# Studies on the Photosensitisation of Animals in South Africa.

IV. The Toxicity of Lopholaena coriifolia (Harv.) Phill. & C.A. Sm. (= L. randii sp. Moore).

By J. I. QUIN, D.V.Sc., Veterinary Research Officer, Onderstepoort, Pretoria.

### INTRODUCTION.

Investigations into an outbreak of "geeldikkop" amongst sheep on a farm near De Aar, Cape Province, revealed the interesting fact that practically no *Tribulus* plants were to be found on the veld where the animals had taken ill. As it had been shown experimentally that in the Karroo area, wilted *Tribulus* frequently caused the disease during the summer months, this outbreak near De Aar necessitated the investigation of other possible factors in the etiology of the condition. From the symptoms and post-mortem lesions shown by the affected sheep, the disease was diagnosed as true geeldikkop as caused by *Tribulus*.

According to the owner and the shepherds on the property, young sheep were frequently seen nibbling at the soft tops of a plant called "Vaalbos". Some of the plant in the late flowering stage was collected and subsequently identified as *Lopholaena coriifolia* (Harv.). Feeding experiments were then commenced at Onderstepoort with plant material forwarded at regular intervals by Government Veterinary Officer Keppel, De Aar.

### FEEDING EXPERIMENTS.

As stabled sheep consistently refused to ingest any of the fresh plant offered to them, it was decided to dry the material, then pulverise and drench through a stomach tube. The plant contained a large amount of tough woody stems and twigs and even the small leaves were fairly leathery. The dry material after being pulverised in a mill emitted a peculiar resinous odour.

Altogether 17 young Merino sheep were used in these experiments. Before being dosed the animals were closely shorn and thereafter kept exposed in sunlight, in order to notice whether symptoms of photosensitisation developed. The following table indicates the results obtained from dosing with the powdered *Lopholaena* plant:—

Sheep No.	Period of Dosing.	Total Amount Dosed.	Symptons Shown,	Post-mortem Findings.	General Remarks.
19435	2 days	400 gm.	Second day: Restlessness and respiratory distress. Third day: Accelerated respi- ration, listlessness, off feed, abdomen distended. Died fourth day.	Pulmonary congestion, de- generation of myocard. Severe fatty changes in liver.	No icterus or oedematous swellings.
19468	3 days	400 gm.	Second day: Listlessness, jerky intermittent breathing resem- bling Cheyne-Stokes. Third day: Breathing un- changed. Fourth day: Animal dead.	Severe fatty changes in the liver.	No leterus or oedematous swellings.
21473	15 days	1250 gm.	Sixteenth day: Progressive falling off in condition, breathing hurried and intermittent, drowsiness, lips and conjunctivae light yellow. Animal killed for post-mortem.	Striking fatty changes in the liver.	Slight icterus. No oedema- tous swellings.
19433	14 days	900 gm.	Twelfth day: Listlessness, un- steady gait, falling off in condition, lips yellowish. Fifteenth day: Animal killed for post-mortem.	Slight generalised ieterus. Extensive fatty changes in the liver.	Slight leterus, no swellings. Di- rect v.d. B. test on serum positive.
21461	10 days	1300 gm.	Ninth day: Facial skin slightly yellowish. Eleventh day: Animal killed for post-mortem.	Slight generalised leterus, Fatty changes in liver and kidneys.	Slight icterus, No swellings.
20962	2 days	Aqueous extract from 800 gm.	Fourth day: No symptoms shown.	_	Watery extract produced no effect.
22421	2 days	400 gm,	Second day: Listlessness. Third day: Animal dead.	Extensive fatty changes of liver.	No interest or swellings.
22418	1 day	200 gm.	Second day: Animal ill, not feeding. Died the same day.	Marked fatty changes of liver. Atony of fore- stomachs.	No leterus or swellings. Death very acute.
22425	1 day	70 per cent. alcoholic extract from 400 g.m.	Fourth day: Animal appears ill, marked dyspnoea. Died during the same night.	Pulmonary congestion. Extensive swelling and fatty changes of the liver and kidneys.	No leterus or swellings.
22391	4 days	300 gm.	Fifth day: Animal very ill and groaning. Mucous membranes injected. Sixth day: Died early in the morning.	Fair generalised icterus. Hydropericardium, cedema of lungs. Very severe fatty changes of liver and kidneys, marked stasis in first part of large intestines.	swellings.
22406	3 days	300 gm.	Third day: Animal died sud- denly during the night.	Marked fatty changes liver, kidneys, and myocard. Fair icterus.	Fair icterus, No swellings.
22393	2 days	200 gm.	Third day: Animal died sud- denly during the night.	Marked fatty changes liver, Pulmonary oedema. Slight leterus.	

From the above table it is clear that the powdered Lopholaena coriifolia (Harv.) plant when dosed to sheep causes severe illness even in comparatively small doses of 200 gm. (Sheep Nos. 22418 and 22393). Some sheep, however, require far larger quantities before showing any symptoms, e.g. sheep No. 21461 received 1,300 gm. in 10 days. The symptoms noted were, laboured and hurried respiration, general debility and in some cases a slight clinical icterus. In no case was photosensitisation noticed, although the animals were kept exposed in sunlight. On post-mortem examination intense fatty changes of the liver and frequently also of the kidneys, were the most important lesions to be found. In some cases stasis in the large intestines were also seen. Both the clinical and pathological pictures agreed very closely with the results obtained in either chloroform or phosphorus poisoning of sheep (see subsequent paper), where intense fatty changes of the liver may be provoked within 24 hours of administering the poison. It has also been ascertained that the toxic agent in Lopholaena coriifolia (Harv.) is insoluble in water but soluble in 70 per cent, cold alcohol which easily extracts it from the plant. The extract has a strong resinous odour.

## CONCLUSIONS.

Lopholaena coriifolia (Harv.), which has been suspected of causing geeldikkop in sheep under field conditions, was dried, pulverized, and dosed to sheep at Onderstepoort. Some animals suffered acute poisoning, while others showed much greater resistance. The symptoms shown did not resemble those of true geeldikkop. The severe fatty change in the liver resembles the action of such poisons as phosphorus and chloroform. In true geeldikkop severe bile stasis and pigmentation of the liver with intense icterus of the whole body are the predominant findings. There is, however, comparatively little fatty change in the liver. From these findings it is evident that Lopholaena coriifolia (Harv.) contains an active liver poison which, however, in drenching experiments has not led to the onset of geeldikkop in sheep.

#### LITERATURE.

FRÖHNER, E. (1919). Lehrbuch der Toxikologie fur Tierärzte. Stuttgart. HAUSMANN, W., and HAXTHAUSEN, H. (1929). Die Lichterkrankungen der Haut. Urban & Schwarzenberg, Berlin u. Wien.

LEWIS, T. (1927). The Blood vessels of the human skin and their responses. Shaw & Sons, London.

QUIN, J. I. (1928). Recent investigations into Geeldikkop affecting sheep and goats in the Cape Province. Jnl. S.A. Vet. Med. Assn., Vol. 1, No. 2, pp. 43-45.

QUIN, J. I. (1929). Further investigations in Geeldikkop (Tribulosis ovium). 15th Rept. Dir. Vet. Serv., Union of S.A., pp. 765-767.

QUIN, J. I. (1930). Further investigations into the problem of Geeldikkop (Tribulosis) in small stock. 16th Rept. Dir. Vety. Serv. and Anim. Indust., Union of S.A., pp. 413-416.

QUIN, J. I. (1931). The photosensitising influence of haematoporphyrin on sheep and goats. 17th Rept. Dir. Vety. Serv. and Anim. Indust., Union of S.A., pp. 645-659.

STEYN, D. G. (1928). Dikoor in sheep. Jnl. S.A. Vet. Med. Assn., Vol 1, No. 2, pp. 47-50.

THEILER, A. (1918). Geeldikkop in sheep. (Tribulosis ovium). 7th and 8th Rept. Dir. Vety. Res., Union of S.A., pp. 1-55.

17 499