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# Toxic ferns of Western Australia

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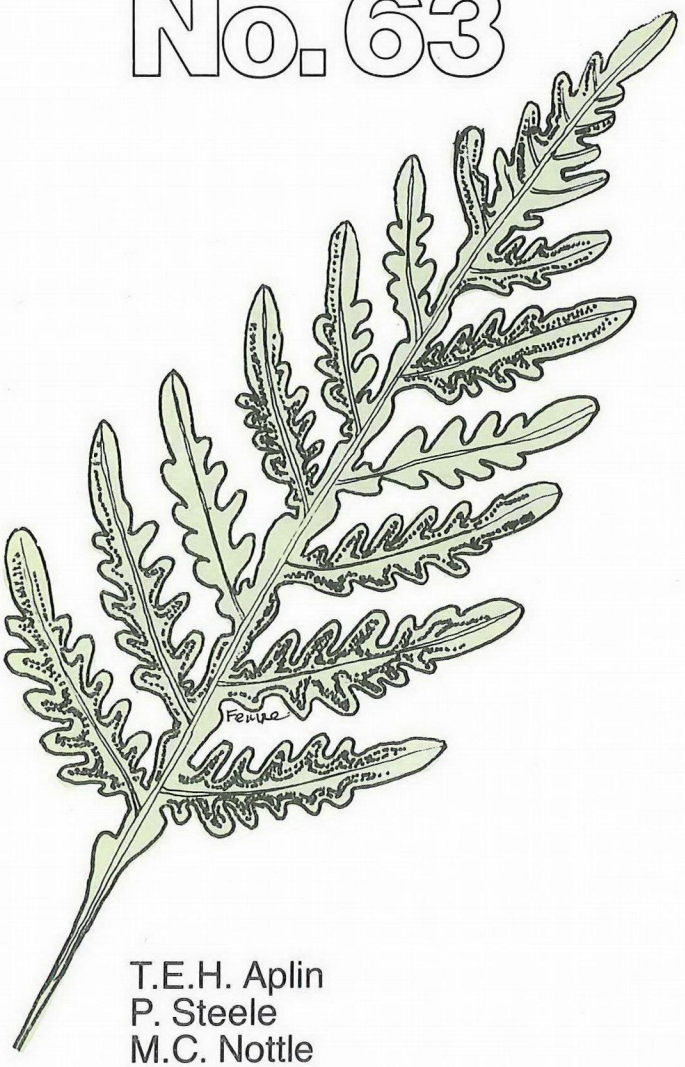


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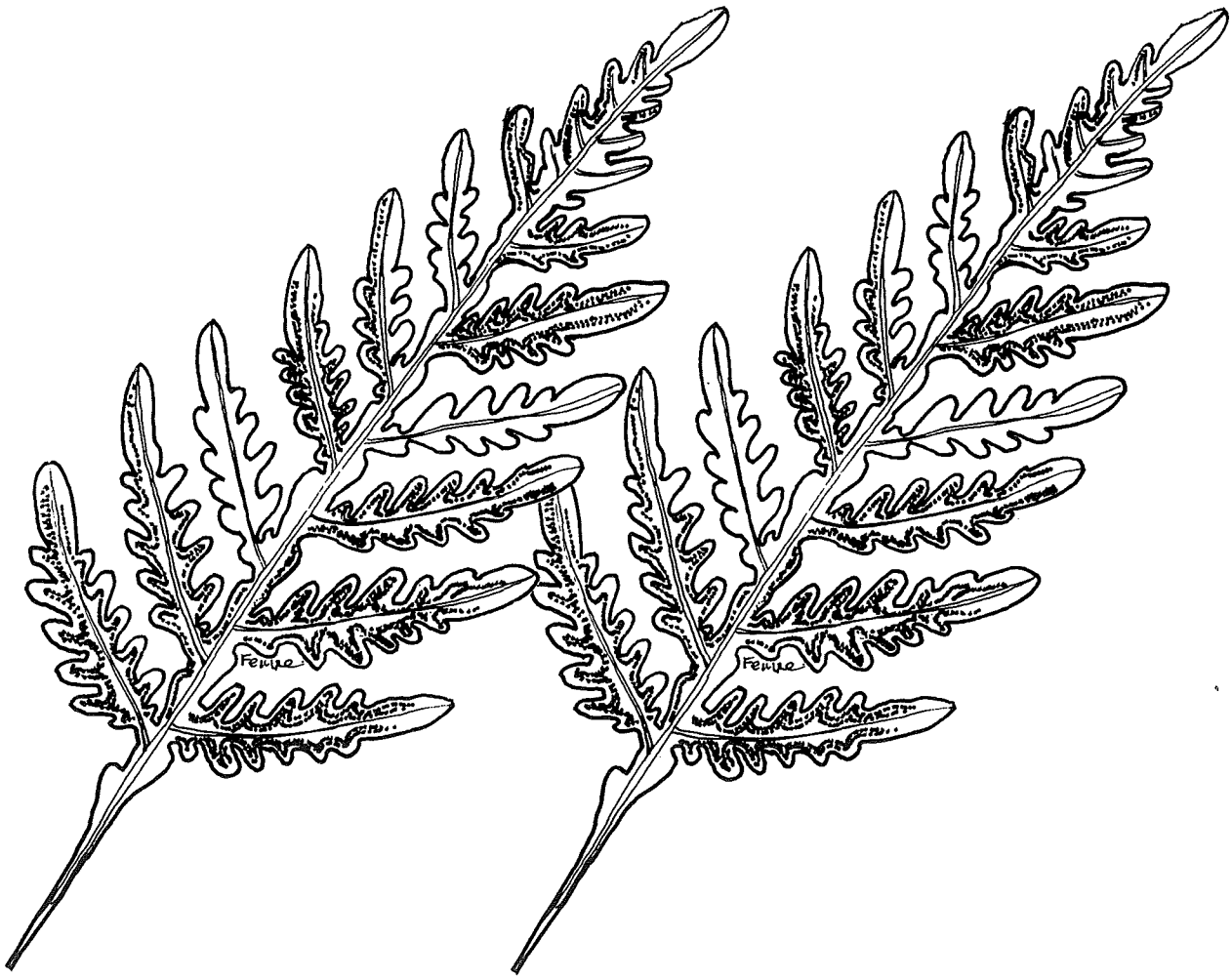
# Introduction

Ferns are a widespread group of plants which were most prevalent in the Carboniferous era some 345 million years ago. Today, there are about 10 000 species of which some 350 are found in Australia. There are 55 species in Western Australia. The majority of species are found in moist areas. Relatively few species of fern are definitely known to be toxic to livestock. In Western Australia, six toxic species have been recorded.



# Toxic ferns

Botanical descriptions of the toxic ferns and their distributions in this State are as follows: (see map 1 for location areas).

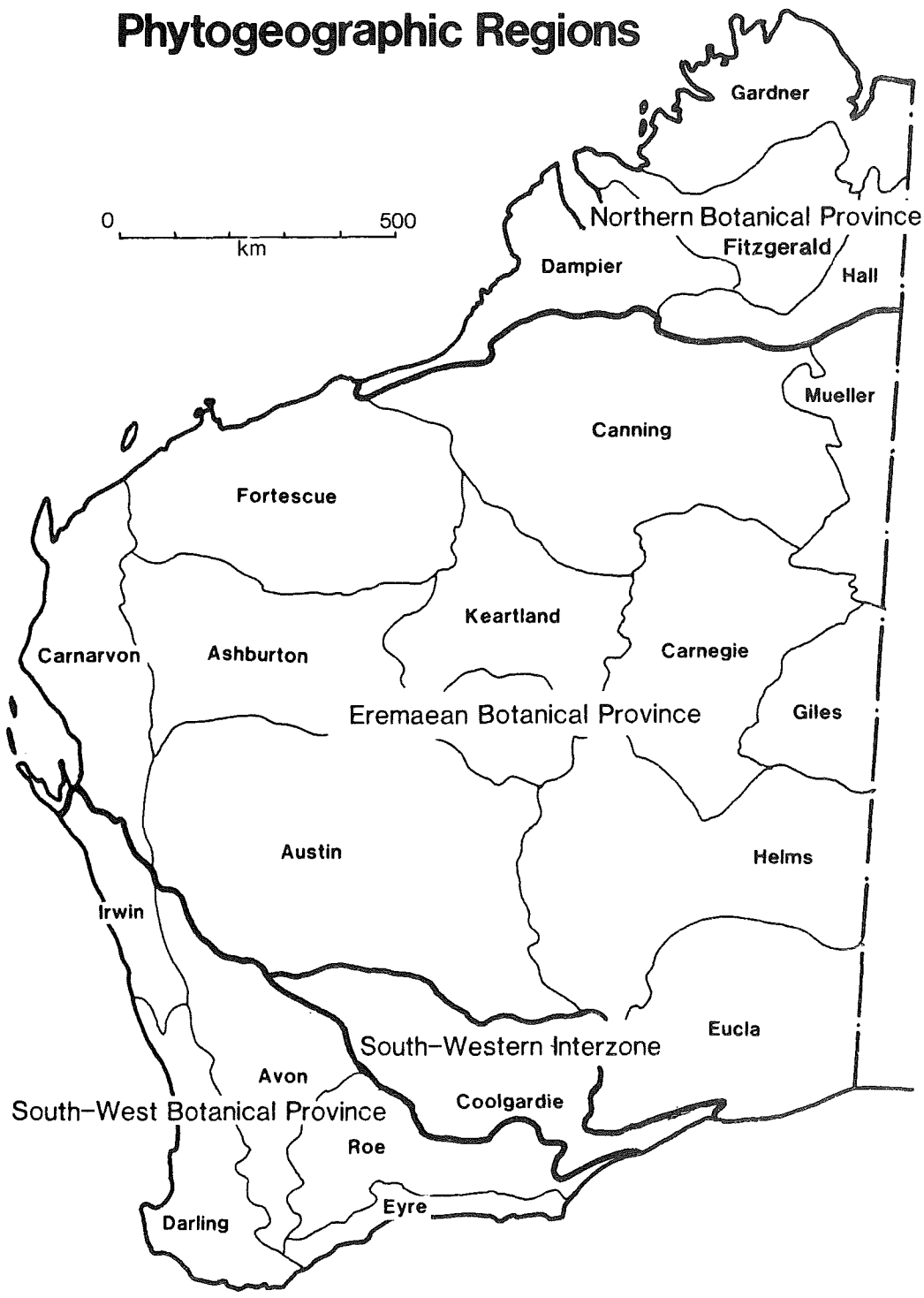
Figure 1.

## *Cheilanthes distans*

(R.Br) Kuntze, Shaggy Rock Fern.

Rhizome short-creeping, wiry, covered with narrow scales; stipes slender, dark, shiny; fronds erect, small, bipinnate; lower pinnae often reducing; pinnules with hair-like scales on undersurface, blunt; sori marginal, becoming confluent and continuous (fig. 1).

Distribution: South-West Botanical Province, general distribution, and the Eremaean Botanical Province, Coolgardie and Eucla.


Phytogeographic Regions
0
500
km
Gardner
Northern Botanical Province
Fitzgerald
Dampier
Hall
Mueller
Canning
Fortescue
Keartland
Carnarvon
Ashburton
Carnegie
Eremaean Botanical Province
Giles
Austin
Helms
Irwin
Eucla
South-Western Interzone
Avon
Coolgardie
South-West Botanical Province
Roe
Eyre
Darling

Map 1.

# *Cheilanthes sieberi*

Kuntze, Mulga Fern.

Rhizome short-creeping, wiry, covered with narrow scales; stipes slender, dark, shiny; fronds erect, small; bi-tripinnate, oblong or narrowly triangular in outline; pinnules narrow, glabrous, blunt; sori marginal, not confluent (fig. 2).

Distribution: South-West Botanical Province, general distribution, and the Eremaean and Northern Botanical Provinces.

Figure 2.

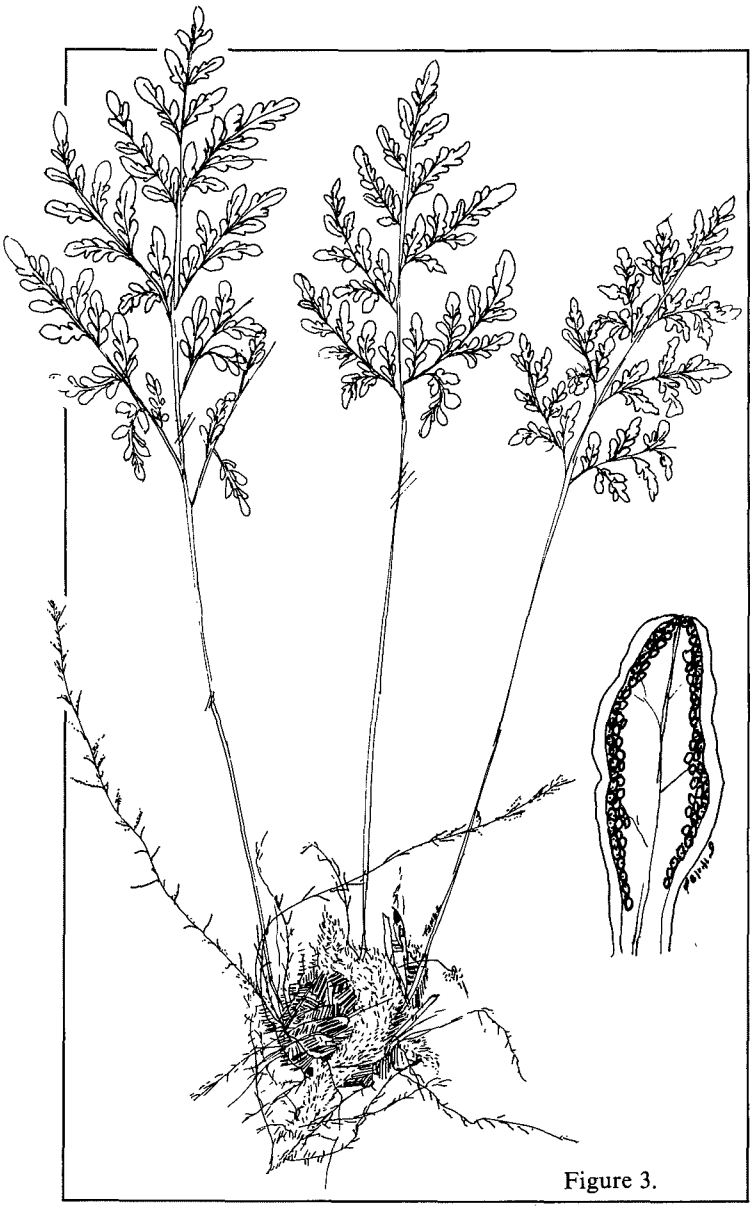


Figure 3.

# *Cheilanthes tenuifolia*

(Burm.f.) Sw., Rock Fern.

Rhizome short-creeping, wiry, covered with narrow scales; stipes slender, dark, shiny; fronds erect, small, bi-tripinnate, broadly triangular in outline; sori marginal, not confluent (fig. 3).

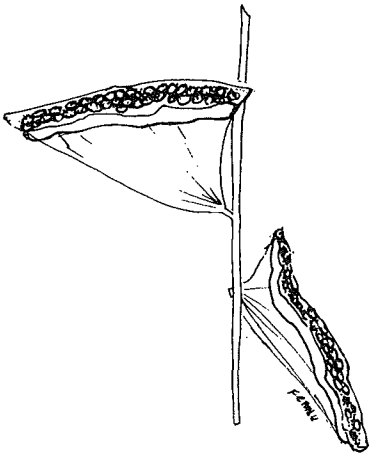
Distribution: South-West Botanical Province, general distribution, and the Eremaean and Northern Botanical Provinces.

# *Lindsaea linearis*

Sw., Screw Fern.

Rhizome short-creeping, wiry; stipes slender, dark, shiny; fronds erect, dimorphic, linear, pale; barren fronds shorter and broader forming a rosette; fertile fronds tall, narrow, erect; pinnae triangular, obliquely deflexed; sori marginal, elongated (fig. 4).

Distribution: South-West Botanical Province, Darling and Eyre.



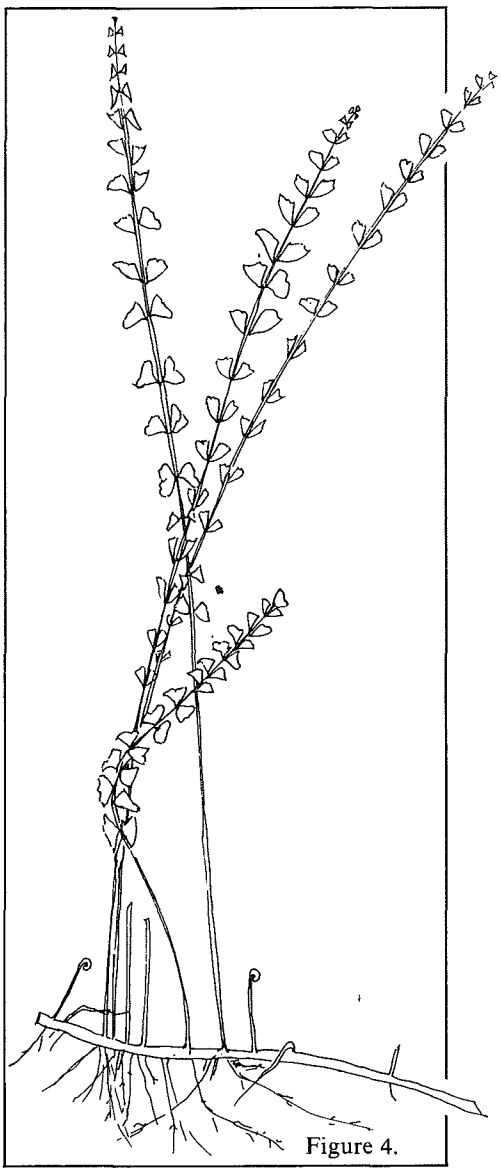


Figure 4.

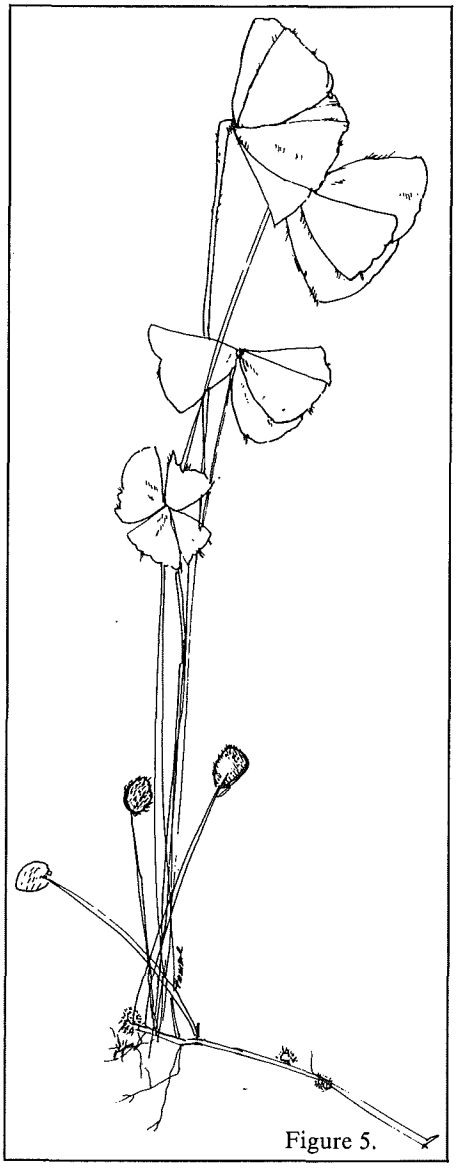


Figure 5.

# *Marsilea drummondii*

A.Br., Common Nardoo.

Leaves arising singly along the rhizome, on stalks up to 30 cm long; leaflets four, floating on or held above surface of water in which plants grow, wedge-shaped, rounded or notched at apex, glabrous or hairy; sporocarps usually arising singly from near the base of each petiole; pedicel of sporocarp longer than conceptacle (fig. 5).

Distribution: South-West and Eremaean Botanical Provinces, general distribution.

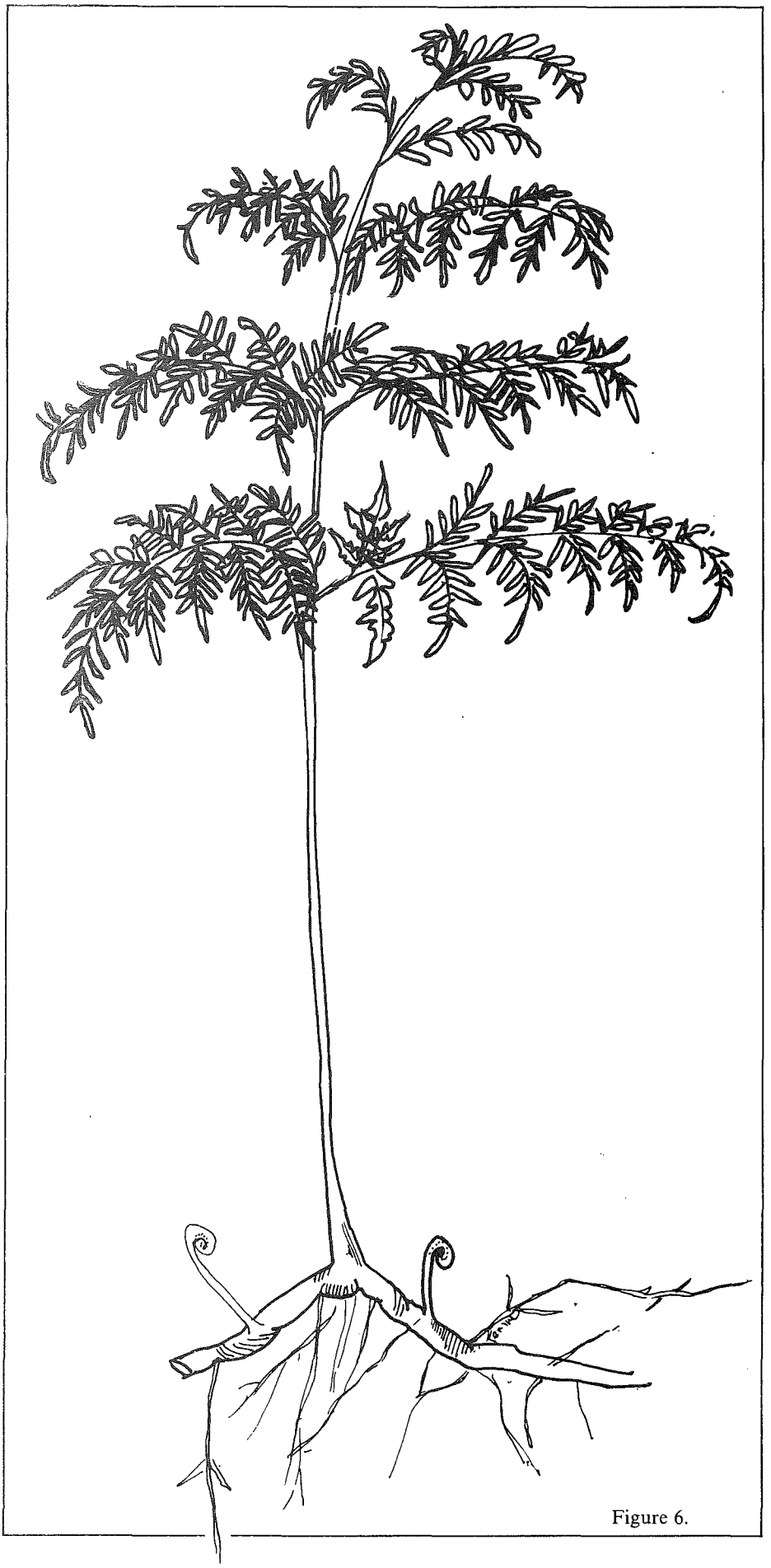


Figure 6.

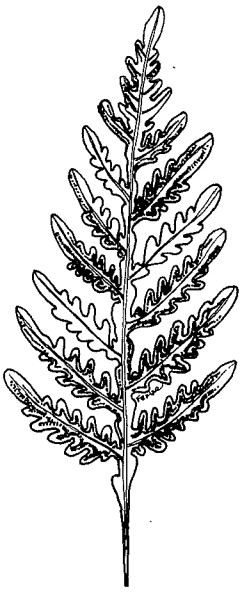
# *Pteridium aquilinum*

(L.) Kuhn var *esculentum,* Common Bracken.

Rhizome long-creeping, much branched, woody, clothed with simple hairs; fronds stiffly erect, broadly triangular, several times divided into narrow segments, glossy above, hairy with appressed fine hairs beneath; sori marginal, linear, continuous, along inrolled margin of pinna (fig. 6).

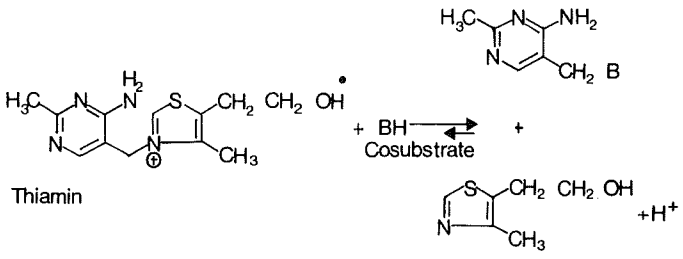
Distribution: South-West Botanical Province, Irwin, Darling, Eyre.

Note: *P. aquilinum* is found world-wide. The variety found in Western Australia is *esculentum.*



# Thiaminase

The existence of an "antithiamin factor" in ferns was demonstrated by Weswig *et al.* (1946) and Thomas and Walker (1949) who discovered that thiamin deficiency symptoms appeared in cattle and rats fed *Pteridium aquilinum,* but because of its thermostability it was not thought to be an enzyme, Evans *et al.* (1950) discoverd the presence of a thermolabile component which they assumed to be an enzyme. Fujita *et al.* (1951) investigated the antithiamin factor in *Pteridium aquilinum* in detail and demonstrated that it contained both thermolabile and thermostable components the former of which they showed to be an enzyme. Two mechanisms of vitamin $B_1$ degradation by thiaminases from several sources were studied. One reaction catalysed by an enzyme subsequently named thiaminase I (thiamin:base 2-methyl-4-aminopyrimidine-5-methenyltransferase, E.C. 2.5.1.2) required a basic substance as a cosubstrate. The reaction is represented in the equation.




A second type of enzyme, thiaminase II (thiamin hydrolase, E.C. 3.5.99.2), has been isolated from certain bacteria, but not from ferns.

## Thiaminase I activity of ferns in Western Australia

The results of analysis of thiaminase I activity in several species of ferns growing in Western Australia using a radiochemical technique (McLeary and Chick, 1977) are presented in table 1. Plant material was collected during periods of lush growth, when it was considered they would be most attractive to livestock.

The thiaminase activity shown in table 1 indicates the maximum thiamin breakdown in the presence of non-limiting amounts of basic cosubstrate. However, actual thiaminase activity in animals eating fern materials depends on the concentration of naturally occurring cosubstrate in the alimentary tract (P. Steele, unpublished data).

Evans (1976) showed that the level of thiaminase I activity in *Pteridium aquilinum* varied with different parts of the plant and with phases of the growing season. Rhizomes during winter possessed a high level of activity; this fell during spring and reached a peak in autumn. Young frond buds in spring had a high thiaminase activity which dropped as the aerial parts of the plant unfolded.

## Table 1

Thiaminase I activity in Western Australian ferns

| Species | Thiaminase activity (mg thiamin hydrolysed/h/g dry weight) |
|---|---|
| *Cheilanthes sieberi*, (Mulga fern) fronds | 108 |
| *Cheilanthes tenuifolia*, (Rock fern) fronds | 52 |
| *Lindsaea linearis*, (Screw fern) fronds | 71 |
| *Marsilea drummondii*, (common Nardoo) fronds | 130 |
| *Pteridium aquilinum* var. *esculentum*, (common bracken) fronds | 34 |
| *Pteridium aquilinum* var. *esculentum*, stems | 11 |
| *Pteridium aquilinum* var. *esculentum*, rhizomes | 42 |

# Thiamin deficiency in livestock

Thiamin deficiencies in animals may be caused by naturally occurring thiamin antagonists and thiamin-splitting thiaminases. *Pteridium aquilinum* caused clinically manifest thiamin deficiency after spontaneous intake in feeding experiments with cattle and horses (Weswig *et al.* 1946; Somoygi 1949; Thomas and Walker 1949). Somoygi and Koller (1959) isolated from bracken a substance with high antithiamin activity *in vitro* which was later identified by Beruter and Somoygi (1967) as caffeic acid (3,4-dihydroxycinnamic acid). It is not known whether, and if so how, caffeic acid induces thiamin deficiency in animals *in vivo*.

In Western Australia, the only spontaneously occurring condition attributed to thiamin deficiency is polioencephalomalacia which is found in sheep, cattle and goats, in grazing and feedlot situations (Gabbedy and Richards 1977; Steele *et al.* 1980). Thiamin produced by ruminal microbiota is, in affected animals, rapidly destroyed by abnormally high levels of thiaminase in the rumen and remainder of the gastrointestinal tract (Edwin and Jackman 1973). Increased ruminal thiaminase I activity has been found in outbreaks of polioencephalomalacia in grazing sheep (Steele and Lambe 1979) and grain fed sheep in Western Australia (Steele *et al.* 1980).

Sheep with clinical signs of the disease usually exhibit disorientation, amaurosis, anorexia and occasionally head pressing. Onset of symptoms is often sudden. Excitation may be followed by depression. Recumbent animals may exhibit opisthotonus, spasticity of the forelimbs and clonic convulsions, and often die within a few days if untreated. Animals which recover may have a residual visual impairment. Body temperature and respiratory rate are normal while heart rate may be decreased. The disease can only be confirmed histologically, but brain swelling and yellowing of the occipital and parietal cortex may be seen at post-mortem examination. Sheep usually respond dramatically to injection of 200 mg thiamin hydrochloride if this therapy is given when the first signs of the disease appear.

# Observations of toxicity due to thiaminase

*Cheilanthes distans* has been suspected in one field case in New South Wales of causing staggers in sheep and in a feeding trial using mixed material of *C. distans* and *C. sieberi* staggering and death occurred (Hurst 1942). Sheep displayed symptoms of ataxia after eating *C. sieberi* and *C. tenuifolia* (Everist 1974, McBarron 1976).

Clinical and histological observations indicate that a disease in sheep associated with *Marsilea drummondii* ingestion was polioencephalomalacia (Pritchard *et al.* 1978). Two clinical syndromes were seen. In the first one, affected sheep isolated themselves and were apparently blind; head-shaking, teeth grinding and body trembling occurred intermittently followed after 24-48 h by recumbency and death within 2-4 d. During recumbency, head-shaking was more vigorous and frothing at the mouth occurred together with trembling, brief tetanic seizures and periods of paddling movements. Affected animals frequently went into a vigorous tetanic spasm or clonic seizure with violent struggling when touched.

The second, more acute syndrome, was seen when sheep were driven. Affected animals fell behind and soon became recumbent. During recumbency, sheep frequently showed signs of marked dyspnoea, then depression, while others died quietly in deep depression in about 12 h. It appears that this acute syndrome was precipitated by the stress of driving animals which, had they been left undisturbed, would have succumbed by the slower course. Significant macroscopic pathological changes were not seen in autopsied sheep but large quantities of *M. drummondii* were consistently identified in the rumen and omasal contents which were considered drier than normal. Specific histopathologic changes which were confined to the brain were typical of polioencephalomalacia. During the investigation of Pritchard *et al.* (1978), 22 affected sheep in various stages of the disease were treated with a single subcutaneous dose of 200 mg thiamin hydrochloride. Sixteen sheep recovered, but the 6 which failed to respond were well advanced cases, recumbent for more than 24 h when treated.

Horses affected by *M. drummondii* ingestion show marked inco-ordination, stumbling and falling, hyperaesthesia and excitement and possible partial blindness. The duration of the disease varied from 3-7 d with either recovery when removed from the plant, or death (McBarron 1976).

Polioencephalomalacia has been reported in sheep consuming dried and milled rhizomes of *Pteridium aquilinum* var. *aquilinum* containing high thiaminase I activity (Evans *et al.* 1975) and in Western Australia has been induced experimentally in sheep fed on a ration containing *P. aquilinum* var. *esculentum* (Bakker *et al.* 1980).

Horses, poisoned by *P. aquilinum*, showed inco-ordination of movement followed by pronounced staggering, awkward stance and arched back. Later, there are severe muscular tremors and the animal goes down. Death is preceded by convulsive spasms and grinding of the teeth. The concentration of thiamin in the blood is very low and the pyruvate concentration is high. At necropsy, congestion and small haemorrhages are

visible in many organs, but there is nothing that is really characteristic to the disease (Kingsbury 1964). The staggering syndrome in horses has been reported in feeding trials with *P. aquilinum* var. *esculentum* (Everist 1974).

Suspected cases of "bracken poisoning" of pigs under field conditions have been reported. The condition has been produced experimentally by feeding pigs on a diet containing powdered rhizomes of *P. aquilinum*. The first signs of poisoning were a rise in blood pyruvate level and a fall in blood transketolase activity, followed by listlessness and reduced appetite. Terminal symptoms of recumbency and dyspnoea appeared suddenly and death occurred within about 6 h. Gross post-mortem lesions were dominated by enlarged, mottled hearts and evidence in other organs of acute heart failure. Microscopically, the hearts revealed lesions similar to those described in experimental thiamin deficiency in pigs (W. C. Evans 1976). No cases of "bracken poisoning" of pigs involving *P. aquilinum* var. *esculentum* has been reported.

# Toxicity other than due to thiaminase

Post-mortem examination of sheep fed on a mixture of *Cheilanthes distans* and *C. sieberi* revealed intensive enteritis, brownish blood and cyanotic lungs (Hurst 1942, Everist 1974). There has been confusion between *C. sieberi* and *C. tenuifolia*, but it appears that field cases of poisonings in New South Wales noted in earlier literature under *C. tenuifolia* apply to *C. sieberi* (Everist 1974). *C. sieberi*, and perhaps allied plants, have been suspected of producing two fairly distinct syndromes.

° Sheep show signs of ataxia, diarrhoea and varying degrees of enteritis (McBarron 1976).

° Cattle show symptoms similar to, but generally more acute than, those noted in bracken-poisoning. The clinical signs may develop weeks after removal of the animal from the plant and death is occasionally the first indication of trouble in the herd. However, sometimes blood-stained urine and scouring, accompanied by raised temperature occurs before death. Post-mortem examination has shown that affected animals can bleed to death internally, with haemorrhages under the skin and in the tissues and organs, with blood filling the lower bowel. Microscopic appearance of the blood was characteristic, with a deficiency of leucocytes and platelets and a prolonged bleeding time characteristic of bracken-poisoning (Clay 1969, Clark and Dimmock 1971).

Consumption of *C. tenuifolia* was considered by McBarron (1976) to be the cause of death in 32 pigs. Deaths continued for one week after removal of pigs from the area where the plant grew. In another case, agisted cattle were exposed to this species, but signs of poisoning were not manifest until they were placed under stress (McBarron 1976).

*Lindsaea* spp. have given positive reactions for hydrocyanic acid, but there are no reports of poisoning (Hurst 1942).

Sickness in cattle has been reported with *Marsilea drummondii*, but no details are available (Hurst 1942, McBarron 1976).

Most of the European research on bracken poisoning has been with *Pteridium aquilinum* var. *aquilinum*. Poisonings associated with *P. aquilinum* var. *esculentum* have been recorded in Australia and New Zealand. In particular, bovine enzootic haematuria caused by the variety *esculentum* has been reported in Australia (Hurst 1942, Skerman and Newton 1952, Everist 1974, McBarron 1976) and in New Zealand (Smith and Beatson 1970, Smith and van der Wouden 1972, Connor 1977).

In feeding trials with *P. aquilinum* var. *esculentum*, a 14 month old steer died suddenly on the 69th day of feeding, after consuming 93 kg of bracken fronds. Another steer fed for the same period showed signs of illness, but recovered when feeding was discontinued (Skerman and Newton 1952).

Experimental poisoning of cattle has shown, in agreement with field experience, that animals seem healthy and may gain weight until the onset of clinical signs of a rise in temperature, blood in the faeces and sometimes bleeding from the nose. In adult cattle, the "enteric type" with haemorrhage occurs, but in calves there is a "laryngitic type" with oedema of the throat and difficulty in breathing, but no external signs of bleeding. In the "enteric type" of poisoning, large multiple haemorrhages occur under the skin and on the surface of the lungs, heart, rumen, intestines, liver and kidneys. There is usually some haemorrhagic gastro-enteritis with ulceration. Blood accumulates in the intestine and frequently the lining of the blind gut (caecum) shows characteristic red or brownish bands (zebra markings). Death can occur within 2-14 d of consuming *P. aquilinum* and there can be destruction and denudation of the gastrointestinal mucosa (Evans 1968). At autopsy in the "laryngitic type" of poisoning, there may be small subsurface haemorrhages in organs, but there is no gross evidence of internal bleeding.

A fall in the level of leucocytes and thrombocytes has been reported in cattle fed on *P. aquilinum* var. *esculentum*, resulting in lowered resistance to infection, thus pneumonia and septicaemia, and spontaneous haemorrhage can occur (Lewis 1963).

Acute haemorrhagic bracken poisoning has been produced experimentally in sheep (Moon and Raafat 1951).

Suspected *P. aquilinum* var. *esculentum* poisoning and a syndrome comparable to that described for cattle has been experimentally induced in sheep in New Zealand (Connor 1977).

"Bright blindness", a progressive retinal degeneration, was induced in sheep fed on *P. aquilinum*. During these experiments, it was shown that prolonged feeding caused death in sheep with typical haemorrhaging (Barnett and Watson 1970, Watson *et al.* 1972).

# Acknowledgments

The figures used in this article were drawn by Ms F. Winfield and the manuscript typed by Mrs V. Blyton, Information Branch, Western Australian Department of Agriculture. The phytogeographic map of Western Australia was taken from J. S. Beard (1980), *Western Australian Herbarium Research Notes* No. 3, 37.

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# Addendum

A sample of *Platyzoma microphyllum* R.Br. from Kununurra, submitted on 26 May 1983, was assayed for thiaminase content. It showed an extremely high activity of 1.287 SI units/g dry wt. This corresponds to a degradation of 20.5 mg of thiamin/h/g of fern.

This indicates that *P. microphyllum* has the potential to cause thiamin deficiency in livestock.

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