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TOXICITY OF THE LEAVES OF *MACROZAMIA* SPP. FOR CATTLE

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SUMMARY.

An account of feeding tests with three groups of cattle is given. One group was fed the leaves of *Macrozamia pauli guilielmi*, a second group mixture of leaves of *M. douglasii* and *M. spiralis*, and a third group the leaves of *M. spiralis*.

Two distinct syndromes were produced. One was liver damage seen in animals in the group fed *M. pauli guilielmi*, and then only when fresh green leaves were used. The other was an ataxia seen in animals in all three groups. This ataxia affected only the hindquarters and was characterised by a swaying to both left and right and a dropping of the rump. This was associated, in particular, with poorly controlled movements of the tibio-tarsal and metatarso-phalangeal articulations. No lesions to account for this disability were found.

The toxicity of the leaves is evidently rapidly lowered by drying and withering on exposure to air.

I. INTRODUCTION.

The following statement on the genus *Macrozamia* has been prepared by Dr. S. T. Blake, Botanist, Queensland Department of Agriculture and Stock.

"*Macrozamia* is a genus of the Cycadaceae that is confined to Australia. About a dozen species are known, of which nine are recognised as occurring in Queensland. All species produce a crown of leaves on a stem, each leaf being composed of very many (25-100) narrow, pointed, tough leaflets on each side of a midrib, each leaflet having several fine longitudinal veins without a distinct midrib. The stem may be bulb-like and subterranean or develop into a tall, stout, usually unbranched trunk.

Macrozamia hopei grows to a height of 60 ft. in the scrubs of north-eastern Queensland. *M. denisonii* is a shorter species (up to about 20 ft.) and occurs in the scrubs of south-eastern Queensland. *M. moorei* is about as high as *M. denisonii* but stouter; it is found in drier country than the other Queensland species, chiefly in the southern part of the Leichhardt District, extending over the Great Dividing Range on to the headwaters of the Maranoa. *M. douglasii*, known only from Fraser Island, sometimes forms a short trunk whereas the other species usually have no stem visible above ground. *M. mountperriensis* is known from the country west of Maryborough through Mt. Perry to Eidsvold. *M. miquelii* has been reported from near Brisbane to near Mackay; it is the common species about Rockhampton. *M. spiralis* is the common species about Brisbane

and other parts of the Moreton District. *M. pauli guiljelmi* is found here and there in "wallum" country as far north as Rockhampton, occurs in the Stanthorpe district and has been reported from the Maranoa. Very little is known about *M. platyrhachis*; it is recorded only from some range near Planet Downs.

In all species the 'flowers' are arranged in more or less woody cones varying from 3-5 in. long in the case of *M. pauli guiljelmi* up to 3 ft. in the case of *M. moorei*. Each plant bears flowers of one sex only, the female cones being thicker and mostly longer than the males. The seeds are large and usually orange or scarlet when ripe; they are shed by the breaking up of the cones into its component scales. The species are distinguished by the presence or absence of spines on the leaf-stalk, the length and breadth of the leaflets and the way they are attached to the midrib, the size and shape of the cones, and the shape of the woody scales that compose them."

Investigations and feeding experiments were carried out many years ago by various observers to determine the relationship of these plants to a disease of cattle, the most significant clinical feature of which appeared to be a disturbance of locomotion known as "wobbles" or "rickets." It is still frequently referred to as "rickets."

In "The Queenslander" newspaper of October 29, 1892, there appeared a statement by Dr. T. L. Bancroft, who was commissioned by the Queensland Government to investigate the "rickets" problem. He said that the eating of the zamia palm (*Macrozamia miquelii* and *M. spiralis*) had been thought by many stock-owners to be responsible and stated:

"If it can be shown that the Zamia is the cause, it would be much better to call the disease 'zamia poisoning', for the name 'rickets' has already been used to designate a very different affection, in which softening of the bones is the main morbid change.

The chief symptom of the disease is loss of proper control over the movements of the hind limbs. A 'rickety' animal may run several yards without showing any peculiarity whatever, when suddenly it may drag its hind limbs much like a dog sick from tick-bite, or knuckle over upon its hind fetlocks, or may fall upon its haunches, immediately afterwards righting itself. . . . Affected animals seem never to recover completely, the weakness of hind-quarters continuing throughout life; but although their infirmity handicaps them in obtaining food, they are capable of being fattened if placed on good feed, and the flesh of those in good condition appears to be quite sound."

Turner (1893) recorded that the disease had been noticed in the district north of the Fitzroy River for at least 20 years. He cited an experiment done by Mr. William Norton, a grazier of Yeppoon, who fed a heifer in a small paddock with scorched zamia plants and fruits. Typical symptoms appeared on the fourteenth day.

Edwards (1894) reported feeding experiments with *M. fraseri* in Western Australia. He reproduced a syndrome similar to that seen naturally. His photographs and description leave no doubt that the condition was very similar to that seen in Queensland.

Lauterer (1898) fed leaves of *M. spiralis* to three calves. Two were given 8 lb. a day for three days and the other 6 lb. a day for six days. The gait was not affected. All three died with severe gastro-enteritis. He concluded that the plant was toxic, but was sceptical about Edwards' conclusions.

Stewart (1899) reported a similar disease in cattle on several properties in the Moruya district of New South Wales. These cattle had leaf fragments of *M. spiralis* in the rumen. His photographs show that the disease was very similar to zamia poisoning in Queensland.

Hunt (1899) said: " 'Rickets' in cattle is unquestionably caused by their eating zamia leaves... Cattle experimentally fed upon the macrozamia—whether upon the leaves, leaf-stems, bulbs, or young male or female fruit—become affected. The first signs of the paralytic condition generally appear after about fourteen days' zamia-feeding—when 2 to 4 lb. of any part of the plant are consumed per day... Cattle removed from contact with zamia after having been even slightly affected with 'rickets' show little or no tendency to spontaneous recovery, neither is their malady progressive." It was stated that the *M. miquelii* and a Cycas palm were the two species experimented with.

Seddon, Belschner and King (1931) reported a severe mortality in sheep which had eaten nuts from *M. spiralis*. There were 2,200 deaths in 6,000 sheep. The first animal died on the day after eating the plant and deaths continued for about three weeks. They confirmed the toxicity of the seeds by experimental feeding to sheep and cattle. The symptoms noted by them included injection of the conjunctivae, a rise in temperature and often an increase in the amount of faeces, which later might be reduced in quantity but remained soft. If the animal died within three days no other symptoms were seen, but if it lived longer icterus was common. Then general depression developed with the animal wandering aimlessly around the pen, tripping over any low object or bumping into the fence.

As part of a programme of investigating the plants poisonous to cattle in Queensland, a series of feeding tests with species of *Macrozamia* was done. The objectives were, briefly:—

- (1) To note any variations in the symptoms produced by the different species of zamia. This was to assist with the differential diagnosis of the ataxias seen in cattle grazed in the area.
- (2) To test the toxicity of as many species as practicable.

II. FEEDING EXPERIMENTS.

In the experiments detailed below the leaves were freshly chopped in a hand chaffcutter and mixed with lucerne chaff.

(1) *Macrozamia pauli guilielmi*.

(a) Test 1.

Plant.—Young leaves of this species were obtained from the Gympie district in November 1951. There was an interval of 2-7 days between collecting and feeding, the leaves therefore being relatively fresh.

*Subject**.—Heifer, in good condition, about 300 lb. weight and approximately nine months old.

It ate the mixture of lucerne chaff and zamia readily for the first two days, 2 lb. of zamia being consumed over this period. On the third day it was obviously sick and would not willingly eat the small ration allowed. All attempts to feed zamia ceased after the sixth day because the animal's condition had rapidly deteriorated and it refused to eat. By this time it had eaten 3½ lb. of zamia and 24 lb. of lucerne chaff.

The animal had now wasted considerably and was put out to graze with other cattle. During the next 10 days it became weaker and grazed little. On the 17th day the temperature was 107.4 deg. F., vision was impaired and shade was sought. The next week saw an abatement of the pyrexia, but the appetite did not improve and the general condition deteriorated further. On the morning of the 28th day it was found dead.

Autopsy.—The carcass was wasted, dehydration being evident. The most prominent lesion was in the liver. This was hard and of an irregular yellow colour; microscopically, it was greatly fibrosed and the parenchyma largely destroyed. There was a severe inflammation of the abomasum, with necrotic areas up to an eighth of an inch in diameter in the mucosa, reddening of the intestinal mucosa and acute congestion of the lungs.

Comment.—Death was undoubtedly due to liver damage. The interval of 22 days between the cessation of feeding and death is compatible with a diagnosis of liver insufficiency.

The amount of 3½ lb. of relatively fresh zamia leaves consumed was sufficient to cause death.

(b) Test 2.

Plant.—As for Test 1, but the interval between collecting and feeding was 11-24 days. The leaves had dried out.

Subject.—Heifer, in good condition, about 350 lb. weight and approximately nine months old.

* The cattle used in these trials were mainly Shorthorn crosses.

The beast was given daily a mixture of 1 lb. of zamia leaves and 7 lb. of lucerne chaff for 13 days. It ate readily and was normal throughout.

Comment.—The amount of leaves (13 lb.) eaten in this test was considerably greater than that which caused death of the animal in Test 1, and on a dry matter basis was still greater. The fact that the test animal showed no symptoms of poisoning suggests that the toxic principle is lost or deteriorates very rapidly when the leaves dry out and wither.

(c) Test 3.

Plant.—As for Tests 1 and 2, but collected in January 1952. The material was fresh, feeding commencing the day after it was collected.

Subject.—Heifer, in good condition, about 350 lb. weight and approximately nine months old.

The zamia leaves were mixed with a greatly reduced amount of lucerne chaff and this was eaten readily over the first two days. On the third day the heifer was obviously sick, ate less, was dull and showed photophobia. By the end of the first six days 4 lb. of zamia leaves ($3\frac{3}{4}$ lb. in the first three days) and $11\frac{1}{2}$ lb. of lucerne had been eaten.

Over the next 12 days, 4 lb. of lucerne chaff daily was allowed, but no zamia. Over this period the condition improved.

Peeling of the muzzle was noted on the 21st day, when a general decline, accompanied by a wobbling gait of the hindquarters, set in. Over the 27th to 29th days violent contraction of the abdominal muscles was noted. The animal was destroyed on the 29th day in a comatose condition.

Autopsy.—The subcutaneous tissue and visceral fat were oedematous and many petechiae were present. The liver was swollen, yellow, hard to cut and apparently fibrosed. Microscopic examination of sections showed an advanced portal cirrhosis and some centrilobular fibrosis. Liver cells were recognisable as such, but most were vacuolated. The mucosa of the abomasum was very oedematous. Microscopic examination of sections showed that most of the epithelial cells had lost their nuclei and the cell walls were ragged in appearance. The blood serum was stained a deep yellow, apparently with bile pigment.

Comment.—The results resemble those of Test 1. The interval between the cessation of feeding and death was 22 days in Test 1 and 23 days in Test 3. The animal had consumed 4 lb. of fresh zamia leaves.

(d) Test 4.

Plant.—As for Test 3. The leaves had been collected 11 days before feeding began and had withered to some extent.

Subject.—Steer, in poor condition, about 300 lb. weight and approximately 15 months old.

At first the daily mixture, which consisted of 1–1½ lb. of zamia leaves plus 4–8 lb. of lucerne chaff, was not eaten very readily, but later, when the zamia was drier, the appetite improved. It was estimated that over the 22 days of the test 18½ lb. of zamia and 100 lb. of chaff were eaten.

Clinically there was no abnormality.

Comment.—The result of this test, when compared with results obtained in Tests 1 and 3, suggests that the toxicity of the leaves is rapidly lost by exposure to air after removal from the plant. It also confirms the results of Test 2, inasmuch as the animal consumed over 18 lb. of relatively dry leaves without ill effect.

(e) Test 5.

An attempt was made to study more closely the factors involved in the production of the liver damage seen in Tests 1 and 3.

Plant.—Leaves from more mature plants than those used in Tests 1–4 were collected in the Gympie district in 1953 and fed within a few days of collection.

Subjects.—Two steers, in good condition, of similar size, each about 650 lb. weight and approximately 18 months old.

Each animal, during May, received a small amount of lucerne chaff (4 lb.) each day. One of them (462) received nothing else, but the other (566) was given as much zamia as it could be induced to eat. A series of liver biopsies was made and the serum of each animal analysed by paper electrophoresis. These examinations yielded nothing of significance.

No. 566, the animal fed zamia plus lucerne, ate well for the first five or six days, but the ration appeared to be distasteful. Feeding of zamia was stopped on the 10th day, up to which time 9 lb. of zamia had been consumed.

No symptoms of any kind were noted.

(f) Test 6.

Plant.—As in Test 5.

Subject.—Steer No. 462 from Test 5 above.

Feeding commenced in July 1953 and for the first 46 days 1 lb. of zamia leaves per day was eaten, then for 54 days, 2 lb. per day. In addition 12–20 lb. of lucerne chaff per day was given. The total amount of zamia eaten over a period of 100 days was 154 lb., all of which the animal consumed quite readily.

Mild symptoms of ataxia were seen on the 86th day; they were advanced when feeding stopped on the 100th day. There were no symptoms of liver damage, but there was clearly a disturbance in locomotion, due largely to an over-extension or increase in tone of the extensor muscles of the hind limbs.

Autopsy.—The animal was killed for autopsy. Spinal cord and brain sections were stained with haemotoxylin and eosin and additional spinal cord sections by the Marchi technique. No macroscopic or microscopic abnormality was found.

(g) Test 7.

Subjects.—Two heifers, in good condition, approximately two years old and weighing 700 lb.

One ate 100 lb. of zamia leaves in 80 days without adverse effect. The other ate 116 lb. of zamia leaves with lucerne chaff over a period of 120 days, also without ill effects. On the 66th day it calved normally and the calf did well.

Comment.—Although ataxia was noted in animal No. 462 (Test 6), which ate 154 lb. of zamia in 100 days, it was clear that the leaves were not nearly as toxic as those received from the same district at the end of 1951 and early in 1952.

(h) Summary of Results.

The results are summarised as follows:—

Test Number.	Number of Animals.	Amount of Leaves Fed. (lb.)	Age of Leaves when Fed. (Days.)	Remarks.
1	1	3 $\frac{3}{4}$	2- 7	Death 28th day. Liver damage
2	1	13	11-24	No symptoms
3	1	4	1- 4	Death 29th day. Liver damage
4	1	18 $\frac{1}{2}$	11	No symptoms
5	1 (one control)	9	4-11	No symptoms
6	1	154	4-11	Ataxia only
7	2	100 and 125	4-11	No symptoms

(2) *Macrozamia douglasii* and *M. spiralis*.

(a) Test 8.

Plant.—Leaves in various stages of growth were received from Fraser Island in April-May, 1952. The two species were about equally represented. There was an interval of 6-14 days between collecting and feeding, and the leaves became partly dried out.

Subject.—Steer, in good condition, about 400 lb. weight and approximately 18 months old.

It readily ate a mixture of 12 lb. of lucerne chaff and 4 oz. of zamia leaves per day for 35 days.

No symptoms were observed from the eating of $8\frac{3}{4}$ lb. of zamia leaves over the test period.

(b) Test 9.

Plant.—As for Test 8.

Subject.—Steer, in fair condition, about 400 lb. weight and approximately 20 months old.

It was given a mixture of 12 lb. of lucerne chaff and up to 12 oz. of zamia leaves per day. The mixture was not readily taken and when zamia feeding was stopped on the 45th day only $22\frac{1}{2}$ lb. of leaves had been consumed, an average of 8 oz. per day.

The animal remained normal until five days after feeding ceased—i.e., the 50th day. Symptoms similar to those seen in mild natural cases were then observed. These included occasional swaying of the hindquarters, plaiting of the hind legs, knuckling of the fetlock and flexing of the hock. This animal was kept under observation for two years. Its gait never returned to normal though the animal appeared to adapt itself to its disability.

(c) Summary of Results.

The following summarises the results obtained with equal parts of *M. douglasii* and *M. spiralis*:—

Test Number.	Number of Animals.	Amount of Leaves Fed. (lb.)	Age of Leaves when Fed. (Days.)	Remarks.
8	1	$8\frac{3}{4}$	6-14	No symptoms
9	1	$22\frac{1}{2}$	6-14	Mild ataxia

(3) *Macrozamia spiralis*.

Young leaves in various stages of growth were obtained in the months of June and July, 1952, from the Toogoolawah district. These were used for Tests 10, 11 and 12 detailed below.

(a) Test 10.

Plant.—There was an interval of 4-11 days between collecting and feeding.

Subject.—Steer, in good condition, about 400 lb. weight and approximately 20 months old.

It was given up to 8 lb. of lucerne chaff per day and various amounts of zamia leaves. The quantity of zamia eaten, however, never exceeded 10 oz. per day.

At the end of the 34th day, when feeding with zamia ceased, the animal had consumed an estimated 18 lb. of zamia leaves. Its condition remained good.

Symptoms were first seen on the 30th day, when the gait became slightly affected. Three days later ataxia was marked. The animal appeared sensitive over the hindquarters and hind-legs down to the coronets. After exercise it seemed abnormally frightened and excited.

On the 35th day, the day after feeding ceased, the animal had to struggle to get on to its feet. Turning was difficult and there was frequent knuckling of the fetlocks of the hind-legs. When the animal was started up quickly, its rump dropped due to abnormal flexion of the lower joints of the hind legs.

On the 36th day the hocks were swollen. Blood serum tests showed phosphate to be 4.6 mg. and calcium 10.5 mg. per 100 ml. The animal ate well while clinically affected.

Autopsy.—Autopsy was carried out on the 48th day. Macroscopically, there were bruising and haemorrhages around the hocks and some superficial bruising of the hindquarters. Sections of spinal cord, mid-brain and cerebellum stained with haematoxylin and eosin showed no abnormality.

(b) Test 11.

Plant.—As for Test 10, but the leaves were allowed to dry out in the air until the bright-green colour had changed to a dull brown.

Subject.—Steer, in good condition, about 560 lb. weight and approximately 18 months old.

It was given 12–16 lb. of lucerne chaff per day. For the first 14 days it received in addition 8 oz. of zamia leaves and thereafter 10 oz. per day. It was estimated that over a period of 103 days 62½ lb. of zamia leaves was consumed. It ate readily.

No abnormality was seen during or after the feeding of zamia ceased.

This result confirms the view that drying-out of the leaves causes rapid loss of toxicity. The difference in toxicity is even more striking when it is considered that the loss of moisture must have appreciably increased the weight of dry matter eaten.

(c) Test 12.

Plant.—As for Test 10, the leaves having been collected 4–11 days before feeding.

Subject.—Steer, in good condition, about 500 lb. weight and approximately 18 months old.

It consumed 12–20 lb. of lucerne chaff plus 4 oz. of zamia daily for 114 days, the total weight of zamia leaves eaten being 28½ lb. It ate readily, remained normal throughout and put on condition.

Strenuous exercise produced no abnormal effects.

(d) Test 13.

Plant.—As for Tests 10, 11 and 12, the plant being collected 4–18 days before feeding.

Subject.—Steer used in Test 4.

An attempt was made in this test to induce the animal to eat as much fresh zamia as possible in order to reproduce the symptoms of liver damage seen in two previous tests (1 and 3) with *M. pauli guiljelmi*. During the first nine days 5 lb. of leaves was consumed. Between the 20th and 27th days another 6½ lb., and between the 69th and 89th day a further 19½ lb., were eaten, making a total of 31 lb. A considerable quantity of lucerne chaff was also eaten.

On the 87th day typical ataxia was noted; the hindquarters swayed from side to side and knuckling of the fetlock was evident. This condition continued until the animal was destroyed on the 110th day.

Autopsy.—Superficial bruising, probably due to the uncontrolled movements, was noted around the hock joints. Microscopic examination of the liver, spinal cord, femoral nerve and sciatic nerve stained by haematoxylin and eosin and the Marchi method showed no abnormality.

(e) Summary of Results.

Test Number.	Number of Animals.	Amount of Leaves Fed. (lb.)	Age of Leaves when Fed. (Days).	Remarks.
10	1	18	4–11	Ataxia
11	1	62½	Dried out	No symptoms
12	1	28½	4–11	No symptoms
13	1	31	4–18	Ataxia

III. DISCUSSION.

One of the most striking features arising from these trials is the great difference in the degree of toxicity of the batches of leaves fed to the experimental animals.

With *M. pauli guilielmi* the first four trials were carried out with leaves collected from the Gympie district in 1951-52, and it was found that up to 4 lb. of fresh leaves (2-7 days old) was highly toxic to two bovines, whereas large quantities of the same batch of leaves, if exposed to air for 2-3 weeks, were practically non-toxic. This suggests that the toxic principle may be volatile or, on the other hand, breaks down readily.

The tests carried out in 1953 with the same species of zamia were with material collected from the same district. Using three animals and relatively fresh plant, mild ataxia was produced only in one animal, but before this symptom appeared the animal had consumed well over 100 lb. of the leaves during a period of nearly three months. This suggests that there may be also a seasonal variation in the toxicity of the species.

Tests with *M. spiralis* produced ataxia in two of the four animals used, and then only after appreciable quantities of the plant had been consumed.

The findings in Test 10, together with field observations, indicate that the damage to the nervous tissues and/or musculature which results in ataxia is permanent, although no microscopic lesions have so far been detected. Hunt (1899) also observed that there was no tendency to spontaneous recovery from the nervous symptoms in this disease.

Field reports frequently refer to damaged horns. Edwards (1894) thought that this was due to necrosis following contraction of the blood capillaries at the base of the horn-core, and Hunt (1899) suggested that it was referable to failure of nutrition of nervous origin. In the experiments reported here the horn was affected, and it is thought that in the field damage to the horn is probably brought about by the animal blundering into trees or stumps.

Lauterer (1898) was evidently able to induce his animals (calves) to eat large quantities of fresh leaves and thereby produce a gastro-enteritis. Attempts by the author to feed relatively large quantities of leaves in admixture with lucerne chaff to older animals were unsuccessful and no gastro-enteritis was observed.

Stewart (1899) and Edwards (1894) both referred to the incessant and involuntary voiding of urine in females. This feature was not seen in either of the two heifers (Tests 1 and 3) which showed symptoms, though poisoning by the swamp grass-tree (*Xanthorrhoea hostile*), which is to be found in some

of the localities where species of zamia grow, does produce in females a constant dribbling of urine as well as an ataxia not unlike that seen in zamia poisoning.

The absence of necrosis, the even distribution of the lesions and the portal fibrosis found in the livers of the experimental animals autopsied in Tests 1 and 3 suggest that the change probably started as a mild degeneration, and that complete destruction and disappearance of many liver cells and their replacement by fibrous tissue followed. Himsworth (1948) pointed out that secondary changes often obscure the primary lesions.

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