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The distribution, diagnoses and estimated economic impact of plant poisonings and mycotoxicoses in South Africa

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ABSTRACT

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Maps are provided showing the national and provincial distributions of many plant poisonings and mycotoxicoses in South Africa. The various poisonings are briefly described and criteria for their diagnoses are given. The annual mortalities of cattle from plant poisonings/mycotoxicoses in South Africa were calculated as *c.* 37 665 head with a current cash value of *c.* R57 627 450, and that of small stock as *c.* 264 851 head with a value of *c.* R46 878 627. The annual total cost of plant poisonings/mycotoxicoses to the livestock industry of South Africa is conservatively estimated at R104 506 077. These figures do not include hidden losses such as diminished production, reproductive failure, the cost of not utilizing toxic pastures and the fall in price of infested land.

Keywords: Diagnoses, distribution, economic impact, mycotoxicoses, plant poisoning

INTRODUCTION

The primary objectives of this study were to plot the distributions of plant poisonings in South Africa and to outline the criteria for their diagnoses. Since southern Africa has a very rich flora with many poisonous plants (Kellerman, Coetzer & Naudé 1988), it is essential, from a diagnostic point of view, to distinguish plant poisonings from other poisonings and from infectious diseases. Knowledge of the local plant poisonings is a prerequisite for rendering a proper diagnostic service in the region.

This study was also prompted by the need for an objective estimate of stock losses from plant poisonings in South Africa. These losses are very difficult to quantify, even in developed countries such as the United States of America. According to Nielsen & James (1992), the "hard-to-define almost impossible-to-account nature of the problem" is a major obstacle in quantifying the damage caused by plant poisonings in that country. Despite the many imponderables, they nevertheless believed that it was necessary, in today's tough economic environment, to put a dollar value to these losses. Knowledge of the economic impact of plant poisonings is particularly important for determining research priorities, evaluating risk and developing or implementing cost-effective control measures.

The losses induced by plant poisonings can be direct or indirect. Direct losses include death, diminished mass and/or milk yield, and reproductive failure (e.g. abortions, stillbirths, birth defects, interference with oestrus, decreased libido, protracted gestation) (Nielsen & James 1992). The effect of plant poisonings on functional efficiency is particularly difficult to identify and evaluate and for this reason, in the current study, only losses as a result of mortality from plant poisonings will be computed.

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Indirect losses include the cost of:

- control measures (fencing, strategic grazing practices, supplementary feeding, veterinary expenses, etc.) (Nielsen & James 1992)
- temporary or permanent non-utilization of toxic pastures
- the diminished value of infested land

METHODS

The distribution maps of the various plant poisonings were prepared in consultation with experienced veterinarians in all the provinces, and the distributions of the relevant plants (Kellerman *et al.* 1988) and veld types (Acocks 1975) were also taken into account.

The economic impact of plant poisonings on the livestock industry of South Africa was computed according to Nielsen & James (1992). The annual mortality of stock from all causes country-wide, after consultation with experienced veterinarians, was estimated at 3% for cattle and 5% for small stock. Ten percent of the total deaths were attributed to poisonous plants in the case of cattle and 15% in the case of small stock.

The value of a bovine animal was computed from a weighted mean reflecting the herd composition (Nielsen & James 1992). The estimated current price of cattle was provided by the Meat Board. The numbers of stock owned by commercial farmers were obtained from the Department of Agriculture and those of traditional farmers from the Development Bank of South Africa. The composition of the herds under the two systems of management was extrapolated from these figures. When the average value of a cattle unit was calculated, the difference in the composition of herds in the commercial and traditional farming sectors was taken into account.

Small stock were valued at R177 per head, irrespective of the composition of the herds.

Sixty percent of stock losses from poisonings were ascribed to the six most important plants for cattle and small stock. These losses were divided amongst the nine provinces according to the prevalence of the poisonings and stock numbers (Tables 1 and 2).

To account for annual and seasonal variation, the estimated annual mortalities from plant poisonings/mycotoxicoses were taken as the mean of, say, 25 years.

RESULTS

General

The estimated total annual stock losses from all causes in South Africa were:

Class	Number	Mortality (%)	Head
Cattle	12 554 982	3	376 649
Small stock	35 313 430	5	1 765 671

The expected annual losses of stock from plant poisonings/mycotoxicoses in South Africa were:

Class	Mortality from all causes (head)	Mortality from plant poisonings (%)	Losses due to plant poisonings (head)
Cattle	376 649	10	37 665
Small stock	1 765 671	15	264 851

The unit prices of cattle were computed as follows:

Commercial sector

Class	% of herd	Price (R)	Weighted value (R)
Bulls	2,25	3 320	74,70
Cows	50,08	1 660	831,33
Heifers	17,08	1 287	219,82
Steers	12,20	1 660	202,52
Calves	18,39	660	121,37
Total			1 449,74

Traditional sector

Class	% of herd	Price (R)	Weighted value (R)
Bulls	11,29	3 320	374,83
Cows	29,79	1 660	494,51
Heifers	24,70	1 287	317,89
Steers/oxen	26,57	1 660	441,06
Calves	7,65	660	50,49
Total			1 678,78

According to our data, commercial farmers owned 64,62% of the national cattle herd, and traditional farmers, 35,38%.

The estimated mean value for a bovine animal, therefore, was:

R1449,74 X 0,6462 = R936,82
R1678,78 X 0,3538 = R593,95
R1 530,77

The estimated annual cost of plant poisonings/mycotoxicoses in South Africa was:

Class	Number	Price/head (R)	Value (R)
Cattle	37 665	1 530	57 627 450
Small stock	264 851	177	46 878 627
Total			104 506 077

The total annual cost of plant poisonings/mycotoxicoses to the livestock industry in South Africa is, therefore, conservatively estimated at R104 506 077.

The estimated losses caused by the six most important plant poisonings/mycotoxicoses were:

Cattle

Poisoning	Mortality (% of plant poisonings)	Head
Cardiac glycosides	33	12 429
Seneciosis	10	3 767
Gifblaar poisoning	8	3 013
Gousiekte	4	1 507
Lantana poisoning	3	1 130
Diplodiosis	2	753
Total	60	22 599

Small stock

Poisoning	Mortality (% of plant poisonings)	Head
Geeldikkop and dikoor	28	74 158
Vermeersiekte	13	34 431
Cardiac glycosides	10	26 485
Seneciosis	5	13 243
Gousiekte	2	5 297
Diplodiosis	2	5 297
Total	60	158 910

The impact of some major plant poisonings and mycotoxicoses on livestock production in South Africa

The six most important plant poisonings/mycotoxicoses affecting cattle are: poisoning by cardiac glycoside-containing plants, seneciosis, gifblaar poisoning, *Lantana* poisoning, gousiekte and diplodiosis.

Sheep are primarily affected by geeldikkop, poisoning by cardiac glycoside-containing plants, vermeersiekte, seneciosis, gousiekte and diplodiosis.

Poisoning with cardiac-glycoside-containing plants

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Poisoning with cardiac-glycoside(GC)-containing plants is collectively the most important plant-associated poisoning of livestock in southern Africa. South African plants contain two types of cardiac glycosides, viz cardenolides and bufadienolides. Cardenolides have a single unsaturated five-member (butenolide) lactone ring on C17 of the steroid molecule, while bufadienolides have a doubly unsaturated six-member (pentadienolide) lactone ring in that position (Naudé 1977). However, cardenolide-containing plants are of little veterinary importance because they are seldom eaten by stock. All the important CG-containing plants have bufadienolides as their active principles (Kellerman *et al.* 1988).

Poisoning by bufadienolide-containing plants may be either acute or chronic, depending on whether the bufadienolides contained by them have a cumulative effect. Notable amongst those plants that cause only acute poisoning, are tulp (*Homeria pallida, H. miniata, Moraea polystachya, M. bipartita*), slangkop (*Urginea* spp.) and witstorm (*Thesium lineatum*). Bufadienolide-containing plants affect the respiratory, cardiovascular, gastrointestinal and nervous systems. The respiratory signs of acute poisoning are manifested as apnoea or dyspnoea, the cardiac signs as tachycardia, runs of arrhythmia and heart-block, the gastrointestinal signs as bloat, diarrhoea or constipation, and the nervous signs as posterior paresis (Steyn 1949; Kellerman *et al.* 1988).

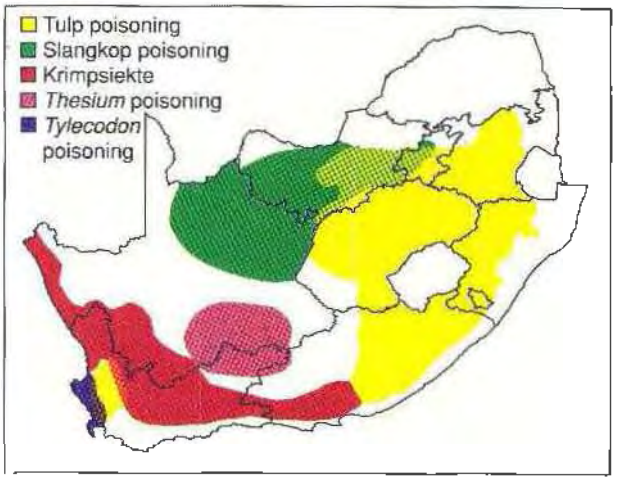

FIG. 1 The distribution of plant-induced cardiac glycoside poisoning in South Africa

Homeria pallida is the tulp most often incriminated in poisoning of stock, and cattle are the species most commonly affected. An important feature of this poisoning is that stock that grow up on tulp-infested veld can learn to avoid the plant. This aversion is strong, therefore poisoning usually occurs only in animals newly introduced from non-infested areas. Since these plants do not lose their toxicity on desiccation, poisoning of stock by tulp-contaminated hay is not uncommon (Steyn 1934; Kellerman *et al.* 1988).

A diagnosis of CG poisoning is made on the grounds of the clinical signs and the presence of appropriate plants showing signs of having been eaten. A diagnosis is confirmed at the laboratory by typical conductive (ECG) changes in the hearts of affected animals (Naudé 1977) and demonstration of cardiac glycosides in rumen content, serum and organs by means of a fluorescent polarization immuno assay (R.A. Schultz, personal communication 1995). The conventional treatment of CG poisoning is activated charcoal administered at a dose of 2 g/kg. Care should be taken not to stress intoxicated animals during treatment as this could lead to fibrillation or heart block (Joubert & Schultz 1982).

TABLE 1 Estimated annual impact of mortalities of cattle from plant poisonings and mycotoxicoses in South Africa

Plant poisoning	South Africa	KwaZulu-Natal		Eastern Cape		Free State		North-West		Mpumalanga		Northern Province		Northern Cape		Western Cape		Gauteng	
	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals
Cardiac-glycoside poisoning	12 429	19	2 362	12	1 492	22	2 734	12	1 491	20	2 486	1	124	8	994	4	497	2	249
Senecíosis	3 767	27	1 017	55	2 072					17	640	1	38						
Gifblaar poisoning	3 013							10	301	10	301	70	2 110					10	301
Gousiekte	1 507	1	15			1	15	40	603	20	301	30	452					8	121
Lantana poisoning	130	40	452	5	56					20	226	15	170					20	226
Diplodiosis	753	16	120			27	204	10	75	27	204	10	75					10	75
Subtotal 60%	22 599		3 966		3 620		2 953		2 470		4 158		2 969		994		497		972
Other plant poisonings 40%	15 066		2 644		2 413		1 969		1 647		2 772		1 979		663		331		648
Total 100%	37 665		6 610		6 033		4 922		4 117		6 930		4 948		1 657		828		1 620
Economic impact @ R1 530/head (R)	57 627 450		10 113 300		9 230 490		7 530 660		6 299 010		10 602 900		7 570 440		2 535 210		1 266 840		2 478 600
No. of cattle in region	12 554 982		2 886 537		2 510 256		1 986 044		1 567 218		1 329 543		1 123 078		453 285		447 436		251 605
Mortalities due to poisonous plants as a % of total number of cattle in region	0,30		0,23		0,24		0,25		0,26		0,52		0,44		0,37		0,19		0,64

TABLE 2 Estimated annual impact of mortalities of small stock from plant poisonings and mycotoxicoses in South Africa

Plant poisoning	South Africa	Eastern Cape		Northern Cape		Free State		Western Cape		Mpumalanga		Kwazulu-Natal		North-West		Northern Province		Gauteng	
	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals	%	No. of animals
Geeldikkop/dikoor	74 158	26	19 281	35	25 955	20	14 832	16	11 865	2	1 483	1	742						
Vermeersiekte	34 431			82	28 233					8	2 755			10	3 443				
Gardiac-glycoside poisoning	26 485	15	3 973	30	7 946	10	2 648	25	6 622	10	2 648	10	2 648						
Senecíosis	13 243	60	7 945							20	2 649	20	2 649						
Gousiekte	5 297									10	530	10	530	65	3 442	10	530	5	265
Diplodiosis	5 297					27	1 430			27	1 430	16	847	10	530	10	530	10	530
Subtotal 60%	158 910		31 199		62 134		18 910		18 487		11 494		7 416		7 415		1 060		795
Other plant poisonings 40%	105 941		20 799		41 423		12 607		12 325		7 663		4 944		4 943		707		530
Total 100%	264 851		51 998		103 557		31 517		30 812		19 157		12 360		12 358		1 767		1 325
Economic impact @ R177/head (R)	46 878 627		9 203 646		18 329 589		5 578 509		5 453 724		3 390 789		2 187 720		2 187 366		312 759		234 525
No. of small stock in region	35 313 430		10 768 568		8 079 083		6 220 174		3 716 208		2 222 542		1 835 397		1 270 185		1 076 631		124 642
Mortalities due to poisonous plants as a % of total number of small stock in region	0,75		0,48		1,28		0,51		0,83		0,86		0,67		0,97		0,18		1,06

Chronic cardiac glycoside poisoning or krimpsiekte, on the other hand, is a paretic condition of small stock (and more rarely of cattle) brought about by members of the family Crassulaceae, which contain cumulative bufadienolides. Included amongst these are *Tylecodon wallichii*, *T. ventricosus*, *Cotyledon orbiculata* and possibly *T. grandifloris*. *Tylecodon wallichii*, the most important of the krimpsiekte-inducing plants, mostly affects animals grazing on south-facing slopes of hills and ridges (where the plant grows abundantly) in the Little Karoo and southern fringes of the Great Karoo. *Cotyledon orbiculata*, the second most important of these plants, being highly ornamental, can poison stock that have access to garden waste anywhere. Although cumulative and non-cumulative bufadienolides have been isolated from *T. grandifloris*, this plant has been incriminated only in acute poisoning of cattle on the Western Cape coast (Kellerman *et al.* 1988).

In chronic poisoning with cumulative bufadienolides, the respiratory, cardiac and gastrointestinal signs are diminished or absent, while the nervous signs are prominent. Affected stock die suddenly, or manifest paresis and paralysis. The animals that survive a large single dose, or repeated small doses, lag behind the flock, assume a characteristic pose (with the feet together and the back arched), lie down frequently and develop protracted paresis/paralysis, often lasting for weeks. A few display persistent torticollis (Steyn 1949; Henning 1926; Kellerman *et al.* 1988).

The lesions of acute or subacute cardiac-glycoside poisoning, namely extracardiac signs of heart failure and scattered foci of myocardial necrosis with round-cell infiltration, are sometimes lacking in krimpsiekte. A diagnosis, therefore, is made on locality, clinical signs and evidence that plakkies on the toxic pasture had been grazed (Kellerman *et al.* 1988).

Krimpsiekte is the only plant poisoning reputed to cause secondary poisoning; humans and animals that eat the meat of krimpsiekte carcases may themselves become affected (Henning 1926).

ECONOMIC IMPORTANCE

TABLE 3 Expected annual impact of mortalities from CG-containing plants on the livestock industry of South Africa

Class	%*	No. of animals	Loss (R)
Cattle	33	12 429	19 016 370
Small stock	10	26 485	4 687 845
Total			23 704 215

* Percentage of all mortalities from plant poisonings and mycotoxicoses

Geeldikkop and Panicum *photosensitization*

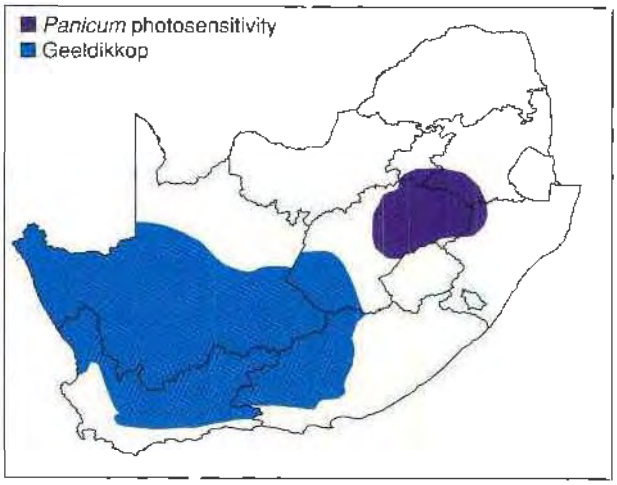

FIG. 2 The distribution of geeldikkop and *Panicum* photosensitivity in South Africa

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Geeldikkop is a hepatogenous photosensitivity disease of sheep and goats grazing on *Tribulus terrestris*, principally in the Karoo (Theiler 1918). More than half a million animals may be affected in a single season (Steyn 1949).

Outbreaks most commonly occur in lambs grazing on young, wilted *T. terrestris*, during hot, dry spells following summer rains (Steyn 1949).

The disease has been experimentally induced by feeding *T. terrestris* (Theiler 1918; Van Tonder, Basson & Van Rensburg 1972) and crude extracts of steroidal saponins from the plant (Kellerman, Erasmus, Coetzer, Brown & Maartens 1991). Hepatogenous photosensitivity results from failure of the liver to excrete phylloerythrin (a photodynamic porphyrin) produced by the degradation of chlorophyll in the gut of herbivorous animals. In geeldikkop, phylloerythrin is believed to accumulate in the blood as a result of the occlusion of bile ducts by crystalloid material (Kellerman, Van der Westhuizen, Coetzer, Roux, Marasas, Minne, Bath & Basson 1980; Coetzer, Kellerman, Sadler & Bath 1983) composed of the Ca^{2+} glucuronides of epismilagenin and episarsasapogenin. The steps proposed for the formation of the "crystals" include hydrolysis of a steroidal saponin from *T. terrestris* to the aglycone (diosgenin) in the rumen, followed by reduction of the double bond on C-5 and epimerization at C-3 (3ß-OH to 3α-OH). The resultant epismilagenin and episarsasapogenin are conjugated with glucuronic acid in the liver, to form glucuronides which precipitate as insoluble calcium salts or "crystals" in the bile ducts (Miles, Wilkins, Erasmus, Kellerman & Coetzer 1994; Miles, Wilkins, Erasmus & Kellerman 1994).

A diagnosis of geeldikkop can be made in small stock which develop photosensitivity and icterus while grazing on *T. terrestris*. The diagnosis is confirmed by the presence of crystalloid material in the liver accompanied by concentric, lamellar periductal fibrosis (Coetzer *et al.* 1983). Affected sheep must be kept in the shade and, where practical, treated symptomatically.

Panicum photosensitivity or dikoor is common in sheep grazing on *Panicum coloratum* pastures on the Highveld of the Free State, Gauteng, Mpumalanga, and KwaZulu-Natal. This nutritious grass grows profusely on soil which has been disturbed, especially for the planting of winter wheat. As in the case of *T. terrestris*, *Panicum* grass periodically becomes toxic under certain specific conditions, for instance when wilted during hot, dry spells following summer rains (Kellerman *et al.* 1988). Workers in New Zealand have shown that the composition of the intrabiliary crystalloid material induced by *P. dichotomiflorum* and *P. schinzii* is similar to that of geeldikkop (Miles, Munday, Holland, Smith, Embling & Wilkins 1991; Miles, Munday, Holland, Lancaster & Wilkins 1992). The clinical signs and lesions of *Panicum* photosensitivity are indistinguishable from those of geeldikkop.

Geeldikkop and dikoor can be distinguished from other hepatogenous photosensitizations on the grounds of the histopathological changes in the liver.

ECONOMIC IMPORTANCE

TABLE 4 Expected annual impact of mortalities from geeldikkop and *Panicum* photosensitivity on the livestock industry of South Africa

Class	%*	No. of animals	Loss (R)
Small stock	28	74 158	13 125 966

* Percentage of all mortalities from plant poisonings and mycotoxicoses

Seneciosis

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Seneciosis is the most notable non-photosensitizing hepatotoxicosis of stock in southern Africa. Of the many toxic species in South Africa, *Senecio latifolius* (Mpumalanga and KwaZulu-Natal) and *S. retrorsus* (Eastern Cape) cause the most losses. Horses seem to be the most susceptible, followed by cattle, sheep and goats, in that order; however, since few horses are kept extensively nowadays, cattle are the species most often affected (Henning 1932; Kellerman *et al.* 1988).

Stock may be either acutely or chronically poisoned, depending on the toxicity, the amount of *Senecio* ingested and the duration of exposure to the plant.

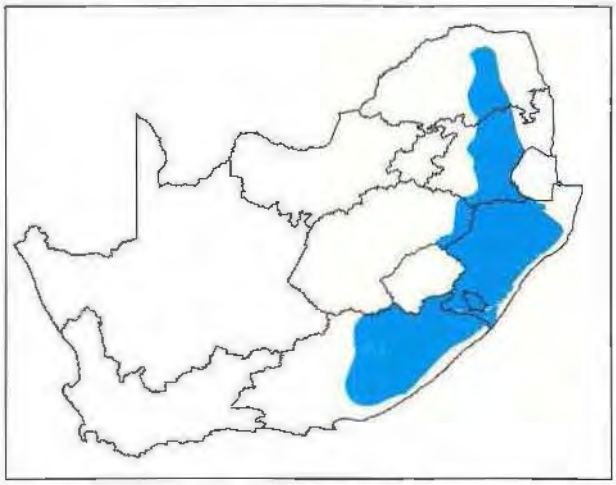

FIG. 3 The distribution of seneciosis in South Africa

Acutely poisoned animals usually start dying within a day or two of showing signs such as depression, weakness, ruminal stasis, constipation and icterus. Chronically affected cattle and horses develop Molteno straining disease and dunsiekte, respectively. Apart from emaciation, which is a feature of both diseases, Molteno straining disease is associated with tenesmus, diarrhoea, and sometime nervous signs. Dunsiekte, in turn, is marked by emaciation, depression, stupor, aimless wandering, locomotory disturbance, and even frenzy. The nervous signs of chronic seneciosis are attributed to hepatic encephalopathy (Henning 1932; Kellerman *et al.* 1988).

The histopathological features of pyrrolizidine alkaloid poisoning are well known and range from centrilobular and lytic necrosis of hepatocytes, haemorrhage, and pooling of blood (acute stage) to severe fibrosis or cirrhosis, characterized by ductular proliferation, veno-occlusive changes, megalocytosis, karyomegaly and nodular hyperplasia (chronic stage) (Jubb & Kennedy 1970).

Outbreaks of seneciosis most commonly occur in stock feeding on newly sprouted *Senecio* plants on veld denuded by droughts, overstocking and burning (Henning 1932; Kellerman *et al.* 1988).

ECONOMIC IMPORTANCE

TABLE 5 Expected annual impact of mortalities from seneciosis on the livestock industry of South Africa

Class	%*	No. of animals	Loss (R)
Cattle	10	3 767	5 763 510
Small stock	5	13 243	2 344 011
Total			8 107 521

* Percentage of all mortalities from plant poisonings and mycotoxicoses

Vermeersiekte

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Vermeersiekte is a major intoxication of ruminants, especially small stock, grazing on *Geigeria* spp. The most important of these, *G. ornativa*, causes widespread vermeersiekte in the dry western parts of the country, while *G. aspera* is responsible for more localized outbreaks on the Highveld (Du Toit 1928; Steyn 1949).

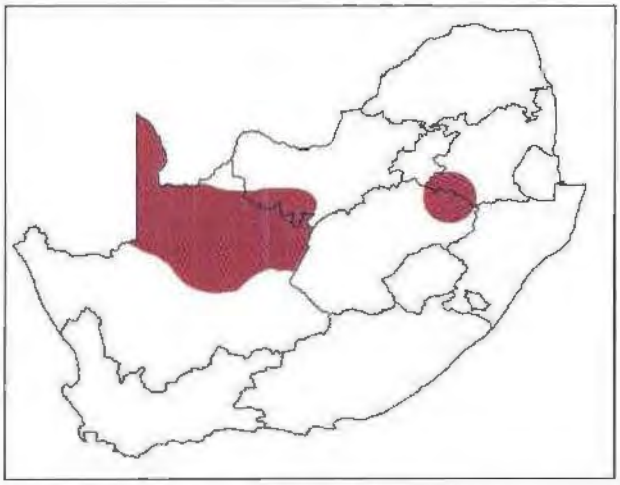

FIG. 4 Distribution of vermeersiekte in South Africa

The principal clinical signs are regurgitation of ruminal content, stiffness, paresis and paralysis. Sheep and goats may manifest one or more of these signs, while cattle contract mainly the paretic form. At necropsy, foreign-body pneumonia, ruminal content in the air passages, and sometimes marked dilatation of the oesophagus are evident (Du Toit 1928; Grosskopf 1964). The primary lesions occur in the skeletal muscles and oesophagus where some fibres are atrophic, degenerated, and necrotic (Pienaar, Kriek, Naudé, Adelaar & Ellis 1973). The toxic principles are α, β-unsaturated-8-sesquiterpene lactones (Anderson, de Kock & Pachler 1967; De Kock, Pachler, Ross & Wessels 1968).

There is no specific treatment. Although mortalities may be high, the prognosis is good if stock are promptly removed from toxic camps. Control of vermeersiekte is based on good pasture management and tactical grazing practices: periods of controlled high-density, non-selective grazing are followed by spells of recuperation on non-infested veld (Grosskopf 1964; Kellerman *et al.* 1988).

A million sheep once died of vermeersiekte in Griqualand West during a single season (Grosskopf 1964).

ECONOMIC IMPORTANCE

Since vermeersiekte is principally a disease of small stock, mortalities of cattle were not estimated.

TABLE 6 Expected annual impact of mortalities from vermeersiekte on the livestock industry of South Africa

Class	%*	No. of animals	Loss (R)
Small stock	13	34 431	6 094 287

* Percentage of all mortalities from plant poisonings and mycotoxicoses

Gifblaar poisoning

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Dichapetalum cymosum, or gifblaar, is a shrublet, about 150 mm tall, with a well developed underground system. The underground stem of a gifblaar plant has been excavated for 30 m to a depth of 12 m. Poisoning usually occurs when the leaves sprout in spring and autumn (Steyn 1949; Kellerman *et al.* 1988).

The toxic principle, monofluoroacetate (Marais 1944), is converted in the body to monofluorocitrate, which blocks the tricarboxylic acid cycle by inhibiting aconitase. A catastrophic loss of cellular respiration results. Affected ruminants typically drop dead from cardiac failure after drinking water or undergoing even mild exertion. On rare occasions, nervous signs are seen. No lesions have been recorded in the hearts of acutely poisoned animals, while chronic poisoning is believed to cause myocardial lesions somewhat similar to those of gousiekte (Steyn 1949; Kellerman *et al.* 1988).

A diagnosis of gifblaar poisoning is made on the grounds of the history, fragments of *D. cymosum* leaves in the rumen and demonstration of mono-

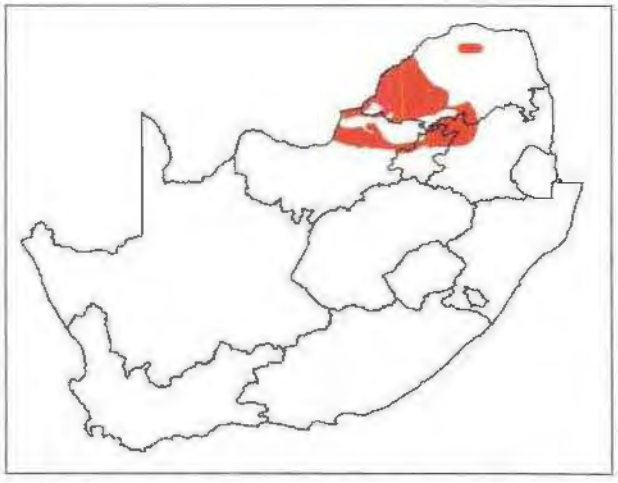

FIG. 5 Distribution of gifblaar poisoning in South Africa

fluoroacetate in rumen contents and livers of poisoned animals (T.W. Naudé, unpublished data 1995)

Animals that have been exposed to gifblaar should be rested and withheld from water for at least 24 h. There is no antidote (Kellerman *et al.* 1988).

Gifblaar poisoning is controlled by fencing off the plant (which virtually does not spread) from stock and/or eradicating it. The plant can be exterminated by excavating a short length of underground stem and immersing the tip in a bottle of 1–2,5% aqueous solution of $CuSO_4$ planted in the soil (Steyn 1949; Kellerman *et al.* 1988). Alternatively, herbicidal sprays containing picloram or terbuthiuron granules may be applied.

ECONOMIC IMPORTANCE

TABLE 7 Expected annual impact of mortalities from gifblaar poisoning on the livestock industry of South Africa

Class	%*	No. of animals	Loss (R)
Cattle	8	3 013	4 609 890

* Percentage of all mortalities from plant poisonings and mycotoxicoses

Gousiekte

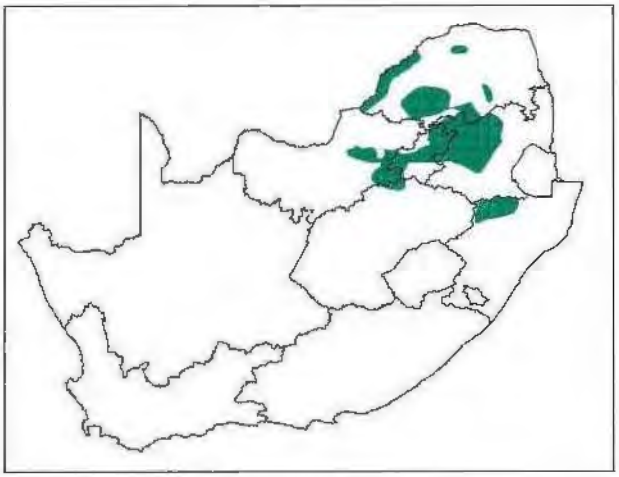

FIG. 6 Distribution of gousiekte in South Africa

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Gousiekte is a disease of ruminants characterized by sudden heart failure 6–8 weeks after the ingestion of certain rubiaceous plants. *Pachystigma pygmaeum* (North-West Province) is the most important of these, followed in descending order by *Fadogia homblei* (central Northern Province, Guateng, the north-eastern portion of Mpumalanga), *Pavetta harborii* (far Northern Province), *Pachystigma thamnus* (KwaZulu-Natal), *Pavetta schumanniana* (mostly Northern Province) and *Pachystigma latifolium* (Mpumalanga). The wildedadel, *Fadogia homblei*, is said to cause stock losses mostly in early summer, and *Pachystigma pygmaeum*, later in the season, while *Pavetta harborii* and *P. schumanniana* are supposed to induce gousiekte throughout the year (Theiler, Du Toit & Mitchell 1923; Kellerman *et al.* 1988; Fourie, Erasmus, Prozesky & Schultz 1994).

Affected stock typically drop dead without warning during even mild exertion or stress. A diagnosis of gousiekte depends on the locality, a history of peracute deaths and the presence of typical lesions such as replacement fibrosis with round-cell infiltration of the endocardial zone (Newsholme & Coetzer 1984; Fourie *et al.* 1994). The toxicity of plants is confirmed by demonstrating the newly isolated (as yet uncharacterized) active principle in their tissue (Fourie 1994).

Subclinically affected animals can be identified by elevations in the activity of serum apartate transaminase during latency, and then be slaughtered. There is no treatment (Fourie, Schultz, Prozesky, Kellerman & Labuschagne 1989).

ECONOMIC IMPORTANCE

TABLE 8 Expected annual impact of mortalities from gousiekte on the livestock industry of South Africa

Class	%*	No.of animals	Loss (R)
Cattle	4	1 507	2 305 710
Small stock	2	5 297	937 569
Total			3 243 279

* Percentage of all mortlities from plant poisonings and mycotoxicoses

Diplodiosis

DISTRIBUTION, DIAGNOSIS AND TREATMENT

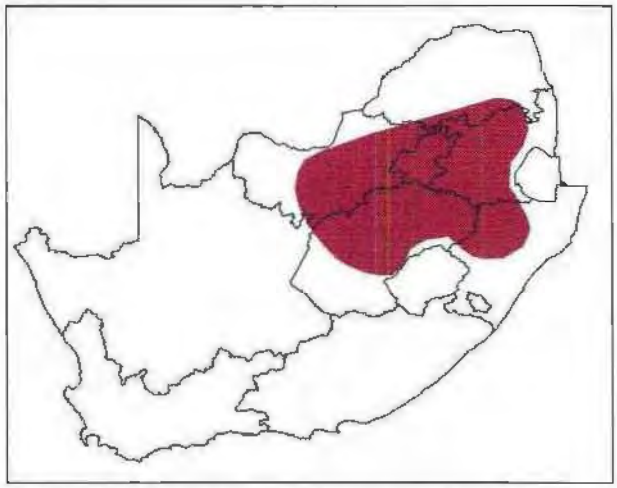

FIG. 7 The principal maize-producing area of South Africa

Diplodiosis is a neuromycotoxicosis of cattle and sheep grazing on harvested maize lands in winter. The disease, caused by the ingestion of maize infected with the common cob-rot fungus, *Diplodia maydis*, is characterized by ataxia (stiff-legged, high-stepping gait), paresis and paralysis. New cases can appear up to 10 d after withdrawal from toxic lands and the prognosis is usually good if stock are removed as soon as the first signs appear. Paretic or paralytic animals may die of hunger or thirst if neglected (Mitchell 1918; Kellerman, Rabie, Van der Westhuizen, Kriek & Prozesky 1985). The unknown neurotoxin produced by the fungus also causes stillbirths and neonatal losses in the offspring of dams exposed to diplodiosis in the second and third trimesters of pregnancy. Even the lambs and calves of dams that have never shown overt signs of diplodiosis may be affected (Kellerman, Prozesky, Schultz, Rabie, Van Ark, Maartens & Lübben 1991). A diagnosis is made on the grounds of the clinical signs and a history of exposure to maize foggage. Diplodiosis in neonates is confirmed histopathologically by the presence of a *status spongiosus* in the white matter of their brains (Prozesky, Kellerman, Swart, Maartens & Schultz 1994). There is no specific treatment. If other roughage is not available, the toxic maize litter may be passed through a hammer mill and fed to the stock. The milled plants are thought to be less toxic because the mouldy cobs are broken up and distributed through the material, thus diluting the toxin (Kellerman *et al.* 1988).

ECONOMIC IMPORTANCE

TABLE 9 Expected annual impact of mortalities from diplodiosis on the livestock industry of South Africa

Class	%*	No. of animals	Loss (R)
Cattle	2	753	1 152 090
Small stock	2	5 297	937 569
Total			2 089 659

* Percentage of all mortalities from plant poisonings and mycotoxicoses

Lantana *poisoning*

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Lantana camara poisoning is the most important hepatotoxicosis of cattle in South Africa, after seneciosis. Significant mortalities from this poisoning occur (often in plantations or along streams) throughout KwaZulu-Natal, Mpumalanga, Gauteng and the Northern Province (Kellerman *et al.* 1988). The active principles are pentacyclic triterpenes, of which icterogenin is probably the best known (Anderson, De Kock & Enslin 1961). Affected cattle may die acutely or show signs of hepatogenous photosensitivity such as icterus, photodermatitis, orange-brown discoloration and swelling of the liver, intestinal impaction and nephrosis. The nephrosis can be severe and accompanied by diarrhoea, supposedly as a result of uraemia. Histopathological examination reveals the hepatocytes to be well delineated, swollen and to have undergone degenerative changes (Fourie, Van der Lugt, Newsholme & Nel 1987; Kellerman *et al.* 1988). A diagnosis is made on the grounds of the clinical signs, the presence of *L. camara* (which has been eaten), and appropriate lesions. Poisoned animals should be removed from infested paddocks, kept in the shade, dosed with activated charcoal and treated symptomatically.

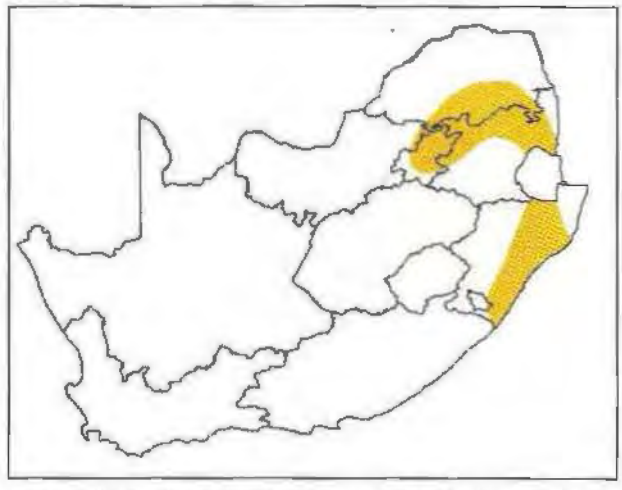

FIG. 8 Distribution of *Lantana camara* poisoning in South Africa

ECONOMIC IMPORTANCE

TABLE 10 Expected annual impact of mortalities from *Lantana* poisoning on the livestock industry of South Africa

Class	%*	No. of animals	Loss (R)
Cattle	3	1 130	1 728 900

* Percentage of all mortalities from plant poisonings and mycotoxicoses

The impact of plant poisonings and mycotoxicoses on livestock production in the provinces

Northern Province

DISTRIBUTION, DIAGNOSIS AND TREATMENT

The most common plant poisonings and mycotoxicoses of the Northern Province, more or less in order of priority, are gifblaar poisoning (Fig. 9), gousiekte (Fig. 9), *Lantana* poisoning (Fig. 8), diplodiosis (Fig. 7), maldronksiekte (Fig. 10), albiziosis (Fig. 10),

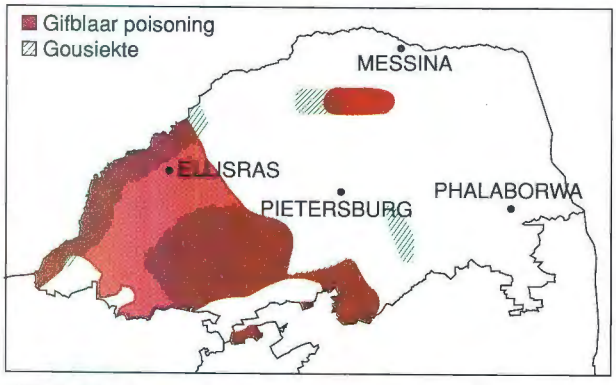

FIG. 9 Distribution of gifblaar and gousiekte in the Northern Province

slangkop poisoning, prussic-acid poisoning, malkopui poisoning, crotalariosis, *Aspergillus clavatus* poisoning, *Sarcostemma viminale* poisoning, aflatoxicosis, *Panicum* photosensitivity (dikoor), seneciosis, *Ornithogalum prasinum* (chink) and *Ficus* poisoning.

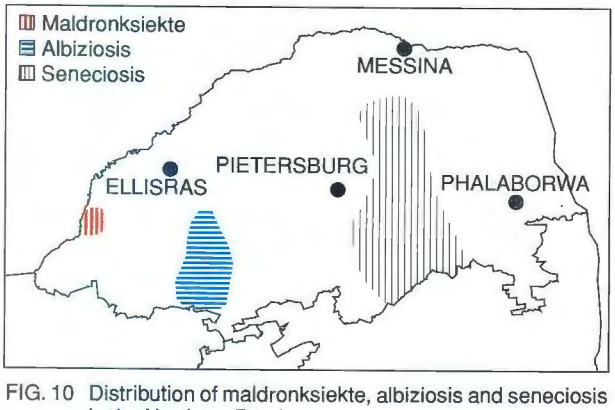

FIG. 10 Distribution of maldronksiekte, albiziosis and seneciosis in the Northern Province

The following poisonings are described under the sections or provinces listed in brackets: *Dichapetalum cymosum* poisoning (Gifblaar poisoning), gousiekte (Gousiekte), diplodiosis (Diplodiosis), *Lantana* poisoning (*Lantana* poisoning), *Dipcadi glaucum* or malkopui poisoning (Northern Cape), *Urginea sanguinea* or slangkop poisoning (Poisoning with cardiac glycoside-containing plants), *Sarcostemma viminale* poisoning (North-West), dikoor (Geeldikkop and *Panicum* photosensitivity), seneciosis (Seneciosis), *Ornithogalum prasinum* or chink poisoning (Western Cape) and *Ficus* poisoning (Gauteng).

Plant poisonings of ruminants in the province are dominated by gifblaar poisoning and gousiekte. Extremely rare intoxications such as those caused by *Ficus* spp., *Abrus precatorius* and vetches (*Vicia* spp.) will not be discussed here.

Maldronksiekte is a nervous disorder of cattle, resulting from the protracted grazing of *Solanum kwebense* growing under geelhaak thorn trees (*Acacia erubescens*) on overgrazed veld in a specific locality near Rooibokkraal (Fig. 10). Animals undergo temporary loss of balance and transient epileptiform seizures induced by a variety of stimuli such as exercise, dipping and fright. When not disturbed, affected animals appear to be quite normal. The condition is irreversible and death ensues from self-trauma or trampling. A diagnosis of maldronksiekte is confirmed by the presence of typical lesions, viz atrophy of the cerebellum accompanied by degeneration and necrosis of Purkinje cells (Pienaar, Kellerman, Basson, Jenkins & Vahrmeijer 1976).

Lantana camara poisoning (in contrast to *Lippia* spp. poisoning) has been diagnosed regularly in many parts of the province, including Thabazimbi.

Like maldronksiekte, albiziosis in South Africa is limited to the Northern Province (Fig. 10). Albiziosis, which results from the ingestion by cattle of *Albizia tanganyicensis* pods blown down from the trees by spring winds, is marked by hypersensitivity, intermittent convulsions and high mortality (Basson, Adelaar, Naudé & Minne 1970). The active principle is a 4-methoxy derivative of pyridoxine. A diagnosis of albiziosis is made on circumstantial evidence such as clinical signs, locality, time of the year, and large quantities of pods in the rumen. The antidote is vitamin B_6 (Gummow, Bastianello, Labuschagne & Erasmus 1992).

Transvaal slangkop, *Urginea sanguinea*, being widely distributed in the province, sporadically poisons stock in various localities (Kellerman *et al.* 1988). The condition is described under Poisoning with cardiac glycoside-containing plants.

Prussic acid poisoning is occasionally responsible for peracute deaths in stock grazing on wilted sorghum coppice (opslag) on the Springbok Flats, wilted twigs of thorn trees knocked down by hail, etc. The diagnosis is confirmed by a qualitative positive picrate test on the ruminal content and leaves of the forage plants. Quantitative determinations are done on rumen content, liver and skeletal muscle preserved by either freezing or 1% $HgCl_2$ (Kellerman *et al.* 1988).

Laminitis and overgrown hooves are associated with cattle and sometimes sheep which have ingested *Crotalaria* spp. Mobility is inhibited, leading to mass loss and starvation. Outbreaks have been reported in different parts of the province, including the Pietersburg district (Steyn 1949; Kellerman *et al.* 1988).

Aspergillus clavatus poisoning is a mycotoxicosis of ruminants, caused by ingestion of sorghum beer residue, maize sprouts or feed pellets infected by the fungus. The clinical signs include hypersensitivity, pronounced muscle tremors, ataxia, progressive paresis and paralysis. Mortality is high and survivors may show permanent locomotory disturbance. Swelling

and necrosis of larger motor neurons in the ventral horns of the spinal cord, medulla oblongata, midbrain and thalamus, corroborate the diagnosis (Kellerman, Pienaar, Van der Westhuizen, Anderson & Naudé 1976; Kellerman, Newsholme, Coetzer & Van der Westhuizen 1984).

Aflatoxicosis occurs from time to time, mostly in pigs fed home-grown ground nuts infected by *Aspergillus flavus* on the Springbok Flats. The condition is diagnosed on the grounds of the typical lesions in the liver and determination of the aflatoxin content of the rations (Kellerman *et al.* 1988).

ECONOMIC IMPORTANCE

TABLE 11 Expected annual impact of mortalities from plant poisonings and mycotoxicoses on the livestock industry of the Northern Province

Poisoning	Cattle	Sheep	Price (R)
Gifblaar	2 110		3 228 300
Gousiekte	452	530	785 370
Lantana	170		260 100
Diplodiosis	75	530	208 560
Cardiac glycosides	124		189 720
Seneciosis	38		58 140
Maldronksiekte	20		30 600
Others			3 122 409
Total			7 883 199

Gauteng Province

DISTRIBUTION, DIAGNOSIS AND TREATMENT

The natural vegetation of Gauteng—the most populous and industrialized province of South Africa—has been significantly disturbed by human activities. This is especially true in the peri-urban areas, where relatively uncommon stock poisonings (such as those caused by algal blooms in eutrophied water or by ornamental plants) are diagnosed from time to time. However, in Gauteng, as in the provinces immediately to its north, east and west, gousiekte and gifblaar poisoning account for many deaths. The plant poisonings and mycotoxicoses of Gauteng, in approximate order of significance, are gifblaar poisoning (Fig. 11), *Lantana camara* poisoning (Fig. 8), gousiekte (Fig. 6), diplodiosis (Fig. 7), poisoning with the blooms of *Microcystis aeruginosa*, *Panicum* photosensitization (dikoor) (Fig. 2), *Aspergillus clavatus* poisoning, slangkop poisoning, prussic-acid poisoning, poisoning with garden waste (e.g. *Ornithogalum* spp., plakkies, *Acokanthera* spp., cabbages, onions, avocado leaves), equine leukoencephalomalacia (LEM), facial eczema, zearalenone poisoning (hyperoestrogenism in pigs), *Acacia nilotica* poisoning, and poisoning of ruminants with wild figs.

The following poisonings are discussed under the sections or provinces listed in brackets: gousiekte (Gousiekte) and gifblaar poisoning (Gifblaar poisoning), *Lantana camara* poisoning (*Lantana* poisoning), diplodiosis (Diplodiosis), dikoor (Geeldikkop and *Panicum* photosensitization), *Aspergillus clavatus* tremors (Northern Province), slangkop poisoning (Poisoning with cardiac glycoside-containing plants), prussic acid poisoning (Northern Province), and facial eczema (Eastern Cape).

FIG. 11 Distribution of gifblaar and gousiekte in Gauteng

FIG. 12 Distribution of tulp and slangkop poisoning in Gauteng

Zearalenone poisoning, manifested as hyperoestrogenism in pigs fed on mouldy maize colonized by the cob-rot fungus, *Fusarium graminearum*, will be discussed with the poisonings of KwaZulu-Natal.

Microcystis aeruginosa is a widely distributed toxic cyanobacterium with pin head-sized colonies composed of minute globular organisms. On calm, hot days, the bloom rises to the surface to form a lettuce-green scum on the leeward banks, where it is ingested by stock. Poisoning of cattle, sheep, donkeys and other animals has been recorded on the Vaal, Hartebeestpoort and Bon Accord Dams. Acutely affected animals might display convulsions and paralysis, but normally only signs of liver damage, such as icterus and hepatogenous photosensitization, are apparent. The histopathological lesions range from massive necrosis of the liver to diffuse fatty metamorphosis accompanied by small foci of hepatocytic necrosis (Stephens 1949; Kellerman *et al.* 1988). The hepatotoxins of *M. aeruginosa* have been identified as cyclic heptapeptides, commonly known as cyanoginosins (Santikarn, Williams, Smith, Hammond, Botes, Tuinman, Wessels, Viljoen & Kruger 1983). A diagnosis is made on the grounds of the history, clinical signs, pathological changes and demonstration of the toxin in the blooms by chemical analysis or biological trials (Kellerman *et al.* 1988) .

Living in proximity to a large human population can expose stock to toxic ornamental plants from other regions, e.g. *Ornithogalum thyrsoides*, *O. saundersii* (see poisonings of the Western Cape), various plakkies (Northern Cape), *Acokanthera* spp. and *Nerium oleander* containing cardiac glycosides. Market-garden waste such as spoilt cabbages (containing S-methylcysteine sulphoxide converted in the rumen to dimethyl disulphide) and onions (n-propyl disulphide) induces fatal haemolytic anaemia in ruminants. Goats, ostriches and other animals that ingest avocado leaves, may die of heart failure. Goats are also prone to prussic acid poisoning induced by *Osteospermum* spp. and nitrate poisoning from eating highly fertilized vegetables. Ripe drupes of *Melia azedarach* (syringa tree) are occasionally responsible for mortalities and signs such as dyspnoea, tremors and paralysis in pigs, cattle and sheep (Kellerman *et al.* 1988).

Leukoencephalomalacia (LEM) must always be considered as a differential diagnosis when horses that have access to maize, die. LEM is a highly fatal neuromycotoxicosis of Equidae, and is caused by the ingestion of maize infected by the ubiquitous fungus *Fusarium moniliforme*. The condition is usually seen in horses grazing on harvested maize fields or fed mouldy home-grown maize rations. Sweating, tremors, changes in temperament, locomotory disturbance, convulsions and death are the notable signs of this intoxication. The onset of signs is usually abrupt and the course short (a few hours to three or four days). Recovery is rare. Coronal sections of the cerebrum reveal large cavities in the cortex where the white matter has undergone liquifactive necrosis. A diagnosis is made on the grounds of nervous signs in horses fed on mouldy maize products, the presence of malacic lesions in the cerebral cortex, and toxic levels of fumonisin B_1 in the rations (Marasas, Kellerman, Pienaar & Naudé 1976; Marasas, Kellerman, Gelderblom, Coetzer, Thiel & Van der Lugt 1988; Kellerman, Marasas, Thiel, Gelderblom, Cawood & Coetzer 1990).

Goats occasionally die or abort after having eaten the pods of felled *Acacia nilotica* trees. Although methaemoglobinaemia is the outstanding feature of this poisoning, the plant does not contain abnormally high nitrate levels (Terblanche, Pienaar, Bigalke & Vahrmeijer 1967).

Facial eczema of cattle has on two occasions been diagnosed on *Lolium perenne*/clover pastures at sewage farms in the Province. The condition will be more fully discussed with those of the Eastern Cape.

Rare outbreaks of neurotoxicosis have been recorded in cattle feeding on *Ficus ingens* and *F. cordata* in times of extreme drought (Myburgh, Fourie, Van der Lugt, Kellerman, Cornelius & Ward 1994).

ECONOMIC IMPORTANCE

TABLE 12 Expected annual impact of mortalities from plant poisonings and mycotoxicoses on the livestock industry of Gauteng

Poisoning	Cattle	Shaep	Price (R)
Gifblaar	301		460 530
Cardiac glycosides	249		380 970
Lantana	226		345 780
Gousiekte	121	265	232 035
Diplodiosis	75	530	208 560
Microcystis	30		45 900
Others			1 039 350
Total			2 713 125

Mpumalanga

DISTRIBUTION, DIAGNOSIS AND TREATMENT

The notable plant poisonings of this Province, roughly in order of importance, are tulp poisoning (Fig. 13), seneciosis (Fig. 13), diplodiosis (Fig. 7), gousiekte (Fig. 14), vermeersiekte (Fig. 13), poisoning by *Gnidia burchellii*, *Dichapetalum cymosum* (Fig. 14), *Panicum coloratum* (Fig. 2), *Lantana camara* (Fig. 8), *Microcystis aeruginosa*, *Cucumis* spp., *Ornithogalum saundersiae*, and *Paspalum* staggers.

The following plants are discussed under the sections or provinces listed in brackets: *Homeria pallida* or yellow tulp (Poisoning with cardiac glycoside-containing plants); *Senecio latifolium* (Seneciosis); *Diplodia maydis* (Diplodiosis); *Pachystigma pygmaeum*, *P. thamnus*, *P. latifolium*, *Fadogia homblei* and *Pavetta schumanniana* (Gousiekte); *Geigeria aspera* (Vermeer-

siekte); *Dichapetalum cymosum* (Gifblaar poisoning); dikoor (Geeldikkop and *Panicum* photosensitization); *Lantana camara* (*Lantana* poisoning); *Microcystis aeruginosa* (alga/cyanobacterium) (Gauteng); *Cucumis* spp. (Free State); *Ornithogalum saundersiae* (chink) (Western Cape); *Paspalum* staggers (Free State).

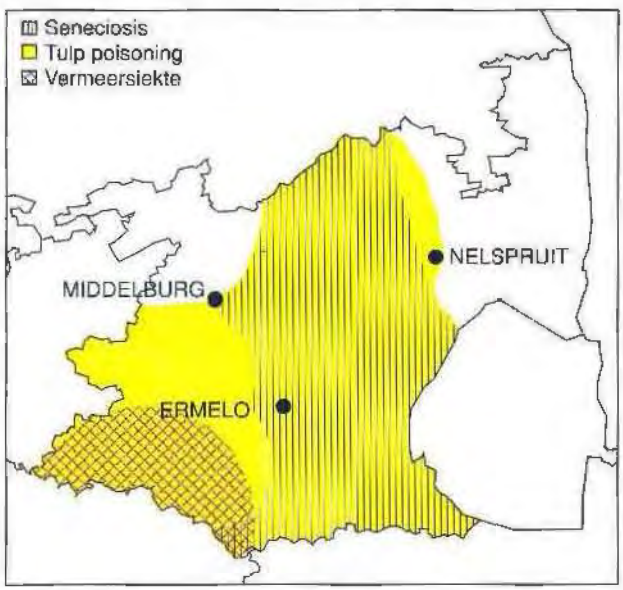

FIG. 13 Distribution of seneciosis, tulp poisoning and vermeersiekte in Mpumalanga

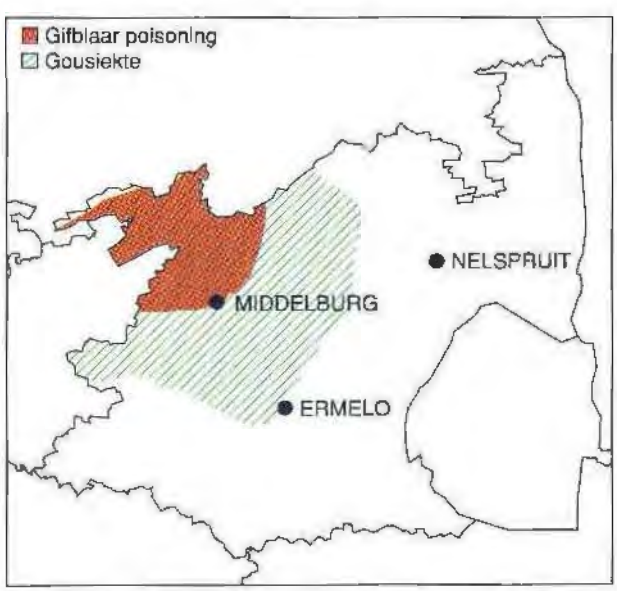

FIG. 14 Distribution of gousiekte and gifblaar poisoning in Mpumalanga

Gnidia burchellii (harpuisbos) poisoning is associated with sheep grazing during winter (particularly on the southern slopes of hills) in the south-eastern parts of the province. Affected animals die acutely or manifest dyspnoea, subcutaneous oedema and sometimes diarrhoea—in more protracted cases. A diagnosis is made on the grounds of the clinical signs and pathological changes such as subcutaneous emphysema, pulmonary oedema, focal necrosis and fibrosis of the myocardium, atrophy of lymphoid tissue and enteritis. Cattle are not known to be poisoned by harpuisbos. There is no specific treatment for the condition (Kellerman *et al.* 1988).

Geigeria aspera is responsible for localized, but often severe, outbreaks of vermeersiekte in the western and northern Free State and Mpumalanga, particularly in the Standerton and southern part of the Ermelo districts (see Vermeersiekte).

ECONOMIC IMPORTANCE

TABLE 13 Expected annual impact of mortalities from plant poisonings and mycotoxicoses on the livestock industry of Mpumalanga

Poisoning	Cattle	Sheep	Price (R)
Cardiac glycosides	2 486	2 648	4 272 276
Seneciosis	640	2 649	1 448 073
Diplodiosis	204	1 430	565 230
Gouslekte	301	530	554 340
Vermeersiekte		2 755	487 635
Gifblaar	301		460 530
Lantana	226		345 780
Dikoor		1 483	262 491
Gnidia		320	56 640
Other			5 540 694
Total			13 993 689

KwaZulu-Natal

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Tulp poisoning (Fig. 16) and seneciosis (Fig. 15) predominate in KwaZulu-Natal. The other plant poisonings and mycotoxicoses of the province, roughly in order of importance, are *Lantana camara* poisoning (Fig. 8), diplodiosis (Fig. 7), gousiekte (Fig. 6), bracken fern poisoning, stootsiekte, kikuyu poisoning, *Cestrum* poisoning, *Panicum* photosensitization (Fig. 2), cynanchosis (Fig. 15), vulvovaginitis (hyperoestrogenism of pigs), and *Aspergillus clavatus* poisoning.

The following poisonings are described under the sections or provinces listed in brackets: tulp poisoning (*Homeria pallida*, Poisoning with cardiac glycoside-containing plants), seneciosis (*Senecio latifolius*, Seneciosis), *Lantana camara* poisoning (*Lantana* poisoning), diplodiosis (Diplodiosis), gousiekte (*Pachystigma pygmaeum* and *P. thamnus*, Gousiekte), dikoor (Geeldikkop and *Panicum* photosensitization), cynanchosis (Eastern Cape), and *Aspergillus clavatus* tremors (Northern Province).

Considerable mortalities in cattle are sporadically caused by bracken fern (*Pteridium aquilinum*), especially in the hilly country of the mist belt. The poisoning also occurs in the more mountainous parts of the adjacent provinces of Mpumalanga and the Eastern Cape. Outbreaks are sporadic, and usually associated with a scarcity of food due to overstocking, drought or injudicious burning. Affected cattle manifest depression and anorexia, bloody mucous discharge from the nose, epistaxis, haemorrhages in the conjunctiva and vagina, melaena, prolonged clotting time, and fever. Haemorrhages and haematomas throughout the body and evidence of bone-marrow depression (including thrombocytopenia and leucocytopenia) are the main necropsy features. A diagnosis is made on the grounds of history, clinical signs and lesions. Symptomatic treatment with antibiotics (for secondary infection), and transfusions with citrated blood may be considered. There is no effective antidote (Tustin, Adelaar & Meldal-Johnson 1968; Kellerman *et al.* 1988).

Stootsiekte (pushing disease) is a neurotoxicosis of cattle, unique to Natal. It is caused by *Matricaria nigellifolia*, a soft-stemmed, water-loving plant with daisy-like flowers, which poisons stock mainly in the districts of Vryheid, Estcourt and Ixopo (Fig. 16) (Andrews 1923; Kellerman *et al.* 1988). The condition is marked by docility, apathy, clumsiness, aimless wandering and pushing against objects. A latent period of several weeks may precede the clinical signs, and the prognosis is poor. Perivascular gliosis and inflammatory cell infiltration in the white matter of the brain are the distinctive lesions of stootsiekte. A diagnosis is made on the strength of the presence of the plant, clinical signs and neuronal changes consistent with the disease (Newsholme, Kellerman & Welman 1984).

Kikuyu (*Pennisetum clandestinum*) poisoning is a condition of cattle, and is characterized by anorexia, depression, copious drooling, ruminal atony and distention, dehydration, sham drinking and incoordination. Outbreaks typically follow about 10 d after invasion of the grass by army worm, *Spodoptera exempta*. An apparently indistinguishable condition has been observed in sheep and cattle grazing on kikuyu pastures in the absence of the worms. In the latter outbreaks, lush growing, highly fertilized pastures that had previously been drought stricken, are thought to predispose to the disease. The most telling lesion in both types of poisoning is ulceration of the epithelium of the forestomachs, especially of the omasum. The aetiology of both diseases remains obscure (Bryson & Newsholme 1978; Newsholme, Kellerman, Van der Westhuizen & Soley 1983; Kellerman *et al.* 1988). Army worm can be controlled by pesticides registered for the purpose such as carbamates and certain pyrethroids. Note that the chemical eradication of army worm does not necessarily render pastures

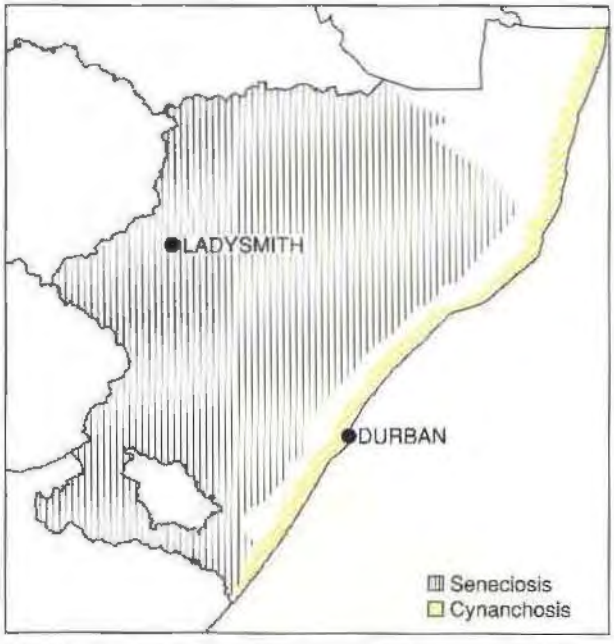

FIG. 15 The distribution of seneciosis and cynanchosis in KwaZulu-Natal

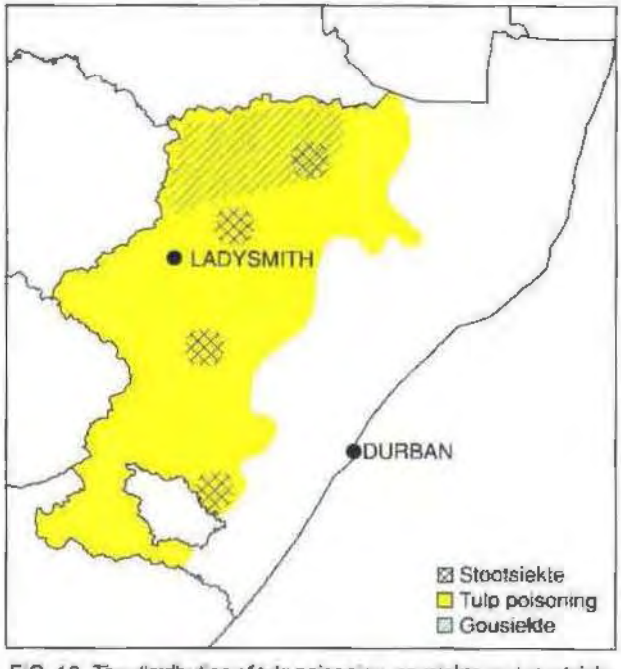

FIG. 16 The distribution of tulp poisoning, gousiekte and stootsiekte in KwaZulu-Natal

safe for grazing and that lightly infested pastures can be as dangerous as heavily infested ones. Cattle must be removed from toxic pastures as soon as the first signs appear, and the pastures are supposed to be rested for 40 d. However, if this period is too long, the toxicity of the pastures can be monitored by grazing a few "tracer" cattle on them for 96 h. Short-term grazing for about 1 h can be considered if other op-

tions are impractical. There is no antidote, and symptomatic treatment has generally been unsatisfactory (Bryson 1982; Kellerman *et al.* 1988).

Cestrum laevigatum poisoning of cattle (Chase Valley disease) is sporadically reported in KwaZulu-Natal and the adjoining provinces, into which the plant has encroached along river banks. The clinical signs of this poisoning include salivation, sunken eyes, arched back, colic, muscle tremor, incoordination, aggression, constipation and icterus. The principal microscopical lesions, situated in the liver, include centrilobular necrosis and haemorrhage, accompanied by infiltration of neutrophils in the necrotic areas. A diagnosis depends on the history and nature of the lesions (Thorburn 1934; Kellerman *et al.* 1988). Recently, parquin and carboxyparquin (compounds very similar in structure to atractyloside and carboxyatractyloside) have been isolated from *C. parqui* (Oelrichs, Pearce, Kudo & Kelly 1994).

Porcine oestrogenism associated with the mycotoxin, zearalenone (F-2) and its metabolite, zearalenol, occasionally occurs in animals fed on maize infected by *Fusarium graminearum*. Maize spoilt by moisture while being stored on the cob under conditions of temperature fluctuation in the lower ranges, is considered to be especially dangerous. Prepubertal gilts are often affected, showing clinical signs such as tumefaction of the vulva, enlargement of mammary glands, and prolapse of the vagina and rectum. Removal of the mouldy ration normally results in full recovery (Aucock, Marasas, Meyer & Chalmers 1980; Kellerman *et al.* 1988).

ECONOMIC IMPACT

TABLE 14 Expected annual impact of mortalities from plant poisonings and mycotoxicoses on the livestock industry of KwaZulu-Natal

Poisoning	Cattle	Sheep	Price (R)
Cardiac glycosides	2 362	2 648	4 082 556
Seneciosis	1 017	2 649	2 024 883
Lantana	452		691 560
Diplodiosis	120	847	333 519
Dikoor		742	131 334
Gousiekte	15	530	116 760
Bracken fern	30		45 900
Stootsiekte	20		30 600
Others			4 843 908
Total			12 301 020

Free State

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Tulp poisoning (Poisoning with cardiac glycoside-containing plants, Fig. 17), ovine hepatogenous photosensitization (Geeldikkop and *Panicum* photosensitivity, Fig. 18) and diplodiosis (Diplodiosis, Fig. 7) are the pre-eminent poisonings in this province. Other poisonings which play a role, are those caused by *Lasiospermum bipinnatum* (Eastern Cape, Fig. 19); *Gnidia burchellii* (Mpumalanga); *Cestrum laevigatum* (KwaZulu-Natal); *Geigeria aspera* (Vermeersiekte, Fig. 17); *G. polycephala* (Northern Cape), *Chrysocoma ciliata*, *Cucumis* spp.; valsiekte; kweek tremors; *Paspalum* staggers, and seneciosis (Seneciosis, Fig. 19). The various poisonings are described under the sections or provinces listed in brackets.

Outbreaks of hepatotoxicosis and photosensitization caused by *L. bipinnatum*, occur sporadically in the east of the province, from Bethlehem to about Zastron (Fig. 19); *G. burchellii* is of relatively minor importance in the north-east and *G. polycephala* in the karoid areas of the south; *C. laevigatum* is sometimes responsible for Chase Valley disease along the banks of the Vaal River, especially in the vicinity of Parys, while seneciosis causes relatively few losses in the broken country of the far north-east.

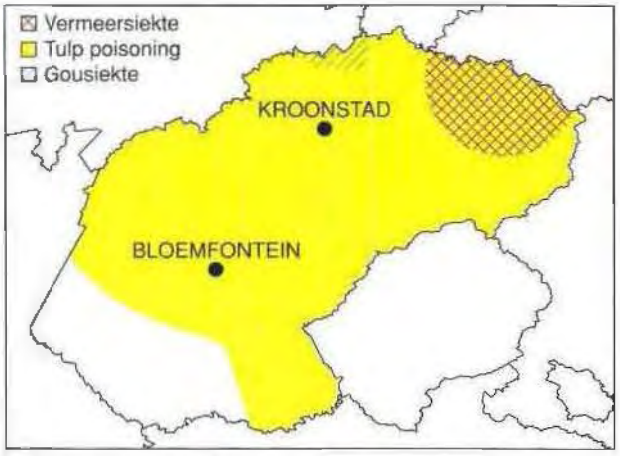

FIG. 17 Distribution of tulp poisoning, vermeersiekte and gousiekte in the Free State

Ewes that graze on *Chrysocoma ciliata* in karoid areas of the southern Free State, intermittently excrete a toxin in their colostrum or milk which induces alopecia in new-born lambs. Affected lambs die of exposure, pneumonia and hairballs of ingested wool. If ewes are kept off veld heavily infested by *Chrysocoma* for about a month prior to and after lambing, the incidence of kaalsiekte is much reduced. Severe diarrhoea has been reported in sheep and cattle that ingest large quantities of the plant. The condition, known as lakseersiekte, occurs on trampled veld during winter in times of drought, when stock graze almost exclusively on *C. ciliata*. Supplementary feeding is said to significantly reduce losses from this poisoning (Steyn 1931; Kellerman *et al.* 1988).

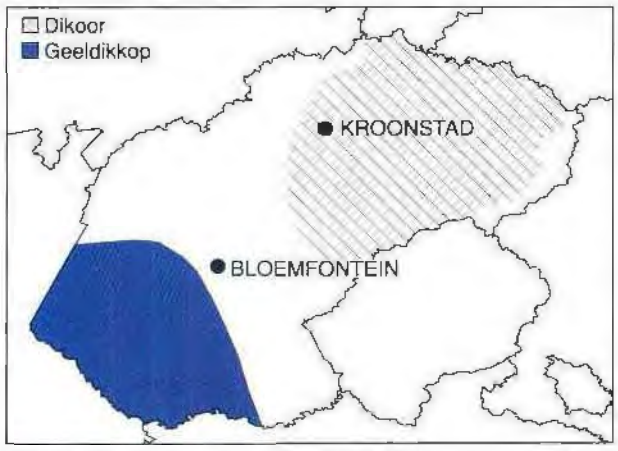

FIG. 18 Distribution of geeldikkop and *Panicum* photosensitization (dikoor) in the Free State

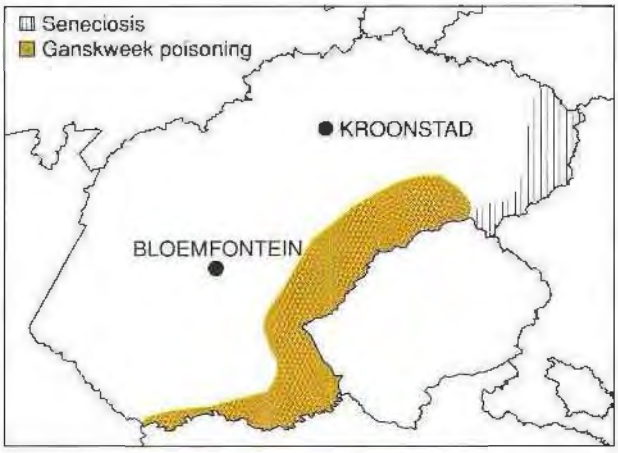

FIG. 19 Distribution of ganskweek poisoning and seneciosis in the Free State

Valsiekte, a disorder of Dorper and cross-breed lambs on *C. ciliata* veld in the districts of Middelburg, Bethulie and Jagersfontein, often occurs in conjunction with kaalsiekte. Lambs aged 2–4 months, drop out of the flock when driven, collapse on their sternums and struggle forward, sometimes on bent knees, dragging their hind-legs behind them. Death may set in suddenly or after a few weeks. Valsiekte resembles swayback in as much as a *status spongiosus* is present, especially in the dorso-lateral tracts of the spinal cord and, more rarely, in the medulla oblongata. A diagnosis is made on the grounds of locality, breed, clinical signs and histopathological lesions in the central nervous system. The liver copper levels are normal (Kellerman *et al.* 1988).

Wild cucumbers (*Cucumis* spp.), containing bitter principles (oxygenated tetracyclic triterpenes and their glycosides), periodically poison stock, especially on old maize lands in winter. Affected cattle and sheep die suddenly or develop diarrhoea and dyspnoea. A diagnosis is confirmed by the presence of large amounts of the characteristic boat-shaped, undigested seed in the gut and rumen (Steyn 1949; Enslin, Joubert & Rhem 1954; Kellerman *et al.* 1988).

A nervous condition of unknown aetiology, known as kweek tremors, sporadically appears in cattle and sheep grazing in winter on rested pastures with abundantly flowering *Cynodon dactylon*. The disease, lasting one day to several weeks, is characterized by tremors, ataxia (stiff-legged gait), paresis and paralysis; affected animals eat and drink normally and the prognosis is good. Outbreaks have been recorded at Tweespruit, Parys, Potchefstroom and Ventersdorp. An ergot or endophyte is suspected of being involved in its aetiology (Kellerman *et al.* 1988).

Paspalum staggers is found in cattle grazing on seeding *Paspalum dilatatum* infected with ergot (*Claviceps paspali*). This moisture-loving grass, found in vleis and wetlands, occurs most commonly in the north-eastern part of the province. The clinical signs, which are brought on or aggravated by exercise, are indistinguishable from those of kweek tremors. Like kweek tremors, the prognosis is excellent if stock are promptly removed from the toxic pastures. The diagnosis is based on circumstantial evidence such as nervous signs associated with ergot-infected *Paspalum* grass (Quinlan 1956; Ehret, Adelaar & Kriek 1968; Kellerman *et al.* 1988).

A condition almost identical to kweek tremors and *Paspalum* staggers is caused by ingestion of the grass, *Melica decumbens*, in the southern Free State (see Eastern Cape).

ECONOMIC IMPORTANCE

TABLE 15 Expected annual impact of mortalities from plant poisonings and mycotoxicoses on the livestock industry of the Frea State

Poisoning	Cattle	Sheep	Price (R)
Cardiac glycosides	2 734	2 648	4 651 716
Geeldikkop and dikoor		14 832	2 625 264
Diplodiosis	204	1 430	565 230
Gousiekte	15		22 950
Others			5 244 009
Total			13 109 168

Eastern Cape

DISTRIBUTION, DIAGNOSIS AND TREATMENT

The plant poisonings and mycotoxicoses of the province, more or less in descending order of importance, are seneciosis (Fig. 20), geeldikkop (Fig. 22), tulp poisoning (Fig. 21), krimpsiekte (Fig. 21), *Lantana camara* poisoning (Fig. 8), *Lasiospermum bipinnatum* or ganskweek poisoning (Fig. 20), cynanchosis (Fig. 22), *Thesium lineatum* poisoning (Fig. 21), kikuyu

poisoning, *Chrysocoma* poisoning, waterpens, slangkop poisoning, bietouw poisoning, facial eczema and *Melica decumbens* tremors.

The following conditions are described with the poisonings of the sections or provinces in brackets: geeldikkop (Geeldikkop and *Panicum* photosensitization); tulp poisoning (Poisoning with cardiac glycoside-containing plants); waterpens or *Galenia africana* poisoning (Western Cape); kikuyu poisoning (KwaZulu-Natal); bietouw or prussic acid poisoning (Northern Province); slangkop (*Urginea altissima*) poisoning (Poisoning with cardiac glycoside-containing plants); *Melica decumbens* or dronkgras tremors (Free State).

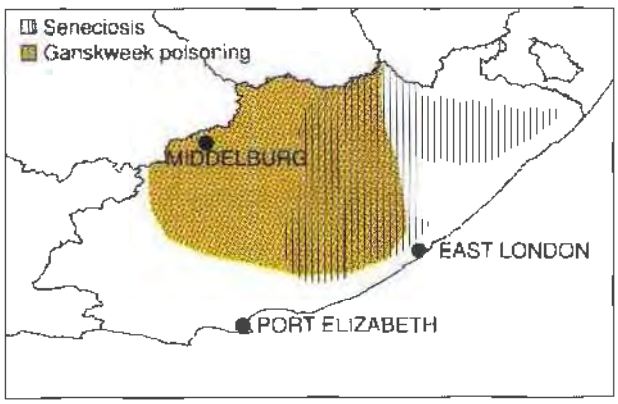

FIG. 20 Distribution of seneciosis and ganskweek poisoning in the Eastern Cape

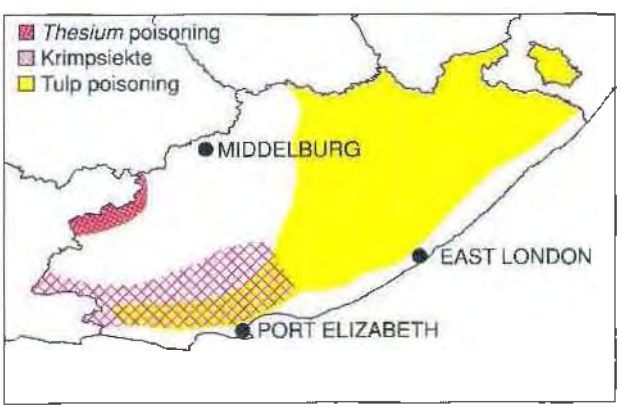

FIG 21 Distribution of krimpsiekte, tulp poisoning and *Thesium lineatum* poisoning in the Eastern Cape

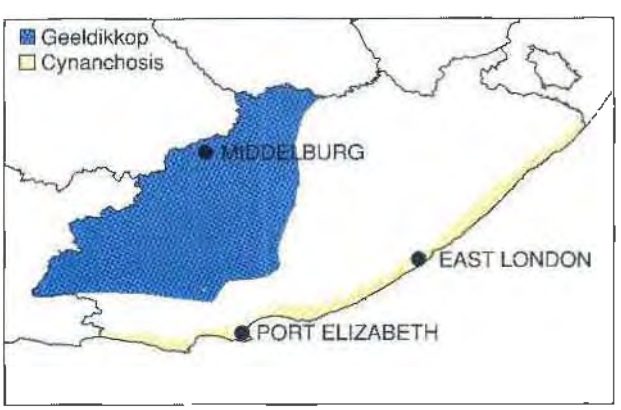

FIG. 22 Distribution of geeldikkop and cynanchosis in the Eastern Cape

Lasiospermum bipinnatum or ganskweek causes significant sheep and cattle losses in the Eastern Cape (Fig. 20), notably during winter (Kellerman *et al.* 1988). The plant contains hepatotoxic furanosequiterpenoids (L.A.P. Anderson, personal communication 1986) which also damage the lungs (Williams 1990). Affected animals show signs of liver involvement, such as icterus and occasional photosensitization; and, as in *Asaemia axillaris* (Northern Cape) and *Athanasia trifurcata* (Western Cape) poisoning, the hepatic lesions are zonally distributed in the lobules. Ganskweek poisoning can be distinguished from other hepatotoxicoses of the region, such as seneciosis and geeldikkop, on the histopathology of the liver (Kellerman, Basson, Naudé, Van Rensburg & Weiman 1973; Coetzer & Bergh 1983; Kellerman *et al.* 1988).

Cynanchum obtusifolium, *C. ellipticum*, and *C. africanum* are palatable, climbing plants causing heavy cattle, sheep, goat and horse losses along the coast, especially in the Eastern and Western Cape Provinces (Fig. 22). Affected animals manifest hypersensitivity, incoordination and recurrent tetanic seizures, followed by protracted paralysis often lasting a week or more. The carcases are bruised (trauma) and *rigor mortis* sets in almost immediately; otherwise there are no consistent lesions. A diagnosis is made on the grounds of clinical signs, the presence of grazed plants, and fragments of *Cynanchum* leaves in the rumen (Kellerman *et al.* 1988). Since the active principles of *Cynanchum* spp. are pregnane glycosides (Steyn, Van Heerden, Vleggaar, Erasmus & Anderson 1988), the clinical signs are indistinguishable from those induced by *Sarcostemma viminale* (Kellerman *et al.* 1988; Vleggaar, Van Heerden, Anderson & Erasmus 1993) in the North-West Province.

Thesium lineatum (witstorm) is a root parasite on a range of Karoo plants such as *Felicia* spp., *Chrysocoma ciliata*, *Pteronia sordida*, *Melianthus comosus* and *Lycium* spp. Outbreaks of witstorm poisoning of ruminants have been recorded in the south-western part of the province and adjacent Western Cape, including the districts of Middelburg, Murraysburg, Graaff-Reinet, Aberdeen and Beaufort West (Fig. 21, 26 and 28). Most deaths occur in winter on the southern slopes of hills, but losses can also be sustained in other seasons and topographical situations. Affected sheep, goats and cattle usually die suddenly, but longer-lived ones develop diarrhoea and dyspnoea. A bufadienolide has been isolated from the plant (Anderson, Joubert, Schultz, Kellerman & Pienaar 1987).

Facial eczema is a hepatogenous photosensitization of ruminants grazing on pastures contaminated by

conidia of *Pithomyces chartarum*. Under favourable weather conditions, numerous conidia, containing a potent hepatotoxic epipolythiadioxopiperazine (sporidesmin), are produced by the fungus growing saprophytically on pasture litter (Synge & White 1959). Wind and water carry the conidia from the litter onto the surrounding herbage, which is eaten by stock. If sufficient conidia-polluted leaves are ingested, facial eczema results (Mortimer, Di Menna & White 1978).

Although the fungus is widely distributed in South Africa, minor, sporadic outbreaks of facial eczema have been recorded only in sheep grazing on perennial ryegrass pastures near Humansdorp and in the winter rainfall area of the Western Cape, in cattle on perennial ryegrass at a sewage farm in Gauteng, and in sheep on oat pastures in the Free State. Gross lesions include signs of photosensitization, hepatosis, icterus, nephrosis and erosions in the gall and urinary bladders. The liver is usually mottled, with yellowish, sunken areas extending into the parenchyma while, in more chronic cases, the organ may be distorted by fibrosis and nodular regeneration. The most important lesions are located in the biliary system, where moderate to severe portal fibroplasia and bile-duct proliferation are accompanied by necrosis and pronounced thickening of the bile ducts by concentric periductal fibrosis. In facial eczema, the photodynamic agent, phylloerythrin, is retained as a result of occlusion of the bile ducts by granulation tissue, periductal fibrosis, necrotic debris and inspissated bile. The latent period of *c.* 9–24 d between ingestion of sporidesmin and the manifestation of photosensitivity, is the time required for the occlusive changes to take place. A diagnosis of facial eczema is made on the grounds of the clinical signs, the nature of the hepatic lesions and elevated spore counts of *P. chartarum* on the pastures. Abroad, the disease is controlled by monitoring the build-up of *P. chartarum* in paddocks, applying fungicides to pastures, and by the prophylactic treatment of stock with zinc or cobalt (Marasas, Adelaar, Kellerman, Minne, Van Rensburg & Burroughs 1972; Kellerman *et al.* 1980; Coetzer *et al.* 1983; Kellerman *et al.* 1988).

ECONOMIC IMPORTANCE

TABLE 16 Expected annual impact of mortalities from plant poisonings and mycotoxicoses on the livestock industry of the Eastern Cape

Poisoning	Cattle	Sheep	Price (R)
Seneciosis	2 072	7 945	4 576 425
Geeldikkop and dikoor		19 281	3 412 737
Cardiac glycosides	1 492	3 973	2 985 981
Lantana	56		85 680
Ganskweek	50		76 500
Others			7 296 813
Total			18 434 136

North-West Province

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Poisoning by cardiac-glycoside-containing plants (*Homeria pallida, Moraea polystachya, Urginea sanguinea*) (Fig. 24), and gousiekte (Fig. 23) are the two major phytotoxicoses of the province. The other poisonings, in approximate order of importance, are vermeersiekte (Fig. 23), gifblaar poisoning (Fig. 23), diplodiosis (Fig. 7), *Sarcostemma viminale* poisoning, fumonisin B_1 poisoning (LEM or leukoencephalomalacia of Equidae), and poisoning with bushveld chink (*Ornithogalum prasinum*).

Poisoning with the following plants is more fully described under the sections or provinces given in brackets: tulp, slangkop (Poisoning with cardiac glycoside-containing plants), vermeersiekte (Vermeersiekte), diplodiosis (Diplodiosis), *Fusarium moniliforme* (LEM, Gauteng), and bushveld chink (Western Cape).

Vermeersiekte in this province (Fig. 23) differs from that seen elsewhere, in that cattle are more often affected here. Cattle usually contract the paralytic form and seldom regurgitate.

Sarcostemma viminale is a succulent climber (or free-standing shrub in karoid areas), sometimes eaten by ruminants in times of scarcity or when the supporting trees are felled. Poisoning with this plant is believed to be more common in the North-West than in other provinces where it occurs. Since the neurotoxic principle of *S. viminale* is a pregnane glycoside, the clinical signs produced by it are indistinguishable from those of *Cynanchum ellipticum* poisoning (see Eastern Cape). A diagnosis is made on the grounds of the history, clinical signs, presence of grazed plants, and fragments of the plant in the rumen (Kellerman *et al.* 1988; Vleggaar *et al.* 1993).

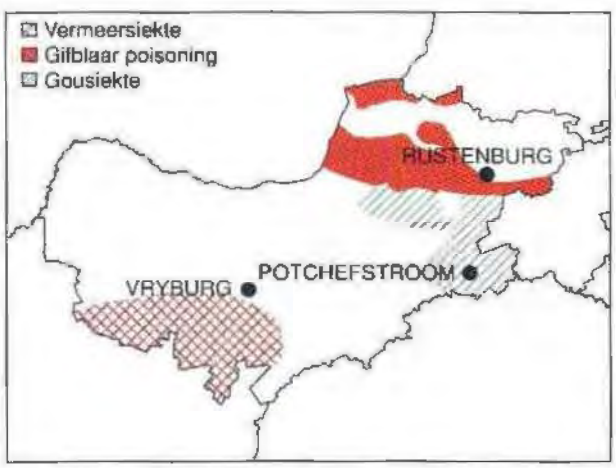

FIG. 23 Distribution of gifblaar poisoning, gousiekte and vermeersiekte in the North-West Province

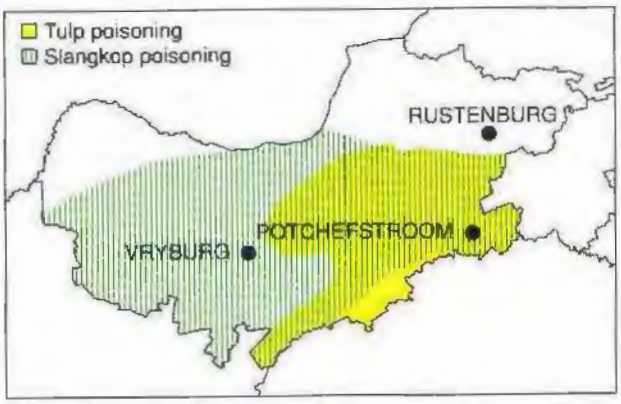

FIG. 24 Distribution of tulp and slangkop poisoning in the North-West Province

ECONOMIC IMPORTANCE

TABLE 17 Expected annual impact of mortalities from plant poisonings and mycotoxicoses on the livestock industry of the North-West Province

Poisoning	Cattle	Sheep	Price (R)
Cardiac glycosides	1 491		2 261 230
Gousiekte	603	3 442	1 531 824
Vermeersiekte		3 443	609 411
Gifblaar	301		460 530
Diplodiosis	75	530	208 560
Others			3 394 821
Total			8 486 376

Northern Cape

DISTRIBUTION, DIAGNOSIS AND TREATMENT

Vermeersiekte (Fig. 25) and geeldikkop (Fig. 25) are the predominant poisonings in this province. The other poisonings, in order of importance, are krimpsiekte (Fig. 26), enzootic icterus (Fig. 27) and poisoning with *Dipcadi glaucum* (malkopui), *Urginea sanguinea*, *U. physodes* (slangkop), *Thesium lineatum* (Fig. 26), *Asaemia axiliaris* (vuursiektebossie), *Hertia pallens* (springbokbos) (Fig. 27), *Pteronia pallens* (Scholtzbossie) (Fig. 27), *Galenia africana* and *Gnidia polycephala*.

The following poisonings are described in the sections or provinces listed in brackets: krimpsiekte and slangkop poisoning (Poisoning with cardiac-glycoside-containing plants), vermeersiekte (*Geigeria* poisoning) (Vermeersiekte), waterpens and *P. pallens* poisoning (Western Cape).

Enzootic icterus or geelsiekte is a chronic copper poisoning of small stock arising from prolonged ingestion of low levels of phytogenous copper on doleritic soils in the Karoo. The epidemiology of this complex disease has not yet been fully elucidated. Since it is a chronic copper poisoning, enzootic icterus can be precipitated by various stressors such as dipping, shearing, and transportation. Old animals are most often affected. Accumulation of copper in the liver is prevented by dosing zinc or molybdenum salts to animals, or providing licks containing these compounds. The distribution of enzootic icterus coincides partially with that of geeldikkop, being bounded in the south by a line of doleritic intrusion almost bisecting the Great Karoo, and in the north by the Kalahari sands (Fig. 27) (Bath 1979; G.F. Bath, personal communication 1995).

Dipcadi glaucum or malkopui is common in the north of the province and the western parts of the North-West and Northern Provinces. This bulbous plant,

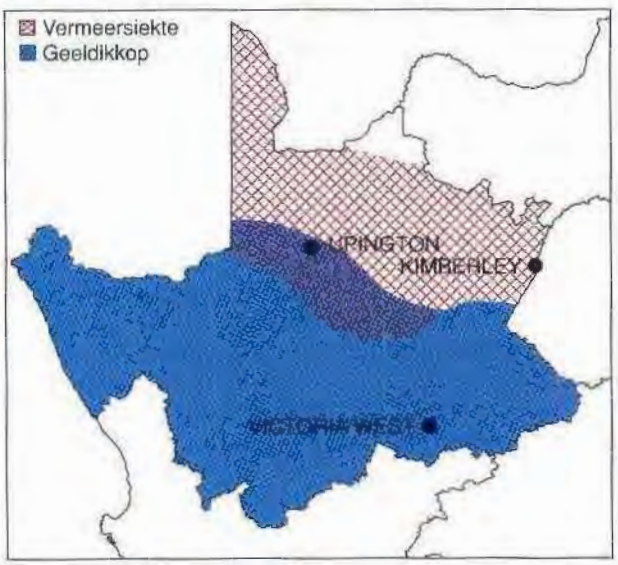

FIG. 25 Distribution of geeldikkop and vermeersiekte in the Northern Cape

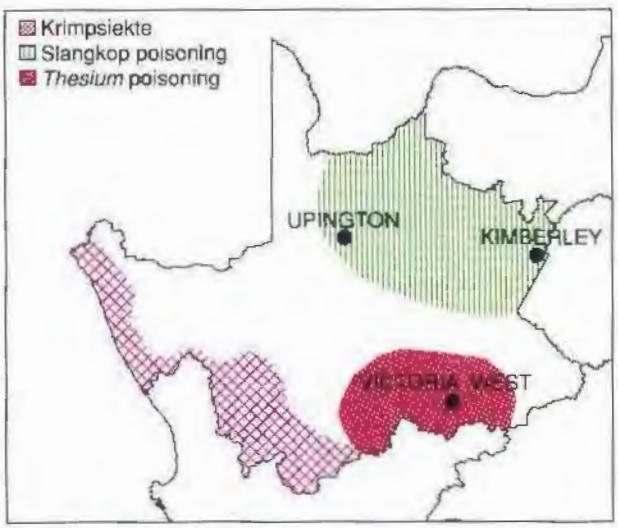

FIG. 26 Distribution of krimpsiekte, slangkop and *Thesium lineatum* poisoning in the Northern Cape

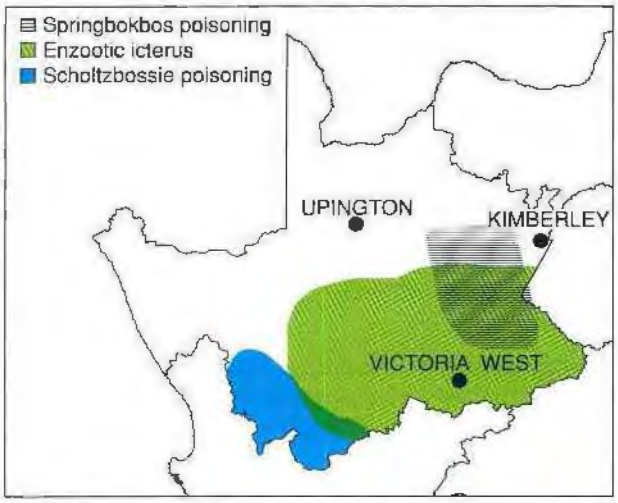

FIG. 27 Distribution of enzootic icterus, springbokbos and Scholtzbossie poisoning in the Northern Cape

which grows rapidly, is particularly troublesome in camps denuded by droughts. Immediately after spring rains, the leaves appear above the ground and, within about a week, the plants can reach a height of *c.* 150 mm. *D. glaucum* poisoning is more common in sheep than in cattle or goats. In sheep, the clinical signs include aimless wandering, pushing against objects, diarrhoea and abortions. Cattle walk about "drunkenly", assume a peculiar rump-down stance with fetlocks in flexion, and lie down with the legs awkwardly disposed. A diagnosis is made on the grounds of circumstantial evidence. There is no known treatment (Steyn 1949; Kellerman *et al.* 1988).

In the north and east of the province, slangkop poisoning is caused mostly by *Urginea sanguinea* (Fig. 1 and 26), and in the south and west by *U. physodes* and the closely related *U. pusilla.* The clinical signs, diagnosis and treatment are similar to those described for tulp poisoning (see Cardiac glycoside poisoning). Cattle suffering from slangkop poisoning are particularly prone to gassy bloat, which must be relieved by trochar (Kellerman *et al.* 1988).

Asaemia axillaris (vuursiektebossie) causes sporadic, localized outbreaks of ovine hepatogenous photosensitivity in the more westerly parts of the province. Although a typical Karoo bush, the vuursiektebossie always grows near water, e.g. in the vicinity of dams or pans. *A. axillaris* is believed to contain furanosesquiterpenoids, therefore the clinical signs and lesions closely resemble those induced by *Lasiospermum bipinnatum* (see Eastern Cape) (Steyn 1949; Kellerman *et al.* 1973).

Hertia pallens is a common pioneer species on denuded soils of the western Free State and the upper Northern Cape Province. Sheep poisoned by this plant (Fig. 27) develop signs such as apathy, anorexia, ruminal stasis, tympany, dyspnoea and cyanosis. Affected sheep often die peracutely, apparently from asphyxia. There is no known treatment (Prozesky, Kellerman, Jordaan & Welman 1986).

Gnidia polycephala poisoning is very similar to that induced by *G. burchellii* (see Mpumalanga).

ECONOMIC IMPORTANCE

TABLE 18 Expected annual impact of mortalities from plant poisonings and mycotoxicoses on the livestock industry of the Northern Cape

Poisoning	Cattle	Sheep	Price (R)
Vermeersiekte		28 233	4 997 241
Geeldikkop		25 955	4 594 035
Cardiac glycosides	994	7 946	2 927 262
Enzootic icterus		2 500	442 500
Others			7 903 761
Total			20 864 799

Western Cape

DISTRIBUTION, DIAGNOSIS AND TREATMENT

The plant poisonings and mycotoxicoses of this province, roughly in order of importance, are geeldikkop (Fig. 29), krimpsiekte (Fig. 28), chink (*Ornithogalum* spp.) poisoning (Fig. 29), waterpens (Fig. 30), tulp (*Homeria miniata*) poisoning (Fig. 28), cynanchosis (Fig. 29), annual ryegrass toxicity and ergotism (Fig. 29), Stellenbosch photosensitivity and *Athanasia trifurcata* poisoning, *Pteronia pallens* poisoning (Fig. 30), *Tylecodon grandiflorum* poisoning (Fig. 28), prussic-acid poisoning, lupinosis, *Phalaris* staggers, *Asaemia axillaris* poisoning, *Trachyandra divaricata* poisoning and facial eczema.

The following poisonings are described under the sections or provinces given in brackets: krimpsiekte (Poisoning with cardiac glycoside-containing plants), geeldikkop (Geeldikkop and *Panicum* photosensitization), cynanchosis (Eastern Cape), prussic acid poisoning (Northern Province), *Asaemia axillaris* poisoning (Northern Cape) and facial eczema (Eastern Cape).

Galenia africana poisoning or waterpens (Fig. 30) is a liver disorder, usually of ewes in poor condition, forced to eat the plant during droughts. The most prominent clinical signs are severe abdominal distension and loss of mass. Habitus and appetite remain fairly normal throughout the course of the disease (sometimes lasting for weeks) and only in the terminal stage do the animals become apathetic and recumbent. At necropsy, severe ascites is evident and the liver is usually discoloured (often greyish-blue) and distorted by nodular hyperplasia and atrophy/hypertrophy of

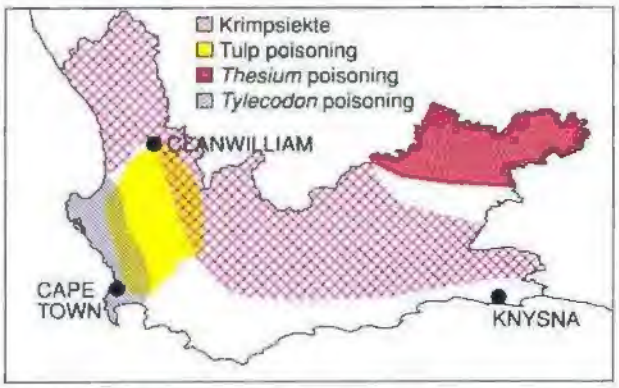

FIG. 28 Distribution of krimpsiekte, *Thesium lineatum* poisoning, *Tylecodon grandiflorus* poisoning and tulp poisoning in the Western Cape

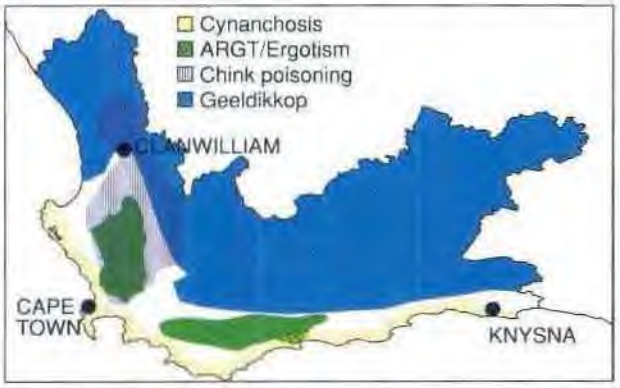

FIG. 29 Distribution of geeldikkop, chink poisoning, cynanchosis, annual ryegrass toxicity (ARGT) and ergotism in the Western Cape

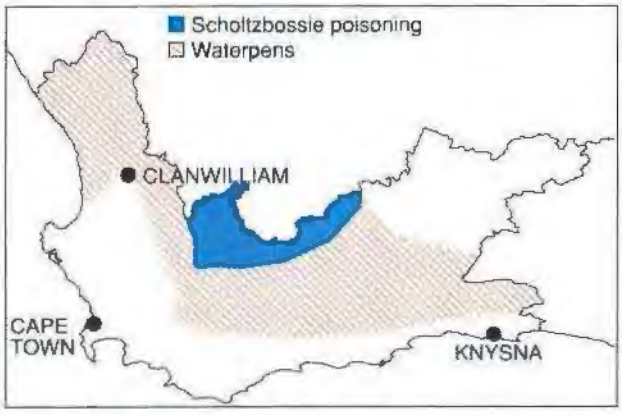

FIG. 30 Distribution of waterpens and Scholtzbossie poisoning in the Western Cape

certain parts. Histopathological examination reveals conspicuous centrilobular fibrosis bridging contiguous lobules, and duplication of the central veins Myocardial lesions have been reported in waterpens but, according to experimental evidence, the heart is affected only in the terminal stage, and the hepatic changes do not result from congestive heart failure (Kellerman *et al.* 1988; Van der Lugt, Schultz, Fourie, Hon, Jordaan & Labuschagne 1992).

Ornithogalum spp. are among the most poisonous plants in South Africa, the dried material having toxicities measured in mg/kg rather than g/kg live mass. *Ornithogalum thyrsoides* and *O. conicum* are most often incriminated in poisoning of stock in the winter rainfall area (Fig. 29), while in the summer rainfall areas *O. saundersiae*, *O. ornithogaloides* (vlei chink) and *O. prasinum* (bushveld chink) are the most important. *Ornithogalum thyrsoides* and *O. saundersiae*, widely used as ornamental plants, cause losses wherever stock have access to garden waste. Since chinks do not lose their toxicity during desiccation, stock are sometimes poisoned by contaminated hay. Chink poisoning is characterized by anorexia, apathy, and persistent watery diarrhoea lasting up to 3 weeks. In addition to the usual clinical signs, cattle may become temporarily or permanently blind (amaurosis). Treatment is symptomatic and activated charcoal gives variable results. A diagnosis is made on the grounds of circumstantial evidence (Quin 1927; Steyn 1949; Kellerman *et al.* 1988).

Annual ryegrass toxicity (ARGT) (Fig. 29) is a nervous disorder of cattle and sheep. It is caused by ingestion of annual ryegrass (*Lolium* hybrids) seed heads containing nematode (*Anguina* sp.) galls infected by a toxin-producing bacterium (*Clavibacter* sp.) (Lanigan, Payne & Frahn 1976). The condition is marked by excitability, muscular twitching, locomotory disturbance and convulsions. Mortality is high and death can ensue within *c.* 4–12 h. The lesions, which are non-specific, include rapid *rigor mortis*, oedema of the lungs and endo- and epicardial haemorrhages. Appropriate nervous signs and the presence of bacterial galls on the seed heads point to ARGT, and positive feeding trials with galls and mice confirm the diagnosis. ARGT is controlled by reducing annual ryegrass infestation of pastures by preventing either the grass from seeding (by mowing or tactical grazing) or by keeping stock off seeded pastures, etc. Affected stock are treated with magnesium sulphate (Schneider 1981).

Ergotism of cattle, induced by *Claviceps purpurea*, assumes two forms, viz gangrenous necrosis of the extremities and hyperthermia. A suspected outbreak of classical ergotism, involving gangrene of the extremities of cattle, has been recorded in the Bredasdorp district. More recently, widespread hyperthermia was manifested during summer and autumn by cattle in the Western Cape (Fig. 29). Affected animals showed symptoms such as elevated temperatures and rapid breathing (often with the mouth open and tongue protruding), accompanied by excessive salivation and a drop in milk yield; they also sought shade and

frequented cool, moist places. Hot weather and exercise aggravated the condition. The outbreaks have been linked with the feeding of barley "screenings" contaminated with ergotized annual rye grass seeds. The fungus, *Claviceps purpurea* (an obligate parasite of grains and grasses), replaces the seeds of the host plant with toxic sclerotia or ergots. These ergots are dark-grey or black, horny structures, usually somewhat larger than the grass seeds. So far, only annual ryegrass (*Lolium rigidum* hybrids), which occurs in this area as a weed, has been incriminated in ergotism in South Africa. Like ARGT, ergotism has been recorded only in the winter rainfall area (D.J. Schneider, personal communication 1995). For control, see ARGT.

It should be noted that syndromes indistinguishable from both types of ergotism have been described abroad in cattle grazing on fescue grass infested by the endophytic fungus *Acremonium coenophialum*.

Stellenbosch photosensitivity is an ovine hepatogenous photosensitization of unknown aetiology encountered in the winter rainfall area, where certain farms are affected almost annually. Sheep, especially lambs, may become photosensitive within 48 h of being introduced onto a toxic pasture. The pastures vary widely in nature and no common factor between the outbreaks has been identified, that might point to a causal agent. The histopathological lesions vary from individual cell necrosis to haphazardly scattered foci of hepatocellular necrosis to diffuse hepatic necrosis or fatty degeneration of the parenchyma. A diagnosis is made on the grounds of location, clinical signs and lesions (Kellerman *et al.* 1988).

Athanasia trifurcata (Kellerman, Coetzer, Schneider & Welman 1983) and *Pteronia pallens* are both hepatotoxic plants, but only the former causes photosensitization. *A. trifurcata*, or Klaaslouwbos, grows near the coast and is believed to contain furanosesquiterpenoids. The clinical signs and lesions of *A. trifurcata* poisoning are, therefore, similar to those of *Lasiospermum bipinnatum* poisoning (Eastern Cape).

Pteronia pallens or Scholtzbossie, on the other hand, frequents the karoid northern part of the province (Fig. 30), where it affects especially naive sheep introduced from non-Scholtzbossie areas. Animals die peracutely or show apathy, anorexia, ruminal stasis and icterus. The liver is enlarged and this is usually accompanied by ascites, hydrothorax, lung oedema and nephrosis. The most constant histopathological feature is centrilobular coagulative to lytic necrosis and haemorrhage in the liver. Farmers try to avoid poisoning by first feeding naive animals before exposing them to Scholtzbossie-infested veld for short periods. A diagnosis is made on the evidence of grazed Scholtzbossie on affected pastures, clinical signs and lesions (Prozesky, Kellerman & Welman 1986; Kellerman *et al.* 1988; Kellerman, unpublished data 1994).

Phalaris staggers is a progressive neurological disorder of sheep and cattle grazing on *Phalaris minor* in the winter rainfall area. *P. minor*, an exotic, cultivated grass, is a troublesome weed in the Swartland on disturbed areas such as reaped lands and the verges of roads (D.J. Schneider, personal communication 1984). The active principles are believed to be tryptamine alkaloids structurally similar to serotonin. Three distinct conditions are produced: a sudden death or cardiac syndrome; acute *Phalaris* poisoning with transient nervous signs; chronic *Phalaris* staggers, a fatal neurological disorder that develops in sheep 2–3 weeks after they have been introduced onto an infested pasture. The clinical signs of chronic poisoning include incoordination, a stiff-legged gait, tremors, convulsions and nystagmus. Signs may last for weeks. The principal histopathological feature is yellowish-brown pigmentation in the cytoplasm of affected neurons. A diagnosis is made on the grounds of locality, the nature of the pasture, clinical signs and histopathological lesions (Hartley 1970; Blood, Henderson & Radostits 1979; Kellerman *et al.* 1988).

Another condition, restricted to the Swartland, is lupinosis, a hepatotoxicosis of sheep and cattle grazing dry, sweet lupins infected by *Phomopsis leptostromiformis*. This fungus is a pathogen which continues to grow saprophytically on senescent or dead lupin plants. The clinical signs are typical of hepatoses, viz apathy, anorexia, icterus, ruminal stasis, constipation and, rarely, photosensitization. The liver, in acute poisoning, is enlarged and greyish-yellow; and, in chronic poisoning, markedly atrophic and fibrotic, with nodular hyperplasia giving rise to so-called "boxing glove" liver. The histopathological lesions in acute poisoning are marked by fatty degeneration of hepatocytes, megalocytosis, anisonucleosis, multinucleation and other nuclear changes (Van Warmelo, Marasas, Adelaar, Kellerman, Van Rensburg & Minne 1970). The active principles (phomopsins) have been identified as linear hexapeptides with ether-bridged macrocyclic rings (Mackay, Van Donkelaar & Culvenor 1986). A diagnosis is made on the grounds of locality, the grazing of *Phomopsis*-infected lupins, clinical signs and the nature of the lesions. Resistant lupin strains offer the best hope for controlling the disease (Kellerman *et al.* 1988).

Trachyandra divaricata is palatable in the early growing season on overgrazed veld in the south-west of the province. The clinical signs of poisoning with this plant are progressive, ascending paresis and paralysis, and the outstanding lesion is yellowish-brown granular pigment in the cytoplasm of neurons in the brain and spinal cord. A diagnosis depends on the clinical signs, presence of the plant and the nature of the neuronal damage. Cattle, horses and sheep are primarily affected (Newsholme, Schneider & Reid 1985; Kellerman *et al.* 1988).

ECONOMIC IMPORTANCE

TABLE 19 Expected annual impact of mortalities from plant poisonings and mycotoxicoses on the livestock industry of the Western Cape

Poisoning	Cattle	Sheep	Price (R)
Geeldikkop		11 865	2 100 105
Cardiac glycosides	497	6 622	1 932 504
Chinks	20	200	66 000
Waterpens		300	53 100
Others			2 568 855
Total			6 720 564

DISCUSSION

The maps were compiled to orientate veterinarians and pasture scientists as to the distribution of plant poisonings in South Africa, as an aid in the estimation of the economic impact of these poisonings, and for purposes of risk management.

Knowledge of the distribution of plant poisonings is particularly important when stock are translocated to new environments. This is necessary because animals learn to avoid some of the poisonous plants in the areas where they grow up. As a result of this acquired aversion, poisoning usually occurs only in naive animals, newly introduced from non-infested veld. A stockman wishing to move cattle from say the bushveld to the Highveld (Fig.1), should, therefore, be mindful of the dangers of tulp poisoning on their arrival.

The maps were drawn in consultation with veterinarians throughout South Africa, and the distribution of the causal plants (Kellerman *et al.* 1988) and veld types (Acocks 1975) were taken into account. The maps were intended to denote the areas of highest prevalence of a poisoning. A specific poisoning may, therefore, from time to time appear outside its designated area, e.g. slangkop poisoning sporadically occurs in the Northern Province, away from its denoted distribution stretching from Gauteng to the Northern Cape (Fig. 1). Geeldikkop (Fig. 2), similarly, has been diagnosed far to the north of the Karoo at Hoopstad in the Free State where sheep were forced by drought to eat *Tribulus terrestris*.

A glance at the maps will show that, for many reasons, the distribution of plant poisonings rarely coincides precisely with those of the causal plants (Kellerman *et al.* 1988). *Solanum kwebense*, for instance, is widely distributed in the Northern Province, Zimbabwe, Botswana and Namibia (Kellerman *et al.*, 1988), yet maldronksiekte has been recorded in only a small area at the confluence of the Crocodile and Marico Rivers (Fig. 10). In this area, overgrazing is believed to have resulted in permanent damage to the ecology. During dry years and when the veld is overgrazed by cattle or game, the palatable *Panicum maximum* grass, growing predominantly in the shade of *Acacia erubescens* trees, is replaced by *S. kwebense*. Once *S. kwebense* becomes established, no grass will grow in its vicinity, with the result that cattle are forced to feed on it. Poisoning with gifblaar is one of the rare exceptions that almost exactly follows the distribution of the causal plant (Fig. 5).

It must always be borne in mind that the distribution of plant poisonings constantly fluctuate, expanding in years that favour them and shrinking in others.

No data are available on the prevalence of plant poisoning in South Africa, and in this respect South Africa is no different from most other countries, including the United States of America (Nielsen & James 1992). The reasons for this dearth of data are various:

- Since plant poisonings are not notifiable diseases, outbreaks are not reported to the authorities.
- Farmers are generally aware of the plant poisonings in their region and tend to stoically accept the losses induced by them as normal natural hazards, like drought.
- Stock owners are sometimes reluctant to report plant poisonings out of fear that the disclosure will reduce the value of their land. The latter problem is not normally experienced with infectious diseases.

The estimations of losses induced by plant poisonings in South Africa are very conservative. According to our calculations, only one out of 300 cattle and one out of 133 small stock die of plant poisoning in this country each year. We have postulated that the normal mortality of stock in South Africa (from all causes including drought, infectious diseases, parasites, etc.) is 3% for cattle and 5% for sheep. In the case of cattle, 10% of the annual death toll was attributed to plant poisonings and, in small stock, 15 %. This is comparable with the estimated annual death toll in 17 western states of the USA of 3% for cattle and 8–10% for sheep. In both cattle and sheep, 33 % of all mortalities in this region were attributed to plant poisonings (Nielsen & James 1992). To allow for periodic massive outbreaks of plant poisonings such as geeldikkop and vermeersiekte (which, respectively, can account for half a million and a million small stock in a single season) the estimated annual losses must be looked upon as the mean over an imaginary 25-year period.

Our estimations of the economic impact of plant poisonings did not take into account production losses such as decrease in mass, ill-thrift, loss of wool; reproductive losses such as poor conception, stillbirths, abortions, birth defects, protraction of gestation periods, lengthening of calving intervals and birth defects (Nielsen & James 1992). The figures, moreover,

do not include the costs of temporary or permanent non-utilization of toxic pastures, impediment to the translocation of stock, interference with pasture management, and the reduced price of infested land. The real cost of plant poisonings to the livestock industry of South Africa probably cannot be computed.

For a more realistic estimate, a country-wide statistical survey of the plant poisonings, spanning a number of years and backed by good diagnostic support, will have to be carried out. Before this can be done, however, the question has to be asked whether the cost of such a survey, in terms of man power and money, is justifiable in a developing country.

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