Impact of Dietary Energy Source on the Responses of Pigs to an Acute Level of Antigen Exposure

T. S. Stahly, professor,
D. R. Cook, assistant research scientist, and
R. C. Ewan, professor,
Department of Animal Science

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Summary and Implications

Two trials were conducted to determine the response of pigs experiencing a low or high level of acute antigen exposure to three dietary energy regimens: a low-fat basal diet, basal diet plus 6% added choice white grease (low linoleic acid), and basal diet plus 6% added corn oil (high linoleic acid).

All pigs were reared via a segregated early weaning scheme to minimize the pigs' exposure to environmental antigens and thus level of immune system activation. Three littermate pigs in each of 24 litters were allotted at 21 days to one of the three dietary energy regimens for 37 days. One-half of the pigs in each trial were administered lipopolysaccharide (LPS), an acute stimulant of the immune system, on day 21 of the trial and again eight days later. LPS administration resulted in a short-term acute depression in pig performance in both trials. The magnitude and duration of the pigs' response to LPS differed between trials and thus results are reported separately

Prior to antigen (LPS) administration, dietary fat additions resulted in faster daily gains in trial 1 and greater efficiency of dietary energy (metabolizable energy, ME) utilization in trials 1 and 2. Responses to the two fat sources were similar in both trials. During the period of acute antigen exposure (day 0 to 4 post-LPS), daily weight gain and gain:ME ratios were similar among the three dietary regimens in both AE groups. Following partial antigen clearance from the body (day 4 to 8 post-LPS), dietary additions of either fat source again resulted in faster daily gains in trial 1 and improved gain:ME ratios in trial 2 in both AE groups. Based on these data, dietary additions of fat (both low and high in linoleic acid content) result in greater growth rates and efficiency of dietary ME utilization in pigs both prior to and following a period of acute antigen exposure.

Introduction

Animal growth is the result of a myriad of biological processes that are regulated by various genetic and environmental factors. These biological processes can be grouped into maintenance functions and growth processes. Maintenance functions include repair of body tissues, fuel for voluntary activity, generation of body heat, and support of body defense (i.e., immune) system. Growth processes include the synthesis of body tissues, organs, and fluids.

Dietary sources of carbohydrates (i.e., starch, lactose, glucose) are utilized efficiently when oxidized for support of either maintenance or growth functions. In contrast, fat calories are utilized less efficiently for maintenance functions and most efficiently for fat tissue synthesis. Furthermore, animals (i.e., rats, chicks) with an activated immune system preferentially utilize glucose calories for metabolic processes as well as preferentially consume high starch versus high fat diets (Kelly et al., 1988, Kiser et al., 1973). Consequently, it is hypothesized that dietary fat calories may be utilized less efficiently in antigen challenged animals and more efficiently in animals with a low level of immune system activation and a high rate of tissue deposition.

The responses of pigs to specific fat sources also may be dependent on the animals' level of exposure to antigens and thus level of immune system activation. Feeding fats high in linoleic acid (i.e., vegetable oils - corn, soy) apparently results in greater cytokine production. Cytokines have been shown to reduce feed intake and rate and efficiency of body growth as well as carcass muscle content in pigs. Linoleic acid is a direct precursor of prostaglandins such as PGE_3 which stimulates the production of cytokines. In contrast, fat sources low in linoleic acid (i.e., animal fats - lard, tallow, some fish oil) do not serve as precursors of prostaglandins and thus minimize cytokine release. Based on these relationships, dietary addition of animal fat low in linoleic acid may result in greater improvements in growth and efficiency of dietary energy utilization than isocaloric additions of vegetable oils in pigs experiencing a low level of antigen exposure and thus level of immune system activation.

The objective of this study was to evaluate the impact of acute antigen exposure (thus immune system activation) and dietary energy source on the rate and efficiency of body growth in pigs.

Materials and Methods

Treatments

The experimental treatments consisted of two levels of acute immune system activation and three dietary energy regimens. The experimental animals were reared via an SEW scheme in order to create animals that initially possessed a low level of antigen exposure and thus immune system activation. The low and high level of acute immune system activation were created, respectively, by administering subcutaneously either 0 or 22.7 g/lb of bodyweight of lipopolysaccharide (LPS), E. coli serotype K-235 phenol extracted, dissolved in .9% NaCl. LPS is a component of the outer membrane of gram-negative bacteria. LPS administration results in a short-term release of cytokines. In turn, cytokines stimulate an acute activation of

the immune system and an acute inhibition of voluntary feed intake and proteinaceous tissue growth.

The dietary energy regimens consisted of a starchy, low-fat basal diet, supplemented with 0% fat, 6% choice white grease (CWG), or 6% corn oil (CO) (Table 1). The basal diet consisted of a wheat, soybean meal, whey, skim milk, and amino acid mixture fortified with minerals, vitamins, and an antioxidant. The three diets were analyzed to contain 1.7, 7.3, and 7.4% fat and .8, 1.4, and 4.2% linoleic acid (Table 2). Wheat was used in the basal diet because of its low fat (1.8 vs 4.0%) and low linoleic acid (.6 vs 2.2%) contents relative to corn. The diets were formulated to meet or exceed the lysine, phosphorous, and essential fatty acid (linoleic acid) needs of the pigs experiencing the low level of immune system activation. Trace minerals and vitamins were provided at concentrations equivalent to 300% of NRC (1988) estimates for 5 to 10 kg pigs.

Table 1. Composition of experimental diets.

Item	Dietary Energy Regimen		
	Basal	CWG	CO
Wheat	29.27	22.71	22.71
Choice white grease[a]	-	6.00	-
Corn oil[b]	-	-	6.00
Soybean meal, dehulled	42.69	43.20	43.20
Whey, dried	20.00	20.00	20.00
Skim milk, dried	5.00	5.00	5.00
Dicalcium phosphate	1.27	1.38	1.38
Limestone	.69	.63	.63
D,L-Methionine	.18	.18	.18
Salt	.25	.25	.25
Trace mineral-vitamin mix[c]	.55	.55	.55
Santoquin	.10	.10	.10
Total	100.00	100.00	100.00
Calculated composition			
ME, Mcal/lb	1.46	1.58	1.57
Fat, %	1.07	6.97	6.97
Protein, %	28.30	27.80	27.80
Lysine, %	1.70	1.70	1.70
Available P, %	.52	.53	.53

[a]National By-Products, Inc., Des Moines, Iowa.
[b]Archer Daniel Midlands, Decatur, Illinois.
[c]Supplied the following per lb of diet: biotin, .014 mg; choline, 218.0 mg; folacin, .08 mg; niacin, 20.5 mg; pantothenic acid, 11.6 mg; riboflavin, 4.4 mg; pyridoxine, .37 mg; thiamin, .25 mg; vitamin B_{12}, 50 mg; vitamin E, 16.9 IU; vitamin A, 2004 IU; vitamin K, .68 mg; Cu, 5.95 mg; Fe, 59.7 mg; Mn 20.5 mg; Se, .11 mg; Zn, 51.1 mg.

Table 2. Analyzed dietary fat composition.[a]

Item	Dietary Energy Regimen		
	Starch	CWG	CO
Dietary fat, %	1.68	7.33	7.38
Dietary fatty acids, %			
C10:0	.022	.027	.027
C12:0	.008	.014	.014
C14:0	.035	.111	.041
C14:1	.006	.006	.006
C16:0	.297	1.665	.885
C16:1	.006	.174	.012
C17:0	.005	.029	.006
C17:1	<.005	.018	.006
C18:0	.071	.817	.108
C18:1	.288	2.797	1.771
C18:2	.815	1.408	4.240
C18:3	.107	.124	.154
C20:0	<.005	.017	.024
C20:1	<.008	.080	.026
C20:2	<.005	.041	.011
C20:3	<.005	.006	.011
C20:4	<.005	.023	.011
C22:0	.007	.007	.013
Dietary unsaturated-saturated fatty acids			
Unsaturated (U), %	1.23	4.64	6.26
Saturated (S), %	.45	2.69	1.12
U:S	2.73	1.73	5.61

[a]Analysis performed by Hazelton Laboratories, Madison, Wisconsin.

Procedures

All pigs were obtained from a single genetic strain and source of origin. Based on previous studies, the pigs' capacity for lean tissue growth from 44 to 242 lb bodyweight was .75 to .80 lb per day. The herd of origin possessed serological titers for mycoplasma hyopneumonia (MP), actinobacillus pleuropneumoniae (APP), porcine reproductive and respiratory syndrome (PRRS), transmissible gastroenteritis (TGE) and swine influenza virus (SIV). All pigs were reared via an SEW scheme to minimize the pigs' initial exposure to antigens and thus level of immune system activation. Pigs were individually penned on slotted floors in 2 ft x 4 ft pens in an environmentally regulated building maintained at 80-85ºF. Pigs were allowed to consume feed and water ad libitum.

Two trials were conducted. In each trial, twelve sets of three littermates were used. Within each littermate set, the three pigs were randomly allotted to one of three dietary energy regimens. On day 21 of the test, six of the twelve littermate sets were administered subcutaneously 22.7 g LPS/lb of bodyweight to create an acute activation of the pigs' immune system. Each of the six littermate sets were administered a second dose of LPS eight days later. Each pig remained on its respective diet for 16 days following the initial LPS administration.

Pig weights and feed consumption were determined at 7-day intervals prior to LPS administration and at 4-day

intervals for 16 days after LPS administration. Immune status of the pigs was estimated via quantification of the acute phase protein, alpha-1 acid glycoprotein, at 0, .25, 1, and 4 days after each LPS administration in Trial 2 but not Trial 1. Serological titers for prevalent antigens in the herd of origin also were determined at the initiation and completion of Trial 2. These pigs were found to be free of antibody titers for actinobacillus pleuropneumoniae (APP), mycoplasma hyopneumonia (MP), and porcine reproductive and respiratory syndrome (PRRS) but possessed antibody titers for swine influenza virus (SIV) and transmissible gastroenteritis (TGE) at the initiation and completion of the study.

Data were analyzed by variance techniques using the General Linear Model procedure of SAS (1995). Data were analyzed as a split-plot design with antigen exposure status considered the whole plot and dietary energy regimen the subplot. The pig was considered the experimental unit. Orthogonal comparisons were made to determine the responses of pigs to low versus high dietary fat concentration (1.7 vs 7.3%) and dietary fat sources containing a low versus high linoleic acid concentrations (1.4 vs 4.2%).

Results and Discussion

Pigs were self-fed their respective experimental diets for a 21-day period prior to immune challenge with LPS. This feeding period was required to allow the dietary fatty acids to be incorporated into the pig's membrane lipids. Upon immune challenge, these membrane lipids are mobilized and potentially contribute indirectly to cytokine production. Linoleic acid (N-6) in membranes can serve as a precursor for prostaglandin production, which stimulates cytokine release. Cytokine release further enhances greater immune system activity but also inhibits voluntary feed intake and tissue growth due to the inhibitory effect of cytokines on anabolic hormones such as IGF-I and somatotropin.

Prior to LPS administration (day 0 to 21 of study, 13 to 30 pounds of bodyweight), dietary inclusion of 6% fat (either CWG or CO) resulted in faster body weight gains in Trial 1 and greater gain:ME ratios in both Trials 1 and 2 (Table 3).

LPS Effect

The response to LPS administration among the three dietary energy regimens differed between trials. Thus, the results of each trial were analyzed separately. LPS administration depressed daily gain and gain:ME ratios to a greater degree and for a longer duration in Trial 1 than that observed in Trial 2. These data would indicate that pigs in Trial 1 experienced a more acute antigen exposure and thus immune system activation than in Trial 2. The failure of LPS to depress ME intake in Trial 1 was unexpected. The authors do not have an explanation for this response.

Table 3. Pig growth and dietary energy utilization – pre-LPS administration.

	Trial No.	LPS, µg/lb	Dietary energy regimen Basal	CWG	CO
No. of pens[a]	1	0	6	6	6
		23	5	6	6
	2	0	5	6	6
		23	6	6	6
Pig weight, lb					
Initial	1	0	14.0	13.9	13.8
		23	13.8	14.0	13.6
	2	0	13.8	13.5	12.8
		23	11.8	12.4	12.6
Day 21	1	0	31.3	32.8	30.8
		23	27.9	33.3	31.5
	2	0	30.4	29.4	29.0
		23	28.3	29.5	31.4
Pre-LPS, day 0 to 21					
Daily ME, Mcal	1	0	1.95	1.93	1.88
		23	1.97	1.96	1.90
	2	0	1.60	1.45	1.46
		23	1.68	1.57	1.65
Daily gain, lb[b]	1	0	.82	.90	.81
		23	.70	.92	.86
	2	0	.80	.76	.78
		23	.79	.81	.90
Gain:ME, g/Mcal[c]	1	0	197	214	199
		23	166	216	208
	2	0	225	237	241
		23	219	240	248

[a]Pigs penned individually.
[b]Basal vs CWG, CO effect, P<.07 in Trial 1.
[c]Basal vs CWG, CO effect, P<.05 in Trials 1 and 2.

Dietary Energy Regimen Effect – Post LPS

Because LPS induces an acute, short-term (1 to 3 day) response followed by a rapid recovery, the trials were designed to analyze the response of pigs during the initial 4 day period immediately following LPS administration and the four day recovery period after LPS administration.

During the initial 4-day periods after LPS administration during which an acute immune response should occur, dietary additions of fat (either CWG or CO) did not alter daily ME intake, weight gain or gain:ME ratios in either antigen exposure group (Table 4). During the 4-day recovery period after LPS administration, during which the LPS should be largely cleared from the body and thus immune system activation minimized, dietary addition of fat (either CWG or CO) resulted in faster daily gains in Trial 1 and improved gain:ME ratios in both Trials 1 and 2.

Based on these data, dietary additions of fat (low or high linoleic acid content) to a low fat diet result in improved daily weight gain and gain:ME ratios in pigs prior to and following a period of acute antigen exposure. The growth response of pigs during a period of acute antigen exposure and thus immune system activation was not altered by dietary fat additions of either a low or high linoleic acid fat source.

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Table 4. Pig growth and dietary energy utilization – post-LPS administration.

	Trial No.	LPS, µg/lb	Dietary energy regimen		
			Basal	CWG	CO
Post-LPS, day 0 to 4 after LPS administrations					
Daily ME, Mcal[a]	1	0	3.02	3.32	3.06
		23	2.94	3.44	2.98
	2	0	3.27	3.15	3.33
		23	2.81	2.79	2.51
Daily gain, lb[b]	1	0	1.50	1.56	1.52
		23	.63	.80	.63
	2	0	1.41	1.39	1.50
		23	1.15	1.01	1.09
Gain:ME, g/Mcal[c]	1	0	228	214	228
		23	101	107	97
	2	0	197	203	206
		23	188	165	209
Post-LPS, day 4 to 8 after LPS administrations					
Daily ME, Mcal	1	0	3.87	4.07	3.71
		23	3.88	4.09	3.59
	2	0	4.28	3.92	3.96
		23	3.98	3.81	3.78
Daily gain, lb[c,d]	1	0	1.53	1.69	1.67
		23	.94	1.18	1.34
	2	0	1.68	1.72	1.64
		23	1.64	1.78	1.71
Gain:ME, g/Mcal[c,e,f]	1	0	185	188	207
		23	110	133	169
	2	0	182	201	190
		23	188	213	208

[a]LPS effect, P<.02 in Trial 2.
[b]LPS effect, P<.01 in Trials 1 and 2.
[c]LPS effect, P<.01 in Trial 1.
[d]Basal vs CWG, CO effect, P<.01 in Trial 1.
[e]Basal vs CWG, CO effect, P<.01 in Trials 1 and 2.
[f]CWG vs CO effect, P<.01 in Trial 1.