

Recent Investigations into the Toxicity of Known and Unknown Poisonous Plants in the Union of South Africa, XI.

By DOUW G. STEYN and S. J. VAN DER WALT, Section
of Pharmacology and Toxicology, Onderstepoort.

(Continued from *Onderstepoort Journal of Veterinary
Science and Animal Industry*, Vol. 15, Nos. 1 and 2).

AMARANTACEAE.

Amarantus paniculatus L.

Registered number.—O.P.H. No. 1833; 23.4.40.

Common name.—Hanekam.

Origin.—Ixopo, Natal.

State and Stage of Development.—The plant was in the late seeding stage and fairly fresh.

Sheep 54332 (6 tooth; 38.2 Kg.) received* 2.7 Kg. of the plant in the course of 3 days.

Result.—Negative.

This plant has repeatedly been suspected of having caused poisoning in pigs and sheep. All the tests conducted by us with the fresh and almost dry plant yielded negative results. However, there is a possibility that, when severely damaged by frost or wilting, the plant may be poisonous.

ASCLEPIADACEAE.

Araya serisifera Brot.

Registered number.—O.P.H. No. 14491; 13.11.40.

Common name.—Motteboom.

Origin.—Carolina, Transvaal.

* Unless where otherwise stated the animals were drenched by means of a stomach tube.

TOXICITY OF POISONOUS PLANTS.

State and Stage of Development.—The plant was in the fresh state and in the late seeding stage.

Rabbit No. 1 (2.25 Kg.) received the juice expressed from 160 gm. of the leaves of the plant in the course of 20 hours.

Result.—Negative.

The latex of the plant was applied to the shaven dorsal aspect of a rabbit's ear. Within 24 hours a slightly painful oedematous swelling had developed. A serum-like fluid exuded onto the surface of the swollen area to form a yellowish scab. Five days after the application of the latex the swelling had subsided but the scab remained.

COMPOSITAE.

Cineraria sp.

National Herbarium number.—26386.

Registered number.—O.P.H. No. 14115; 22.2.40.

Origin.—Bethlehem, Orange Free State.

State and Stage of Development.—The plant was in the dry state and in the flowering and seeding stages.

Sheep 50056 (6 tooth; 44.6 Kg.), sheep 51436 (6 tooth; 38.7 Kg.), sheep 55603 (2 tooth; 37.8 Kg.) and sheep 55497 (6 tooth; 35.5 Kg.) each received 9.4 Kg. of the plant in the course of 25 days.

Result.—Negative.

Senecio pterophorus D.C.

Registered number.—O.P.H. No. 14462; 12.11.40.

Origin.—Umtata, Cape Province.

State and Stage of Development.—The plant was in the dry state and in the flowering stage.

Sheep 57241 (6 tooth; 36.4 Kg.) received 2.9 Kg. of the plant in the course of 9 days.

Symptoms.—By the ninth day after the first dose had been administered a severe icterus had developed. The animal was listless and off its feed and died the same night.

Post-mortem appearances.—Severe icterus; severe hyperaemia and oedema of the lungs; extensive haemorrhages in the pericardium, epicardium, subcutaneous tissues, subscapular tissues and in the costal pleura; severe degeneration and yellowish pigmentation with initial cirrhosis of the liver; yellowish pigmentation and degeneration of the kidneys and adrenals; haemorrhagic and necrotic foci, both of varying sizes, of the abomasum; pronounced gelatinous infiltration of the wall of the pyloric portion of the abomasum; excessive

quantities of ingesta in all four stomachs; haemorrhages of the small intestine which contains no ingesta but a fair amount of blood; constipation of the caecum and initial portion of the colon; urine turbid and greenish in colour.



Fig. 1.—*Cineraria* species.

Histology.—Liver: The lobuli are separated into islands of hepatic cells by broad bands of rapidly proliferating fibroblastic tissue in which collagen has already formed, although not to a very great extent. These islands vary from more or less normally shaped lobuli to small groups of hepatic cells in which all lobular structure is lost. There is practically no formation of delicate intralobular fibres of collagen. Associated with this process is a marked and extensive bile duct proliferation.



Fig. 2.—*Senecio pterophorus* D.C.

The hepatic cells themselves are deformed and either swollen or compressed. The nuclei are swollen and vesicular. Large fat droplets are present in most of the cells especially around the periphery of the existing lobuli. The bile capillaries are markedly dilated. Granulae of bile pigment are present in many of the liver cells. Some of the von Kupffer cells contain a large amount of fine pigment granules. The endothelial cells are swollen and prominent.

Periportal lymphatic gland.—There is a total disappearance of the secondary follicles. A large number of cells are present which were apparently desquamated from the liver. They are full of fine pigment granules and resemble Gaucher cells.

Spleen.—Except for a marked dilation with blood no other change was noted.

Kidney.—Large fat droplets are present in most of the proximal convoluted tubules becoming fewer and smaller in the descending area of Henle's loop.

Lung.—Hyperaemia is very marked and there is a moderate degree of oedema.

Myocardium.—Fine fatty droplets are present in the myofibrils.

Adrenal.—There is a larger fat content than normal especially in the outer cortical zone.

Thyroid.—No pathological changes observed.

Abomasum.—The one portion merely shows a marked oedema of the submucosa whereas in the other portion lesions of a deep necrotic abomasitis are present. The mucous membrane has become necrotic and the submucosa is swollen as a result of the large quantities of fibrin, serum and polymorphonuclear cells present.

Senecio scleratus sp. nov. Schweickerdtd.

Registered number.—O.P.H. No. 14242; 6.11.40.

Origin.—Zoekmekaar; Transvaal.

State and Stage of Development.—The plant was in the dry state and in the flowering and seeding stages.

Sheep 57240 (full mouth; 36.9 Kg.) received 500 gms. of the plant in the course of 2 days.

Symptoms.—Listlessness; severe icterus; pulse somewhat accelerated and fairly weak; anorexia; ruminal movements in abeyance. As the disease progressed the sheep went down and refused to rise. The respiration became laboured with a double expiratory movement accompanied by groaning. The animal died 53 hours after the administration of the first dose.

Post-mortem appearances.—General cyanosis; severe icterus; tremendous number of petechiae and ecchymoses in the subcutaneous tissues, costal pleura, parietal peritoneum, omentum and mesenterium; blood tinged fluid in the pericardium, thorax and abdomen; petechiae and ecchymoses of the epi- and endocardium; regressive changes of the myocardium; slight hyperaemia and emphysema of the lungs; severe degeneration and pigmentation of the liver; haemorrhagic infiltration of the wall of the gall bladder which was distended with partially coagulated blood; regressive changes, pigmentation and slight hyperaemia of the kidneys; tremendous number of petechiae and ecchymoses in the alimentary tract from the rumen right up to the rectum; pronounced stasis in the caecum, the caecal contents being covered with blood.

Histology.—Liver: Only small islands of hepatic cells have remained around the portal tracts; the rest of the parenchyma had undergone disintegration of the cytoplasm, and pyknosis and karyorrhexis of the nuclei with consequent formation of large central blood pools. The surviving cells show advanced fatty changes as well as nuclear degeneration. Bile pigment in granular and rod shape has accumulated in these liver cells to a moderate degree. Stasis of blood is very marked, the sublobular veins being most widely dilated and filled with blood. The intrahepatic branches of the portal vein and the sinusoids as well as the central veins are similarly distended. The latter can be seen lying in pools of blood in which a few disintegrating hepatic cells, a number of neutrophils, a few eosinophils and macrophages are the only structural elements visible.

There is active fibroblastic proliferation starting from the portal tracts without however the formation of collagen. Bile ducts have also proliferated but to a lesser degree.

Unlike the usual irregular histological picture of senescence this one is markedly regular.

Gall-bladder.—There is oedema and extensive haemorrhage especially in the muscular layer. Blood has apparently oozed into the lumen since the mucous membrane is covered by a layer of blood in many parts.

Kidney.—There is a moderate hyperaemia especially of the venules and arterioles. Fatty changes are marked mostly so in the proximal convoluted tubules and to a lesser extent in the distal convoluted tubules. As a rule the fat droplets are large and basally situated. In the epithelium of the distal part of the collecting tubules and in the basal epithelium of the renal pelvis diffusely spread fat droplets occur.

Adrenal.—Abnormal quantities of fat in the shape of large globules are found in the outer edge of the cortex and occasionally also in the zone bordering on the medulla. Phagocytosis of fat by the endothelial cells of the sinusoids has occurred.

Lung.—There is marked dilation of the larger vessels and capillaries. Haemorrhages have occurred under the pleura and in various parts into the alveoli. Moderate emphysema is present.

Myocardium.—The myofibrils of the atria and ventricles contain large numbers of medium sized fat droplets. Haemorrhages have occurred under the epi- and endocardium.

Spleen.—The spleen is markedly distended with blood with the result that the red pulp predominates and the splenic corpuscles appear small and atrophic. The germ centres reveal an open structure due to paucity of cells. Pyknosis and karyorrhexis occur in these germ centres either in light or moderate degree.

Pancreas.—No pathological changes observed.

Abomasum and intestines.—Extensive haemorrhages have occurred in the submucosa as well as numerous small haemorrhages in the mucosa.

Thyroid.—No pathological changes observed.

Hypophysis.—Hyperaemia is the only change noted.

ARE THE *Senecio* ALKALOIDS THE CAUSE OF " DUNSIKTE "
(*Senecio* POISONING)?

Watt (1909) isolated two alkaloids *senecifoline* ($C_{18}H_{27}O_8N$) and *senecifolidine* ($C_{18}H_{25}O_7N$) from "*Senecio latifolius*". In 1909 the identification of species of *Senecio* was very unsatisfactory and we can safely assume that, at that time, the name *Senecio latifolius* DC included *Senecio latifolius*, *Senecio retrorsus* and *Senecio barbellatus*. Hence it is impossible to say from which species of *Senecio* Watt isolated the two above alkaloids (Steyn, 1934). *Senecifolidine* has the same empirical formula as *isatidine* isolated by de Waal (1939). Cushny (1911) injected *senecifoline*, isolated by Watt, into frogs, white rats, cats and rabbits and also administered it *per os* to cats. The symptoms and post mortem appearances were the same whether the alkaloid was drenched or injected. According to Cushny the cirrhosis of the liver in chronic *senecifoline* poisoning in cats resembled that found in *Senecio* poisoning in cattle. From the results of his experiments he concluded that there can be no question that *Senecio* disease is caused by the *Senecio* alkaloids.

Davidson (1935) injected *retrorsine*, which was obtained from *Senecio retrorsus* and which was supplied to him by Professor G. Barger, University of Edinburgh, subcutaneously into white rats, and found that the primary effect of the alkaloid would appear to be vascular. This was followed by cirrhosis with regeneration of the liver cells and bile ducts, the ultimate picture being that of an interstitial hepatitis or cirrhosis.

Hosking and Brandt (1936) isolated the alkaloid *jacobine* ($C_{18}H_{25}O_6N$) from *Senecio jacobaea* growing in New Zealand. They

state that this alkaloid "is very probably identical with the alkaloid *jacobine* previously isolated from Canadian ragwort by Manske, to which was given the formula $C_{18}H_{23}O_5N$ ". The *jacobine* isolated by Hosking and Brandt was drenched to guinea-pigs and injected subcutaneously into rats by Hopkirk and Cunningham (1936). They found guinea-pigs very resistant to the alkaloid. From the results of their experiments upon rats they concluded that "the rat lesions, therefore, are similar in type to those seen in larger animals which have eaten *Senecio jacobaea*".

Chen, Harris and Rose (1940) injected the *Senecio* alkaloids *platyphylline* ($C_{18}H_{23}O_5N$) intravenously into mice, rats, guinea-pigs and monkeys and also into the lymph sacs of frogs, and *seneciophylline* ($C_{18}H_{23}O_5N$) intravenously into mice, rats and guinea-pigs. The animals, particularly the rats and mice, injected with acutely sublethal doses of *seneciophylline* (delayed death) showed necrosis around the central veins of the liver with infiltration of leucocytes and vacuolation of the cytoplasm cells. Also the kidneys showed cloudy swelling with venous and glomerular congestion. On the other hand, they were unable to detect any hepatic or renal damage in the mice, rats and guinea-pigs injected with acutely sublethal doses of *platyphylline*.

In spite of the above information which certainly lends support to the contention that the said *Senecio* alkaloids are responsible for the production of *Senecio* disease ("dunsiekte" in horses and Molteno cattle disease in South Africa, "Sirasyke" in Norway, Pictou disease of Nova Scotia, Winton disease of New Zealand) it is felt that the definite proof or disproval of this contention could be brought forward in one way only, and that is to administer the suspected alkaloids *per os* either to horses or cattle and to produce, in this way, a disease similar to that seen under natural conditions. It has been our experience, in many cases, that the results of oral administration of a poison, cannot be deduced from those obtained in parenteral administration of that poison and *vice versa*, and also, that the results of an experiment conducted upon one species of animal very frequently differ from those obtained upon other species of animals. It has, for example, been established that the rabbit's liver is much more resistant to injury by *Senecio* alkaloids than the horse's or dog's liver. It is for these reasons that we selected horses for our experiments in order to establish whether the *Senecio* alkaloids are responsible for *Senecio* disease.

The following is a summary of our experiments conducted at Onderstepoort.---

A. *Isatidine* and *retrorsine*.

These two alkaloids were isolated by Mr. J. J. Blackie, Helyrood Road, Edinburgh, Scotland, from *Senecio isatideus* DC and *Senecio retrorsus* DC respectively (Steyn, 1937).

Horse 21305 (8 months old) was given 2.0 gm. *isatidine* in a small quantity of bran by means of a balling gun on 10.3.36, and 5.0 gm. of *retrorsine* in a similar manner on 11.5.36.

Horse 21304 (8 months old) received in the same way as horse 21305, 0.1 gm. *isatidine* daily (except Sundays) from 10.3.36 to 27.4.36, and 0.2 gm. of this alkaloid daily from 28.4.36 to 16.5.36, that is a total of 8.2 gm. in a period of ten weeks.

Neither of these animals developed any symptoms of ill health. *Horse* 21305 was killed on 12.6.36 and 21304 on 26.6.36. Autopsy revealed no lesions which could, in any way, be associated with *Senecio* poisoning. According to information obtained from subsequent experiments it would appear that the above negative results are due to the fact that the quantities of *isatidine* and *retrorsine* administered to the horses were too small. Besides *isatidine* readily undergoes decomposition when stored.

B. *Retrorsine* ($C_{18}H_{25}O_6N$) and *isatidine* ($C_{18}H_{23}O_7N$) isolated from *Senecio retrorsus* DC by Dr. H. L. de Waal, (1939) Organic Chemist, Onderstepoort.

Horse 21676 (120 Kg., 1 year and 3 months old).—Received per stomach tube 19.0 gm. *isatidine* and 14.0 gm. *retrorsine* (equivalent to approximately 1 Kg. of dry *Senecio retrorsus*), shaken up in a litre of water, on 29.4.40. From 6.5.40 the animal developed symptoms typical of acute *Senecio* poisoning namely icterus, general weakness, apathy, fever attacks, swaying gait and dragging the hindlegs, haemorrhages on the conjunctivae and membrana nictitans, walking into stationary objects (abrasions on head and other prominent parts of the body), pulse accelerated, weak and irregular, and yawning repeatedly. The animal died on 10.5.40. Autopsy revealed a dilated stomach, general icterus and an acute parenchymatous hepatitis as seen in *Senecio* poisoning.

C. *Isatidine* and *retrorsine* isolated by Dr. H. L. de Waal (1939).

Horse 21971 (150 Kg., 1½ years old).—Received per stomach tube 14.0 gm. *isatidine* isolated from *S. isatideus* in 300 c.c. of tap water on 1.8.40. Only transient symptoms of apathy developed. A specimen of blood examined by Mr. G. C. S. Roets, Section of Chemical Pathology, Onderstepoort, on 23.8.40 showed no increase in bile pigments. On this date the animal was drenched with 15.0 gm. of *retrorsine* isolated from *S. retrorsus* (no more *isatidine* was available). As the animal was apparently still in good health on 13.9.40, it received another 15.0 gm. of *retrorsine per os*. From approximately 10.10.40 distinct and progressive loss in condition, and apathy, set in. General weakness, swaying gait and gradual loss of appetite followed. From the end of December the animal, at times, walked into stationary objects. At no time was general icterus detectable. The horse was down and unable to rise on 14.1.41. Towards the late afternoon it was unconscious, and breathing rapidly and superficially, and it was decided to destroy it. The post mortem appearances were typical of those of chronic *Senecio* poisoning (dunsiekte), namely cachexia, dilated stomach, pronounced hydroperitoneum, and hydropericardium, very pronounced atrophic cirrhosis of the liver, and pronounced oedema of the wall of the caecum. The blood showed 6.25 van den Berg units according to Mr. Roets.

Horse 21972 (150 Kg., 1½ years old) received per stomach tube 14.0 gm., 14.0 gm. and 15 gm. of *retrorsine*, isolated from *S. retrorsus*, on 1.8.40, 23.8.40 and 13.9.40 respectively. The animal developed the same symptoms at the same rate as horse 21971 and it was killed *in extremis* on 15.1.41. The post-mortem appearances were identical with those described in horse 21971. Blood collected at death and examined by Mr. G. C. S. Roets showed 2.25 van den Bergh units.

The results of the above experiments conducted upon horses with *isatidine* and *retrorsine* are definite proof that these alkaloids, and most probably also other chemically closely related alkaloids present in the various poisonous species of *Senecio*, are responsible for the causation of *Senecio* disease in stock.

A large number of rats have been injected subcutaneously with various alkaloids isolated by de Waal (1939) from different species of *Senecio* and the results of these experiments will be published at a later date.

Addendum.

The following description of *Senecio sceleratus* was supplied by Dr. H. A. Schweickerdt:—

“*Senecio sceleratus* Schweickerdt sp. nov.; affinis *S. glaberrimus* DC, sed foliis multioribus, foliis summis maioribus, marginibus foliorum manifestius dentatis, anastomoso prominulo nervorum differt.

“*Senecio sceleratus* Schweickerdt sp. nov.; affinis *S. glaberrimus* DC, sed foliis multioribus, foliis summis maioribus, marginibus striatus, rectus vel undulatus, vulgo simplex, apice laxo polycephalo corymboso. Internodia 2-7 cm. longa. Folia caulina 8-10, usque ad 10 cm. longa et 3 cm. lata, coriacea, ellipticooblonga vel lanceolata, apice acuta, margine paucidentata; nervus intermedius prominentus in pagina inferiora; costae adscendentes, anastomoso prominulo folia inferiora petiolata mox decidua, basi non valde auriculata; superiora ca. 1.5 cm. longa et 0.5 cm. lata, lanceolata, gradatim minora, sessilia, basi auriculata; auricula aliquantum decurrentes, parce cordato-amplexicaules. Bractae ca. 2-3 mm. longa, lanceolatae. Corymbus fastigiatus, polycephalus, capitulis laxo depositis. Pedunculi usque ad 2.5 cm. longi, subesquamati. Capitula parce calyculata, campanulata; involucri segmenta 8-9, aequalia vel subaequalia, linearia vel oblonga, obtusa vel sub-acuta, marginibus late vel anguste membranaceis, cilliata in tertia parte superiore. Fl. ligulae 4-5 lutea, semper 4-nervosae, extus infraque fauce papillis minutis munitae; rami stylium glabri; achaenia striata glabra. Fl. disci 12-17, lutei; tubus infra cylindratus supra dilatatus; rami stylium apice penicillati; achaenia striata glabra.

Pappi setae copiosae albae.

Transvaal Zoutpansberg dist., roadside Tzaneen Phillips 3265 (type). Lydenburg distr., Burt Davy 7261. Lydenburg, in town on vacant lot, Burt Davy 416. Pietersburg (without exact locality) collected by the Government Veterinary Officer in *Nat. Herb.* Pretoria 12954.”

Erratum.—On page 457 of Toxicology of Plants in South Africa (Steyn, 1934) *Senecio scleratus* Schweickerdt sp. nov. should read *Senecio sceleratus* Schweickerdt sp. nov.

Tagetes minuta Lam.

Registered number.—O.P.H. No. 13507; 31.8.37.

Common name.—Kakiebos, Khakiweed.

Origin.—Pretoria, Transvaal.

State and Stage of Development.—The plant was in the fresh state and in the preflowering stage.

Sheep 48879 (6 tooth; 36.5 Kg.) and sheep 48966 (6 tooth; 38.0 Kg.) received 40.8 Kg. of the plant in the course of 32 days.

Result.—Negative.

Subsequently sheep 50341 (4 tooth; 34.5 Kg.) received in the course of 28 days 33.6 Kg. of the fresh plant, which was in the flowering stage.

Result.—Negative.

The plant contains the essential oil-oleum tagetes. It was therefore decided to determine the toxicity of this oil.

Sheep 49124 (4 tooth; 35.0 Kg.) received 60 c.c. and sheep 48739 (full-mouth; 52.0 Kg.) 150 c.c. oleum tagetes.

Both sheep only exhibited transitory symptoms of listlessness, anorexia and tympanites.

CRASSULACEAE.

Kalanchoe paniculata Harv.

Registered number.—O.P.H. No. 2507; 4.5.40. and 4724; 4.6.40.

Origin.—Kimberley, Cape Province.

State and Stage of Development.—The first consignment of the plant (O.P.H. No. 2507; 4.5.40) was in the fresh state and flowering stage whilst the second consignment of the plant (O.P.H. No. 4724; 4.6.40) was in the fresh state and early seeding stage.

Sheep 54249 (6 tooth; 41.0 Kg.) received 1.0 Kg. of the fresh leaves of the first consignment of the plant.

Symptoms.—Six hours after dosing the following was observed: Apathy; slight tympanites; pulse accelerated and weak; respiration accelerated and laboured and accompanied by groaning; ruminal movements in abeyance. No symptoms of cotyledonosis were observed even after forced exercise. The animal died overnight on the day of dosing.

Post-mortem appearances.—Slight post-mortem changes; general cyanosis; marked tympanites; hydropericardium; numerous petechiae of the epi- and endocardium; regressive

changes of the myocardium; hyperaemia and oedema of the lungs; extensive haemorrhages of the trachea; hyperaemia and regressive changes of the liver and kidneys; slight tumor splenis; slight hyperaemia of the mucosa of the abomasum, small intestine and caecum.

Sheep 52632 (6 tooth; 44·0 Kg.) received in the course of 18 hours 2·0 Kg. of the fresh leaves of the first consignment of the plant.

Symptoms.—Five days after the administration of the first dose the animal started to pass blood. The quantity of blood passed increased and the animal showed listlessness associated with anorexia, laboured respiration and gradual loss of condition. The sheep finally went down, refusing to rise and died 14 days after the first dose had been administered.

Post-mortem appearances.—Emaciation; anaemia; hydropericardium; slight hyperaemia of the lungs; regressive changes in the myocardium; regressive changes in the liver and kidneys; ecchymoses in the abomasal mucosa; ecchymoses in and ulceration of the mucosa of the caecum.

Histology.—Lung: Hyperaemia.

Kidney.—Slight hyperaemia; fatty changes in isolated tubules in the medulla.

Liver.—A more open network of cytoplasm and vacuoles in the peripheral hepatic cells. No significant fatty changes.

Heart.—A low grade chronic interstitial myocarditis was observed.

Caecum.—Infiltration with round cells and neutrophiles; necrosis of the epithelial cells at the bases of the crypts opposite the haemorrhages, many crypts being packed with necrotic debris.

Sheep 51348 (6 tooth; 52·0 Kg.) received in the course of 3 days 7·6 Kg. of the fresh leaves of the second consignment of the plant.

Symptoms.—Listlessness; anorexia; ruminal movements in abeyance; laboured respiration; accelerated weak pulse; haemorrhagic diarrhoea. The animal died 4 days after the administration of the first dose.

Post-mortem appearances.—General cyanosis; tympanites; hydropericardium; petechiae epicardium; hyperaemia of the lungs; regressive changes in the liver and kidneys; hyperaemia of the mucosa of the abomasum and caecum.

Sheep 54544 (6 tooth; 44·6 Kg.) received in a single dose 400 gm. of the fresh seed heads of the second consignment of the plant.

Symptoms.—The animal died two hours after dosing after having developed a severe tympanites.

Post-mortem appearances.—General cyanosis; severe tympanites; hyperaemia, emphysema and oedema of the lungs; regressive changes in the myocardium; hyperaemia of and regressive changes in the liver and kidneys; hyperaemia of the mucosa of the small intestine.

Sheep 55525 (4 tooth; 42.3 Kg.) received in a single dose 200 gm. of the fresh seed heads of the second consignment of the plant.

Symptoms.—One-and-a-half hours after dosing a severe tympanites had developed which was relieved by means of a trocar and canula. In spite of this however the respiration remained extremely laboured and the animal died 2 hours after dosing.

Post-mortem appearances.—General cyanosis; fair tympanites; hydropericardium; hydrothorax; hydroperitoneum; hyperaemia and emphysema of the lungs; regressive changes in the myocardium; hyperaemia of and regressive changes in the liver and kidneys; slight hyperaemia of the mucosa of the abomasum and small intestine.

Sheep 55389 (4 tooth; 40.0 Kg.) received in a single dose 100 gm. of the fresh seed heads of the second consignment of the plant.

Symptoms.—Two hours after dosing the animal was shivering slightly. A slight degree of tympanites had developed whilst the respiration was extremely laboured. The animal died $\frac{1}{2}$ hour later.

Post-mortem appearances.—General cyanosis; slight tympanites; severe emphysema of the lungs; regressive changes in the myocardium; hyperaemia of and regressive changes in the liver and kidneys; slight hyperaemia of the abomasal and duodenal mucosa.

Sheep 59061 (6 tooth; 32.8 Kg.) received in a single dose 50 gm. of the fresh seed heads of the second consignment of the plant.

Symptoms.—Listlessness; pulse slightly accelerated and strong; respiration laboured. Tympanites did not develop and the animal died during the night following the day of dosing.

Post-mortem appearances.—Fairly advanced post-mortem changes; general cyanosis; hydropericardium; hyperaemia, oedema and emphysema of the lungs; hyperaemia of the liver and kidneys (regressive changes, if any, masked by the post-mortem changes); hyperaemia of the mucosa of the caecum and initial part of the colon.

Sheep 51436 (6 tooth; 47.5 Kg.) received 50 gm. of the fresh seed heads of the second consignment of the plant in two doses on 11.6.40 and 12.6.40.

Symptoms.—11.6.40. 4 p.m.: Slight tympanites; respiration somewhat laboured.

12.6.40. 3 p.m.: Severe tympanites; respiration very laboured; pulse accelerated and weak; anorexia; ruminal movements in abeyance; listlessness. Tympanites was completely relieved by means of a trocar and canula. The sheep died overnight on 12.6.40.

Post-mortem appearances.—General cyanosis; tympanites; hydropericardium; hyperaemia and emphysema of the lungs; regressive changes in the myocardium; hyperaemia and regressive changes in the liver and kidneys; severe hyperaemia of the abomasum; hyperaemia of the duodenal mucosa; caecal contents fluid.

The leaves and seed heads of the second consignment of the plant were tested biologically for the presence of cotyledon toxin according to the method of Steyn (1932). Both the leaves and seed heads were found to contain a small quantity of cotyledon toxin.

The plant therefore contains either insufficient cotyledon toxin to cause cotyledonosis with the quantities drenched or the action of the cotyledon toxin is overshadowed by the action of the other poisonous principle(s) present in the plant.

Kalanchoe thyrsiflora Harv.

Registered number.—O.P.H. No. 7110.: 15.7.40.

Origin.—Port Shepstone, Natal.

State and stage of development.—The plant was in the fresh state and in the flowering stage.

Sheep 57241 (6 tooth; 40.0 Kg.) received 5.0 Kg. of the fresh leaves of the plant in the course of 2 days.

Symptoms.—Five days after the administration of the first dose an haemorrhagic diarrhoea developed which persisted for 3 days after which the animal made a complete recovery. Except for the diarrhoea no other symptoms of any importance were observed.

Sheep 57165 (6 tooth; 30.0 Kg.) received in a single dose 500 gm. of the fresh inflorescences of the plant.

Symptoms.—Within four hours of dosing the following symptoms were observed: Cyanosis; pronounced dyspnoea; very slight tympanites; frothing at the mouth. The animal died $7\frac{1}{2}$ hours after dosing.

Post-mortem appearances.—The carcass was in a very advanced state of decomposition.



Fig. 3.—*Kalanchoe thyrsiflora*, Harv.

GERANIACEAE.

Erodium cicutarium L'Hérit.

Registered number.—O.P.H. No. 14871, 25.11.40.

Common name.—Heron's Bill.

Origin.—Norvals Pont, Cape Province.

State and stage of development.—The plant was in the dry state and in the seeding stage.

Sheep 55091 (6 tooth; 46.9 Kg.) and sheep 59063 (fullmouth; 54.6 Kg.) both received 5.6 Kg. of the plant in the course of 17 days.

Result.—Negative.

LEGUMINOSAE.

Castanospermum australe.

Registered number.—O.P.H. No. 15366; 18.3.40. and 2722; 22.5.40.

Common name.—Australian Chestnut tree, Black Bean, Moreton Bay Chestnut.

Origin.—Forestry Department, Pietermaritzburg, Natal.



Fig. 4.—Flower and seed head of *Kalanchoe thyrsiflora*, Harv.

State and stage of development.—Both consignments of the plant (O.P.H. No. 15366; 18.3.40. and 2722; 22.5.40) were in the dry state and in the late seeding stage. The fruit were still fairly green.

Sheep 55091 (6 tooth; 41.9 Kg.) and sheep 54682 (6 tooth; 41.9 Kg.) received in the course of 14 days 4.0 Kg. and sheep 55228 (4 tooth; 31.9 Kg.) and sheep 55525 (4 tooth; 42.3 Kg.) received in the course of 7 days 2.0 Kg. of the leaves of the plant.

Result.—Negative.

Sheep 55389 (4 tooth; 40.0 Kg.) received in the course of 3 days 5.6 Kg. and again 2 months later 5.2 Kg. of the unripe beans (the husks were discarded) in the course of 3 days.

Result.—Negative.

Hindmarsh and Hart (1937) found that the unripe seeds caused a severe gastro-enteritis in cattle whereas the ripe seeds appeared to be non-toxic. The discrepancy between the results of the experiments of these authors and those of our experiments may possibly be due to the seeds used in our experiments having been more mature than those used in the experiments of Hindmarsh and Hart, or (and) to differences in soil and climatic conditions.

Crotolaria rhodesiae Baker f.

Registered number.—O.P.H. No. 4610; 4.6.40.

Origin.—Pietersburg, Transvaal.

State and stage of development.—The plant, which was somewhat mouldy, was in the dry state and in the seeding stage.

Sheep 59062 (6 tooth; 61.4 Kg.) received 3.4 Kg. of the plant in the course of 9 days.

Symptoms.—On the tenth day after commencement of dosing the gait of the animal was somewhat stiff especially in the fore-limbs. This stiffness disappeared after driving the sheep around for some time. On no other occasion was the stiffness again observed.

LILIACEAE.

Albuca floribunda Dryand var. *fastigiata* Baker.

Registered number.—O.P.H. No. 9165; 17.11.39.

Origin.—East London, Cape Province.

State and stage of development.—The plant was in the fresh state without flowers or fruit.

Sheep 51158 (6 tooth; 41.9 Kg.) received 3.6 Kg. of the bulbs of the plant in the course of 30 hours.

Result.—Negative.

Ornithogalum saundersiae Baker.

Registered number.—O.P.H. No. 4182A; 28.5.40. and 4832; 7.6.40.

Common name.—

Origin.—Trichardt, Transvaal.

State and stage of development.—Both consignments of the plant (O.P.H. No. 4182A; 28.5.40. and 4832; 7.6.40.) were in the fresh state and in the post-seeding stage.



Fig. 5.—*Crotonia rhodesiae* Baker, f.

Rabbit A (2.9 Kg.) received 90 gm. of the bulbs of the plant in the course of 24 hours.

Symptoms.—The animal died overnight on the second day of dosing.

Post-mortem appearances.—Hyperaemia and oedema of the lungs; hydrothorax; hydroperitoneum; dilatation and hyperaemia of the stomach; severe degenerative changes in the liver; congestion of the kidneys.

Sheep 55603 (4 tooth; 48.2 Kg.) received in a single dose 800 gm. of the bulbs of the plant.

Symptoms.—Apathy; salivation; accelerated and strong pulse; dyspnoea; anorexia; ruminal movements in abeyance; slight tympanites; foetid diarrhoea; lying down and refusing to rise. The animal died 36 hours after dosing.

Post-mortem appearances.—Fairly advanced post-mortem changes; general cyanosis; severe tympanites; subcutaneous haemorrhages; hydropericardium; petechiae of the epicardium; pronounced hyperaemia and oedema of the lungs; degeneration (partially masked by the post-mortem changes) and hyperaemia of the liver and kidneys; hyperaemia of and haemorrhages in the abomasum; hyperaemia of the mucosa of the small intestine and caecum.

Histology. Liver: Slight peripheral fatty changes and bile stasis.

Kidneys: Well marked fatty changes of the renal tubules and active interstitial nephritis especially of the cortex.

The bulbs are used for poisoning rats according to the farmer who submitted the plant for investigation. The minced bulbs are mixed with porridge for this purpose and the mixture is made up into pellets which are placed where the rats have free access to them.

Pseudogaltonia clarata (Baker) Phillips.

Registered number.—O.P.H. No. 13677; 12.2.40. and 14596 18.11.40.

Common name.—Inappropriately termed slangkop.

Origin.—Mariental, South West Africa.

State and stage of development.—The first consignment of the plant (O.P.H. No. 13677; 12.2.40.) was in the fresh state and in the post-seeding stage whilst the second consignment of the plant was in the fresh state and in the flowering stage.

Sheep 54173 (6 tooth; 35.5 Kg.) received 4.9 Kg. of the fresh bulbs of the first consignment of the plant in the course of 48 hours.

Symptoms.—The animal had become pot-bellied and died 54 hours after having received the first dose. For a few hours before death marked apathy was observed.

Post-mortem appearances.—Anaemia; slight cyanosis; hydropericardium; subepicardial petechiae; subendocardial ecchymoses in the left ventricle; hyperaemia and oedema of the lungs; hyperaemia of the mucosa of the posterior part of the trachea; marked regressive changes in the myocardium; tumor splenis; patchy hyperaemia of the mucosa of the small intestine; petechial haemorrhages in the abomasum; tympanites.

TOXICITY OF POISONOUS PLANTS.

Sheep 51158 (6 tooth; 42·8 Kg.) received 9·0 Kg. of the fresh bulbs of the plant in the course of 8 days. The first dose was administered on 19.2.40.

Symptoms.—24.2.40. and 25.2.40. Urine dark reddish-brown in colour; slight apathy; ruminal movements in abeyance; constipation.

26.2.40. and 27.2.40.—Fairly marked apathy; urine dark red and turbid; conjunctivae yellow; rapid and shallow respiration; rapid and weak pulse; anorexia. For the rest as on the previous day.

28.2.40. and 29.2.40.—As for previous day but the conjunctivae were paler although still icteric. Urine dirty brown and turbid.

1.3.40.—As on the previous day. Drenched with 2·0 litres physiological saline followed by 100 c.c. ol. ricini and 200 c.c. ol. lini.

2.3.40.—Drenched with 100 c.c. ol. ricini and 200 c.c. ol. lini. As on the previous day.

3.3.40.—Lively; urine normal; profuse defaecation. Recovery of the animal from 3.3.40. was uninterrupted.

Mr. G. C. S. Roets of this Institute kindly made the following determinations on the blood and urine of the animal:

26.2.40.—The urine contained 0·548 gm. of haemoglobin per 100 c.c. No methaemoglobin was present in the urine.

The blood contained 8·1 gm. of haemoglobin per 100 c.c.

The serum contained 0·4784 gm. of haemoglobin per 100 c.c. and had a bilirubin content of 1·5 van den Bergh units.

27.2.40.—The urine contained 0·4485 gm. of haemoglobin per 100 c.c.

28.2.40.—The haemoglobin content of the urine was 0·004 gm. per 100 c.c.

29.2.40.—The haemoglobin content of the urine was the same as that of the previous day.

Sheep 57164 (6 tooth; 37·3 Kg.) received 3·3 Kg. of the bulbs of the second consignment of the plant in the course of 30 hours.

Result.—Negative.

SOLANACEAE.

Datura stramonium L.

Registered number.—O.P.H. No. 13510; 31.8.37.

Common name.—Stinkblaar, olieboom, white-stramonium.

Origin.—Pretoria, Transvaal.

State an stage of development.—The plant was in the fresh state and in the flowering stage.

Sheep 51758 (6 tooth; 36·0 Kg.) received in the course of 32 days 25·8 Kg. and sheep 52826 (6 tooth; 35·5 Kg.) received in the course of 24 days 18·0 Kg. of the fresh plant.

Result.—Negative.

Sheep 50397 (full-mouth; 38·7 Kg.) received 3·0 Kg. of the mature seeds of the plant in the course of 9 days.

Symptoms.—A few days after commencement of dosing the animal became somewhat restless and excitable. Dilatation of the pupils was observed and it was most pronounced during the afternoon. For the rest the animal was perfectly healthy.

VERBENACEAE.

Lantana camara L.

Registered number.—O.P.H. No. 3016; 13.5.40.

Origin.—Durban, Natal.

State and stage of development.—The plant was in the fresh state and in the flowering and seeding stages with numerous immature and a few mature fruit.

Sheep 55091 (6 tooth; 40·0 Kg.) received 300 gm. of the fresh leaves and young shoots of the plant at 3 p.m. on 14.5.40.

Symptoms.—15.5.40. Received 300 gm. fairly fresh leaves. Conjunctivae very red; serum intensely yellow in colour.

16.5.40.—Received 100 gm. of the dry leaves. Conjunctivae dark red. Voided elongated dark pellets of faeces covered with mucus (constipated).

17.5.40 and 18.5.40.—Exactly as described in sheep 54682.

19.5.40.—Improving but conjunctivae still icteric.

20.5.40.—Feeding. Severe icterus still present.

21.5.40.—The skin covering the ears and face hardening whilst that of the nose was hard, light orange in colour and painful. There was a fairly profuse flow of mucus from the nose and the animal was still constipated.

23.5.40.—The conjunctivae and the skin covering the face, ears and nose was dark yellowish brown. For the rest as on the previous days.

24.5.40.—Icterus decreasing.



Fig. 6.—*Lantana camara* L.

28.5.40.—Icterus almost disappeared. The skin of the nose, face and ears was very hard and was peeling off. At this stage the animal was very emaciated but was feeding well.

The further recovery of the animal was uninterrupted but the skin around the eyes and of the nose remained pink for a long time, hence the name "pink nose".

Sheep 54682 (6 tooth; 35.0 Kg.) received 600 gm. of the fresh leaves and young shoots of the plant at 3 p.m. on 14.5.40.

Symptoms.—15.5.40. Conjunctivae dark red; ruminal movements in abeyance; dyspnoea.

16.5.40.—Voided dark elongated pellets of faeces covered with mucus (constipated). Conjunctivae yellowish-red. The animal received a further 200 gm. of the dry leaves.

17.5.40.—and 18.5.40. Grinding of the teeth; continuous licking of the lips; repeated shaking and scratching of the head; conjunctivae orange in colour; the skin of the face red and warm and swollen; buccal mucous membrane slightly yellowish.

19.5.40.—The animal presented a typical picture of acute photosensitisation and died over-night.



Fig. 7.—*Lantana camara* L. poisoning. NOTE.—Mummification and peeling of the skin on the muzzle, being after-effects of *Lantana* photosensitisation.

Post-mortem appearances.— Advanced post-mortem changes; severe general icterus; infiltration of the subcutaneous tissues of the ears and submaxilla with a yellowish oedematous fluid; hydrothorax; hydroperitoneum; subepicardial and subpleural haemorrhages; severe stasis in the caecum and colon (hard balls of faeces); elongated dry and hard pellets of faeces with partially coagulated blood in the rectum; severe pigmentation of and degenerative changes in the liver.

According to Tucker *Lantana camara* is poisonous but Seddon *et al* (1927-28) had reason to believe that he was dealing with *Lantana crocea* and not with *Lantana camara*. Pound found a plant termed by him *Lantana Sellowiana* poisonous, but Seddon *et al* (1927-28) were informed by C. T. White, Government Botanist, Queensland, that also this plant was *Lantana camara*. Seddon *et al* (1927-28) fed the fresh green shoots of *Lantana camara* with very few flowers and fruits to a Jersey cow. She consumed 80½ lb. of the plant mixed with oaten chaff in twenty-three days without suffering any ill-effects. A ten-months old calf was then fed in the same way as the cow and it consumed 5¾ lb. of the fresh plant during nine days. A third beast (adult cow) then received fresh *Lantana camara* obtained from a different source. This animal was fed in a darkened box for thirty days, but on five of these days no *Lantana* was available. During the thirty days the cow consumed 396 lb. of the plant. At the end of 15 days feeding and again after 30 days feeding, the animal was taken out of the box and exposed to bright sunshine. At no time did the animal develop any symptoms of ill-health.

McIntosh and White (1935) describe the following symptoms in stock poisoned by *Lantana camara*: General weakness, constipation, discharge from the eyes and nose, peeling of the skin, decrease in milk yield, and eventually death. They make no mention of general icterus. On the other hand Mettam (1933) states that *Lantana camara* appears according to the work of Seddon, Carne and McGrath, to be innocuous when fed to stock in massive doses (80½ lb. and 396 lb. in two experiments).

Seddon and Carne (1929) succeeded in proving *Lantana crocea* poisonous for stock. This plant is the cause of the so-called "pinknose" in cattle in Queensland, Australia. Seddon and Carne drenched the water extract of 14 ounces of the green fleshy seeds to a sheep with negative results. They also fed the plant (young green shoots with flowers and green fruit) to a bull, three calves and a cow. The bull ate 30 lb. of the plant in three days and 1 lb. daily for the following ten days. He showed rapid loss in condition and frothing at the mouth but recovered. The three calves (1½ years old) consumed 93½ lb. of the plant in ten days. One died on the 19th day, one was killed and the third one recovered after three weeks. The symptoms and post-mortem appearances described in the calves are very similar to those observed in the sheep dosed with *Lantana camara* at Onderstepoort, the only exception being that Seddon and Carne do not describe the symptoms of photosensitisation, which were so pronounced in our experiments at Onderstepoort. The lactating cow fed by Seddon and Carne ate 22 ounces of *Lantana crocea* leaves, chopped up with the ordinary ration of green peas and green barley, in three days. The milk yield rapidly declined and there were loss of condition, constipation and general icterus. On the third day the milk was watery and showed a faint pinkish

tinge and on the fourth day it was lemon coloured and "when allowed to stand a thick layer of curdy material floated to the top. Examination of this showed it to consist of very numerous polymorphonuclear leucocytes with at times numbers of small lymphocytes present also, and a granular debris. No bacteria were seen on microscopic examination, but on inoculation of cultures staphylococci were observed in the milk from certain quarters". One quarter with altered milk showed no bacteria in the cultures. The milk also contained bile pigment. This animal furthermore showed hyperaemia and subsequent necrosis of the epithelium of the muzzle. These symptoms were undoubtedly the result of photosensitisation caused by the plant, but erroneously ascribed by Seddon and Carne to irritation with some irritant in the plant. In addition to the above animals, Seddon and Carne offered the plant mixed with wheaten chaff to a nine-months calf and a sheep, but both refused to eat it.

Walker (1928) states that *Lantana salvifolia* has been proved non-poisonous for sheep. On the other hand Mettam (1933) was able to kill a sheep with 2.7 lb. (=1225 gm.) of the plant collected during the dry season. The animal died very suddenly without any symptoms having been noticed. The autopsy revealed large quantities of straw-coloured fluid in all three serous cavities, numerous petechiae in the upper air passages and alimentary tract, marked oedema and hyperaemia of both lungs and subendocardial haemorrhages in the left ventricle. It is possible that this animal would have developed symptoms of photosensitisation had it not died so suddenly. A second sheep, which received 6.2 lb (=2820 gm.) of the plant collected during short rains failed to develop any symptoms of poisoning. In 1934 one of us (D.G.S.) drenched a rabbit (3 Kg.) with 60 gm. of the fresh immature fruit of *Lantana salvifolia* with negative results.

From *Lantana crocea* Blackie (1932) isolated a trace of an alkaloidal substance, which, he stated, is being investigated. His "Plant extracts" which were injected into animals failed to produce the characteristic symptoms of *Lantana crocea* poisoning.

DISCUSSION.

From the above information it appears that there has been some doubt about the identity of *Lantana camara* and *Lantana crocea*.

Lantana camara is very generally grown as an ornamental plant (hedges, etc.) and is of very widespread occurrence on the Natal coast. In the course of investigations conducted into a disease among dairy cattle near Durban we suspected poisoning with this plant as the most prominent symptoms exhibited by the cows were general icterus and photosensitisation. From information collected subsequently it appears that *Lantana camara* poisoning in cattle is not uncommon in the Durban area.

TOXICITY OF POISONOUS PLANTS.

The symptoms of poisoning with *Lantana camara* are very similar to those described by Quin (1933) in cases of poisoning with *Lippia Rehmanni* and *Lippia pretoriensis*.

SUMMARY.

The toxicity of sixteen plants was investigated. According to the available literature the following three plants were proved for the first time to be toxic: *Senecio pterophorus* DC; *Kalanchoe paniculata* Harv., *Kalanchoe thyrsiflorae* Harv.

The results obtained in the experiments with *Crotolaria rhodesiae* Baker f. and *Pseudogaltonia clavata* (Baker) Phillips are not considered sufficiently conclusive to prove these two plants to be poisonous.

The latex of *Arauya serisifera* Brot. was proved to possess irritant properties when applied to the skin.

The Senecio alkaloids, *isatidine* and *retrorsine*, administered *per os* to horses induced symptoms, post-mortem appearances, and histological lesions typical of those of "dunsiekte" in horses, and these alkaloids are undoubtedly the active principles of the species of Senecio from which they were isolated.

ACKNOWLEDGEMENTS.

Our thanks are due to Dr E.P. Phillips, Chief, Division of Botany and Plant Pathology, and to Mr. A. O. D. Mogg, Dr. R. A. Dyer and Miss Verdoorn, botanists in the Division of Botany and Plant Pathology, for the identification of plant specimens. To Mr. H. P. A. Boom of the Section of Pathology, Onderstepoort, we are indebted for the histological examination of animal organs. We wish, also, to thank Mr. M. G. van Niekerk and Mr. P. Strydom for assistance rendered in the course of the experiments.

REFERENCES.

- BLACKIE, J. W. (1932). Toxic principle of *Lantana crocea*. *The Analyst* vol. 57, p. 782.
- CHEN, K. K., HARRIS, P. N., AND ROSE, C. L. (1940). The action and toxicity of platyphylline and seneciophylline. *Jour. Pharm. and Exp. Ther.* Vol. 68, p. 130-140.
- CUSHNY, A. R. (1911). On the action of Senecio alkaloids and the causation of the hepatic cirrhosis of cattle (Picton, Moltano or Winton disease). *Jour. Pharm. and Exp. Ther.* Vol. 2, p. 531.
- DAVIDSON, J. (1935). The action of retrorsine on rat's liver. *Jour. Path. and Bact.* Vol. 40, p. 285-293.
- DE WAAL, H. L. (1939). The Senecio alkaloids Pt. I. The isolation of isatidine from *Senecio retrorsus* and *Senecio isatideus*. *Onderstepoort J.*, Vol. 12, pp 155-163.

- HINDMARSH, W. L., AND HART, L. (1937). *Castanosperum australe*, green seeds poisonous to stock. *N.S.W. Veterinary Research Report No. 7*, pp. 109-114.
- HOPKIRK, C. S. M., AND CUNNINGHAM, I. J. (1936). Biological experiment with Jacobine extracted from ragwort by J. R. Hosking and C. W. Brandt. *N.Z. Jour. Sci. and Tech.* Vol. 17, pp. 645-648.
- HOSKING, J. R. AND BRANDT, C. W. (1936). The toxic principle of ragwort (*Senecio jacobaea* L.). *N.Z. Jour. Sci. and Tech.* Vol. 17, pp. 638-644.
- McINTOSCH, K. S. AND WHITE, C. F. (1935). Lantana (*Lantana camara*) and poison peach (*Trema aspera*). Their effects on stock. (Queensland Agric. Jour. Vol. 43, 1935, p. 369-373). *Nutr. Abstr. and Rev.* Vol. 5, p. 554.
- METTAM, R. V. M. (1933). Some poisonous plants of Kenya. *Vet. Jour.* Vol. 89, p. 37-47.
- QUIN, J. I. (1933). Studies on the photosensitisation of animals in South Africa. V. The toxicity of *Lippia Rehmanni* Pears and *Lippia pretoriensis* Pears. *Onderstepoort J.*, Vol. 1, p. 501-504.
- SEDDON, H. R., AND CARNE, H. R. (1929). Feeding tests with Lantana camara. *Science Bull. No. 33, Dept. Agric. N.S. Wales*, p. 91-96.
- SEDDON, H. R., CARNE, H. R., AND McGRATH, T. T. (1927-28). Feeding tests with Lantana camara. *Vet. Res. Rept. No. 5, N.S. Wales*, p. 94-95.
- STEYN, D. G. (1932). A study of the factors concerned in the determination of the toxicity of *Cotyledon orbiculata* L. *18th Rep. Dir. Vet. Serv. and Anim. Ind. Union of South Africa, 1932*, pp. 899-938.
- STEYN, DOUW G. (1934). The toxicology of plants in South Africa. Central News Agency Ltd., Cape Town.
- STEYN, DOUW, G. (1937). Recent investigations into the toxicity of known and unknown poisonous plants in the Union of South Africa VII. *Onderstepoort J.*, Vol. 9, pp. 111-124.
- WALKER, J. (1928). *Lantana salvifolia*. Annual Rept. Dept. Agric., Kenya Colony. p. 162.
- WATT, H. E. (1909). The alkaloids of *Senecio latifolius*. *Proc. Chem. Soc.* Vol. 95, p. 466.