THE TOXIC PLANTS OF WESTERN AUSTRALIA

GARDNER
AND
BENNETTS

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of

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by

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and

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FOREWORD

From the very beginning of agricultural development in Western Australia—when mysterious losses in sheep occurred on the York Road through the Darling Ranges—to the most recent development on "light lands" around the west and south coasts, the fear of poison plants (often only suspected) has worried settlers and added to the cost of development.

From time to time, since the beginning of this century, valuable data has been collected and published, covering, however, a comparatively few proved poison plants, and "The Toxic Plants of Western Australia" is the first systematic account of all the known poison plants of the State.

This has been only possible through some thirty years of active systematic research and personal collection in the field by one of the authors, C. A. Gardner, whose position as Government Botanist and Curator of the State Herbarium provided unique opportunities for this work.

The authors have achieved in a single book of moderate size the most difficult task of presenting botanical data in a manner to satisfy scientific readers, while permitting those knowing little of botany—but who are interested in the toxic properties of suspected plants—to establish their identity and to recognise the symptoms of poisoning in stock which may have eaten them.

This collection of valuable records may never have been assembled if the authors as young men thirty years ago had not only been brought together officially but had found also social interests which led to a deep and lasting friendship. A better team would be difficult to imagine, an outstanding botanist prepared to literally tramp the wilds in search of specimens and a keenly trained veterinary pathologist to observe and record authoritatively the toxological data. Dr. H. W. Bennetts, C.B.E., is Principal of the Animal Health & Nutrition Laboratory, Department of Agriculture, Perth.

In 1934 sufficient botanical data had been collected to enable a large scale project being commenced at the Avondale Research Station, Beverley, where some fifty known and suspected plants were tested. The careful description of symptoms shown by stock ingesting various poisons, together with numerous line drawings and colour prints, will assist the stock owner in tracing the cause of losses suspected as being due to poioning.

Stock losses may not be confined to the known dangerous poison plants, so the authors have included accounts of all plants which may be toxic under certain conditions; some of these plants are surprising, as they may be quite useful fodder plants under normal conditions, for instance, Burr Trefoil, Alsike Clover, and even the ubiquitous Subterranean Clover.

The book should be of value and interest to a wide circle of readers, to botanists throughout the world, to Agricultural Institutions generally, to Public Libraries, and particularly to pastoralists and farmers in

Western Australia, the Northern Territory, and also Queensland, as it contains accounts of both North-West and Kimberley plants which are also found across the North of Australia.

Careful attention has been paid in the preparation of the book to its value as a practical reference dictionary of all the known poison plants in Western Australia, and reliance has been placed on line drawings and original water colour drawings for the recognition of these poison plants rather than upon their botanical description. These are truly works of art, and represent hundreds of hours of painstaking effort over many years.

Thirty-seven original water-colour drawings by Edgar Dell have captured the distinctive colour tints of these indigenous poison plants, particularly the typical blue and grey greens of the Gastrolobiums, and the effect of the harsh dry foliage peculiar to the Oxylobiums.

One of the authors, C. A. Gardner, has increased these water-colour paintings to fifty-two and has included forty-five line drawings, many of which have appeared in the Journal of Agriculture of Western Australia, although a number have been prepared specially for this book.

The presentation of a book including technical language, intricate drawings, and the mysteries of colour plates—which to be of value must be perfect in reproduction—calls for unusual patience in the human contacts and the mastery of the whole science of book production.

The Manager and Staff, Periodicals Division of West Australian Newspapers Ltd., have succeeded in presenting a book pleasing to the artist, appealing to the taxonomic botanist, and understandable to all who come under the heading of "the practical man." Such a book will find a place in many libraries.

Perth,
Western Australia.

W. K. Baron-Hay

Director of Agriculture.

PREFACE

THE purpose of this book is to give a reliable and comprehensive record of the toxic plants which are endemic to Western Australia, and to supply stockowners with a means of recognising both native and introduced species which may be poisonous, and their effects on animals.

To attain this purpose satisfactorily the use of some scientific terms and of botanical keys is unavoidable, but an attempt has been made to illustrate and describe all the more important plants so that they can be recognised by the layman; a glossary of the terms used is included to facilitate an understanding of the structures and effects described. Where possible common as well as botanical names have been used and indexed.

Yet before proceeding to specify what is exactly meant by "toxic plants" and how such may affect stock, we have thought it advisable to consider those who may use this book.

These potential readers seem to fall into three classes:

a. Those who, because of scientific interests, desire to refer to an authoritative source;

b. Stockowners who, knowing little or nothing of botany, are faced with stock losses possibly due to toxic plants;

c. Those who may desire to know what toxic plants may be found on land in a certain area, and how to identify these.

Readers with scientific knowledge will find that by use of the coloured plates, line drawings where available, and text matter, any information sought regarding these toxic plants can be readily found.

In the case of a stockowner who finds any of his animals sick or dead, and suspects that this may be due to their having eaten what is commonly called "a poison plant," it is suggested that he turns first to the section dealing with "Distribution." Under that heading will be found a list of plants which may be the cause of loss in the area in which his property is situated. There may be one or more of these. He should then turn to the section dealing with each and note the symptoms described. (The symptoms in the case of **Gastrolobium** and **Oxylobium** are substantially the same for all species and are dealt with on pages 79 and 80 **et seq.**) If he is able to decide on one (or more) as likely to produce the symptoms noted in his own sick or dead stock, he should then turn to the coloured plates. From these he should be able to recognise any such plants growing on his property, or make a search to ascertain if such grow there, and not least important—if the plant or plants in question have been available to the stock.

Beyond that, only a post-mortem examination is likely to prove informative. Alternatively to the above procedure he may use the "List of the Indications of Poisoning by Specific Toxic Plants" (page 228). By reference to this, from the symptoms noted in his affected animals he should be able to decide what plant or plants are likely to be responsible for the poisoning of his stock, subject, of course, to their being available to the animals.

Lastly, those who for various reasons are interested to discover what toxic plants may be expected to be found in any given area, should similarly turn to "Distribution." Having then read the information relating to each species which may be native or introduced to the area, and examined the coloured plate relating to each plant, it should be possible to examine the flora of the property to discover whether it carries any of the toxic plants referred to.

The State Herbarium and the Animal Division of the Department of Agriculture are readily available to those desiring further information concerning the identification of suspected plants and the signs of poisoning resulting from their consumption by stock.

The "poison plants" native to Western Australia, by reason of their multiplicity, variety and wide distribution, and because of the very highly toxic properties of many species, constituted a serious obstacle to the early agricultural development of the State.

This applied particularly to the genera **Oxylobium** and **Gastrolobium**, the species of which are with one exception confined to Western Australia; no less than 32 toxic species are now recognised, most of them being exceedingly poisonous in certain stages of growth.

Records of poisoning of man and animals by native plants (notably Zamia) even preceded the date of the settlement of the Swan River Colony in 1829, but the literature of the subject commenced in 1840 with accounts by James Drummond, the first Government Botanist. A number of "poison plants" were described by Bentham (1864).

Taxonomic work has continued up to the present time and this book contains the first really authentic account of the numerous toxic species of **Oxylobium** and **Gastrolobium**, the outcome of many years' work by C.A.G.

The signs and post-mortem appearances induced in animals by some toxic plants were investigated and recorded by medical men—notably Rosselloty (1899) and J. B. Cleland (1912)—and by early Government Veterinary Officers (notably Edwards and Crawley).

The first publication collecting data relative to the common "poison plants" of Western Australia was that of Morrison, published in 1909 as Bulletin No. 32 of the Western Australian Department of Agriculture. Subsequently Herbert (1921) collected and edited the information available, to that time, in Bulletin No. 69, "The Poison Plants of Western Australia." This bulletin was revised by Carne, Gardner and Bennetts in a second edition published in 1926.

Up till then much of the information available concerning the effects on animals was based on reports from stockowners, including data collected by a former Agricultural Editor of "The Western Mail," the late W. Catton Grasby, and on the results of feeding tests with laboratory

animals. Much of this information was confusing and inaccurate, particularly the data concerning the symptoms and post-mortem appearances of poisoning by species of **Oxylobium** and **Gastrolobium,** and their identification.

In 1934 the two authors became associated in an investigation of the toxicity of endemic species and their effects on stock, notably sheep. During that year one of us (C.A.G.) collected the plant material, where available, and dispatched it in a fresh condition to Beverley where feeding tests, with sheep, were carried out at the Avondale Research Station (by H.W.B.); here the laboratory which had been provided earlier for the investigation of a sheep disease (entero-toxaemia) proved a valuable asset.

In this series of experiments, reported by Bennetts (1935) some 50 plants were tested. In many instances several samples of the same species from different sources, or comprising different forms of the plant, were included; 24 of the plants tested were found to be poisonous, the majority of these being species of **Oxylobium** and **Gastrolobium.** Following this investigation a variety of suspected plants have been tested, from time to time, by administration to sheep, at the Perth Laboratories of the Department of Agriculture. Further information has been collected from various sources referred to in the text; Steyn (1934), Hurst (1942) and Webb (1948) have been fully consulted.

Following the cultivation, many years ago, of the more fertile agricultural areas in the South-West Division, endemic toxic plants were largely eradicated from the areas used for the permanent depasturage of stock. Up to the present, however, very considerable mortalities of sheep and cattle have occurred in the pastoral areas from time to time, particularly in travelling mobs along the stock routes, from poisoning by species of **Euphorbia**; stock gaining access to uncultivated "bush country" are not uncommonly poisoned. Poisoning by plants has been commonly experienced during the early stages of development of "light country," extensive areas of which are now being brought into agricultural production as a result of modern knowledge of fertilizers and agricultural procedures. Cases of poisoning with garden species and newly introduced plants, moreover, are by no means uncommon.

It is evident, therefore, that a more comprehensive account of the toxic plants occurring in Western Australia than has been available previously is an important necessity. The two authors for many years had intended to carry out this project but for a number of reasons this has not been practicable until now.

There is a regrettable lack of knowledge concerning the nature of the toxic principles responsible for the poisonous properties of native species. In most instances these have not been isolated and their chemical nature is entirely unknown. This is notably so in the case of **Oxylobium** and **Gastrolobium**; Mann (1905 and 1906) claimed to have isolated toxic alkaloids cygnine and lobine from **G. calycinum** and **O. parviflorum** respectively, but subsequent workers, using the same methods, have been unsuccessful in isolating any toxic principle from these and other highly toxic species of either genus. This open field constitutes a challenge to the chemist and pharmacologist.

In the meantime, knowledge regarding the extremely varied and important toxic flora of Western Australia has steadily increased and it

is hoped that the present volume will not only form a sound basis on which to build a more complete structure, but will also be of practical value to those concerned with the husbandry of animals.

Our knowledge of the species which are toxic, and of the effects produced by some of the plants known to be toxic, is still far from complete and much remains to be done. In particular further information regarding the native species which may be cyanogenetic is very desirable, as has been evidenced by stock mortalities, presumed to result from HCN poisoning, which have occurred recently on newly developed "light land" areas and, also, under exceptional seasonal conditions.

INTRODUCTION

DEFINITION

For general purposes a toxic plant may be defined as one which detrimentally affects the health of man or animals when eaten in such amounts as would be taken normally or under special circumstances, as for example, restriction of choice of diet or extreme hunger.

It is difficult, however, to find an entirely satisfactory definition of a toxic or poisonous plant.

There are many plants, not normally cultivated for fodder, which constantly contain one or more principles which render the plants toxic when eaten by animals. On the other hand some plants which are regarded as good pasture or forage plants, under certain conditions of growth may elaborate dangerous amounts of toxic principles. Species of **Sorghum**, for example, may be cyanogenetic, leading to stock mortalities from HCN (prussic acid) poisoning, and the grass **Phalaris tuberosa,** under circumstances referred to later, may induce "phalaris staggers."

Furthermore a plant accepted and widely used as a good pasture or forage plant may cause poisoning, and even serious mortalities of stock, if taken as the main constituent of the diet over a prolonged period of time **(e.g.** subterranean clover, **Trifolium subterraneum** and the Western Australian blue lupin, **Lupinus varius).**

Animals eating plants containing certain pigments may become abnormally sensitive to sunlight. Some plants have a purely local irritant effect on the skin, inducing a dermatitis; these plants will not be considered in the text but a list of those known has been compiled for the purpose of general information.

Plants which cause purely physical ("mechanical") damage may affect the health of stock, even to a serious degree. Some of these plants, although not strictly poisonous, cannot altogether be excluded on definition. It is not proposed, however, to include them in the subsequent account of the toxic plants of Western Australia.

Suffice it to say that some plants, or some plant structures, may cause local injury which may provide a portal of entry for bacterial infection **(e.g.** grass seed abscess). Stinkwort **(Inula graveolens)** was shown by Bennetts (1932) to predispose to enterotoxaemia, a fatal disease of sheep, because of the damage to the bowel inflicted by the barbed pappus hairs of the seed when ingested; a number of fibrous or hairy types of plant **e.g.** Guildford grass **(Romulea rosea)** and "flannel bushes" **(Solanum spp.)** may give rise to "hair" balls in the gastro-intestinal tract of horses, cattle and sheep. These hair balls may, at times, cause obstruction of the stomach or bowels, resulting in death.

From what has been said it is evident that it is not always possible to draw a clear distinction between what constitutes on the one hand a toxic plant, and, on the other, a good forage or pasture species.

TOXIC PRINCIPLES

The chemical nature of the principles contained in plants, which cause poisoning in animals, is extremely variable, as are the effects induced.

The toxic principles may be classified, chemically, as **alkaloids, e.g.** strychnine which occurs in **Strychnos spp.,** nicotine or nor-nicotine, found in **Duboisia spp.** and **Nicotiana spp.;** as **glucosides,** notably the cyanogenetic glucosides which give rise to HCN poisoning; as **toxalbumins,** such as ricin, in **Ricinus communis,** the castor oil plant; as **mineral poisons,** such as oxalic acid which may occur in relatively high concentration in such **plants as Oxalis spp.** and parakeelia **(Calandrinia spp.),** or **selenium,** which can be taken up by certain plants in dangerous amounts.

In many cases the toxic principles have not been isolated and their chemical composition is unknown, although their presence is deduced from the effects produced in poisoned animals, as with the toxic species of **Oxylobium** and **Gastrolobium.**

FACTORS INFLUENCING TOXICITY OF PLANTS

The toxicity of a plant may be influenced by a number of factors, concerning both the plant and the animal.

(a) PLANT FACTORS

The toxicity of the same species of plant, related to the concentration of toxic principles within the plant tissues, may be subject to very great variations. The toxicity may vary considerably according to the locality and environment in which the plant is grown; to the variety or form of the plant (see **Isotropis spp.**); to the stage of growth; and to the parts of the plant which are eaten.

The toxic principles of many plants may be concentrated in flowers, seeds, or in the young leaves — in **Oxylobium spp.** and **Gastrolobium spp.** these last named are invariably the parts of the plant which are most highly poisonous to animals.

In addition to variation in the concentration of toxic principles within the plant, the nature of the main principles present may also differ according to the environment in which the plant is grown. This seems to apply particularly to plants containing alkaloids.

It has been shown, for example, that the main alkaloid found in Pituri **(Duboisia Hopwoodii),** when growing in the Eastern States, is nor-nicotine, whereas plants from Western Australia have been found by Bottomly, Nottle and White (1945) to yield both nicotine and nor-nicotine, the relative concentrations of either alkaloid varying according to locality. In the case of **Duboisia Leichhardtii** it was found that hyoscine appeared to be the dominant alkaloid in the northern range of the plant's distribution, while another alkaloid, hyoscyamine, preponderated in the southern range.

(b) ANIMAL FACTORS

The species of animal eating the plant, is a consideration. Plants which are definitely toxic for some animals may be quite harmless to others. **Indigofera enneaphylla,** for example, causes Birdsville disease in horses, but appears to be harmless to sheep and cattle although readily eaten by them.

The palatability of the plant and the amounts eaten may be important considerations. Some toxic plants are eaten in quantity only by hungry stock (**e.g.** "native tobacco," **Nicotiana spp.**), or by animals which are not accustomed to them (**e.g.** Cape tulip, **Homeria spp.**). Other plants may constitute good forage for horses, cattle and sheep under ordinary conditions, but heavy mortalities may result when large amounts of the same plants are eaten in a short space of time by hungry travelling stock.

Other factors in the animal's environment may predispose to plant poisoning. The driving of stock in some instances may reveal or precipitate signs of poisoning (**e.g.** "rye grass staggers"): if sheep are left undisturbed after eating **Oxylobium spp.** and **Gastrolobium spp.** losses may be light, but if the mob is disturbed, or frightened by dogs, etc., heavy mortalities from poisoning may result.

In some instances plant poisoning may become evident after stock have watered.

The state of health of the animal may be a factor in determining poisoning; photosensitization may result from the consumption of chlorophyll, the normal green pigment contained in common pasture (and other) plants, by animals **in which the liver function is altered by disease.**

In general, the effects induced in animals by the toxic plants of Western Australia will be considered together with the description of the plants concerned, which are arranged in the text according to the systematic botanical sequence.

There are, however, a number of plants which, although quite unrelated botanically, still induce the same "group effects" in poisoned animals—notably (a) cyanogenetic plants, and (b) plants causing photosensitivity.

A general account of poisoning with plants causing these effects will be considered here in order to avoid much repetition in the text.

CYANOGENETIC PLANTS

Cyanogenetic plants can be defined as those which, at least under certain circumstances, may give rise to hydrocyanic (prussic) acid poisoning in grazing animals.

Such plants, which are widely distributed throughout the plant kingdom, produce substances known as cyanogenetic glucosides during their growth. The glucosides are not themselves poisonous but, in the presence of appropriate enzymes, are broken down giving off free hydrocyanic acid (HCN) which is, of course, highly toxic.

Poisoning of the grazing animals is related to three main considerations:—

1. The concentration of cyanogenetic glucosides in the plant.
2. The presence of an enzyme (ferment) which will hydrolyse the glucoside in question, so that free hydrocyanic acid is liberated.
3. The amounts eaten and the rate of consumption by the animal.

These factors may now be considered in some detail:—

The Concentration of Cyanogenetic Glucosides.

Some plants normally contain cyanogenetic glucosides, but in others they occur only under certain conditions. This applies particularly to cyanogenetic grass, **q.v.**

Climate appears to be the most important factor influencing the concentration of glucosides in the plant, but soil factors and stage of growth also have an effect. Wilting has been shown to cause a large increase in concentration as does frosting or damage from insects, disease, or mechanical means. It appears that periods of rapid regrowth of plants following a check or caused by rain after a period of drought are particularly favourable to toxicity.

The highest concentration of HCN occurs in the early stages of growth and decreases as the plant matures. Soil conditions, too, may influence the HCN content of the plant; low soil moisture, high nitrogen content and low phosphorus content are all factors favourable to HCN production. Plants grown on good soils are likely to be more highly toxic than those grown on poor soils.

The strain or variety of the plant can also greatly influence the HCN content, as has been shown with sorghum and with white clover. In the case of white clover the investigations of Corkill (1942) and Melville **et al.** (1940) demonstrated that there may be wide hereditable differences in both the cyanogenetic glucoside and enzyme content of different strains.

Some species of plants **(e.g. Eremophila maculata)** generally contain dangerous concentrations of cyanogenetic glucosides; other species do so only under certain circumstances, whereas some species **(e.g.** white clover) rarely, if ever, yield a concentration of HCN which is sufficient to cause poisoning in grazing animals.

The Presence of Appropriate Enzymes.

As indicated, the fresh growing plant does not usually contain free HCN* but a cyanogenetic glucoside or glucosides which when acted upon by a suitable enzyme will yield the poisonous acid.

The chemical nature of these glucosides varies considerably, and each glucoside can be broken down only by a specific (suitable) enzyme. This phenomenon has been likened to a lock and key mechanism.

The appropriate enzyme may be contained in the same plant containing the corresponding glucoside, or in some cases the enzyme may occur in a completely different plant species. Finnemore (1931), for example, found that the native fuchsia, **Eremophila maculata,** which contains the glucoside prunasin, may not contain the necessary "key" enzyme to liberate HCN. This, however, is found in other plants, including gidgee. It was alleged that on the Georgina River stock could eat the leaves of native fuchsia without being poisoned until they also ate other plants or gidgee pods.

It does seem evident that the **non-ruminant** animal must consume plants containing both the glucoside and its "key" enzyme for poisoning to occur, whereas in **ruminants** it may be otherwise, as has been demonstrated by Coop and Blakley (1949). Their investigations proved that the main cyanogenetic glucoside of white clover **(lotaustralin)** is rapidly hydrolysed by the ruminal flora, the presence of a plant enzyme thus not being necessary for the production of free HCN. It seems most probable that the same may apply to other cyanogenetic plants containing different glucosides, and that in sheep, cattle (and other ruminants), HCN poisoning may occur in the absence of enzymes in herbage ingested.

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* If glucoside and enzyme occur in the same plant, free HCN may be present in the plant tissues when these are bruised, cut or eaten, because this mechanical damage permits of free admixture of the cyanogenetic glucoside and the enzyme which acts upon it.

The Amounts Eaten and the Rate of Consumption by the Animal.

As HCN is rapidly excreted by the animal body no detrimental effects will be produced unless the rate of absorption exceeds that of excretion.

For this reason severe mortalities from cyanogenetic plants are more commonly experienced with travelling, or hungry, stock which are more prone to eat large amounts of the plants in a short period of time, thus absorbing prussic acid more rapidly than they are able to eliminate it. Local animals, grazing over the same area, are not so likely to be poisoned because they normally consume smaller amounts over longer periods of time.

Seddon and King (1930) concluded that a plant should be regarded as dangerous if on analysis it is found to yield at least 0.02 per cent HCN (green basis); a sheep would need to eat about 1 lb. of such a plant within about one hour for a fatal result to ensue.

Signs of poisoning, not uncommonly, are shown by animals after drinking.

The elimination of absorbed HCN in sheep was shown by Coop and Blakley **(loc. cit.)** to be due to the exhalation of free HCN in the breath (10 per cent) and to detoxication, mainly in the liver, by conversion into the sulphur-containing thiocyanate. This conversion requires the presence of "sulphur donors" such as cysteine.

In Western Australia serious mortalities in travelling sheep and cattle have resulted from the ingestion of "balsam" **(Euphorbia Drummondii)** and more particularly "Gascoyne Spurge" **(Euphorbia boophthona)** as recorded by Gardner (1942). He attributed the death of 57 bullocks and more than 1,000 sheep on a stock route near Cue, during one week in 1935, to poisoning with **E. boophthona.**

SYMPTOMS

In acute poisoning, following the ingestion of cyanogenetic plants, the symptoms are related to the dose of HCN and to the period of time during which it is taken. The dose is, of course, related to the glucoside content of the plant, the amounts eaten, and to the rapidity of the production of free HCN by suitable enzymes.

This poison acts very quickly. Large amounts cause almost instantaneous death with spasms and respiratory failure. Smaller doses cause accelerated and deepened respiration, an irregular and weak pulse, bright red membranes (the lining of the eyes, nose and mouth), which later turn purplish in colour (cyanosis). There is salivation, frothing at the mouth, muscular twitching and spasms, a staggering gait, dilatation of pupils and pronounced bloat followed by coma and death from respiratory failure; the heart usually continues to beat for some time after breathing has ceased.

Chronic poisoning is described in human beings, but there seems to be considerable doubt about its occurrence in animals.

It has been suggested by a number of workers that bloat (hoven) in sheep and cattle may be due to ingestion of pasture plants, for example white clover, which contain insufficient HCN to cause death or other noticeable symptoms in grazing animals. The mechanism inducing the well known condition of bloat is, however, not yet fully understood.

POST-MORTEM APPEARANCES

The post-mortem appearances of HCN poisoning are not very striking. In very acute cases the blood may be bright red (due to the formation of cyanhaemoglobin). In less acute poisoning it may be very dark (cyanosis). If examined soon after death there is a smell of bitter almonds, particularly evident in the rumen which is also markedly distended with gas.

(For list of cyanogenetic plants see Appendix.)

PLANTS CAUSING PHOTOSENSITIVITY

As a result of eating certain plants stock may become abnormally sensitive to light (photosensitive) so that on exposure to sunlight a more or less severe inflammation of the skin ensues; the areas affected are those which are not well protected by pigment, hair or wool and are most exposed to light. This condition is known as **photosensitization.**

The state of abnormal sensitivity to light has been shown to result from the absorption of certain plant pigments.

A few plants normally contain a specific pigment which is absorbed and cannot be completely eliminated by animals in the processes of digestion. There is then no barrier to this effective agent reaching the skin, through the blood stream, and sensitizing it. Such plants are St. John's wort **(Hypericum perforatum)** and buckwheat **(Fagopyum esculentum)** which for long have been known to cause photosensitization when eaten by animals.

In another type of photosensitization, the sensitizing agent is phylloerythrin, a derivative of chlorophyll, the green colouring matter of plants. In the healthy animal this pigment is readily eliminated from the body. In animals affected with liver disease, however, the chlorophyll derivative may not be excreted or destroyed, and may cause the animal to be photosensitive.

Facial eczema, a serious sheep problem in New Zealand, is a notable example of photosensitization of this type. Here the sensitizing agent is derived from the chlorophyll in rye grass pastures—the cause of the liver damage is undetermined.

We had occasion to investigate, some years ago, an occurrence of photosensitization in sheep at Beverley. The mob affected comprised a small consignment of sheep, recently overlanded by rail from the Eastern States, and grazed together with sheep bred on the property. The pastures contained no plants unusual to the area and cases of photosensitization, several of them fatal, were confined to the imported section of the flock. It was deduced that the photosensitization resulted from failure of liver function in the travelled stock, the sensitizing agent being, presumably, a pigment contained in normal pasture plants.

Photosensitization is not known to have been responsible for any serious stock losses in Western Australia. Trefoil dermatitis has been encountered at times in sheep and cattle grazed on pastures of burr clover **(Medicago denticulata).** It has been recorded in horses affected with Kimberley horse disease and is seen also in some cases of lupinosis in sheep. In both of these conditions marked liver damage is constantly present. Other plants occurring in Western Australia which have been known to cause photosensitization elsewhere are Lantana **(L. camara)** and Caltrop **(Tribulus terrestris).**

In view of the relative unimportance of photosensitization in Western Australia the condition will be dealt with only briefly here. A comprehensive review of the subject has been made by Clare (1952).

SYMPTOMS

General signs are uneasiness and evidence of skin irritation, and the affected animals seek shade. Inflammation of the skin develops only on unpigmented, or white areas.

In cattle, white patches wherever they occur but especially those along the back, are affected; the skin of the udder, the escutcheon and face may also suffer.

In horses white markings of the muzzle, face and lower portions of the limbs are the parts affected.

In sheep the muzzle, face, ears and the lower parts of the legs, uncovered by wool are affected.

In the early stages the skin is hot, red and thickened as a result of the collection of fluid under it. This may result in the affected parts, notably the face and ears of sheep, becoming considerably swollen ("bighead"). If the condition is severe, large areas of skin may die and be shed, or rubbing—due to the irritation—may lead to erosion of skin and scab formation.

Following the absorption of the fluid the skin becomes dry and shrivels, thus causing a marked distortion of the ears, notably in sheep.

Jaundice may be present in cases of photosensitization associated with liver damage.

Recovery frequently follows photosensitization, but serious stock mortalities may result particularly when the plant responsible contains toxic principles in addition to the pigments causing the skin reaction (e.g. the liver damage in facial eczema and poisoning with **Tribulus terrestris**).

POST-MORTEM APPEARANCES

The manifestations of photosensitization are confined to the skin, but where plants responsible contain other toxic principles, internal organs, notably the liver, may be abnormal.

(For list of plants causing or suspected of causing photosensitivity see Appendix III.)

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