The Toxicity of Sodium Chlorate.

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INTRODUCTION.

VERY favourable results with regard to sodium chlorate as a weed-killer have been reported from New Zealand [Editorial (1930) and Lyons (1930)] and the United States of America [Editorial (1931)]. In New Zealand sodium chlorate has been officially recommended in a 4 to 5 per cent. aqueous solution as an efficient killer of ragwert (Senecio spp.), whilst in the United States of America it is advocated as a general weed-killer, being especially destructive to thistles and many graminaceous weeds. The spraying of weeds during early stages of development is advised as they are less resistant than fullgrown plants and also less spraying material is required.

In New Zealand the effects of calcium chlorate as a weed-killer have been compared with those of sodium chlorate and the latter was found to be cheaper and more effective than the former.

Since Senecio poisoning is of very widespread occurrence in the Union of South Africa, it has been decided to conduct some experiments in order to test the value of sodium chlorate as a weed-killer under South African conditions with special regard to Senecio spp. and other poisonous plants (Dichapetalum cymosum Hook, Pachystigma pygmaeum Robyns, etc.).

The toxicity of such a substance to stock is of the utmost importance in the determination of its value as a weed-killer. This statement needs no lengthy elucidation, as it is wellknown that some arsenical preparations are excellent weed-killers but are at the same time a grave danger not only to stock but also to the persons handling such poisonous preparations. It might be mentioned here that the general practice of eradicating prickly pear by means of arsenic pentoxide has been responsible for serious losses in stock.

It is clear that an ideal weed-killer should be highly toxic to the weed or weeds to be destroyed and relatively non-toxic to edible and useful plants and to stock and human beings.

According to reports from New Zealand and the United States of America, sodium chlorate seems to fulfil these requirements better than any known weed-killers.

It was thought advisable to ascertain the toxicity of sodium chlorate under South African conditions before recommending it as a general weed-killer.

REVIEW OF LITERATURE.

Fröhner (1919) administered 25 grams of potassium chlorate to a 35 Kg. wether with negative results, whilst 50 grams caused transient depression, inappetence and cessation of rumination. He found that amounts of 30 and 40 grams had no effect on horses and that a cow, which had received 50 grams, and two days later, 100 grams of potassium chlorate, developed no symptoms of poisoning.

Zimmerman (quoted by Fröhner 1919) states the following to be the lethal doses of potassium chlorate: horse 250 grams; cow 500 grams; sheep 100 grams; dogs 60 grams.

Seddon and Mcgrath (1930) fed a lick containing two parts of sodium chlorate and one part of bonemeal to a bovine. On the fourteenth day of the experiment after it had ingested 260 grams of sodium chlorate the animal exhibited inappetence and depression, the faeces being dark and mucoid. An intense icterus developed and the animal was destroyed on the tenth day of illness.

Seddon and Mcgrath found the toxic dose of sodium chlorate for sheep to be from 50-75 grams. Thirty-three grams of this salt had no effect on sheep whilst one sheep died from 50 grams and another one developed no symptoms after the administration of the latter amount.

Lipschitz (1932) fed cats for weeks on a diet to which 0.5 gram of sodium and potassium chlorate per kilogram body weight had been added daily without producing any symptoms of poisoning. A single dose of 1.13 grams potassium and sodium chlorate per kilogram body weight caused slight symptoms, whilst 1.35 to 1.94 grams of sodium chlorate per kilogram body weight produced pronounced dyspnoea, methaemoglobinaemia and death in cats.

Symptoms of Poisoning.

(a) Acute poisoning.—Large amounts of sodium and potassium chlorate cause death one to a few hours after administration, the proximate cause of death being asphyxia due to severe methaemoglobinaemia which was diagnosed spectroscopically. There is also a certain degree of haemolysis.

On post-mortem examination the blood and organs (especially the lungs) are found to be dark chocolate brown in colour. If the blood be centrifuged the supernatant serum is of a reddish tinge due to haemolysis.

(b) Subacute poisoning. -When small amounts of the chlorates are repeatedly administered the following symptoms may be seen: laboured respiration; accelerated heart action, which becomes progressively weaker; gastro-intestinal irritation; inappetence; cessation of rumination; intense icterus (haematogenic): haemoglobinaemia: haemoglobinuria: anuria: uraemia: opisthotonus; uraemic spasms; disappearance of the petallar and corneal reflexes; coma and death.

The *post-mortem* reveals icterus of a varying degree depending on the period of illness: enlarged and copper coloured liver: pigmented and swollen kidneys; gastro-enteritis and cystitis.

On pastures treated with sodium chlorate the subacute and chronic forms of poisoning with this weed-killer are more likely to occur than the peracute and acute forms.

ONDERSTEPOORT EXPERIMENTS.

The experiments conducted at Onderstepoort are recorded in the following table. Commercial sodium chlorate was administered per stomach tube in a 10 per cent. aqueous solution. The commercial preparation was used as it is cheaper than chemically pure sodium chlorate and will, therefore, be used in preference to the latter as a weed-killer.

THE EFFECTS OF SODIUM CHLORATE ON HORSES, SHEEP AND RABBITS.

D.O.B. No. of animal.	Weight in Kg.	Quantity of NaC10 ³ given, and period of dosage.	Total amount of NaC10 ³ given.	Result.
Rabbit A.	2.0	l gm. daily* for 20 days; 3 gm. daily for 6 days; 15 gm. in one dose	Gm. 53	The 1 gm. doses continued for 20 days had no effect; likewise the 3 gm. doses over a period of 6 days. 15 gm. caused dyspnoea 1 hour after dosage; then pronounced apathy; weak and accelerated heart beat; brownish conjunctiva; passage of dark brown urine; fever; weakness and death within 10 hours after dosage. Post-mortem appearances: Blood and all organs (especially the lungs) of an intense dark chocolate brown colour; haemolysis; haemoglobinuria.
Rabbit B.	2.2	3 gm. daily for 20 days; 6 gm. daily for 3 days; 10 gm. daily for 2 days; 12 gm. in one dose	110	The 3 gm. doses over a period of 20 days had no effect; likewise the three 6 gm. doses. The 10 gm. doses produced symptoms as described above and diarrhoea. The 12 gm. dose caused death after 8 hours. Post-mortem appearances: As in Rabbit A plus acute catarrhal gastro-enteritis.
Rabbit C.	2.0	5 gm. daily for 20 days; 7·5 gm. daily for 3 days	122.5	The twenty 5 gm. doses caused no ill-effects. The third dose of 7.5 gm. caused symptoms as in Rabbit A and death 6 hours after administration. Post-mortem appearances: Pronouned hydraemia; blood of a slight brownish tinge; hyperaemia of the lungs and liver.
Rabbit D.	2.3	7.5 gm. in one dose; 10 gm. in one dose; 12 gm. in one dose	29.5	A few hours after the 7.5 gm. dose the animal passed dark brown urine and showed laboured respiration; accelerated and weak heart beat. More severe symptoms were produced by the 10.0 gm. dose. The animal died 55 hours after the 12.0 gm. dose after having exhibited apathy, pronounced weakness, pronounced dyspnoea, accelerated and weak heart action. Post-morten appearances: Cachexia; pronounced hydraemia; extreme dilatation of atria and ventricles of heart.

^{*} Except Sundays.

THE EFFECTS OF SODIUM CHLORATE ON HORSES, SHEEP AND RABBITS (continued).

AND RADDITS (communical).						
D.O.B. No. of animal.	Weight in Kg.	Quantity of NaC10 ³ given, and period of dosage.	Total amount of NaC10 ³ given.	Result.		
Rabbit E.	2.1	10 gm, in one dose	Gm. 10	Symptoms similar to those described in Rabbit A produced by the 10 gm. dose were exhibited, death occurring one hour after dosage. Post-mortem appearances: Blood and organs of an intense dark chocolate brown colour; haemolysis.		
Rabbit F.	2 · 2	10 gm. in one dose	10	Developed severe symptoms of poisoning but recovered.		
Sheep 32313 (full mouth)	40.0	15 gm. daily for 3 days	45	The first two doses had no discernible effects. Three hours after the third dose the animal exhibited dyspnoea; accelerated heart beat, which became progressively weaker; haemoglobinmaemia, temperature 160° F.; visible mucous membranes; conjunctiva and unwoolled parts of the skin dirty brown in colour; the animal died 20 hours after the third dose. Post-mortem appearances: Blood and organs (especially lungs) of an intense dark chocolate brown colour; haemoglobinaemia; hae-		
				moglobinuria; pronounced oedems and hyperaemia of lungs; oedems of all lymph glands; marked tumor splenis; degenerative changes in the liver; subendocardial hae morrhages; numerous haemorr- hages in mucous membrane of caecum and colon.		
Sheep 28203 (full mouth)	45	30 gm, daily for 2 days	60	The first dose of 30 gm. caused slight dyspnoea and accelerated heart beat which passed off a few hours. The second dose produced the same train of symptoms as that described in sheep 32313 (temp 108° F.) death following within 20 hours of the record dose. Post-mortem appearances: As in sheep 32313.		
Sheep 31599	35	7.5 gm. daily for 20 days; 10 gm. daily for 3 days	180	After the second dose of 7.5 gm petechiae were noticed on the conjunctiva and disappeared within 24 hours. Inappetence and diar rhoea set in on the twelfth day of dosage; complete recovery, how ever, had occurred on the seventeenth day of the experiment in spite of the fact that daily dosage was continued. The three 10 gm doses produced no clinical symptoms.		

THE EFFECTS OF SODIUM CHLORATE ON HORSES, SHEEP AND RABBITS (continued).

D.O.B. No. of animal.	Weight in Kg.	Quantity of NaC10 ³ given, and period of dosage.	Total amount of NaC10 ³ given.	Result.
Horse 18526 (aged)	450	30 gm. on 4/4/32 60 gm. on 11/4/32 90 gm. on 18/4/32 120 gm. on 2/5/32 130 gm. on 16/5/32	Gm. 430	The doses of 30 and 60 gm. caused slight brownish discolouration of the conjunctiva and laboured respiration, recovery occurring after 36 hours. The 90 gm. dose produced dark dirty brown discolouration of the conjunctiva with eechymoses and dyspnoea which persisted for about 2 days. The 120 and 130 gm. doses caused a pronounced discolouration of the conjunctiva; somnolence; inappetence; accelerated and strong pulse; temparature of 102° F.; dyspnoea. The animal appeared normal after a further two days.

From the above table it is evident that sodium chlorate given in daily amounts up to 5 grams in a 10 per cent. solution produced no appreciable illeffects on rabbits, whilst 10 grams in a single dose sufficed to produce death one hour after administration in one rabbit, and on another rabbit severe symptoms of poisoning which, however, did not prove fatal.

In sheep three daily doses of 15 grams each produced death twenty hours after administration of the last dose, whilst two 30 gram doses sufficed to cause death. Again in the course of the administration of twenty daily doses of 7.5 grams transient diarrhoca and inappetence appeared, the animal recovering in spite of the fact that dosing was continued and the daily quantity increased to 10 grams for the last three days of dosage.

A horse developed fairly severe symptoms of poisoning after the administration of 120 and 130 grams of sodium chlorate respectively, whilst 60 grams produced only slight methaemoglobinaemia.

The proximate cause of death in acute sodium chlorate poisoning is asphyxia due to severe methaemoglobinaemia, death being accelerated by haemolysis.

In protracted cases methaemoglobinaemia becomes a less prominent symptom and may not be manifested clinically whilst hydraemia and uraemia enter into the symtom complex.

It must be mentioned that in cases of sodium chlorate poisoning no reference to elevation of body temperature is made in the literature, while in the cases produced at Onderstepoort high fevers were recorded in all animals, especially in severe cases of poisoning.

Dr. G. de Kock, head of the department of Pathology, Onderstepport, who has kindly examined the organs of rabbits and sheep poisoned by sodram chlorate reports that the former animals showed hyperaemia of the different organs and fibrosis of the myocard. In the case of sheep all the organs were hyperaemic and the kidneys showed a peculiar pigmentation, which was not due to haemosiderin or haemoglobin.

DISCUSSION.

In discussing weed-killers the most important point that arises is the possibility of their causing poisoning in stock. It is obvious that weed-killers which are to be extensively used on pastures should not be so toxic as to cause poisoning in such amounts as are likely to be ingested with the vegetation. The toxicity of arsenical compounds precludes their use as weed-killers except in special cases, for example, in localised spots.

Another point of the utmost importance in the extensive application of weed-killers to pastures is the degree of damage they will cause to the edible and valuable vegetation in such solutions as will destroy the weeds. This relative destructive value of weed-killers to weeds and edible vegetation is perhaps of more value than the degree of toxicity of the weed-killers to stock, as the poisoning of stock could be prevented by not allowing them access to treated pastures until after heavy rains have fallen.

From the results of experiments conducted by Fröhner (1919), Seddon and Mcgrath (1930) and at Onderstepoort, it would appear that sodium chlorate is relatively speaking, not very toxic to stock. As reports from New Zealand and the United States of America record it to be an efficient weed-killer, sodium chlorate would best seem to satisfy the requirements of a suitable weed-killer to be utilised on pastures.

However, before its use as a general weed-killer on pastures can be advocated, it is essential to conduct experiments in order to determine its relative destructive capacity for the weed or weeds to be killed and for the pasture plants. It is on this property that the suitability of sodium chlorate as a weed-killer on pastures depends.

SUMMARY.

In New Zealand sodium chlorate is recommended as an efficient destroyer of ragwort, while reports from the United States of America state it to be effective as a general weed-killer.

The fact that sodium chlorate is of comparative low toxicity to stock would seem to warrant the conducting of experiments with a view to ascertaining its value as a general weed-killer on South African pastures.

LITERATURE.

EDITORIAL (1930). Attack on Ragwort. The Auckland Weekly News (New Zealand), 30 April, 1930, p. 31.

EDITORIAL (1931). Weed-killers. Pharmacent, Jul. and Pharmacist. June 1931, p. 520.
 FRÖHNER, E. (1919). Lehrbuch der Toxikologie für Tierärzte. 4te Auflage. Stuttgart. Verlag von Ferdinand Enke, pp. 132–133.

LANDER, G. D. (1926). Veterinary Toxicology. 2nd Edition. London. Baillière, Tindall & Cox, p. 92.

LIPSCHITZ, W. (1932). Über die Toxizität des Chlorats. Arch. f. Expt. Path. n. Pharm, pp. 570-575.

LYONS, J. (1930). Annual Report for 1929-1930; New Zealand Dept. of Agric., p. 14.

SEDDON, H. R., & McGRATH, T. T. (1930). Toxicity of Sodium chlorate. Austral. Vet. Jul., Vol. 6, No. 3, p. 112.

WITTHAUS, R. A. (1911): Manual of Toxicology, London. Ballière, Tindall & Cox. 2nd Edition, pp. 690-701.