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SELENIUM POISONING IN HORSES IN NORTH QUEENSLAND

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SUMMARY.

A disease of horses in Cape York Peninsula known locally as "change hoof disease" is described.

The gross pathology in field cases, involving laminitis, loss of hair from the mane and tail and hoof sloughing, parallels that reported in chronic selenium poisoning elsewhere.

Levels of selenium found in organs and tissues from field cases are comparable with those found in selenised horses in the U.S.A. and Ireland. Selenium contents of 2.6, 12.0, 6.0 and 6.8 p.p.m. respectively were found in the liver, mane, tail and hoof of one horse, and the mane and tail of another contained 2.5 and 1 p.p.m. selenium respectively.

The selenium content of the incriminated plant, Morinda reticulata, **varied from 1.5 to 1,141 p.p.m. on a dry-matter basis but was generally well in excess of the minimum required to cause chronic selenosis.**

The feeding of 138 lb. of M. reticulata, **containing 13.3 g. Se, to a horse over an 82-day period caused lameness and loss of hair. Analyses of samples taken at autopsy showed 9.8 p.p.m. Se in the liver and the following levels in hair grown during the trial—body hair 11.2, mane 19.4 and tail 45.4 p.p.m.**

I. INTRODUCTION.

Selenium, discovered by Berzelius in 1817, was shown to be toxic to animals in 1842 (Japha 1842). Cameron (1880) demonstrated the ability of plants to absorb this element.

A disease of dragoon horses at Fort Randall, Nebraska, in 1856, characterised by lameness and loss of hair from the mane and tail (Madison 1860), was the first report of the disease later known as "alkali disease". Subsequently this disease was shown to occur over large areas of semi-arid plains of North America and to affect all grazing stock. A similar disorder resulted in animals fed grains grown in these areas. A much more rapidly fatal disease, "blind staggers", caused stock losses, particularly in Wyoming.

The connection between these apparently unrelated enzootic diseases and selenium was not suspected till Franke (1934) postulated a rare toxic element in grain from affected areas. The presence of selenium in this grain was demonstrated by Robinson (1933). Independent investigation in Wyoming showed that "blind staggers" was also due to the ingestion of herbage containing selenium (Beath, Eppson and Gilbert 1935).

Since then selenosis has been reported from many areas of the United States (Russel and Duncan 1956); from Saskatchewan, where the disorder "frozen feet" occurs in conditions of extreme cold (Williams, Lakin and Byers 1941); from Mexico, where the local name is "soliman disease" (Williams, Lakin and Byers 1940); and from Colombia (Ancizar-Sordo 1947).

The first reported case of selenosis outside the Americas was an isolated occurrence in County Limerick in Ireland (Walsh, Fleming, O'Connor and Sweaney 1951). Later, selenosis was demonstrated in Counties Tipperary and Meath (Fleming and Walsh 1957).

In Queensland a condition in horses involving laminitis and loss of mane and tail hair had been known locally in northern Cape York Peninsula since the area was first settled in 1864. This condition, called "change hoof disease", was brought to our attention when the Department of Native Affairs attempted to establish cattle at the Bamaga Settlement in the Cape York area. In March 1954 the Superintendent of Bamaga reported: "A horse muster has been completed on known settlement horses. Total mustered 9 including 3 draught horses. Two more are in the bush too lame to come in. All these with the exception of one draught are in various stages of changing their hoofs and are very lame. They will not be fit for work for several months".

In December 1955, Bamaga Settlement was visited at the request of the Department of Native Affairs. As a result of observations in the area and discussion with local residents the plant *Morinda reticulata* was suspected of causing the disease.

Records of the Queensland Herbarium show that a plant specimen identified as *M. reticulata* had been submitted from the Temple Bay area in 1923 and was reputed to cause founder and hoof abnormalities in horses.

In January 1956, McGavin (unpublished Departmental report) reported "change hoof disease" at the Arukun Mission at the mouth of Archer River. Again it was observed that the disorder occurred in an area carrying *M. reticulata.*

The results of the 1955 field investigations and subsequent feeding trials and laboratory findings are presented here.

II. FIELD INVESTIGATIONS.

Bamaga Settlement lies in a zone of 70 in. annual rainfall which is characterised by a predominantly December to March incidence. Monthly averages for 32 years show that 90 per cent. of the total falls during this period.

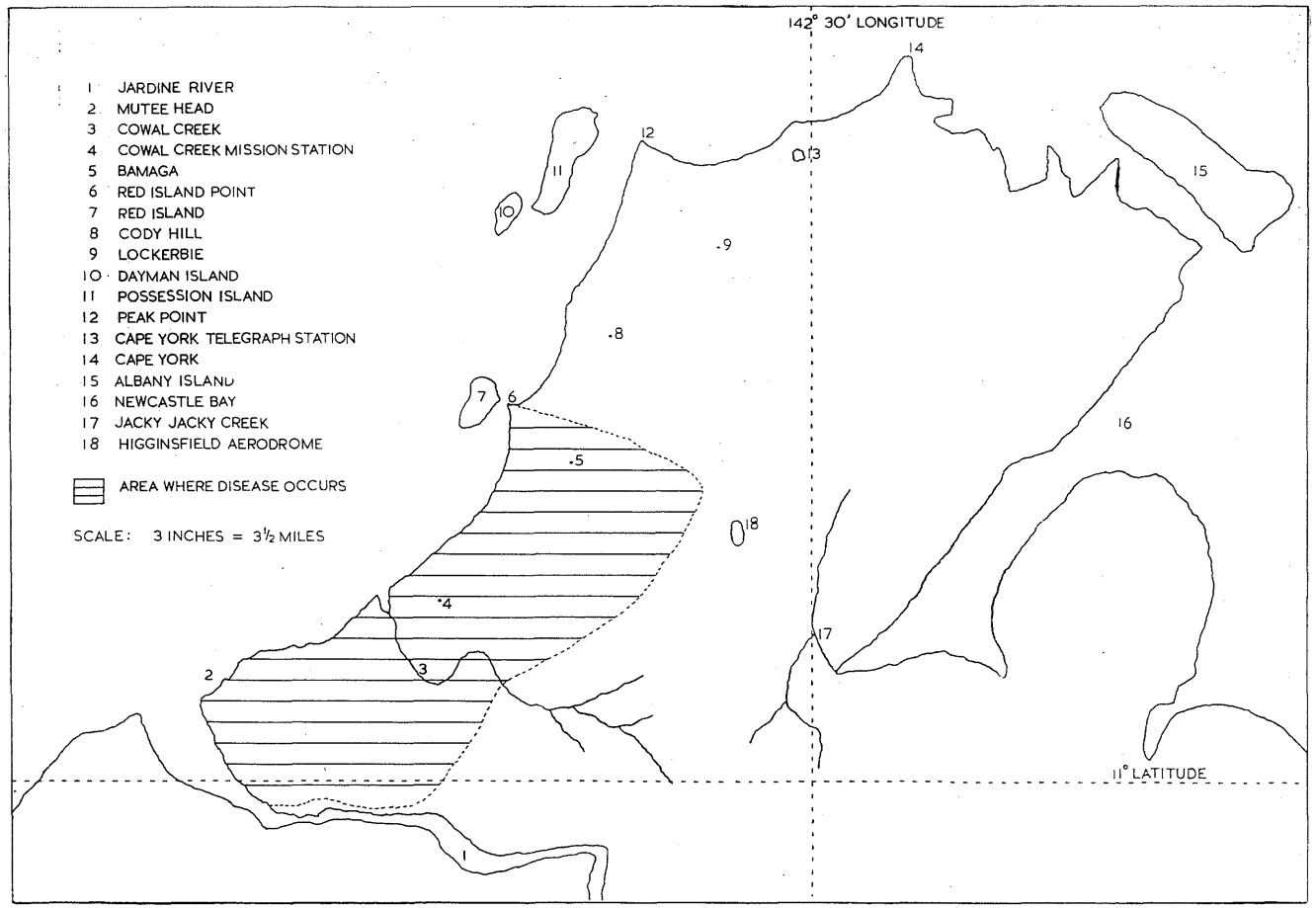

Fig. 1.

Distribution of "Change Hoof Disease" in the Bamaga Area.

Outbreaks of "change hoof disease" always occurred in the spring and early summer. The disease was confined to horses and only horses grazing certain areas were affected.

Most cases occurred in the Bamaga and Cowal Creek areas (Fig. 1). No cases occurred north of Red Island Point and consequently horses at Lockerbie and Cody Hill Stations were not affected. The vegetation over the whole area alternates between dense rain-forest and eucalypt forest country. The pasture at Bamaga was similar, but slightly inferior, to that at Lockerbie and Cody Hill. The striking botanical difference was that *M. reticulata* was very prevalent at Bamaga and was scarce on Lockerbie and Cody Hill.

Field evidence focused attention on *M. reticulata* for the following reasons:—

(*a*) In the area, pastures are burnt off in the spring and a rapid regrowth of *M. reticulata* follows early storm rains. It is succulent and relatively abundant at the period when outbreaks of the disease occur. It later becomes coarse and unpalatable.

(*b*) There was ample evidence that the plant had been grazed.

(*c*) The distribution of the plant closely followed that of recorded occurrences of the disease.

A sample from a mature plant was taken for identification and analysis. The sample contained 76 p.p.m. selenium in the dried plant.

Horses in the area were not under close supervision and loss of hair from the mane and tail was the first abnormality usually seen. Local experience was that horses were affected for several months. Most survived if food and water were readily available but some died during the acute phase of laminitis, probably due to inability to feed and reach water. In the majority of cases hoof shedding was a gradual process whereby the old hoof was replaced by new horn growing down from the coronet. Some cases had been reported to shed their hooves during the acute stage of laminitis. Horses of all ages and both sexes were affected and an attack apparently conferred no immunity, as certain horses had been affected twice or more.

At the time the area was visited three horses were affected, but all were past the acute stage of the disease.

Case 1.—This was a young unbroken filly whose front hooves each displayed a crack and transverse corrugations. The hind feet were normal and lameness had practically disappeared. Loss of hair from the mane and tail was evident. As this was a chronic mild case no samples were taken.

Case 2.—An aged bay mare was examined at Cowal Creek. This mare had shown symptoms earlier than the other two cases. All four feet showed evidence of previous laminitis, the front ones being more severely affected The near front hoof was in the process of being shed. Hair on the mane and tail was scanty. Hair from mane and tail contained 2·5 and 1·0 p.p.m. selenium respectively. The condition of this mare is shown in Figs. 2-5.

Fig. 2.

Front Hooves of Cowal Creek Mare, showing Transverse and Vertical Separation.

Fig. 3.

Hind Hooves of Cowal Creek Mare, showing Transverse Cracking.

Case 3.—An aged stallion in fair condition displayed a stilted gait and unnatural stance. All four hooves showed a prominent transverse separation just distal to the coronet. The wall of the hooves had transverse corrugations, while proximal to the separation new horn was beginning to grow down. The front feet were more severely affected. Most of the mane and a good deal of the brush of the tail had fallen out. This subject was autopsied. An affected front hoof is shown in Fig. 6. The visceral organs appeared normal but samples were taken for histopathological examination and for chemical analysis. No unusual abnormality was detected microscopically and the following levels of selenium were determined in tissues and organs:—

	p.p.m.
Liver	2·6
Mane	12·0
Tail	6·0
Hoof	6·8
Hoof	8·0

III. FEEDING TRIALS.

Trial 1.

In 1956, McGavin (unpublished Departmental report) fed an aged mare (weighing 972 lb.) 165 lb. of *M. reticulata* over a period of 56 days.

The plant fed early in the trial contained 25·5 p.p.m. selenium while that fed later contained only 1·5 p.p.m. selenium. In this trial the clinical syndrome was not produced, nor was any abnormality detected at autopsy or on histopathological examination, but analysis showed the following amounts of selenium in the animal's tissues:—

	p.p.m.
Muscle (psoas)	Nil
Heart muscle	Nil
Spleen	1·1
Kidney	4·0
Liver	3·2
Tail hair grown before experiment commenced	Nil
Tail hair grown during experiment	4·0
Hoof grown during experiment	2·0

Trial 2.

In 1957, *M. reticulata* was fed to an aged gelding in fair condition and weighing 826 lb. at the beginning of the experiment.

The plant used was identified by the Government Botanist as *M. reticulata* and was from the property of the Department of Native Affairs, Bamaga.

The leaves were stripped from the stems, minced finely and added to oaten chaff at first and later to bran, oaten chaff and ground limestone.

Symptoms were first noticed 56 days after feeding commenced by which time the animal had eaten 72 lb. of *M. reticulata* containing approximately 7·3 g. of selenium. During the following 26 days, 66 lb. of *M. reticulata* containing approximately 6 g. of selenium was eaten.

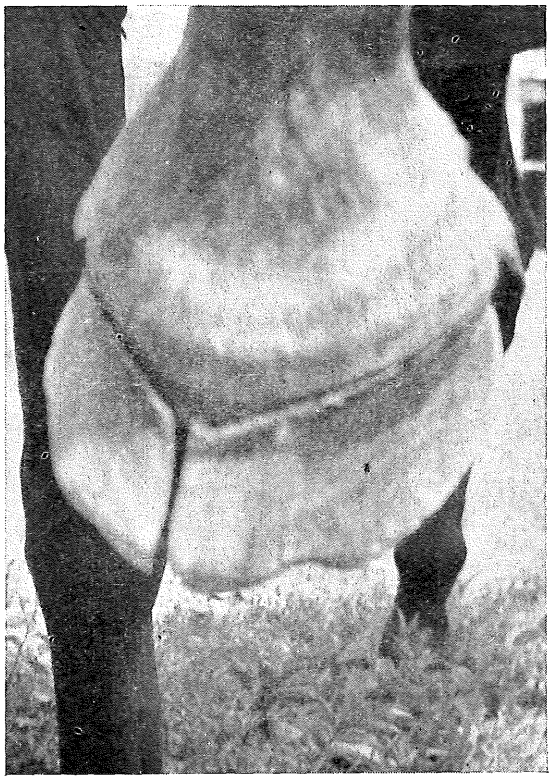

Fig. 4.

Close-up of Near-side Front Hoof of Cowal Creek Mare. Hoof in the process of being shed.

The first symptom was slight lameness in one hind foot. Four days later the horse was very lame in all four feet. It was frequently noted lying down, presumably in an attempt to ease its painful feet. When standing, the animal would place its forefeet close together and partially flex the hind limbs. Oedema was present over the ventral chest region one week after the first symptoms were seen. At this period the haemoglobin level fell from 15 g. to 12·6 g. per 100 ml. blood and the serum bilirubin rose to 2·0 mg. per 100 ml., which was the highest figure recorded during the trial.

Lameness was less marked two weeks after the animal was affected. Stranguria was noticed and was most evident when the animal was very lame.

The hoof formed during the trial was uneven, scaly and pale in colour. Horn development of all four feet was irregular for a distance of ½ in. to ¾ in. below the coronet. Distal to the abnormal horn there was a crack in the wall (Fig. 7).

A small amount of mane and tail hair was lost towards the end of the experiment. The remaining hair was fragile and broke near the skin when pulled. The animal was destroyed for autopsy on the 82nd day. The liver showed a few fibrinous tags on its parietal surface. These are common in aged horses in Queensland. The cartilage of the proximal articular surface of the left humerus was eroded, but microscopic examination did not show any inflammatory reaction of the adjacent bone. Microscopic examination of the haematoxylin and eosin stained sections of the cervical cord, thoracic cord, sciatic nerve, tibial nerve, lung, heart, spleen, adrenal and bladder did not show any abnormality. The Kupffer cells of the liver were more conspicuous than usual. In the kidney some of the glomeruli appeared swollen, completely occluding or narrowing the space between them and Bowman's capsule. However, these are common features of the liver and kidney of horses.

Analysis revealed the following amount of selenium in the tissues and organs of this animal at autopsy:—

	p.p.m.
Left lobe liver	8·6
Middle lobe liver	9·8
Left kidney	8·2
Right kidney	8·2
Spleen	2·0
Lung	0·8
Muscle (psoas)	2·9
Muscle (semitendenosis)	1·6
Blood	4·1
Left front hoof	11·4
Body hair grown during feeding	11·2
Mane hair grown during feeding	19·4
Tail hair grown during feeding	45·4

IV. LABORATORY FINDINGS.

The methods used in the analyses of soils, plant and animal tissues were those described by Trelease and Beath (1949).

Eleven samples of *M. reticulata,* taken from the area where the disease occurs (Fig. 1), were analysed. Selenium levels varied from 1·5 to 1,141 p.p.m. selenium on moisture-free plant material. Details of these findings are shown in Table 1.

Table 1.

SELENIUM CONTENT OF *Morinda reticulata*.

Specimen.	Time Collected.	Se Content as Received.	Se Content on Moisture-free Basis.	Remarks.
		p.p.m.	p.p.m.	
Old mature leaf	Dec. 1955	..	76.0	Composite sample
Fibrous leaf	Feb. 1956	16.3	25.5	Composite sample used in feeding trial 1
Fibrous leaf	Mar. 1956	0.7	1.5	,, ,, ,,
Old mature leaf	Jan. 1957	..	720	Used in feeding trial 2
Old mature leaf	Jan. 1957	..	610	,, ,, ,,
Old mature leaf	Mar. 1957	93	109	,, ,, ,,
Old mature leaf	Mar. 1957	201	259	,, ,, ,,
*Succulent young leaf ..	Nov. 1956	407	1,141	From a single plant
*Green mature leaf ..	Nov. 1956	320	422	From the same plant
*Old mature leaf	Nov. 1956	138	151	,, ,, ,,
*Rootstock	Nov. 1956	387	610	,, ,, ,,

* Soil samples in association with this plant were taken.

Two specimens from a single plant were used to assess the type of selenium compound in the plant. The results are given in Table 2.

Table 2.

TYPE OF SELENIUM COMPOUND PRESENT IN *Morinda reticulata*.

Sample.	Total Se.	Water Soluble Se.	Selenite Se.	Selenate Se.
	p.p.m.	p.p.m.	p.p.m.	p.p.m.
Green mature leaf	422	63.1	..	..
Old mature leaf	151	23.4	..	..

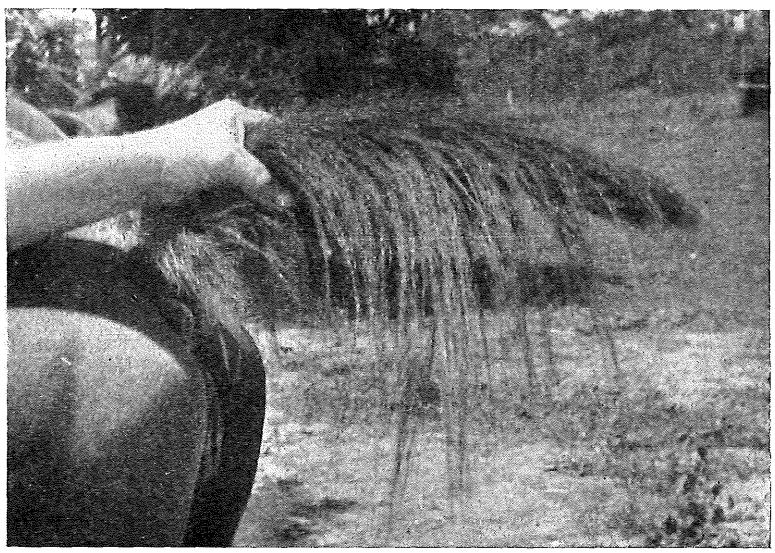

Fig. 5.
Tail of Cowal Creek Mare, showing Scant Hair on Butt of Tail.

The only other non-pasture species eaten to any extent when the disease occurs in *Breynia cernua*. A specimen of this plant growing in association with seleniferous *M reticulata* did not contain selenium.

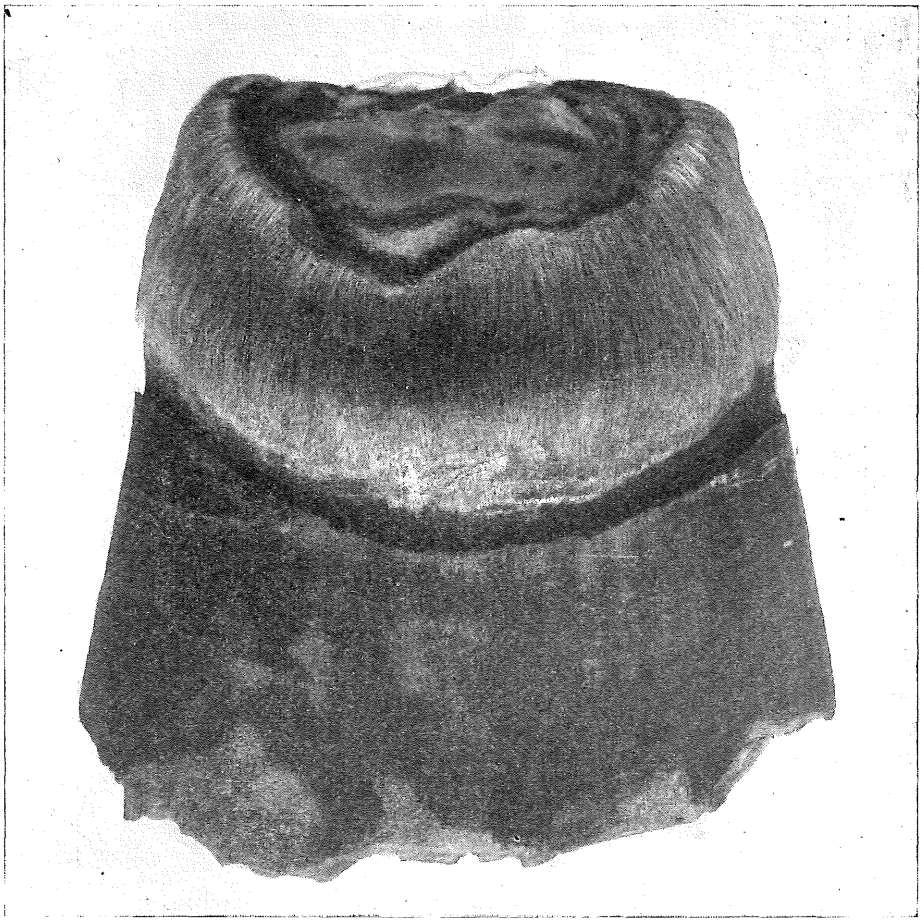

Fig. 6.

Affected Front Hoof of Bamaga Stallion, showing Transverse Separation

The soils associated with *M. reticulata* in the Bamaga area are brown sandy loams overlying a reddish-brown loam to clay loam and are probably derived from the Blythesdale formation of freshwater sandstones of Lower Cretaceous age.

Four soil samples were taken from the Bamaga area. Selenium was detected only in a sample containing debris of *M. reticulata*. Analytical data are shown in Table 3.

Table 3.

SELENIUM CONTENT OF SOIL SAMPLES TAKEN FROM BAMAGA AREA.

Specimen.	Time Collected.	Se in Air-dried Soil.
		p.p.m.
Brown sandy loam from surface of area carrying scattered *M. reticulata* *	Dec. 1955	Less than 0.02
Red brown sandy loam containing dry plant debris from surface under plant of *M. reticulata* *	Nov. 1956	0.1 to 0.4, depending on amount of debris taken
Reddish-brown clay loam taken at 12 in. depth associated with root of *M. reticulata* *	Nov. 1956	Less than 0.01
Reddish sandy loam from surface not associated with *M. reticulata*	Nov. 1956	Less than 0.01

* Analysis of this plant is given in Table 1.

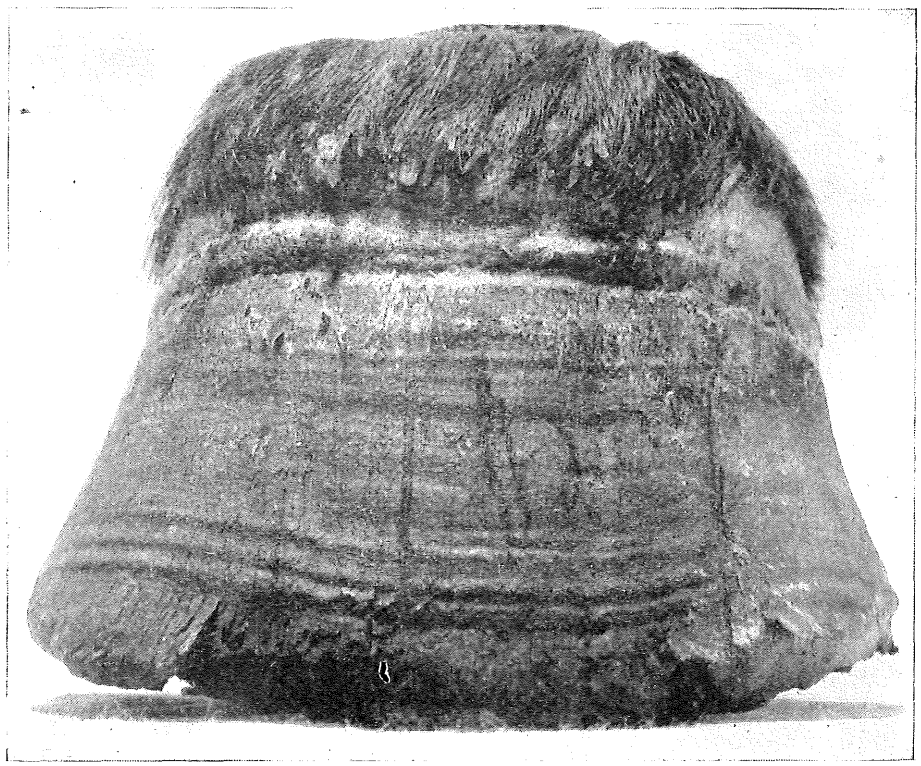

Fig. 7.

Hoof of Horse in 1957 Feeding Trial, showing Early Cracking.

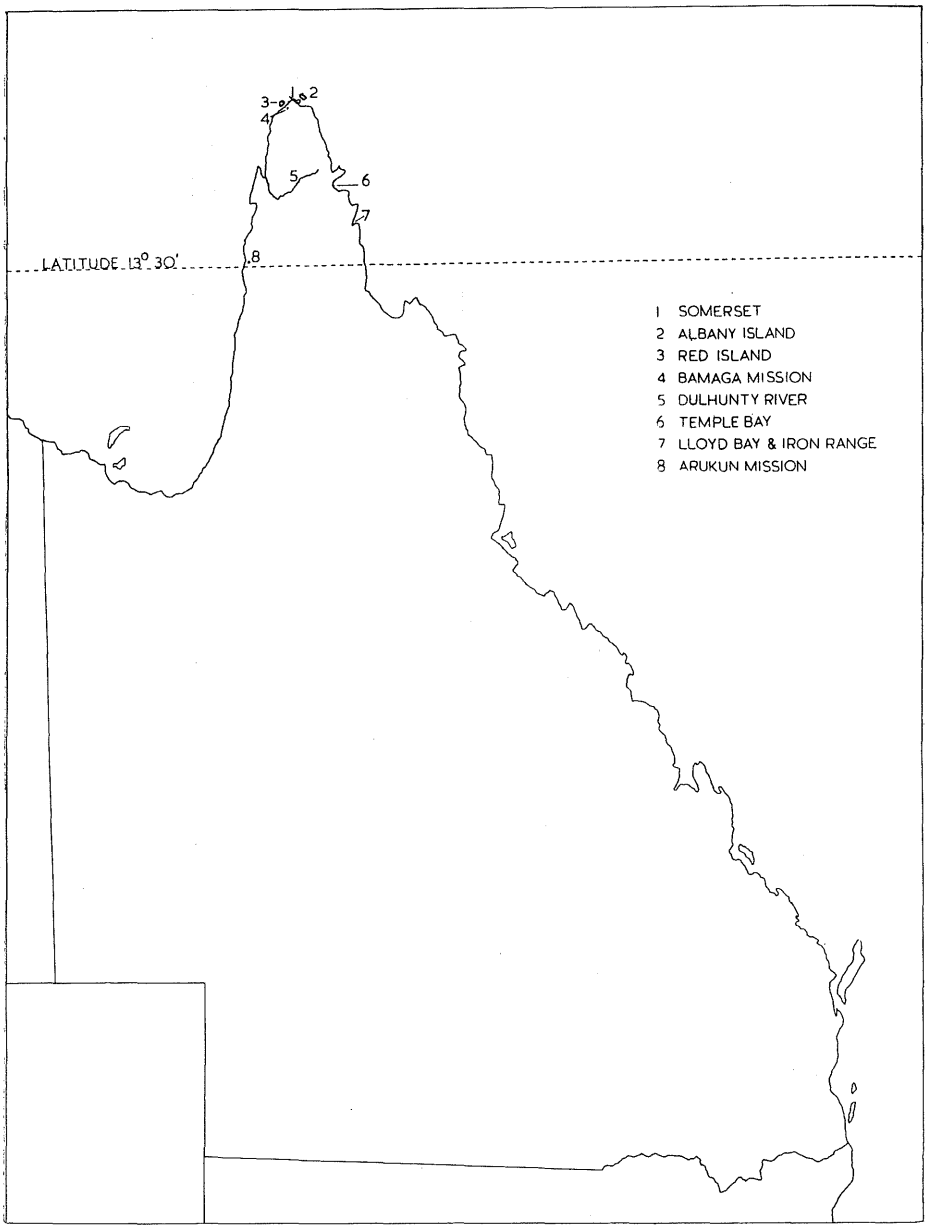

Fig. 8.

Distribution of *Morinda reticulata* as shown by Herbarium Specimens. Known distribution is confined to the Cape York area.

V. DISCUSSION.

The field evidence and feeding trials reported show that *M. reticulata* is the cause of "change hoof disease." Chemical analysis shows that the plant contains toxic levels of selenium and that the disease is chronic selenosis.

M. reticulata belongs to the family *Rubiacae,* no member of which has been previously reported to contain selenium. Eight species of *Morinda* occur in Queensland, some widely distributed in populated areas. Two are rare, including *M. reticulata,* which is confined to Cape York Peninsula, as shown in Fig. 6. This distribution is based on specimens in the Queensland Herbarium but the region is not well known botanically and it is possible that future collection may extend the known range. The plant is a low, sparse bush, growing 1–3 ft. high, with several little-branched, pale-green stems arising from a much thickened, irregular-shaped, rather woody rootstock.

Clinical changes seen at Bamaga, involving laminitis, affected hooves and alopecia of the mane and tail, follow the classical pattern for chronic selenosis in horses.

Analytical data for tissues from the horse autopsied at Bamaga (hoof 8·0, mane 12·0, tail 6·0 p.p.m. selenium) are comparable with those recorded in other countries. Fleming and Walsh (1957) reported 9·3, 8·7, 7·5, 7·3, 7·1 and 6·2 p.p.m. in sloughed hooves, 12·7 p.p.m. in the mane, and 5·4 p.p.m. in the tail of a selenised pony in Ireland. A level of 8·0 p.p.m. selenium was recorded in the hoof of an "alkali horse" from South Dakota (Dudley 1936).

No lesions were evident either macroscopically or microscopically in the liver and kidney, but the autopsied horse was said to be "recovering" and some regeneration may have taken place. Analyses of leaves of *M. reticulata* showed a wide variation in selenium content, but young succulent leaves showed selenium levels much higher than old mature leaves. This suggests that the degree of exposure of stock to this source of selenium may be related to stage of growth of this plant. This would explain the seasonal nature of the disorder, the rapid onset of symptoms and the gradual recovery in affected horses.

Clinical symptoms were not produced in the 1956 feeding trial (Trial 1). Although 165 lb. of *M. reticulata* was fed to a horse over a period of 56 days, subsequent analyses showed that the selenium content of the plant fed initially was 25·5 p.p.m. and of that fed later 1·5 p.p.m. This lower level would not be expected to induce selenosis. The analyses of tissues and organs at autopsy showed significant levels of selenium.

Plant material used in the 1957 feeding trial gave a much higher level of selenium in the diet, and in the test animal a syndrome resembling that found in the field was produced. Comparison of the amount of selenium ingested (13·3 g.) with the levels in tissues and organs indicates that an appreciable amount of the selenium as it occurs in *M. reticulata* is absorbed and retained by horses eating this plant. The concentrations of selenium in the tissues, particularly the horn and hair formed during feeding (hoof 11·4, body hair 11·2, mane 19·2 and tail 45·4 p.p.m. selenium respectively) are

comparable with those in the naturally affected animal (hoof 8·0, mane 12·0, tail 6·0 p.p.m. selenium). The uneven pale and scaly horn produced, as well as the fragility of the mane and tail hair, compare with the symptoms produced by dosing horses with sodium selenite. Miller and Williams (1940) fed sodium selenite for a period of 17 months; 96 p.p.m. selenium in the ration was given for the last two months and 48 p.p.m. selenium for the previous 9 months. This produced death and lesions in the heart, liver and kidney resembling field cases seen in U.S.A. In the *M. reticulata* feeding trial the average selenium content of the ration (13·3 g. Se in 280 Kg. feed) was 47·5 p.p.m. fed for 82 days but no internal lesions were produced.

The break in the hoof was not as marked as in the natural case, but if the animal had been allowed to survive the affected hoof may have cracked as it grew down. Affected hooves from a natural case and the 1957 feeding trial are shown in Figs. 3 and 4.

It is probable that the production of abnormal horn began within a few days of commencing to feed *M. reticulata* but no lameness was noticed till the 57th day. This could be associated with the structure of the hoof. In the unaffected hoof there would be a normal distribution of pressure at the stratum germinativum of the hoof where new horn is being formed or this horn would be abnormally shaped. Trautmann and Fiebiger (1952) described how most of the horn of the hoof is formed by the coronary stratum germinativum and it might be expected that some time will elapse before affected horn has grown down sufficiently for its weakness to cause abnormal pressure on the sensitive underlying tissues. Moxan and Rhian (1943) stated that in cattle a drop in haemoglobin can be used to detect early cases of "alkali disease". In the feeding trial there was no conspicuous drop in the haemoglobin, but the lower haemoglobin and higher bilirubin on the 60th day could be associated with some loss of red cells.

The stranguria was probably due to the painful feet causing considerable discomfort when the animal attempted to assume normal urinating posture.

In attempting to assess the extent and nature of the problem at Bamaga only the broadest general conclusions can be drawn. The remoteness and isolation of the outbreak, the sparse population and the lack of detailed geological and botanical knowledge of the area have militated against a critical discussion of factors influencing "change hoof disease".

Information available indicates the following conclusions:—

(1) The common pasture species have no significant amount of selenium for the greater part of the year, since cattle are not affected and the occurrence of selenosis in horses is seasonal.

(2) The level of selenium in the soil samples analysed has been extremely low. This is in keeping with the suggestion of a selenium accumulator plant in association with a non-seleniferous pasture and resembles conditions

in "alkali" areas in U.S.A. In Ireland a high selenium content was recorded in all pasture species growing on soils rich in organic matter containing up to 1,200 p.p.m. selenium (Fleming and Walsh 1957).

(3) A level of total selenium in *M. reticulata* commonly in excess of 200 p.p.m. is higher than levels reported for most pasture species in Irish toxic areas where soil levels of selenium are high (Fleming and Walsh 1957), and much higher than levels reported for normal forage species in "alkali" areas in U.S.A. where soil levels of selenium are relatively low (Moxan and Rhian 1943). This suggests that *M. reticulata* may be classified as an "accumulator" plant. The fact that 15 per cent. of the selenium in dried mature leaves is in the form of water-soluble organic selenium suggests the plant as a selenium accumulator comparable with secondary selenium accumulators in U.S.A. (Trelease and Beath 1949).

(4) In contrast with "alkali" areas in U.S.A. the area where the disease occurs is one of high rainfall, but observations indicate it is unlikely that the source of selenium could be a water-soluble form accumulating in wet areas. Such a source was incriminated in Ireland (Fleming and Walsh 1957).

(5) The fact that the outbreaks at Bamaga and Arukun and an unconfirmed outbreak at Temple Bay were each locally associated with *M. reticulata* suggests this plant as the major cause of selenosis in Cape York Peninsula.

VI. ACKNOWLEDGEMENTS.

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