POISONING OF WILDLIFE IN SOUTHERN AFRICA*

P.A. BASSON**

ABSTRACT: Basson P.A. **Poisoning of wildlife in Southern Africa.** *Journal of the South African Veterinary Association* (1987) **58** No. 4, 219-228 (En). P.O. Box 81, Grootfontein 9000, South West Africa/Namibia.

Wildlife can be poisoned by both plants and chemicals. The co-evolution of wildlife and toxic plants has resulted in an increased resistance to these substances as compared with domestic animals. Both groups of animals are however susceptible to chemical poisons. The results of experimental poisonings with *Dichapetalum cymosum, Urginea sanguinea, Senecio retrorsus, Nicotiana glauca* and prussic acid are discussed. The effect of poisoning of wildlife with *Crotalaria* spp. *Geigeria spp, Lantana camara,* chlorinated hydrocarbons, organophosphates, carbamates, strychnine, heavy metals and other plants and chemicals is reviewed.

Key words: wildlife, poisoning

INTRODUCTION

Over the past two decades game farming has become increasingly popular as a means of meat production, for the provision of animals either for sale or trophy hunting, and for recreation purposes. The reintroduction of animals and their translocation to strange habitats has become a frequent operation, sometimes with unnecessary or unforeseen problems and losses. These developments occurred in an age of ever increasing environmental pollution which is threatening life in various forms. All these factors highlighted the effects of environmental pollution on wildlife and in particular have focussed our attention on wildlife poisoning.

As with their domestic counterparts, wild animals can be poisoned either by natural toxicants in plants or by unnatural substances such as pesticides, pollutants (including heavy metals) and other chemicals. Also, certain animals are known to be more susceptible to the side-effects of some drugs. However, for the purpose of this communication, pharmaceutically induced poisoning will be excluded.

POISONING BY PLANTS

The co-evolution of wildlife with their natural enemies, be they other animals, micro-organisms, parasites or toxic plants, usually leads to the improvement of specific defence mechanisms up to a point where co-existence is possible. In such a well-balanced ecosystem, local populations are being kept hardy, virile and well-adapted to their surroundings by natural selection. Those that are most susceptible to diseases, parasites and poisonous plants are regularly removed by predators or die before they can reproduce. The relative lack of information in the literature on subjects such as plant poisoning in wildlife, especially in Southern Africa, is therefore not surprising. Cases of such poisoning which have been reported include the following (Table 1):

Steyn described stiffness among some free-living antelopes during years of abundance of *Crotalaria* spp[32]. The numbers and species concerned were not specified. Lewis, Wilson and Hill (1973)[22] recorded three cases of chronic laminitis in the common duiker *(Sylvicapra grimmia)* in Zimbabwe due to *C. barkae.* Grosskopf reported cases of *Geigeria* poisoning in springbok *(Antidorcas marsupialis)*[14]. In one instance they were seemingly so weak that they could be caught by man on foot. Confirmation at autopsy or by histopathological examination was not mentioned. Grosskopf also described cases of bloat in waterbuck *(Kobus ellipsiprymnus)* that died in lucerne fields on the Limpopo river[13]. Various other abundant species such as kudu *(Tragelaphus strepsiceros)*, impala *(Aepyceros melampus)*, steenbok *(Raphicerus campestris)* and bushbuck *(Tragelaphus scriptus)* which grazed in the same fields were never seen to be affected. Myocardial fibrosis was observed in springbok near Potchefstroom that dropped dead while running (Prozesky L, Veterinary Research Institute (VRI), Onderstepoort, unpublished data). These antelopes were confined to overgrazed camps with abundant gousiekte bossie *(Pachystigma pygmaeum).* It seemed evident that the springbok were dying of "gousiekte" under these circumstances. Suspected cases of gifblaar *(Dichapetalum cymosum)* poisoning in kudu occurred under similar circumstances of confinement and overutilization of camps (Naude TW, Faculty of Veterinary Science (FVS), University of Pretoria, unpublished data). Sudden deaths occurred but no carcasses were presented for autopsy. *Lantana camara,* a well-known declared exogenous weed, was suspected of being responsible for photosensitization in gemsbok *(Oryx gazella)* (Coetzer JAW, VRI, Onderstepoort, unpublished data), but again no carcasses were obtained for confirmation.

In a series of experiments in SWA/Namibia since 1974, some of the most toxic plants of Southern Africa were used as models to determine the intake and susceptibility of some indigenous wild ungulates[4 5 26]. Eland *(Taurotragus oryx),* kudu, springbok, gemsbok and one giraffe *(Giraffa camelopardalis)* were used in a comparative study with domestic ruminants. In order to minimise or exclude the effect of stress which could lead to capture myopathy, shock and injuries, the majority of animals were caught and subjected to a period of adaptation which lasted for several months or years. In fact, many of the experimental animals, especially eland, were born in captivity. The antelopes were kept in small camps on natural bushveld and their diets supplemented with horse cubes or lucerne hay. Usually, before each dosing trial, both antelopes and domestic

* Paper delivered at a symposium on the current status of knowledge of wildlife diseases in southern Africa

** P.O. Box 81, 9000 Grootfontein, South West Africa/Namibia

0038-2809/87/04/0219-0228

Table 1: Wildlife poisoning by plants

Animal	Poison	Area	Malnutrition	Mortality	Clinical Signs	Pathology	Confirmed	Reference
Springbok Eland (R + +) Kudu (R + +) Gemsbok	Gifblaar *(Dichapetalum cymosum)*	South West Africa/Namibia		Some	Interrupted recumbency, inappetence, depression, sudden death	Cardiomyopathy Hepatosis Nephrosis Gall bladder oedema	Experimental	5
Kudu	Gifblaar	Farm (C) Transvaal	Yes	Few	Sudden death		No	Naudé T.W. pers. comm. 1987
Springbok (R + +) Eland(R +) Kudu (R +) Gemsbok(?)	Slangkop *(Urginea sanguinea)*	South West Africa/Namibia		Some	Transient diarrhoea, inappetence, listlessness, sudden death	Cardiomyopathy Hepatosis Nephrosis Enteritis	Experimental	5
Springbok Eland(R +) Kudu	*Senecio retrorsus*	South West Africa/Namibia		Some	Wobbly, pushing syndrome, depression	Necrosis and haemorrhage of liver Pulmonary oedema	Experimental	5
Giraffe Springbok Eland (R + +) Kudu (R + +)	Prussic acid	South West Africa/Namibia			Eland: ataxia, imbalance, drowsiness, sedation, sternal recumbancy. Mild spasms rare.		Experimental	5
Gemsbok	*Nicotiana glauca*	South West Africa/Namibia	Yes	Yes	Hypersensitivity Spasms Paresis	 Myocardial, hepatic degeneration	No Experimental	Ebedes H. 1974 unpublished data Basson *et al* 1974 *unpublished* data
Springbok	*Pachystigma pygmaeum*	Farm (C) Potchefstroom	Yes	Numbers unknown	Dropped dead while running	Myocardial fibrosis (sub-endocardial region)		Prozesky L. pers. comm. 1987
Various antelopes	*Crotalaria* spp				"Langklou", stiffness			32
Common duiker	*Crotalaria barkae*	Zimbabwe		3 cases	Elongated hoofs			22,
Springbok	*Geigeria* sp			Not recorded	Weakness Easily caught		No	14
Blou duiker	*Cestrum laevigatum*							37
Gemsbok	Lantana?				Photosensitization		No	Coetzer J.A.W. pers comm. 1987
Waterbuck	Lucerne	Transvaal, RSA		Few	Bloat		Yes	13
Various antelopes (Kudu)	Tannins	Confinement	Yes				Experimental	35, 36

R +, R + + = Increasing degrees of resistance (R)
? = Questionable
C = Camps

animals were starved and withheld from water for approximately 18 - 24 hours. Each animal's mass was accurately determined and, with the exception of a few antelopes, restraint in a crush was preferred to chemical immobilisation in order to exclude possible interactions between immobilising agents and the toxic principles concerned. Dosing was done by stomach tube with the aid of a suction pump. The experiments were conducted in the Mangetti area approximately 150km north of Grootfontein and continued for six years mainly because of the limited number of available antelopes at a specific time. A few antelopes were experimented on in the Etosha National Park and a few sheep at Onderstepoort. The following plants were used in these experiments:

Gifblaar (D. cymosum): This plant contains monofluoroacetate (MFA)[32] which is converted in the body to fluorocitrate, a potent cardiac poison which

blocks the Krebs cycle[27]. Voluntary intake of the plant was determined by offering sublethal doses of specific quantities of counted leaves to starved antelopes. This procedure was followed periodically for more than one year. Initially a remarkable avoidance was noticeable in eland but later, prolonged experiments indicated that in captivity these antelopes will ingest varying quantities of gifblaar leaves. Some individuals were more cautious than others. Kudu, however, were much more wary and their intake was both exceptional and limited. It is noteworthy that the later experiments were conducted after the animals had been in captivity for a few years. By this time some could have lacked parental education and others could have lost some of their avoidance behaviour.

In the dosing trials, because of the prolonged experimental period and the consequent variation of MFA content in the various batches — of which only some 50 per cent were able to be assayed — a comparison between animals was only valid within the same period or whenever the MFA content could be calculated. The total numbers of animals used for the dosing trials were 32 goats, ten eland, four kudu, six springbok and two gemsbok (Table 2). The *c.*LD100 for domestic goats was found to be equivalent to 1,01 - 1,6 mg/kg. Springbok and gemsbok were as susceptible as goats but confirmation is needed for the latter in order to eliminate a possible interaction between MFA and the immobilising drugs which had to be used to make this species more tractable.

Table 2: Mortalities caused by *D. cymosum*

Dosage c.MFA mg/kg	No. of deaths/No. dosed				
	Goats	Springbok	Gemsbok	Kudu	Eland
0,4-0,8	0/4	1/1	–	–	–
1,0-1,1	3/4	1/1	–	–	0/1
1,6	1/1	–	–	–	0/1
2,2-3,5	9/9	4/4	2/2	0/2	0/4
4,5-5,9	5/5	–	–	0/1	0/1
6-8	1/1	–	–	1/1	1/2

The most important lesions were degeneration of the myocardium and liver. Myocardial lesions were usually more frequently encountered in the papillary muscles and subendocardially. Replacement fibrosis occurred in animals that survived for two or more days[3].

Slangkop (Urginea sanguinea): This plant, which contains cardiac glycosides[32], is widespread throughout the country and causes serious losses in domestic stock during early spring. Either the inflorescence or bulbs during various growth stages were used as a blend in tap water. A total of 39 goats, 14 eland, nine kudu, six springbok and four gemsbok were used in the trials (Table 3). The plant material did not always provoke identical reactions and the impression was gained that the early inflorescence and bulbs of the seed-bearing stages were more toxic than the other stages. The *c.*LD50 and *c.*LD100 for goats were 3 - 4 g/kg and 4 - 5 g/kg respectively. A transient diarrhoea and inappetance were noticed in eland and kudu at 5 g/kg but they only started dying at 6 g/kg. One eland died at 7 g/kg without showing any diarrhoea. Springbok showed no symptoms even at their maximum dose of 6 g/kg. The gemsbok that died (at 2,5 - 4 g/kg) showed complications of shock and capture myopathy. One of these gemsbok (4 g/kg) had a diarrhoea. However, another gemsbok which received a higher dosage of 5 g/kg did not develop any symptoms.

It was evident that the resistant antelopes have either a superior ruminal degradation or superior systemic detoxification or both.

The most important lesions caused by slangkop were confined to the heart and could not be distinguished from those caused by gifblaar[3]. Subacute to chronic lesions of replacement fibrosis were encountered in animals that survived for more than six days[3,5].

Senecio retrorsus: Finely ground dried material obtained from the Eastern Cape was dosed to eight sheep, six goats and six eland (both males and females), and to one kudu heifer and one springbok ram (Table 4). All five sheep given 5 g/kg died but none of the goats even at 8 g/kg succumbed. One eland heifer and one young eland bull were given 8 g/kg. The heifer became wobbly five days after treatment, showed the pushing syndrome and died on the same day. Both kudu (8,7 g/kg) and springbok (7 g/kg) died on day three post dosing. Lesions typical of *Senecio* poisoning were found both macro- and microscopically[17]. It was therefore established that whereas the *c.*LD50 for sheep was between 2,5 - 5 g/kg (Table 4), the MLD for goats was more than 8 g/kg and that of eland about 8 g/kg.

Senecio spp. are well-known for their production of pyrrolizidine alkaloids. In general some ruminal degradation of alkaloids can occur in animals. Pyrrolizidine alkaloids, which as such are nontoxic, are, after absorption, converted in the liver by microsomal enzymes to pyrroles which are hepatotoxic[12]. Rapid pyrrole production is therefore also correlated with increased susceptibility. It is of interest to note that Dean and Winward (1974) (quoted by Fowler[12]) have also reported that black-tailed deer *(Odocoileus hemionus columbianus)* can consume large quantities of *Senecio* without ill-effects.

Prussic acid (HCN): Potassium cyanide powder was used as a source of HCN because of the practical problems in procuring fresh plant material with adequate cyanogenetic glycosides. Thirteen cattle, two sheep, 28 goats, 20 eland, two kudu, five springbok and one giraffe were given dosages of calculated HCN ranging from 2 - 7 mg/kg. For fear of losing some of the limited number of animals, an antidote consisting of an aqueous solution of 25% sodium thiosulphate and 2,5% sodium nitrite was invariably given by intravenous route (40 ml/200 kg) during severe convulsive stages or sometimes also in animals with milder clinical signs (Table 5). The onset of clinical signs was slower in those animals such as the eland which were less susceptible. It varied from 1 - 6 minutes (*ca.*2,6) in cattle at 2 - 4 mg/kg, 2 - 15 minutes (*ca.* 4,2) in goats at 3,7 - 7 mg/kg and 4 - 18 minutes (*ca.* 8,4) at 4 - 7 mg/kg in eland. The clinical signs noticed included lip-licking, chewing, salivation, foaming at the mouth, drowsiness, ataxia, imbalance, respiratory distress, recumbency, severe convulsions or intermittent spasms. Severe spasms were commonly seen in cattle at 3 mg/kg and in goats at 5 - 7 mg/kg but only once in one eland at 6 mg/kg. Eland were more inclined to show ataxia, imbalance, drowsiness, sedation and sternal recumbency. Recovery

Table 3: Mortalities caused by *U. sanguinea*

Growth stage	Parts dosed	Dosage g/kg	No. of deaths/No. dosed				
			Goats	Eland	Kudu	Springbok	Gemsbok
Early flowering	Bulbs	2	0/5	–	–	–	–
Early flowering	Bulbs	2,5	–	0/1	0/1	–	1/1?
Late flowering	Bulbs	3	3/8	–	–	0/3	–
Early flowering	Inflorescence	3	0/3	–	–	–	–
Early flowering	Inflorescence	4	7/8	0/3	0/3	0/1	2/2?
50% Flowering	Bulbs	4	3/6	0/1	0/1	–	–
50% Flowering	Bulbs	5	5/5	0/5	0/1	–	–
Early flowering	Inflorescence	5	–	–	0/1	–	0/1
Seed-bearing	Bulbs	6	4/4	2/3	2/2	0/2	–
Seed-bearing	Bulbs	7	–	1/1	–	–	–

? Complications : Shock, capture myopathy.

Table 4: Mortalities caused by experimental *S. retrorsus* poisoning

Dosage mg/kg	No. of deaths/No. dosed				
	Sheep	Goats	Eland	Kudu	Springbok
2,5	0/1	–	–	–	–
5	5/5	0/2	–	–	–
6	2/2	0/1	–	–	–
7	–	0/1	0/4	–	1/1
8	–	0/2	1/2	–	–
8,7	–	–	–	1/1	–

with high dosages, however, sometimes took an hour or more. One eland died two hours after a dosage of 7 mg/kg. Three eland (at 6 - 7 mg/kg), which received the antidote 19-245 minutes after dosing, took longer than the other animals to recover completely (45 - 50 minutes). Results from the other animals used are rather inconclusive because of their limited numbers, but indications were obtained that kudu and springbok are less susceptible than cattle. Antidotes were probably administered too soon in both springbok and giraffe because losses were feared in their very limited numbers. The HCN levels in the blood showed some correlation with the duration of clinical signs but none with the dosage or severity of clinical signs.

Plants which may cause HCN poisoning are known to contain cyanogenetic glycosides which, by a process of hydrolysis under the action of ruminal organisms, enzymes liberated from macerated plant material and suitable pH, liberate cyanide[42]. Free HCN, however, can also occur in plants that are stunted, wilted or damaged by frost, hail or trampling[32]. It is also of interest to note that some herbicides could increase toxicants such as cyanogenetic glycosides in plants[42].

Resistance to cyanogenetic glycosides could imply either a very rapid systemic detoxification by conversion to thiocyanate or a beneficial ruminal process whereby microbial hydrolysis is retarded or by both. As potassium cyanide instead of plant material with cyanogenetic glycosides was used, the results need not be a true reflection of the susceptibility of antelopes to the natural compounds. It can merely be concluded that some antelopes, such as eland, have a more efficient systemic detoxification process than domestic ruminants.

Wild tobacco (Nicotiana glauca): This commonly occurring introduced poisonous plant[32] was suspected of causing mortalities in gemsbok in the western regions of SWA/Namibia (Ebedes H, National Zoological Gardens, Pretoria, 1974, unpublished data). Consequently plant material was collected and a blend prepared with tap water and dosed to two gemsbok (Basson P A, Ebedes H and Norval A G, 1974, unpublished data). Muscular twitching, hypersensitivity and spasms were noticed approximately 5 - 10 minutes after dosing. Respiratory and cardiac arrest occurred within 30 minutes in an antelope that received 5 g/kg of the plant material. The other gemsbok at 2 g/kg developed posterior paresis and torticollis with sporadic spasms. It died approximately 17 hours after dosing.

The most important pathological findings were degenerative changes in the myocardium and liver, glial swelling and mild status spongiosus of the brain, haemorrhages and oedema of the urinary bladder, abomasal oedema, and kariorrhexis in the spleen and lymph nodes. Disseminated intravascular coagulopathy in the myocardium associated with necrotic fibres was a striking feature in the gemsbok that had survived for 17 hours.

Other secondary plant compounds: Stahl (1888) (quoted by Fowler[12]) introduced the concept that plants could produce compounds, other than direct toxicants, which serve as defence mechanisms against herbivores. Essential oils as well as other bacteriostatic compounds which are highly unpalatable and which interfere with digestion were given as examples[12]. In South Africa, Van Hoven[35 36] proved that bushes can increase their production of unpalatable tannins in response to overutilisation. The tannins react with digestive enzymes and proteins rendering them either inactive or indigestable and in this manner cause indigestion and star-

Table 5: Number of animals experimentally dosed with potassium cyanide powder which showed spasms and number which were given antidotes (AD)

Dosage mg/kg	No. with spasms/No. dosed (No. given AD)						
	Cattle	Sheep	Goats	Springbok	Giraffe	Kudu	Eland
2-2,5	0/1	0/1					
2,8	1/2(1)						
3-3,7	6/9(6)		0/7(2)	0/2	0/1(1)	0/1	0/3
4	1/1(1)	1/1(1)	1/7(4)	1/2(2)		0/1	0/2
4,8			0/1(1)				
5			5/6(5)	0/1(1)			0/6
6			3/6(3)				1/7(2)
7			2/2(2)				0/2(1)

vation. Confinement and overstocking of browsers and browser-grazers could therefore lead to starvation and death in spite of an adequate food supply. Although this need not necessarily be regarded as direct poisoning *per se*, such chronic effects and indirect ways of affecting the well-being of herbivores need more emphasis.

POISONING BY MYCOTOXINS

Only one record was obtained where two cases of suspected ergotism were diagnosed in impala in the Midmar Game Camp, Natal (Lewis AR, Natal Parks Board, 1976, unpublished data). However, only *Claviceps paspali* and not *C. purpurea* was identified from very heavily parasitised *Paspalum dilatatum*, a very common species in the Park.

POISONING BY BACTERIAL TOXINS

Under anaerobic conditions *Clostridium botulinum* type C can produce toxins in carcass and other organic material in water, which can poison waterbirds such as wild ducks and wild geese[16,34]. The toxins, which are probably contained within the bacteria, cause paralysis and polydipsia in birds. They usually lie with outstretched necks and legs and drooping wings. Paralysis of the nictitating membranes is apparently a very characteristic feature. The toxin can also cause malfunctioning of the supraorbital gland because of its blocking action on the facial nerve. The gland has an extrarenal excretory capacity which, with a small dose of toxin, can easily be overloaded. In this way death can be precipitated by excessive salt. Intravenous treatment with antiserum seems to be very effective[16].

POISONING BY PESTICIDES, POLLUTANTS AND OTHER CHEMICALS

Poisoning of wild animals by unnatural substances (Table 6) is more important than poisoning by natural toxicants contained in plants because animals usually lack resistance against these substances. Knowing that some animals have an innate resistance to MFA, the commercial product 1080 is one of the exceptions in this respect.

Chlorinated hydrocarbons (CHC) are well-known, persistent, poorly degradable pesticides whose residues accumulate in an ecosystem[6]. Their effects are particularly noticeable in birds of prey that are at the top of a food chain. The worldwide decline in raptor populations due to direct mortality or indirect effects such as increased fragility of eggs, terminal death of embryos and a delay in onset of breeding due especially to DDE, the principal metabolic product of DDT, is well-documented[25,29].

In South Africa, Wiese and Basson[39], Wiese *et al*[41] and Basson[2] studied the effects of certain levels of dieldrin and photodieldrin on birds such as guinea-fowl *(Numida meleagris)* and established that they are more susceptible to these compounds than domestic fowl. Wiese *et al*[40] also described very high mortality rates in blesbok *(Damaliscus dorcas)* and springbok on fields sprayed with dieldrin for termites. Typical acute clinical signs were described as well as a dumb syndrome characterized by stupor, lack of fear and apparent blindness. Myocardial and muscular lesions as well as glial swelling and brain oedema were reported. It was concluded that these antelopes are more susceptible to these compounds than any vertebrates reported on previously (Table 8). Rams were also more susceptible than ewes. Clinical signs described in birds with CHC poisoning were inappetance, tremors, incoordination, spreading of the rectrices, spasms, salivation and muscular dystrophy[25,39]. Dieldrin proved to be more powerful than DDT in delaying the onset of breeding in birds[29]. Such a delay consequently reduces the chances of reproductive success. Records of CHC poisoning, especially in raptors, occur worldwide, including many in Southern Africa[6,16,30,33,38].

The polychlorinated biphenyls (PCB), which are widely used in industry as plasticizers and in paints and lubricants, resemble DDT in molecular structure and produce similar physiological actions in animals[29]. They can also cause thinning of egg shells but, as with dieldrin, are more effective in delaying the onset of breeding. The PCBs are also released when plastic materials are burned and are consequently widely distributed over the earth.

Organophosphors (OP), although more easily degradable than the CHCs, can cause acute deaths and high mortalities in birds and mammals[20]. In sheep-farming areas, some farmers use concentrated insecticides such as diazinon on carcasses in order to control blowflies. By doing so many scavenging birds and mammals can be killed. Periodic mortalities of grain-eating wild birds by different OP pesticides in various parts of Southern Africa have been encountered from time to

Table 6: Wildlife poisoning by pesticides

Animal	Poison	Area	Mortality	Clinical Signs	Pathology	Confirmed	Reference
Blesbok Springbok	Chlorinated hydrocarbons (Dieldrin Photo-dieldrin)	Republic of South Africa	High (100%)	Violent form: excitement, spasms Dumb form: Fearlessness blindness	Muscular and myocardial degeneration Brain swelling Degeneration of liver and kidney	Yes	40
Birds	Chlorinated hydrocarbons	Zimbabwe				Yes	38
Guinea-fowl	Dieldrin	Transvaal		Inappetence, incoordination, spreading of rectrices, seizures, salivation. Eggs: low chick survival		Experimental	39, 41
Herons, raptors	Chlorinated hydrocarbons	Transvaal					30
Birds	Dieldrin, Photo-dieldrin					Experimental	2
Guinea-fowl	Organophosphors	South West Africa	Many			Yes	Grant R, Basson P.A. Unpublished data
Spurwing	Organophosphors ("Monocrotophos")	Natal	31			Yes	Bath G 1987 Pers. comm.
Egyptian goose, guinea-fowl, dove, crowned crane, francolin, redbilled teal, sacred ibis	Organophosphors ("Monocrotophos")	Natal	2 7 +			Yes	Bath G 1987 Pers. comm.
Coot	Organophosphors ("Triazophos")	Natal	1			Yes	Bath G 1987 Pers. comm.
Vultures	Organophosphors ("Parathion")	Natal	2			Yes	Bath G 1987 Pers. comm.
Geese, Guinea-fowl	Organophosphors ("Diazinon")	Natal	+			Yes	Bath G 1987 Pers. comm.
Vultures	Organophosphors ("Dioxathion")	Kruger National Park	c.50				Kellerman T.S. 1987 Pers. comm.
Birds (Blue Crane)	Organophosphors ("Diazinon")	Cape	19				Schneider D.J. 1987 Pers. comm.
Vultures	Organophosphors	Zimbabwe	>60			Yes	21
Birds Elephant Zebra Giraffe Jackal Fish Frogs etc.	Carbamate (Aldicarb) ("Temik")	Transvaal (River)	5 1 2 1 6 Many Many	Found dead near or in water.		Yes	Loock T.J. 1987 Pers. comm.
Vultures	Carbamate (Carbofuran) ("Curaterr")	Caprivi	250			Yes	20
Raptors, scavenging birds, brown hyaena, honey badgers, bat-eared foxes	Strychnine					Yes	20
Vultures	Strychnine	RSA, SWA	>44			Yes	8, 9, 31
Vultures	Strychnine	Botswana	>100			Yes	20

Vultures	Strychnine	Transvaal	37			Yes	20
Wahlbergs eagle	Strychnine	Transvaal	1			Yes	10
White-backed Vulture	Cyanide	Kruger National Park	c.50			Yes	Kellerman T.S. 1987 Unpublished data
Cape vulture Tawny eagle Bateleur eagle Spotted Hyaena Black-backed Jackal	Cyanide	Kruger National Park	1 2 1 1 Several			Yes	Kellerman T.S. 1987 Unpublished data
Vultures, crows (Feeding on primary victims)	Monofluoroacetate (1080, "Blougif")						21
Eland	Arsenic					Yes	Basson P.A. 1972 Unpublished data
Black rhino	Creosote			Weals on skin	Hepatic necrosis Gastric ulcers		43
Grants gazelle Dorcas gazelle	Corrosive weedkiller used to eradicate kikuyu grass	Zoo	Few	Necrobacillosis	Necrosis: Buccal cavity, rumen, liver	Yes	Basson P.A. and Smit J. (1970) Unpublished data
Rock Lobster	Organic pollution (fish factories)	Western Cape	Thousands			Yes	24
Fish	Organic pollution (fish factories)	Western Cape	Many			Yes	24
Birds	*Clostridium botulinum* type C toxin	Transvaal Orange Free State Western Cape		Paralysis, polydipsia, paralysis of nictitating membrane		Yes	16, 34 Schneider D.J. (1987) Pers. comm.
Waterfowl	*Oil*			*Incoordination* Tremors	*Lipid pneumonia*, fatty liver, *nephrosis*, gastro-enteritis	Experimental	15
Impala	Ergot?	Natal	2	Limping	Necrosis lower extremities	No	Lewis A.R. 1976 unpublished data

Table 7: Susceptibility of animals to various poisons

	MFA (mg/kg)	Urginea (g/kg)	HCN (mg/kg)	Senecio (g/kg)
Sheep	0,25-0,5[4]	–	2-2,3[4]	5[6]
Goat	0,3 -0,7[4] 1,0 -1,6[6]	3-4[5]	3-6[3]	8[1]
Cattle	0,25-0,6[4]	–	3[3]	–
Eland	6-8[2]	6[2]	6-7[3]	8[2]
Kudu	6-8[2]	6[2]	4[1]	±8?[2]
Giraffe	–	–	±3?[1]	–
Springbok	±0,84[2]	6[1]	±4-5[3]	±7?[2]
Gemsbok	±0,7[2]	5?[1]	–	–

? Only one animal dosed or complications encountered.
1 = Nil deaths 3 = anticipated MLD 5 = c.LD 50
2 = Onset of death (MLD) 4 = LD 50 6 = c.LD 100

time over many years. Poisoning is carried out by contaminating grain with various OP pesticides, especially parathion, and then putting it out for the birds to eat. This intoxication has been repeatedly confirmed in guinea-fowl, Egyptian geese *(Alopochen aegyptiacus)*, blue cranes *(Anthropoides paradisea)* and other wild birds (Kellerman T S, VRI, Onderstepoort, 1987, pers comm). In a recent incident in the northern Kruger

Table 8: Arbitrary orders of susceptibility to some toxicants (high to low)

Poison	5	4	3	2	1
Dieldrin	Pheasant	Guinea-fowl	Domestic fowl		
Dieldrin and photodieldrin	Blesbok	Springbok	Domestic ruminants		
Gifblaar	Dog	Domestic Ruminants, Springbok			Eland, Kudu
Slangkop	Goats, sheep			Eland, Kudu Gemsbok	Springbok
Senecio	Horses	Cattle, Sheep	Springbok	Eland, Kudu Goats?	Goats
Prussic Acid	Cattle, Sheep	Giraffe	Springbok Goat	Eland, Kudu	

National Park, about 50 vultures were found dead in a deliberate "muti"-killing and the OP dioxathion was found in the possession of the person suspected of causing the incident (Kellerman T S, VRI, Onderstepoort, 1987 pers comm). When the control of redbilled quelea finch *(Quelea quelea)* swarms is deemed necessary, the Department of Agriculture uses OPs by extensive aerial application to the swarms from aircraft. This is done at night when the swarms are sleeping. Initially parathion was used but now fenthion methyl ("Queletox") is used instead. This particular OP has the advantage over parathion that it is more toxic to birds than mammals and is furthermore more easily adsorbed onto organic matter, which inactivates it (Bot J, Plant Protection Research Institute, Pretoria, 1987, pers comm). Fenthion methyl is extensively used as, amongst others, the agricultural remedy "Lebaycid" and as the veterinary ectoparasiticides "Tiguvon" and "Bayopet Spotton".

Carbamates are generally not as poisonous as OPs although some extremely toxic compounds are available and have caused severe mortalities. Carcasses are also treated with carbamates such as carbofuran ("Curaterr") in order to control predators (Naude T W, FVS, Onderstepoort, 1987, pers comm). Ledger[20] quoted one example in the Caprivi where 250 vultures were killed.

The injudicious use of strychnine to control predators is suspected to be a major cause of the near extinction of the Cape vulture *(Gyps coprotheres)* in SWA/Namibia[6, 9]. In one incident in Botswana 100 vultures were killed by strychnine stuffed into a carcass[7]. Clinical signs reported were regurgitation, extension of the necks and aggressive behaviour. Deaths of other scavenging birds and mammals and even birds not known as carrion-eaters, such as the Wahlberg's eagle *(Aquila wahlbergi)*, have also been recorded[10].

"Blougif" (1080) (commercial MFA) is odourless and tasteless, extremely stable, and notorious for poisoning scavengers when placed in carcasses. Vultures or crows feed on animals that have died of such poisoning and may be secondarily poisoned[21].

Heavy metals such as lead, mercury and cadmium may be so concentrated in industrial wastes that they present a hazard to both wildlife and man[25]. Alkyl-mercury compounds previously used as fungicides in seed dressings are more poisonous because of their solubility in both water and fat. In Europe, widespread mortalities occurred mainly in granivorous birds but also in raptors due to secondary poisoning[25]. It was also established that mercury could reduce the production and hatchability of eggs and the viability of chicks. Cases of mercurial poisoning in wildlife in Southern Africa could not be traced, but one case of arsenical poisoning was diagnosed in an eland (Basson P A, 1972, unpublished data).

A serious outbreak of cyanide poisoning in the Kruger National Park has been confirmed recently where cyanide was put in a carcass in order to kill vultures (Kellerman T S, VRI, 1987, pers comm). Approximately 50 vultures were killed as well as some raptors and predators.

An unspecified corrosive weedkiller, used to eradicate kikuyu grass in the Pretoria Zoo, caused buccal and pharyngeal lesions with secondary necrobacillosis in Grant's gazelle *(Gazella granti)* and dorcas gazelle *(Gazella dorcas)* (Basson P A and Smit J, 1970, unpublished data).

A suspected fatal case of creosote poisoning in a black rhino *(Diceros bicornis)* has been reported in the Transvaal[43].

Organic pollution from fish factories evidently caused the death of thousands of rock lobsters *(Jasus lalandii)* in the Western Cape[24]. This was seemingly due to a drastic depletion of oxygen caused by the degradation of accumulated organic matter. Toxicity of oil to waterbirds involves external oiling as well as ingestion of oil during preening[15]. It was established experimentally that lipid pneumonia, gastro-intestinal irritation, fatty hepatosis and toxic nephrosis were some of the most common lesions in such cases.

DISCUSSION

In order to establish the susceptibility of wild animals to poisonous plants of Southern Africa, Basson *et al*[5] have dosed 221 individual animals over a period of six years under difficult circumstances in the field. Although the numbers in some of their trials were still inadequate, valuable information was obtained (Table 7). It became apparent that, wherever co-evolution with toxic plants existed, antelopes usually showed some superior detoxifying mechanisms or innate resistance. It is of interest to note that gifblaar areas generally fall within the natural habitat of both eland and kudu which are mainly browsers or browser-grazers and which proved to have a relatively high resistance against MFA. On the contrary springbok, which are as susceptible as goats, occur in habitats where gifblaar is absent. They are, however, very resistant to slangkop which is found in semi-arid as well as subtropical areas. Various farmers have also reported that the common duiker, steenbok, springhare

(Pedetes capensis) and porcupine *(Hystrix africaeaustralis)* feed on the bulbs of slangkop and other bulbous plants without being poisoned.

In Australia, very interesting work has been done on MFA by Oliver[27, 28], King[18, 19], Mead[23] and Aplin[1]. They studied the metabolism and detoxification of MFA and established, amongst others, that the substance is only detoxified systemically and not by ruminal degradation. Susceptibility of various indigenous animals was determined by administering MFA systemically and subsequently measuring the plasma citrate levels[27]. This was a superior and more reliable method than the one used by Basson *et al.* By these means the co-evolutionary role of MFA in plant-animal interactions was studied and resistance to the compound used as a genetic marker to trace the evolutionary history in their country. Several animals such as the brush-tailed possum *(Trichosurus vulpecula)* from Western Australia where MFA-producing plants occur, were 150 times more resistant than the same species in Southern Australia.

In an excellent review of plant poisoning in free-living wild animals, Fowler (1983)[12] described various factors influencing the susceptibility of wild animals to toxicants. They can cope with these toxicants by avoidance, by eating many other plants and thus diluting the toxicants, by ruminal degradation and by systemic detoxification. Genetically controlled food identification, parental education, interaction with con-specifics, own experience and availability of adequate food supply are all factors that play a role. Starved animals are more likely to consume toxic plants than those with adequate food supply. Also well-nourished animals have a healthier and more balanced gastro-intestinal microflora to assist them in the degradation of toxicants than under-nourished animals or those on maintenance rations. Ingestion of bacteriostatic compounds will consequently also have an inhibitory effect on micro-organisms and thus indirectly increase the susceptibility of animals where ruminal degradation of toxicants plays an important role.

Fowler further stated that effective detoxification is dependant on adequate periodical microsomal stimulation[12]. The translocation of wildlife or plants from their natural habitats to strange surroundings and the confinement of animals to smaller areas than they were used to, are therefore fraught with many dangers. Not only may wild animals ingest strange toxicants and die as a result of a lack of proper defence mechanisms, but inadequate stimulation or exercise of the systems or processes involved in detoxification can lead to poisoning should these animals subsequently be exposed to more poisonous plants. This means that either ruminal degradation or systemic detoxification can become less effective with inadequate stimulation. In this respect it is of special interest to mention the successful ruminal degradation by specific bacteria of the toxicant mimosine, contained in *Leucaena leucocephala* which is an indigenous plant in South America where poisoning of domestic stock by this plant is unknown (Jones R, CSIRO, Townsville, Australia, pers comm). However, poisoning was experienced where this high protein-yielding shrub has been cultivated in Australia[11].

Young animals with poorly developed microsomal systems are more susceptible than adults, and stress factors which are invariably imposed on captured animals can also lead to inadequate detoxification[12].

Research both overseas and in Southern Africa has indicated that, apart from behavioural safeguards, wild animals usually have either an innate resistance to or very efficient ruminal degradation of natural toxicants occurring in their habitats. These features, together with other beneficial factors such as hardiness and more efficient and diverse use of plants (especially by browsers), make them ideal animals in their own domain. This is so because of strong long-term natural selection.

Poisoning of wildlife by pesticides, pollutants and other chemicals is currently of more importance than poisoning by natural toxicants because wild animals lack resistance against these substances. Over the past decades it has therefore become increasingly clear that, in the production of chemicals for the control of parasites and diseases, preference should be given to those that disturb the eco-systems the least. These compounds should be more parasite specific and preferably easily degradable. Education of the public in their use should continue but this is no safeguard. Naturally more emphasis is needed on biological conrol. At a recent symposium on the "Use/Misuse of Poison in our Farming Community" in SWA/Namibia, the following question was posed: "Do we want a monoculture of humans and sheep or a variety of life-forms?" (Van Heerden, 1986, quoted by Ledger[21]). It may well be asked whether we want life at all if we refuse to consider pollution in general as a serious worldwide hazard.

It is evident that further research is required on toxicological aspects of wildlife. By doing so and applying our knowledge to restocking and translocation of animals, mortalities will be minimized.

ACKNOWLEDGEMENTS

To all those colleagues whose unpublished data were used in order to make this effort worthwhile, a special word of thanks. The assistance of Professor T W Naude, FVS, Onderstepoort, and Dr T S Kellerman, VRI, Onderstepoort in reviewing and Miss K Hare, Veterinary Technician, Grootfontein, for editing and typing the manuscript is equally gratefully acknowledged.

REFERENCES

1. Aplin T E H, King D R, Olivier A J 1983 The distribution and ecology of the toxic species of *Gastrolobium* and *Oxylobium* in South-Western Australia in relation to the tolerance of native animals to fluoroacetate. Toxicon, Suppl. 3:21-24
2. Basson N C J 1971 Effect of dieldrin and its photoisomerization product 'photodieldrin' on birds. Phytophylactica 3:115-124
3. Basson P A 1987 Acute and chronic cardiomyopathies caused by monofluoroacetate in gifblaar *(Dichapetalum cymosum)* and cardiac glycosides in slangkop *(Urginea sanguinea)*. Journal of the South African Veterinary Association (in press)
4. Basson P A, Norval A G, Hofmeyr J M, Ebedes H, Schultz R Anitra 1982 Antelope and poisonous plants: 1. Gifblaar *Dichapetalum cymosum* (Hooker) Engler and Prantl containing monofluoroacetate. Madoqua 13(1):59-70
5. Basson P A, Norval A G, Hofmeyr J M, Ebedes H, Schultz R Anitra, Kellerman T S, Minne J A 1984 Antelopes and poisonous plants. Proceedings of XIIIth World Congress on Diseases of Cattle, Durban, Republic of South Africa, Vol 2:695-701
6. Billing K J, Phelps R J 1972 Records of chlorinated hydrocarbon pesticide levels from animals in Rhodesia. Proceedings and Transactions of the Rhodesia Science Association 55:6-9
7. Boshoff A 1985 Mass poisoning of vultures in Botswana. Vulture News 13:13
8. Brown C J 1985 The status and conservation of the Cape Vulture in SWA/Namibia. Vulture News 16:10-20
9. Brown C J 1986 Biology and conservation of the Lappetfaced Vulture in SWA/Namibia. Vulture News 16: 10-20.
10. Carlyon J 1985 Wahlberg's eagle dies of strychnine poisoning. African Wildlife 39(6):239-241

11. Everist Selwyn L 1981 Poisonous plants of Australia. Angus and Robertson Publishers: 460-462
12. Fowler M E 1983 Plant poisoning in free-living wild animals: A review. Journal of Wildlife Diseases 19(1):34-43
13. Grosskopf J F W 1964a Advances in research on acute frothy bloat in ruminants. Journal of the South African Veterinary Medical Association 35(2):169-178
14. Grosskopf J F W 1964b Our present knowledge of "Vermeersiekte" *(Geigeria* poisoning). Technical Communication No 21. Department of Agricultural Technical Services, Onderstepoort
15. Hartung R, Hunt G S 1966 Toxicity of some oils to waterfowl. Journal of Wildlife Management 30(3):564-570
16. Hay Cheryl M E, Van der Made H N, Knoetze P C 1973 Isolation of *Clostridium botulinum* type C from an outbreak of botulism in wild geese. Journal of the South African Veterinary Association 44(1):53-56
17. Jubb K V F, Kennedy P C, Palmer N 1985 Pathology of domestic animals, Vol 2:296-298
18. King D R, Oliver A J, Mead R J 1978 The adaptation of some Western Australian mammals to food plants containing fluoroacetate. Australian Journal of Zoology 26:699-712
19. King D R, Oliver A J, Mead R J 1981 *Bettongia* and fluoroacetate: A role for 1080 in fauna management. Australian Wildlife Research 8:529-536
20. Ledger J 1985 The poison people. African Wildlife 39(1):6-8
21. Ledger J 1986 "Superjakkals" and the poison people. African Wildlife 40(3):85-89
22. Lewis A R, Wilson V J, Hill R R 1973 Crotalariosis in the common duiker *(Sylvicapra grimmia)*. Rhodesian Veterinary Journal 4:29-33
23. Mead R J, Oliver A J, King D R 1979 Metabolism and defluorination of fluoroacetate in the Brush-tailed possum *(Trichosurus vulpecula)*. Australian Journal of Biological Science 32:15-26
24. Newman G G, Pollock D E 1973 Organic pollution of the marine environment by pelagic fish factories in the Western Cape. South African Journal of Science 69:27-29
25. Newton I 1979 Population ecology of raptors. T & A D Poyser, Berkhamsted, England: 229-262
26. Norval A G, Basson P A 1974 Eland avoid gifblaar. Journal of the South African Veterinary Medical Association 45:233
27. Oliver A J, King D R, Mead R J 1977 The evolution of resistance to fluoroacetate intoxication in mammals. Search 8(4): 130-132
28. Oliver A J, King D R, Mead R J 1979 *FLuoroacetate tolerance*, a genetic marker in some Australian mammals. Australian Journal of Zoology 27:363-372
29. Peakall D B 1970 Pesticides and the reproduction of birds. Scientific American 218(10):255-261
30. Peakall D B, Kemp A C 1976 Organochlorine residue levels in herons and raptors in the Transvaal. Ostrich 47(2 & 3):139
31. Smit W 1984 Cape Vultures poisoned in the Eastern Cape, S.A. Vulture News 12:5-7
32. Steyn D G 1949 Vergiftiging van mens en dier. J L van Schaik, Pretoria: 68,131
33. Thomson W R 1984 DDT in Zimbabwe. International Council for Bird Preservation, Bulletin 1:41-47
34. Van Heerden J 1974 *Botulism in the Orange* Free State Goldfields. Ostrich 45:182-184
35. Van Hoven W 1984a Bome kom in verset teen plundery van koedoes. Tukkie-Werf (Sept.): 4-6.
36. Van Hoven W 1984b Tannins and digestibility in greater kudu. Canadian Journal of Animal Science 64 (Suppl.):177-178
37. Von Ketelhodt H F 1977 The lambing intervals of the blue duiker *Cephalophus monticola* Gray, in captivity, with observations on its breeding and care. South African Journal of Wildlife Research 7(1):41-43
38. Whitwell A C, Phelps R J, Thomson W R 1974 Further records of chlorinated hydrocarbon pesticide residues in Rhodesia. Arnoldia Rhodesia 6(37):1-8
39. Wiese I H, Basson N C J 1967 The oral toxicity of dieldrin to the crowned guinea-fowl, *Numida meleagris* (L). South African Journal of Agricultural Science 10:697-706
40. Wiese I H, Basson N C J, Basson P A, Naude T W, Maartens B P 1973 The toxicology and pathology of dieldrin and photodieldrin poisoning in two antelope species. Onderstepoort Journal of Veterinary Research 40(1):31-40
41. Wiese I H, Basson N C J, Van der Vyver J H, Van der Merwe J H 1969 Toxicology and dynamics of dieldrin in the crowned guinea-fowl, *Numida meleagris* (L). Phytophylactica 1:161-176
42. Williams M C, James L F 1983 Effects of herbicides on the concentration of poisonous compounds in plants: A review. American Journal of Veterinary Research 44(12):2420-2422
43. *Young E (Editor)* 1973 *The capture* and care of wild animals. Human and Rousseau, Cape Town & Pretoria: 159