

## Poisoning of Human Beings by Weeds con- tained in Cereals (Bread Poisoning.)

By D. G. STEYN, B.Sc., Dr. Med. Vet., Veterinary Research Officer, Onderste-  
poort.

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\* Only those plants marked with an x are indigenous.

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I. INTRODUCTION.

In this article the term "bread poisoning" signifies poisoning caused by the ingestion of bread prepared from wheat contaminated with extraneous seeds. Mention will, however, also be made of poisoning which is liable to occur when other articles of diet, e.g. beans and mealies, become contaminated with extraneous seeds.

The first report of an obscure disease known as "bread poisoning" was made to the Union Government Health Department in 1918 from Albertyn (Willmot and Robertson, 1920), although it had apparently been occurring in the Mossel Bay-George-Riversdale area for quite a number of years. Willmot and Robertson (1920) investigated the disease in the George district and suspected some form of poisoning. As the disease was most prevalent in families belonging to the poorer classes, whose diet consisted mainly of bread, their attention was directed to the wheat and bread consumed by the affected people. Willmot and Robertson at once suspected *Senecio* poisoning, as they knew that certain species of *Senecio* had been established as the cause of the Molteno Cattle Disease, Winton Disease in New Zealand, and Pictou Disease in Nova Scotia, which diseases, in common with the above disease in human beings, show cirrhosis of the liver.

They found *Senecio Burchellii* DC and *Senecio ilicifolius* Thunb. growing on the wheatlands and suggested that at the time of threshing the seeds and other portions of these weeds find their way into the wheat when threshing machines and mills not fitted with efficient winnowing appliances are used.

The ages of the patients examined by Willmot and Robertson varied from eleven to nineteen years and the period that elapsed from the onset of symptoms up to the time of death ranged from fourteen days to two years. The disease is most prevalent amongst young people, although both sexes of all ages may be affected.

The following are the symptoms, post-mortem appearances and histological changes as described by Willmot and Robertson and as verbally communicated to the author by Dr. Shanks of Humansdorp and Drs. van Zyl and G. Muir of Riversdale: A feeling of discomfort over the epigastrium, abdominal pain, nausea, vomiting, especially after meals, the vomit at times containing blood, ascites enlarged liver, apathy, extreme emaciation and diarrhoea with blood in the stools. In one of the Humansdorp cases Dr. Shanks saw very slight yellowish discolouration of the conjunctivae.

*Post-mortem appearances.*—Enlarged liver with well defined, slightly raised areas of a deeper colour than normal on the surface: on section these areas, which varied in size from a hazelnut to a walnut, were hyperaemic; in more advanced cases the liver, which may or may not be enlarged, showed cirrhotic changes and similar sized areas, which were of a lighter colour than the liver substance. In some cases the contents of the stomach, which as a rule was normal in size, were dark-brown. Dark coloured circular spots varying in size from a pin's head to a pea, were noticed on the gastric mucosa. On closer examination these spots were found to be small ulcers the bases of which appeared to be covered with blood.

The stomach may in some cases be dilated. The kidneys showed marked congestion, whilst the remaining organs appeared normal.

*Histology of the liver: Recent cases.*—The central veins and the capillaries between the hepatic cells are distended: the liver cells are reduced in size, some containing a brown pigment and others fatty particles.

*More advanced cases.*—These showed the usual round cell infiltration and increased formation of fibrous tissue as seen in cirrhosis of the liver from other causes.

Dr. G. de Kock, Head of the Department of Pathology, Onderstepoort, examined a portion of the liver of one of the suspected "Drabok" cases, which had occurred in the Clanwilliam District (Willmot and Silberbauer, 1931) and found the histological picture very similar to that seen in the livers of horses poisoned with *Senecio spp.* This liver specimen was kindly submitted by the Somerset Hospital, Capetown.

The majority of affected people die unless early treatment is applied and the causative agent removed. Numbers of people who suffer from ascites due to bread poisoning are "tapped" at fortnightly or three-weekly intervals and may recover. It stands to reason that once the liver has been severely damaged over prolonged periods pronounced irreparable changes (e.g. cirrhosis and degeneration), which markedly decrease the functions of this organ, will set in. It is hardly possible that such affected patients, if they recover from an acute attack, will ever return to the normal state of health. What is more, in such patients the function of the liver as chief detoxicator of the system, is very much inhibited, consequently such human beings will be more susceptible to the effects of poisons, both those of exogenous and endogenous origin.

Having suspected poisoning with *Senecio Burchellii* DC. and *Senecio ilicifolius* Thunb. Willmot and Robertson (1920) proceeded to conduct some feeding experiments upon guinea pigs and white rats. The dried flowerheads and "seeds" of the above two *Senecio spp.* were added to the diet of these animals. Willmot and Robertson reported as follows: "All the experimental animals became very emaciated, in spite of the fact that they consumed a normal amount of food. One guinea pig out of twelve under experiment died after feeding for ten weeks on various quantities of dried ground-up seed-heads

and tops of a plant identified as "*Senecio ilicifolius*." The post-mortem findings in this guinea pig were almost identical to those in the human subject referred to above, viz: liver mottled, showing to the naked eye well-marked areas of a lighter colour than normal, which on microscopic examination were found to be due to round celled infiltration both intra- and interlobular with the formation of new fibrous tissue. The stomach and upper part of the duodenum contained dark brown fluid (altered blood), and many small specks of blood were found adherent to the stomach wall, chiefly in the neighbourhood of the pyloric end; on washing the blood away, numerous minute ulcers could be made out with a hand lens."

Very similar lesions were found in three white rats which succumbed after having been fed daily for almost four months on three grams of ground-up heads of *Senecio ilicifolius* Thunb. and in one rat which had for three weeks received three grams daily of *Senecio burchellii* DC. The livers of the three rats fed on *Senecio ilicifolius* Thunb. showed cirrhosis, whilst the liver of the rat which had consumed *Senecio burchellii* DC. was congested but not cirrhotic.

The stomach and intestinal contents of all the rats were dark brown and blood-stained and minute pin-point ulcers covered with blood were detectable on the gastric mucosa.

Willmot and Robertson concluded their publication with the following remark:—

"We recognise the incompleteness of our investigations which were unfortunately interrupted by the epidemic of influenza which swept South Africa in 1918. Further enquiry and research are necessary, but it seems desirable to place our preliminary investigations on record."

On 15/2/28 two specimens (53657 and 53658) of wheat and one (specimen 53656) of meal were forwarded to Onderstepoort by the Chief, Division of Plant Industry, Pretoria, with the following remark (Onderstepoort File 144 2282): "These samples have all been examined but there appears to be no trace of *Senecio* present, in fact, the samples of wheat may be said to be clean."

These specimens of wheat and meal were submitted by the Magistrate, Riversdale, as the result of the death of a European girl at Corrente River, Riversdale District, from suspected *Senecio* poisoning. The suspected wheat was grown on the farm where the girl took ill.

Very small amounts of wheat and meal were submitted. Fungus-free seeds of darnel (drabok), barley and oats were found in the two samples of wheat. 200 grams of the meal (specimen 53656) fed to a rabbit caused inappetence, pronounced diarrhoea, extreme weakness, apathy and death within three days after ingestion. The post-mortem revealed marked hyperaemia and slight oedema of the lungs; colourless gelatinous infiltration of the pericardium, mesenterium and wall of the big intestine, the mucosa of which showed numerous small haemorrhages and an acute catarrhal gastro-enteritis. The histological examination of the heart, liver, and kidneys was negative.

150 and 80 grams of wheat (specimen 53657) were fed to two rabbits respectively. Both developed symptoms of severe gastro-intestinal irritation, the former dying within twenty hours and the latter within twenty-four hours after ingestion of the material.

As the samples of wheat and meal caused severe gastro-intestinal irritation and as arsenical poisoning is so common in South Africa the small amount of material that was left of each was examined for the presence of arsenic. The result was negative.

100 grams of wheat (specimen 53658) had no ill-effects on a rabbit.

Each of two control rabbits ingested 450 grams of ordinary wheat in the course of four days without suffering any ill-effects.

Unfortunately the small quantities of the above samples submitted only allowed of these few preliminary experiments being conducted.

More material was requested but with the fresh consignment of wheat no poisoning of rabbits could be induced with the result that the cause of poisoning by the original samples remained a mystery. The following seeds were found in this wheat: *Senecio* spp., a *Silene* sp., barley, oats, drabok, a *Rumer* sp., and *Raphanus* spp.

On 21/4/31 the Principal, Grootfontein School of Agriculture, Middelburg, Cape Province, forwarded to Onderstepoort two samples (405A and 406A) of wheat and two (405B and 406B) of meal, which had been submitted by Dr. Shanks of Humansdorp, who suspected *Senecio* poisoning. One European woman and her two children and four native servants were affected, exhibiting symptoms of gastric disturbances, namely, pain, vomiting and constipation (Onderstepoort File 144 68).

The suspected wheat was produced on the farm where the disease occurred. The samples of wheat, which were too small to allow of any reliable experiments being conducted, contained a large amount of drabok. A small amount of vetch seeds, barley, oats, *Senecio* flowerheads, and seeds of *Silene gallicia* L. were also present.

At the request of the Director of Veterinary Service two bags (specimens 1007 and 1008) of this suspected wheat were forwarded to Onderstepoort for further investigation. In this wheat a large percentage ( $\pm$  5 per cent.) of drabok *Lolium temulentum* L. was found, as well as a small amount of seed of *Lithospermum arvense* L.

Four rabbits ingested large amounts of this wheat in the course of fourteen days without any deleterious effects. In addition the drabok was sorted out and fed to a rabbit, which consumed 1,550 grams in the course of fifteen days without any ill-effects. 35 grams of the *Lithospermum arvense* L. seeds were drenched to a rabbit with negative results.

It was realised that no progress in the investigations of the problem of bread poisoning could be made by collecting specimens of wheat and meal and submitting them to laboratory tests at the time cases of suspected bread poisoning occur. As bread poisoning is essentially a chronic malady it stands to reason that the particular samples of wheat and meal collected at the time the disease is reported must not necessarily have been concerned in the causation of poisoning. The bread containing the harmful weeds may have been eaten weeks and even months before the disease is reported. The disease is mostly of a chronic nature and in addition disease is very rarely or never reported to the medical people until fairly pronounced symptoms have developed.

Another important fact which renders an investigation into the cause of bread poisoning of very little value at the time such cases are reported is that only some bags of wheat (meal), and of these again some more so than others, are contaminated with the seeds and portions of poisonous weeds.

In the Humansdorp, Riversdale and Clanwilliam districts the author has seen wheat lands infested with *Senecio Burchellii* DC. and *Senecio ilicifolius* Thunb. In quite a number of cases the *Senecio* plants were found growing luxuriantly amongst the wheat in a portion of the lands whilst none or very few of these plants were seen on the rest of these lands.

It was for the above reasons that the importance of investigating the prevalence of weeds on the wheatlands of farms, where suspected bread poisoning occurs, was realised.

The outcome of the co-operation between Sir E. N. Thornton, Assistant Health Officer and Director of Medical Services, Pretoria, and Dr. P. J. du Toit, Director of Veterinary Services, Onderstepoort, in their eagerness to solve this very serious problem, was that the author was requested to visit farms in the Humansdorp, Riversdale, and Clanwilliam districts, where cases of suspected bread poisoning had occurred, in order to study the occurrence and prevalence of poisonous weeds on the wheatlands concerned.

The following weeds were collected by the author on wheatlands in the Humansdorp, Riversdale, and Clanwilliam districts, where cases of suspected bread poisoning had occurred :—

BORRAGINACEAE.

*Lithospermum arvense* L.

CARYOPHYLLACEAE.

*Scleranthus annuus* L.

*Silene gallica* L.

COMPOSITAE.

*Crepis polyodon* Phillips.

*Osteospermum muricatum* E. Mey.

*Senecio arenarius* Thunb. (Clanwilliam only).

*Senecio burchellii* DC. (not in Clanwilliam).

*Senecio ilicifolius* Thunb.

*Senecio laevigatus* Thunb. (Riversdale only).

*Senecio rigidus* L. (Riversdale only).

*Senecio rosmarinifolius* L. f. (Riversdale only).

CRUCIFERAE.

*Raphanus* sp. (pink flower) (Humansdorp only).

*Raphanus raphanistrum* L.

EUPHORBIACEAE.

*Euphorbia helioscopia* L. (not in Clanwilliam).

*Euphorbia peplus* L. (not in Clanwilliam).

GRAMINEAE.

*Lolium temulentum* L.

LEGUMINOSAE.

*Vicia sativa* L.

PAPAVERACEAE.

*Fumaria officinalis* L.

POLYGONACEAE.

*Rumex acetosella* L.

PRIMULACEAE.

*Anagallis arvensis* L.

## SCROPHULARIACEAE.

*Hebenstreitia integrifolia* L.

It must be mentioned here that the plant which was identified by Hutchinson (Kew) and Pillans (Bolus Herbarium) as "*Senecio ilicifolius*" (teste Dr. J. Muir) and which occurs in the wheatfields in the Riversdale district, appears to be different from the plant of the Humansdorp, George, and Clanwilliam wheat-fields and which was also identified as *Senecio ilicifolius*. Field observations make it difficult to believe that the two are the same species. Specimens of Hutchinson's and Pillans's "*Senecio ilicifolius*" collected by the author on wheatfields at Corrente River, Riversdale, were identified by the Division of Botany, Pretoria, as *Senecio Rehmanni* Bolus.

It is with the Riversdale plant that Willmot and Robertson (1920) conducted their experiments on guinea pigs and white rats (teste Dr. J. Muir).

Muir (1928) made a valuable contribution to the study of *Senecio* spp. and other weeds in relation to the so-called bread poisoning, which occurred to an alarming extent in the Riversdale, George, and Mossel Bay districts.

Muir recorded forty-six species of *Senecio* growing in the Riversdale Division and of these he found the following as weeds on cultivated lands: "*Senecio burchellii*" (abundant), "*Senecio levigatus*" (fairly common), "*Senecio ilicifolius*" (abundant), "*Senecio rosmarinifolius*" (fairly common).

In the course of the above investigation the author made arrangements for large amounts of weeds which were liable to find their way into threshed wheat, to be forwarded to Onderstepoort for experimental purposes, but with a few exceptions, it was a difficult task to obtain any material at all.

The weeds and weed-seeds obtained and experimented with, as well as other plants, which may find their way into human foodstuffs, will now be discussed.

**II. PLANTS DISCUSSED IN THIS ARTICLE.**

## A. BORRAGINACEAE.

*Lithospermum arvense* Linn.

*Registered number*.—Onderstepoort Spec. No. 6118 (*b*): 19/2/32.

*Common and vernacular names*.—Cromwell-corncockle: clove bush; maaldjie bos.

*Origin*.—Wheatlands at Modderfontein, Humansdorp.

*Parts of plant tested*.—"Ripe seeds." When ground these emitted an unpleasant oily smell.

*Two rabbits*.—Each received per stomach tube a totale amount of 200 grams of these "seeds" in the course of sixty-eight days.

*Result*.—Negative.

No record of the toxicity of this plant could be found in the literature. Rosenthal (1862) mentions that the roots, which are of a reddish colour when the plant is immature, are used by the peasant girls of the Northern Countries as a "paint." Huseman and his co-publishers (1882) refer to the red colouring matter which had been isolated by Ludwig and Kromayer from the roots of *Lithospermum arvense* Linn.

The fruit of this plant is used medicinally in gonorrhoea, diarrhoea and as a cathartic (Dragendorff, 1898). Pammel (1911) states that *Lithospermum arvense* Linn. is very frequently attacked by the fungus *Puccinia rubigo-vera*.

## B. CARYOPHYLLACEAE.

*(a) Agrostemma Githago* Linn.

*Common and vernacular names.*—Koringroos: Corn cockle: Kornrade (German): corn rose: corn campion.

*Habitat.*—The Flora Capensis (Harvey and Sonder, 1859) describes it as a plant 1 to 2 feet high and having purple flowers: and mentions that it occurs in corn fields and was introduced from Europe. Burt-Davy (1926) records this plant as having occurred in a patch of European vetches at Groenkloof, Pretoria.

*History.*—Thunberg (1823) writes: "*Agrostemma githago* inter *Triticum satum* juxta Fransche Hoek et alibi, ex Europa advena. Versus finem anni floret."

Rosenthal (1862) states that this plant contains a saponin and an amorphous poisonous substance githagin, and that the seed is used as a diuretic, expectorant and anthelmintic and the root as a remedy for haemorrhoids and eczematata. At this early date the seed which frequently finds its way into wheat was known to be detrimental to health. Kobert (1906) refers to this plant at length. In the seed the poison (agrostemmasapotoxin) is situated in the "embryo and in the cotyledons and not in the seed protein." Horses, bovines, goats, dogs, cats, rabbits, fowls, doves, canaries, and rats are susceptible to this poison when administered per os or subcutaneously. Pigs appear to develop a tolerance to the poison when initial small doses are given.

The cheaper grades of flour in Europe are frequently adulterated with corn-cockle (Pammel, 1911). Pammel, quoting Millspough, cites a case of two calves which died after having been fed  $14\frac{1}{2}$  oz. of wheaten flour containing 30 per cent. and 45 per cent. of corn-cockle seeds respectively. These seeds cause severe gastro-intestinal irritation and death in ducks and geese. According to Pammel (1911) Chestnut stated that all parts of the plant are poisonous, but the kernel contains most poison. Pammel (1911) says: "The poisoning is generally produced by a poor grade of flour made from wheat containing cockle seeds. Machinery is used to remove these seeds from the wheat, but the difficulty of separating is so great that the result is not entirely accomplished." And again: "Flour containing a smaller amount has often been made into bread and eaten, sometimes with fatal results, the baking not always being sufficient to decompose the poison. The effect may be acute, or, if a small quantity of the meal is eaten regularly, it may be chronic. In the latter case it is sometimes known as a disease under the name of "githagism."

With regard to the amount of *Agrostemma Githago* Linn. seed required to cause poisoning Pammel (1911) again quoting Chestnut, says: "A person eating 1,200 grains of bread made from flour containing only one-half per cent. of corn-cockle seed would consume six grains of cockle seed, an amount which the author believes beyond a doubt to be poisonous in its effects."

Sapotoxin, the toxic principle of corn-cockle, is stated to be only partially destroyed by baking.

Long (1917) mentions that when ground-up with wheat these seeds impart a greyish tint and disagreeable odour to bread made from it. The various investigators have had different results with regard to the effects of corn-cockle seeds on domestic animals. Long, quoting Pesch, says *(a)* that the amount of poison in the seed varies: *(b)* animals develop a tolerance to the poison: *(c)*

the susceptibility of animals to the poison varies both with the species and the individual"; (d) young animals are more susceptible than older ones; (e) "it is believed that rodents and sheep are not susceptible; and, as far as is known, grown cattle are only slightly or not at all affected by the poison; (f) calves, swine, horses, and especially dogs, are more or less susceptible"; and (g) "concerning birds and fowls there is some doubt."

According to Long (1917) the toxic principle is a glucoside which has an acrid taste and which in the course of time has received the following names: Githagin, saponin, agrostemmin, sapotoxin, agrostemma-sapotoxin, and smilacin. Long, quoting Cornevin, gives the following as letha doses of corn-cockle seed per 100 lb. live weight of animal:

Calf	: 0.25 lb.
Pig	: 0.10 lb.
Dog	: 0.90 lb.
Fowl	: 0.25 lb.

According to Thomson and Sifton (1922) corn-cockle seed has caused so much trouble in the United States of America that in certain States laws have been passed prohibiting the marketing of feeds which are contaminated with even the smallest amounts of these seeds. Referring to these seeds they state that "before the days of modern machinery they often found their way into the flour with disastrous results."

Bernhard-Smith (1923) gives the active principle of corn-cockle as smilacin. De Wilde (1932) followed the saponin content of *Agrostemma Githago* and found that there was an increase in the amount of saponin as the plant ripened. Watt and Brandwyk (1930) also shortly refer to poisoning with corn-cockle in man and animal, quoting work done by Brandl, Brandl and Mayr, Wedekind and Knecke, and Wedekind and Schicke.

*Symptoms of poisoning.*—The following symptoms are described in the literature: Kobert (1906), Pammel (1911), Long (1917), Fröhner (1919), Thomson and Sifton (1922), and Pugh (1932).

(1) *Human beings: Chronic form.*—It occurs when small amounts of corn-cockle seed are taken over prolonged periods, and is almost invariably the only form met with in human beings. It is characterised by marasmus, weakness, dyspnoea, vomiting, abdominal pains, chronic diarrhoea, and affections of the nervous system.

*Acute form.*—Intense irritation of the gastro-intestinal tract, nausea, vomiting, headaches, diarrhoea, fever, also vertigo, pains in the spine, impaired locomotion, dyspnoea, delirium, and coma, which may be followed by death.

The poison is said to be destroyed by good baking of meal contaminated with corn-cockle seed.

(2) *Animals: Chronic form.*—It is rarely met with in animals except the pig. The symptoms closely resemble those met with in chronic corn-cockle poisoning in human beings.

*Acute form.*—Long (1917) describing the symptoms in the different classes of stock says: "In the horse, if a small quantity only is taken, there is yawning, heavy colic, stamping and evacuation of rather soft faeces. If larger quantities are taken, the symptoms, which commence in about an hour, are salivation, frequent yawning, and turning of the head, colic, pale mucus, hurried and weak pulse, rise in temperature and accelerated respiration. Some time later

there are muscular tremors succeeded by pronounced rigidity, and the faeces are diarrhoeic and foetid. The animal lies down and getting up is painful, it falls into a kind of coma, stretches itself to the utmost, and death takes place without convulsions."

In cattle symptoms were observed within one hour after ingestion of corn-cockle seed. The animals showed restlessness, salivation, grinding of the teeth, excitement, colic, coughing, followed within five to eight hours by a period of coma. Furthermore, there is permanent decubitus, repeated foetid diarrhoea, hurried and plaintive respiration, accelerated and progressively weakening pulse, progressive loss of motor and sensory powers and a gradual fall in temperature. Death may occur within twenty-four hours.

Pigs show grunting, salivation, nausea, vomiting, foetid diarrhoea, which may be bloody, clonic muscular spasms, paralysis, coma, and death.

In pregnant animals abortion may occur.

*Susceptibility.*—Young animals are more susceptible than full grown ones. Sheep, goats, and rodents (rabbits) appear not to be susceptible at all to corn-cockle poisoning; also full-grown cattle are very slightly susceptible. Most susceptible are dogs, horses, pigs, calves, and fowls.

*Post-mortem Appearances:* (1) *Human beings.*—In the available literature no mention is made of specific lesions, but presumably they will be those of a gastro-enteritis of variable degree and in chronic cases extreme cachexia with the characteristic lesions accompanying it.

(2) *Animals.*—Blood is dark and tarry in consistence; furthermore lesions characteristic of severe gastro-intestinal irritation are present; frequently also hyperaemia of the brain and spinal cord and exudates in the cavities of the central nervous system.

*Histology.*—No information with regard to the histology of the organs of victims of corn-cockle poisoning could be found in the available literature.

*Treatment.*—Symptomatic treatment (demulcents and stimulants) must be applied; Pamme! (1911) mentions that *Digitalis* antagonises the poisonous action of corn-cockle.

*Detection of corn-cockle in meal and gastro-intestinal contents.*—Fröhner (1919) describes methods, both botanically and chemically, of detecting corn-cockle in meal and in gastro-intestinal contents.

(b) *Silene gallica* L.

*Registered number.*—Onderstepoort Spec. No. 6118 (a), 19/2/32.

Nat. Herb. No. 14270.

*Common and vernacular names.*—Eierbossie; gunpowder weed.

*Origin.*—Wheatlands at Modderfontein, Humansdorp.

*Parts of plant tested.*—Ripe seeds and the dry plant in the late seeding stage.

Two rabbits received per stomach tube 235 grams and 940 grams of the ripe seeds (capsules + seeds) in the course of fifty-four days respectively.

*Result.*—Negative.

Sheep, 31599, received per stomach tube 4,600 grams of the whole plant in the dry state and late seeding stage in the course of eleven days.

*Result.*—Negative.

*History*.—The gunpowder weed is recommended as a snake-bite cure (Dragendorff, 1898). The following passage is quoted from Burtt-Davy (1926): “Two cases of horse poisoning, one at Johannesburg in 1909 and one at Bloemfontein in 1913, have been attributed to hay or forage containing a considerable admixture of this weed: in the first case the animal became listless, dull, without evidence of spirit and refusing to eat; in the latter case violent purging and colic resulted.”

## C. COMPOSITAE.

(a) *Centaurea picris* DC.

*Registered number*.—Onderstepoort Spec. No. 4594; 8/12/31.

*Common names*.

*Origin*.—On cultivated lands, Carolspoort, De Aar.

*Parts of plant tested*.—Whole plant; dry and in the flowering and early fruiting stage.

The owner of the farm Carolspoort suspected this plant of having caused mortality in sheep grazing on the harvested lands, where there was abundant growth of it. It is also maintained that during harvesting the people, handling crops contaminated with this plant, were affected. No symptoms were however, described.

This plant is also referred to in an article on “Plant poisoning in Stock and the development of Tolerance” published elsewhere in this report.

300 grams of the plant given per stomach tube to sheep on each of two consecutive days invariably caused salivation, hoven, pronounced laboured respiration, groaning, fever, diarrhoea, cyanosis, accelerated pulse, which became progressively weaker, apathy and death within eighteen hours after the second dose had been administered. Cyanosis, asystolic heart and pronounced hyperaemia of the lungs were found at autopsy. In some cases there was, in addition to the described lesions, hydroperitonium, hydrothorax, hydropericardium; dilatation of both heart ventricles; slight oedema of the lungs; acute catarrhal gastro enteritis with numerous haemorrhages in the mucous membrane of the small intestine; pronounced hyperaemia of and haemorrhages in the retropharyngeal, bronchial and mediastinal lymph glands, and degenerative changes in the liver.

*Histology*.—No specific microscopic lesions were detected in the myocard, liver, kidney, spleen and lymph glands.

In the course of the above experiments it was found that a high degree of tolerance could be induced in sheep by drenching the animals with non-toxic and increasing amounts of the plant.

*History*.—No reference to the toxicity of *Centaurea picris* DC. is made in available literature. Muir (1928) recorded the fact that *Centaurea melitensis* L. occurs on cultivated lands in the Riversdale district. The latter plant is used as a stomachic (Dragendorff, 1898).

According to Dopheide *Centaurea cyanus* L. (corn-flower) caused complete paralysis in a cow (Fröhner, 1919).

(b) *Senecio arenarius* Thunb.

*Registered number.*—Onderstepoort Spec. No. PN ; 15/11/31.

*Vernacular name.*—Hongerblom.

*Habitat.*—Cultivated lands, Ou-dam, Clanwilliam.

Repeated attempts were made to obtain some plant material for experimental purposes but without success. No reference to the toxicity of this plant is made in the literature.

(c) *Senecio Burchellii* DC.

*Registered number.*—Onderstepoort Spec. Nos. 4319 ; (17/11/31) and 114 ; (6/4/32).

*Vernacular name.*—Ragwort : sprinkaanbos (in some parts of the South-western Cape Province).

*Origin.*—Commonage, Humansdorp.

It was realised that in order to obtain the most reliable experimental results, it would be essential to use plants growing on wheatlands on farms where suspected bread poisoning occurs. As attempts to obtain *Senecio Burchellii* DC. from such sources failed, the Extension Officer of the Department of Agriculture stationed at Humansdorp was approached and he kindly collected and forwarded to Onderstepoort the plant used in the under-mentioned experiments.

*Part of plant tested.*—Whole plant. Dry and in the flowering and " seedling " state. The results of the experiments conducted at Onderstepoort are recorded in the following table :—

TABLE I.  
*Experiments with Senecio Burchellii, VC.*

Animal.	Method of Administration of Plant.	Period of Administration.	Total Amount of Plant Taken.	Loss in Weight of Animal.	RESULT.
Rabbit A.....	Per stomach tube...	11 days.....	20 gm. (at rate of 2 gm. daily)*	None.....	From the 4th day of experiment there was laboured respiration, apathy, accelerated heart beat and decreased appetite. These symptoms progressed until death on the 11th day of the experiment. <i>Post-mortem appearances.</i> —Vessels of conjunctivæ injected, hyperæmia and œdema of the lungs, congestion of the liver. <i>Histology.</i> —Congestion of the liver, no specific changes in myocard, kidney, and spleen.
Rabbit B.....	Per stomach tube...	172 days.....	772 gm. (i.e. 2 gm. daily for 102 days and 10 gm. daily for last 70 days of the experiment)	None.....	This animal appeared to be in normal state of health up to 27-6-32 when it was killed, i.e. 10 days after discontinuation of the experiment. <i>Post-mortem appearances.</i> —All organs appear normal. <i>Histology.</i> —Nothing abnormal.
Rabbit C.....	Per stomach tube...	174 days.....	944 gm. (i.e. 4 gm. daily for 104 days and 10 gm. daily for last 70 days of the experiment)	None.....	At no time were any symptoms of poisoning noticed except slight dyspnoea and inappetence. Animal was killed within 8 days after discontinuation of the experiment. <i>Post-mortem appearances.</i> —All organs appear normal. <i>Histology.</i> —Nothing abnormal.
Rabbit D.....	Per stomach tube...	95 days.....	820 gm. (i.e. 10 gm. daily for 95 days)	28 per cent.....	Progressive dysnoea, cachexia, inappetence, accelerated heart beat and apathy set in from the 6th day until death supervened on the 95th day of the experiment. At times a slight yellowish discolouration of the conjunctivæ, which were markedly injected, was seen. <i>Post-mortem appearances.</i> —Anæmia, cachexia, hydroperitœnum, pronounced œdema and hyperæmia of the lung, heart in systole, atrophy, congestion and cirrhosis of the liver, chronic catarrhal gastritis. <i>Histology.</i> —Liver: Hyperæmia, atrophy, interstitial hepatitis.
Rabbit E.....	Per stomach tube...	122 days.....	1,810 gm. (i.e. 40 gm. daily for 28 days and 10 gm. daily for last 94 days of experiment)	None.....	This animal developed no symptoms of poisoning. Killed within 14 days after discontinuation of experiment. <i>Post-mortem appearances.</i> — <i>Histology.</i> —Nothing abnormal.
Dog 1044 (6 months old) (mixed breed)	Per stomach tube...	92 days.....	400 gm. (i.e. 5 gm. daily for 92 days)	None.....	In the course of the experiment and up to the time of publishing this article (that is six months after the discontinuation of the experiment) no symptoms of poisoning were noticed in this animal.
Dog 1045 (10 months old)	Per stomach tube...	92 days.....	1,350 gm (i.e. 15 gm. daily for 62 days and 20 gm. daily for 30 days)	None.....	In the course of the experiment and up to the time of publishing this article (that is six months after the discontinuation of the experiment) no symptoms of poisoning were noticed in this animal.
Sheep 31599 (6 tooth)....	Per stomach tube...	14 days.....	5,200 gm. (i.e. 400 gm. daily)	None.....	In the course of the experiment and up to the time of publishing this article (that is six months after the discontinuation of the experiment) no symptoms of poisoning were noticed in this animal.

\* Except Sundays.

## POISONING BY WEEDS CONTAINED IN CEREALS.

Five rabbits, two dogs, and one sheep were used in this experiment. Of these animals only two rabbits died of which one (rabbit D) may possibly have succumbed to the effects of *Senecio Burchellii* DC., although this appears doubtful in view of the results obtained with *Senecio ilicifolius* Thunb. and *Senecio isatideus* DC. The experiments with the two latter plants proved that rabbits are much less susceptible than dogs to *Senecio* poisoning.

It is unfortunate that the experiments with *Senecio Burchellii* DC. could not be conducted with specimens of the plant collected on lands of farms where cases of suspected bread poisoning have occurred, as cultivation, fertilisation and type of soil may influence the toxicity of plants to a considerable extent.

It was attempted to drench dogs with larger quantities of the plant than those recorded in the above table, but vomiting invariably occurred after drenching young dogs with amounts exceeding 20 grams.

*Histology.*—Rabbit D, which had received 10 grams of the plant daily for 95 days, showed hyperaemia and atrophy of the liver and an interstitial hepatitis. No specific lesions were detectable in the livers of the remaining four rabbits.

*History.*—Chase, Verney, and Robertson were the first investigators to prove *Senecio Burchellii* DC., and *Senecio latifolius* DC. (now shown to be *S. retrorsus* DC) poisonous to horses. They attributed “dunsiekte” in horses to poisoning with these two *Senecio* spp. [Theiler, 1918 (a)]. Watt (1909) isolated two alkaloids (Senecifolin and Senecifolidine) from *Senecio retrorsus* DC. (then incorrectly named *Senecio latifolius* DC).

Cushny (1911), who experimented with these two alkaloids says: “The symptoms and post-mortem findings in animals poisoned with these alkaloids resemble so closely those described by Gilruth, Chase, Pethick and others, in cattle and horses, that there can be no question that the cause is the same in each and that Pictou, Winton, or Molteno disease is really more or less chronic poisoning with *Senecio* alkaloids.”

Theiler [1918 (a) and 1918 (b)] discussed at length *Senecio* poisoning in horses.

Willmot and Robertson (1920) fed one white rat for three weeks with *Senecio Burchellii*. On post-mortem this animal showed a congested but not cirrhotic liver (see Introduction).

Van Es and his co-publishers (1929) found “*Senecio fremonti*” and “*Senecio Riddellii*” poisonous to horses.

Craig, Kearney, and Timoney (1930) refer to the toxicity of *Senecio latifolius* DC. and *Senecio Burchellii* DC. (South Africa), and *Senecio Jacobea* L. (Ireland, Great Britain, Europe, New Zealand, Canada, and Asiatic Russia).

Jalving (1930) succeeded in producing liver lesions in calves by feeding them on “*Senecio aquaticus*” and “*Senecio Jacobea*.”

Further experiments were conducted by De Kock, Du Toit, and Steyn (1931) in order to ascertain whether “dunsiekte” in horses and *Senecio* poisoning were identical.

Ewart (1931) writes that Molteno disease due to prolonged feeding with '*Senecio latifolius*' may be caused by the saponin content of this plant.

On the farm Modderfontein, Humansdorp district, where several cases of suspected bread poisoning had occurred, the author found *Senecio Burchellii* DC. growing luxuriantly amongst the wheat in a corner of the land concerned. Only two specimens of *Senecio ilicifolius* Thunb. were present on this land.

The owner of this farm requested me to examine a horse, which had been ailing for the past few months. This animal exhibited symptoms which could not be distinguished from those produced at Onderstepoort by feeding and drenching horses with *Senecio retrorsus* DC. (*Senecio latifolius* DC., now *S. retrorsus* DC.) and *Senecio isatideus* DC.

This horse was stabled and allowed to run in a small land near the home-stead. The land was found to be heavily overgrown with *Senecio Burchellii* DC.

Manske (1932) has isolated the alkaloid retrorsine from "*Senecio retrorsus*" which he obtained from South Africa and the alkaloid jacobine from "*Senecio jacobaea*." Of great interest is that Manske found the former *Senecio* to contain 1.3 per cent. of alkaloid and the latter only 0.04 per cent. of alkaloid.

(d) *Senecio ilicifolius* Thunb.

*Registered number.*—Onderstepoort Spec. No. 6179: 23/2/32.

*Nat. Herb. No.* 14269.

*Vernacular names.*—Sprinkaanbos: kovanna (guano-) bos (Clauwilliam).

*Origin.*—D. Botha, George.

The plant material was collected from a land on a farm where cases of suspected bread poisoning had occurred.

*Parts of plant tested.*—Whole plant; dry and in the flowering and "seeding" stage.

The experiments conducted at Onderstepoort are recorded in the following table:—

POISONING BY WEEDS CONTAINED IN CEREALS.

TABLE 2.  
*Experiments with Senecio Nicifolius Thunb.*

Animal.	Method of Administration of Plant.	Period of Administration.	Total Amount of Plant Taken.	Loss in Weight of Animal.	RESULT.
Rabbit F. ....	Per stomach tube...	4 days.....	4 gm. (at rate of 1 gm. daily)	None.....	On the 4th day of the experiment the animal showed dyspnoea accelerated heart beat, and inappetence. It became very restless, pushed the head violently and repeatedly against the cage walls and died on the same day during such a fit. <i>Post-mortem appearances.</i> —Cyanosis, pronounced dilatation of both heart ventricles, hyperaemia and emphysema of lungs, hyperaemia of liver, which showed a marked resemblance to that in acute senesiosis in horses, stomach: Wall covered with thick greyish mucus and a dark reddish brown substance, which spectroscopically proved to be changed blood, in addition the stomach showed fairly extensive ulceration. <i>Histology.</i> —Liver: Slight hyperaemia and multiple localised necrosis. Myocard: Spleen and kidney showed no lesions.
Rabbit G. ....	Per stomach tube...	4 days.....	8 gm. (at rate of 2 gm. daily)	2.3 per cent....	Died on the 4th day of the experiment. Symptoms and post-mortem appearances as in rabbit F, with the exception of ulceration of the stomach. <i>Histology.</i> —Liver: Fatty changes, hyperaemia, central necrosis and several neutrophils present. Spleen, myocard, and kidneys showed no lesions.
Rabbit H. ....	Per stomach tube...	121 days.....	1,040 gm. (at rate of 10 gm. daily) *	—	Developed no symptoms of poisoning. Killed 10 days after discontinuation of the experiment. <i>Post-mortem appearances.</i> —All organs appeared normal. <i>Histology.</i> —Nothing abnormal.
Rabbit J. ....	Per stomach tube...	120 days.....	554 gm. (at rate of 1 gm. daily for 62 days and 10 gm. daily for 58 days)	—	No symptoms of poisoning developed. Killed on the day the experiment was discontinued. <i>Post-mortem appearances.</i> —All organs appeared normal. <i>Histology.</i> —Nothing abnormal.
Rabbit K. ....	Per stomach tube...	120 days.....	608 gm. (at rate of 2 gm. daily for first 62 days and 10 gm. daily for last 58 days)	—	No symptoms of poisoning appeared. Killed 14 days after discontinuation of the experiment. Animal gained in weight. <i>Post-mortem appearances.</i> —All organs appeared normal. <i>Histology.</i> —Nothing abnormal.
Rabbit L. ....	Per stomach tube...	120 days.....	716 gm. (at rate of 4 gm. daily for first 62 days and 10 gm. daily for last 58 days)	—	Apart from pronounced loss in weight and dyspnoea, no symptoms of poisoning were exhibited. Killed within 14 days after discontinuation of the experiment. Animal gained in weight. <i>Post-mortem appearances.</i> —All organs appeared normal. <i>Histology.</i> — Nothing abnormal.

\* Except Sundays.

TABLE 2.—(continued).

Animal.	Method of Administration of Plant.	Period of Administration.	Total Amount of Plant Taken.	Loss in Weight of Animal.	RESULT.
Dog 1042 (12 months old) (mixed breed)	10 gm. of ground-up plant mixed in food daily	90 days.....	Approximate 180 gm.....	27 per cent.....	<p>Diarrhoea was noticed on the 27th day of the experiment, then followed progressive loss in condition, anæmia, apathy, inappetence, repeated vomiting; <i>N.B.</i>—At no time was icterus seen. On the 91st day of the experiment the animal was found prostrate, vomiting foaming mucus these were chronic spasms of the front legs and at intervals of a few seconds clonic spasms of the neck causing the head to be drawn tight in between the front legs, the cornea reflex was decreased. Death occurred the same day.</p> <p><i>Post-mortem appearances.</i>—Pronounced cachexia and anæmia, marked hydroperitoneum (150 c.c.), pronounced cirrhosis (atrophic) and degeneration of the liver (not pigmented), extensive ulceration of the gastric mucosa, especially in the pyloric portion with hæmorrhages, dark reddish brown mucous substance in the stomach.</p> <p><i>Histology.</i>—Liver: Cirrhosis and degeneration. Blood and spleen smears.—Negative.</p>
Dog 1043 (9 months old) (mixed breed)	Per stomach tube and 10 gm. of ground-up plant mixed in food daily	45 days.....	Approximate 480 gm. (at rate of 10 gm. per stomach tube daily and approximate 90 gm. taken with the food)	37.5 per cent..	<p>Diarrhoea appeared on the 16th day of the experiment. There was progressive inappetence, cachexia, dyspnoea and apathy. Diarrhoea was followed by constipation and this again by diarrhoea, pronounced injection of conjunctivæ and eye ball vessels, pulse weak and accelerated, fever, staring coat, retching, vomiting, extreme cachexia and weakness; on the 45th day of the experiment there was slight general icterus, which was very pronounced on the day of death, which occurred on 47th day of the experiment.</p> <p><i>Post-mortem appearances.</i>—Pronounced general icterus, anæmia, extreme cachexia, blood very dark and not coagulated, slight hyperæmia of the lungs, diastolic heart, pronounced hydroperitoneum (185 c.c.), pronounced cirrhosis and degeneration of the liver, pronounced sub-acute catarrhal gastritis, pyloric portion of the stomach covered by a dark reddish brown mucous substance, which spectroscopically and chemically proved to be changed blood, a fair number of pi-point ulcerations were present on the gastric mucosa, subacute catarrhal enteritis, the intestine containing a large amount of dark reddish brown mucous substance, which proved to be changed blood. Straw and sand in stomach and intestine.</p> <p><i>Histology.</i>—Liver: Cirrhosis and degeneration. Blood and spleen smears.—Negative.</p>

POISONING BY WEEDS CONTAINED IN CEREALS.

TABLE 2—(continued).

Animal.	Method of Administration of Plant.	Period of Administration.	Total Amount of Plant Taken.	Loss in Weight of Animal.	RESULTS.
Dog 1047 (15 months old) (mixed breed)	Per stomach tube and 5 gm. of ground-up plant mixed in food daily	82 days.....	Approximately 435 gm. (at rate of 5 gm. per stomach tube daily and approximately 80 gm. taken with the food).	18.7 per cent..	<p>Diarrhoea appeared on the 19th day of the experiment. The symptoms markedly resembled those described in dog 1043. The conjunctivae showed a slight yellowish discoloration on the 79th day of the experiment. There was a pronounced general icterus on the day of death, which occurred on the 83rd day of the experiment.</p> <p><i>Post-mortem appearances</i>.—Intense general icterus, cachexia, pronounced hydroperitonium (200 c.c.), marked dilatation of both heart ventricles, tumor splenis with extensive haemorrhages, pronounced pigmentation (dark orange-yellow), degeneration, swelling and cirrhosis of the liver, haemorrhages in periportal lymph glands, straw and sand in stomach, haemorrhages in and slight ulceration of the pyloric portion of the stomach, subacute catarrhal enteritis, extensive haemorrhage into mucous membrane of colon.</p> <p><i>Histology</i>.—Liver: Cirrhosis and degeneration. Blood and spleen smears.—Negative.</p>
Dog 1051 (12 months old) (mixed breed)	5 gm. of ground-up plant mixed in food daily	51 days.....	30 gm.....	—	<p>Feeding of plant was discontinued on the 51st day of the experiment, when the animal had developed distinct symptoms of Senecio poisoning (loss in weight, diarrhoea, constipation, inappetence and pronounced anathy), in order to study the further course of the disease. Improvement in the condition of the animal set in within 14 days and the animal appeared to be in normal health within 10 weeks after the discontinuation of feeding <i>Senecio bicoloratus</i> Thunb.</p>

Six rabbits and four young dogs were used in the above experiments.

*Rabbits.*—Rabbits F and G which had received 4·0 and 8·0 grams of plant respectively, died on the fourth day of the experiment with symptoms of dyspnoea, inappetence, heart weakness and violent excitement (probably due to asphyxia). The following lesions were found at post-mortem: General cyanosis, dilatation of the heart ventricles; hyperaemia and emphysema of the lungs; hyperaemia and degenerative changes in the liver, and ulceration of the gastric mucosa in one rabbit. (F). The stomach of rabbit F contained a fair quantity of a dark reddish brown mucous substance, which spectroscopically and chemically proved to be changed blood.

*Histology.*—The liver of rabbit F showed slight hyperaemia and multiple localised necrosis whilst fatty changes, hyperaemia, central necrosis and several neutrophils were present in the liver of rabbit G. No microscopical lesions were detectable in the spleen, kidney and myocard of these rabbits.

The remaining four rabbits, which developed no symptoms of poisoning having after received 1,040 grams, 554 grams, 608 grams, and 716 grams respectively over prolonged periods, were killed within different periods, varying up to fourteen days, after discontinuation of the experiment. No macroscopic and microscopic lesions were present at post-mortem.

*Dogs.*—All four dogs, two of which were drenched and also received the plant in their food while the other two received the plant in their food only, developed symptoms of poisoning. Three of these dogs died after having showed the following symptoms: Progressive cachexia, general weakness, inappetence, apathy, anaemia, repeated vomiting, diarrhoea, constipation, weak and accelerated pulse, allotriophagia, dyspnoea, general icterus not in dog 1042. In addition to these symptoms dog 1042 showed clonic spasms of the front legs and, at intervals of a few seconds, clonic spasms of the neck.

*Post-mortem Appearances.*—Pronounced general icterus (dogs 1047 and 1013), anaemia, extreme cachexia, pronounced hydroperitonem, dilatation of both heart ventricles, hyperaemia of the lungs, pronounced cirrhosis, degeneration and pigmentation of the liver (*N.B.*—the liver of dog 1042 showed no pigmentation), subacute catarrhal gastro-enteritis, haemorrhages in and ulceration of the mucosa of the pyloric portion of the stomach, the stomach and intestine contained a dark reddish-brown mucous substance, which spectroscopically and chemically proved to be changed blood, in addition sand and straw were found in the gastro-intestinal tract, extensive haemorrhage into the mucosa of the colon.

*Histology.*—This aspect of seneciosis is being fully discussed by Dr. G. de Kock, head of the Department of Pathology, Onderstepoort Laboratories, in a paper dealing with the pathology of Senecio poisoning. It, therefore, suffices to state here that chronic *Senecio* poisoning in dogs is characterised by pronounced cirrhosis (atrophic) and degeneration of the liver.

From the above table it is evident that rabbits are much less susceptible than dogs to poisoning with *Senecio ilicifolius* Thunb. 180 grams of this plant taken by dog 1042 over a period of ninety days sufficed to cause death, whilst rabbits receiving quantities of plant varying from 554 grams to 1,040 grams developed no symptoms of poisoning.

In view of the insusceptibility of rabbits H, J, K, and L it is doubtful whether rabbits F and G succumbed to *Senecio* poisoning unless we except an enormous hypersusceptibility in these two cases.

It appears that dogs receiving very small quantities of the plant over prolonged periods are not liable to develop general icterus, while this symptom is very pronounced in dogs, receiving larger amounts of the plant over shorter periods.

Young dogs which have developed fairly distinct symptoms of *Senecio ilicifolius* Thunb. poisoning, will recover provided the feeding of the plant is discontinued and the liver has not been damaged beyond repair. It is, however, doubtful whether such damaged livers will completely recover as a certain amount of cirrhosis is bound to persist.

*History.*—Willmot and Robertson (1920) produced liver lesions in one of twelve guinea pigs and three white rats which had been fed with "*Senecio ilicifolius*" for almost four months (see Introduction). As explained previously Dr. J. Muir verbally informed the author that Willmot and Robertson conducted their experiments with "*S. ilicifolius*" collected on wheatlands at the farm Corrente River, Riversdale district. The author collected specimens of this plant in the presence of Dr. Muir (Riversdale). These specimens were identified by the Division of Botany, Pretoria, as *Senecio Rehmanni* Bolus. (N.H. No. 14267).

Willmot and Silberbauer (1931) describe four cases of ascites in male European adults and state "in view of the absence of gastric disturbance and pain associated with *Senecio* poisoning, Drabok poisoning was almost certainly the cause." These cases occurred on the farm Palmietfontein (Oudam), Clanwilliam district, and will be referred to more fully under *Lolium temulentum* L. (Drabok) and under "Discussion."

Muir (1931), who was invited by the Editor of the Journal of the Medical Association of South Africa to add a note to the article on "Darnel (*Lolium temulentum*) or Drabok Poisoning" by Willmot and Silberbauer (1931), writes: "I will merely note in passing that partial reliance by Drs. Willmot and Silberbauer on the absence of pain for the diagnosis of drabok poisoning is in conflict with the extract given above. It is further the experience of my colleague, Dr. J. W. van Zyl, District Surgeon here, that certain cases of *Senecio* disease occur where pain is not a prominent feature or is even absent."

In November, 1931, the author visited the above farm Palmietfontein (Oudam), Clanwilliam district, where the cases of suspected drabok poisoning (Willmot and Silberbauer, 1931) have occurred. On inspecting the land where the suspected wheat was grown, *Senecio ilicifolius* Thunb., locally known as kovanna-(guano)-bossie(bos) was found growing quite abundantly in one corner, whilst only a few specimens of this plant were found on the remaining portion of this land. In addition a neighbouring fallow land was overgrown

with *Senecio ilicifolius* Thunb. The owner (Mr. Smit) of the farm promised to forward two bags of this plant to Onderstepoort for experimental purposes, but in spite of repeated reminders the promise was not fulfilled.

As deaths in mules had been reported by Mr. van Zyl, Paardekop, a farm adjoining Palmietfontein, it was decided to investigate the cause of the disease. Twelve mules and one horse had died in the course of a few months after having shown symptoms (described by Mr. van Zyl) very closely resembling those produced at Onderstepoort in horses by feeding and drenching them with *S. retrorsus* DC. (*Senecio latifolius* DC.) and *Senecio isatideus* DC. There was abundant growth of *Senecio ilicifolius* Thunb. on the lands where the oats fed to the above affected animals were grown and Mr. van Zyl admitted that some of the sheaves of the suspected oats were heavily contaminated with this plant.

(c) *Senecio isatideus* DC.

*Registered number*.—Onderstepoort Spec. No. 5789 : 11/2/32.  
Nat. Herb. No. 10848.

*Common and vernacular names*.—Dan's cabbage : Inkanga (Zulu) : Poisonous ragwort.

*Origin*.—Greytown, Natal.

*Parts of plants tested*.—Whole plant ; dry and in preflowering stage.

*Senecio isatideus* DC. is of widespread occurrence in South Africa, namely, from Uitenhage through the Eastern Province and Natal into the Transvaal. It was, therefore, thought advisable to discuss here also the toxicity of this plant as it is quite possible that it may find its way on to wheatlands and in this way become a menace to the health of human beings.

In the following table are recorded the results of experiments conducted at Onderstepoort upon dogs and rabbits :—

POLSONING BY WEEDS CONTAINED IN CEREALS.

TABLE 3.  
Experiments with Senecio Isatideus DC.

Animal.	Method of Administration of Plant.	Period of Administration.	Total Amount of Plant Taken.	Loss in Weight of Animal.	RESULT.
Rabbit M. ....	Per stomach tube...	78 days.....	134 gm. (at rate of 2 gm. daily)*	17 per cent.....	Progressive inappetence, anaemia, dyspnoea, apathy, weak and accelerated heart beat and cachexia until death on the 134th day of the experiment. <i>Post-mortem appearances.</i> —Enaetation, flabby heart, hydroperitoneum, hyperaemia and oedema of the lungs, extensive haemorrhages into mesentery and subserosal tissues of big intestine, ulceration of gastric mucosa, atrophic cirrhosis of the liver. <i>Histology.</i> —Liver: Hyperaemia, multiple necrotic areas. Myocard spleen and kidney showed no lesions.
Rabbit N. ....	Per stomach tube...	13 days.....	48 gm. (at rate of 4 gm. daily)	8 per cent.....	Symptoms like those seen in Rabbit M, death occurring on the 13th day of the experiment. <i>Post-mortem appearances.</i> —Pronounced hydroperitoneum, flabby heart, liver swollen, nutmeg-like in structure. <i>Histology.</i> —Liver: Slight hyperaemia, multiple necrosis, slight increase in connective tissue. Myocard, spleen, and kidney showed no lesions.
Rabbit O. ....	Per stomach tube...	2 days.....	20 gm. (at rate of 10 gm. daily)	None.....	Apathy, inappetence, laboured respiration and death on the 3rd day of the experiment. <i>Post-mortem appearances.</i> —Cyanosis, slight hydroperitoneum, hydrothorax, pronounced hyperaemia of lungs, flabby heart, dark reddish brown (changed blood) mucus on gastric mucosa, liver swollen and dark red, spleen swollen. <i>Histology.</i> —Liver: Slight hyperaemia, no specific changes. Hyperaemia of kidney and spleen.
Rabbit P. ....	Per stomach tube...	34 days.....	300 gm. (at rate of 10 gm. daily)	22 per cent.....	Progressive apathy, inappetence, anaemia, dyspnoea, and cachexia until death on the 34th day of the experiment. <i>Post-mortem appearances.</i> —Hyperaemia and oedema of the lungs, very flabby heart, subserosal haemorrhages along gastro-intestinal tract, large number of small dark reddish brown spots and ulcers on gastric mucosa, liver: Consistence very firm and of a diffuse light greyish colour. <i>Histology.</i> —Nothing abnormal.

\* Except Sundays.

TABLE 3—(continued).

Animal.	Method of Administration of Plant.	Period of Administration.	Total Amount of Plant Taken.	Loss in Weight of Animal.	RESULT.
Dog 1041 (8 months old) (mixed breed)	Per stomach tube and 5 gm. of ground-up plant in the food daily	20 days.....	Approximately 110 gm. (at rate of 5 gm. per stomach tube daily for 20 days and approximately 1 gm. taken with the food daily)	37 per cent.....	Inappetence, apathy, alternating diarrhoea and constipation, ulcerative stomatitis, pronounced progressive emaciation, weak and accelerated pulse, dyspnoea, prostrate for last four days, showing regular spasmodic jerks of all four legs; the animal died in a comatose state on the 20th day of the experiment. <i>Post-mortem-appearances.</i> —Ulcerative stomatitis, extreme cachexia, pronounced degenerative changes in the liver, multiple hydronephrosis and cirrhosis of the kidney, ulcerative gastritis, dark brown mucous substance in small intestine. <i>Histology.</i> —Liver: Degenerative changes. Spleen of blood smears.—Negative.
Dog 1050 (8 months old) (mixed breed)	5 gm. of the ground-up plant mixed in the food daily	82 days.....	Approximately 50 gm.....	—	Symptoms of poisoning (except icterus) very similar to those described in Dog 1048 commenced on the 11th day of the experiment. Feeding of the plant was discontinued on the 92nd day of the experiment, when the animal had lost 30 per cent. of its weight and appeared very ill. Improvement was discernible within three weeks after discontinuation of the feeding of the plant. Three months later the animal appeared to be in normal health again.
Dog 1046 (12 months old) (mixed breed)	5 gm. of the ground-up plant mixed in the food daily	77 days.....	Approximately 50 gm.....	58 per cent.....	Symptoms as those described in Dog 1048, with the exception that icterus never seen in dog 1046. The animal died on the 78th day of the experiment. <i>Post-mortem appearances.</i> —Extreme cachexia and anaemia. <i>N.B.</i> no icterus, hydropertoneum, slight hyperaemia of lungs, extensive ulceration of pyloric portion of stomach, some ulcers having healed again; subacute catarrhal enteritis; pronounced cirrhosis (atrophic) of liver (no pigmentation); straw and sand in gastro-intestinal tract. <i>Histology.</i> —Liver: Pronounced cirrhosis.

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POISONING BY WEEDS CONTAINED IN CEREALS.

TABLE 3—(continued).

Animal.	Method of Administration of Plant.	Period of Administration.	Total Amount of Plant Taken.	Loss in Weight of Animal.	RESULT.
Dog 1048 (12 months old) (mixed breed)	Per stomach tube and 10 gm. of ground-up plant mixed in the food daily	31 days.....	Approximately 332 gm. (at rate of 10 gm. per stomach tube daily and 2 gm. taken with the food daily)	25 per cent.....	<p>Inappetence, yawning, progressive apathy and cachexia, alternating diarrhoea and constipation, dyspnoea, general icterus appearing 2 days before death, weak and accelerated pulse, retching and vomiting at intervals, death, preceded by a comatose state, occurred on the 31st day of the experiment.</p> <p><i>Post-mortem appearances.</i>—Intense general icterus, marked hydroperitonium, hyperaemia of the lungs, intense pigmentation, degeneration and cirrhosis of the liver, which was extremely firm in consistency, haemorrhages in and ulceration of gastric mucosa, acute catarrhal enteritis, which contained a large amount black blood-like mucous substance, which chemically and spectrophotically proved to be changed blood, straw and sand in gastro-intestinal tract.</p> <p><i>Histology.</i>—Liver: Cirrhosis and degeneration.</p>
Dog 1049 (14 months old) (mixed breed)	10 gm. of ground-up plant mixed in food daily	42 days.....	Approximately 84 gm.....	45.1 per cent..	<p>The symptoms resembled those in dog 1048. Slight icterus appeared on the 56th day of the experiment and increased in intensity up to the time of death, which occurred on the 62nd day of the experiment.</p> <p><i>Post-mortem appearances.</i>—Intense general icterus; pronounced hydroperitonium (500 c.c.); slight hyperaemia of the lungs; pronounced pigmentation (dark-orange yellow), degeneration, swelling and cirrhosis of the liver; dark greenish, mucous, flocculent bile; hyperaemia and ulceration of the pyloric portion of the stomach with haemorrhages, subacute catarrhal enteritis with a dark reddish brown mucous substance in the stomach and intestine, cirrhosis of the kidneys, extensive haemorrhages in mucosa of rectum, subpleural haemorrhages in left thoracic cavity.</p> <p><i>Histology.</i>—Liver: Pigmentation, degeneration, and cirrhosis.</p> <p>Blood and spleen smears.—Negative.</p>
Dog 1082 (16 months old) (mixed breed)	5 gm. of ground-up plant mixed in food daily	18 days.....	Approximately 20 gm.....	—	<p>On the 13th day of the experiment diarrhoea appeared and a certain degree of inappetence and listlessness was present, loss in condition was noticeable and feeding of the plant was discontinued on the 18th day of the experiment in order to study the further course of the disease. Marked improvement in the condition of the animal had occurred three weeks after discontinuation of feeding of the plant and the animal appeared quite normal within a further six weeks.</p>

*Rabbits.*—All four rabbits drenched with *Senecio isatideus* DC. died after having exhibited the following symptoms: Pronounced cachexia (except rabbit O, which died on the third day of dosing), progressive inappetence, anaemia and apathy, laboured respiration, and weak and accelerated heart beat.

*Postmortem appearances.*—Emaciation (except rabbit O) hydroperitoneum, hydrothorax, pronounced hyperaemia and slight oedema of the lungs, extensive haemorrhage into the mesentery and subserosal tissues of the big intestine; ulceration of the gastric mucosa: dilatation of both heart ventricles; atrophic or hypertrophic cirrhosis of the liver with no pigmentation; hyperaemia of kidneys and spleen.

*Histology.*—Liver: hyperaemia, multiple necrotic areas, cirrhosis.

*Dogs.*—Both the dogs that had been drenched and fed and those that had been fed only died from *Senecio isatideus* DC. poisoning. There is, however, some doubt in the case of dog 1041, which died from uraemia caused by severe disease of the kidneys. Whether the kidney lesions described were caused by the plant or not, is impossible to say.

The following symptoms were developed by dogs 1050, 1046, 1048, and 1049: Progressive cachexia, inappetence and listlessness, anaemia, diarrhoea, yawning, constipation, retching, vomiting, allotriophagia, general icterus (not seen in dogs 1050 and 1046), weak and accelerated pulse: death (dogs 1046, 1048, and 1049) was preceded by coma.

*Post-mortem appearances.*—(Dogs 1046, 1048, and 1049): Extreme cachexia intense general icterus (absent in dog 1046), pronounced hydroperitoneum, hyperaemia of the lungs, pronounced pigmentation (absent in dog 1046), degeneration and cirrhosis of the liver, extensive ulceration of and haemorrhages in the mucosa of the pyloric portion of the stomach: subacute catarrhal enteritis, straw and grit in gastro-intestinal tract, the stomach and intestine contained a dark reddish-brown mucous substance, which proved to be changed blood.

*Histology.*—Liver: Cirrhotic and degenerative changes are characteristic of chronic *Senecio isatideus* DC poisoning in dogs.

From the above table it appears that rabbits, although susceptible to *Senecio isatideus* DC. poisoning, require much larger quantities of the plant to cause death than do dogs.

There is a marked resemblance in the symptoms and post-mortem lesions in dogs poisoned by *Senecio ilicifolius* DC. and in those poisoned by *Senecio isatideus* DC. As in *Senecio ilicifolius* DC. poisoning it was found that small amounts of *Senecio isatideus* DC. administered over prolonged periods did not produce general icterus, which invariably occurred in dogs taking larger amounts over shorter periods.

Dogs poisoned with *Senecio isatideus* DC. are liable to recover provided the administration of the plant is discontinued before the liver has been damaged beyond repair.

*History.*—Steyn (1931) proved *Senecio isatideus* DC. toxic to sheep and a horse. The latter animal exhibited symptoms which were indistinguishable from those of natural cases of "Dunsiekte" in horses.

(f) *Senecio laevigatus* Thunb.

(g) *Senecio rigidus* L.

(h) *Senecio rosmarinifolius* L. f.

Muir (1928) and also the author have collected these three species of *Senecio* on wheatlands in the Riversdale district. What is more they were present on lands on farms where cases of suspected bread poisoning occurred.

Marloth (1917), records *Senecio rigidus* L. under the common name of "poisonous ragwort."

Watt and Brandwyk (1932) state that they have proved *Senecio rigidus* L. poisonous to a rabbit. From experiments conducted at Onderstepoort on a large number of rabbits it would appear that rabbits are the animals least suitable for use in the determination of the toxicity of *Senecio* spp.

There is a greater possibility of *Senecio laevigatus* Thunb. and *Senecio rosmarinifolius* L. f. finding their way into threshed wheat than there is of *Senecio rigidus* L. doing so, as the latter plant is very tall and is almost exclusively found growing in very moist patches of the lands.

#### CRUCIFERAE.

##### *Raphanus raphanistrum* L.

*Registered number.*—Onderstepoort Spec. No. 6628R, 9/5/32.

*Nat. Herb. No.* 14274.

*Common and vernacular names.*—Knopherik, ramenas, jointed or white charlock, wild radish, field wall-flower.

*Origin.*—Sorted from a bag of wheat obtained from the Langkloof Roller Mills, Joubertina.

*Parts of plant tested.*—Ripe seed (+capsule).

Rabbits were drenched as follows:—

Rabbit A.—Received 105 grams of the above seed in the course of twenty-two days at the rate of 5 grams daily (except Sundays).

Rabbit B.—Received 225 grams of the above seed in the course of seventeen days at the rate of 15 grams daily (except Sundays).

Rabbit C.—Received 360 grams of the above seed, moistened twelve hours before dosing, in the course of twenty-seven days at the rate of 15 grams daily (except Sundays).

Rabbit D.—Received 310 grams of the above seed, moistened twelve hours before dosing in the course of thirty-six days at the rate of 10 grams daily (except Sundays).

*Result.*—Rabbit A died on the twenty-seventh day, Rabbit B on the seventeenth day, Rabbit C on the twenty-seventh day, and Rabbit D on the thirty-sixth day of the experiment after having exhibited inappetence, apathy and diarrhoea with gradual loss in condition.

*Post-mortem appearances.*—Hyperaemia and slight oedema on the lungs, pronounced subacute catarrhal gastro-enteritis and, in one case, subserosal haemorrhages in the peritoneal cavity.

*Histology.*—No specific lesions were found in the liver, kidneys, and heart.

*History.*—The seed of *Raphanus raphanistrum* L. contains small amounts of an irritant substance. The seed is used in tympanites, rheumatism and sciatica (Dragendorff, 1898).

Kobert (1906) writes that according to Sjollema *Brassica napus* L. and *Brassica rapa* L. contains more than one glucoside, which all develop mustard oil. These mustard oils are responsible for the production of chronic enteritis with tympanites, haemorrhagic diarrhoea, colic, stimulation of the brain, and abortus when horses and cattle ingest rape cakes over prolonged periods. Haematuria and the accumulation of haemorrhagic fluid in the thorax and peritoneal cavity were also seen. Kobert furthermore mentions that probably similar glucosides are contained in *Raphanus raphanistrum* L. which is known to have caused poisoning in animals.

Long (1917) writes: "Wild radish (*Raphanus raphanistrum* L.). As in the case of charlock, the seeds of wild radish are very acrid, and susceptible of introducing intestinal troubles if eaten by animals when mixed with cereals."

Fröhner (1919) states that *Raphanus raphanistrum* L. is one of the plants which, owing to its containing mustard oil or similar substances, will cause poisoning when ingested in large amounts.

Elaine (1922) described poisoning in horses with grain, which contained a third or a fourth part of the seeds of the wild radish. The horses showed colic and inappetence and recovered after treatment.

Wehmer (1929) writes that the seed of *Raphanus raphanistrum* L. contains 30 to 40 per cent. of fatty oil and no sinigrin, but a sinalbin-like sulphur-containing glucoside and myrosin.

Petri (1930) states that mustard oils are excreted by the lungs and kidneys and that they are severe irritants to the mucous membranes, and skin. Haematuria may be caused after the ingestion of mustard and rape cakes. The liver of animals poisoned with mustard oil are yellowish grey and soft in consistency, and microscopically show necrosis of the parenchyma. Petri mentions that Carlau saw disintegration of the liver and haemorrhages in guinea pigs and rabbits which had succumbed to mustard oil poisoning.

#### E. EUPHORBACEAE.

##### (a) *Euphorbia helioscopia* L.

*Registered number.*—Onderstepoort Specimen No. 5179; 9/10/30.  
Nat. Herb. No. 14267.

*Vernacular names.*—Melkgras; melkbos; wolfsmelk; milkweed; spurge.

*Origin.*—Cultivated lands, vicinity of Capetown.

*Habitat.*—Cultivated lands southern and western Cape Province.

*Parts of plant tested.*—Whole plant; fairly fresh and tested in the pre-flowering, flowering, and seeding stages.

It was suspected of having caused poisoning in stock running on cultivated lands. Three sheep received respectively per stomach tube 2,000 grams, 2,100 grams, and 1,750 grams of the plant in the different stages of development in the course of a few days without suffering any ill-effects (Steyn, 1931 and 1932).

*History.*—The plant itself as well as the "bark" was used as a purgative under the name of Herba et Cortex Esulae vel Tithymali. Latterly the latex, which is slightly irritant, was used in the treatment of syphilis (Rosenthal, 1862). Dragendorff (1898) states that this plant is poisonous and Kobert (1906) that it causes poisoning in children.

Pammell (1911) writes that Lehmann lists "*Euphorbia helioscopia*" as a poisonous plant.

According to Long (1917) sun spurge (*Euphorbia helioscopia* L.) has caused fatal poisoning to a boy who ate it" and in Germany cows were poisoned through pasturing in stubble in which the plant was growing, but there were no deaths."

Fröhner (1919) states that the following species of *Euphorbia* are toxic to stock: "*E. cyparissias*," "*E. peplus*," "*E. helioscopia*," "*E. marginata*," and "*E. lathyris*," and that their toxic properties are due to euphorbin-acidanhydride. The milk of goats which has partaken of *Euphorbia helioscopia* L. is stated to have caused poisoning in human beings. Bernhard-Smith (1923) mentions euphorbin as the toxic principle of *E. helioscopia* L. and *E. peplus* L.

*E. helioscopia* L. has been reported to cause constipation and narcosis in stock (Onderstepoort File 144/512, 14/9/28).

Dunning (Onderstepoort File 144/250, 10/9/31) writes that in a feeding experiment a sheep ingested 161 ounces of this plant in the young seeding stage without developing any symptoms of poisoning.

(b) *Euphorbia peplus* L.

*Vernacular names.*—Wolfsmelk, spurge.

*Habitat.*—Cultivated lands southern and western Cape Province.

*History.*—In the early centuries this plant under the name of Herba Esulae rotundifoliae was used as a remedy for dropsy (Rosenthal, 1862). According to Kobert (1906) it causes blisters on the skin and inflammation of the mouth and intestine and according to Pammel (1911) Lehmann records it as a poisonous plant. Long (1917) and Fröhner (1919) also refer to the toxicity of this plant.

Seddon (1929) produced salivation and blood stained faeces, but not death, in a calf drenched with a watery extract of 4 lb. of the fresh green plant in the early flowering stage.

*Symptoms of Euphorbia poisoning.*—On the skin the latex causes itching, redness, pimples, and in bad cases gangrene. The seeds cause symptoms of severe gastro-intestinal irritation, namely, inappetence, salivation, nausea, constipation, vomiting, pronounced diarrhoea, which may be haemorrhagic, colic, tympanites, palpitation of the heart, apath, dizziness, convulsions, unconsciousness, collapse and death.

*Post-mortem appearances.*—Severe acute catarrhal or haemorrhagic gastro-enteritis with ulceration of the mucosa.

(c) *Ricinus communis* L.

*Common names.*—Kasterolie boom, castor oil tree.

*Habitat.*—A native of Southern Asia; ubiquitous; on cultivated lands.

As the castor oil plant is of frequent occurrence on cultivated lands there is a possibility of its seed contaminating mealies, beans, etc., especially those foodstuffs grown by natives and irresponsible Europeans. The danger is greatest when the harvested crops are stacked and threshed on lands where castor oil plants grow, as the possibility of contamination is greater than when the crops are threshed outside such infested lands.

It has frequently occurred that maize stored in the same hold of the ship as castor oil seed has become mixed with the latter with the result that serious mortality has occurred in cattle and horses (Legal Notes, 1931 and 1932).

With regard to cakes and meals as feedingstuffs for stock a content of 0.02 per cent. seed is regarded as dangerous.

*History.*—Rosenthal (1862) states that *Ricinus communis* L. occurs in many varieties of which one of the most wellknown ones is *Ricinus africanus* Willd. Castor or ricinus oil is one of the most extensively used mild oily purgatives.

In 1864 Tuson mentioned that the seeds of *Ricinus communis* L. contains ricinin and described a method of isolating it (Husemann, 1882).

Castor seeds also contain the toxalbumin ricin; the root of this plant is used in kidney troubles and the leaf in abscesses (Dragendorff, 1898).

The seeds contain colouring matter and a large amount of oil, proteins, enzymes, and ricin (Kobert, 1906).

Long (1917) writes that “according to Cornevin four seeds suffice to cause accidents in man, eight lead to very grave results and beyond that number death may ensue.” He also mentions that the seeds have been found as an impurity in linseed cake and maize meal.

Pammel (1911), Long (1917), Fröhner (1919), Byam and Archibald (1921), Thomson and Sifton (1922), Lander (1926), and Petri (1930) all refer to the toxicity of the castor oil plant.

Heffter (1924) refers extensively to the methods of isolating ricin from castor seeds and to its actions in vitro and in vivo.

Cases of asthma resulting from the inhalation of castor bean dust in a castor oil factory have been described by Figley and Elrod (1928).

Ratner and Gruehl (1927–1928) produced anaphylaxis in guinea pigs by allowing them to inhale castor bean dust and injecting them intravenously with an extract of this dust after an incubation period of three weeks. Some guinea pigs died from ricin poisoning caused by the inhalation of castor bean dust and showed a severe haemorrhagic condition of the lungs.

Dodd (1932) made a valuable contribution to the study of the presence of castor seed in feedingstuffs. Dodd realises that the determination of the percentage of castor seed in feedingstuffs is not of great value as in practically all cases the castor seed is unevenly distributed. As new contracts with regard to feedingstuffs specify limits of contamination with castor seed, analysts are, however, forced to state percentages. With regard to the possibility and probability of the various castor seeds varying in toxicity Dodd rightly remarks that all castor seeds should be regarded as deadly. He describes the method employed by him in the detection and identification of castor seeds in cakes and states that seeds which might easily be mistaken for castor include grape or raisin seed, neuhuba seed, croton seed and curcas seed. The two former seeds are harmless whilst the two latter are more poisonous than castor seed. He says: “It is usual to return castor, croton or curcas as castor seed.” The percentage of castor seed present in the cakes is calculated by multiplying the weight of the husk found by 5.

*Symptoms of poisoning.*—According to the above-mentioned authors and Bornemann (1922) and Mariott (1922) the following are the symptoms of castor bean poisoning in human beings and animals.

(1) *Human beings*.—The symptoms are those of very severe gastro-intestinal irritation, namely burning pains in the throat and stomach, salivation, nausea, vomiting, colic, diarrhoea, thirst, small rapid pulse and also cramps of the calves and abdominal muscles, drowsiness, measles-like eczematata, cyanosis, and icterus.

*Post-mortem appearances*.—Acute gastro-enteritis sometimes accompanied by erosions in the stomach.

(2) *Animals*.—Haemorrhagic enteritis with its accompanying symptoms, staggering, dullness of vision, marked apathy, heart weakness, paralysis, somnolence, convulsions, muscular spasms, fever, shivering, coma and death. In some cases toxic laminitis was noticed. Death occurs within one to three days in acute poisoning.

*Post-mortem appearances*.—Acute gastro-enteritis; haemorrhages in the cortex of the kidneys and subpleural tissues of the lungs; degeneration of the myocard; subendocardial and subepicardial haemorrhages; haemorrhages in the serosa of the body cavities and in the organs; haemorrhagic ascites.

*Histology*.—Fatty degeneration of the myocard, congestion haemorrhages and accumulation of fat in the liver and irregular areas of parenchymatous disintegration; chronic poisoning causes cirrhosis of the liver; spleen shows increased pulp but owing to the disappearance of the lymphocytes the follicles are few and small, the haemorrhages and areas of disintegration in the spleen are due to occlusion of the capillaries by fibrin; the bonemarrow is very soft and mottled due to hyperaemia and haemorrhages; the marrow appears to produce abnormal blood elements; the circulating blood shows eosinophile leucecytosis and polychromasia (due to damaged blood forming apparatus); the kidneys, which excrete ricin, show fatty degeneration and necrosis of the tubuli epithelium.

*Toxic principle*.—The active principle of castor oil seed is the toxalbumin ricin, which is a very severe irritant and is much more poisonous when administered parenterally, than when taken per os. By reason of its proteid character ricin when injected subcutaneously produced an immunity to castor bean poisoning.

*Toxicity of castor seeds*.—Miessner (Fröhner, 1919) gives the following quantities as the lethal doses of castor bean seed administered in one dose:—

Horses.....	0·1	gram per Kg. body weight.
Cattle.....	2·0	” ”
Sheep.....	1·25	” ”
Goats.....	5·5	” ”
Pigs.....	1·4	” ”
Rabbits.....	1·0	” ”
Geese.....	0·5	” ”
Fowls.....	14·0	” ”

If the feeding of small quantities of castor bean seed is continued it has a cumulative action.

The seed is much more poisonous when administered subcutaneously.

At Onderstepoort the fresh immature seeds were found to be highly toxic to rabbits.

*Treatment*.—Symptomatic treatment must be applied as no specific antidote is known.

*Detection of castor bean in foodstuffs*.—This can be done botanically and biologically by the precipitation and conglutination tests.

## F. GRAMINEAE.

(a) *Lolium temulentum* L. var. *macrochaeton* A. Br.

*Registered No.*—Onderstepoort Spec. No. 6628 Q; 22/3/32. Nat. Herb. No. 14265.

(b) *Lolium temulentum* L. var. *muticum* Boiss.

*Registered No.*—Onderstepoort Spec. No. 6574; 22/3/32. Nat. Herb. No. 14266.

*Common names.*—Drabok, darnel, cheat, "Tares" of the Bible. German: Taumellolech, Tobkrout.

*Habitat.*—Occurs extensively in cultivated lands especially in the southern and western Cape Province.

*Origin.*—Spec. 6628 Q (N.H. No. 14265) sorted from a bag of wheat contaminated with weed seeds obtained from the Langkloof Roller Mills, Joubertina.

*Specimen 6574* (N.H. No. 14266).—Sorted from a consignment of wheat contaminated with weed seeds obtained from the Roller Mills, Riversdale.

*Specimen 6628Q.*—These darnel seeds were undersized and discoloured and had a mouldy smell. The degree of fungus infection was 100 per cent. When chewed they at first had a slight sweetish starchy taste, which after a few minutes became bitter.

Dr. A. C. Leemann of the Division of Plant Industry examined this specimen of drabok mycologically. The results of his investigation are incorporated in an article published elsewhere in this report.

Two rabbits ingested 3,370 grams and 3,225 grams of these fungus-infected darnel "seeds" in the course of thirty-eight days respectively. No additional food was given to these rabbits during the period of experimentation.

*Result.*—Not only did these "seeds" prove harmless but they provided also in all the necessary nutritive substances essential for the maintenance of health and growth of the experimental rabbits.

Pig 789 ingested 4,000 grams of this fungus-infected drabok in three days without suffering any ill-effects. No additional food, except a small quantity of milk mixed with the drabok meal was given.

Dog (no number) took 1,250 grams of this drabok meal in the course of ten days without any detrimental effects. The raw meal was made into porridge with a small quantity of milk.

The above experiments would have been conducted over longer periods had more drabok been available.

*Specimen 6574.*—These darnel "seeds" were of normal size and appeared healthy. Three rabbits ingested in the course of seventy days 5,585 grams, 4,655 grams, and 6,065 grams of the "seeds" respectively. No additional food was given.

*Result.*—These seeds also proved harmless and provided all the food requirements essential for the maintenance of health and growth of the experimental animals.

In addition to the above experiments with darnel a number of other feeding experiments with the seed have been conducted on rabbits and horses at Onderstepoort in the course of the last five years, with negative results.

*History.*—For centuries this plant has been regarded as harmful to health. It is held to be the “tares” mentioned in the Bible which were sown amongst the enemy’s wheat.

Huseman and his co-publisher (1882) mentions that an impure bitter substance, named loliin, had been isolated from the seed of *Lolium temulentum* L.

Dragendorff (1898) states the seeds to be poisonous and that the meal was used as an analgesic and in the treatment of skin diseases. The seeds contain temulentin, loliin and temulentie acid and according to Hofmeister temulin.

Kobert (1906) correctly remarks that many of the cases of poisoning in Russia, Germany, France, etc., ascribed to darnel could have been caused by other extraneous material contained in the wheat.

The cases of darnel poisoning referred to by Willmot and Silberbauer (1931) will be dealt with under “Discussion.”

For further references see Leemann’s article: “A short summary of our botanical knowledge of *Lolium temulentum* L.” published elsewhere in this report.

*Symptoms of poisoning.*—[Kobert (1906); Pammel (1911); Long (1917); Fröhner (1919); Byam and Archibald (1921); Thomson and Sifton (1922); Lauder (1926)].

(a) *Human beings.*—Pronounced apathy; sleepiness; staggering; giddiness; trembling; mydriasis; a feeling of pressure in the epigastrium; nausea; vomiting; and later, painful cramps of the stomach; diarrhoea; heartweakness.

Barger (1931) (p. 29) writes: “In Germany and elsewhere the darnel (“*Lolium temulentum*,” zizania, the tares of Scripture?) was considered by some to be the cause of the Kriebelkrankheit and more plausibly, since this grass does indeed contain a narcotic poison. Husa observed a number of cases of actual poisoning by rye containing 16 to 22 per cent. of darnel seeds; the symptoms were frontal headache, giddiness, rumbling in the ears, gastric pains, twitching of the tongue, difficulty in swallowing and in speech, vomiting, diarrhoea, fatigue, cold sweat, and trembling of the limbs. The patients declared that they felt completely intoxicated. There is here a slight resemblance to some of the symptoms of ergotism, but various observers agree that the effects of “*Lolium*” poisoning are of short duration; after a sound sleep Husa’s patients were practically normal next day.”

*Post-mortem appearances.*—Darnel very rarely or never causes death in human beings. No specific lesions are described in the available literature.

(b) *Animals.*—Mydriasis; vertigo; uncertain gait; trembling; laboured respiration; slow and small pulse; convulsive movements of the head and limbs; paralysis (in pigs); unconsciousness; colic; spasms.

*Post-mortem appearances.*—As a rule the post-mortem is negative; rarely slight gastro-enteritis; hyperaemia of the lungs and hyperaemia of the brain and spinal cord are seen.

*Toxic principle.*—It is at present generally held that darnel is poisonous only when infected with fungi. According to Wehmer (1929) the active principle of darnel is the alkaloid temulin.

This plant is referred to in the article “Fungi in Relation to Health in Man and Animal” published elsewhere in this report.

*Toxicity of darnel.*—Cornevin's (Lander, 1926) lethal doses are :—

Horse.....	7 grams per Kg. body weight.
Dog.....	18           "           "

Ruminants and birds are supposed to be less susceptible.

Pammel (1911), quoting Hackel, says that the toxicity of darnel is due to a narcotic principle, loliin, which causes trembling, eruptions, and confusion of sight in man and flesh-eating animals, and particularly in rabbits, but it has no effect on swine, horned cattle or ducks.

*Treatment.*—Symptomatic.

#### G. LEGUMINOSAE.

##### *Vicia sativa* L.

*Registered number.*—Onderstepoort Spec. No. MN ; 18/4/32. Nat. Herb. No. 14271.

*Vernacular names.*—Common vetch ; wilde wieke ; "tares."

*Origin.*—Seed collected on the suspected wheatland at Modderfontein, Humansdorp, and sown at Onderstepoort. Experiments were conducted with the seed collected at Onderstepoort.

On moistening the ground up seed with water a typical bitter almond smell was emitted, and the picrate paper test for hydrocyanic acid was strongly positive. Dr. C. Rimington, Onderstepoort, found these seeds to contain 64·8 mgm. of hydrocyanic acid per 100 grams of seed.

A rabbit drenched with 20 grams of these seeds developed typical symptoms of hydrocyanic acid poisoning within five minutes and died within one and a quarter hours of dosage.

The post-mortem appearances were those of hydrocyanic acid poisoning. A large amount of this acid was detectable in the stomach contents by the picrate paper test.

*History.*—*Vicia sativa* L. is cultivated as a fodder plant especially for the feeding of cattle. Husemann and his co-publishers (1882) described methods of isolation of vicin and convicin from *Vicia sativa* L. peas. When vicin is heated in dilute sulphuric acid divicin sulphate crystallises out on cooling.

Dragendorff (1898) mentions that among other things cholin and betain are contained in the seed of this plant.

Kobert (1906) writes that *Vicia sativa* L. has caused poisoning in horses, cattle, and pigs with symptoms similar to those of lupinosis. Horses fed with this plant developed pronounced icterus and at post-mortem there was enlargement of the liver, which was of an orange yellow colour. In other cases horses showed loss in condition, alopecia and icterus ; the postmortem lesions were enteritis, brownish discolouration and swelling of the liver. Similar symptoms and post-mortem lesions were caused in pigs, which had partaken of this plant.

Pammel (1911) states that this is another weed commonly found in wheat screenings, and that it is harmful to pigs but not to cows. Fröhner (1919), quoting Wenke and Mason, describes weakness, paralysis of hindquarters, blindness, laminitis, trismus and death in horses poisoned with *Vicia sativa* L.

Anderson, Howard, and Simonson (1925) investigated the toxicity of *Lathyrus sativus* L. and state their experiments to indicate that *Lathyrus sativus* L. itself is harmless and that the toxicity ascribed to it is due to contamination with the seeds of *Vicia sativa* L. They, furthermore, state that the seeds of the latter plant contain a glucoside, vicin, and a cyanogenetic glucoside, vicianin, which is closely related to amygdalin. Vicin on hydrolysis yields a base divicine and d-glucose.

The authors were able to kill guinea pigs by injecting them subcutaneously with 0.6 mgm. of divicine per Kg. body weight. Violent and continuous clonic convulsions of the whole body were exhibited for about an hour and then progressive paralysis set in until death supervened within some hours of injection. Intense congestion of and about the brain and spinal cord was found at post-mortem.

Owing to the small amount of vicin and vicianin they were unable to determine whether these substances are toxic or not.

Anderson and his collaborators produced poisoning in ducks and monkeys by feeding them on a diet containing a high percentage (50 per cent.) of *Vicia sativa* L. seeds.

The ducks exhibited ataxy, walking in circles, convulsions, paresis and a kind of writhing contortion of the whole body with extreme retraction of the head and neck, and died from the thirteenth to the hundred and twenty-fifth days of the experiment.

*Post-mortem lesions.*—Oedema of the subcutaneous tissues of the head and haemorrhages over the surface of the skull; superficial congestion of the brain and hyperaemia of the cerebellum, medulla and upper cord.

In the monkeys the symptoms appeared from the sixth to the five-hundredth day of the experiment. The animals became less active, crouched in the cages, were unable to sit up and were constantly grinding the teeth. They showed fibrillary twitchings of the muscles of the arms, legs and flanks and violent convulsions of the whole body lasting from five to ten minutes. They yawned frequently, were hyperexcitable and showed symptoms of paralysis. They, furthermore, state that vicin itself is apparently non-toxic, but that during digestion in the stomach it is hydrolysed into divicin, which they proved poisonous.

Wehmer (1929) states that the seeds of *Vicia sativa* L. contain vicin, con-vicin, and that they yield prussic acid and benzaldehyde.

Wernery (1929) mentions that the seeds of different species of *Vicia* contain vicianin, which through enzyme-action liberate prussic acid.

Deaths in mules and horses running on harvested lands in the Western Cape Province have been ascribed to this plant. These animals showed a stiff gait and became paralysed, particularly in the fore-quarters. Patchy inflammation of the intestine was the only lesion seen at post-mortem. (Onderstepoort File 144/3234, 5/12/31).

A number of pigs which had been fed "Chilean peas," which consisted mainly of the common vetch (*Vicia sativa* L.) became ill and died suddenly.

The post-mortem revealed gastritis and patchy enteritis. On incubating these peas with water 0.018 per cent. prussic acid was found in them and this apparently was the cause of death (Clough, 1931).

Stockman (1931) isolated a "poisonous acid" from the seeds of the common vetch. By injecting this acid subcutaneously he produced marked general weakness and paralysis on monkeys, rabbits, and frogs.

## H. POLYGONACEAE.

*Rumex acetosella* L.

*Vernacular names.*—Boksuring, steenboksuring, dock, Sheepsorrel.

*Habitat.*—Cultivated lands. Very prevalent in southern and western Cape Province.

The "seeds" of this plant were found in specimens of wheat received from the southern and western Cape Province.

*History.*—Rosenthal (1862) and Dragendorff (1898) mention that the root and seed of this plant are used as toxic-astringents in diarrhoea, and the leaves, which contain potassium oxalate as an antiseptic and remedy in scurvy.

According to Kobert (1906) *Rumex acetosella* L. contains acid potassium oxalate.

Long states that children have been poisoned by eating large amounts of *Rumex acetosa* L.

Fröhner (1919) reports poisoning in sheep grazing on harvested lands heavily overgrown with *Rumex acetosella* L.

Graig and Kehoe (1921) fed 147 lb. of *Rumex acetosa* L. for a month to a bull with negative results.

Wehmer (1929) states that *Rumex acetosella* L. contains potassium oxalate.

*Symptoms of poisoning.*—The symptoms resemble those of subacute or chronic oxalic acid poisoning.

*Animals.*—Drunkenness, swaying gait, salivation, muscular tremors, dilatation of the pupils, feeble, slow and intermittent pulse, convulsive contraction of the lips, accelerated and stertorous breathing, tetanic contraction of the muscles of the neck, back, and limbs, profuse sweating, inappetence, diarrhoea, apathy, symptoms of paralysis, death occurs in convulsions.

The milk of affected cows is said to be made into butter with difficulty.

*Post-mortem appearances.*—Acute catarrhal gastro-enteritis with hæmorrhages on the gastro-intestinal mucosa.

*Treatment.*—Limewater, calcium carbonate, diuretics, furthermore symptomatic treatment.

Oxalic acid and oxalate poisoning are fully described by Witthaus (1911), Petri (1930), and Glaister (1931).

## I. SOLANACEAE.

(a) *Datura stramonium* L.

*Vernacular names.*—Stinkblaar, olieblaar, olieboom, thorn apple, Pietjie Laporte, Jimson weed.

*Habitat.*—Very common on cultivated and waste lands and along river.

At Onderstepoort a rabbit received 100 grams of the ripe seed on one day and a sheep 1,000 grams of the ripe seed administered at the rate of 500 grams daily without developing any symptoms of poisoning (Steyn, 1931).

*History.*—*Datura stramonium* L. was used in witchcraft practice in olden times and was later extensively used in homicidal poisoning (Lewin, 1920).

Rosenthal (1862) states that the leaves and seed contain a most poisonous narcotic alkaloid, daturin. This plant is used as a remedy in cases of neuralgia, spasms, epilepsy, stomach cramps, chronic rheumatism and is smoked to relieve asthma.

Hutcheon (1903) writes: "The seeds and young growing plants of *Datura stramonium* or Stinkblaar, as it is called, are very poisonous to young ostriches; I have seen them die off very rapidly from eating the leaves of the young plants.

Veterinary Surgeon Sinclair reports (*vide Agric. Jour.*, Vol. 13, p. 550) equally serious cases resulting from ostrich chicks eating the seeds obtained from the fruit of the previous season."

No marked lesions were present as the poison acts on the central nervous system causing a dull, sleepy condition terminating in complete collapse. Some birds showed delirious excitement and staggered about before the comatose condition set in. The treatment recommended is castor oil and strong coffee.

Kobert (1906) mentions that the active principle of the thorn apple is atropine.

According to Bryant (1909) the leaves of this plant are freed from the mid-ribs and then laid over painful wounds and sores.

According to Pammel (1911) the seeds are the most poisonous part of the plant.

South African specimens of "*Datura stramonium*" examined at the Imperial Institute contained 0.49 per cent. of total alkaloid, the chief alkaloidal constituent being hyoscyamine (Editorial, 1916).

Mitchell (1923) records a case of *Datura* poisoning in human being at Vrede, Orange Free State.

Watt and Brandwyk (1927) recorded poisoning in mine boys in South-West Africa through eating beans contaminated with seeds of *Datura stramonium* L.

Beyers (1930) reported cases of poisoning in natives in the Somerset East district due to the eating of "boermeal" admixed with seeds of *Datura stramonium* L.

*Symptoms of poisoning.*—(Kobert, 1906; Witthaus, 1911; Long, 1917; Fröhner, 1919; Byam and Archibald, 1921; Thomson and Sifton, 1922; Watt and Brandwyk, 1927; Gimlette, 1929; Beyers, 1930; Petri, 1930; and Glaister, 1931.)

(1) *Human beings.*—The symptoms vary according to the size of the dose and the age of the victim and may appear within a few minutes after ingestion of the seeds. The following symptoms may be exhibited: Giddiness, yawning, dryness of the throat and thirst, attempts to swallow provoke spasms of the pharynx and may resemble hydrophobia to a certain extent, bitter taste and burning sensation in the mouth; impairment of vision, power of standing is lost and on attempting to walk patient staggers as if intoxicated, progressive restlessness, which may develop into wild delirium, pupils widely dilated, difficult and incoherent speech, affected people try to climb walls and trees, pull on imaginary ropes, children run about naked and on all fours, pick at imaginary objects, and search the bedding most vigorously for some lost article, "tries to thread imaginary threads and tries to pick them from the tips of his fingers or he constantly gazes at his fingers and keeps passing his thumb over them in a most peculiar way" (Gimlette, 1929), laughing is common, and there is a tendency to discard clothes, when trying to read "the letters run over the pages

like so many ants," flushed (scarlatinal) face and a scarlatinal rash with itching may appear upon the skin of the body and extremities, the pulse is at first hard and full and very much accelerated (up to 200 per minute) and in the course of time becomes intermittent, irregular and ultimately imperceptible, the skin may be hot or cold and dry, vomiting is uncommon, all reflexes are exaggerated, delirium passes into exhaustion, coma supervenes and terminates in death. Watt and Brandwyk (1927) describes vomiting and purging in a few of the victims.

*Post-mortem appearances.*—Dilatation of the pupils, hyperaemia and oedema of the lungs, hydrothorax, pronounced congestion of the meninges of the brain, with bloody serum in the ventricles, and punctiform haemorrhages in the brain substance, hyperaemia of and haemorrhages in mucosa of stomach and small intestine.

*After effects of Datura poisoning.*—As the toxic principles of *Datura stramonium* L. are excreted slowly the after effects may persist for quite a number of days. There may be general weakness, dilatation of the pupils, thirst, impairment of the memory and difficulty in walking.

*Toxicity.*—It is impossible to state definitely the toxic dose of the seed as the amount of active principle varies considerably in the plant growing in different localities.

*Treatment.*—Treatment of cases of atropine poisoning and chemical analysis of the gastro-intestinal contents of such victims are described by Glaister (1931) and Byam and Archibald (1921).

(2) *Animals.*—Cattle and horses are considered equally susceptible, whilst they are held to be less susceptible than carnivora.

The symptoms in ostriches have already been referred to (Hutcheon, 1903).

Other animals show dryness of the tongue and buccal mucous membrane, dilated pupils, an accelerated, irregular and weak pulse, excitement followed by paralysis, tympanites, staggering and death from asphyxia and heart failure.

*Post-mortem appearances and histology.*—The appearances in acute cases are essentially those of asphyxia.

In chronic cases of atropine poisoning wheals, blisters, petechiae and scarlatina-like eczematata appear on the skin. Anima and Metzner found enlarged alveoli and also diminished alveoli with shrivelling of the ducts in the salivary glands of animals poisoned with atropine. The blood picture appears normal in atropine poisoning (Petri, 1930). Agapi (Petri, 1930) saw extensive haemorrhages in the parenchym of lungs of mice repeatedly injected with atropine.

*Toxic principle.*—In all parts of the plant hyoscyamine is the chief alkaloid and a small amount of atropine and scopolamine is also present (Wehmer, 1929). It is quite possible that the ratio of the percentages of these three active principles in the seed of this plant growing in different localities may vary as this is known definitely to occur in other plants with more than one active principle (for example, *Digitalis* glucosides).

*Differential diagnosis.*—Clinically and pathologic-anatomically meat and fish poisoning resemble atropine poisoning. In the case of suspected Datur poisoning, especially in natives and coloured people, methyl alcohol poisoning is differential diagnostically of great importance.

(b) *Datura Tatula* L.

*Common name.*—Purple thorn apple, purple stramonium, blou stinkblaar, blou olieboom.

*Habitat.*—As in *Datura stramonium* L.

Two sheep received per stomach tube 750 grams of fresh leaves of the plant in the late flowering stage on each of two consecutive days and 500 grams of the ripe seeds on each of two consecutive days respectively.

*Result.*—Negative (Steyn, 1929).

*History.*—Pammel (1911), referring to the toxicity of this plant remarks that the seeds are especially poisonous.

Thomson and Sifton (1922) state that this plant has similar effects to those of *Datura stramonium* L.

According to Bernhard-Smith (1923) the active constituents of *Datura tatula* L. are atropine, hyoscyamine, and hyoscine.

Beyers (1930) referring to *Datura stramonium* L. poisoning writes: "In conclusion it may be of interest to state that about fifteen years ago I attended a few small children who were similarly affected after sucking the honey from the nectar of the large purple flowers of the stinkblaar."

### III. LEGAL ASPECT.

Regulation 12 (7) of the Food, Drugs, and Disinfectants Act, No. 13 of 1929, states: "Every mill in which grain is milled for human consumption shall be provided with efficient sieving and winnowing appliances so as completely to remove the seeds of *Senecio* (Sprinkaanbos) and any other poisonous or unwholesome seeds or matter. Any person selling any flour or meal containing such seeds or matter shall be guilty of an offence."

As all the evidence, both circumstantial and experimental, gathered in the past and present investigations, points very strongly to bread poisoning being due to the presence of portions of *Senecio* plants in the wheat used for household purposes, and as no evidence whatsoever has as yet been brought forward to disprove this contention, it would seem advisable to introduce legislation whereby all growers of foodstuffs for man and animal will be forced to keep the lands free from all species of *Senecio*. The eradication of *Senecio* from cultivated lands will not entail an unreasonable amount of work or expense as these plants are large and very easily uprooted.

The author has seen fallow lands overgrown with *Senecio ilicifolius* Thunb. and no attempt was made to eradicate this weed. It is due to such irresponsible persons that weeds are allowed to spread instead of being eradicated.

It is a custom among many wheatgrowers to market their best and clean wheat and to use for household purposes that which is contaminated with weeds. The enforcement of the above Regulation 12 (7) would be difficult in these cases. Under these circumstances a much safer procedure would appear to be the proclamation of those species of *Senecio* growing on cultivated lands as noxious weeds.

## IV. DISCUSSION.

At Onderstepoort seeds of the following weeds were sorted from wheat obtained from areas where cases of suspected bread poisoning occurred: *Lithospermum arvense* L. (very rare), *Silene gallica* (2 per cent.), unripe and ripe *Senecio* seed heads (very rare), *Eruca sativa* Mill. (2 per cent.), *Rephanus raphanistrum* L. (2 per cent.), *Lolium temulentum* L. (up to 33 per cent.), *Vicia sativa* L. (1.5 per cent.), *Malva parviflora* L. (0.5 per cent.), and *Rumex acetosella* L. (rare). In addition to these seeds the following substances were found: Legs and other parts of lizards, parts of beetles, bird droppings, small stones, stems of plants up to two inches long, and parts of leaves of different kinds of plants.

Furthermore, oats, barley, and lucerne seed were present in some specimens of wheat.

## A. PLANTS CONCERNED IN BREAD POISONING AND IN POISONING BY OTHER FOODSTUFFS CULTIVATED ON LANDS.

(a) *Agrostemma Githago* Linn.

Thunberg (1823) recorded this plant is wheat grown near Fransch Hoek and according to Burt-Davy it was found on cultivated land near Pretoria. The author is unaware of the presence of this plant on cultivated lands in areas where "bread poisoning" occurs. From the foregoing information it is, however, evident that this plant when growing on wheat lands constitutes a grave danger to man.

(b) *Centaurea picris* DC.

This plant was proved poisonous to sheep and as it grows on cultivated lands it is likely to find its way into foodstuffs which are grown on such infested lands and which are threshed carelessly.

(c) *Senecio burchellii* DC.

The plant obtained from the Humansdorp Commonage produced no symptoms of poisoning in rabbits, dogs, or sheep at Onderstepoort. Chase, however, proved it poisonous to horses, consequently it should be regarded as a dangerous weed on wheat lands. The plant was most probably responsible for the typical symptoms of *Senecio* poisoning exhibited by a horse belonging to Mrs. de Bruyn, Modderfontein, Humansdorp district. The cases of suspected bread poisoning on this farm were most probably due to *Senecio Burchellii* DC., which grows very abundantly in one corner of the wheat land. There is, however, a possibility of *Senecio ilicifolius* Thunb. also having played a role in these cases of poisoning, as a few specimens were found on the land through not acutally mixed with the wheat and it is quite likely that in some years it may be found growing amongst the wheat.

(d) *Senecio ilicifolius* Thunb.

This plant obtained from lands on a farm where "bread poisoning" occurred, caused symptoms of poisoning and post-mortem lesions in dogs very similar to those seen in cases of "bread poisoning" in human beings. Rabbits were apparently not affected by large amounts of this plant. The results of experiments upon dogs show that small amounts of the plant administered over prolonged periods do not cause clinical icterus nor was pigmentation of the liver seen at post-mortem, whilst larger quantities of this plant administered over comparative short periods invariably produced sever clinical icterus and at post-mortem intense pigmentation of the liver was present.

(e) *Senecio isatideus* DC.

This plant has not yet been recorded as occurring on cultivated lands in areas where bread poisoning occurs, it is however, discussed here as it possibly may spread and some day find its way on to wheatlands. The plant caused poisoning in dogs and rabbits with symptoms and post-mortem lesions closely resembling those seen in dogs poisoned with *Senecio ilicifolius* DC. The latter plant was found to be much less poisonous than *Senecio isatideus* DC. in all the experiments conducted at Onderstepoort.

Like *Senecio ilicifolius* Thunb., this plant when given in small amounts, produced no clinical icterus in dogs and no pigmented liver at post-mortem, whilst with larger amounts clinical icterus and a pigmented liver were present.

These results would tend to explain the extremely rare occurrence of icterus in suspected cases of *Senecio* poisoning in human beings, as it must be accepted that the victims of bread poisoning ingest very small amounts of *Senecio* with the bread. Dr. G. de Kock, Deputy Director of Veterinary Services, Onderstepoort, is engaged upon an investigation into the pathology of *Seneciosis* and an article treating with this aspect of *Senecio* poisoning will be published by him.

(f) *Raphanus raphanistrum* L.

This plant is known to be poisonous and at Onderstepoort its seed (+ seed capsules) sorted from wheat was proved to be poisonous to rabbits.

(g) *Euphorbia helioscopia* L.

It is recorded as toxic. Experiments with the plant received from Capetown and with material grown at Onderstepoort were negative.

(h) *Euphorbia pepylus* Linn.

No tests have been conducted at Onderstepoort. It is, however, recorded as toxic.

(i) *Ricinus communis* L.

There are a number of cases on record of the seed of this plant having been found in maize and of its having caused serious poisoning in stock fed with such maize. Castor oil trees should, therefore, not be allowed to grow on or in the neighbourhood of cultivated lands.

(j) *Lolium temulentum* L.

All the experiments conducted at Onderstepoort on horses, pigs, and rabbits with fungus-free and fungus-infected drabok yielded negative results. Rabbits are considered to be the animals most susceptible to drabok poisoning.

The fact that large numbers of human beings, especially the coloured people, in the southern Cape Province constantly eat bread prepared from wheat very heavily contaminated with drabok without suffering any or very slight ill-effects tends to prove that drabok poisoning is of very rare occurrence or does not occur at all.

Several people have informed the author that they are well acquainted with the symptoms of poisoning by bread containing a high percentage of drabok. These symptoms, which are more liable to occur when such bread is eaten soon after being baked and when still warm, are dizziness, headache, and sleepiness.

The cases of suspected bread poisoning which occurred at the farm Palmietfontein (Oudam), Clanwilliam district, and which were suspected by Willmot and Silberbauer to be drabok poisoning, were most probably cases of *Senecio ilicifolius* Thunb. poisoning according to the symptoms and post-mortem appearances described in the affected cases. During subsequent investigations *Senecio ilicifolius* Thunb. was found growing amongst the wheat.

It is, however, generally held by authorities in Europe that drabok is poisonous when infected with a fungus termed *Endoconidium temulentum*. The symptoms and post-mortem appearances attributed to poisoning with drabok are of such a nature that they cannot be confused with *Senecio* poisoning.

(k) *Vicia sativa* L.

At Onderstepoort a rabbit was killed by a small amount (20 grams) of this seed with symptoms typical of prussic acid poisoning. Chemical tests revealed the presence of a large amount of prussic acid in the form of a cyanogenetic glucoside, vicianin.

Furthermore, the plant and its seed have been proved poisonous by other investigators and the toxic principles considered to be divicine and an acid.

(l) *Rumex acetosella* L.

Although this plant is considered poisonous and it is recorded that children were poisoned by partaking of it, it is not likely to find its way into bread in such amounts as would be detrimental to health, except in cases of gross carelessness.

(m) *Datura stramonium* L.

This is a known poisonous plant. Experiments conducted at Onderstepoort with the ripe seed and the green plant on sheep and rabbits yielded negative results.

The presence of the seed has been recorded in beans and wheat (and may also find its way into maize) and has caused poisoning in human beings.

(n) *Datura tatula* L.

The above information is also applicable to this plant.

Attention should also be paid to *Centaurea picris* DC. in view of the fact that it has been proved poisonous to sheep.

*Osteospermum moniliferu* L. (bieton, boete bossie, bok berries, brother berries, bushtick berry) and *Malva parviflora* L. (mallow, kiesieblaar), should be mentioned here. The former plant was found by Muir (1928) on wheatlands and he states that it is regarded as toxic. *Malva parviflora* L. is held by many farmers to cause shivers in stock, especially in horses when they are worked soon after having ingested the plant. Dodd and Henry (1923) proved this plant as one of the causes of shivers or staggers in stock.

B. ARE SPECIES OF *Senecio* CONCERNED IN THE SO-CALLED "BREAD POISONING" IN HUMAN BEINGS?

The one and only way to prove definitely that the species of *Senecio* proved toxic to animals are also the cause of bread poisoning in human beings will be to experiment on human beings. As this method of investigation is out of the question, and as we have to rely on the results of experiments on animals, we can only state that this or that plant, whose actions on human beings are

unknown, will "most probably" be poisonous to human beings as it causes poisoning in animals. The problem is more complicated by the fact that the different species of animals vary to a considerable extent in their susceptibility to poisons. Furthermore, some poisons attack different organs in the different animals (for example, *Crotalaria dura* and *Crotalaria burkeana* poisoning in horses and cattle). It is for the latter reasons that we should be cautious in drawing conclusions as to the effects on human beings of substances, which have been proved poisonous to animals.

The following facts, however, point very strongly to *Senecio spp.* being concerned in bread poisoning :—

- (a) There is a marked similarity between the symptoms and post-mortem appearances and also in the microscopical lesions found in livers in cases of bread poisoning and those seen in animals, especially dogs, poisoned by species of *Senecio*.
- (b) Cases of bread poisoning have only occurred in those areas where *Senecio spp.* grew on the wheatlands and usually in families belonging to the poorer classes, who paid no or very little attention to the presence of weeds in the wheat used for household purposes.
- (c) A natural case of what was most probably Seneciosis in a horse due to the ingestion of *Senecio Burchellii* DC. was seen on a farm (Modderfontein, Humansdorp district) where seven cases of suspected bread poisoning had occurred. A corner of the land on which the wheat for household purposes was grown, was found heavily overgrown with this plant.
- (d) Muir (1931) reported "that since measures were taken to ensure better sifting of wheat, we have had a cessation of cases of *Senecio* poisoning here (Riversdale area). We are hoping that this is not a coincidence, but there has been no reason to think that it is."

#### C. CIRCUMSTANCES FAVOURING BREAD POISONING.

These are :—

- (a) Threshing and grinding wheat in machines fitted with deficient winnowing and sieving appliances which will allow to pass through seeds and other parts of weeds. Even machines with fairly well adjusted winnowing and sieving appliances will allow to pass through weed seeds of approximately the size of wheat grains. Such weed seeds are darnel, *Agrostemma githago* L., unripe and ripe flower heads of *Senecio spp.*, etc. Small portions of the plants other than the seed may also find their way into the wheat. Specimens of wheat used for human consumption and examined at Onderstepoort were found to contain sticks up to two inches long. This is not surprising when it is considered that quite a number of wheat growers, especially those belonging to the poorer classes, thresh and grind the wheat themselves in old fashioned machines. It is quite customary amongst many wheat growers to sell the best and cleanest wheat and retain that contaminated with extraneous weeds for their own use. Furthermore, millers are instructed by some wheat growers not to adjust the above-mentioned appliances too finely as that would result in the loss of too much wheat.

- (b) Dry years: In years of deficient rainfall the wheat does not grow to normal height and it is then reaped close to the ground with the result that there is a greater possibility of the wheat becoming contaminated with poisonous weeds.
- (c) A grave danger is that poisonous weeds (especially *Senecio spp.*) frequently occur in patches on wheatlands with the result that during threshing some bags of wheat become heavily contaminated with these seeds, whilst the remaining bags may not contain a trace of these weeds ("pocket contamination").

It is for this reason and also because bread poisoning is essentially a chronic disease that the collecting of specimens of wheat and meal at the time cases of bread poisoning occur, is of comparatively little value in the investigation of the prevalence of weeds in the wheat concerned.

I would like to draw attention to the fact that in the usual course of events suspected wheat samples are examined for the presence of *Senecio* seed heads, and that in some areas (or in some years in the same area) the *Senecio* plants may not have reached the flowering stage at the time of reaping. This was the case when the author visited the farm Oudam, Clanwilliam district. On this farm the wheat was milled in an old fashioned type of mill with no screening appliance at all. It is, therefore, possible and probable that parts of the leaves and stems of *Senecio ilicifolius* Thunb. which was found growing amongst the wheat in a corner of the land, found their way into the wheat and were responsible for the cases of suspected bread poisoning in the Smit family.

- (d) It is quite possible that in some years the toxic weeds may be much more poisonous than in other years as variations in the toxicity of plants are well known. From the results obtained at Onderstepoort in experiments with *Senecio spp.* it appears that these plants are most poisonous in the early stages of development.
- (e) Several poisonous weeds contaminating bread at the same time may by virtue of their synergistic effects or other actions markedly increase each other toxicity. It is, for example, quite likely that wild mustard (*Raphanus raphanistrum* L.) will, owing to its irritating effect on the gastro-intestinal tract, render *Senecio spp.* more poisonous by facilitating the passage of the active principles of these plants through the damaged mucosa.

#### D. EFFECT OF THE PROCESS OF PREPARATION OF BREAD IN THE TOXICITY OF WEEDS CONTAMINATING THE MEAL.

##### (a) *Moistening the Meal and Kneading the Dough.*

These processes will undoubtedly render the active principles (especially those soluble in water) of poisonous weeds more readily absorbable by the intestinal mucosa. Furthermore, cyanogenetic glucosides (for example vicianin contained in *Vicia sativa* L.) will be acted upon by enzymes and liberate prussic acid and those weeds which contain mustard oil compounds as active principles, for example, *Raphanus raphanistrum* L., will liberate mustard oils.

*(b) Baking of Bread.*

Baking is said to partly destroy the active principle of *Agrostemma githago* L., and all the evidence at our disposal seems to indicate that the active principles of *Senecio spp.* are not affected to an appreciable extent by temperatures such as are encountered in the baking of bread. As soon as fresh supplies of poisonous *Senecio spp.* are available this point will be definitely settled.

High temperatures will destroy all enzyme action thus preventing further development of prussic acid from cyanogenetic glucosides (*Vicia sativa* L.) and will at the same time expel from the bread (dough) the already liberated prussic acid.

Likewise baking will stop the liberation of mustard oils from plants containing mustard oil compounds by destroying enzyme action, and the already liberated mustard oils, being volatile, will escape from the bread during the time of baking.

Insufficiently baked bread may still contain a certain amount of the above poisonous substances which would have escaped during thorough baking or the production of which would have been rendered impossible by thorough baking.

E. THE CAUSE OF DEATH IN *SENECIO* POISONING.

The active principles of *Senecio spp.* must be considered primarily as liver poisons. Whether their immediate effects on the remaining organs are of any value in contributing to the cause of death is a point yet to be elucidated. Whether the gastrointestinal disturbances (inappetence, constipation, diarrhoea, ulceration of the gastric mucosa and catarrhal enteritis) are primary or secondary effects of *Senecio* poisoning is difficult to state.

In the light of our present knowledge of *Senecio* poisoning it appears that the function of the liver, especially as detoxicator and as excretor of harmful substances, is partially, or, in advanced cases of *Senecio* poisoning, completely destroyed. Poisonous substances, some of which are present under normal circumstances in the intestinal tract, will then be allowed to pass into the circulation and exert their harmful effects on the whole system and ultimately cause death.

Chronic *Senecio* poisoning therefore appears to cause death indirectly by destroying functions of the liver, which is the main protector of the system as far as poisoning is concerned. In acute *Senecio* poisoning there is marked destruction of the liver cells and the absorption of cell products liberated in this way will undoubtedly aid in poisoning the system.

Of interest is the following passage quoted from Wright's (1931) Applied Physiology: "The liver is thus mainly concerned with the excretion of bile pigment: the elaboration of the pigment is chiefly carried out in the bone marrow, to a less extent in the spleen and to a very slight extent in the liver (Kupfer cells)." It would, therefore, appear that in the case of a liver with an impaired function an accumulation of bile pigments in the system will occur. In high concentrations these pigments will have a detrimental effect on the system. In addition there is every reason to believe that the glycogen-glucose-lactic acid balance will be disturbed in a system with a damaged liver, the degree of disturbance depending on the degree of damage present in the liver.

## V. SUMMARY.

- A.—Poisonous weeds, which are liable to find their way into wheat and cause poisoning in human beings are discussed.
- B.—Circumstances favouring bread poisoning, the effect of the process of preparing and baking bread on the toxicity of weeds contained in the meal, and the cause of death in *Senecio* poisoning are discussed.
- C.—It would seem advisable to proclaim species of *Senecio* growing on cultivated lands as noxious weeds in addition to enforcing Regulation 12 (7) of the Food, Drugs, and Disinfectants Act, No. 13 of 1929.

## VI. ACKNOWLEDGEMENTS.

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## VII. LITERATURE.

Onderstepoort File	144/2282	(1931).
"	"	144/68 (1931).
"	"	111/22 (1930-1931).
"	"	144 481 (1931).
"	"	144 3163 (1931).
"	"	144/3032 (1931).
"	"	144 2589 (1931).
"	"	144/2282 (1931).
"	"	111/60 (1930-1931).
"	"	144 2587 (1931).
"	"	144/512 (14/9/28).
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