

## AN OVINE HEPATOTOXICOSIS CAUSED BY THE PLANT *PTERONIA PALLENS* (ASTERACEAE) L.F.

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### ABSTRACT

PROZESKY, L., KELLERMAN, T. S. & WELMAN, WILHELMINA, G., 1986. An ovine hepatotoxicosis caused by the plant *Pteronia pallens* (Asteraceae) L.f. *Onderstepoort Journal of Veterinary Research*, 53, 9-12 (1986).

The hepatotoxicity of *Pteronia pallens* was demonstrated in 5 sheep which developed lesions that ranged from centrilobular necrosis to diffuse hepatocellular degeneration. Botanical, clinical and pathological data are given and the lesions are briefly compared with those caused by other hepatotoxic plants in South Africa.

### INTRODUCTION

Little is known about the toxicity of *Pteronia pallens*. According to Steyn (1929), stock losses often occur when animals, unfamiliar with the plant, are introduced into areas where it grows. Steyn (1949) described the clinical signs and gross lesions in a sheep dosed with the plant.

The purpose of this report is to describe the clinical signs, clinical pathology and pathology of 5 sheep experimentally intoxicated with the plant and to compare the lesions with those caused by other hepatotoxic plants in South Africa.

### DESCRIPTION, DISTRIBUTION AND ECOLOGY OF *P. PALLENS*

*Family:* Asteraceae (Compositae).

*Name:* *Pteronia pallens* L.f.

*Common name:* Scholtzbossie, Joggenscholtzbossie, Stolsbossie, aasvoëlbossie, witgatbossie (Smith, 1966).

*Description:* (Fig. 1 & 2) Small, robust, much-branched perennial bush up to 0,6 m high; branches glabrous, bark greyish-white. *Leaves* opposite, slightly connate at the base, linear-acicular, obtuse, with a narrow groove on the upper side, 10-40 mm long, about 1 mm thick, glabrous, bright green. *Capitula* about 15 mm long, solitary or in threes at the tips of the branches, shortly pedunculate, homogamous, discoid with 12-15 bright yellow to orange-yellow florets. *Involucre* turbinate-campanulate, bracts close-pressed, in about 6 series, increasing upwards, the outermost ovate-elliptic, about 1,5 mm long, the innermost linear, 7 mm long, about 1,25 mm broad, all rounded at the apex and shortly ciliate, slightly keeled, not membranous. *Receptacle* flat, 3 mm in diameter, deeply honeycombed and rather long-setose. *Florets* with corolla 8 mm long, gradually widened upwards, glabrous; lobes 5, narrowly triangular, obtuse, scarcely 1 mm long; anthers 3 mm long, linear, obtuse at the base, with lanceolate, membranous, apical appendage; ovary more or less obovoid; style terete, branches lanceolate at the hairy apex. *Achenes* 3 mm long, densely villous, pappus of 2 rows of bristles, connate at the base, light straw-coloured, 6 mm long. *Flowering time* from September to February, but mostly in October.

The distinctive characters of *P. pallens* are the greyish-white stems, the long, narrow opposite leaves and the discoid flower heads (Harvey, 1865; Hutchinson & Phillips, 1917).



FIG. 1 & 2 *P. pallens*

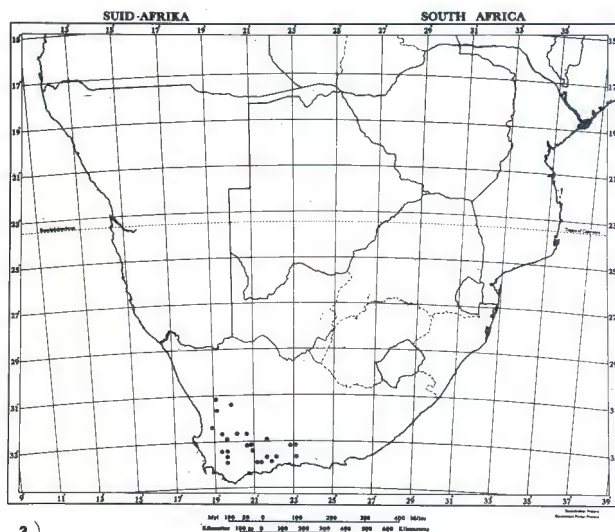


FIG. 3 Distribution of *P. pallens*

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TABLE 1 Dosing regimen, clinical signs and pathology of sheep dosed with *P. pallens*

Sheep		<i>P. pallens</i>					Dosing regimen		Fate	Clinical signs	Clinical pathology	Pathology
No.	Sex	Age	Source	State	Approximate temperature of storage (°C)	Approximate length of storage (months)	Dose (g/kg × n)	Period dosed (days)				
1	W	MT	Calvinia <sup>(1)</sup>	Green	-10	14	5 × 1 5 × 1	0 7	8	Died overnight without clinical signs being observed	No notable change	Mild ascites and oedema of the gall bladder wall. Liver: Centrilobular to midzonal necrosis and haemorrhages. Oedema of the portal triads. Lungs: Oedema
2	W	MT	Calvinia <sup>(1)</sup>	Green	-10	14	2.5 × 3	0-2	3	Died overnight without clinical signs being observed	γ-GT 137 μg/l, AST 120 μg/l, Total bilirubin 1.5 mg/100 ml (Day 2)	Prominent hydrothorax with coagulation of fluid on exposure to air. Mild icterus. Liver: Centrilobular to midzonal necrosis with mineralization of necrotic hepatocytes. Oedema of portal triads. Lungs: Oedema
3	W	MT	Calvinia <sup>(1)</sup>	Green	-10	14	1 × 4	0-3	4	Apathy, tendency to lie down, anorexia, ruminal stasis (Day 2)	γ-GT 145 μg/l, AST 1 220 μg/l (Day 2)	Ascites and oedema of the mesenterium and gall bladder wall. Liver: Centrilobular to midzonal necrosis and mineralization of necrotic hepatocytes. Kupffer's cell activation, oedema of the portal triads and a mild bile ductular proliferation. Lungs: Oedema and congestion. Kidneys: Nephrosis and perirenal oedema
4	FM	4T	Calvinia <sup>(1)</sup>	Green	-10	15	1 × 7	0-9	10	Apathy, icterus (Day 10)	γ-GT 72 μg/l, AST 148 μg/l (Day 10)	Mild icterus and oedema and haemorrhages of the gall bladder wall. Liver: Diffuse degeneration of hepatocytes, Kupffer's cell activation, oedema of the portal triads, and a mild bile ductular proliferation. Kidneys: Nephrosis. Lungs: Congestion
5	F	FM	Calvinia <sup>(2)</sup>	Dry	-10	4	5 × 2	0-1	2	Inappetence, ruminal stasis, apathy, mild icterus	γ-GT 104 μg/l, AST 2 800 μg/l. Total bilirubin 2 mg/100 ml (Day 2)	Mild icterus and oedema of the gall bladder wall. Liver: Centrilobular necrosis and haemorrhages, mineralization of necrotic hepatocytes, Kupffer's cell activation and oedema of the portal triads. Kidneys: Nephrosis, perirenal oedema, oedema and haemorrhages of renal lymph nodes. Lungs: Congestion. Gastrointestinal tract: Stasis.

W = Wether  
M = Male  
F = Female  
g/kg × n = number of daily administrations  
MT = Milk tooth  
FM = Full mouth

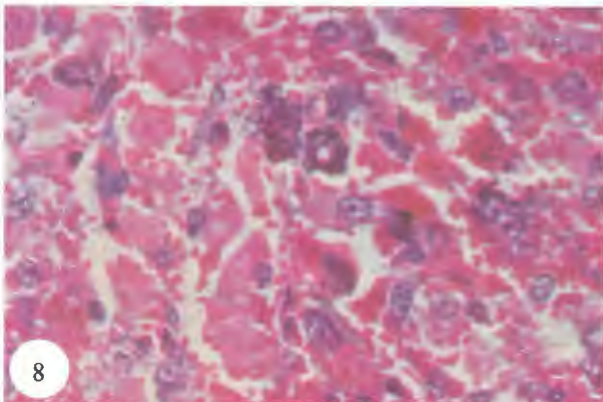
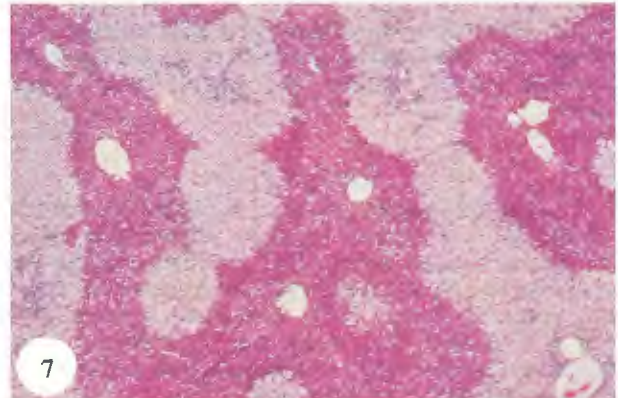
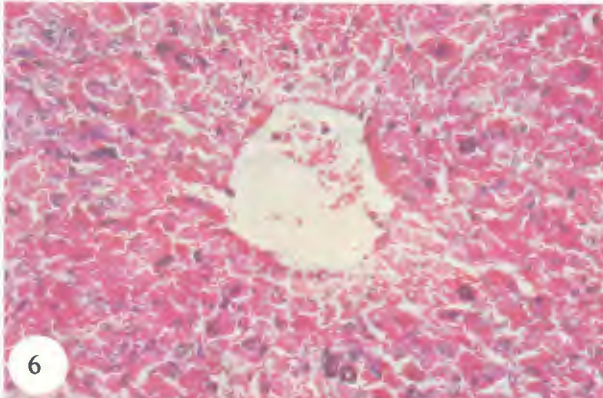
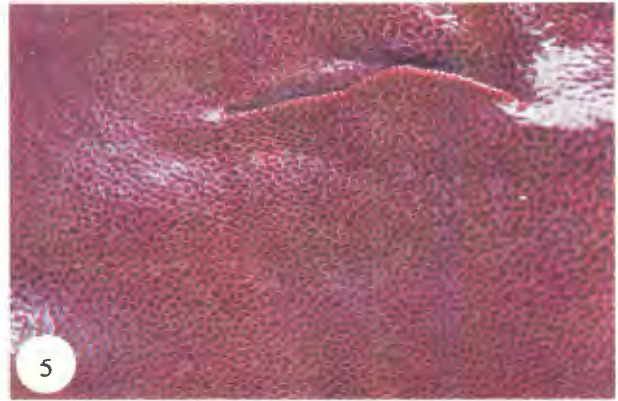
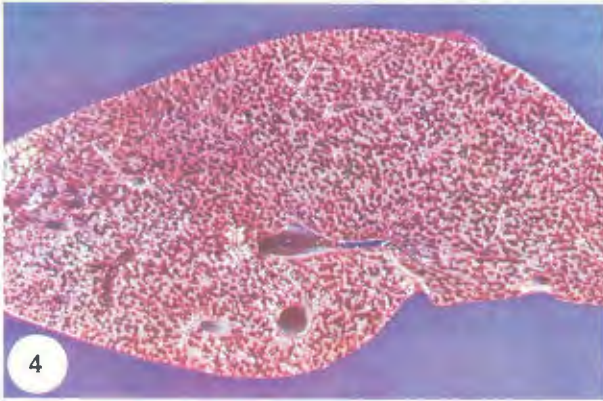


FIG. 4 & 5 Liver with accentuated lobulation

FIG. 6 & 7 Centrilobular necrosis with bridging of adjacent lobules: HE  $\times 200$ ;  $\times 28$

FIG. 8 Mineralization of necrotic hepatocytes: HE  $\times 240$

**Distribution** (Fig. 3): The plant is common in the south-western part of the Great Karoo and also in the Little Karoo, from Calvinia to the Mossel Bay district. It has been recorded in the following districts: Calvinia, Clanwilliam, Ceres, Sutherland, Worcester, Laingsburg, Prince Albert, Montagu, Ladysmith, Calitzdorp, Oudtshoorn, Riversdale, Mossel Bay.

**Ecology:** *P. pallens* can be found on rocky ridges, hills and slopes (particularly the western slopes), but also on the flats. It often grows in dry and bare areas on sandy or stony soil as well as shale and lime-rich soil, and occurs from about 300 m to 1 000 m above sea-level. It is found in the Western Mountain Karoo and succulent Karoo vegetation types and, according to Acocks (1975), is a characteristic shrub on the hillsides of veld Type No. 26 B, namely, the Little Karoo of the Karoid Broken Veld.

#### MATERIALS AND METHODS

##### Dosing trials

Five Merino sheep were dosed per stomach tube with milled plant material at the levels and intervals outlined in Table 1.

The sheep were examined daily, and periodically the following routine chemical pathological determinations were done on the blood: gamma glutamyl transpeptidase ( $\gamma$ -GT), aspartate transaminase (AST), total bilirubin, urea nitrogen, red blood cell volume and haemoglobin.

At necropsy, specimens of various organs were collected in 10 % buffered formalin, routinely processed and stained with haematoxylin and eosin. Additional staining techniques applied to various liver sections included Hall's bile stain and Dahl's alizarin method (Anon, 1968).

#### RESULTS

**Clinical signs:** Two sheep died without clinical signs being observed. Apathy, ruminal stasis and icterus were evident in the other 3 (Table 1).

**Chemical pathology:** Typical changes, related to liver involvement, including elevation of  $\gamma$ -GT, AST and total bilirubin, were recorded (Table 1).

**Pathology:** In all the animals, the livers were swollen and light-brown to dark-red, with accentuated lobulation, giving the organ a mosaic appearance (Fig. 4 & 5). Oedema of the gall bladder was evident in 4 of the sheep and 3 were icteric. In 3 cases the kidneys were pale, and perirenal oedema was present in 2 sheep (Table 1). The lungs of 3 sheep were oedematous.

The most consistent microscopical lesion in 4 of the sheep was centrilobular coagulative to lytic necrosis, accompanied by congestion and haemorrhages. The lesion often extended into the midzonal areas (Fig. 6 & 7). In 3 animals many of the necrotic hepatocytes were mineralized (Fig. 8). The hepatocytes surrounding the portal triads were degenerated (hydropic degeneration and cloudy swelling) and interspersed with single necrotic hepatocytes, which were often surrounded by neutrophils. In one of the sheep the hepatocytes were diffusely affected by cloudy swelling, hydropic degeneration and severe fatty metamorphosis, and individual necrotic hepatocytes were randomly distributed throughout most lobules. Other lesions in the animals included mild Kupffer cell activation (hypertrophy and proliferation), an increase in neutrophils in the sinusoids, bile ductular proliferation and oedema of the portal triads (Table 1).

The epithelial lining of the convoluted tubules of 3 animals was degenerated (cloudy swelling and hydropic degeneration) and protein casts were present in the lumina.

#### DISCUSSION

*P. pallens* is a hepatotoxic plant in South Africa which hitherto has received little attention. According to Steyn (1929) stock losses are experienced when animals are newly introduced into areas where the plant occurs. Henning (cited by Steyn, 1949) intoxicated a sheep with 300 g *P. pallens*. The sheep died 30 h later without symptoms, but showing gross lesions such as cyanosis, hepatomegaly and lung congestion.

In 4 of the 5 animals in the current experiment, the hepatic lesions consisted of centrilobular necrosis and haemorrhages. No zonal pattern of necrosis was evident in Sheep 4. Instead, there was diffuse degeneration of the parenchyma of this animal. Although Sheep 3 and 4 were intoxicated at a dosage level of 1 g/kg, the intervals between doses varied, which could explain the difference in the hepatic lesions between the 2 animals (Table 1). Our results show that the insult in *P. pallens* poisoning is mainly directed at the hepatocytes, although the kidneys and lungs may also be affected.

Intoxication induced by 3 plants namely, *Asaemia axillaris* (Coetzer & Bergh, 1983), *Athanasia trifurcata* (Kellerman, Coetzer, Schneider & Welman, 1983) and *Hertia pallens* (Prozesky, Kellerman, Jordaan, Welman & Joubert, 1985) can possibly be confused with *P. pallens*. According to Steyn (1929), the lesions in sheep intoxicated with *H. pallens* and *P. pallens* are very similar. Contrary to his findings, Prozesky *et al.* (1985) reported that in ovine *H. pallens* poisoning a diffuse hepatocellular degeneration was the most constant lesion and

centrilobular necrosis occurred rarely. Different zonal patterns of hepatic necrosis in sheep intoxicated with *Asaemia axillaris* and *Athanasia trifurcata*, were described by Coetzer & Bergh (1983) and Kellerman *et al.* (1983). Considering the wide spectrum of hepatic lesions associated with these 2 plants, it is clear that the lesions may in some cases be indistinguishable from those seen in *P. pallens* poisoning in sheep. Necrosis of the periportal hepatocytes and hepatocellular unrest, (depicted as hepatocytic anisonucleosis, mitoses and hepatocytes with 2-3 nuclei), associated with *A. axillaris* and *A. trifurcata* (Coetzer & Bergh, 1983; Kellerman *et al.*, (1983), were not seen with *P. pallens* poisoning.

#### ACKNOWLEDGEMENTS

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