

THE TOXICOLOGY AND PATHOLOGY OF DIELDRIN AND PHOTODIELDRIN POISONING IN TWO ANTELOPE SPECIES

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ABSTRACT

WIESE, I. H., BASSON, N. C. J., BASSON, P. A., NAUDÉ, T. W. & MAARTENS, B. P., 1972. The toxicology and pathology of dieldrin and photodieldrin poisoning in two antelope species. *Onderstepoort J. vet. Res.* 40(1), 31-40 (1973)

Blesbuck (*Damaliscus dorcas phillipsi*) and springbuck (*Antidorcas marsupialis*) were found to be more susceptible to dieldrin and its photoisomer than any vertebrates reported on previously in the literature. In blesbuck the mean cumulative lethal dose of dieldrin at various dietary levels was 9,07 mg/kg and of photodieldrin 1,90 mg/kg. Under field conditions the calculated median lethal dose of photodieldrin to blesbuck was 1,21 mg/kg; to springbuck it was 3,97 mg/kg. In both species rams were more susceptible to photodieldrin than ewes. It was proved that antelope mortalities on veld sprayed with dieldrin for harvester termite (*Hodotermes mossambicus*) control was principally due to photodieldrin intoxication.

In addition to the usual violent nervous signs encountered in chlorinated hydrocarbon intoxication a dumb syndrome, in which blindness was rather characteristic, was seen, especially in the field cases.

The pathological changes in the blesbuck and springbuck were similar. Degenerative lesions typical of various conditions, including chlorinated hydrocarbon poisoning, were encountered in the skeletal muscles, myocardium, kidneys and liver of cases of both dieldrin and photodieldrin poisoning. The severity and widespread nature of the lesions in the skeletal muscles and myocardium of natural cases were reminiscent of white muscle disease and capture myopathy. Congestion, oedema and glial swelling of the brain were constant features.

INTRODUCTION

The aerial application of dieldrin as a cover spray at the rate of 87 g/ha for the control of the harvester termite, *Hodotermes mossambicus* (Hagen, 1853), is currently registered as an approved procedure with the South African Department of Agricultural Technical Services. This level provides a wide safety margin for sheep and cattle (Wiese, Minne & Naudé, unpublished data) and permits the raising of healthy calves and lambs even when the recommended 9-month stock-withdrawal period is ignored (Wiese, Basson & Van der Merwe, 1970).

During the past few years, however, isolated reports of blesbuck (*Damaliscus dorcas phillipsi* Harper, 1939) mortalities following dieldrin spraying have come to the notice of the Department of Agricultural Technical Services. In all authenticated cases it was established that the aircraft made use of landing strips in camps being sprayed and during loading operations some spillage of the emulsifiable concentrate, which was used until 1967 and which is reputedly attractive to game, might have occurred. As this explanation was not altogether satisfactory, twelve blesbuck were placed on an experimental site in the Lydenburg district which was commercially sprayed in July 1967. During the spraying of the 500 ha area the buck, which had only been on the site for 6 weeks, took fright and ran amok, sustaining multiple injuries from charging into barbed-wire fences and gullies. Within a week of spraying all the buck were dead. *Post mortem* investigation showed signs of myocardial degeneration while analysis of various organs revealed very low dieldrin residues. It was accordingly inferred that the buck had died of injuries, exhaustion and shock.

During the winter of 1968 only one incident of game mortality was reported from veld subjected to harvester

termite control. This was in a camp of 80 ha which had been sprayed at the recommended rate with the new ultra-low volume formulation, hence eliminating the possibility of gross contamination by the emulsifiable concentrate. Fifteen blesbuck were present in the camp during spraying. A week later one buck was found dead in the camp. The site was visited on the 28th day after spraying and in the preceding week a further eight buck had died. According to the owner the buck appeared to be in a stupor without any fear of humans and apparently blind. Some had charged through the barbed-wire fence of the camp and had been badly mutilated. Year-old lambs in particular were afflicted. Unlike the Lydenburg incident the buck were not unduly excited during spraying operations. Necropsies were performed on the ninth and tenth buck to die on the 28th and 30th day after spraying respectively. These investigations revealed macro- and microscopic lesions which in many respects resembled those of capture myopathy (white muscle disease syndrome) of game (Basson, McCully, Kruger, Van Niekerk, Young, De Vos, Keep & Ebedes, 1971). However, subsequent selenium determinations appeared normal. Dieldrin and photodieldrin residues, in ppm, were as follows: grass taken on the 28th day after spraying 1,3; 9,1; brain 0,22; 0,02; liver 6,05; 0,12; muscle 0,21; 0,01; marrow 17,00; 0,35; ruminal content 0,58; 0,02. Dieldrin residue on grass was as expected; photodieldrin was somewhat higher than the 3 to 5 ppm expected at 30 days (Wiese *et al.*, 1970).

The question therefore arose whether blesbuck and possibly other species of antelope are in fact hypersensitive to either dieldrin or its photoisomer, photodieldrin (Fig. 1), of which considerable quantities are formed by solar radiation under field conditions in South Africa (Wiese *et al.*, 1970).

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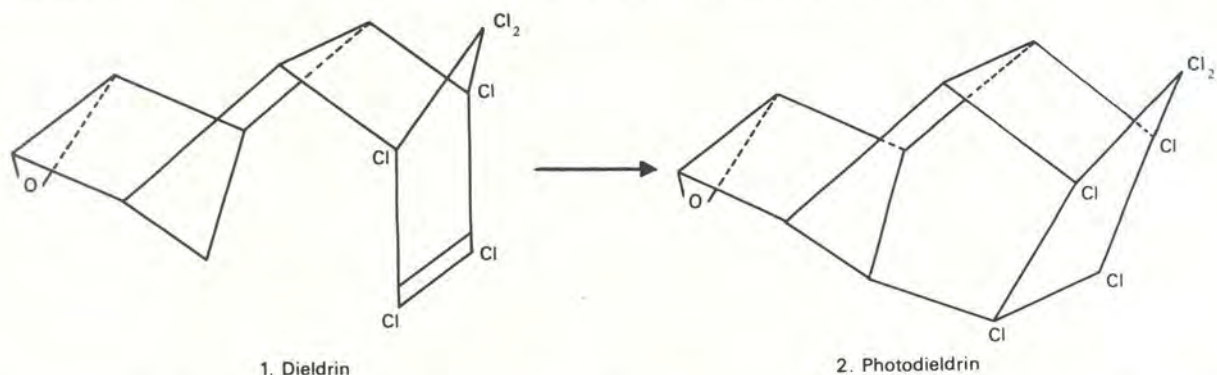


FIG. 1 Relevant structural formulae (1) 1, 2, 3, 4, 10, 10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4,5,8-dimethanonaphthalene (HEOD) (2) 1,1,2,3,3a,7a-hexachloro-5,6-epoxy, decahydro-2,4,7-methano-1H cyclopenta (a) pentalene (PHOTO-HEOD)

TABLE 1 Toxicity to blesbuck of dieldrin incorporated into feed

Dietary level (ppm)	Sex	Live mass at commencement of exposure (kg)	Period till death (days)	Lethal dose expressed as total toxicant intake (mg/kg)
5	♂, ♀	42,6 } 42,6 }	No mortality after 90 days	15,40 } 15,40 } $\bar{x} > 15,40$
15	♂, ♀	40,2 } 40,2 }	do	43,20 } 43,20 } $\bar{x} > 43,20$
25	♂, ♀	39,0 } 33,1 }	24 } 20 } $\bar{x} = 22,0$	7,89 } 10,21 } $\bar{x} = 9,05$
35	♂, ♀	39,9 } 42,5 }	11 } 14 } $\bar{x} = 12,5$	7,14 } 7,70 } $\bar{x} = 7,42$
50	♂, ♀	42,0 } 41,6 } 37,1 }	9 } 10 } 8 } $\bar{x} = 9,0$	12,58 } 12,10 } 7,52 } $\bar{x} = 10,73$

EXPERIMENTAL INVESTIGATIONS

1. Oral toxicity of dieldrin to penned blesbuck

Materials and methods

Year-old blesbuck were captured and, after a 3-month taming period, individually penned. Adequate shelter was provided in order to minimize stress in this shy antelope. A mixture of one part chopped teff hay and two parts chopped lucerne hay was provided *ad libitum*. After acclimatising (as judged by feed intake) in the pens for at least 2 weeks the antelope were exposed to dieldrin (85% HEOD and 15% related compounds) incorporated at known concentrations in their ration (Wiese & Basson, 1967). Feed intake was recorded daily in order to calculate the total toxicant intake during the exposure period.

Results

These are presented in Table 1.

Three animals died overnight without any clinical signs being observed. In the others the following nervous signs were seen: hypersensitivity (4 cases), imbalance (1 case) and intermittent clonic convulsive attacks over a period of 48 h prior to death (1 case).

The temperatures of two animals showing clinical signs were recorded as 40,7 and 41,1°C.

Animals not dead after 90 days' exposure were sacrificed. *Post mortem* investigations were performed on all test animals. The results of the pathological investigations on this group as well as the other two groups are presented separately below.

Tissue samples were taken and chemically analysed as outlined by Wiese *et al.* (1970). As fat reserves in the antelope were minimal, marrow drawn from the femur and tibia replaced mesenteric fat. Tissue burdens of dieldrin are presented in Table 2.

TABLE 2 Tissue burdens of dieldrin in blesbuck exposed to dieldrin-treated feed

Dietary level (ppm)	Tissue burdens (ppm)		
	Muscle	Liver	Marrow
5	0,20	3,3	17,2
15	0,33	8,2	36,4
25	0,75	9,4	29,9
35	1,08	15,1	33,0
50	1,69	18,4	57,0

Initial deposits of dieldrin on veld following aerial treatment for harvester termite control are in the vicinity of 15 to 20 ppm (Wiese *et al.*, 1970). These residues degrade fairly rapidly. Data contained in Table 1 point to a considerable safety margin at these levels. Comparison of tissue burdens of dieldrin in blesbuck exposed to varying dietary levels with those of antelope which had succumbed on veld treated for harvester termites suggested exposure to only sublethal doses of the latter and the cause of death of the blesbuck on treated veld was, therefore, not explained.

In the light of the above observations it was deemed essential to evaluate the hazards to antelope of dieldrin applied for harvester termite control critically under field conditions.

2. Blesbuck and springbuck herds exposed to commercially-treated grazing

Materials and methods

The 250 ha experimental site, situated on the farms Lusthof 1 456 and Fraaiuitzicht 1 453 in Kroonstad District, Orange Free State, at 27° 30'S, 27° 40'E, was at an altitude of 1 450 m with vegetation classified as transitional *Cymbopogon-Themeda* veld (Acocks, 1953). The site had a resident population of 35 blesbuck and 20 yearling springbuck (*Antidorcas marsupialis* (Zimmermann, 1780)) which were introduced some 2 to 3 months prior to treatment. The veld was very heavily infested with termites and the grass in patches reduced to stubble. The chances of detrimental effects showing up in the antelope were therefore optimal. The terrain was aerially sprayed on 30 June 1969 with a solution of

13% (m/m) technical dieldrin in a mixture of 70% xylene/styrene polymer and 30% Shellsol R. The insecticide was delivered by four rotary atomisers giving a distribution of seven droplets/cm² with mass median diameter of 280 μ and a deposit of 160 g dieldrin/ha (160 μ g/cm²), i.e. approximately double the concentration recommended for termite control. During spraying operations care was taken not to excite the antelope unduly so as to avoid a repetition of the Lydenburg experience. Residue determinations on the veld were as outlined by Wiese *et al.* (1970).

Results

The residue determinations on the veld are given in Table 3 and are graphically illustrated in Fig. 2.

By an arbitrary logarithmic residue/square root time transformation the three persistence curves can be shown to have a rectilinear trend as illustrated in Fig. 3. These results are comparable with those of Wiese *et al.* (1970).

On the 3rd day a change in the behaviour of the antelope was noticed particularly in the blesbuck which

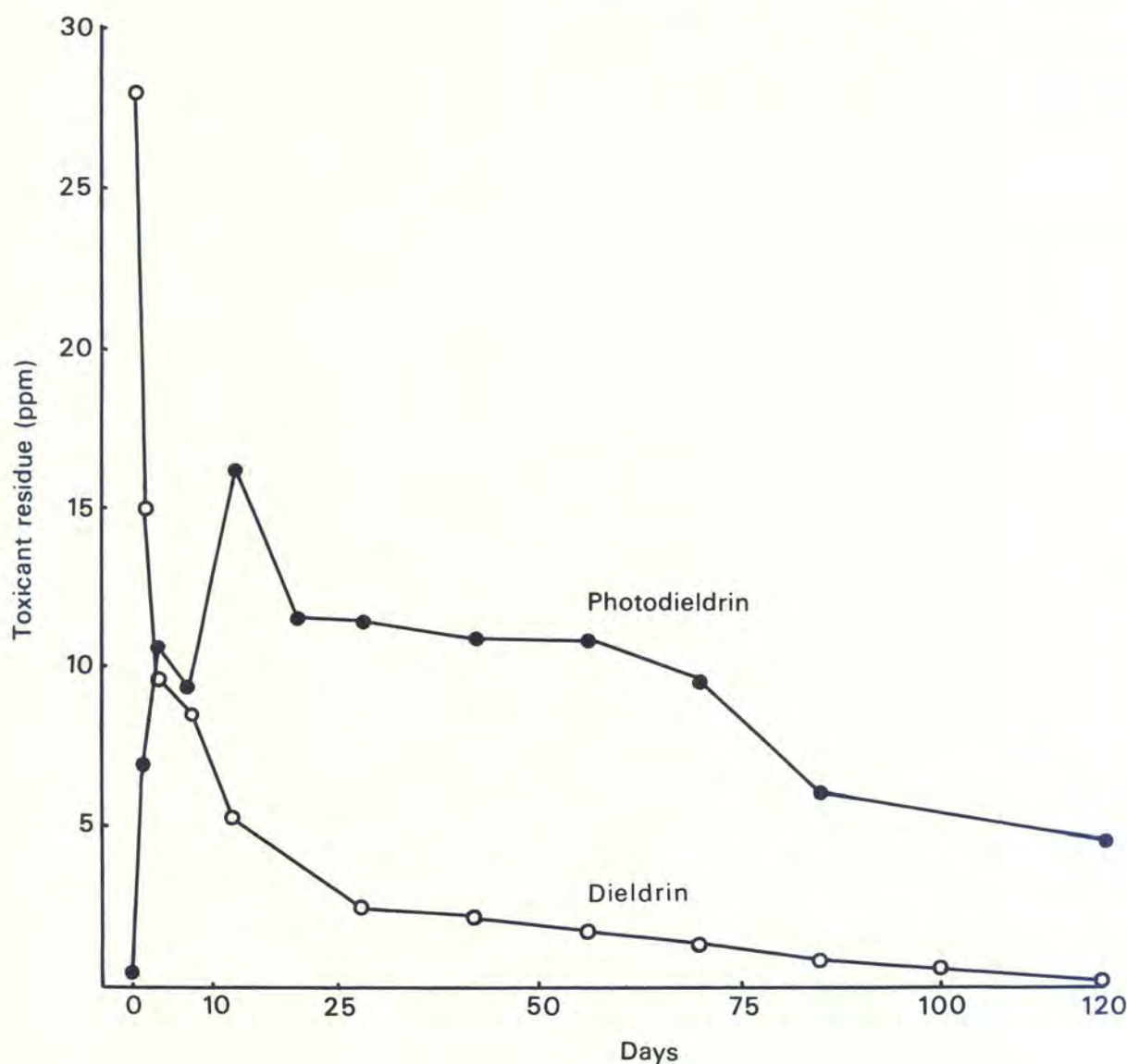


FIG. 2 Toxicant residues on veld after aerial application of dieldrin

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TABLE 3 Dieldrin and photodieldrin residues on veld at progressive intervals after treatment

Days	Dieldrin residues (ppm)	Photodieldrin residues (ppm)	Total residue, dieldrin plus photodieldrin (ppm)
0	27,60	0,52	28,12
1	15,00	6,93	21,93
3	9,50	10,92	20,42
7	8,42	9,25	17,67
14	5,04	16,13	21,17
21	3,43	11,54	14,97
28	3,28	11,59	14,87
42	2,45	10,93	13,38
56	2,13	11,00	13,13
70	1,17	9,70	10,87
85	0,75	6,70	7,45
120	0,23	4,93	5,16

appeared stupefied and periodically galloped aimlessly back and forth. Further nervous signs were exhibited which could be divided into violent and dumb syndromes. The former occurred from the 4th day onwards and varied from restlessness, mild to severe hyperaesthesia and wild galloping to muscular fasciculations and spasms and even intermittent clonic convulsive attacks. The dumb syndrome was characterised by an apparent loss of fear and by a state of stupor in which impaired vision, varying from partial and intermittent to

complete and continuous blindness, was very characteristic. This resulted in animals running or stumbling blindly into fences and through gullies, causing severe trauma and even death. Some animals were drowned in a dam in this way.

Mortalities in the blesbuck occurred from the 4th day onwards, the entire population being killed by the 19th day.

The springbuck were less obviously affected but followed the same pattern. Mortalities occurred from the 6th to the 13th days, by which time 70% had succumbed. Surviving springbuck recovered with no apparent after-effects and two ewes lambed normally in the spring following winter treatment.

Mortality data were subjected to probit analysis (Finney, 1952), the assumption being made that progressive mortalities were independent. These data are given in Table 4 and Fig. 4 and 5.

Residues of the two toxicants in the tissues of the two antelope species are presented in Table 5.

These data support the earlier inference that dieldrin alone was unlikely to have been responsible for the mortality among blesbuck (and probably springbuck), the total dieldrin intake in the field test being well below the levels resulting in mortality among penned antelope. It was therefore concluded that either photodieldrin or a combination of the two toxicants was responsible for antelope mortality and this led to the third phase of the investigation.

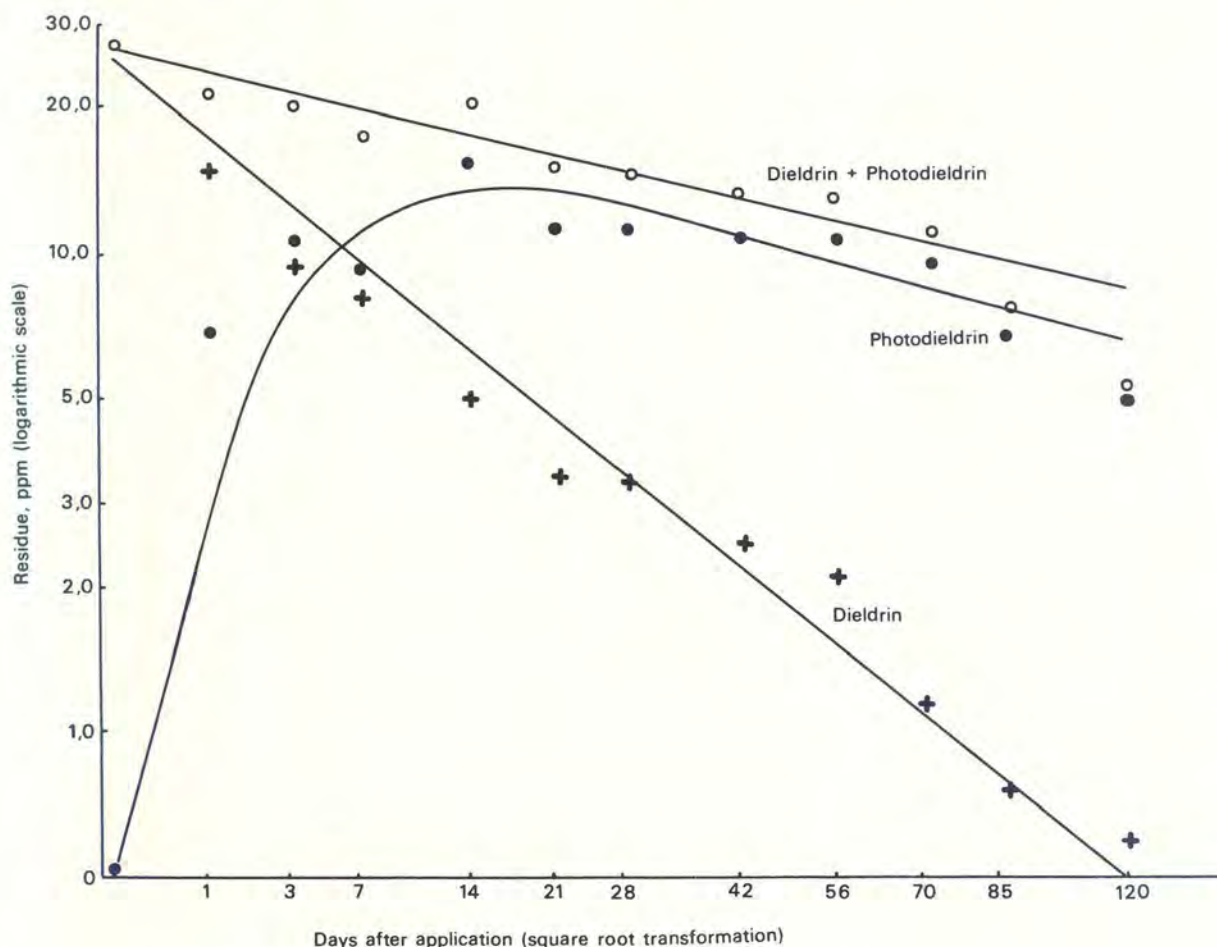


FIG. 3 Toxicant residues on veld after aerial application of dieldrin (log. residue/square root time transformation)

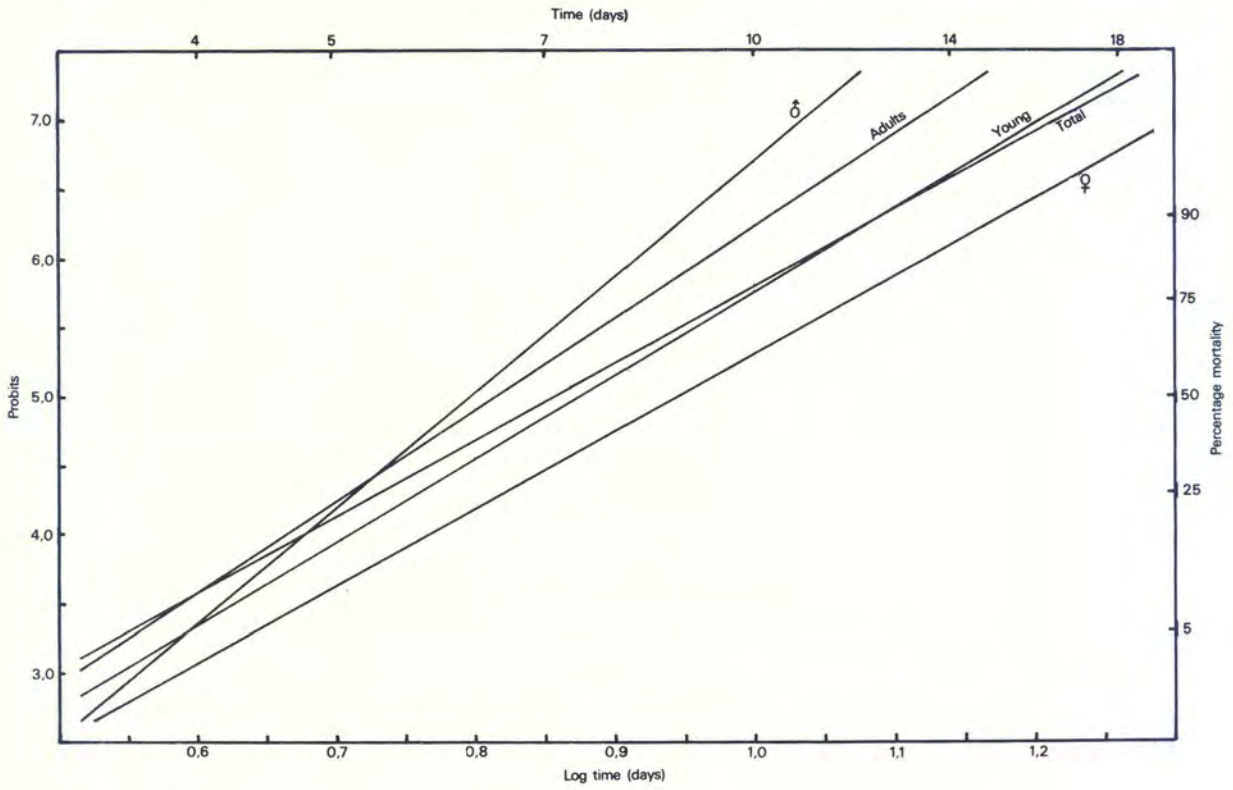


FIG. 4 Mortality of blesbuck exposed to dieldrin-treated veld

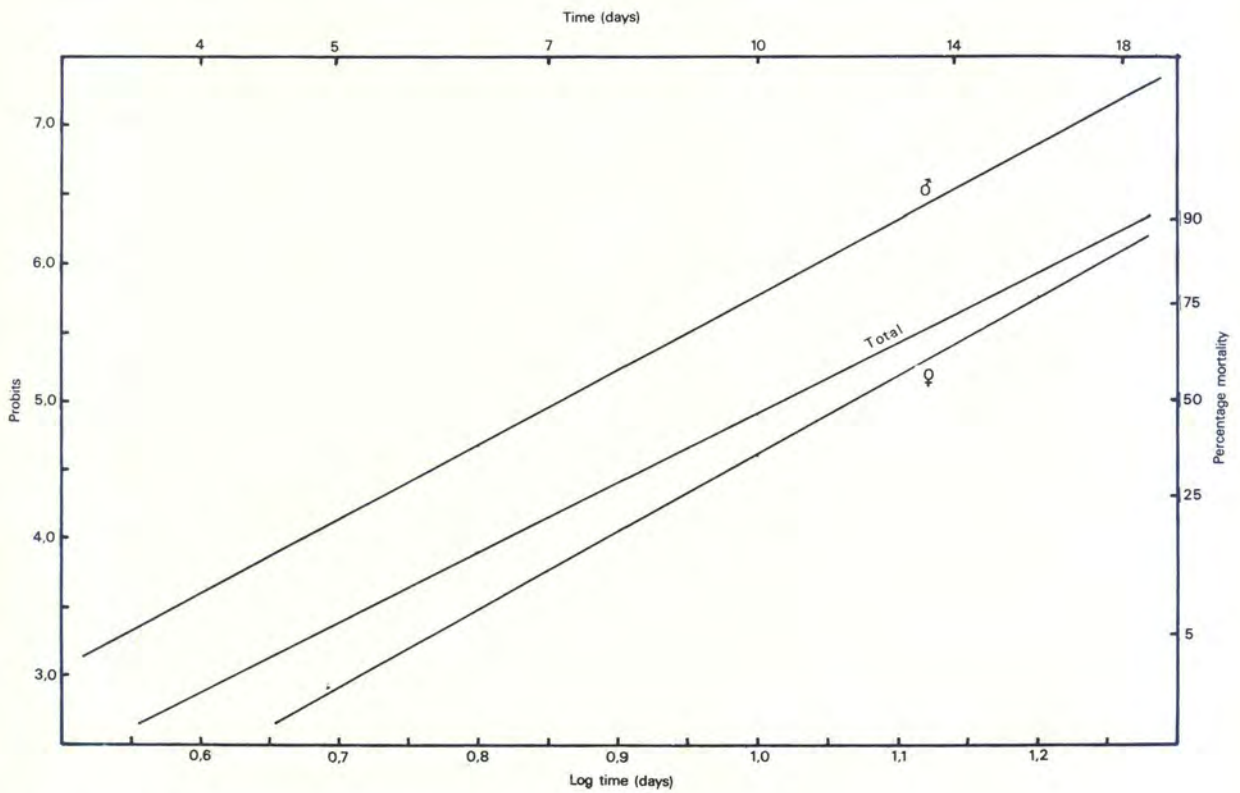


FIG. 5 Mortality of springbuck exposed to dieldrin-treated veld

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TABLE 4 LT₅₀ values, fiducial limits of LT₅₀ values and calculated toxicant intake of blesbuck and springbuck exposed to dieldrin-treated veld

Species	LT ₅₀ (days)	5% Fiducial limits	Total toxicant intake (mg/kg)*		
			Dieldrin	Photodieldrin	Total
<i>Blesbuck</i>					
Total	7,08	6,61-7,59	1,82	1,21	3,03
Rams	6,17	5,37-7,08	1,70	1,00	2,70
Ewes	8,70	7,94-9,55	1,98	1,60	3,58
Adults	7,41	5,62-9,77	1,90	1,27	3,17
Juveniles	6,46	5,60-7,36	1,78	1,05	2,83
<i>Springbuck</i>					
Total	10,47	8,83-12,42	4,53	3,97	8,50
Rams	7,24	5,89-8,91	3,71	2,49	6,20
Ewes	11,75	9,12-15,14	4,64	4,64	9,28

*Based on an assessment of mean herbage intake of healthy adult antelope (blesbuck: 1,45 kg/adult/day (Du Plessis, 1968); springbuck: 0,96 kg/adult/day (J. H. M. van Zyl, Div. Nature Conservation, Tvl. Prov. Admin., personal communication, 1970); modified by progressive change in herbage intake following intoxication as measured by mass of rumen content in relation to body mass over mortality period.

TABLE 5 Dieldrin and photodieldrin burdens in blesbuck and springbuck exposed to dieldrin-treated veld

Period between dieldrin appli- cation and sam- pling (days)	Tissue burdens (ppm)									
	Muscle		Liver		Marrow		Rumen		Abomasum	
	D*	P**	D	P	D	P	D	P	D	P
<i>Blesbuck</i>										
Control	0,007	<0,005	0,023	<0,005	0,081	<0,005	0,003	<0,005	0,005	<0,005
3	0,550	0,052	6,400	0,581	—	—	2,000	0,114	0,938	0,052
6	0,962	0,042	9,380	0,136	42,000	2,580	1,685	0,054	1,350	0,112
7	0,938	0,051	9,000	0,420	38,250	2,130	1,000	0,039	0,844	0,033
10	0,732	0,032	7,905	0,364	52,125	2,475	0,788	0,033	0,729	0,076
13	0,763	0,017	7,500	0,113	35,000	1,000	0,500	0,080	0,319	0,006
19	0,750	0,024	9,500	0,130	36,000	0,580	0,410	0,094	—	—
<i>Springbuck</i>										
Control	0,006	0,005	0,030	<0,005	0,091	<0,005	0,008	<0,005	0,019	<0,005
3	0,725	0,069	9,380	0,108	33,500	3,550	3,250	1,000	3,500	1,010
8	0,953	0,056	8,940	0,160	38,000	2,290	1,995	1,101	2,500	0,721
13	0,738	0,025	9,380	0,050	26,250	1,060	1,590	0,513	—	—

*Dieldrin
**Photodieldrin

TABLE 6 Toxicity to blesbuck of photodieldrin incorporated into feed

Dietary level (ppm)	Sex	Live mass at commencement of exposure (kg)	Period till death (days)	Lethal dose expressed as total toxicant intake (mg/kg)
15	♂ ♂ ♂	37,6 34,9 34,8	4 } 5 } 8 } $\bar{x} = 5,7$	1,34 } 1,10 } 1,90 } $\bar{x} = 1,45$
25	♂ ♂	34,4 41,2	7 } 6 } $\bar{x} = 6,5$	2,37 } 2,73 } $\bar{x} = 2,55$
50	♂ ♂	38,9 36,7	3 } 3 } $\bar{x} = 3,0$	1,35 } 2,06 } $\bar{x} = 1,71$

3. Oral toxicity of photodieldrin to penned blesbuck

Materials and methods

Year-old blesbuck were exposed to photodieldrin (99% 1, 1, 2, 3, 3a, 7a - hexachloro - 5,6 - epoxy decahydro - 2, 4, 7 - methano-IH cyclopenta (a) pentalene) using the methods described in Experiment 1.

Results

The results of this experiment appear in Table 6.

The clinical signs exhibited by these animals were similar to those seen in the dieldrin group except for one animal in the 15 ppm group in which the dumb syndrome and impaired vision were encountered.

Tissue burdens of photodieldrin in penned blesbuck are presented in Table 7.

TABLE 7 Tissue burdens of photodieldrin in blesbuck exposed to photodieldrin-treated feed

Dietary level (ppm)	Tissue burdens (ppm)		
	Muscle	Liver	Marrow
15	0,03	0,30	0,38
25	0,03	0,30	1,60
50	0,11	0,46	5,25

4. Study of the Pathology

Macroscopic Examination

Penned animals receiving dieldrin (Expt. 1) and photodieldrin (Expt. 3)

No major difference was found between the changes caused by dieldrin and photodieldrin poisoning, except that more widespread petechial haemorrhages, especially subepicardially, occurred in the latter. This is probably because the photodieldrin group died sooner (between 4 and 8 days) after feeding was commenced, than the dieldrin group (between 7 and 21 days). Consequently serous atrophy of the fat and hydrothorax was seen only in the dieldrin group, whereas atony of the forestomachs occurred in about half of the photodieldrin group. The feeding level of each specific substance and the duration of feeding also apparently did not alter the macroscopic picture significantly. However, no reliable statistical conclusions could be drawn as the numbers were too small.

The most consistent findings were general congestion, particularly of the liver, lungs and kidneys; cyanosis; mild to marked pulmonary oedema; mild to moderate localized muscular and subcutaneous haemorrhages, many of which were unilateral and traumatic; mild subendocardial haemorrhages, either in the left or right ventricle or in both, and prominent congestion of the brain. The spleen was usually contracted and atrophied. Myocardial and skeletal muscular degeneration was suspected in approximately 50% of the cases in both groups, but was only unequivocal in the back and upper thigh muscles of one case. This is in contrast to the natural cases where these lesions were very striking.

Lesions encountered either less frequently or occasionally were a mild hydropericardium; mild myocardial haemorrhages; swelling of the optic chiasma; mild haemorrhages in the lymph nodes, on the pulmonary artery and in the pleura; mild oedema of the pulmonary valves and suspected hepatic degeneration and nephrosis. Degeneration of the liver and kidneys was evidently masked at times by the fairly constant and prominent congestion. The *longissimus dorsi* was torn in one animal.

Field cases exposed to commercially-treated grazing (Expt. 2)

Examinations were done on a total of five blesbuck and two springbuck which were either destroyed or found dead on Day 7 of the experiment.

The findings were similar to those described for the penned animals except the following:

Traumatic injuries were very much more extensive. Subepi- and subendocardial haemorrhages were usually severe and degeneration of the myocardium was striking, as were haemorrhages in and degeneration and necrosis of the skeletal musculature. In addition varying degrees of constipation were seen in approximately half of the cases examined.

Microscopic Examination

Materials and methods

Tissues of six natural cases (four blesbuck and two springbuck), three of the seven blesbuck in the experimental dieldrin group and four of the six blesbuck in the experimental photodieldrin group were examined microscopically. The specimens were fixed in 10% buffered formalin. Sections of 3 to 6 μ thickness were cut and stained with haematoxylin and eosin (HE). Oil red-O stain (ORO) was used as a special method for determining lipids.

Results

The lesions in both the natural and the two experimental groups were very similar and the pathology will therefore be described in general.

Skeletal muscles: The lesions were very constant and varied from mild degeneration to severe, fairly widespread degeneration accompanied by haemorrhages and oedema. The mildest changes were cloudy swelling and hydropic degeneration. Zenker's degeneration, rarefaction, lysis, fragmentation and necrosis were present in more severe and advanced cases. Sarcolemmal proliferation, very mild neutrophil infiltration and mild macrophage mobilization followed these changes. Mineralization of necrotic fibres was frequently present. In the haemorrhagic lesions necrotic vascular lesions and very mild vasculitis were noticeable occasionally within some of the medium-sized veins. Swelling and degeneration in the walls of small vessels were also encountered.

Myocardium: Fairly constant lesions very similar to the muscular lesions were encountered, but they were usually not as severe and advanced. Segmental rarefaction of fibres, cloudy swelling and hydropic degeneration occurred most frequently. Typical Zenker's necrosis, however, was rare and localized. In one case necrotic changes were present within some of the capillaries between the muscle fibres. The lesions were sometimes fairly widely distributed and could have caused cardiac failure.

Liver: Congestion was a constant feature and often very marked. Very mild to mild cloudy swelling, hydropic and fatty changes were noticed in most of the cases and were sometimes more pronounced centrilobularly. Mild centrilobular haemorrhages were seen in one animal.

Kidneys: Usually moderately to markedly congested. Varying degrees of nephrosis, mostly mild or very mild, were present in many animals (more than 50%) and the changes included cloudy swelling, hydropic and fatty changes and an abundance of proteinaceous fluid in the tubuli and renal corpuscles. Tubular necrosis (kariorrhesis) and glomerular oedema were seen in a few cases.

Lungs: Invariably congested and frequently oedematous. However, the oedematous fluid was usually very poor in protein and consequently stained either very slightly or mildly with eosin.

Spleen: Almost invariably contracted with a relatively small number of erythrocytes and leukocytes in the red pulp. The splenic corpuscles were mostly small and kariorrhetic necrosis of the germinal centres was evident in a few cases.

Lymph nodes: Congestion and haemorrhages were often seen and very mild kariorrhesis of the follicles occurred in one case.

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TABLE 8 Distribution ratios of dieldrin and photodieldrin in blesbuck and springbuck

Species	Toxicant	Tissue		
		Muscle	Liver	Marrow
<i>Blesbuck</i>	Dieldrin only	1,00 0,07 0,02	13,5 1,0 0,3	42,8 3,2 1,0
	Photodieldrin only	1,00 0,16 0,02	6,1 1,0 0,1	42,3 6,9 1,0
	Dieldrin when administered with photodieldrin	1,00 0,10 0,02	10,4 1,0 0,2	49,0 4,7 1,0
	Photodieldrin when administered with dieldrin	1,00 0,14 0,02	7,1 1,0 0,1	53,9 7,6 1,0
<i>Springbuck</i>	Dieldrin when administered with photodieldrin	1,0 0,09 0,02	11,5 1,0 0,3	41,1 3,6 1,0
	Photodieldrin when administered with dieldrin	1,0 0,05 0,02	21,2 1,0 0,5	46,0 2,2 1,0

Brain: Severe or moderate congestion, frequently accompanied by mild haemorrhages and mild or moderate oedema, was usually present. The presence of a slightly eosinophilic substance in the perivascular spaces was taken as evidence of oedema. Brain swelling as evidenced by glial changes was a constant feature. These glial changes were represented by varying degrees of swelling (such as distinct visibility and an increase mainly of the cytoplasm); intracytoplasmic, eosinophilic granularity; nuclear pyknosis and kariorrhexis. The glial cells in the cerebellum and thalamus were most commonly affected.

A few deeply eosinophilic necrotic neurones and a few others containing prominent, single or multiple intracytoplasmic vacuoles were noticeable in the *medulla oblongata* of a minority of animals. Pallor, tigrolysis, suspected swelling and rarefaction of neuronal cytoplasm were frequently seen and regarded as doubtful lesions, because similar changes are common non-specific features which may also reflect physiological depletion of Nissl substance or autolysis.

DISCUSSION AND CONCLUSIONS

From data in Tables 1 and 6 it is apparent that photodieldrin is considerably more toxic to blesbuck than dieldrin. On account of the limited number of antelope available for toxicity trials, no median lethal doses could be calculated but only lethal doses for each individual. The mean cumulative lethal dose at the various dietary levels for dieldrin was 9,07 mg/kg; for photodieldrin it was 1,90 mg/kg. In other words photodieldrin is about five times more toxic to blesbuck than dieldrin. Under field conditions the calculated median lethal dose (m_D) of photodieldrin to blesbuck was 1,21 mg/kg. On the other hand the calculated m_D to springbuck was 3,97 mg/kg, suggesting that springbuck are three times less sensitive to photodieldrin than blesbuck. In both these antelope species rams were found to be significantly more susceptible to photodieldrin than ewes. Although young blesbuck appeared to be more susceptible than adults, the difference was not statistically significant. Brown, Robinson & Richardson (1967) reported on the acute toxicity of photodieldrin

to a variety of vertebrates. LD₅₀ values recorded by these authors range from about 3 mg/kg in guinea-pigs to 120 mg/kg in beagle hounds. In the case of dogs, males were found to be less sensitive than females.

The distribution of the two toxicants in the various tissues examined is interesting (Table 8).

From these data it appears that the distribution ratios in blesbuck are not affected when the two isomers are ingested either individually or in combination. The distribution of the two toxicants in both antelope species is also similar. The foregoing distribution ratios point to a low storage level in the muscle of antelope when compared with storage ratios in cattle and sheep (Wiese *et al.*, 1970). The low storage level in muscle tissue in comparison with liver and marrow can probably be ascribed to the low percentage of fat in the meat of antelope species. Glees (cited by Von la Chevallerie, 1970) reports that, with the exception of waterbuck and warthog, the fat content in canned meat of various wild ungulate species was below 1% while similarly prepared meat from cattle had a fat content of between 12 and 16%.

Furthermore Von la Chevallerie (1970) notes that fat constitutes but 1,7% of the adult springbuck buttock, while Kroon, Van Rensburg & Hofmeyr (1972) report that the ether extract of entire blesbuck carcasses varies from 2,09% in early spring to 7,80% in autumn. The two antelope species studied therefore exhibit low fat deposition tendencies. It can therefore be presumed that the low fat content is a prime reason for acute

TABLE 9 A comparison of the approximate acute lethal dose of dieldrin and photodieldrin to some ruminant species

Toxicant	Approximate acute LD (mg/kg)		
	Dorper wethers	Merino wethers	Blesbuck
Dieldrin	178*	24*	9,07**
Photodieldrin	—	12,9*	1,90**

*LD₅₀
**Mean cumulative LD

sensitivity of the antelopes, fat being considered to act as a buffer to dieldrin and photodieldrin intoxication (Wiese *et al.*, 1970).

As can be seen from Table 9 this assumption is further strengthened when these results are compared with those obtained in a recent study on Dorper and Merino sheep by Wiese, Basson, Basson, Naudé & Maartens (unpublished data).

Although these results are not strictly speaking, comparable (LD₅₀ and mean cumulative LD) an interesting trend can clearly be seen. The considerable fat reserve in the Dorper breed protects it from the effects of the highly lipid-soluble dieldrin and makes it seven times less susceptible than the leaner Merino breed. Blesbuck, which have even sparser fat deposits, are twice as susceptible to dieldrin and six times more susceptible to photodieldrin than Merino sheep.

While analysis of ruminal and abomasal content suggests that photodieldrin is absorbed more rapidly than dieldrin, the storage ratio of photodieldrin in the three tissues examined from these two antelope species is low in comparison with dieldrin. This latter observation supports the findings of Wiese *et al.* (1970) in respect of cattle and sheep.

The degenerative and necrotic lesions in the liver, kidneys, myocardium and skeletal musculature are in accordance with those mentioned for chlorinated hydrocarbon poisoning in stock in general by Garner (1961), Clarke & Clarke (1967) and for dieldrin toxicosis in the rat by Harr, Claeys, Bone & McCorcle (1970). However, in addition to brain oedema, brain swelling was also observed here. Furthermore, although mentioned by other workers, in these experiments the lesions in the skeletal and cardiac musculature were so conspicuous and widespread that they could have caused this poisoning to be confused with other syndromes such as capture myopathy. The disparity in the degree of this damage between penned blesbuck and the field cases can probably be attributed to the more stressful conditions, with freedom of movement, under which the latter were living.

In many of the field cases partial or total blindness was a conspicuous sign. Apart from Mellanby (1967), who mentions apparent blindness in foxes intoxicated by eating birds killed by dieldrin, this has not been described by other workers. It might possibly be ascribed to brain oedema and/or swelling of the optic chiasma. Unfortunately no specimens were collected for eye studies.

The lesions encountered are in accordance with those which may result from hyperthermia, spasmodic convulsions and excessive muscular exertion, hypoxia due to cardiac degeneration and possibly the suppression of certain vital centres in the brain such as the heat control, vasomotor, cardiac and respiratory centres. In the latter event terminal shock may, in some animals, increase the likelihood of death or the animal may die of either cardiac or respiratory arrest. Many of the lesions seen are compatible with shock and cardiac arrest.

In the field experiment conducted in this investigation commercial application resulted in 160 g dieldrin/ha being used instead of the advocated 87 g/ha. The photo-

dieldrin residues were still ≈ 10 ppm 70 days after spraying (Table 3 & Fig. 2). Where the recommended concentration is used a maximum photodieldrin concentration of 7.13 ± 1.66 ppm was recorded 6 days after spraying and the residue was still 5.67 ± 0.77 ppm at 21 days (Wiese *et al.*, 1970).

The lowest concentration of photodieldrin fed experimentally to penned blesbuck was 15 ppm and at this level the buck died after 4 to 8 days' exposure (average 5.7 days, Table 6), whereas in the field experiment the last animals only died 19 days after spraying. The actual level of this toxicant encountered after spraying at the recommended concentration was, therefore, not tested experimentally but it was concluded that dieldrin application for harvester termite control presents a definite hazard to antelope populations and its registration for use on open grassland inhabited by antelope has accordingly been withdrawn.

ACKNOWLEDGEMENTS

We wish to express our sincere thanks to the Divisions of Nature Conservation of both the Transvaal and Orange Free State Provincial Administrations for the antelope used in these experiments; to the Shell International Chemical Centre for the photodieldrin; to Prof. T. F. Adelaar for his interest and advice; to the technical staff of the Sections of Toxicology and Pathology, Veterinary Research Institute, Onderstepoort for their able and willing assistance and to Mrs. R. A. Schultz for her assistance in preparing the figures.

REFERENCES

- ACOCKS, J. P. H., 1953. Veld types of South Africa. *Mem. bot. Surv. S. Afr.*, 28, 192 pp.
- BASSON, P. A., MCCULLY, R. M., KRUGER, S. P., VAN NIEKERK, J. W., YOUNG, E., DE VOS, V., KEEP, M. E. & EBEDS, H., 1971. Disease conditions of game in southern Africa: Recent miscellaneous findings. *Vet. med. Rev.*, 2/3, 313-340.
- BROWN, V. K. H., ROBINSON, J. & RICHARDSON, A., 1967. Preliminary studies on the acute and subacute toxicities of a photo-isomerization product of HEOD. *Fd. Cosmet. Toxicol.*, 5, 771-779.
- CLARKE, E. G. C. & CLARKE, MYRA L., 1967. Garner's veterinary toxicology, 3rd ed. London: Baillière, Tindall & Cassell.
- DU PLESSIS, S. S., 1968. Ecology of blesbok, *Damaliscus dorcas phillipsi*, on the Van Riebeeck Nature Reserve, Pretoria, with special reference to productivity. D.Sc. Thesis, Univ. Pretoria.
- FINNEY, D. J., 1952. Probit analysis: A statistical treatment of the sigmoid response curve, London: Camb. Univ. Press.
- GARNER, R. J., 1961. Veterinary toxicology, 2nd ed. London: Baillière, Tindall & Cox.
- HARR, J. R., CLAEYS, R. R., BONE, J. F. & MCCORCLE, T. W., 1970. Dieldrin toxicosis: rat reproduction. *Am. J. vet. Res.*, 31, 181-189.
- KROON, FREDERIKA, E., VAN RENSBERG, W. J. J. & HOFMEYER, H. S., 1972. Seisoenale veranderinge in chemiese samestelling van volwasse blesbokramme (*Damaliscus dorcas phillipsi*) van die Van Riebeeck Natuurreservaat. *S. Afr. J. Anim. Sci.*, 2, 41-44.
- MELLANBY, K., 1967. Pesticides and pollution, London: Collins.
- VON LA CHEVALLERIE, M., 1970. Meat production from wild ungulates. *Proc. S. Afr. Soc. Anim. Prod.*, 9, 73-87.
- WIESE, I. H. & BASSON, N. C. J., 1967. The oral toxicity of dieldrin to the crowned guinea-fowl, *Numida meleagris* (L.). *S. Afr. J. Sci.*, 10, 697-706.
- WIESE, I. H., BASSON, N. C. J. & VAN DER MERWE, J. H., 1970. Dynamics of dieldrin and its photo-isomerization product, photodieldrin, on veld and in livestock exposed to dieldrin-treated veld. *Phytophylactica*, 2, 33-48.