CASE REPORT

Fescue-associated oedema of horses grazing on endophyte-inoculated tall fescue grass (*Festuca arundinacea*) pastures

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A new form of toxicity called equine fescue oedema is described. The clinical signs included inappetence, depression, and subcutaneous oedema of the head, neck, chest and abdomen. Affected horses had very low plasma albumin values.

The toxicity affected 48 of 56 horses on six farms in different states of Australia, and 4 horses have died. All horses were grazing pastures that had been sown with varieties of Mediterranean tall fescue (*Festuca arundinacea*) that carry the endophyte known as Max P or Max Q. It is proposed that a pyrrolizidine alkaloid, N-acetyl norloline, which is produced by the Max P endophyte, may be responsible for this new toxicity in horses.

Keywords endophyte; fescue; horses; oedema; poisoning; pyrrolizidine alkaloid; toxicity

Abbreviations EFO, equine fescue oedema

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Figure 1. **Tall fescue grass (*Festuca arundinacea*), a perennial pasture species sown in temperate climate zones.**

Tall fescue (*Festuca arundinacea*) is a perennial pasture grass that originated in Europe and North Africa (Figure 1). Commercially available varieties are divided into Continental and Mediterranean genotypes, the former displaying summer growth and the latter having winter growth activity.[1,2] Until 2001 all tall fescue varieties sown internationally were the Continental type and then the Mediterranean genotypes, including selected endophyte-inoculated seed lines, were released for commercial use, initially in South America in 2001, then in both North America and Australia in 2003. Fescue toxicosis has been observed in horses, cattle and sheep grazing Continental tall fescue infected with wild strains of the endophyte *Neotyphodium coenophialum* for at least 100 years.[3] Wild strains of this fungus can produce a range of toxic ergopeptide alkaloids of which ergovaline is the predominant compound.[4] They can also produce two groups of unrelated alkaloids called peramines and lolines.[5,6] Each of these alkaloids contributes to the good health of the fescue plant by acting as insecticides. The toxicity of ergovaline in farm livestock is well established, but there are as yet no reports of studies in which the potential toxicity of peramines or lolines in farm livestock has been investigated.

Fescue toxicosis in horses typically presents as either a reproductive problem or a summer ill-thrift problem.[3] In cattle and sheep, fescue toxicosis most commonly presents as summer ill-thrift,[3] characterised by reduced feed intake, reduced live weight gain, reduced milk production, hyperthermia and, in some cases, diarrhoea. On rare occasions during the winter period, fescue toxicosis in ruminants can present as peripheral gangrene and because this usually involves the distal limbs it is often referred to as 'fescue foot'.[3] Ergot alkaloids are responsible for most manifestations of fescue toxicoses, but their specific contribution to 'fescue foot' remains unclear. Reproductive problems can occur in ruminants grazing fescue, but are more commonly encountered in horses and include failure of embryos to implant, death of early stage embryos, late term abortions, small birth weights, increased numbers of stillbirths and an increase in neonatal deaths during the first 24 h.[3]

Between October 2007 and December 2008 a new toxicosis, equine fescue oedema (EFO), occurred in horses grazing pastures that contained tall fescue grass at Scone, Cootamundra, Albury and Canowindra in New South Wales, at Kangarilla in South Australia and at Katanning in Western Australia. A total of 56 adult horses had grazed these toxic pastures and of these 48 became intoxicated and 4 died. Each of the pastures associated with the toxicosis had been sown with Mediterranean tall fescue varieties infected with a specific strain of *N. coenophialum*, AR542 (Max P or Max Q; Grasslanz Technology–PGG–Wrightson Seeds) that did not produce ergovaline. Continental tall fescue infected with wild endophyte producing ergopeptide alkaloids was not involved. The amount of fescue present in each toxic pasture varied from as little as 8% to as much as 95% of the feed on

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Figure 2. Equine fescue oedema. The horse appears depressed and has mild subcutaneous oedema of the head and neck.

Figure 3. Equine fescue oedema, showing subcutaneous oedema of the ventral abdomen.

offer. In toxic pastures where the fescue component was only small, owners of affected horses reported that it had been preferentially grazed.

Two of the six outbreaks of this new toxicosis are reported in detail here, and they are the first recorded accounts of EFO.

Case reports

Case report 1

This outbreak of EFO involved five, mixed age, Thoroughbred mares with foals at foot that ranged in age from 1 to 2 weeks, grazing a 1.8-ha pasture paddock at Kangarilla in the Adelaide Hills of South Australia in November 2007. The paddock consisted predominantly of annual ryegrass pasture oversown in July 2007 with a mixture of Flecha Max P tall fescue, cocksfoot and white clover. The pasture germinated following late winter rains, but went through a dry period during September and October. Rain (20 mm) fell in early November and by 26 November the feed on offer was a mixture of the four species, as well as self-sown barley grass, fog grass, cape weed and wire weed. Tall fescue only constituted 8% to 10% of the total feed on offer, but presumably because it was the greenest and most palatable species present it appeared to have been preferentially grazed. Three of the mares and their foals were placed in the paddock on 21 November and the remaining mares and foals introduced on 24 November. On 26 November one mare ('Reave') was observed to be depressed and inappetant. On closer inspection it was found that she had a slightly swollen head consistent with subcutaneous oedema. The eyelids and lips were the most affected, but there was also some oedema of the neck (Figure 2). The mare was brown in colour and had a fully pigmented head and body, so photosensitisation seemed unlikely. Later that day a second mare ('Mignon') was observed to be showing similar signs. By 27 November a third mare ('Daisy') had become affected, as had three of the five foals. One of the affected foals had a moderate amount of swelling of the vagina with exteriorisation of the mucosa.

All of the horses were moved out of the paddock on this day and placed onto a ryegrass pasture and provided a high-protein extruded feed supplement at a rate calculated to provide 150% of the lactation requirement. Over the following 5 days the subcutaneous oedema in the three affected mares moved to the chest and along the ventral abdomen (Figure 3). A previously unaffected mare that had only been on the toxic pasture for 4 days also developed ventral oedema by the 5th day after removal from the pasture. Heart rates, respiratory rates and rectal temperatures for all mares remained normal throughout this period. The biochemistry and haematology results recorded for three of the affected mares are presented in Tables 1 to 4. Affected horses had low plasma concentrations of total protein, particularly albumin.

None of the mares were pregnant when they grazed the toxic pasture. The four affected mares lost considerable condition over the following 2 weeks despite being fed a supplement. The growth rate of their foals was reduced, possibly because the mares were making less milk. The condition of the foals only improved when they started to eat the supplement. Following the toxic event the mares were unresponsive to a prostaglandin injection 20 days postpartum, and when examined by ultrasound on 12 December two of the affected mares had obvious oedema of the uterus and echogenic inclusions in the endometrium similar in appearance to fibrin strands. In one of these mares the inclusions also occupied 30% of the uterine lumen, giving a honeycomb appearance to a body that was 12 to 15 cm long and 2 to 6 cm in depth. There were no obvious changes in the four other mares, but none of the mares cycled over the next 6 weeks. After this period the four mares started to cycle, but none fell pregnant after insemination on four cycles. The worst affected mare continued to not cycle and, together with the other mare showing abnormal changes in the uterus on 12 December 2007, was re-examined in October 2008. Ultrasound examination at this time indicated little resolution. The three affected foals remained significantly smaller than other similarly aged and managed foals.

Table 1. **Biochemistry results for the Kangarilla mare 'Mignon', first observed to be affected by equine fescue oedema on 26 November 2007**

Parameter	Reference range	26/11/07	29/11/07	13/12/07	10/1/08
Sodium	132–152 mmol/L	137	136	142	141
Potassium	2.8–5.0 mmol/L	3.8	4.5	3.9	4.9
Chloride	98–110 mmol/L	95	98	98	99
Bicarbonate	23–32 mmol/L	33	31	30	27
Na : K ratio	>27.0	36.1	30.2	36.4	28.8
Urea	3.6–8.9 mmol/L	6.0	4.0	4.1	5.9
Creatinine	110–170 umol/L	110	90	110	114
Calcium	2.50–3.60 mmol/L	1.90	2.19	3.00	3.42
Phosphate	0.80–1.77 mmol/L	1.90	0.98	1.27	1.70
Total protein	58–76 g/L	27	30	64	73
Albumin	28–38 g/L	8	11	23	32
Globulin	26–40 g/L	19	19	41	41
Total bilirubin	4–100 µmol/L	23	15	19	23
GLDH	<21 U/L	–	4	3	3
ALP	50–250 IU/L	207	229	174	189
AST	150–400 IU/L	161	260	15	17
CK	60–240 IU/L	550	451	327	264
GGT	20–38 IU/L	12	8	15	17
Cholesterol	1.8–3.6 mmol/L	1.8	1.5	2.2	2.3

ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, gamma-glutamyl transferase; GLDH, glutamic dehydrogenase.

Table 2. **Haematology results for the Kangarilla mare 'Mignon', first observed to be affected by equine fescue oedema on 26 November 2007**

Parameter	Reference range	26/11/07	27/11/07	29/11/07	3/12/07	13/12/07	10/1/08
RBC	$6.50–12.50 \times 10^{12}$/L	11.9	11.30	9.33	9.45	10.7	10.5
Hb	110–190 g/L	195	186	153	134	173	171
PCV	0.32–0.52 L/L	0.56	0.50	0.43	0.37	0.49	0.51
MCV	34–58 fL	47	45	46	39	46	49
MCH	12–18 pg	16	17	16	14	16	16
MCHC	310–370 g/L	348	372	358	366	354	334
Platelet count	$100–500 \times 10^9$/L	237	242	199	348	–	–
WBC	$5.5–12.5 \times 10^9$/L	16.4	15.1	13.4	10.8	9.0	6.3
Neutrophils %	22–72%	86	78	76	68	42	21
Neutrophils abs	$2.5–8.0 \times 10^9$/L	14.1	11.8	10.2	7.3	3.8	1.3
Lymphocytes %	17–68%	13	21	19	26	55	75
Lymphocytes abs	$1.5–5.5 \times 10^9$/L	2.1	3.2	2.5	2.8	4.9	4.7
Monocytes %	<15%	1	1	3	6	2	2
Monocytes abs	$<0.6 \times 10^9$/L	0.2	0.1	0.4	0.7	0.2	0.1
Eosinophils %	<11%	0	0	2	0	1	2
Eosinophils abs	$<1.1 \times 10^9$/L	<0.1	0	0.3	0	0.1	0.1
Basophils %	<5%	0	0	0	0	0	0
Basophils abs	$<0.3 \times 10^9$/L	<0.1	0	0	0	0	0
Fibrinogen	1.0–4.0 g/L	2.0	5	6.0	3.0	7.0	3.0

abs, absolute; Hb, haemoglobin; MCV, mean cell volume; MHC, major histocompatibility complex; MCHC, mean corpuscular Hb concentration; PCV, packed cell volume; RBC, red blood cells; WBC, white blood cells.

Table 3. Biochemistry results for the Kangarilla mares 'Reave' and 'Daisy', first observed to be affected by equine fescue oedema on the 26 and 27 November 2007, respectively

		'Reave'			'Daisy'		
Parameter	Reference range	29/11/07	3/12/07	13/12/07	2/11/07	3/12/07	13/12/07
Sodium	132–152 mmol/L	140	143	141	139	138	140
Potassium	2.8–5.0 mmol/L	4.8	4.2	4.3	4.0	4.1	4.8
Chloride	98–110 mmol/L	102	108	99	103	107	100
Bicarbonate	23–32 mmol/L	30	24	32	25	22	26
Na : K ratio	>27.0	29.2	34.1	32.8	34.8	33.7	29.2
Urea	3.6–8.9 mmol/L	6.0	4.8	4.0	6.2	3.9	3.9
Creatinine	110–170 umol/L	124	126	125	84	80	101
Calcium	2.50–3.60 mmol/L	2.7	2.91	3.02	2.39	2.83	2.94
Phosphate	0.80–1.77 mmol/L	0.85	0.63	1.01	0.79	0.70	1.11
Total protein	58–76 g/L	37	53	59	37	57	64
Albumin	28–38 g/L	12	18	22	11	16	22
Globulin	26–40 g/L	25	35	37	26	41	42
Total bilirubin	4–100 umol/L	15	15	21	39	15	18
GLDH	<21 U/L	8	6	3	3	9	3
ALP	50–250 IU/L	175	189	151	346	252	230
AST	150–400 IU/L	338	302	238	178	251	251
CK	60–240 IU/L	456	384	226	1300	305	208
GGT	20–38 IU/L	7	13	13	4	9	11
Cholesterol	1.8–3.6 mmol/L	1.9	1.8	2.2	1.4	2.0	1.9

ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, gamma-glutamyl transferase; GLDH, glutamic dehydrogenase.

Case report 2

The second outbreak involved a group of 10 fillies (2- and 3-year-olds used for polo) that were grazing a 30-ha paddock at Canowindra in the central west of New South Wales in March 2008. The paddock had been sown with a seed mix of Flecha Max P tall fescue, phalaris, lucerne, white clover and subterranean clover in June 2005. In March 2008 there was also some stink grass (*Eragrostis cilianensis*), crumb weed (*Chenopodium carinartum*) and blown grass (*Panicum* sp.) growing in the paddock. There had been storm activity and mild temperatures in January 2008, but this had been followed by a hot and dry February. By March plant growth over most of the paddock was dry and dormant; however, in an approximately 3 ha depression that ran across the paddock and formed a natural water drainage zone, moisture conditions were much better and plants were green and actively growing. Approximately 30% to 60% of the ground cover across this section was tall fescue and the owner reported that this area had been the preferred grazing location for the horses in the affected group.

A different group of horses grazed the same paddock in July and August 2007 without any problems. The affected group of 10 horses was introduced to the paddock on 1 March 2008. On 7 March the owner discovered one of the horses had died. Three days later four other horses were noticed to be depressed, lethargic and inappetant, and all presented with oedematous swelling of the head. Two ('Billy Jean' and 'Moet') also had swelling of the neck and the other two ('Sybil' and 'Kiki') also had prolapse of the rectum. Sheep were grazing the paddock at the same time as the affected horses, but were not affected. The horses were removed from the paddock on 10 March and had fully recovered by 1 April. The biochemistry and haematology results recorded for the four affected horses are presented in Tables 5 and 6. Affected horses had low plasma concentrations of total protein, particularly albumin.

Discussion

Both case reports demonstrate that EFO is characterised by the development of dependent subcutaneous oedema and that this oedema is associated with the presence of a low total plasma protein value, in particular a very low albumin value. In normal animals albumin is synthesised by the liver as a constant process with no storage capacity.[7] In EFO, the plasma albumin concentration can fall below the reference range within 5 days or less of first ingesting the toxic plant. This is very rapid, and possibly too rapid to be caused simply by the inhibition of synthesis. An alternative possibility is a rapid loss of albumin; however, random urinalyses in other cases of EFO not reported here failed to indicate protein loss via urine. Three severely affected horses in other outbreaks presented with signs of subcutaneous oedema and colic and subsequently died. Those animals were necropsied and found to have severe oedematous thickening of the right dorsal colon wall, as well as varying degrees of secondary peritonitis (D Radecleft,

Table 4. **Haematology results for the Kangarilla mares 'Reave' and 'Daisy', first observed to be affected by equine fescue oedema on the 26 and the 27 November 2007 respectively**

		'Reave'			'Daisy'			
Parameter	Reference range	29/11/07	3/12/07	13/12/07	27/11/07	29/11/07	3/12/07	13/12/07
RBC	6.50–12.50 × 10^{12}/L	7.95	9.12	8.32	7.03	6.15	5.93	11.2
Hb	110–190 g/L	146	166	147	132	117	113	150
PCV	0.32–0.52 L/L	0.38	0.44	0.41	0.34	0.31	0.29	0.43
MCV	34–58 fL	48	48	49	49	50	49	38
MCH	12–18 pg	18	18	18	19	19	19	13
MCHC	310–370 g/L	380	381	358	384	380	390	352
Platelet count	100–500 × 10^{9}/L	–	–	–	189	–	360	–
WBC	5.5–12.5 × 10^{9}/L	8.8	8.5	6.5	21.7	22.0	10.1	13.3
Neutrophils %	22–72%	73	69	65	96	92	72	77
Neutrophils abs	2.5–8.0 × 10^{9}/L	6.4	5.9	4.2	20.8	20.2	7.3	10.2
Lymphocytes %	17–68%	24	25	31	3	6	23	19
Lymphocytes abs	1.5–5.5 × 10^{9}/L	3.1	2.1	2.0	0.7	1.3	2.3	2.5
Monocytes %	<15%	1	4	1	0	1	4	3
Monocytes abs	<0.6 × 10^{9}/L	0.1	0.3	0.1	0	0.2	0.4	0.4
Eosinophils %	<11%	1	0	2	0	1	1	1
Eosinophils abs	<1.1 × 10^{9}/L	0.1	0	0.1	0	0.2	0.1	0.1
Basophils %	<5%	1	2	1	1	0	0	0
Basophils abs	<0.3 × 10^{9}/L	0.1	0.2	0.1	0.2	0	0	0
Fibrinogen	1.0–4.0 g/L	9	7	7	7	8	8	5

abs, absolute; Hb, haemoglobin; MCV, mean cell volume; MHC, major histocompatibility complex; MCHC, mean corpuscular Hb concentration; PCV, packed cell volume; RBC, red blood cells; WBC, white blood cells.

Table 5. **Biochemistry results on 11 March 2008 for the four Canowindra horses first observed to be affected by equine fescue oedema on 10 March 2008**

Parameter	Reference range	'Billy Jean'	'Moet'	'Sybil'	'Kiki'
Sodium	133–150 mmol/L	126	136	128	133
Potassium	3.0–5.3 mmol/L	4.1	3.1	3.0	3.9
Chloride	97–109 mmol/L	99	107	101	108
Urea	3.6–8.9 mmol/L	6.5	7.3	6.5	7.2
Creatinine	71–194 umol/L	64	94	75	75
Calcium	2.60–3.22 mmol/L	2.46	2.60	2.41	2.44
Phosphate	0.58–1.81 mmol/L	1.01	0.37	0.45	0.94
Total protein	56–79 g/L	39	45	35	33
Albumin	19–32 g/L	10	12	8	9
Globulin	24–47 g/L	29	34	26	25
Total bilirubin	0–60 µmol/L	13	2	21	15
ALP	10–326 IU/L	249	306	137	146
AST	100–600 IU/L	172	882	297	186
CK	10–350 IU/L	340	274	385	205
LDH	250–2070 IU/L	1049	>2800	–	1397
GGT	0–87 IU/L	10	25	1	2

ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, gamma-glutamyl transferase; LDH, lactate dehydrogenase.

Table 6. Haematology results on 11 March 2008 for the four Canowindra horses first observed to be affected by equine fescue oedema on 10 March 2008

Parameter	Reference range	'Billy Jean'	'Moet'	'Sybil'	'Kiki'
Hb	11.0–19.0 g/dL	17.7	13.6	13.6	13.4
Hct	32.0–52.0%	48.9	38.7	39.1	36.4
MCHC	30.0–36.9 g/dL	36.2	35.1	34.8	36.8
Platelet count	90–350 × 10^9/L	293	307	217	322
WBC	6.0–12.5 × 10^9/L	13.6	15.5	13.2	14.0
Neutrophils %	22–72	68	61	85	69
Neutrophils abs	2.8–8.0 × 10^9/L	9.2	9.4	11.2	9.6
Lymphocytes %	17–68	32	39	15	31
Lymphocytes abs	2.1–7.0 × 10^9/L	4.4	6.1	2.0	4.4

abs, absolute; Hb, haemoglobin; Hct, haematocrit; MCHC, mean corpuscular Hb concentration; WBC, white blood cells.

A Begg, K Clayton and J Allen, personal communication). Without knowing the protein content of the interstitial fluids in the subcutis and colon, it is not possible to determine whether the subcutaneous and colonic oedema was because of loss of albumin into these tissues or simple osmotic loss of plasma into the interstitium because of the low concentrations of total protein and albumin in the plasma.

Observations of horses affected in all six outbreaks indicated that the recovery time after EFO is variable, with some affected horses returning to normal within 3 to 7 days, but others remaining affected for as long as 21 days. Case one raises the possibility of longer term reproductive problems in some brood mares affected by EFO, presumably related to the stage of pregnancy or the stage of the reproductive cycle of the mare when intoxicated. The mares had only recently foaled when they became intoxicated.

The two outbreaks highlight the difficulty that some livestock owners and their veterinarians may experience in making a direct link between clinical disease and fescue pasture toxicity. In case one the toxicity occurred within 5 days of a group of horses being placed in a new pasture paddock and so the horse owner immediately surmised that the pasture was in some way toxic. In case two, on the other hand, confusion was created in the horse owner's mind because several other groups of horses had grazed the same paddock the previous winter without experiencing any health problems. It is significant that the period of pasture toxicity in both cases followed a protracted period of dry weather that ended with sufficient rainfall to initiate pasture growth. In all of the outbreaks so far there has been an extended period of dry weather, varying in length from 4 to 6 weeks, prior to toxicity developing. A similar circumstance has also been observed in the period prior to outbreaks of kikuyu grass poisoning in cattle.[8] Kikuyu poisoning is another disorder that appears to involve a grass–endophyte association, but in the case of kikuyu poisoning the suspected fungal endophyte is *Fusarium torulosum*.[9]

Sudden onset of inappetence, lethargy, weakness and depression, together with variable signs consistent with gastrointestinal pain, can occur in some cases of EFO and may also be seen in the non-steroidal anti-inflammatory disorder that can occur in horses treated with phenylbutazone,[10,11] in the equine form of acute arsenic poisoning,[12] and in poisoning following ingestion of acorns (*Quercus* spp.).[13] Subcutaneous oedema, in addition to several of the aforementioned signs, can be seen in cases of African horse sickness,[14] equine viral arteritis,[14] equine infectious anaemia[14] and poisoning following ingestion of walnut (*Juglans* spp.) shavings.[15] All of these toxic or infectious causes were excluded in the present cases.

This new form of fescue toxicity is clinically different to other forms of toxicity previously reported for livestock grazing fescue pastures and different to any known effects of ergovaline or related ergot alkaloids. Ruminants do not appear to be affected by the new toxin at the dose level that affects horses. In fact, in one case not reported in detail here horses were affected by a toxic pasture and were removed; it was then grazed by cattle for 2 weeks without ill effect, but when another group of horses were subsequently placed on it they became intoxicated. Likewise in case two, sheep were grazing the toxic pasture together with the affected horses but remained unaffected.

Pastures containing Continental tall fescue varieties with the Max P endophyte have been widely planted, both nationally and internationally since 2001 yet there have been no cases of this new toxicosis in horses grazing these pastures. Similarly, there have been no reports of this condition in horses grazing Mediterranean tall fescue varieties without the Max P endophyte. The emergence of this new condition only in association with pastures sown with Mediterranean tall fescue varieties with the Max P endophyte suggests that a unique interaction is occurring in this specific pairing. Whether this interaction is resulting in the Max P endophyte producing the causative toxin or the presence of Max P is stimulating the grass to produce it remains to be determined.

Max P produces the alkaloids N-acetyl norloline and peramine, whereas many other *N. coenophialum* strains produce N-acetyl loline, N-formyl loline and N-acetyl norloline, and peramine.[16] Ball and Tapper compared loline production with different strains of endophyte, and with different species of fescue and ryegrass, and concluded that both the endophyte strain and the grass genotype have a significant influence over the type and amount of lolines produced.[16] Consistent with this has been the finding in preliminary comparative analyses of both Mediterranean and Continental tall fescue varieties in

Australia, with and without the Max P endophyte, that N-acetyl norloline production by the Max P endophyte in Mediterranean tall fescue is up to 8-fold greater than when the Max P endophyte is in Continental tall fescue (Qawasmeh, Bourke, and Wheatley, unpublished data). In these preliminary analyses, N-acetyl norloline was present in concentrations of up to 2000 mg/kg in Mediterranean tall fescue with the Max P endophyte, whereas Ball and Tapper[16] found concentrations of only 200 to 300 mg/kg in Continental tall fescue with the Max P endophyte. These results indicate a unique interaction between the Max P endophyte and Mediterranean tall fescue and suggest N-acetyl norloline might be involved in the aetiology of EFO. N-acetyl norloline is a saturated pyrrolizidine alkaloid and consequently is not hepatotoxic,[17] but little is known about other biological activities that either it or one of its metabolites might express in the horse. Peramine values were also established in toxic pastures associated with outbreaks of EFO not reported here. However, in two outbreaks the concentrations were only 5.7 and 6.0 mg/kg dry matter (Bourke and Watson, unpublished data) and in the third it was only 25.9 mg/kg dry matter (Bourke and Allen, unpublished data). These low values are similar to those found in endophyte-infected Continental fescues[5] and are of doubtful significance. The findings of this report would indicate that the biological activity of N-acetyl norloline in the horse warrants investigation.

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References

1. Gibson DJ, Newman JA. *Festuca arundinacea* Schreber (*F. elatior* L. ssp. *arundinacea* (Schreber) Hackel). *J Ecol* 2001;89:304–324.
2. Reed KFM, Clement SL, Feely WF, Clark B. Improving tall fescue (*Festuca arundinacea*) for cool-season vigour. *Aust J Exp Agric* 2004;44:873–881.
3. Burrows GE, Tyrl RJ. *Toxic plants of North America*. Iowa State University Press, 2001:895–902.
4. Panaccione DG. Origins and significance of ergot alkaloid diversity in fungi. *FEMS Microbiol Lett* 2005;251:9–17.
5. Bush LP, Wilkinson HH, Schardl CL. Bioprotective alkaloids of grass-fungal endophyte symbioses. *Plant Physiol* 1997;114:1–7.
6. Leuchtmann A, Schmidt D, Bush LP. Different levels of protective alkaloids in grasses with stroma-forming and seed transmitted *Epichloe/Neotyphodium* endophytes. *J Chem Ecol* 2000;26:1025–1036.
7. Ruot B, Breuille D, Rambordin F et al. Synthesis rate of plasma albumin is a good indicator of liver albumin synthesis in sepsis. *Am J Physiol Endocrinol Metab* 2000;279:244–251.
8. Bourke CA. A review of kikuyu grass (*Pennisetum clandestinum*) poisoning in cattle. *Aust Vet J* 2007;85:261–267.
9. Ryley MJ, Bourke CA, Liew ECY, Summerell BA. Is *Fusarium torulosum* the causal agent of kikuyu poisoning in Australia? *Aust Plant Dis Notes* 2007;2:133–135.
10. MacKay RJ, French TW, Nguyen HT, Mayhew IG. Effects of large doses of phenylbutazone administration to horses. *Am J Vet Res* 1983;44:774–780.
11. MacAllister CG. Effects of toxic doses of phenylbutazone in ponies. *Am J Vet Res* 1983;44:2277–279.
12. Pace LW, Turnquist SE, Casteel SW et al. Acute arsenic toxicosis in five horses. *Vet Pathol* 1997;34:160–164.
13. Anderson GA, Mount ME, Vrins AA, Ziemer EL. Fatal acorn poisoning in a horse: pathological findings and diagnostic considerations. *J Am Vet Med Assoc* 1983;182:1105–1110.
14. Geering WA, Forman AJ. African horse sickness. In: *Animal health in Australia*. Vol. 9: *Exotic diseases*. Bureau of Rural Science, Department of Primary Industries and Energy, Canberra, 1987;21–25, 104–106.
15. Uhlinger C. Black walnut toxicosis in ten horses. *J Am Vet Med Assoc* 1989;195:343–344.
16. Ball OJP, Tapper BA. The production of loline alkaloids in artificial and natural grass/endophyte associations. In: *Proceedings of the 52nd New Zealand Plant Protection Conference*, 1999:264–269.
17. Cheeke PR. Toxicity and metabolism of pyrrolizidine alkaloids. *J Anim Sci* 1988;66:2343–2350.

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