Efficacy of Pantothenic Acid as a Modifier of Body Composition in a Porcine Model of Obesity Development

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Summary and Implications

Our group's previous research has shown that pantothenic acid (PA) fed in amounts above that needed to maximize body growth effectively reduce fat tissue accretion in pigs. In the current study, the efficacy of PA to minimize fatty tissue accretion in a porcine model of obesity development was determined. Heavy weight pigs (156 kg) were allotted to one of four dietary regimens consisting of a basal diet (8 ppm PA) supplemented with 0, 80, 800, 8000 ppm added PA. The basal diet contained a dietary nutrient mix representative of the American diet (34 % of calories from fat) at daily caloric intakes equivalent to 1.8 times the animal's maintenance needs for 144 days.

A state of obesity development occurred over the duration of the study. Specifically, pigs accrued 73 kg of body weight, of which 48 % was fat tissue. Whole body fat tissue content responded quadratically to increasing PA additions. Body fat percentage was reduced by .9 percentage units by the 80 ppm added PA and increased by 1.6 and 1.1 percentage units by the 800 and 8000 ppm added PA. Based on these data, PA is not an efficient modifier of body composition in a porcine model of obesity development induced by a high fat dietary regimen.

Introduction

Obesity is reaching epidemic proportions worldwide. It occurs when the balance of energy intake and energy expenditure is disrupted over a period of time and thus leading to high adipose tissue accretion. Research is focusing on preventative and therapeutic methods to alleviate the problem.

Pantothenic acid (PA) has become of interest due to research showing its' and its derivatives lipid-mediating effects. Previous work at our research station demonstrated that PA additions in amounts greater than that needed to maximize BW gain resulted in linear reductions in subcutaneous carcass fat depth and increases in the carcass lean (fat-free) tissue content of growing pigs. Overall, the subjects in the above studies consumed low fat, high starch diets ad libitum, thus de novo lipogenesis (DNL) would be expected to play a large role in body fat accretion. In contrast, Americans typically consume a diet that is high in fat, which would limit the role of DNL relative to direct fat deposition in body fat accretion.

The objective of the current study was to determine the efficacy of dietary PA as a modifier of body composition in a porcine model of obesity development induced by a high fat dietary regimen.

Materials and Methods

Experimental treatments consisted of a basal diet supplemented with four levels of pantothenic acid (0, 80, 800, 8000 ppm) added as d-calcium pantothenate. The basal diet contained 8 ppm of PA that met or exceeded the current estimated needs of pigs and humans. The 80 ppm PA addition closely represented the amounts per unit of estimated body energy expenditure that have been shown to effectively reduce body fat accretion and increase body lean accretion in growing pigs consuming a high starch diet ad libitum. The basal diet was formulated to provide a calorie mix representative of that typically consumed by adults in the US and to provide nutrient intakes that met or exceeded the animal's needs for body maintenance and expected tissue growth. The calorie sources in the diet, expressed as % of total calories, were 33 % fat, 17 % protein and 9 % fiber.

Pigs were provided daily dietary caloric intakes equivalent to 1.8 times their estimated body maintenance (110 kcal ME/ BW kg $^{.75}$/ day) needs based on the weekly mean BW of each animal. Their daily feed allocation was divided equally into 0700 and 1600 hr feedings. Pigs were penned individually and fed their respective diets from 156 to 240 kg BW. Seventeen barrows from a high lean strain were evaluated per dietary treatment. Pigs were randomly allotted to dietary treatment by BW and date on test (block).

After 144 d, pigs were transported to the abattoir, electrically stunned and killed by exsanguination. Two tissue depots (carcass and internal organs with associated fat tissues) were isolated, weighed and subsequently scanned by dual-energy x-ray absorptiometry for determination of body tissue content. Subcutaneous fat depth at the midline of each carcass also was measured at the first rib, last rib, last lumbar vertebrae. Liver samples from representative pigs in the 0 and 8000 ppm treatments were evaluated for acetyl-CoA carboxylase, acyl-CoA oxidase, and fatty acid synthetase (all major enzymes in fat metabolism) mRNA expression by real-time PCR.

For estimation of the pig's initial body composition, thirteen pigs representative of the starting weights of the pigs on test were sacrificed and the composition of the body depots were determined using the same procedures as above.

Results and Discussion

A state of obesity development occurred over the duration of the study. Animals accrued 72.85 kg BW consisting of 67.49 kg of carcass and 5.36 kg of organ tissues. Total body fat accretion was 34.88 kg and represented 48 % of the accrued body weight. Of the accrued body fat, 32.3 kg or 93 % was deposited in the whole carcass depot and 2.58 kg or 7 % were accrued in the organ depot. Although the proportion of BW gain accrued as fat was 48 % compared with 27 % fat tissue present prior to the initiation of the study, the proportional distribution of body fat among the carcass and organ depots remained the same.

As expected, PA additions did not alter daily body weight gain, feed intake, or the efficiency of feed utilization in pigs growing from 156 to 240 kg BW. Cold carcass weights and subcutaneous backfat depths did not differ among PA treatment groups. Gastrointestinal tract and left kidney weights increased linearly as dietary PA additions increased ($P < .13$). Longissimus muscle area at the tenth rib decreased quadratically as PA additions increased ($P = .01$).

Carcass fat tissue content, expressed as kg of tissue ($P = .10$) or % of carcass weight ($P = .14$), responded quadratically as dietary PA additions increased. Carcass fat was lowered by .9 percentage units with the incremental addition of 80 ppm PA and increased by 1.8 and 1.0 percentage units by the additions of 800 and 8000 ppm PA. Organ fat tissue as well as total organ weights increased quadratically ($P < .03$) as PA additions increased. Organ fat content, expressed as a % of organ weight, was numerically lowered by .6 percentage units with the 80 ppm PA addition and increased by .7 and 2.7 percentage units by the 800 and 8000 ppm PA additions. These shifts in organ fat content were largely due to changes in the fat content of the gastrointestinal tract depot. Whole body fat tissue increased quadratically ($P < .09$) as dietary PA additions increased. Whole body fat percentages were shifted by -.8, +1.6 and +1.1 percentage units with the addition of 80, 800 and 8000 ppm PA, respectively.

In the current obesity development study, body fat tissue contents were reduced by a smaller magnitude and less consistently with a ten fold increase in PA than that observed in pigs with a high capability for de novo fat synthesis induced by high carbohydrate feeding. The lower response in the current study is hypothesized to be due in part to a low rate of de novo fat synthesis (DNL) occurring relative to direct deposition of ingested fat. The high fat diet also probably limited the ability to see a difference in mRNA expression of the fat metabolism enzymes between the 0 and 8000 ppm PA treatments due to the enzyme's assistance in DNL. The liver in pigs also may not contribute much to fat accretion through DNL, which occurs mainly in the adipose tissue.

In conclusion, a porcine model of obesity development was effectively created by feeding a high dietary fat regimen at 1.8 times body maintenance needs. Dietary PA additions in this model resulted in quadratic responses in fat tissue content in carcass and organ depots but did not affect hepatic fat metabolism enzyme gene expression. A ten fold increase in PA intake above that needed to maximize BW gain resulted in small reductions in body fat content; whereas, 100 and 1000 fold increases in PA intake resulted in increases in body (carcass and organ depots) fat content.

Table 1. Tissue accretion by body depot in porcine model of obesity development. [a]

Body Depot	Tissue Component	Tissue Component Weight, kg Initial[b]	Tissue Component Weight, kg Final	Accrued Tissue, kg Mean	Accrued Tissue, kg SE	Accrued Tissue % of Depot Accrued Weight
Carcass[c]						
	Fat	15.63	31.89	16.26	.65	47
	Lean	39.56	57.26	17.70	.64	51
	BMC	0.99	1.55	0.56	.02	2
	Total	56.18	90.64	34.46	.71	
Organ[d]						
	Fat	2.83	5.41	2.58	.11	48
	Lean	11.80	14.57	2.77	.28	52
	BMC	0	0	0	0	0
	Total	14.62	19.98	5.36	.30	
Whole Body[e]						
	Fat	34.86	69.74	34.88	1.34	48
	Lean	93.36	130.12	36.76	1.39	50
	BMC	1.92	3.13	1.21	.05	2
	Total	130.14	202.99	72.85	1.54	

[a]LSMeans values reported for all 54 pigs used in the study regardless of dietary treatment.
[b]Predicted from relationship of DEXA estimated tissue weight and body weight of pigs at the initiation of the study.
[c]Right carcass half
[d]Lungs-heart and liver and associated thoracic fat, and gastrointestinal tract with spleen and associated mesenteric and omental fat
[e]Whole body= right carcass half, kg + (percentage of component in right carcass half* left carcass half weight, kg) + organs, kg

Table 2. Effect of dietary pantothenic acid additions on DEXA estimated carcass tissue content. [a]

Criteria	Added pantothenic acid, ppm 0	80	800	800	SEM	P= DL [b]	DQ [b]
Carcass Tissue, kg							
Fat	30.86	30.87	33.48	32.68	.61	.42	.10
Lean	56.09	57.71	56.11	56.48	.61	.88	.66
BMC [c]	1.54	1.56	1.57	1.53	.02	.65	.71
Total	88.27	90.15	91.16	90.67	.73	.57	.26
Carcass Tissue, % Total Carcass Tissue Weight							
Fat	35.02	34.13	36.77	35.98	.56	.52	.14
Lean	63.47	64.13	61.50	62.35	.55	.50	.11
BMC [c]	1.75	1.73	1.72	1.69	.03	.46	.84

[a]LSMeans reported. Values of 14, 13, 14, 13 pigs for the four treatment groups, respectively.
[b]Linear (L) and Quadratic (Q) response to dietary (D) pantothenic acid additions.
[c]Bone mineral content.

Table 3. Effect of dietary PA additions on DEXA estimated organ tissue content. [a]

	Added pantothenic acid, ppm					P=	
Criteria	0	80	800	8000	SEM	DL [b]	DQ [b]
Total Organ Tissue, kg [c]							
Fat	4.80	5.17	5.56	6.08	.08	<.01	.01
Lean	13.41	14.74	14.87	14.74	.24	.41	.15
Total	18.21	19.90	20.44	20.82	.25	.02	.03
Total Organ Tissue, % Total Organ Weight [c]							
Fat	26.76	26.15	27.37	29.47	.43	.01	.58
Lean	73.23	73.84	72.62	70.52	.43	.01	.57

[a]LSMeans reported. Values of 14, 13, 14, 13 pigs for the four treatment groups, respectively.
[b]Linear (L) and Quadratic (Q) response to dietary (D) pantothenic acid additions.
[c]Total organ tissue= GI Tract(gastrointestinal tract with contents and associated mesentery and omental fat and spleen) + LHL (lungs-heart and associated thoracic fat and liver).

Table 4. Effect of dietary pantothenic acid additions on DEXA estimated whole body tissue content. [a,d]

	Added pantothenic acid, ppm					P=	
Criteria	0	80	800	8000	SEM	DL [b]	DQ [b]
Whole Body Tissue, kg[c]							
Fat	67.17	67.56	72.87	71.82	1.26	.30	.09
Lean	126.87	131.39	127.64	128.31	1.29	.89	.73
BMC	3.11	3.15	3.15	3.07	.05	.56	.80
Total	197.15	202.09	203.66	203.19	1.55	.47	.22
Whole Body Tissue, % of Total Body Tissue[c]							
Fat	34.2	33.4	35.8	35.3	.005	.41	.14
Lean	64.2	65.1	62.6	63.2	.005	.43	.13
BMC	1.58	1.56	1.55	1.51	<.001	.36	.76

[a]LSMeans reported. Values of 14, 13, 14, 13 pigs for the four treatment groups, respectively.
[b]Linear (L) and Quadratic (Q) response to dietary (D) pantothenic acid additions.
[c]Whole body tissue=right carcass half, kg + (percentage of component in right carcass half* left carcass half weight, kg) + organs, kg
[d]Equivalent mean whole body weights determined gravimetrically for the four treatment groups were 197.46, 202.98, 204.66 and 203.93 kg with a SEM of 1.55 (DL= .47 and DQ= .22)

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