

Phalaris Toxicity in Sheep Involves Two Separate Diseases

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Introduction

Phalaris toxicity has been recorded in sheep flocks in New South Wales since at least 1942. During the 1960's several toxic compounds were identified in phalaris plants and collectively referred to as tryptamine alkaloids.

It was suggested at that time that these poisons were responsible for three supposedly related phalaris diseases called Peracute Phalaris Toxicity ("Sudden Death"), Acute Phalaris Toxicity and Chronic Phalaris Toxicity ("Staggers"). Subsequently, attempts were made to breed varieties of phalaris plants which produced much smaller amounts of these alkaloids (eg. "Sirolan") compared to the original phalaris varieties (eg. "Australian"). However, phalaris toxicity has continued to be a problem in sheep grazing phalaris dominant pastures regardless of the phalaris variety being grazed.

Research at AR & VC, Orange

The results of recent research work carried out at the Agricultural Research and Veterinary Centre, Orange have thrown new light onto the phalaris problem. This work has shown that there are only two phalaris diseases, a sudden death disease and a nervous disease. These two diseases are not related. The plant poison that causes the sudden death disease remains unknown but the available evidence suggests that it may be a cyanide compound. The plant poisons that cause the nervous disease are a mixture of tryptamine and beta carboline alkaloids. Reducing the levels of tryptamine alkaloids in the plant has not stopped the nervous disease. Instead it has delayed the speed with which the disease develops and increased the number of cases that occur weeks or months after phalaris grazing has ceased. This is probably because the

breeding of cultivars with less tryptamine content has favoured an increase in beta carboline content.

Sudden Death Disease

The sudden death disease is easily recognised, it involves the death of sheep within 12 to 24 hours of being moved onto a fresh phalaris pasture. The number of affected sheep is often quite large and frequently there is a history of prior transportation of the mob hence feed and water deprivation for a day or so beforehand. Alternatively, there may be a history of rotationally grazing the mob, with the episode of sudden death occurring when the sheep have been moved off a bare paddock and put onto a fresh one. Drying out of short phalaris growth or frosting of short phalaris growth seems to increase the risk of this disease and the autumn period is probably the highest season of risk. The best way to prevent this disease is to make sure that mobs of sheep are not hungry when first put onto a fresh phalaris pasture. Sheep will adapt to the phalaris poison during the first 48 hours of grazing, and thereafter, if left undisturbed, seem to be able to successfully detoxify the substance. Sending dogs around the mob, or disturbing them in other ways, will cause any marginally affected sheep to suddenly collapse, these animals display an abnormal heart beat, disturbed breathing and darkening of the skin around the gums and eyelids. If left to lie quietly by themselves, some will quickly recover.

Nervous Disease

The nervous disease is reasonably easy to recognise, but it may sometimes be confused with the nervous disease caused by the fungal poison in ryegrass. Individual sheep affected by the nervous disease may display many different nervous signs and may be affected for highly variable lengths of time, ranging from days to weeks to months. The most common sign is twitches of the skin of the face and ears together with fine tremors (or shakes) of the head. The animal may have a proppy gait, it may be weak in the legs and refuse to travel, some sheep display bounding, hopping or jumping movements, whilst others tend to go down onto their knees, or walk on their knees, some sheep develop tremors (or shakes) of the body, others may knuckle over at the fetlock joints. Sheep that collapse may struggle vigorously to stand up again and if helped to their feet are often able to bound away and

rejoin the mob. Many sheep affected by the nervous disease will eventually die as a result of it, however, sheep that are only mildly affected, if moved off the phalaris and left undisturbed, will return to normal over the ensuing weeks. One of the more confusing aspects of the nervous disease is the fact that sheep can appear to be perfectly normal when they are removed from phalaris pastures and yet still develop nervous signs several weeks or months later.

Use of Cobalt to Prevent Sudden Death Disease

It is possible to prevent the development of the nervous disease by the oral administration of long acting cobalt pellet **before** sheep are grazed on phalaris paddocks. However, the greater the level of alkaloid in the plant, the greater the amount of cobalt required. In some districts there is already a sufficient trace element mixture in the phalaris plants to allow

the sheep's stomach to detoxify the phalaris poisons. Phalaris growing on the red basalt soils of the Orange Plateau in central New South Wales is a good example. However, in adjacent districts such as Molong, where the soil type changes, the trace element mixture in the phalaris is different and the risk of the nervous disease is increased.

In situations in New South Wales where the nervous disease is known to occur, the oral administration of a cobalt pellet to each sheep, each year prior to their being depastured on phalaris dominant paddocks should suffice. The addition of cobalt to fertiliser pasture topdressings, or the use of cobalt in lick blocks, are not recommended approaches to the prevention of the nervous disease, because they are not reliable methods of ensuring that all sheep in the mob have sufficient cobalt in their stomachs all of the time when they are grazing phalaris pastures. The use of cobalt in any form does not offer any protection against the sudden death disease.