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Tolerance of pigs to sorghum ergot (*Claviceps africana*) during growth and finishing, and effect on conception of replacement gilts

J. S. Kopinski[A,C], *B. J. Blaney*[A] *and J. A. Downing*[B]

[A]Department of Primary Industries and Fisheries, LMB No. 4, Yeerongpilly, Qld 4105, Australia.
[B]Department of Animal Science, University of Sydney, Camden, NSW 2570, Australia.
[C]Corresponding author. Email: John.Kopinski@dpi.qld.gov.au

Abstract. Two batches of sorghum infected with ergot were incorporated into nutritionally balanced grower and finisher diets that contained 0, 5 or 10 mg alkaloid/kg (0, 4 or 8 mg dihydroergosine/kg), or 10 mg alkaloid/kg (8 mg dihydroergosine/kg) plus 1% zeolite. The contents of ergot sclerotia in the 10 mg/kg diets were ~2% in one batch and 4% in the other; the latter batch had a heavy secondary fungal infection of *Cerebella* sp., which tends to limit alkaloid accumulation. These diets were each fed to four male and four female pigs as they grew from 20 to 90 kg. There were no deleterious effects on growth, feed intake and conversion even with lower plasma prolactin of 0.1 μg/L in ergot-fed pigs compared with ~1 μg/L in the control pigs. Zeolite did not counteract the ergot reduction of prolactin and had no effect on performance. Male pigs were then slaughtered, but females continued to be fed the diets for a further 3 months, when they were brought into oestrus and artificially inseminated. One month after pregnancy was confirmed, they were slaughtered and fertility was assessed. There were no significant differences in the numbers of corpora lutea or embryos between pigs fed ergot and control diets.

Additional keyword: mycotoxin.

Introduction

Sorghum ergot was identified in Australia in early 1996 (Ryley *et al*. 1996), and in the following year several piggeries reported agalactia in sows fed ergot, resulting in the death of piglets (Blaney *et al*. 2000*b*). In subsequent experiments, diets containing 1.5% sorghum ergot (7 mg ergot alkaloids/kg) caused complete agalactia (Kopinski *et al*. 2007), arising from reduced plasma prolactin concentrations. It appeared to be possible that sorghum ergot could also affect the performance of growing pigs.

In a previous pig growth experiment involving feeding ergot for 4 weeks (Blaney *et al*. 2000*a*), plasma prolactin was reduced, and this reduction was accompanied by significant reductions in feed intake, feed conversion efficiency and growth in pigs fed 5% sorghum ergot (70 mg alkaloid/kg), but not in pigs fed 2.5% (35 mg alkaloid/kg). Digestibility studies subsequently indicated that the availability of the nutrients in ergotised sorghum could explain the observed responses, especially as reduced digestible energy (DE) for pigs is a common result of heavy ergot infection of sorghum (Kopinski and Blaney 1999).

Secondary infection of honeydew-releasing sorghum heads by *Cerebella* spp. is common, particularly when warm, humid conditions prevail during sorghum maturation, and this appears to restrict development of sclerotia and limit alkaloid concentrations (Blaney *et al*. 2003). It was not known if infection of sorghum with *Cerebella* sp. affects toxicity or palatability *per se*.

Zeolites and other aluminosilicates have been used successfully to bind certain mycotoxins such as zearalenone (Smith 1980; Lemke *et al*. 1998) and aflatoxin (Harvey *et al*. 1989; Ramos and Hernandez 1996, 1997) and prevent their absorption in the gut (see Huwig *et al*. 2001 for a review of adsorbents), although bentonite was ineffective for zearalenone and nivalenol (Williams *et al*. 1994). Aside from ergotamine binding by calcium montmorillonite (Huebner *et al*. 1999), it is not known if such binders might also decrease absorption of ergot alkaloids and counteract the lowering of plasma prolactin caused by ergots.

The current study had several aims. Firstly, to determine if diets with 5 and 10 mg ergot alkaloids/kg (1–4% ergot depending on the batch) were tolerated by pigs if fed for the entire grower and finisher periods, providing that the diets were formulated to minimise nutritional imbalances. These alkaloid levels are within the range of those reported (Blaney *et al*. 2003) for natural ergot infection. Secondly, to determine whether this long-term ergot feeding would affect the subsequent fertility of females retained as replacement gilts. Thirdly, to compare two different batches of ergotised sorghum, one with a heavy secondary fungal infection with *Cerebella* sp., to determine whether the pigs performed differently. Finally, to assess whether the use of zeolite at 1% of the diet would improve pig performance by binding the alkaloids and thereby preventing any lowering of prolactin.

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Materials and methods

Ergot-infected sorghum and diets

Sorghum grain infected with ergot was sourced from two farms in the Monto region of Queensland where cases of poisoning of pigs had been observed (Blaney *et al*. 2000*b*). These were designated Batches M and V. Ergot sclerotia concentrations were estimated by visual separation and weighing of several subsamples. Batch M contained 4% ergot and Batch V contained 10% ergot, with most of the sclerotia in Batch V also having a black, sooty appearance because of an extensive secondary fungal infection with *Cerebella* sp.

The batches were mixed extensively to ensure homogeneity because previous observations (Blaney *et al*. 2003) showed that the distribution of ergot throughout a batch of sorghum could vary widely. After hammer milling 1-tonne batches of grain, subsamples were taken and assayed for ergot alkaloids by high-performance liquid chromatography using the method described in Blaney *et al*. (2003). Batch M contained 38 mg alkaloid/kg, which included 32 mg dihydroergosine (DHES)/kg, 5 mg dihydroelymoclavine/kg and 1 mg festuclavine/kg, while Batch V contained 58 mg alkaloid/kg, which included 42 mg DHES/kg, 14 mg dihydroelymoclavine/kg and 2 mg festuclavine/kg. The control sorghum contained no detectable alkaloid.

In accordance with these results, sorghum Batch M was incorporated into diets at 12.5% (M1) and 25% (M2). Batch V was incorporated into diets at 10% (V1) and 20% (V2). The DE of these sorghum batches had been determined in a previous experiment (Kopinski and Blaney 1999). By having the ingredient specification DE values for ergot sorghum set at 12.6 MJ/kg for batch M and 12.5 MJ/kg for batch V, the diet formulation (using AUSPIG Feedmania, CSIRO, Armidale, NSW) compensated for this lower energy content of ergot-infected sorghum, thus minimising the risk of a performance effect resulting from a nutrient imbalance. Tables 1 and 2 give details of the grower diets (14 MJ DE/kg; lysine/DE ratio of 0.63) and finisher diets (13.5 MJ DE/kg, lysine/DE ratio of 0.58). Zeolite at a 1% inclusion level was included as a separate treatment group for both higher alkaloid (8 mg DHES/kg) diets (M2 + zeolite and V2 + zeolite). The mixed diets were steam-press pelleted and stored in sealed bulk bins of 700–800 kg capacity. Thus, there were seven diets in all: control, M1, M2, M2 + zeolite, V1, V2 and V2 + zeolite. After pelleting, all diets were sampled and assayed for alkaloids, and within the bounds of sampling and analytical variation these results matched the alkaloid concentrations predicted from original grain assays and formulation. The alkaloid concentrations in the control, M1 and M2 diets were <0.1, 5 and 10 mg alkaloids/kg, providing <0.1, 4 and 8 mg DHES/kg, respectively. Diets V1 and V2 contained 6 and 12 mg alkaloids/kg, which also provided 4 and 8 mg DHES/kg, respectively.

Experimental design and treatments

The experimental design consisted of two phases: phase I was a grower/finisher pig performance assessment using males and females, while phase II examined the effects of ergot on the fertility of the females to model the situation that could

Table 1. Composition of diets, nutrient supply estimates and alkaloid contents for the grower phase I
DE, digestible energy

	Control	V1	V2	V2 + zeolite	M1	M2	M2 + zeolite
Ingredient (kg)							
Sorghum control	596	561	528	510	537	512	453
Sorghum Batch V	0	100	200	200	0	0	0
Sorghum Batch M	0	0	0	0	125	250	250
Millrun	150	100	50	50	100	0	50
Lysine HCl	1.88	2.32	2.77	2.71	2.31	2.72	2.65
Methionine	0.51	0.69	0.87	0.90	0.73	0.75	0.88
Threonine	0.19	0.31	0.43	0.44	0.32	0.36	0.42
Salt	2.0	2.0	2.0	2.0	2.0	2.0	2.0
Vitamin and mineral premix[A]	2.0	2.0	2.0	2.0	2.0	2.0	2.0
Bloodmeal 85	25	25	25	25	25	25	25
Meat and bone meal 50	52	55	57	58	55	58	58
Canola meal	84	67	49	46	62	71	50
Soybean full fat meal	86	85	83	88	89	76	88
Zeolite	0	0	0	10	0	0	10
Choline chloride	0.36	0.36	0.36	0.36	0.36	0.36	0.36
Fat	0	0	0	5	0	0	8
Total	1000	1000	1000	1000	1000	1000	1000
Estimated DE (MJ/kg)	14.0	14.0	14.0	14.0	14.0	14.0	14.0
Estimated avail. Lys/DE (g/MJ)	0.63	0.63	0.63	0.63	0.63	0.63	0.63
Ergot content (%)	0	1	2	2	0.5	1	1
Total alkaloids (mg/kg)	<0.1	6	12	12	5	10	10
Dihydroergosine (mg/kg)	<0.1	4	8	8	4	8	8

[A]1 kg of premix provided the following levels of vitamins and minerals: vit. A 5.5 mIU, vit. D_3 1 mIU, vit. E 27.50 g, vit. K 1.50 g, thiamine 0.75 g, folic acid 0.5 g, niacin 12.5 g, calcium pantothenate 8 g, riboflavin 3 g, vit. B_6 1.75 g, vit. B_{12} 15 mg, biotin 75 mg, Se 150 mg, Co 200 mg, Cu 10 g, Fe 30 g, Mn 22.5 g, Zn 50 g, I 0.75 g, antioxidant 50 g.

potentially arise if they were to be retained as replacement gilts. In phase I, there were 14 treatments (consisting of the seven diets × two sexes) by four blocks, giving a total of 56 pigs. However, an additional four females were fed the control diet in phase I to ensure a balanced design for the control group in phase II.

Sixty large white (LW) purebred pigs (28 entire males and 32 gilts) of 5–6 weeks of age and an initial weight of 18–22 kg were selected from a larger pool of pigs after ranking on liveweight and discarding a few that were weight outliers (light or heavy). Pigs were ear-tagged for identification, then stratified and grouped on sex and liveweight into four blocks. Male pigs remained on their allocated ergot treatment through the change-over from grower to finisher diets until they were slaughtered at the end of phase I of the trial. All female pigs continued on the same diets across phases I and II and were slaughtered at the end of phase II.

In phase II, the seven treatments from phase I were used plus the additional controls and each block of four pigs was group housed and fed for logistical and welfare reasons. However, the group housing restricted the replication, so for statistical analysis, alkaloid level alone was taken into account, with different sorghum batches combined. Consequently, the analysis was for a randomised layout of eight pens, with two replicates × four treatments: control; M1 combined with V1; M2 combined with V2; and M2 + zeolite combined with V2 + zeolite.

Housing

In phase I, pigs were individually housed in grower pens (2.4 m × 1.2 m) constructed of galvanised weldmesh with 30% of the floor as solid concrete and 70% of the floor as clay slat (150 mm wide with a 20 mm space between each slat). Each pen was provided with a drinking nipple that met the recommended welfare guidelines (SCARM 1998; section 3.2). Each pen was also provided with a stainless steel feeder large enough to accommodate *ad libitum* feeding. Control of the environment was by an automatic temperature-controlled shutter system set at 22°C. Although pigs were housed in individual pens, they were able to interact visually with one another. During phase II of the trial, females were housed in groups of four in pens (4 m × 2.4 m) that had 90% clay slats flooring and 10% solid concrete.

Feeding

Pigs were fed the grower diet for the first 6 weeks, which was then substituted with the finisher diet (containing an equivalent alkaloid level) for the next 5 weeks. Individual weekly diet allocations were weighed out at the start of the week into a bin for each animal. Spillage from each animal was collected and placed in a smaller waste bin for each pig. At the end of each week the feed was removed from the pigs for 12 h before weighing to assess liveweight gain for the week. The remaining contents of the feed bin and waste bin of each pig was weighed separately and recorded to get an accurate figure for intake (wastes dried, with intake calculations on the basis of the same dry matter as feed).

Following the conclusion of the finisher phase and 2 weeks after the males had been removed, the females had reached 19 weeks of age and were placed in the group pens with their respective three replicates from phase I. Females would usually

Table 2. Composition of the diets, nutrient supply estimates and alkaloid contents for the finisher phase I and phase II
DE, digestible energy

	Control	V1	V2	V2 + zeolite	M1	M2	M2 + zeolite
Ingredient (kg)							
Sorghum control	625	589	554	587	577	521	555
Sorghum batch V	0	100	200	200	0	0	0
Sorghum batch M	0	0	0	0	125	250	250
Millrun	200	150	100	50	125	75	25
Lysine HCl	1.93	2.36	2.8	2.83	2.32	2.78	2.81
Methionine	0.29	0.36	0.42	0.43	0.3	0.41	0.42
Threonine	0.33	0.44	0.55	0.57	0.38	0.53	0.55
Salt	2	2	2	2	2	2	2
Vitamin and mineral premix[A]	2	2	2	2	2	2	2
Bloodmeal 85	25	25	25	25	25	25	25
Meat and bone meal 50	41	44	46	48	44	47	49
Canola meal	59	45	30	31	63	36	37
Soybean meal 48%	43	40	37	41	34	38	41
Zeolite	0	0	0	10	0	0	10
Choline chloride	0.4	0.4	0.4	0.4	0.4	0.4	0.4
Total	1000	1000	1000	1000	1000	1000	1000
Estimated DE (MJ/kg)	13.5	13.5	13.5	13.5	13.5	13.5	13.5
Estimated avail. Lys/DE (g/MJ)	0.58	0.58	0.58	0.58	0.58	0.58	0.58
Ergot content (%)	0	1	2	2	0.5	1	1
Total alkaloids (mg/kg)	<0.1	6	12	12	5	10	10
Dihydroergosine (mg/kg)	<0.1	4	8	8	4	8	8

[A]1 kg of premix provided the following levels of vitamins and minerals: vit. A 5.5 mIU, vit. D_3 1 mIU, vit. E 27.50 g, vit. K 1.50 g, thiamine 0.75 g, folic acid 0.5 g, niacin 12.5 g, calcium pantothenate 8 g, riboflavin 3 g, vit. B_6 1.75 g, vit. B_{12} 15 mg, biotin 75 mg, Se 150 mg, Co 200 mg, Cu 10 g, Fe 30 g, Mn 22.5 g, Zn 50 g, I 0.75 g, antioxidant 50 g.

be allocated to a gilt developer ration at this stage, but as the proposed nutrient composition of the gilt developer was very similar to our finisher diets, we continued to feed the finisher diets *ad libitum* to each group of four females.

Health monitoring

Feed consumption was monitored twice per day along with observations of general alertness, appetite, health and overall condition, and any abnormalities were recorded. Pigs were removed from the trial if they failed to gain weight over a week or if they appeared sick and did not respond to treatment. Backfat was measured on two occasions using an ultrasonic backfat tester at the conclusion of the grower and finisher phase of the trial.

Artificial insemination and fertility assessment

Females retained for the second phase of the study were given several days of close proximity to a training boar to enable maximum synchronisation of pubertal oestrus (Brooks and Cole 1970) and those in apparent oestrus were artificially inseminated (AI) at 31 weeks of age. This procedure was repeated for those returning to oestrus. Pregnancy was confirmed by observation of non-return to oestrus at ~34 weeks of age. At ~36 weeks, pigs were slaughtered and the reproductive tract was removed intact and the corpora lutea (CL) and embryos were counted. Insemination was not successful with some pigs, largely because of lameness (this results in agitated sows that are less receptive to AI; additionally this discomfort leads to the release of adrenalin, which counteracts the oxytocin release that normally stimulates uterine contractions and fertilisation).

Blood collection

Pigs were bled before commencement of the trial (~56 days of age), at ~84 days of age (before completion of the grower phase) and following the finisher phase (133 days of age), and in the case of females also at ~211 days of age. Blood was collected either from the anterior vena cava (when <40 kg) or from the auxiliary vein (Muirhead 1981), with a maximum of 10 mL collected. Time of restraint of the animal was minimised by having two persons hold the pig while another bled. Plasma prolactin concentrations were determined by radioimmunoassay (Blaney *et al*. 2000*a*).

Animal ethics approval

This research was carried out with adherence to the 'Australian code of practice for the care and use of animals for scientific purposes' (NHMRC 1997) and the 'Model code of practice for the welfare of animals, Pigs' (SCARM 1998). Animal ethics approval from the Animal Research Institute Animal Ethics Committee for the conduct of the study was gained before commencement of the work (Approval No. ARI/034/2000-1).

Statistical analysis

A randomised blocks analysis of variance (ANOVA) model (phase I) and a completely randomised one-way ANOVA model (phase II) were used to test for treatment differences using an error term estimated from pen to pen variation. The ergot treatment means were compared using the protected least significant difference (l.s.d.) test operating at a $P = 0.05$ level of significance. Hence, in the result tables, no pairwise testing of the means was carried out if the F-value for treatments was not significant. Plasma prolactin data were cube-root transformed (prolactin concentration$^{1/3}$) before ANOVA. Prolactin assay results below the detection limit of 0.15 ug/L were allocated a value of 0.075 µg/L before transformation. Embryo implantation and CL data were square-root transformed (count + 0.5)$^{1/2}$ before ANOVA. Ratios of embryos : CL were inverse sine [(percentage/100)$^{1/2}$] transformed before ANOVA.

Results

All diets were readily accepted by most pigs. Persistent and severe feed refusal was observed in only two pigs, both of which were found to have gastric ulcers. One of these pigs was removed from the trial on welfare grounds and, following euthanasia, a category-3 oesophageal gastric ulcer (healing) (see Kopinski and McKenzie 2007) was observed, which due to stenosis was constricting the stomach opening and limiting the entry of food. The distribution of gastric ulcers across different treatment groups, including the control, indicated no relationship to ergot.

In Phase I the performances of pigs in the grower phase (20–50 kg) and the finisher phase (50–90 kg) are shown in Table 3. The only significant difference noted was an improved average daily gain for the V2 + zeolite treatment compared with

Table 3. Performance of pigs fed various batches and levels of sorghum ergot in phase I (day 0 to day 77) of the study

Within columns, means followed by a different letter are significantly different at $P = 0.05$. There was no pair-wise testing of means if the F-value for the treatments was not significant. DHES, dihydroergosine

Treatment	Alkaloid (mg/kg)		Liveweight (kg)		Daily feed intake (kg/day)			Average daily gain (kg/day)			Feed conversion (feed:gain)			Backfat (mm)	
	Total	DHES	Initial	Final	Grower period	Finisher period	Entire trial	Grower period	Finisher period	Entire trial	Grower period	Finisher period	Entire trial	End grower period	End finisher period
Control	0	0	18.5	87.8	1.58	2.40	1.96	0.83	0.98ab	0.90ab	1.91	2.45	2.17	8.7	10.9
M1	5	4	18.6	89.0	1.56	2.41	1.95	0.86	0.98ab	0.92ab	1.81	2.45	2.12	9.4	12.7
V1	6	4	18.4	93.2	1.67	2.57	2.07	0.89	1.07bc	0.97bc	1.87	2.41	2.14	8.9	11.9
M2	10	8	18.9	91.1	1.67	2.50	2.05	0.89	1.00ab	0.94abc	1.89	2.50	2.18	10.0	13.0
V2	12	8	19.0	86.0	1.52	2.25	1.85	0.82	0.93a	0.87a	1.86	2.44	2.14	9.0	12.4
M2 + zeolite	10	8	19.1	89.8	1.59	2.44	1.97	0.84	1.01abc	0.92ab	1.89	2.41	2.15	9.5	13.0
V2 + zeolite	12	8	18.8	95.0	1.67	2.60	2.10	0.89	1.11bc	0.99c	1.89	2.35	2.12	9.2	12.3
l.s.d. ($P = 0.05$)			1.29	5.99	0.142	0.256	0.178	0.066	0.103	0.071	0.096	0.142	0.104	1.52	2.05

the control and V2 treatment groups over the entire trial period. There was also a significant difference during the finisher phase for the V2 + zeolite compared with the V2, but it was not a significant difference when compared with the control treatment. Zeolite did not improve gain in the M2 treatment.

The performance of group-housed gilts in Phase II is shown in Table 4, with no significant differences observed in gilt liveweights and feed conversions. There was, however, a significantly higher daily feed intake in M1 + V1 gilts than in the control pigs. A lower daily feed intake in the M2 + V2 was also observed, but this was not significantly different to the control group. The pattern of daily gain reflected daily feed intakes of gilts in phase II, but the differences between treatments in daily weight gain were not significant.

Table 4. Performance of gilts fed various levels of sorghum ergot in phase II (day 78 to day 194) of the study

Within columns, means followed by a different letter are significantly different at $P = 0.05$. There was no pair-wise testing of means if the F-value for the treatments was not significant. DHES, dihydroergosine

Treatment	Alkaloid (mg/kg)		No. of sows	Liveweight (kg)						Daily feed intake for 119 days	Daily gain	Feed conversion
	Total	DHES		Day 0	Day 17	Day 36	Day 64	Day 86	Day 116	(kg/sow)	(kg/sow)	
Control	0	0	8	90	107	123	147	167	192	3.24bc	0.879	3.75
M1 + V1[A]	5–6	4	8	93	109	126	153	175	203	3.50a	0.943	3.71
M2 + V2[A]	10–12	8	8	89	103	121	144	161	197	3.14c	0.855	3.68
M2 + V2 + zeolite[A]	10–12	8	8	93	110	128	153	174	201	3.39ab	0.931	3.64
l.s.d. ($P = 0.05$)				9.6	16.3	16.3	21.7	22.2	19.5	0.223	0.136	0.34

[A]Two group-housed pens of four pigs per treatment. The analysis was based on alkaloid content, ignoring batches V or M.

Table 5. Plasma prolactin concentrations in pigs fed sorghum ergot during growth and finishing

Within columns, means followed by a different letter are significantly different at $P = 0.05$. There was no pair-wise testing of means if the F-value for the treatments was not significant. DHES, dihydroergosine

Treatment	Alkaloid (mg/kg)		Plasma prolactin concentration (μg/L)							
	Total	DHES	Phase I[A]		Phase 1[A]		Phase 1[A]		Phase 2[B]	
			Pretreatment		End of grower period		End of finisher period		Gilt period	
			Geometric mean[C]	Transformed mean[D]	Geometric mean	Transformed mean	Geometric mean	Transformed mean	Geometric mean	Transformed mean
Control	0	0	1.04	1.01	1.04	1.01a	1.39	1.12a	1.27	1.08
M1	5	4	1.30	1.09	0.22	0.61b	1.14	1.04a	1.96	1.25
V1	6	4	1.44	1.13	0.34	0.70b	1.77	1.21a		
M2	10	8	1.19	1.06	0.11	0.48c	0.43	0.76b	0.46	0.77
V2	12	8	0.63	0.86	0.08	0.44c	0.22	0.60bc		
M2 + zeolite	10	8	0.95	0.98	0.11	0.48c	0.11	0.48c	0.58	0.83
V2 + zeolite	12	8	0.85	0.95	0.09	0.46c	0.14	0.52c		
l.s.d. ($P = 0.05$)				0.204		0.121		0.219		0.59

[A]Statistically analysed as seven treatments with four replicates × two sexes × seven diets = 56 individual pigs in a randomised block layout.
[B]Treatments were combined for statistical analysis in phase II: M1 + V1; M2 + V2; M2 + zeolite + V2 + zeolite.
[C]Back-transformed mean.
[D]Cube-root transformed (prolactin concentration$^{1/3}$).

Table 6. Embryo implantation and corpora lutea in gilts fed various sorghum ergot alkaloid levels

Within columns, means followed by a different letter are significantly different at $P = 0.05$. There was no pair-wise testing of means if the F-value for the treatments was not significant. DHES, dihydroergosine

Treatment	Alkaloid (mg/kg)		No. of corpora lutea		No. of embryos		Percentage embryo/corpora lutea	
	Total	DHES	Geometric mean[A]	Transformed mean[B]	Geometric mean	Transformed mean[B]	Geometric mean	Transformed mean[C]
Control	0	0	13.7	3.77	10.0	3.25	76.2	1.06
M1 + V1	5–6	4	14.0	3.81	10.6	3.33	82.0	1.13
M2 + V2	10–12	8	13.0	3.67	9.3	3.13	80.1	1.11
M2 + V2 + zeolite	10–12	8	16.6	4.14	13.6	3.75	83.5	1.15
l.s.d. ($P = 0.05$)				0.59		0.54		0.35

[A]Back-transformed mean.
[B]Square-root (count + 1/2) transformed.
[C]Inverse sine [(percentage/100)$^{1/2}$] transformed.

Prolactin results are given in Table 5. There were significant reductions in prolactin compared with the controls in all ergot treatments by the end of the grower phase, but only in the higher ergot treatments by the end of the finisher phase. Zeolite did not have any effect in countering this reduction.

In the later stage of phase II, the size of the pigs and the smooth concrete floors produced some lameness, which impacted on the insemination and subsequent collection of data from those gilts. As a result of lameness, two pigs were culled early before AI could be performed (one from the control group and one fed the V2 diet). The lameness was not considered to be treatment-related and close examination ruled out any lesions suggestive of gangrenous ergotism in the feet and ear tips. Of the 30 gilts remaining, 24 were artificially inseminated at the third heat after induction by exposure to a boar. Four other gilts were inseminated 2 weeks later at their third heat. The remaining two pigs (from the control and the V2 groups), both with slight reddening of the vulva suggestive of heat, would not stand for AI despite two attempts.

The reproductive performance of gilts is shown in Table 6. The numbers of CL and embryos were very similar across all treatment groups.

Discussion

Growth and feed utilisation

The results indicated no adverse effect of feeding either batch of ergotised sorghum on the performance of the growing pig. The presence of the *Cerebella* sp. infection also appears to have had no adverse effect on the growth of pigs. Our current results support previous conclusions (Blaney *et al*. 2000*a*) that grower and finisher pigs are able to tolerate diets containing at least 10 mg/kg of sorghum ergot alkaloids (1–4% ergot depending on the batch) for all of the growth and finisher period (and up to 35 mg/kg for shorter periods: Blaney *et al*. 2000*a*). In contrast, Dignean *et al*. (1986) observed that pigs fed diets with 0.4% rye ergot-infected barley (containing 4 mg alkaloid/kg: 48% ergocristine) converted feed less efficiently to gain despite greater consumption, and suggested that the poor conversion might result from the ergot interfering with hepatic functions. An alternative hypothesis could be that this was a response to lower dietary energy in the ergotised diets, which was not examined by Dignean *et al*. (1986). Our results indicate that growth and feed utilisation will be normal if diets are compensated for the lower DE value of sorghum grain infected by ergot and if palatability is addressed.

Palatability and feed refusal

We found no significant feed refusal by the pigs when offered the sorghum ergot diets containing either 4 or 8 mg alkaloid/kg, while Blaney *et al*. (2000*a*) found mild feed refusal from 9 to 35 mg alkaloid/kg, persisting only in pigs fed 70 mg/kg. Two pigs developed feed refusal later, but this was found to be because of gastric ulcers, unrelated to ergot feeding, as grinding and pelleting of diets increases the risk of ulcers (Chamberlain *et al*. 1967; Nielsen and Ingvartsen 2000).

In the current study and in the study of Blaney *et al*. (2000*a*), full-fat soybean meal was included in grower diets to increase palatability, and previously flavouring agents have been used to overcome the initial feed refusal displayed by sows fed up to 16 mg alkaloids/kg (Kopinski *et al*. 2007). Similarly, Whittemore *et al*. (1977) reported the need to use higher levels of palatable ingredients (such as fishmeal) in diets to counteract the distastefulness of rye ergot-contaminated grain. Without such additives, feed refusal could have been more severe at higher ergot inclusion levels, as has been observed on-farm (Blaney *et al*. 2000*b*).

Comparison of toxicity of sorghum ergot to rye ergot

The studies of Friend and MacIntyre (1970) have shown an apparent effect of rye ergot (equivalent to a range of alkaloid contents of 1.5–59 mg/kg, mainly ergocristine) on pig performance, with a lower coefficient of digestibility for nitrogen as well as a lower retention of nitrogen. Friend and MacIntyre (1970) also observed severely reduced feed intakes in growing pigs, and subsequently found a trend for reduced feed intake with 1.5–3 mg alkaloids/kg. Whittemore *et al*. (1976) induced pigs to consume diets containing up to 10% rye ergot (300 mg alkaloids/kg), but intakes remained severely reduced, and prolonged acceptance of diets was only achieved at less than 14 mg/kg. Later Whittemore *et al*. (1977) also found when using a different source of rye ergot that feed intakes of pigs fed 24 mg alkaloid/kg (80% ergotoxine) were not markedly lowered, but at 49 mg/kg appetite was noticeably reduced with some alimentary tract lesions. These researchers suggested that the conflicting responses of animals to ergot were a consequence of the amounts eaten, the toxicity of the sclerotia and the extent of absorption of toxin from the intestinal tract.

Other studies suggest that very young pigs are particularly susceptible to the effects of ergot alkaloids. Mainka *et al*. (2005*a*, 2005*b*) found that the maximum level of rye ergot alkaloid in diets for piglets ranged from 4.7 to 11 mg/kg, and Oresanya *et al*. (2003) found that feed intake of weanling pigs was reduced with only 1 mg alkaloid/kg and 2 mg/kg affected daily gain.

Frederickson *et al*. (1991) has suggested that the alkaloids of sorghum ergot are less toxic than the alkaloids of rye ergot based on laboratory animals. Comparing the above studies to our own, it appears that grower pig feed intakes may be similarly affected by rye and sorghum ergot at alkaloid levels up to 10–15 mg/kg, with some initial feed refusal that can be successfully masked with palatable ingredients and by pelleting diets. However, as ergot alkaloid concentrations increase past ~30 mg/kg, some unique toxic effects decrease the consumption of rye ergot (Whittemore *et al*. 1976), which have not yet been observed with sorghum ergot, although they both affect prolactin similarly.

Plasma prolactin

Ergot consumption reduced prolactin levels substantially and there were trends in the higher alkaloid diets for the lowering of plasma prolactin to be maintained for a longer period. Zeolite did not counteract the prolactin reduction observed and apparently did not prevent absorption of the alkaloid. It is feasible that other binding agents might have more success if they were developed to have a more specific action, as reported for montmorillonite binding of ergotamine (Huebner *et al*. 1999).

The prolactin concentrations determined in this study were similar to those found for grower pigs in our earlier study (Blaney *et al*. 2000*a*), which ranged from 0.09 to 0.9 μg/L and are also comparable to the study of Diekman *et al.* (1983), which ranged from 1.1 to 1.7 μg/L. These prolactin levels are very low in comparison to those detected in peri-parturient sows, which can rise to 50–400 μg/L (Dusza and Krymowska 1981; Kopinski *et al*. 2007). Diekman *et al.* (1983) also observed that the average concentration of serum prolactin did not vary significantly from 10–18 weeks of age (2.1–3.5 μg/L) and likewise in a group of gilts from 19–25 weeks of age (4.6–6.6 μg/L).

Conception and implantation

Several studies in rats and mice have suggested that ergot feeding may influence successful conception and implantation. There are reports of abortion (Nordskog and Clark 1945), pregnancy maintenance, failure or blockage (Shelesnyak 1958; Edwardson and MacGregor 1969; Mantle 1969), while Grauwiler and Schon (1973) reported effects on maternal weight gain and increased prenatal mortality (especially after implantation).

In contrast, pig studies indicate that ergot has little impact on implantation and pregnancy (Campbell and Burfening 1972; Bailey *et al*. 1973; Dignean *et al*. 1986), although Carslon (1986) reported work carried out by Wiernusz and Schneider (1984) of abortion and fetal resorption in gilts. Studies with bromocriptine showed prolactin reduction, but no effect on conceptus survival or maintenance of pregnancy (Young *et al*. 1989; Szafranska and Ziecik 1990*a*, 1990*b*). Wrathall (1975) has suggested that the reason ergot affects mice and rats differently from pigs is that prolactin is required to maintain CL in early pregnancy in mice and rats but is not required for this in pigs.

The results of our study indicate that long-term feeding of ergot sorghum had no effect on implantation and conception, despite substantial lowering of plasma prolactin.

Conclusions

Diets containing sorghum ergot alkaloid at up to 12 mg/kg (8 mg DHES/kg) were not toxic to grower or finisher pigs, with normal performance observed for the entire baconer production cycle, and the fertility of female pigs retained for breeding purposes was not impaired.

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