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NEWSLETTER

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It has been a pleasure to note that this year prices for lamb and beef cattle are still good and that even the price of wool continues to improve – not before time. We hope by the time you receive this that the autumn break rains will have fallen and that our members will have a considerably less worrying winter than those endured lately.

Lately I have been the recipient of "Focus on Salt" which is a record of the considerable research and producer effort that has gone into overcoming the problems of salinity. Although it has been clearly shown that woodland is the most assured control of salinity, research has shown that perennial pastures are a very good answer to salinity. One of our members (and contributors) Dr. Brian Dear has been heavily involved in overcoming salinity in the Murray Darling Basin. He is also involved in breeding a productive acid-tolerant lucerne for salt-affected areas.

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that allows those family farmers and their communities to remain financially and socially viable".

There was an interesting article on pages 8-11 of The Land on March 16 in which Bruce Stannard warns that the environmental lobby has built up an inner city political support base by seeking out some environmental issues in rural areas. Dr Ray Johnson (CEO of NSW Farmers Association) states "— it is important that farmers have an ongoing and constructive dialogue with many environmental groups". A difficult task but it is important for members to convince urban groups that our members care every bit as much for the environment as anyone.

Your committee looks forward to again meeting many of our members at our annual conference in Wagga Wagga on July 25-27 (note the later date than usual). Dr Belinda Hackney and her committee have been working hard putting the programme together and we are grateful for their efforts.

Haydn Lloyd-Davies
Editor

PHALARIS POISONING SYNDROMES

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Phalaris aquatica with its numerous cultivars is a much-valued perennial grass species widely used in improved pastures across south-eastern Australia. However in certain circumstances, it does have the potential to become a toxic pasture plant, producing a variety of unrelated syndromes which manifest either as neurological or cardiac disturbances, presumably involving different toxins. The poisonous potential of *Phalaris aquatica* is dynamic and is a function of interacting plant, animal, environmental and management factors. Currently it is generally accepted that there are three distinct syndromes: chronic phalaris staggers, cardiac sudden death and 'PE (polioencephalomalacia)-like' sudden death, although recent evidence suggests that PE is not involved in the latter syndrome and a urea cycle disorder has been proposed.

Chronic phalaris staggers

This neurological syndrome results from the repeated or protracted ingestion of methylated tryptamine alkaloids present in *P.aquatica*. The compound accumulates in the CNS to directly interact with serotonergenic receptors in the motor and sensory nerve nuclei of the brain and spinal cord. This causes a functional rather than structural nervous derangement, which is demonstrated by the clinical signs being precipitated with disturbance of the flock. Animals are paretic, ataxic, have a generalised muscle tremor including head nodding and jaw champing. They display incoordination and proprioceptive deficits with frequent falling over. Some lack the ability to rise and may appear hyperaesthetic and struggle when approached. Knee-walking is frequently seen and the animals may 'bunny hop'. Cardiorespiratory signs can be seen with the nervous forms of intoxication, probably due to the increased effort and strain on the cardiovascular system due to the nervous incoordination, rather than any direct effect of the toxin on myocardial function The affected animals remain conscious throughout, however if recumbent for a prolonged period, may become comatose and develop cerebral convulsions. Death or recovery can occur over the ensuing weeks or months, depending on the chronicity of ingestion and the severity of clinical signs. Clinical signs can develop as soon as 1-3 weeks following the introduction to the pasture especially with the older, high tryptamine cultivars. However, with

the new, low tryptamine varieties such as Sirolan, much longer periods of grazing (3-4 months) may be needed to induce staggers (Bourke et al 2003) plus a delay in development of clinical signs can occur even after being removed from the incriminating pasture, with cases developing up to 3-4 months later.

Gross pathology may reveal a green-grey discolouration of the lateral geniculate body in the brain and brainstem, with this discolouration also sometimes seen in the renal medulla. Characteristic histopathological lesions include intracytoplasmic brown pigment granules in the nerve cell bodies of the brain sections, being most concentrated in the lateral geniculate body. Wallerian degeneration may also be seen associated with the white matter (axons) of the brain and spinal cord. These lesions can usually only be detected in cases greater than several weeks duration (Bourke et al 1988).

There is no effective treatment, but animals should be immediately moved to phalaris-free pastures. Protection against this form of intoxication via intraruminal Cobalt bullets has proven protective as ruminants are able to detoxify the toxin when intraruminal Cobalt (Co) levels are high enough to match the toxic challenge. It is advised that two bullets are given to prevent a calcium carbonate coating building up around the bullet, which would decrease effective absorption of Co. Intraruminal grinders are also available for this purpose. Two bullets should be given every three years. Alternatively, top dressing the pasture with Co or individually drenching each sheep so a minimum of 28mg per head per week is given will allow potentially toxic pasture to be grazed with no adverse consequences (Blood et al 2000).

Acute Cardiac Sudden Death Syndrome

The cardiac from of sudden death form on phalaris pastures involves a sudden onset of a cardiorespiratory disorder without neurological signs. The toxin responsible is unknown, although it is considered that ruminants are able to detoxify this toxin provided it is not ingested too rapidly or in excess (Bourke et al 1988). To produce the signs seen, the toxin must act either on the cardiorespiratory centres in the medulla oblongata or on the vagal nerve endings as they innervate the heart. Toxic levels of cyanide (20mg or greater/100g of hydrocyanic acid) have been measured in phalaris plants from toxic pastures (Bourke & Carrigan 1992), thus a cyanogenic poison has been investigated. Nitrate compounds have also been postulated as the causative agent as it has been documented that phalaris pastures can attain nitrate nitrogen concentrations >2920µg/g, with the potentially toxic concentration for sheep only 1000µg/g (Bourke & Carrigan 1988). It has also been noted that the incidence of this form

of phalaris sudden death may be associated with seasonal increases of N-methyltryamine in *P.aquatica* (Bourke et al 2003).

Outbreaks can occur as soon as 24 hours following introduction to the pasture, however in some reports sheep had been grazing the toxic pastures for 2 weeks before outbreaks occurred. The clinical course of the disease ranges from minutes to hours; clinical signs being induced by flock disturbance or when the animals are forced to exert themselves. Cardiac disturbances include ventricular fibrillation and cardiac arrest, followed by syncope. The animals suffer from respiratory distress, their mucous membranes becoming cyanotic. Most affected sheep die, however some may spontaneously recover. As mentioned, no nervous signs are seen with this form of phalaris poisoning, nor are there any obvious gross or histopathological lesions. The prevalence is usually about 1%, being much lower than seen with cases of PE-like sudden death (Bourke & Carrigan 1992).

Again there is no treatment and stock should be removed immediately from the paddock with as little stress as possible to avoid eliciting further mortalities. Once moved, there should be no more new cases. Intraruminal Co administration is not preventative for these cases. The incidence of cardiac sudden death syndrome does appear to be greatest during the first few months of new growth, typically autumn to early winter (Bourke & Carrigan 1992): thus it is wise avoid grazing phalaris dominant pastures during this period.

Peracute PE-like sudden death

'PE-like sudden death' involves an acute onset of neurological signs and death that differ greatly from those of phalaris staggers. The animals display ataxia, decreased awareness, cerebral blindness, aimless walking and head pressing and often die in and episode of cerebral convulsions with opisthotonos. No disturbance is needed to precipitate the clinical signs. The greatest mortalities occur within 48 hours following the introduction to the pasture, with the highest incidence of disease seen during autumn through to late winter. The noxious pasture is only poisonous for several weeks during this season though.

As with cardiac sudden death, the toxin responsible for this condition is unknown. Suggestions include agents known to produce thiamine-deficient PE in sheep such as thiamine antagonists (thiaminases) or amine co-substrates. A pyridoxine antagonist has also been suspected. However more recently a mechanism involving hyperammonaemia due to the causative toxin interfering with the urea cycle has been proposed. This postulated pathogenesis simulates citrullinaemia

seen in Holstein-Friesian calves and was initially suspected because of the identical histopathological lesions seen in sections of cerebral cortex submitted from Citrullinaemia (Harper et al, 1986) and PE-like phalaris sudden death cases. The lesion seen is diffuse spongiform change involving astrocytes and sparing neurones, the latter being affected in thiamine-deficient PE. Elevated levels of ammonia levels in aqueous humor of these cases is similar to that seen in plasma in Citrullinaemia, suggesting compromise of the urea cycle in PE-like phalaris sudden death.

There is no treatment or consistent method of preventing outbreaks of 'PE-like' sudden death. Investigations into prevention have included prophylactic administration of thiamine and pyridoxine. This was based on the idea that the causative toxin, as mentioned above could be some form of thiamine or pyridoxine antagonist. The study in question failed to demonstrate any protective effect of these substances, however did not completely dismiss the possibility of their use for prophylaxis. This was based on a number of reasons outlined in the paper such as the rate of action of the toxic antagonistic agent was too rapid for the dose administered of the prophylactic agent (Bourke et al 2003).

'PE-like' sudden death outbreaks occur more commonly when hungry stock are put on phalaris dominant pastures that have been spelled or involved in rotational grazing where an abundance of new shoots has been available. The toxic potential of phalaris pastures also seems to increase when rain has followed a period of moisture stress. Therefore it is advised that the phalaris pastures are continuously grazed or set-stocked to keep the new growth during autumn/winter to a minimum, and that hungry sheep should not be placed on previously spelled phalaris dominant pastures, especially not following periods of moisture stress or heavy frosts. From autumn through to late winter it may be wise to test the toxic potential of a paddock by placing a group of sentinel sheep onto the paddock 48 hours before the entire flock is given free access. If no clinical cases have been seen within this time, the pasture is generally considered safe, and it is assumed that the animals can adequately adapt to the toxic challenge.

Epidemiology of phalaris toxicity

The poisonous potential of phalaris pastures is dynamic. It has been proven that the level of noxious alkaloids responsible for the chronic staggers syndrome are increased during certain periods, this being influenced by interacting plant, animal and environmental factors. As the toxins responsible for the other conditions remain unknown, there has been speculation on associations between increased incidence of outbreaks and these interacting factors. Increased alkaloid content in

the foliage of *P.aquatica* has been measured during periods of moisture stress, frost conditions and decreased light intensity, such as overcast weather or shading. Fertile soils such as those nitrogen-enriched with leguminous plants, or fertilised with superphosphate have also been found to have higher levels of the tryptamine alkaloids. New shoots are also more concentrated sources of the toxic alkaloid, with poisonous potential of the pasture rapidly declining after it has reached a certain height. The new cultivars such as Sirolan and Sirosa are lower alkaloid strains than older varieties such as Holdfast. Other potential risk factors include s the soil type, with limestone soils inherently low in cobalt and associated with increased incidence of phalaris staggers. Basaltic soils are high in cobalt and hence staggers is not common in areas where these soils dominate. It appears that animals have the ability to adapt to the toxic agent across the spectrum of disease syndromes. Animals that are newly introduced to phalaris and those with alterations in feed intake, as occurs in cell grazing systems, are considered at greater risk of intoxication.

Control or Prevention

Consideration of these risk factors suggests that producers should aim to avoid putting hungry stock on freshly-shooting phalaris dominant pastures, especially following periods of frosts or moisture stress. If the stock have been transported or yarded for a period of time without access to food, they should be fed before being placed on the pasture. Continuously grazing or set-stocking pastures to keep new growth at a minimum especially during the autumn and winter months may assist. Intraruminal cobalt bullets are also an effective measure to protect against Phalaris staggers, and allow potentially toxic pastures to be utilised and grazed. It is important to remember however that they serve no purpose in the prevention of the other forms of toxicosis. As the phytotoxins responsible for the acute poisonings are yet to be identified the only way to prevent the occurrence of acute intoxication is to adhere to the management strategies that have been proven to be sound over many years.

References

Blood DC, Gay CC, Hinchcliff KW & Radostits OM *Veterinary Medicine: A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats and Horses,* 9th Ed, W. B. Saunders, London 2000 '*Diseases caused by major phytoxins'* pg 1652-1653.

Bourke CA & Carrigan MJ (1992) Mechanisms underlying Phalaris aquatica 'sudden death' syndrome in sheep. *Australian Veterinary Journal* 69:165-167.

Bourke CA, Carrigan MJ, & Dixon RJ (1998) Experimental evidence that tryptamine alkaloids do not cause Phalaris aquatica sudden death syndrome. *Australian Veterinary Journal* 65:218-220.

Bourke CA, Colegate SM, Rendell D (2003) *Efficacy of the prophylactic use of thiamine and pyridoxine in sheep during an outbreak of Phalaris aquatica 'Polioencephalomalacia-like sudden death' poisoning. Australian Veterinary Journal* 81:637-638.

Bourke CA, Colegate SM & Rendell D (2003) Clinical observations and differentiation of the peracute Phalaris aquatica poisoning syndrome in sheep known as 'Polioencephalomalacia-like sudden death'. *Australian Veterinary Journal* 81:698-700.

Healy PJ, Harper PAW & Dennis JA (1990) Bovine citrullinaemia: a clinical, pathological, biochemical and genetic study. *Australian Veterinary Journal* 67: 255-258.

GRASSLAND SOCIETY OF NSW
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TALL FESCUE ENDOPHYTES – TOXIC OR TERRIFIC

A summary of a talk presented to ASAP & NSW Farmers in November 2005

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The word endophyte simply means within the plant (endo=within, phyte=plant). Therefore an endophyte is an organism (in this case a fungi) that lives in the plant. The endophytic fungi live between the plant cells of many grass species. The endophyte is not visible to the naked eye and can only be detected via a microscope or other laboratory methods

The endophyte and the grass plant have a symbiotic relationship where the plant provides the endophyte with a place to live, nutrients to grow and a means of dispersal through the plants seed. There are a number of reported benefits to the plant including, reduced insect attack, improved plant growth, increased persistence, better drought tolerance and improved nutrient uptake. However, grazing endophyte infected tall fescue pasture may cause animal health problems and production losses.

The endophytic fungi associated with tall fescue are *Neotyphodium coenophailum*. The life cycle of the tall fescue endophytic fungi is relatively simple and follows the growth and development of the host plant. The fungus is present in the seed and as the seed germinates the fungus begins to grow extending into the young shoot by growing through the space between plant cells. During the vegetative growth phase the fungus is located in crown, leaf sheaths and shoot apexes. As reproduction of the plant approaches the fungus grows into the flower stem and ultimately invades the plants ovaries and ovules where the fungus becomes encapsulated in the seed.

The endophytes produce chemicals called alkaloids that effect animals and insects. Not all endophyte-grass combinations produce the same range of alkaloids, and concentrations of alkaloids vary with season, part of the plant, water & nitrogen levels. The same endophyte can produce differing levels of alkaloids in separate cultivars. All alkaloids increase markedly from mid spring to greatest concentrations over summer and autumn. The three main alkaloids isolated from tall fescue endophyte are Ergovaline, Peramine and Loline. Ergovaline is an Ergopeptide alkaloid associated with animal disorders. It is also known to deter insect feeding. Peramine is a Guanidinium alkaloid, known to act

as a feeding deterrent to insects. Loline, also known as Festucine acts as a feeding deterrent to insects.

Ingestion of endophyte infected tall fescue pastures has been associated with a number of animal conditions as a result of the toxic alkaloids. One condition is summer ill-thrift, also called fescue toxicosis or summer slump. This condition occurs over summer when ergovaline levels are their highest. The condition is characterised by unthrifty appearance and poor production over summer. Symptoms include elevated body temperatures, excessive salivation, and rough coat hair. As a result of the increased body temperatures the animal seek shade and water to wallow in to cool themselves and spend less time grazing leading to reduced feed intake and reduced weight gains. The condition is also associated with mild inflammation of the intestines, diarrhoea, increased presence of dags and reproductive problems

The other major animal condition associated with tall fescue endophyte is fescue foot. Fescue foot is a highly visible disorder and is a dry gangrenous condition most often associated with lameness and the loss of ear tips and tail tips. In acute situations the feet, hooves or entire limb can drop off. Fescue foot usually occurs in cattle grazing tall fescue in the cooler seasons.

In Australia the first documented case of fescue toxicity appeared in the Australian Veterinary Journal in 1950 – the case was of lameness in cattle grazing tall fescue in SA. There have been additional sporadic anecdotal reports of tall fescue toxicity in NSW, Vic and SA. However, there have been very few documented/scientifically analysed cases and no documented cases in Australia since 1960. This is largely due to the fact that the current tall fescue varieties of tall fescue available in Australia are free of wild type endophyte. Older varieties (pre the release of Demeter) e.g. Alta, Fawn or Kentucy-31 may contain high levels of endophyte so in some areas with a long history of tall fescue use such as the Northern Tablelands there is potential for some endophyte problems. It is important to note that all tall fescue turf varieties contain endophyte.
In collaboration with the Bozo Landcare group (near Uralla NSW), Dr Lewis Kahn, Jeff Lowien and the author received funding from MLA to survey pastures on the Northern Tablelands of NSW to determine the presence and level of toxic endophyte alkaloids in pastures across the year.

The survey had 2 components; perennial ryegrass and tall fescue. In this article only the data from the tall fescue component is discussed.

Table 1. Ergovaline levels (ppm) in tall fescue samples and mixed pasture samples collected from 6 properties in the northern New England areas of NSW.

Property	Tall Fescue Samples			Mixed Pasture Samples		
	Nov 03	Mar 04	May 04	Nov 03	Mar 04	May 04
1	ND	ND	ND	ND	ND	ND
2	ND	ND	ND	ND	ND	ND
3	ND	ND	ND	ND	ND	ND
4	0.6	<0.1	ND	<0.1	ND	<0.1
5	<0.1	NS	NS	0.2	NS	NS
6	ND	NS	NS	ND	NS	NS

ND=not detected, NS=not sampled

Clinical "threshold" for ergovaline
Sheep: 0.8-1.2 ppm, Cattle: 0.4-0.8 ppm, Horses: 0.4-0.6 ppm

Table 1 shows the levels of ergovaline detected in tall fescue samples and mixed pasture samples between Ben Lomond and Liston (near the Qld border). As you can see there was no ergovaline detected at 4 of the 6 sites. At one of the sites – property 4 there was relatively high levels of ergovaline at 1 sampling. This property has a long history of tall fescue use dating back to the early 1900's including use of Alta etc so potentially tall fescue sampled was this older variety.

In the USA, the tall fescue endophyte issue is quiet a different situation to Australia. There are approximately 14 million ha of tall fescue based pastures in USA particularly in the humid areas of eastern USA. The majority of this fescue is Kentucky-31 a locally adapted ecotype that was selected and released with much success in the 1940s. Although this tall fescue cultivar established and persisted successfully the livestock production was not as expected and there were a number of losses to the fescue foot condition. With the discovery of tall fescue endophyte in the 1970s wide-spread testing detected that over 90% of the tall fescue pastures contained wild-type endophyte with a 75% mean level of infection. It is estimated that the cost to the beef industry alone in the USA of the tall endophyte is over 600 M per annum

Due to the large losses experienced in the USA and other countries due to wild-type endophyte several research organisations and seed companies have been

working on a solution. They have developed 'novel' or safe" endophytes that produce lower or nil levels of the toxic alkaloids like ergovaline but still produce 'plant friendly' alkaloids such as peramine. As these endophytes produce low or nil levels of the toxic alkaloids they are safer to the grazing animal. In Australia the 'safe endophyte' technology is traded as MaxP and is available in 5 tall fescue varieties i.e. Advance MaxP, Jesup MaxP, Quantum MaxP, Flecha MaxP and Resolute MaxP.

Safe endophyte tall fescue varieties are relatively new to NSW and as such, there is limited data available on the performance and persistence of these varieties in NSW. However, early data indicates that the Mediterranean safe endophyte varieties have the potential to extend the zone of adaptation of tall fescue into lower more marginal rainfall areas. By contrast, temperate safe endophyte varieties are not showing the same potential for improved production and persistence in high rainfall areas.

FEED QUALITY PROVES A CHALLENGE

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A Quick Quiz

Anyone who feeds animals is concerned with feed quality. Indeed many of us are professionals who make our living by feeding animals but how well do we understand an animals feed requirements and do we know the quality of feed we provide?

During 2005 the Topfodder silage team prepared a small quiz asking farmers and advisors to match a series of 6 feed samples with their feed test results.

The quiz was presented at the 2005 NSW Grasslands Society annual conference as well as many field days including Tocal, Mudgee, Henty and Orange .

The samples were 2 silages, 2 hays, barley grain and white cottonseed, each very different and typical of their type.

Standard test results showed dry matter %, crude protein, metabolisable energy, digestibility and fibre (NDF) for each sample And in one special case % fat. The challenge was to match each sample with its test results - surely a simple ask for professional farmers and advisors.

The quiz proved to be more of a challenge than expected. From the thousands of people who viewed the samples we had 247 enter the quiz and 33 were correct. Another 20 correct answers were received from DPI staff or after some extra 'help' - in total 20% of entrants could match 6 very different feed samples with their feed test results.

So what is the answer?

The six feeds were a ryegrass silage cut when leafy, before heading, an oaten silage showing seed heads (flowering), a wheaten hay (no grain in heads), a lucerne hay, barley grain and white (fuzzy) cotton seed. The test results were:

Sample	Dry Matter %	Crude Protein %	Metabolisable Energy (ME)	Digestibility %	NDF %
A	88.8	10.2	12.8	86.7	(ADF) 5.1
B	47.3	21.8	11.0	75.3	46.1
C	91.9	21.1	13.0	65.0	(%Fat 20.6)
D	90.2	11.8	8.1	57.3	66.3
E	44.4	10.4	9.4	65.1	54.0
F	89.4	20.4	9.7	67.2	39.3

By knowing the basics of feed quality and thinking about all the numbers provided we quickly get to some simple choices.

First look at the dry matter % samples - B and E are much wetter than the others therefore must be the silages. Looking at the ME and crude protein we see that sample B is high in ME and protein and is the high quality leafy ryegrass silage. Sample E is higher in fibre, lower in energy and protein which is consistent with lots of stem and seed head in a cereal (oats). The other 4 samples have similar dry matter % so look at other features.

Samples A and C have very high metabolisable energy (ME) compared with D and F so they are the grain and cottonseed. Sample A has the lower protein, low

fibre and very high digestibility. A is the barley grain. Sample C had high protein and a fat level of 20.6% (extra hint) which is typical of white cottonseed.

This leaves D and F as the hays and it should be no surprise that sample F with 20.4% crude protein was the lucerne hay.

Who is correct?
It was pleasing to see that the most correct answers were received at the Grasslands Conference where 33% of entries, i.e. 12 out of 36, were correct . . . but what about the other couple of hundred at the conference???

People who had completed a Prograze course were also better than average at the quiz with up to 40% Prograze people correct in some groups.

We were very pleased to see that farmers who had completed the Topfodder silage course were also more confident with basic animal nutrition with up to 75% of Topfodder silage participants correct.

So what does it mean?
Our little quiz has raised quite a few questions which professional farmers, graziers and advisors should consider.

How much money is being spent or profits lost by not understanding basic animal nutrition and feed quality (in our Topfodder silage program we have a small exercise which shows that most enterprises can increase income by $10,000's by improving the quality of fodder they use)?

Some people have very little idea of feed quality and what it looks like. One of the most common 'confusions' were people mixing up the test results for wheaten hay (D) and barley grain (A) . . . two very different feed types!

Does feed testing matter? Does the old saying *"you can't improve what you can't measure"* still apply?

Obviously most farmers and advisors are not comfortable using feed test results. Is it enough just to know the basic effect on feed quality of plant species, growth stage etc. or could more profits be made by a better understanding of the quality of what we are feeding and what the feed requirements of our animals are?

Can members of the Grasslands Society help answer these questions or come up with other ideas. All suggestions would be welcome.

PERENNIAL RYEGRASS ENDOPHYTES: Effects on Dairy Cattle

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Introduction

Plant toxicoses represent a challenge for producers, researchers and clinicians. Effects on dairy cattle of the large group of potential toxins associated with the ryegrass endophyte *Neotyphodium lolii* are particularly challenging to evaluate. There are a number of excellent reviews of the effects of ryegrass endophyte on cattle and sheep, however, the complex interactions between plant cultivar and endophyte do not readily allow extrapolation of research findings to field situations and vice-versa.

Production of toxins is greatest when plants are stressed and in summer. There are seasonal changes in the nutritional value and environment that confound field observations of effects of ryegrass toxins on production in cattle. Specifically, ryegrass nutritional quality is poor at the same time as toxin levels are highest.

Further, there are possible effects of endophyte toxins and pasture sward composition, specifically clover content and grazing patterns, that make it difficult to measure the effect of specific toxins. Lastly, the role of prolactin (PRL) in cattle in milk production is largely confined to periods during growth and just before calving. Ergovaline acts to decrease PRL concentrations and this may have very substantial effects on production of susceptible cattle. However, such effects will be difficult to detect in the field. Despite these difficulties, there is ample evidence that infection of ryegrass with specific strains of *Neotyphodium lolii* has the potential to be one of the most significant factors reducing dairy production in Australia.

This paper provides a review of evidence supporting observed and suggested effects of *Neotyphodium lolii* on dairy cattle. Problems that impair effective estimation of the economic and health effects of ryegrass endophyte are discussed and opinion is provided on the possible magnitude of the toxicosis.

Table 1 provides a summary of all papers that provide information on milk production responses of dairy cattle to endophytes.

Table 1. Milk production responses reported in studies reporting on the effects of ryegrass endophyte on dairy cattle.

Trial and year	**Milk or Milk Solids Production Low E (N)**	**Milk or Milk Solids Production High E (N)**	**SD – pooled**	**Comments on design**
Valentine et al 1993 Expt 1	21.2 +5% milk production (12)	20.2 (12)	0.4	Australia: Ellet ryegrass with high or nil endophyte – November
Valentine et al 1993 Expt 2	17.7 +14% milk production (12)	15.5 (12)	0.4	Australia: Ellet ryegrass with high or nil endophyte – March
Clark et al 1996 Expt 1	+10.3%, production ~ 8-10 litres, solids <1 kg (40)	- (40)	-	NZ: Short term study – clover free grass and silage, significantly differed, high EV also significantly increased body temperature
Clark et al 1996 Expt 2	+ 2.7% Milk Solids (40)	- (40)	-	NZ: Clover present in grass and silage, short term, not significant
Keogh et al 1999	+23% Milk Solids, production ~ 20 litres (16)	- (16)	-	Northland NZ, period October to Mid April – not control matched pasture, significantly differed, temperature and humidity and time interaction with grass type 1997-8
Keogh and Blackwell 2001	NA	NA		Extension of Keogh et al 1999, but a systems approach that modified grazing and other systems. This resulted in significantly more production in the low endophyte group, but is potentially biased in 1999-2000

Thom et al 2001 Year 1	1.01 Milk Solids, - 3.9%	1.05	0.03	NZ: Comparison is between existing high endophyte pasture and new low endophyte mixed sward
Year 2	0.96 Milk Solids, + 35.2%	0.71	0.03	
Year 3	0.94 Milk Solids, + 16%	0.81	0.02	
Year 4	1.08 Milk Solids, +2.8%	1.05	0.02	
Lean 2001	31.2 L +14.5% (80)	26.5 L (80)	0.46 L 1.75 H	Australia: Observational study, historical controls, silage feeding
Bluett et al 2005	+6.7% Milk Yield, production range between 20 to 7 litres per day (15)	-	-	NZ: Comparison with AR1 and nil endophyte, significant difference pooled over 3 periods

Discussion

The papers identified in Table 1 have significant flaws in demonstrating a causal relationship between ryegrass endophyte and lower milk production. These flaws include, confounding between pasture composition and outcome. Specifically, pasture swards used in the studies were not identical except for the presence of *Neotyphodium lolii*. Often swards differed in botanical composition (Table 1), especially clover content that would have altered digestibility and feed value of dry matter intake. Milk production generally increases in association with increased clover content of pasture. Researchers and labour used in projects were not blinded from group allocation, i.e. they were aware which pasture was which. A lack of documented randomization of cattle and internal replication of pastures means that the observations were more like a comparison than a truly randomized study.

Nonetheless, papers in Table 1 provide significant evidence of associations between the presence of grass infected with *Neotyphodium lolii,* especially wild type variants of the fungus, and decreased milk production. There are, despite weaknesses present, a number of strengths of evidence also present in these data. There is also evidence of an underlying biological cause for clinical signs observed. Physiological mechanisms by which *Neotyphodium lolii* infection and specifically ergovaline, may decrease milk production, that is by vasoconstriction (constriction of blood flow) and lowered PRL concentrations have been demonstrated in randomized controlled studies.

Fletcher (1998) suggested that concentrations of ergovaline that exceed 0.5 Φg per gm of dry matter can have marked effects on the health and productivity of livestock. However, conclusions on a dose-response seem premature in the context of the number of potentially adverse effects of ergovaline and the sensitivity of cattle at different stages of development to the ergopeptine alkaloid. It is possible that doses < 0.5 Φg per gm of dry matter may have a negative effect in some sensitive cattle, for example heifers or cows before calving.

Milk production is strongly influenced by mammary blood flow (Bequette et al 1998). The action of ergovaline as a vasoconstrictor, therefore, may influence milk production by reducing blood flow to the mammary gland and splanchnic bed, and by increasing heat stress. As important to the industry is the potential for ergovaline, a dopamine agonist, to decrease PRL concentrations. Prolactin does not play an important role in maintenance of lactation in dairy cattle, but is a major mediator of mammary tissue differentiation and development before lactation. In my opinion, the potential for ergovaline from ryegrass to influence mammary development and reduce milk production potential in autumn calving cows, in spring calving cattle fed silage and in growing heifers is very substantial and may explain some of the lower milk production observed Australasia. At present, it is impossible to determine the amount of loss, because critical studies are not available.

There is a vast array of potential toxins, including neurotoxic alkaloids that may be present in ryegrass infected with *Neotyphodium lolii* (Lane 1999). One of the challenges in researching this area is that the presence of a causal agent may not always demonstrated during a study (eg Auldist and Thom 2000 Table 2). Further, it can be argued that causal agents may be present that have either not yet been identified in the example of neurological disorders and immune suppression or have not been, to date, associated with specific conditions observed in cattle. One example is the lysergyl group alkaloids that could influence behaviour of cows.

Table 2

Potential for Negative Effect	**Causal link**	**Trial Evidence (Strength of evidence)**	**Assessment of evidence**
Heat stress	Ergovaline – Vasoconstriction, decreased plasma prolactin	Easton et al 1996 W, Keogh et al 1999 M, Stuedemann and Thompson review SA, Easton and Couchman 1999 M, Browning Jr and Leite-Browning M	Strong physiological evidence from many sources, Field data moderate with increased respiratory rates and salivation (eg Easton and Couchman 1999), injections of ergotamine and ergonovine cause symptoms
lower milk production	Ergovaline – Vasoconstriction, decreased plasma prolactin	Valentine et al 1993 M Clark et al 1996 expt 1 S, exp 2 M, Lean 2001 W, Thom et al 2001 W, Keogh et al 1999 W, Bluett et al 2005 M, Hannah et al al MA 1990	Moderate evidence is available that milk production is decreased. Physiological evidence suggests that cattle could be susceptible to PRL related under-development of mammary gland, decreased fibre digestion
lower weight gain	Ergovaline – Vasoconstriction, Lolitrem B	Bluett et al 2005 M, Stuedemann and Thompson review SA, Hamilton-Manns and Crothers W, Easton and Couchman 1999 M	Bluett et al 2005 found no significant difference in heifers, Bulls and heifers lost weight (Hamilton-Mann and Crothers 1999), Moderate by analogy with strong sheep ryegrass and fescue studies on cattle

Increased somatic cell count	Ergovaline – Vasoconstriction, decreased plasma prolactin	Lean 2001 W, Auldist and Thom 2000 W against an effect	There were low levels of ergovaline in the trial of Auldist and Thom 2000 that found no effect of endophyte
Reduced clover content	Possible phytotoxin release	Cunningham et al 1993 M, Keogh and Blackwell 2001 W	
Scouring	Ergopeptine alkaloids? Eg ergotamine, Paxilline and Lolitrem B	Fletcher 1993 SA, McLeay and Smith 1999 S, Fletcher et al, 1999 MA, Hannah et al al MA 1990	Sheep studies and basic science showing smooth muscle effects of Praxilline and Lolitrem B, also changes in rumen outflow rates and decreased fibre digestion
Fat Necrosis	Vasoconstriction	Found with fescue toxicosis Stuedemann and Thompson review WA	
Selective Ingestion of pasture	Lolitrem B, ergovaline? Other compounds	Cosgrove et al 2002, MA Field observation (Lean unpublished) W	Mouse studies show feed rejection potential of lolitrem B (Munday- Finch and Garthwaite 1999)
Lameness	Selective eating, ergovaline vasoconstriction	Field observation (Lean unpublished) W	Acidosis with altered forage: concentrate intake – New South Wales, South Australia, Victoria, possible ‘fescue foot’.
Polioencephalo-malacia	Alkaloid Thiaminases	Dougherty et al 1991 WA	Thiamin treatment alleviated signs of clinical toxicosis (fescue)

Reproductive Performance	Ergovaline	Burke et al 2001 WA, Porter 1995, Westwood and Norriss (2000) W	Decreased reproductive performance with heat stress. Altered ovarian function in beef. Dairy study (Westwood and Norriss 2000) in different cultivars
Reduced Immune Function	Lower prolactin concentrations		
Central Nervous Disorders	Lolitrem and other lolium alkaloids eg ergine	Easton 1999, Lane et al 1999, Field observation (Lean unpublished) W, Fletcher et al 1999 SA	Mouse studies confirm neurological effects of lolitrem B (Munday- Finch and Garthwaite 1999)

Strength of evidence code: W – weak, includes analogy from Fescue studies; M – moderate; S – strong; A - analogy

While a comprehensive review of other putative effects of endophyte toxins was not conducted, there is ample evidence of effects of the toxins on health (Table 2). Strong evidence, from many sources, is available on the effects of Lolium alkaloids on neurologic disorders and especially grass staggers. There is also evidence that other neurologic disorders may arise. The evidence of effects of endophyte toxins on immune function, polio-encephalomalacia (PEM), fat necrosis, and somatic cell count are weak, although potential pathways exist for effects to be exerted. There are few studies in dairy cattle on reproductive performance in relation to endophyte toxins, but basic studies in beef cattle with fescue endophytes and ergovaline provide evidence of potential to cause harm. There is moderate evidence of scouring, weight loss, selective appetite, lameness and reduced clover content and again good physiological evidence for these putative effects. Lastly, there is strong evidence that ergovaline and Lolitrem produced by *Neotyphodium lolii* cause heat stress in cattle.

The high prevalence of wild-type endophyte in grassland areas of Australia ensures that many dairy cattle will be exposed as mature cows or heifers to these potential toxins. It appears, more likely that the potential effect of these toxins has been significantly underestimated than overestimated in our production system. Of particular concern is the potential to influence mammary gland development,

through effects around calving on dam and calf and during puberty and gestation in the heifer. The potential for interactions between ergovaline and heat stress should not be under-estimated in the summer months, especially in irrigation districts. Behavioural problems with either Lolitrem or other neurotoxic alkaloids are observed by producers. It is clear from the magnitude of loss in some studies (Table 1) that the disorder may be of substantial magnitude attributable to milk production loss in the summer and autumn and due to costs associated with behavioural problems.

Plant poisonings pose special problems in definition. Toxins, may not be always present or may not be well characterized, leading to difficulties in conducting studies on a dose-response basis. Randomisation, controlling for confounding effects (eg more fertile paddocks, effects on other plant species) and blinding are difficult when dealing with herds of lactating cows. Physiological studies demonstrate mechanism, but not necessarily magnitude of effect in the field. Notwithstanding these problems, it appears that there is ample evidence that ryegrass endophyte could be a major factor limiting pastoral production in Australia.

(Note: References are available from the author)

Subscriptions for 2007 will be due July 2006.

Reminder notices will be sent out in May 2006.

From the President's desk

Autumn is well upon us and once again dry weather is causing concern over much of the state. Parts of the northern tablelands have fared reasonably well while elsewhere rain is needed asap to ensure pasture growth before winter sets in. Cropping areas in central and southern NSW are also looking for the much quoted "autumn break" to kick off winter cereal and oil seed programs. It seems dry autumns are becoming more normal than occasional, presenting problems for weed management in pasture and crop as well as the more obvious moisture accumulation. Pasture and grazing management strategies are also disrupted significantly with irregular rain where ground cover is threatened and productive species decline.

Planning for the Wagga conference scheduled for 25th to 27th July is well under way. Belinda Hackney and her team have been hard at work now since late last year putting together a stimulating program. This year the emphasis is on weed control and all aspects of successful weed management. Topics will explore new and existing weeds, chemical and biological control, pasture and animal weed interactions and the role of fodder conservation. This is a conference not to be missed by anyone with a concern for current and future weed challenges. A feature of the program will be the producer experiences in managing practical weed control programs.

While on the subject of the conference, I would very much like to acknowledge our many sponsors. They will be listed in the next newsletter when sponsorships have been finalized, as well as in the conference proceedings. Without the seed, fertilizer, agri-business and chemical companies who so generously support the Society we would simply be unable to function at our current level. A big thank you to all these companies.

Please put 25th to 27th July, Wagga into your diaries now. A great program of technical and social interaction is assured.

Best wishes to all.

Mick Duncan

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A unique blend of people with a common interest in developing our most important resource – our Grasslands

The Grassland Society of NSW was formed in March 1985. The Society now has 490 members and associates, 75% of whom are farmers and graziers. The balance are agricultural scientists, farm advisers, consultants, and executives or representatives of organisations concerned with fertilisers, seeds, chemicals and machinery.

The aims of the Society are to advance the investigation of problems affecting grassland husbandry and to encourage the adoption into practice of results of research and practical experience. The Society holds an annual conference, publishes a quarterly newsletter, holds field days, and is establishing regional branches throughout the State.

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