Gangrenous ergotism in cattle grazing fescue (*Festuca elatior* L.) in South Africa

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ABSTRACT

The 1st outbreak of fescue toxicosis in South Africa was recently confirmed in a Brahman herd at Perdekop, near Standerton, in Mpumalanga province, South Africa. Within 3 weeks of being placed on a fescue pasture in mid-winter, 50 of 385 cattle developed lameness and/or necrosis of the tail. The farmer had established *Festuca elatior* L. (tall fescue, Iewag variety) on *c*. 140 ha for winter grazing. Fescue may be infected by an endophyte, *Neotyphodium coenophialum*, which produces ergot alkaloids, in particular ergovaline. Ergovaline concentrations in basal leaf sheaths and grass stems collected during the outbreak ranged from 1720–8170 ppb on a dry-matter basis.

Key words: cattle, ergotism, ergovaline, fescue, Festuca elatior, Neotyphodium coenophialum.

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INTRODUCTION

Festuca elatior L. (= *F. arundinacea* Scribe.), commonly known as tall fescue or 'lang swenkgras', is one of the most widely utilised perennial, cool-season, bunch grasses in the world (Fig 1). This grass, probably originating from western Europe, was introduced into South Africa and currently is naturalised all over the country. This versatile perennial grass is used as pasture, hay and silage. Tall fescue produces most abundantly under irrigation and high nitrogen fertilisation, and remains green in winter. It is also tolerant of continuous close grazing and is superior to many other cool-season grasses in livestock-carrying capacity^{1, 6}.

Although tall fescue compares favourably with other cool-season grasses, it has certain shortcomings. The forage is of low palatability to livestock and performance of animals grazing it is erratic and less than satisfactory. Cattle grazing fescue occasionally develop lameness and sometimes lose portions of their tails or hooves when ambient temperatures are low. It has been noted that these signs are com-

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parable to poisoning in animals following ingestion of *Claviceps purpurea* ergots. Since these signs were observed during the cool seasons (autumn and winter) when *C. purpurea* sclerotia were not present, ergots of *C. purpurea* were ruled out as causative agent^{1, 22}.

After years of research, the association of fescue toxicity with the infection of tall fescue with an endophytic fungus was finally established². This clavicipataceous endophyte was originally identified as Epichloë typhina, later renamed Acremonium coenophialum and now known as Neotyphodium coenophialum (Morgan-Jones & Gams)^{1,2,16,18}. This non-sporulating fungus is non-phytopathogenic and inhabits the intercellular spaces (only microscopically visible) of fescue leaf sheaths, the base of grass stems as well as the seeds. The endophyte imparts both advantageous and disadvantageous qualities to tall fescue. The endophytic fungus does not appear to have any adverse effect on the plant and in fact live in mutualistic symbiosis with the plant^{1, 9}. The fungus derives its nutrients and a method to reproduce (via infected seeds) from the plant and in turn imparts hardiness and persistency to infected fescue through mechanisms such as reduced herbivory, increased drought tolerance and increased resistance to insects and nematodes. The main disadvantage of endophyte-infected tall fescue is that, in some instances, it causes fescue toxicosis in livestock^{3,9}.

POISONOUS PRINCIPLES

A number of chemical classes of compounds have been associated with endophyte-infected tall fescue, including perloline, halostachine, β -carboline alkaloids, clavine alkaloids, lysergic acid amides, loline alkaloids and ergopeptine alkaloids such as ergovaline 22 . Fescue toxicosis was finally attributed to the ergopeptine alkaloids. Ergovaline contributes 84–97% of the total ergopeptine alkaloids present in *Neotyphodium*-infected tall fescue 10 .

MECHANISM OF ACTION

These ergopeptine alkaloids are structurally similar to the biogenic amines serotonin, dopamine, adrenalin and noradrenalin and have affinity for their receptors. Interaction of the ergopeptines with these biogenic amine receptors will alter the normal homeostatic mechanisms, resulting in hyperthermia or gangrenous ergotism^{3, 22}.

In an in vitro study, it was demonstrated that ergopeptines (ergotamine and ergosine) cause constriction of the dorsal pedal vein of cattle. Ergotamine probably causes peripheral vasoconstriction via α-adrenergic receptors²⁰. The vasoconstrictive properties of these ergopeptine alkaloids may help explain some of the signs observed in animals. The peripheral cutaneous vasoconstriction prevents heat loss and results in hyperthermia during the warm summer months, which is also referred to as 'summer syndrome'. During the winter the existing vasoconstriction is exacerbated causing ischaemia of the extremities (tail, feet, ears), necrosis and dry gangrene³.

Studies by Macleod and Lehmeyer (1974)¹¹ have shown that ergopeptines reduce prolactin secretion, at the pituitary level. Ergopeptines were shown to interact with dopamine (D₂) receptors¹⁹. Physiologically, the suppression of prolactin secretion is regulated mainly by tonic inhibition through activation of the D2-receptors by dopamine. Therefore, agonistic interaction of ergopeptine alkaloids with the D₂-receptors will result in decreased prolactin secretion^{11,19,21}. Decreased serum prolactin concentra-

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tions were also documented in animals consuming endophyte-infected tall fescue¹⁰. The severe drop in milk production in dairy cows is probably the result of decreased feed consumption. Prolactin secretion during the peri-parturient period is essential for maximal milk production post partum, as prolactin is important in lactogenesis (establish milk secretion) and mammogenesis, but not for galactopoiesis (production of milk)14. Lower conception rates and short term infertility are also attributed to the decreased serum prolactin concentrations as prolactin is required for the maintenance of the corpus luteum and thus gestation¹⁷.

FIELD OUTBREAK

An outbreak of fescue toxicosis was confirmed in a Brahman herd on the farm Slangfontein (27°05′ S, 29°40′ E) at Perdekop, near Standerton, in Mpumalanga province, South Africa. The farmer had established fescue (*Festuca elatior*, Iewag strain) pastures (*c*. 140 ha) to be utilised in winter. Fifty of 385 adult cattle, that grazed on the fescue pastures for less than 3 weeks in midwinter, developed claw lesions, lameness and dry gangrene of the tail (Fig 2). Decreased weight gains were also noted.

During the outbreak, 9 tufts of grass taken from 4 different pastures, seed heads, harvested seed screenings and threshed seeds were collected on the farm. The tufts of grass were broken up to separate individual grass stems with roots. The roots were removed and the basal grass stems, including the leaf sheaths, (5–10 cm sections) were collected in quantities of 80–110 g. These grass basal stems were freeze-dried to constant weight before being forwarded to the analytical laboratory in Missouri, USA. Following extraction, the ergot alkaloids were identified and quantified according to a standard high-performance liquid chromatographic (HPLC) procedure¹⁵.

In addition, during the farm visit specimens of the grass were collected and submitted for botanical identification. Tufts of grass from the pastures were transplanted and established in the Poisonous Plant Garden, Faculty of Veterinary Science, Onderstepoort.

RESULTS

The grass was identified as *Festuca elatior* L. (= *F. arundinacea* Schreb.) by the National Botanical Institute, Pretoria. Ergovaline concentrations ranging from 1720–8170 ppb DM were measured in the basal stems and leaf sheaths. In the seeds, seed screenings and seed heads ergovaline concentrations ranged from 995–4600 ppb DM (Table 1). Lower concentrations



Fig. 1: Panicle of Festuca elatior L. (= F. arudinacea Schreb.).



Fig. 2: Dry gangrene of the tail of a Brahman bull.



Fig. 3: Claviceps purpurea sclerotia on seed heads of tall fescue.

Table 1: Ergopeptine concentrations determined in various plant parts.

Farm Slangfontein	Ergopeptine concentrations (ppb DM)					
	Ergovaline	Ergosine	Ergotamine	Ergocornine	Ergocryptine	Ergocristine
Pasture 1						
Grass stem A	7605	_	_	_	_	_
Grass stem B	4455	_	_	_	_	_
Grass stem C	5310	_	_	_	_	_
Pasture 2						
Grass stem A	6750	_	_	_	_	_
Grass stem B	5590	_	_	_	_	_
Grass stem C	8170	_	_	-	-	_
Pasture 3						
Grass stem A	1720	-	_	_	_	-
Pasture 4						
Grass stem A	nd	_	_	_	_	_
Grass stem B	2610					
Seed	4600	3305	290	685	840	185
Seed heads	995	nd	nd	nd	nd	nd
Seed screenings	1130	680	130	270	270	110

nd = none detected.

of ergosine, ergotamine, ergocornine, ergocryptine and ergocristine were also detected in the seed screenings and seed samples, although collectively these ergopeptine alkaloids exceeded the concentration of ergovaline.

DISCUSSION

This is the 1st recorded outbreak of gangrenous ergotism (fescue foot) in cattle following ingestion of endophyteinfected fescue in South Africa. It is estimated that fescue toxicosis is induced in cattle when ergovaline concentrations exceed 400-750 ppb^{8,23}. This threshold level, before dry gangrene will be precipitated, is also dependent on low environmental temperatures²³. The high concentration of ergovaline, the principal ergot alkaloid produced by Neotyphodium coenophialum, in the samples analysed supported a diagnosis of fescue toxicity. However, lower concentrations of ergosine, ergotamine, ergocornine, ergocryptine and ergocristine, that are usually associated with Claviceps sclerotia, were also detected in the seed screenings and seed samples.

Subsequently, *Claviceps purpurea* sclerotia were observed in the tufts of grass transplanted at Onderstepoort. (Fig. 3). These ergopeptines most probably also contributed in inducing the syndrome.

In light of the exceptionally high ergovaline concentrations determined in the fescue samples augmented by ergopeptines present in *C. purpurea* sclerotia, it is astonishing that outbreaks of bovine ergotism from this source had not been reported previously. It is interesting to note that during 1993, a suspected outbreak of fescue toxicosis, manifesting as summer syndrome in dairy cows,

occurred in the Humansdorp district, Eastern Cape Province. Endophytes were observed in the fescue variety, but no chemical analysis was undertaken (FA van Niekerk, private practitioner, Humansdorp, pers. comm., 1996).

In addition to the lesions associated with gangrenous ergotism, cattle consuming endophyte-infected tall fescue have been reported to show some or all of the following responses in comparison with those grazing non-infected fescue: reduced feed intake, lower weight gain, decreased milk production, higher respiratory rates, elevated body temperatures, rough hair coat, more time spent in water or in the shade, less time spent grazing, excessive salivation, reduced serum prolactin concentrations and diminished reproductive performance 13,14. This manifestation of fescue toxicosis, which predominantly occurs in the summer months, has been referred to as summer syndrome or summer slump³.

In 1993, summer syndrome (or hyperthermia) was diagnosed in cattle in the Western Cape Province, South Africa, after feeding barley screenings (broken and undersized barley and grass seeds) heavily contaminated with annual ryegrass (*Lolium* sp) seeds and *C. purpurea* sclerotia¹⁷.

A similar syndrome in dairy cattle occurred in 1996/97 following feeding of maize silage or tef hay contaminated with *Cyperus esculentus* (yellow nutsedge, 'geeluintjie') containing numerous sclerotia of the fungus *C. cyperi*¹².

If fescue toxicosis is suspected, 200 g of fresh seed, seed heads or basal grass stems (remove close to level of the soil) should be submitted for analysis. The presence of the fungus in fescue can be confirmed indirectly by the detection of ergovaline.

Analytical techniques such as thin-layer chromatography (TLC)¹⁶, enzyme-linked immunosorbent assay (ELISA) screening methods¹⁸ and HPLC quantification⁵ can be used to determine ergot alkaloid concentrations. In addition to screening for ergovaline concentrations, tillers for fungus identification and determination of percentage of endophyte infection can also be submitted. Urinary ergot alkaloid excretion can be used to confirm fescue toxicosis in cattle and provides a more accurate assessment of animal health compared with serum prolactin concentrations⁷. However, urinary alkaloid analysis (ELISA) can only aid in the diagnosis if the cattle are still grazing on infected fescue, as the urinary ergot alkaloid concentrations decrease rapidly if cattle are removed from the source for a day or two. Thus, the test cannot be used to diagnose what happened the previous week.

The most obvious method of prophylaxis is to prevent cattle from grazing endophyte-infected tall fescue pastures. Ergovaline concentration is highest during seeding (and Claviceps ergotisation may additionally be present then); thus, grazing during this phase should be avoided. High mowing of the seeding grass will remove the seed heads, a major source of ergot alkaloids. Stock owners should also prevent heavy grazing of the grass, as higher concentrations of endophytes occur in the basal stems near the roots. Farmers could also be advised to establish clover and fescue pastures together, thereby reducing the consumption of fescue. Cutting and baling of fescue, before seeding, would most probably also prevent ergotism³. Ammoniation of endophyte-infected tall fescue hay with anhydrous ammonia alleviated the negative

effects on animal performance often associated with *Neotyphodium*-infected fescue⁴. With long-term storage of seed the endophyte will eventually die¹. Since the infection can only be transmitted via endophyte-infected seed, new fescue pasture could be established with endophyte-free seed. However, the endophyte-free fescue is not that hardy and may not survive drought stress. It is probably more advisable to plant the new 'friendly endophyte-infected fescues' that are currently available and which do not produce ergovaline, but the good qualities are retained.

Fescue pastures have been widely cultivated throughout South Africa and there is a grave possibility of this endophytic fungus being present in other fescue pastures. The situation can be exacerbated by a concurrent *C. purpurea* infection. Private practitioners should be aware of the potential problems posed by such pastures and farmers should be alerted to the existence of this disease in South Africa.

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