The use of extrusion to reduce dietary ergot toxicity in growing pigs

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SUMMARY

Ergot, produced by the fungi of the Claviceps genera, infect cereal crops such as rye and wheat when conditions are favourable. Consumption of ergot alkaloids by livestock results in reduced feed intake and growth and a dramatic reduction in the hormone prolactin, which is important for milk production. Previous research at the PSC demonstrated that extreme processing in the form of steam explosion reduced ergot toxicity, however, it is not always practical. Therefore, this trial set out to utilize extrusion as an example of a more commonly used processing technique to determine potential effects on ergot toxicity in growing pigs. A total of 160 grow-finisher pigs were fed 1 of 4 dietary treatments for 56 days in a 2 x 2 factorial design with main effects of ergot level (0 or 4000 ppb) and extruded or not.

Extrusion changed the ergot alkaloid chemistry, specifically a decrease in the R:S epimer ratio. Some reports indicate that toxicity is due primarily to the R epimer, thus a decrease in the ratio would indicate decreased toxicity of the ergot. Pigs fed ergot had decreased growth in phase 1 and overall. There was a tendency for reduced feed intake when the pigs were initially fed the ergot contaminated diets, and a significant decrease from d 29 to 42, throughout phase 2 and overall. There was no effect of ergot on feed efficiency. There were no ergot by extrusion interactions. Serum prolactin was reduced dramatically in the pigs receiving the ergot contaminated feed, however, there was no effect of extrusion, and no ergot by extrusion interaction.

The available data indicates that extrusion does not reduce ergot toxicity. The dramatic decline observed in serum prolactin observed with ergot alkaloids in the diet indicates that these diets should not be fed to sows or gilts destined for the breeding herd.

INTRODUCTION

Ergot alkaloids infect grasses and cereal crops such as rye, wheat, triticale and barley. There are "R" and "S" epimers of each alkaloid and it has been reported in the early literature that only the "R" epimer is toxic. Symptoms of toxicity range from reduced feed intake to gangrene in the extremities. Synthesis or release of the hormone, prolactin, is especially sensitive to ergot toxicity. The negative effects of ergot on prolactin are responsible for the decrease or complete cessation of milk production observed following ergot ingestion by sows.

There is some evidence suggesting that ergot toxicity can be reduced by feed processing, possibly by reducing the R:S ratio. Previously we showed that extreme processing, steam explosion, apparently reduced ergot amount and toxicity. However, steam explosion is not commonly used. Therefore, this trial set out to examine the potential of extrusion as an example of a more commonly used processing technique to determine potential effects on ergot toxicity in growing pigs. This information will be used to develop recommendations on potential processing strategies for pork producers when ergot toxicity is suspected.

Figure 1. Extruder used for this experiment, located at the Canadian Feed Research Centre

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EXPERIMENTAL PROCEDURES

Trial 1: A total of 160 grow-finisher pigs (65 ± 4 kg initial BW) were housed in groups of 5 per pen in a randomized complete block design. The pens were randomly assigned to 1 of 4 dietary treatments arranged in a 2 x 2 factorial design with main effects of ergot level (0 or 4 ppm) and extruded or not. The ergot levels were achieved by formulating a "clean" premix or a premix that contained ergot contaminated wheat screenings to achieve ergot levels of 0 and 240 ppm respectively. Premixes were either extruded at 90 ˚C with 80-100 psi pressure or not. These premixes were then included into the diets to achieve the final 0 or 4000 ppb levels. Diets were typical Western Canadian wheat-barley-soybean meal diets formulated to be isonitrogenous and isoenergetic to meet or exceed the recommended nutrient requirements for growing pigs in each phase (NRC, 2012). Diets were formulated for phase 1 to be fed to 60 to 90 kg BW pigs and phase 2 to be fed to 90 to 120 kg BW pigs.

Blood samples were collected on d7 and d56 for prolactin analyses, and pigs were weighed each week for the first 4 wks of the 56-d period and every 14 d thereafter until the heaviest pig reached market weight. Weight gain and feed disappearance were measured for each period, and feed efficiency (gain:feed) calculated.

Trial 2: A total of 32 growing pigs (75 ± 5 kg initial BW) were individually housed in metabolic crates in a randomized complete block design. Each metabolic crate had a urine collection tray underneath. Pigs were assigned to one of 4 dietary treatments in a 2 x 2 factorial design with main factors of ergot and extrusion. The diets were isonitrogenous and isoenergetic and were offered to provide 3 x maintenance energy requirement, which approximates ad libitum intake.

A 7-d adaptation to the experimental diets was followed by a 4-d collection of fecal and urine samples. These samples were analyzed for N content to allow a determination of the effect of treatment on N balance as an estimation of protein deposition.

RESULTS AND DISCUSSION

In our previous experiments, steam explosion drastically reduced total ergot content. In contrast, total ergot content in the extruded samples were reduced only by about 14%. However, the R:S ratio of the 6 measured ergot alkaloids in the non-extruded sample was 1.1, in contrast to 2.5 in the extruded sample. There was a 12% increase in the "R" epimer and a 50% decrease in the "S" epimer.

Table 1 describes the performance of growing pigs fed diets contaminated with 4 ppm ergot alkaloids, extruded or not, from 65 kg until market. Pigs fed ergot had decreased growth in phase 1 and overall. There was a tendency for reduced feed intake when the pigs were initially fed the ergot contaminated diets, and a significant decrease from d 29 to 42, throughout phase 2 and overall. There was no effect of ergot on feed efficiency, indicating that the effects of ergot were primarily due to the decrease in feed intake. Interestingly, there were no ergot by extrusion interactions for any of the performance data. An interaction would indicate a differential effect of ergot when the screenings were extruded, which would have supported our hypothesis. Thus, the lack of an interaction indicates that despite the changes observed in ergot epimer profile due to extrusion, extrusion had no effect on ergot toxicity in this experiment.

Figure 2. Room used for grow-finisher pig research at the Prairie Swine Centre

Serum prolactin was reduced dramatically in the pigs receiving the ergot contaminated feed, however, there was no effect of extrusion, and comparable to the performance data, no ergot by extrusion interaction (Table 2). The dramatic decline observed in serum prolactin observed with ergot alkaloids in the diet indicates that these diets should not be fed to gilts or sows used for reproductive purposes.

Nitrogen retention was unaffected by ergot alkaloid content of the diet, regardless of extrusion (data not shown).

IMPLICATIONS

The lack of an effect on feed efficiency indicates that ergot alkaloids exert their main effect on growth through decreases in feed intake. Extrusion had no effect on ergot toxicity in this experiment, despite changing the epimer profile. This implies either that changes in the epimer profile were not of sufficient magnitude to observe changes in performance, or that both epimers have toxic properties. Recent in vitro work by others favours the latter conclusion. Unlike what has been reported in the older literature, analysis and reporting of the ergot alkaloids should include both epimers.

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Table 1. The response of growing pigs to diets contaminated with 4 ppm ergot alkaloids, unprocessed or extruded.

Parameter	Ergot, ppm		Extrusion		Pooled SEM	P values[a]	
	0	4	No	Yes		Ergot	Extrusion
BW, kg							
Initial	65.94	65.82	65.87	65.9	1.54	0.84	0.97
d7	72.38	70.89	71.66	71.61	1.53	0.06	0.94
d14	79.5a	77.64	78.73	78.41	1.63	**0.03**	0.70
d21	88.10	85.89	87.30	86.6	1.18	**0.03**	0.55
d28	96.47	93.03	94.91	94.60	1.03	**0.002**	0.76
d42	112.56	109.55	111.33	110.77	1.15	**0.01**	0.62
ADG, kg/d							
d0-7	0.81	0.63	0.72	0.72	0.07	0.09	0.65
d8-14	1.21	1.12	1.20	1.13	0.11	0.65	0.57
d15-21	1.22	1.18	1.21	1.18	0.35	0.13	0.79
d22-28	1.18	1.02	1.08	1.12	0.28	0.35	0.81
d29-42	1.15	1.66	1.17	1.14	0.12	**0.004**	0.82
d43-56	0.92	1.00	0.98	0.93	0.07	0.14	0.17
phase 1[b]	1.07	0.95	1.02	0.99	0.02	**0.001**	0.34
phase 2[b]	0.94	0.96	0.96	0.94	0.12	0.40	0.40
Overall	**1.07**	**1.03**	**1.05**	**1.04**	**0.01**	**0.02**	**0.31**
ADFI, kg/d							
d0-7	2.1	1.92	1.98	2.03	0.07	0.09	0.65
d8-14	2.45	2.38	2.37	2.46	0.11	0.65	0.57
d15-21	3.32	3.06	3.17	3.21	0.38	0.13	0.80
d22-28	3.51	3.39	3.44	3.47	0.28	0.36	0.81
d29-42	3.23	2.92	3.06	3.09	0.12	**0.004**	0.82
d43-56	3.07	2.92	3.07	2.93	0.07	0.15	0.43
phase 1[b]	2.62	2.45	2.51	2.56	0.12	0.18	0.63
phase 2[b]	3.22	3.02	3.15	3.11	0.05	**0.01**	0.54
overall	2.97	2.78	2.88	2.89	0.05	**0.02**	0.95
G:F							
d0-7	0.40	0.33	0.36	0.36	0.02	0.08	0.66
d8-14	0.50	0.48	0.52	0.47	0.02	0.52	0.08
d15-21	0.37	0.39	0.39	0.37	0.02	0.48	0.44
d22-28	0.34	0.31	0.32	0.33	0.02	0.12	0.68
d29-42	0.36	0.40	0.39	0.37	0.01	**0.01**	0.36
d43-56	0.30	0.34	0.32	0.32	0.02	**0.02**	0.97
phase 1[b]	0.41	0.40	0.41	0.39	0.01	0.39	0.12
phase 2[b]	0.33	0.34	0.34	0.33	0.01	0.26	0.79
Overall	**0.36**	**0.37**	**0.37**	**0.36**	**0.01**	**0.19**	**0.37**

a Interaction ergot by extrusion P > 0.10. , b Phase 1, day 0-21; phase 2, day 22 to 56.

Table 2. Serum prolactin (ng, ml) of growing pigs fed diets contaminated with 4 ppm ergot alkaloids, unprocessed or extruded

Parameters	Ergot		Extrusion		Pooled SEM	P-values[a]	
	0	40	No	Yes		Ergot	Extrusion
d7	1.67	0.57	1.17	1.07	0.16	**< 0.001**	0.49
d56	0.71	0.43	0.52	0.62	0.11	**0.03**	0.38

a Interaction ergot by extrusion P > 0.10.