Vermeersiekte caused by *Geigeria burkei* Harv. subsp. *burkei* var. *hirtella* Merxm. in the Northern Province of South Africa

C J Botha^a, T A Gous^b, Mary-Louise Penrith^c, T W Naudé^{a,d}, Leonie Labuschagne^d and Elizabeth Retief^e

ABSTRACT

The 1st field outbreak of vermeersiekte induced by *Geigeria burkei* Harv. subsp. *burkei* var. *hirtella* Merxm. is reported. It is also the first recorded outbreak of this disease in the arid sweet bushveld of the Northern Province of South Africa. The toxicosis was experimentally reproduced in a sheep following daily intraruminal administration of 2.5–5.0 g/kg dried, milled plant material for 18 consecutive days. Neither the sheep in the field outbreak nor the ewe in the experiment exhibited any signs of regurgitation of rumen contents (vermeersiekte). All developed only the stiff or paretic/paralytic forms of the disease. Serum activities of CK and GGT were slightly raised in clinically affected sheep (n = 11) during the field outbreak, and serum activities of AST, GLDH, GGT, LDH and CK increased in the ewe dosed with the plant material. Analysis of dried, milled *Geigeria* plant material confirms that this species is moderately nutritious.

Key words: Geigeria burkei, paralysis, paresis, sheep, stiffness, vermeersiekte.

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INTRODUCTION

Vermeersiekte following ingestion of different Geigeria species is a major intoxication of small stock in South Africa. Geigeria ornativa is the most important cause of vermeersiekte in the dry Griqualand West area of the Northern Cape^{11,15}. Vahrmeijer¹³ reported that during 1929-1930 as many as a million sheep died in Griqualand West. Kellerman et al.¹¹ calculated the economic impact of plant poisonings and estimated that in 1996 monetary terms the annual loss due to vermeersiekte mortalities exceeds 6 million Rand. The economic loss could be considerably higher, as vermeersiekte is known to be an erosive disease that causes various production and reproduction losses⁶. *G. aspera* is associated with more localised poisoning on the highveld of Mpumalanga and the northern Free State^{10,11}. Under experimental conditions *G. burkei* subsp. *burkei* var. *zeyheri* induced vermeersiekte, although there is no confirmed report of any natural field outbreak¹⁰. Various α , β -unsaturated-8-sesquiterpene lactones have been isolated from different *Geigeria* species and are implicated as the toxic principles^{2,10,11}.

The toxicity of the 3 species mentioned varies. It has been estimated that G. burkei subsp. *burkei* var. *zeyheri* is 3 times and *G*. aspera 10 times more toxic than G. ornativa^{6,10}. Sheep in feeding experiments with G. ornativa hay ingested the plant for prolonged periods and clinical signs were noticed only after 3 weeks6. Clinically, 4 forms of the disease are recognised, namely regurgitation ('vomition'), stiffness, bloat and paresis/paralysis¹⁰. Regurgitation is not a common finding, and sheep usually exhibit stiffness, particularly of the hindquarters, in the early stages of the toxicosis^{10,15}. Macroscopical lesions are usually absent, but a megaoesophagus may be encountered in some cases and sheep may die from a foreign body pneumonia if rumen fluid is aspirated^{10,11}. Van der Lugt and Van Heerden¹⁴ reported hypertrophy and vacuolation of myofibres of the skeletal system, diaphragm and oesophagus, as well as foci of myocardial degeneration and necrosis.

Vermeerbos (the common name for plants of the genus *Geigeria*) is reported to be moderately nutritious¹⁵. Grosskopf⁶ stated that vermeerbos, in limited quantities, appears to be an excellent feed for sheep, as it retains its protein concentration during winter, whereas that of grasses rapidly decreases after the growing season.

FIELD OUTBREAK

During the end of winter (August 1996) approximately 50 sheep in a flock of 200 Dorpers on the farm Drielingbosch (23° 17′ S, 29° 41′ E), near Bandolierkop in the Soutpansberg district of the Northern Province, exhibited listlessness, stiffness in the hindquarters, lameness and lagging behind the rest of the flock. A decline in their condition was also noticed by the farmer and some animals became paretic/paralytic. Some sheep were unable to rise and remained in sternal or lateral recumbency. When sheep were assisted to stand the limbs trembled and a staggering gait was observed. Two weeks before the investigation the farmer instituted supplementary feeding consisting of a salt and flowers-of-sulphur lick, maize stover and poultry litter. The farmer incriminated a small, green, herbaceous bush as the cause of the paretic condition. On inspection of the camp and observation of the animals it was noticed that the sheep eagerly ingested the incriminated herb (Fig. 1), which was the only greenery available at the time. The plant was recognised as a Geigeria species and was collected and submitted for identification. The presence of Helichrysum argyrosphaerum in the camp was also noted. The farmer reported that the flock was moved 8 months previously to another farm, Potgietersrand (23° 15' S, 29° 43' E), where the veld was trampled and the grazing sparse, although abundant green Geigeria was present. The sheep grazed on this particular farm for 4 months before being returned to the present property. Blood

^aDepartment of Pharmacology and Toxicology, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

^bVeterinary Laboratory, Private Bag X2408, Louis Trichardt, 0920 South Africa.

^cPathology Section, Onderstepoort Veterinary Institute, Private Bag X05, Onderstepoort, 0110 South Africa.

^dToxicology Section, Onderstepoort Veterinary Institute, Private Bag X05, Onderstepoort, 0110 South Africa.

^eNational Botanical Institute, Private Bag X101, Pretoria, 0001 South Africa.

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Fig. 1: Sheep eagerly ingesting *Geigeria burkei* Harv. subsp. *burkei* var. *hirtella* Merxm. on a farm in the Northern Province.



Fig. 2: Geigeria burkei Harv. subsp. burkei var. hirtella Merxm.

samples from 21 sheep, ranging from severely affected (n = 11) to apparently healthy animals (n = 10), were collected from the jugular vein. The blood was allowed to clot, the serum collected and submitted for determination of aspartate transaminase (AST), gamma-glutamyl transferase (GGT), lactate dehydrogenase (LDH) and creatine kinase (CK) activities. These serum enzyme activities were determined by an automated chemical analyser (Technicon RA-1000 Analyser, Technicon Instruments Corporation) using the manufacturer's methods and reagents, except LDH, where the German-recommended method was used (Boehringer Mannheim). To rule out carboxylic ionophore antibiotic poisoning, samples of the poultry litter (n = 2)were also submitted for thin layer chromatography (TLC) and calorimetric analysis⁵. A necropsy was performed on a sheep that was euthanased and samples of brain, lung, liver, spleen, kidney, heart and skeletal muscle including oesophagus were preserved in 10 % buffered formalin and submitted for histopathological examination. The tissues were routinely processed and stained with haematoxylin-eosin (HE). A provisional diagnosis of vermeersiekte was made and plant material was collected, air-dried, milled and stored in a cold room (-6 °C) for dosing trials.

Laboratory trial

A 1-year-old Dorper ewe, with a cannula fitted into the rumen, was housed in a concrete pen at the Laboratory Animal Facility of the Toxicology Section at the Onderstepoort Veterinary Institute (OVI). She had free access to water and was fed oats hay and a maize meal-based pelleted concentrate. During a 2 week adaptation period and throughout the experimental period, regular clinical examinations and ECG recordings (Ectromed) were performed. Before and during the trial, blood samples were collected twice a week from the jugular vein and submitted for determination of serum activities of glutamate dehydrogenase (GLDH), AST, GGT, CK and LDH. Dried, milled plant material collected from the field outbreak was administered via the rumen cannula on consecutive days (n = 18) according to the dosing regimen presented in Table 1. Dosing ceased once clinical signs were observed. The ewe was weighed weekly and the intraruminal dose adjusted accordingly. A sample of the dried, milled plant material was also submitted for feed analysis.

RESULTS

Plant identification

The incriminated plant was identified as *Geigeria burkei* Harv. subsp. *burkei* var. *hirtella* Merxm.^{7,12} (Fig. 2) by the National Botanical Institute, Pretoria.

Family

Asteraceae

Common name

Most probably also vermeersiektebossie.

Description^{7,12}

A several-stemmed perennial herb with a woody rootstock, forming a round bush, semi-woody, 0.2–0.3 m tall. Stems densely covered with crinkled, whitish, multicellular hairs, branching in upper part with more than one branch from the apex of main stem, brownish. Leaves alternate, sessile, linear, $20-50 \times 0.5-1.5$ mm, margin strongly revolute, densely covered with glands and white, crinkled multicellular hairs. Flower heads in axils of branches with leaves involucrate. Involucre globose to campanulate, $10-20 \times 8-10$ mm, 4seriate, outer bracts long, leaf-like, appendiculate, inner bracts brownish, acuminate-lanceolate, margin in upper part ciliate. Receptacle setose. Ray florets female, 8–10 mm long, yellow, deciduous. Disc florets 5-6 mm long, bisexual, brownish yellow. Achenes narrowly turbinate, densely silky-strigose, 1.5 mm long. Pappus usually of 10 broad scales.

Habitat

Grassland.

Distribution

Pietersburg (2329 CD); Haenertsburg (2329 DD); Potgietersrus and Percy Fyfe Nature Reserve (2429 AA); Nelspruit (2530 BD); Barberton (2531 CC) (Fig. 3).

Autopsy findings

The carcass was in good condition and gross lesions included mild anaemia, muscle pallor, dull, pale areas in the myocardium, moderate hydropericardium, brain oedema, enlargement and oedema of the mesenteric lymph nodes, severe lung oedema with petechial haemorrhages, multifocal petechiae in the thymus, congestion of the spleen, kidney and liver and cholestasis. Microscopical examination of the tissue samples submitted revealed multifocal hyaline degeneration and some fragmentation of oesophageal and skeletal muscle. Myocardial lesions consisted of multifocal hyaline necrosis and myofibrolysis with mononuclear cell infiltration accompanied by mild interstitial fibrosis. Lung tissue was characterised by severe congestion, oedema and emphysema, with accumulation of alveolar macrophages.

Serum enzyme activities

The serum enzyme activities of the clinically affected and apparently healthy sheep selected on the farm where the outbreak occurred are presented in Table 2. Mean serum activities of CK and GGT were slightly raised in clinically affected sheep (n = 11). The clinical chemistry parameters determined for the sheep in the experiment are presented graphically (Fig. 4). Notable increases in AST, GGT, CK and GLDH activities were detected from Day (D) 10 and LDH activity raised conspicuously on D 13.

Analysis of poultry litter

The TLC screen for the presence of carboxylic ionophore antibiotics in the poultry litter detected salinomycin. Following quantification, using the calorimetric method, the salinomycin concentrations in the 2 samples of poultry litter were 19 and 21 ppm, respectively.

Experimental case

The ewe dosed with milled *G. burkei* subsp. *burkei* var. *hirtella* lagged behind the other sheep and exhibited stiffness in the hindquarters and recumbency from



Fig. 3: Distribution of *Geigeria burkei* Harv. subsp. *burkei* var. *hirtella* Merxm. in the Northern and Mpumalanga Provinces of South Africa. (■ = known localities.)

Table 1: Dosing regimen and body weight of the ewe during the laboratory trial.

| Experimental day | Body weight (kg) | Dosing regimen | | | |
|------------------|---------------------|------------------|---------------------------|---------------------|--|
| | | Dose g/kg × n | Total dose per day (g) | Period dosed Day | |
| 0 | 38.0 | 2.5 × 7 | 95 | 0–6 | |
| 7 | 39.0 | 5 × 7 | 195 | 7–13 | |
| 14 | 39.7 | 5×4 | 198.5 | 14–17 ^ª | |
| 20 | 36.9 | _ | _ | _ | |
| 27 | 38.1 | — | — | — | |

^aDosing discontinued after Day 17.

Table 2: Serum enzyme activities of clinically affected and apparently healthy sheep during the field outbreak.

| Enzyme | Clinically affected sheep (n = 11) | | Apparently healthy sheep (<i>n</i> = 10) | | Normal value ^a |
|------------------------------------|------------------------------------|------------------|--|------------------|---------------------------|
| | \bar{x} (SD) | Range | \overline{x} (SD) | Range | |
| CK (U/ℓ 25 °C) | 101.0 (34.34) | 40–166 | 84.9 (27.3) | 49–129 | <21 |
| AST (U/ℓ 25 °C) | 57.82 (11.05) | 41–83 | 55.3 (8.04) | 41–69 | <60 |
| LDH (U/ℓ 25 °C) GGT (U/ℓ 25 °C) | 474.36 (80.04) 37.09 (6.43) | 343–654 25–53 | 452.6 (32.12) 30.6 (4.32) | 392–499 23–41 | <530 <32 |

^aSchmidt M, Forstner D 1986 *Laboratory testing in veterinary medicine. Diagnosis and clinical monitoring.* Boehringer Mannheim, Mannheim

D 14 which progressed to a severe stiffness, lameness and paresis on D 17 and D 18. The dosing was discontinued on D 18 as the clinical signs observed were considered to be the stiff and paretic

forms of vermeersiekte. From D 19 locomotion improved. Throughout the experiment, no significant ECG abnormalities were detected and the habitus of the animal remained unaffected. During



Fig. 4: Clinical chemistry parameters of the ewe dosed with dried, milled plant material.

the adaptation period and the initial 2 weeks of dosing the body weight of the animal increased. However, during the last week of dosing a 7 % decrease in body weight occurred (Table 1). The ewe was considered fully recovered on D 27 based on the disappearance of clinical signs, the return of chemical pathology parameters to baseline activities and an increase in body weight.

Analysis of dried *Geigeria* plant material

The results of the analysis of the dried, milled plant material dosed are tabulated (Table 3). A metabolisable energy value of 8.5% and a crude protein value of 6.77% denote a fairly good nutritional quality.

DISCUSSION

The morphological relationship of G. burkei Harv. subsp. burkei var. hirtella Merxm. with related species and its distinguishing characteristics are as follows^{7,12}: G. burkei subsp. burkei var hirtella is characterised by its narrow, revolute leaves, densely covered not only with glands but also with white, multicellular hairs. Geigeria can be divided roughly into 2 main groups on basis of leaf structure. G. burkei, G. aspera Harv., G. filifolia Mattf., G. ornativa O. Hoffm. and G. acaulis (Sch.Bip.) Benth. & Hook.f. ex Oliv. & Hiern, for example, have long, linear, narrow leaves, while species such as G. pectidea (DC.) Harv. and G. obtusifolia L. Bolus have much shorter and broader, elliptical leaves. G. filifolia and G. acaulis are rosette plants (stemless), G. ornativa occurs as rosette plants or as erect, branched herbs with flower heads close Table 3: Analysis (Weende) of dried, milled *Geigeria burkei* Harv. subsp. *burkei* var. *hirtella* Merxm.

| Macro-constituents | % 'As is' |
|--|-----------|
| Moisture | 10.60 |
| Dry matter (DM) | 89.40 |
| Ash | 10.84 |
| Nitrogen | 1.08 |
| Crude protein | 6.77 |
| Crude fibre | 29.83 |
| Crude fat | 1.01 |
| Calcium | 1.04 |
| Phosphorus | 0.12 |
| Metabolisable energy (ME) (MJ/kg) (Rum.) | 8.50 |
| Nitrogen free extract (NFE) | 40.95 |
| Total digestible nutrients (TDN) | 56.85 |

together. G. burkei subsp. diffusa (Harv.) Merxm. and G. aspera var. rivularis (J M Wood & M S Evans) Merxm. are more or less prostrate, whereas the other taxa are erect, usually many-stemmed from the base, as is G. burkei subsp. burkei var. hirtella. The latter taxon is distinguished from G. burkei subsp. burkei var. burkei and var. elata Merxm. in having the leaves more densely distributed along the stem and in the lower stature. G. burkei subsp. fruticulosa Merxm. and G. burkei subsp. burkei var. zeyheri (Harv.) Merxm. are more diffusely branched and the flower heads are smaller than those of *G. burkei* subsp. burkei var. hirtella. G. burkei subsp. valida Merxm. and G. burkei subsp. burkei var intermedia (S Moore) Merxm. have the leaves much more densely crowded along the stem and the flower heads are usually much larger than those of G. burkei subsp. burkei var. hirtella. G. burkei subsp. burkei var. hirtella can be distinguished from

G. aspera by its involucre, which is globose or campanulate and not narrowly obovoid; its stems are usually simple below and branched above, not tending to branch from ground level.

Although there is experimental evidence for the toxicity of *G. burkei* subsp. *burkei* var. *zeyheri*¹⁰ this is the first confirmed field outbreak of *G. burkei* subsp. *burkei* var. *hirtella* poisoning in southern Africa. The prevailing poor grazing conditions at the end of winter and the availability of lush, green *Geigeria* might explain why the plant was so eagerly consumed by the sheep (Fig. 1). This is also the first recorded outbreak of vermeersiekte in the arid sweet bushveld of southern Africa¹ and emphasises that wherever this genus is grazed it should be regarded as potentially toxic.

Neither the sheep in the field outbreak nor the ewe in the experiment exhibited signs of regurgitation of rumen contents, but developed the characteristic stiff and paretic/paralytic forms of the disease. It is generally accepted that not all clinically affected sheep will develop typical 'vermeersiekte' (regurgitation)^{10,15}, and Dorpers are considered to be less susceptible when compared to breeds such as Merino and Karakul¹⁰.

The sharp rise in serum AST, GLDH, GGT and LDH activities of the sheep in the experiment indicate hepatocyte and biliary damage. Increased AST and GGT activities have also been observed in sheep dosed with G. aspera (N Fourie, Biocon Research Laboratories, pers. comm., 1997). Van Heerden et al.¹⁵ were of the opinion that GGT and AST may be of diagnostic value in *G. ornativa* poisoning. The Dorper ewe dosed with the plant material exhibited severe stiffness on Days 17-18, and the increased AST and LDH activities observed could reflect muscle damage, which was confirmed by the rise in CK activity (Fig. 4). In the field outbreak, mean CK activity was slightly higher in the clinically affected group (n =11) compared to the apparently healthy sheep (n = 10). A more substantial elevation in CK activity was anticipated, as the sesquiterpene lactones are primarily myotoxins. However, Joubert9 reported that serum activities of CK, glutamic oxaloacetate transaminase (GOT = AST) and GGT did not increase in correlation with the symptomatology during experimental G. filifolia poisoning in sheep, and Van Heerden et al.¹⁵ reported no increase in serum CK activity despite the presence of muscle pathology in sheep with vermeersiekte induced by G. ornativa.

Helichrysum argyrosphaerum (wild everlasting) also occurred on the farm and can induce paresis/paralysis and amaurosis in sheep^{3,10}. Since this plant was not eaten and none of the sheep were blind, *Helichrysum* poisoning was ruled out. The sheep also received a supplementary ration that included poultry litter. The carboxylic ionophore antibiotics are often present in poultry litter and may result in ionophore poisoning in ruminants, especially maduramicin⁴. In sheep, ionophore poisoning causes skeletal and cardiac muscle damage with locomotory disturbances, weakness and recumbency⁴. However, the poultry litter contained only an average of 20 ppm salinomycin, which is within the range of 15–20 ppm recommended for use as a growth promotant in ruminants⁸.

Results of the analysis of dried, milled plant material indicated that the nutritional quality of this particular Geigeria species is fairly good. This might explain why the plant was so eagerly consumed by the sheep (Fig. 1). A crude protein value of 6.77 %, crude fibre value of 29.93 % and metabolisable energy value of 8.5 % was determined. Van Heerden et *al.*¹⁵ reported an average crude protein value of 11.25 % and crude fibre value of 23.05 % for dried, milled G. ornativa (n =2). Myburg and co-workers, cited by Grosskopf⁶, analysed 84 samples of G. africana (= G. ornativa) obtained from Griqualand West and determined a mean protein percentage of 7.08 % (±1.2) and mean fibre percentage of 28.82 % (±4.7). They also analysed 2 specimens of G. aspera and determined an average protein percentage of 6.51 % and crude fibre of 27.5 %. Geigeria spp. are therefore nutritious in small quantities¹⁰, but become dangerous when they constitute a major element in the pasture.

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REFERENCES

- Acocks J P H 1988 Veld types of South Africa. *Memoirs of the Botanical Survey of South Africa no 57*. Botanical Research Institute, Department of Agriculture and Water Supply, South Africa
- 2. Anderson L A P, De Kock W T, Pachler

K G R, Brink C v d M 1967 The structure of vermeerin. A sesquiterpenoid dilactone from *Geigeria africana* Gries. *Tetrahedron* 23: 4153–4160

- Basson P A, Kellerman T S, Albl P, Von Maltitz L J F, Miller E S, Welman W G 1975 Blindness and encephalopathy caused by *Helichrysum argyrosphaerum* DC. (Compositae) in sheep and cattle. Onderstepoort Journal of Veterinary Research 42: 135–148
- 4. Fourie N, Bastianello S S, Prozesky L, Nel P W, Kellerman T S 1991 Cardiomyopathy of ruminants induced by the litter of poultry fed on rations containing the ionophore antibiotic, maduramicin. I. Epidemiology, clinical signs and clinical pathology. Onderstepoort Journal of Veterinary Research 58: 291–296
- 5. Golab T, Barton S J, Scroggs R E 1973 Calorimetric method for monensin. *Journal of the Association of Analytical Chemistry* 56: 171–173
- 6. Grosskopf J F W 1964 Our present knowledge of "Vermeersiekte" (*Geigeria* poisoning). *Technical Communication no. 21*. Department of Agricultural Technical Services, Pretoria
- Hilliard O M 1977 Compositae in Natal. University of Natal Press, Pietermaritzburg
 Immediate A (ed.) 1006 Craveth stimulants
- 8. Immelman A (ed.) 1996 Growth stimulants. Index of Veterinary Specialities 34: 75–77
- 9. Joubert J PJ 1983 Attempted prevention and treatment of *Geigeria filifolia* Mattf. poisoning (vermeersiekte) in sheep. *Journal of the South African Veterinary Association* 54: 255– 258
- 10 Kellerman T S, Coetzer J A W, Naudé T W 1988 Plant poisonings and mycotoxicoses of livestock in southern Africa (1st edn). Oxford University Press, Cape Town
- 11. Kellerman T S, Naudé T W, Fourie N 1996 The distribution, diagnosis and estimated economic impact of plant poisonings and mycotoxicoses in South Africa. *Onderstepoort Journal of Veterinary Research* 63: 65–90
- Merxmuller H 1953 Compositen-studien III. Revision der gattung Geigeria Griesselich. Mitteilungen der Botanischen Staatssammlung Munchen 7: 239–316
- 13. Vahrmeijer J 1981 Poisonous plants of southern Africa that cause stock losses. Tafelberg, Cape Town
- 14. Van der Lugt JJ, Van Heerden J 1993 Experimental vermeersiekte (*Geigeria ornativa* O Hoffm. poisoning) in sheep. II: Histological and ultrastructural lesions. *Journal of the South African Veterinary Association* 64: 82–88
- 15. Van Heerden J, Van der Lugt J J, Durante E 1993 Experimental vermeersiekte (*Geigeria* ornativa O Hoffm. poisoning) in sheep. I: An evaluation of diagnostic aids and an assessment of the preventative effect of ethoxyquin. Journal of the South African Veterinary Association 64: 76–81