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EFFECTS OF LOW PROTEIN DIETS ON LEAN GENOTYPE PIGS – GROWTH, FEED/ N-INTAKE AND FAT INDICATORS

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ABSTRACT

The EU nitrate directive and the increasing cost of protein sources are leading farmers to reduce the nitrogen content in livestock feed. UK pig production often employs high protein ration to ensure high growth rate and low fat deposition. The aim was to compare the performances of pigs of a lean genotype fed with a conventional (C) or 2 low protein (LP) diets, lysine supplemented (LP1) or not (LP2). 64 animals on each diet were reared from 40 to 115 kg and fed *ad-libitum*. Liveweights (LW) and feed intake (FI) were recorded and after slaughter backfat (P2 site) thickness was measured and samples of *longissimus* muscle were analysed for total fatty acids. Pen-based data were analysed examining diet and batch as main factors. There were no significant diet effects on FI. Pigs on LP2 had a lower average daily weight gain (ADG) and higher feed conversion ratio (FCR) than C or LP1 from 60 kg onwards. Between diet strategies there were no significant differences in backfat thickness, but body fat deposition was higher in the LP2 group, followed by the LP1, and C the lowest. Results confirm that the LP1 strategy allows growth performance similar to the C diet but with 11% higher intramuscular fat (IMF). Pigs on LP2 diet show an increased body fat and IMF, although subcutaneous fat thickness was little affected. LP1 results indicate that reduced nitrogen intake (C vs. LP1 by 11 to 15%) can be achieved without compromising the growth performance, however feed conversion is significantly poorer (–6%) compared to the C diet probably due to amino acids (AA) deficiencies.

Key words: pigs / animal nutrition / growth performance / low protein diet / lean genotype / N pollution

1 INTRODUCTION

Consumer demand for meat with low fat, the payment method to the farmers based on the carcass fat thickness and the widely held belief that reducing N levels in pigs diet may reduce growth rate and raise the content of fat in the carcass has led to intense selection for lean genotypes and the formulation of high protein diets for UK pigs. However, the increasingly stringent environmental legislation (EU nitrates directive, 1991) is focused on decreasing the protein content in livestock diets to reduce both cost of feed protein sources and environmental pollution. The imposed limits on both N-emissions and numbers of animals reared per hectare are forcing farmers to change feeding strategies, husbandry and genetics of the pig production units.

In pig production, protein in the feed is the most important component of daily weight gain, as it controls production efficiency and quality of muscle, which is the most desirable and economically valuable body tissue. The whole body of the pig consists of 15–18% protein with dietary proteins being the only source of essential AA, which are required for maintenance of body functions including protein turnover, urine and gut losses

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and protein retention to build body protein mostly in the form of muscle.

The formulation of commercial diets supplies excess dietary protein in order to satisfy the need for the first limiting AA (Lenis, 1989). Additionally, there is the idea that protein retention corresponds to the maximum available dietary protein and the protein retention increases linearly with increasing supply of ideal protein and energy. Unfortunately, due to the relatively low efficiency of nutrient utilisation in pigs, from 60 to 70% (depending on genotype, age and sex) of the N consumed is excreted as urine and faeces (e.g. Whittemore *et al.*, 2001; Shirali *et al.*, 2012), causing N pollution.

There is evidence (e.g. Canh *et al.*, 1998) that reducing dietary protein levels in the diet by 1 to 2% could reduce N excretion by up to 20% without compromising pig performance, whilst maintaining similar levels of essential AA (e.g. Latimier and Dourmad, 1993). However, it remains unclear if this could be done without increasing fat deposition (e.g. Teye *et al.*, 2006) and which fat deposits will be affected.

This work aimed to test the effects of low protein rations on the performance of growing pigs of a lean commercial genotype, with the main goals to clarify if the low protein rations compromise the carcass quality, growth rate or feed conversion efficiency or if they lead to increased fat deposition in undesirable fat depots.

2 MATERIALS AND METHODS

Four batches of 48 male commercial slaughter pigs from a JSR lean genotype [Synthetic Pietrain × (Large White × Landrace)], were transported from JSR Farms Ltd. (Driffield, East Yorkshire, UK) to an SAC rented farm near Edinburgh. Sixty four pigs, balanced across batches, were assigned to each feeding strategy: a control diet (C); a low protein diet supplemented with high levels of lysine (LP1); and a low protein, low lysine ration (LP2) (Table 1). All diets were formulated to the same net energy (9.7 MJ/kg). Pigs were reared in pens of 4 on straw and fed *ad libitum*.

Table 1: *Diets* [1]

Weight-range (kg)	C		LP1		LP2	
	CP[2]	L[3]	CP	L	CP	L
40–60	189	11.1	166	11.3	167	11.3
60–85	171	9.8	147	10.0	145	6.9
85–115	152	8.1	127	8.4	113	5.4

[1] All diets net energy = 9.7 MJ/kg; [2] CP: Crude protein (g/kg); [3] L: Lysine (g/kg); Values averaged over batches.

Pigs were weighed on arrival (at an average weight of ~36 kg) and allocated randomly to pens/treatments. All pigs were weighed weekly and FI was recorded on a pen basis. Samples of diet were collected at the feed mill and analysed for protein, AA composition, lipid, fibre and predicted net energy and digestible energy.

Experimental diets were introduced at an average LW of 40 kg. Approximately 3 weeks later, the pigs reached the target average weight of 60 kg and after 4 weeks the second target weight of approximately 85 kg was reached. Four to 5 weeks later the target average weight of 115 kg was reached and pigs were transported to the Tulip abattoir (Spalding, Lincolnshire, UK) and slaughtered.

P_2 fat thickness was measured on the carcasses using an intrascope at the head of the first rib, 65 mm from midline of back, left side. Joints were transported to the University of Bristol where samples of *longissimus* muscle, at the last rib position, were removed from the loin. Total fatty acids (TFA) were extracted using methanolic potassium hydroxide and the methyl esters were quantified by gas liquid chromatography.

2.1 STATISTICAL ANALYSIS

Collected data were analysed using the general linear model procedure (GLM) in SAS 9.1 (SAS Inst. Inc., Cary, NC). FI and feed conversion data were analysed taking pen as the experimental unit, but expressing the results on an individual animal basis (pen average). The traits analysed were LW at 40 kg, 60 kg, 85 kg and 115 kg; individual FI between each of these milestones; ADG and FCR (FI/ADG) between each milestone weight. ADG and FCR, as well as total FI, were also calculated for the period from the 40 kg weight until slaughter.

The statistical model applied was:

$$Y_{ijkl} = \mu + LW_i + B_j + R_k + D_l + e_{ijkl}$$

where: μ = overall mean; LW = liveweight at the start of the finishing period (co-variate); B = batch; R = row (excluded from the model for slaughter data); D = diet; e = residual error.

3 RESULTS AND DISCUSSION

Diet effects (Table 2) on FI and FCR were not significant before the period of growth between 60 and 85 kg, although the P values reached borderline values for ADG and FCR for the period between 40 to 60 kg (P = 0.061 and 0.056 respectively). Subsequently there was some

differentiation between the groups regarding ADG, FI and FCR. Contrasts between C and LP1 occasionally also reached significance, but overall these differences seem much smaller than the contrasts C vs. LP2. The results (Table 2) suggest that the animals on diet LP2 had lower growth rates (and consequently lower slaughter weights) and higher FCR (less efficient growth) than animals on the C diet, at least from 60 kg onwards, probably mainly due to the lower dietary lysine level. Pigs on the LP1 diet had similar growth rates as those on the C diet, but significantly higher FCR from 60–85 kg and over the total period from 40 kg to slaughter. This seems to be caused by the lower dietary protein level in the LP1 compared to the C group leading to AA deficiencies, especially for the branched-chain AA (valine, isoleucine and leucine) from the second period (60–85 kg) until slaughter.

LW at the start of the finishing period (LW st), batch and row exhibited a significant effect on most of the traits (Table 2), especially on LW and FI, and thus required accounting for in the model. Analysing data on LW shows that there were no significant differences between diet groups from the start until the weight at the 60 kg mark, with a clear group difference at 85 kg and at slaughter. The contrast between the C and LP1 group remained not significant, whereas LP2 reached significantly lower values than both other diets.

Nitrogen intakes (NI) were affected by the diet (Table 2) and by the batch, LW st influenced the NI significantly in the 40–60 kg period. During the first two

Table 2: *Least-squares (LS) means for different diets for liveweights (LW), feed intake (FI), average daily gain (ADG), feed conversion ratio (FCR) and nitrogen intake (NI) in the different periods, across batches 1–4 data expressed in kg*

	C LS-Mean	LP1 LS-Mean	LP2 LS-Mean	av SE	*Diet* P-value	*LW st* P-value	*Batch* P-value	*Row* P-value
LW st	36.18	36.18	36.49	0.336	0.746	*	0.0125	0.366
LW 40	40.52	40.73	40.53	0.189	0.689	< 0.0001	< 0.0001	0.2712
FI st-40	9.35	9.24	9.34	0.122	0.799	< 0.0001	< 0.0001	0.082
ADG st-40	0.7640	0.7970	0.7790	0.0293	0.720	0.982	< 0.0001	0.1403
FCR st-40	2.334	2.233	1.689	0.382	0.449	0.685	0.8164	0.4824
LW 60	59.54	59.04	58.57	0.410	0.264	< 0.0001	< 0.0001	0.3454
FI 40-60	38.28	39.16	38.16	0.454	0.247	0.0001	0.0002	0.6287
ADG 40-60	0.874	0.852	0.808	0.0192	0.061	0.72	0.0009	0.672
FCR 40-60	2.059	2.158	2.224	0.0470	0.056	0.04	0.792	0.833
NI 40-60	1.157[a]	1.040[b]	1.038[b]	0.013	< 0.0001	0.0001	0.0002	0.6653
LW 85	87.82[a]	87.35[a]	84.54[b]	0.590	0.0006	< 0.0001	< 0.0001	0.039
FI 60-85	68.44[a]	71.25[ab]	71.90[b]	1.09	0.07	0.162	0.0053	0.194
ADG 60-85	1.0020[a]	1.0000[a]	0.8950[b]	0.017	< 0.0001	0.357	0.0006	0.156
FCR 60-85	2.430[a]	2.540[b]	2.788[c]	0.0260	< 0.0001	0.439	0.0004	0.488
NI 60-85	1.872[a]	1.676[b]	1.725[b]	0.027	< 0.0001	0.1492	0.0059	0.2273
LW sl[2]	117.81[a]	116.50[a]	108.88[b]	1.065	< 0.0001	0.0289	0.0359	0.0101
FI 85-sl	83.62	87.11	84.99	1.802	0.394	0.97	< 0.0001	0.657
ADG 85-sl	0.9750[a]	0.9430[a]	0.7990[b]	0.0270	< 0.0001	0.72	< 0.0001	0.0865
FCR 85-sl	2.844[a]	3.015[a]	3.559[b]	0.0732	< 0.0001	0.959	0.0013	0.849
NI 85-sl	2.033[a]	1.770[b]	1.497[c]	0.039	< 0.0001	0.5452	< 0.0001	0.5776
FI Total 40-sl	190.34	197.52	195.05	2.53	0.138	0.277	0.0108	0.428
ADG 40-sl	0.9790[a]	0.9590[a]	0.8680[b]	0.0134	< 0.0001	0.6295	0.0173	0.0215
FCR 40-sl	2.469[a]	2.608[b]	2.857[c]	0.0290	< 0.0001	0.506	0.272	0.0943
NI 40-sl	5.063[a]	4.486[b]	4.260[c]	0.059	< 0.0001	0.239	0.071	0.347

* Not estimated for the liveweight at start (LW st), as LW st was only a co-variate for all other traits not for itself.
LS-means within row sharing a common character in their superscript are not significantly different (P > 0.05).
[1] st = start of recording period (on arrival in Edinburgh); [2] sl = slaughter (at ~115 kg on average).
Note: data are based on pen as a unit (pen averages were used) as feed intake was measured on a per pen basis (N = 48, n = 12 per feeding group).

periods (40–60 and 60–85 kg) the LP groups had different NI values in comparison with pigs on the C strategy (approximately −10%). From 85 kg until slaughter and for the overall period 40-sl all the diets differed for NI values, with C the highest and LP2 the lowest. In comparison with C the reductions were 13 and 26% for LP1 and LP2, respectively for the 85-sl period, 11 and 16% for the 40-sl period.

There were no significant group differences in P_2 backfat thickness (Table 3). However IMF in the loin muscle was found to be 11 and 46% higher in LP1 and LP2, respectively, than in C. The proportion of linoleic acid was highest in the loin muscle of pigs from treatment C, and lowest in those fed LP2, indicating that body fat deposition was in the order LP2> LP1> C. By lowering the dietary lysine level and keeping the same net energy level, an energy excess responsible for the higher fat deposition has been created in the LP2 group. For the LP1 group, the essential AA deficiencies seem to have limited the protein synthesis leading to a slight energy excess responsible for the higher IMF compared to the group C.

Table 3: *Least-squares means for different diets for post-mortem fat measurements*

	C	LP1	LP2	P value
P_2 fat thickness (mm)	13.1	13.8	13.7	0.280
Total Fatty Acids (TFA) (mg/100 g)	1055^{c}	1177 ^{b}	1543 ^{a}	< 0.001
C18:2 (n-6) (g/100 g TFA)	16.6^{a}	14.7 ^{b}	11.6 ^{c}	< 0.001

^{abc} Means in rows with a common letter in their superscript are not significantly different (P > 0.05).

4 CONCLUSIONS

Pigs on the LP2 diet reached the lowest weights and showed the least efficient growth. On the contrary, growth performance and slaughter weights were similar for pigs on the C and LP1 feeding strategies.

The LP1 diet was designed to reduce dietary protein, and thus N excretion, without affecting performance. This aim was largely achieved, as these animals maintained comparable growth rate with the C group.

There is some evidence of slightly greater fat deposition in muscle of LP1 animals compared with C, but low lysine levels in LP2 further increased muscle fat, although subcutaneous fat thickness (P_2) was not significantly affected. Assuming the reduced nitrogen intake (C vs. LP1 by 11 to 13%, Table 2) corresponds to a similar reduction in N excretion, substantially lower excretion values on the LP1 diet can be achieved without compromising the growth performance. However, the feed needed to produce 1 kg of LW gain (40 kg to slaughter) in LP1 is significantly higher (6%) compared to the C diet.

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