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### Blood prolactin depression in growing pigs fed sorghum ergot (*Claviceps africana*)

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*Abstract.* The toxicity of sorghum ergot (*Claviceps africana*) was assessed in young pigs over 28 days. Fortyeight pigs of both sexes and 2 breeds (Large White and Duroc) were allocated across 6 grower diets, balanced for fibre and predicted digestible energy, and containing 0, 0.3, 0.6, 1.3, 2.5, or 5% ergot sclerotia [the 5% sclerotia diet contained 70 mg alkaloids/kg (>90% dihydroergosine)]. Blood samples taken on Days 0 and 28 were analysed for prolactin and clinical, biochemical, and haematological indices of health. Feed consumption and liveweight were individually monitored. There were no clinical signs of illness attributable to ergotism in the pigs. Blood prolactin concentrations were significantly depressed in pigs receiving 9 mg alkaloids/kg (0.6% sclerotia) and by >80% in pigs receiving 35 and 70 mg alkaloids/kg, clearly indicating a potential to interfere with lactation in sows. Reductions in feed intake and poor feed conversion were observed over the first 7 days with >9 mg alkaloids/kg, but some tolerance developed later. Feed refusal was more pronounced for pigs of the Duroc breed. Over the full trial period, growth was reduced by about 30% in pigs receiving 70 mg alkaloids/kg, as a result of poor feed intake and feed conversion. Digestible energy of diets containing ergot was later found to be lower than predicted, which contributed to this result.

Additional keywords: mycotoxin, fungus.

#### Introduction

Sorghum ergot (*Claviceps africana*) was first identified in Australia in April 1996, and within 6 months had been found in all sorghum-producing regions in Queensland and New South Wales. It has also recently entered South and North America (Bandyopadhyay *et al.* 1996). Ergot fungi infect the plant during flowering and after a growth cycle characterised by release of modified plant sap (honeydew) and release of spores, the infected florets develop hard sclerotia (ergots), which either fall to earth or are harvested with the grain. *C. africana* sclerotia are small, grey/white, and elongated, about half the size of sorghum grain and with a rough surface. Sclerotia are a mass of simple fungal cells, composed largely of chitin, and containing alkaloids.

Sorghum ergot produces a different range of alkaloids than the ergot of rye (*C. purpurea*) (Mantle 1977). Alkaloids of rye ergot interfere with milk production in sows (Nordskog and Clark 1945) by inhibiting release of prolactin, and reduce pig growth (Whittemore *et al.* 1977). However, pigs appear to be relatively resistant to the impaired peripheral circulation that can produce gangrene of the extremities in cattle and other species.

In contrast to rye ergot alkaloids, several published reports have suggested that the main alkaloid of sorghum ergot (dihydroergosine) was much less toxic and did not affect lactation (Frederickson *et al.* 1991). Indeed, there were no reports of disease in livestock caused by sorghum ergot anywhere in the world (Mantle 1977) until recent cases of agalactia in sows and cows in central Queensland (Blaney *et al.* 2000*b*). In these cases, apart from agalactia, severe feed refusal and poor growth of pigs was reported.

The objective of this study was to test whether sorghum ergot was toxic or in any way detrimental to pigs growing from 20 kg to 40 kg. Some of the preliminary results have been presented at conferences (Blaney 1997; Blaney and Kopinski 1998).

#### Materials and methods

#### Source of ergot

A batch of ergot-infected grain (7 tonne) was obtained from Mutdapilly in south-eastern Queensland in mid 1996 (the same crop used for a cattle grazing trial—Blaney *et al.* 2000*a*) and the ergot-rich fraction of the grain was separated by flotation in 10% salt, and air dried. This ergot-rich sorghum was estimated by microscopic examination to contain about 12% of sclerotia by weight.

#### Chemical and nutritional analyses

The total alkaloid content of the ergot-rich sorghum, of the diet containing 40% of ergot-rich sorghum, and of a few mature ergot sclerotia selected from the infected sorghum were determined spectroscopically after solvent extraction, clean-up, and reaction with van Urk's reagent. Individual alkaloids were separated by thin layer chromatography and detected by spraying with Erhlich's reagent. Dihydroergosine was quantified by high performance liquid chromatography (HPLC).

Since the wet, cool conditions favouring ergot infection might have also favoured infection by *Fusarium* species, several trichothecene mycotoxins were assayed in the diet containing most ergot. A slight modification of the official method of the Association of Official Analytical Chemists for deoxynivalenol, with detection by gas-liquid chromatography (Helrich 1990) was used. Zearalenone was assayed by HPLC (Helrich 1990).

Proximate and amino acid analyses were conducted on the ergotrich sorghum as an aid to feed formulation, using methods given in Williams and Blaney (1994).

#### Formulation of diets

Ergot-rich sorghum was incorporated into pig grower diets at concentrations of 0, 2.5, 5, 10, 20, and 40%, which was equivalent to 0, 0.3, 0.6, 1.25, 2.5, and 5% sclerotia and to 0, 4.5, 9, 18, 35, and 70 mg/kg of alkaloids, respectively. An attempt was made to balance fibre and pre-

dicted digestible energy (DE) between diets. In formulating diets containing ergot, the intention was to minimise differences in nutrient composition, particularly with respect to fibre and digestible energy, so as not to confound any toxic effects. It was anticipated that the high fibre in these diets might limit intake and growth to some extent. Full fat soy meal was added in order to increase the palatability of the diet. Oil was added to increase the DE, but also to reduce dust. The diets formulated are given in Table 1.

There was no indication in the literature that the alkaloids present in ergot were unstable, but to guard against this possibility, the crude ergot was kept at  $-20^{\circ}$ C after milling until the diets were mixed. Following preparation, mixed diets were bagged and stored at  $4-10^{\circ}$ C until required. Only 3–4 days supply was kept at room temperature while feeding the experimental animals.

Representative samples of all diets and their components were taken, hammer-milled, and assayed for nutrients, gross energy, and amino acids.

#### Experimental design and pig husbandry

Forty-eight pigs of both genders and 2 breeds (Large White and Duroc) and about 20 kg liveweight were stratified by liveweight and divided into a heavy and light group, each containing 6 pigs of each gender and each breed. The heavy and light blocks were allocated each to one side of a single pig grower shed equipped with individual pens. Within each block, pigs of each gender and breed were randomly allocated to individual pens, and pens randomly allocated to each of the 6 diets. After a settling-in period of 5 days when all pigs were offered a standard grower mash diet based on sorghum, the experimental diets were offered for 28 days, during which pig performance was monitored. Feed consumption was individually monitored daily for the first week and weekly thereafter, while liveweight was monitored weekly. Each pig was bled from the anterior *vena cava* on Days 0 and 28 of the trial, and the samples were analysed for prolactin and general clinical, biochemical, and haematological parameters.

	Diet no.:	1	2	3	4	5	6
		Con	nponent				
Ergot-rich sorghum		0	25	50	100	200	400
Sorghum (9% crude protein)		244	278	317	336	346	404
Wheat pollard		410	354	286	240	180	0
Lucerne meal		100	100	100	87	50	0
Tuna meal (56% crude protein)		70	70	70	73	75	85
Full fat soybean meal		74	80	90	87	87	79
Vegetable oil		90	81	75	65	50	20
L–lysine HCl		2.5	2.5	2.5	2.5	2.5	2.5
DL-methionine		1	1	1	1	1	1
L-threonine		1.2	1.2	1.2	1	1	1
Dicalcium phosphate		5	5	5	5	5	5
Salt		2.5	2.5	2.5	2.5	2.5	2.5
Vitamin/mineral premix							
		Nu	trients				
Ergot sclerotia (g/kg)		0	3	6	13	25	50
Ergot alkaloids (mg/kg)		0	4.5	9	18	35	70
Crude fibre (g/kg)		67	67	67	67	67	70
Crude protein (g/kg)		174	174	173	173	174	173
Calcium (g/kg)		8.9	8.9	8.8	9.0	8.7	8.9
Phosphorus (g/kg)		6.3	6.2	6.0	6.0	6.0	6.0
Digestible energy (MJ/kg)		14.6	14.5	14.6	14.5	14.6	14.6
Lysine/energy (g/MJ)		0.67	0.67	0.67	0.67	0.67	0.67

Table 1. Formulation of diets (g/kg) and predicted nutrient composition

#### Measurement of nutrient digestibility

The digestibility of 4 of the formulated diets was determined retrospectively after the growth trial due to the limitation of ergot-rich sorghum. Eight entire male Large White pigs of about 20 kg liveweight were used to assess faecal dry matter and energy digestibility, and nitrogen retention. The design was an incomplete  $4 \times 2$  latin square for 4 treatments assessed over 2 periods, and using 8 pigs (2 replicates pigs/treatment.period) individually penned in a randomised block layout of pens in each period. Pigs were penned in metabolism crates kept in a draught-free, air-conditioned room maintained at 21°C. Pigs were offered feed and water twice daily at 0730 hours and 1530 hours, to a total of 900 g feed plus 2.7 L of water (Period 1) and a total of 1100 g feed plus 3.3 L of water (Period 2). Following an initial adaptation period of 7 days, total feed intake was recorded for another 5 days known as the feeding period, with collection and retention of all faecal output from that 5-day feeding period using the ferric oxide dye procedure. In this procedure, upon the initiation of the 5-day feeding period, red dye (10 g ferric oxide/kg of diet) was included and mixed well into the first meal fed to the pigs. Upon appearance of the red dye in the faeces, all red faecal material and that following was collected twice daily, bulked, and frozen. Ferric oxide was included in the first meal given to the pigs following the conclusion of the 5-day feeding period. When this second dose of red dye appeared in the faeces, all red faecal material and that following were excluded from the 5-day collection period.

#### Clinical, biochemical, and haematological analyses

Blood samples were assayed for haemoglobin, packed cell volume, erythrocyte count, mean corpuscular haemoglobin, mean corpuscular volume, and leucocyte count. Plasma samples were assayed for calcium, magnesium, albumin, globulin, creatinine, urea, bilirubin, gamma glutamyl transferase, glutamate dehydrogenase, aspartate aminotransferase, and creatine phosphokinase using standard methods.

#### Hormone assays

Prolactin concentrations were determined by radioimmunoassay (Downing et al. 1995). The antiserum (AFP-753180) was diluted in phosphate buffer to give an initial dilution of 1:20000. All prolactin standard reference preparations (AFP-4835B) and the quality control samples were prepared in prolactin-free plasma. Prolactin (AFP-4835B) was iodinated by the chloramine-T method. For use in the assay, the label was diluted in phosphate buffer with EDTA to give 15000 cpm in 50 µL. All samples were assayed in triplicate using 10–50  $\mu$ L of plasma. The sensitivity of the assay (n = 5) at 90% displacement is 0.045 ng/tube. The intra-assay and inter-assay coefficients of variation were estimated using 3 quality controls: low (5.8  $\mu$ g/L), 12.4% and 15.3%; medium (16.8 µg/L), 3.6% and 10.9%; and high (54.9  $\mu$ g/L), 7.6% and 10.5%, respectively. The detection limit for the method as used with these samples was <0.15 µg/L, and when prolactin was below the limit of detection, the actual value was taken to be 0.08 µg/L for statistical purposes.

#### Statistical analyses

The main effects of diet, sex, and breed, and their interactions, were tested using a randomised-blocks analysis of variance model. The individual pig (pen) was the experimental unit, so error was estimated from pig-to-pig variation. Main effect and interaction means were compared using the protected l.s.d. procedure at P = 0.05. Prolactin concentrations were transformed to the logarithmic scale prior to analysis. The log prolactin v. ergot concentration response curve was modelled using non-linear regression.

#### Results

#### Ergot and feed analyses

The total alkaloid content of a few mature ergot sclerotia was found to be 2200 mg/kg (0.22%). The primary component (>90%) was dihydroergosine, which was consistent with *C. africana* (Frederickson *et al.* 1991). The alkaloid content of the ergot-rich sorghum was assayed at 150 mg/kg, indicating a lower average alkaloid content of sclerotia than the few selected for assay. The diet estimated as containing 5% sclerotia was assayed and contained 70 mg total alkaloids/kg. This diet was also assayed for trichothecenes and zearalenone with negative results (<0.1mg/kg of deoxynivalenol, nivalenol, and HT-2 Toxin; <0.15 mg/kg of T-2 Toxin and diacetoxyscipenol; and <0.025mg/kg of zearalenone).

Proximate and amino acid assay results on the ergot-rich sorghum were (g/kg dry matter): sodium chloride, 50; ash minus sodium chloride, 35; crude fibre, 127; ether extract, 47; protein (N × 6.25), 127; lysine, 5.6; histidine, 2.5; arginine, 6.3; aspartic acid, 10.2; threonine, 5.0; proline, 8.6; serine, 5.9; glutamic acid, 19; glycine, 5.2; alanine, 9.1; valine, 6.3; methionine, 1.42; isoleucine, 6.1; leucine, 12.5; tyrosine, 4.0; phenylalanine, 5.6; cystine, 1.67; and gross energy, 18.4 MJ/kg.

#### Clinical observations and husbandry

During the trial, all pigs were observed daily for clinical signs associated with rye ergotism (lameness, peripheral gangrene, nervous disorder, etc.) or other abnormal signs, and none were detected, apart from one pig with lameness from a mild abrasion of the footpad that healed quickly after application of topical antibiotic cream. No behavioural changes were observed in the pigs fed ergot compared with controls. Minimum ambient temperatures during this trial ranged from 17 to 25°C (average 22.1°C). Maximum temperatures ranged from 27 to 39°C (average 31.3°C). Water misting equipment was used to cool pigs in hot weather (>30°C, approximately).

#### Biochemical and haematological results

There were no significant treatment effects (P > 0.05) with respect to any of the haematological indices. Of the biochemical indices, there were only minor, albéit significant treatment effects (P < 0.05) for plasma albumin, calcium, and glucose, which are shown in Table 2. The values shown are for the second bleeding, after treatment, and also as the difference between these values and those taken pre-treatment.

Plasma prolactin was significantly depressed (P < 0.05) by treatment as shown in Table 3 and the trends are also depicted in Fig. 1.

#### Feed intake, growth, and feed digestibility measurements

The feed intake and growth performance data are shown in Table 4. There were no significant (P > 0.05) main effects of

Parameter		l.s.d.					
	0	4.5	9	18	35	70	(P = 0.05)
Albumin (g/L)							
Post-treatment	35.7a	33.3ab	32.3ab	34.7a	35.4a	29.6b	3.7
Change	2.5	3.3	3.0	2.3	1.7	-3.8	5.0
Calcium (mM/L)							
Post-treatment	2.98	2.92	2.89	2.95	2.93	2.83	0.11
Change	0.06a	0.10a	0.12a	0.05a	0.07a	-0.11b	0.13
Glucose mM/L							
Post-treatment	8.8	9.5	7.9	9.2	8.7	7.7	1.4
Change	3.0ab	4.4a	3.1ab	2.9bc	2.8bc	1.6c	1.4

 Table 2.
 Effects of sorghum ergot alkaloids on plasma albumin, calcium, and glucose following treatment, and the change (post-treatment minus pre-treatment) in these parameters over the 28-day experimental period

 Within rows, means followed by the same letter are not significantly different

Table 3.	Effects of varving	levels of sorohum	ergot alkaloids on	nlasma prolactin
Table 5.	Effects of varying	icvers of solghum	ci got ainaioius on	$\mu_{1}a_{3}\mu_{1$

Means followed by the same letter are not significantly different

Parameter	Ergot alkaloids in diet (mg/kg)						l.s.d.
	0	4.5	9	18	35	70	(P = 0.05)
Initial prolactin $[-\log_e(1+x)]^A$	0.861	0.434	0.570	0.885	0.653	1.011	0.745
Initial prolactin $(\mu g/L)^{B}$	0.42	0.65	0.57	0.41	0.52	0.36	0.000
Final prolactin [–log <sub>e</sub> (1+x)] <sup>A</sup> Final prolactin (µg/L) <sup>B</sup>	0.075a 0.93	0.939b 0.39	1.457b 0.23	1.566bc 0.21	2.340cd 0.10	2.396d 0.09	0.800

<sup>A</sup>Transformed mean values. <sup>B</sup>Back-transformed mean values.



**Fig. 1.** Blood prolactin concentrations of pigs before and after feeding sclerotia of *Claviceps africana* (sorghum ergot) for 28 days.

sex or sex × treatment interaction effects. Feed intakes, feed conversion, and weight gain were significantly (P < 0.05) worsened only in the 70 mg alkaloids/kg group. At this ergot concentration, mean feed intake over the initial 7 days was sig-

nificantly (P < 0.05) reduced in the Duroc breed, but not in Large Whites. This pattern continued over the trial period, and after 28 days, the mean feed intake of Duroc pigs was 0.929 kg/day compared with 1.228 kg/day for Large White pigs.

Feed digestibility and nitrogen retention data are shown in Table 5. Digestibility of both energy and nitrogen were significantly (P < 0.05) poorer in pigs fed 70 mg ergot alkaloids/kg.

#### Discussion

#### Implications of prolactin depression

The most obvious finding was that plasma prolactin concentrations were significantly depressed in pigs receiving diets with 9 mg alkaloids/kg (0.6% sclerotia). In pigs receiving 35 and 70 mg alkaloids/kg, this depression was >80%, clearly indicating a potential to interfere with lactation if breeding sows are fed C. africana sclerotia. Trends shown in Fig. 1 suggest depression even with 4.5 mg alkaloids/kg (0.3% ergot sclerotia), but the high mean prolactin for the control pigs compared with pre-treatment values confounded the issue. At the same time as these results became known, reports were received from several piggeries in central Queensland of severe feed refusal and death of piglets (Blaney et al. 2000b). Sows were reported as farrowing normally and producing apparently healthy piglets but failing to lactate. Samples of sorghum from these piggeries contained 1-31% by weight of crude ergot. Concentrations of alkaloid

Variable Ergot alkaloids in diet (mg/kg) l.s.d. (P = 0.05)0 4.5 35 70 9 18 22.0 21.7 20.721.121.720.8Initial weight (kg) 1.4 39.4a 37.9ab 36.9b 37.7ab 37.6ab 32.1c 2.3 Final weight (kg) Feed intake days 1-7 (kg/day) Large White 1.104a 1.061ab 1.175a 0.996ab 0.986ab 1.061ab 0.211 Duroc 1.089a 1.039ab 0.764c 1.082a 0.857bc 0.698c Feed intake days 1-28 (kg/day) 1.330a 1.078b 1.388a 1.376a 1.315a 1.339a 0.139 FCR days 1-28 (kg/kg) 2.229a 2.377a 2.285a 2.262a 2.336a 2.715b 0.225 Weight gain days 1-28 (kg/day) 0.623a 0.580a 0.578a 0.594a 0.406b 0.069 0.569a

 Table 4.
 Effects of varying levels of sorghum ergot alkaloids on feed intake, feed conversions, and weight gain of pigs

Within rows (main effects) or 2 rows (2-way interaction table of means), means followed by the same letter are not significantly different

Table 5. The digestibility of dry matter (DM), gross energy (GE), and nitrogen (N), and digestible energy (DE) contents of 4 selected diets with different ergot alkaloid concentrations

Within rows, means followed by the same letter are not significantly different

Attribute	Е	()	l.s.d.		
	0	9	35	70	( <i>P</i> = 0.05)
DM digestibility (%)	70.4b	73.7a	72.0ab	70.6b	2.2
GE digestibility (%)	70.0b	72.8a	70.7ab	68.9b	2.1
N digestibility (%)	73.6a	75.0a	70.3a	63.0b	4.8
DE (MJ/kg, 'as is' basis)	13.16a	13.22a	12.35b	11.51c	0.38

in the farm diets ranged from 5 to 40 mg/kg (Blaney *et al.* 2000*b*). When these observations are considered in conjunction with the experimental evidence shown here of reduced plasma prolactin concentrations, there can remain little doubt that sorghum ergot can have similar effects to rye ergot in reducing the milk production of pigs.

#### Palatability and feed refusal

Reductions in feed intake were observed over the first 7 days with diets containing 9 mg alkaloids/kg (0.6% ergot sclerotia) and more, but tolerance appeared to develop over time. In the cases of sorghum ergot intoxication in central Queensland piggeries, the extent of feed refusal varied from farm to farm, at worst depressed by about 50% (Blaney et al. 2000b). However, this was confounded by the fact that farms were changing from a barley-based diet to sorghum-based, which normally does produce a minor reduction in voluntary feed intake lasting several days. Our experimental pigs were accustomed to sorghum-based diets, and the test diets did include full-fat soybean meal, which has a pleasant nutty flavour (to human taste) in the hope that it would overcome any palatability problems. Whittemore et al. (1977) found that pigs fed 2.5% rye ergot sclerotia (75 mg alkaloids/kg) had severe and persistent feed refusal, although control pigs were also intake-restricted so the full extent of refusal was not evaluated.

In broad terms, appetite of pigs tends to be controlled by demand for energy. DE contents of the diets ranged from 11.51 MJ/kg in the highest ergot diet to 13.16 MJ/kg in the

control diet (Table 5). However, voluntary feed intake of this class of pig (20 to 50 kg liveweight) is limited by gut capacity and has been shown to be unaffected by dietary DE concentration from 11.8 to 15.5 MJ/kg (Campbell and Dunkin 1990). The persistently reduced feed intakes of pigs at higher ergot inclusion levels was, therefore, not likely a consequence of the lower digestible energy of those diets. On the other hand, the effects on intake of poor palatability usually do not persist to this extent. The main alkaloid in sorghum ergot (dihydroergosine) has been reported to have a sedative effect on rodents (Manev et al. 1989), which might have reduced intakes. But some ergot alkaloids, such as ergotamine, are known to produce nausea and emesis, which would have the same effect on intakes. Trichothecene mycotoxins, such as deoxynivalenol and nivalenol, produce severe and persistent feed refusal in pigs (Williams and Blaney 1994). These were assayed in the diet containing most ergot, with negative results.

#### Feed utilisation and growth

Over the full period of the trial, growth was reduced by about 30% in pigs receiving 70 mg alkaloids/kg (5% ergot sclerotia), largely as a result of poor feed intake and poor feed conversion (Table 4). Whittemore *et al.* (1976, 1977) also found reductions in growth of pigs fed rye ergot that were not solely attributed to reduced feed intake. Poor feed conversion might have an infectious, a toxicological, or a nutritional cause. With regard to an infectious or toxicological cause of poor feed utilisation, there was no sign of illness including diarrhoea in any of our experimental pigs. It is possible that the poorer digestion of nutrients measured may have been partly a result of increased peristalsis through the colon due to ergot alkaloids (Nickerson 1970). The high salt content of the 70 mg/kg alkaloid diet (2.5% on a DM basis) might have exacerbated this, although pigs can tolerate up to 13% salt if abundant fresh water in available (Williams 1990).

With regard to a nutritional cause of poor feed utilisation, all diets had been formulated in an attempt to balance nutrients, but the availability of those nutrients was uncertain. The digestibility results, obtained after the growth trial was completed (Table 5), showed a lower actual DE content of the diets than that predicted using assumed feed values for the dietary ingredients. Whatever the cause of the poor DE, to ensure near-maximum energy utilisation and growth performance, grower diets are recommended (Campbell and Dunkin 1990) to contain 14 MJ DE/kg for *ad libitum* fed pigs (20–50 kg). It seems that in the current experiment, poor feed conversion in the high ergot diet could simply be attributed to a low DE intake per day as a consequence of low feed intake and lower DE value of diets.

The digestibility data in Table 5 showed a significantly poorer nitrogen digestibility and retention in pigs fed the diet containing 70 mg alkaloids/kg. The 10% depression in plasma albumin in this group of pigs suggests that protein metabolism was inhibited. This might have been due to poor protein quality (discussed next section), or to energy intake being limiting (discussed previous paragraph). Low blood glucose would have supported the latter explanation, but all pigs were hyperglycaemic at the second bleeding date, for which we have no explanation other than stress; possibly the pigs reacted more to the stress of bleeding on the second occasion, being older. There was a definite trend for the difference in glucose concentration between bleedings to be reduced with increasing ergot concentrations; put another way, if stress was the cause of the hyperglycaemia, then ergot reduced either the basal glucose concentrations or else the stress of bleeding. The slight reductions in calcium corresponding to reduction in albumin may simply reflect the fact that calcium is transported bound to albumin.

#### Nutrient changes

The most prominent differences in nutrient composition of the ergot-rich sorghum compared with that expected of sorghum grain of this protein content were as follows. The crude fibre (20–30 g/kg is usual for sorghum) and ash (20 g/kg is usual) were high, while the ether extract and gross energy were normal. A high fibre concentration was expected because of the cell wall components of the glumes and stalk fragments. The main component of the ash was salt introduced during the ergot-separation process, but the glumes may also have a higher ash content than grain. Protein calculated from total nitrogen appeared normal, but the poor nitrogen digestibility (Table 5) in pigs fed the highest ergot alkaloid diet raises doubts about protein quality. The amino acids were variable: lysine (3 g/kg is usual) and threonine (3.8 g/kg is usual) were higher than expected, while glutamic acid was depressed; others were either normal, or slightly above or below normal. A low glutamic acid concentration has been observed in mouldy grain previously, and may reflect utilisation by the ergot fungus (Mannion *et al.* 1987). A closer examination of the effect of the ergot fungus on the nutritional value of grain is needed.

#### Circulatory and other toxic effects

There were no clinical signs of lameness, or lesions to the feet, tail, or ears suggestive of decreased peripheral circulation. Whittemore et al. (1977) similarly found no sign of peripheral necrosis in any of the extremities of pigs fed up to 75 mg rye ergot alkaloids/kg diet, but they did report evidence of a toxic insult, including erosive gastritis, enteritis, and hepatic fibrosis. However, their descriptions of these abnormalities are difficult to interpret clearly. We did not necropsy our trial pigs, but there was no evidence of illness in the blood indices. The vascular effects of some ergot alkaloids are made worse by extremes of temperature, leading either to peripheral gangrene in persistent sub-zero temperatures or hyperthermia in tropical conditions. Bakau et al. (1988) considered this aspect by feeding rye ergot to pigs housed at either 15°C or 35°C. While the higher temperature exacerbated the reduced feed intake, there was no evidence of vasoconstriction or gangrene, supporting previous reports that pigs are more resistant than cattle to this expression of ergot poisoning. Moreover, it has been reported that dihydrogenated alkaloids (such as dihydro-ergosine) are less active than the parent alkaloids in interfering with peripheral circulation.

#### Conclusions

The value of fungus-infected grain is generally a consequence of affects on palatability, nutritional content, and risk of toxicity (Blaney and Williams 1991). The current results indicate that grower pigs over 20 kg in weight and accustomed to sorghum based diets were able to tolerate up to 35 mg alkaloids/kg (2.5% sorghum ergot sclerotia). As a consequence, there is some justification for raising the regulated limit for sorghum ergot in stock feeds above the 0.3% currently set in Australia. However, performance in longer feeding studies should be investigated.

More investigation is also required to assess whether different batches of infected grain have different palatability to pigs. It must be stressed however, that the use of ergot in sow feed is likely to have drastic consequences in terms of deaths and reduced growth of piglets as a consequence of inhibited lactation, and further research is required to determine the maximum amount tolerated by pregnant and lactating sows.

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This trial was approved by the Animal Ethics Committee of the Animal Research Institute.

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