

LIPOID COMPOSITION OF CARCASS TISSUE FROM TRANSGENIC PIGS

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SUMMARY

Total lipid, fatty acid profiles and cholesterol content of whole carcass ground tissue were compared for transgenic (T) pigs expressing a bovine growth hormone gene and control (C) pigs. Pigs were slaughtered at five different weights: 14, 28, 48, 68 and 92 kg. Total carcass fat followed a different pattern of deposition in T-pigs than in C-pigs. Carcass fat increased with increasing live weight in T-pigs compared to a continuous increase (carcass fat) in C-pigs. A dilution effect on fatty acids was observed in T-pigs with increasing live weight, whereas, fatty acid concentrations increased in C-pigs. Cholesterol content was not different between T- and C-pigs at any of the slaughter weights.

INTRODUCTION

The technology for introducing recombinant genes into laboratory animals has been available only since 1980. Gene transfer techniques have evolved from the first successful gene transfer experiments carried out in mice in 1980 (GORDON et al, 1980). Success in mice prompted similar experiments in farm animals. HAMMER et al (1985) described the first production of transgenic livestock by pronuclear injection. Most of the gene transfer experiments carried out with farm animals during the late 1980s attempted to manipulate growth and related production characteristics such as feed efficiency. However, little is known regarding body/carcass composition and lipid composition of transgenic livestock. Therefore, the purpose of this study was to determine what effect the introduction of recombinant bovine growth hormone (rbGH) gene into pigs has on lipid and carcass composition.

MATERIALS AND METHODS

Animals and Feed. Fifty-two transgenic and control pigs were G2 generation descendants of transgenic founder 31-04 and G3 and G4 generation descendants of transgenic founder 37-06. The founders of these two transgenic lines were produced by microinjection of fertilized ova with a MT-bGH fusion gene (PURSEL et al, 1989). Subsequent generations were produced by artificial insemination of non-transgenic crossbred females with fresh or frozen epididymal spermatozoa recovered from transgenic boars after euthanasia. Presence of the transgene was established by hybridization of dot blots of DNA from tail biopsies (HAMMER et al, 1985). Expression of the transgene was established by radioimmunoassay of plasma collected at 1 week of age or older using a bGH radioimmunoassay that did not measure pGH (MILLER et al, 1989). Plasma bGH concentrations were >1000 ng/ml in transgenic pigs of the 31-04 line and 25 to 200 ng/ml in transgenic pigs of the 37-06 line. Pigs were weaned at 4 to 8 weeks of age and fed *ad libitum* a corn-soybean meal diet containing 18% crude protein, supplemented with .25% vitamin E. Pigs were slaughtered at five different live weights: 14, 28, 48, 68, and 92 kg.

Experimental. The left side of each intact carcass (skin and hair included) was ground in a whole-carcass grinder and random samples of ground tissue were collected. Tissue samples

were analyzed for proximate-chemical composition, fatty acid composition, and cholesterol content.

Isolation of Lipids. The FOLCH et al (1957) procedure was employed to obtain lipid extracts of each sample. Preparation and isolation procedures were previously described by SOLOMON et al (1990). Fatty acids were converted into methyl ester derivatives prior to analysis. Methylation and preparation of samples for GLC analysis were previously described (SOLOMON et al, 1990). Cholesterol was converted to a trimethylsilyl ether derivative. Derivatizing procedure and sample preparation were previously described (SOLOMON et al, 1990).

Data Analysis. Data were analyzed by the analysis of variance technique (SAS, 1985) to determine the significance of variation between transgenic and control pigs at the five different weight groups for a 2 x 5 factorial arrangement which included treatment x weight group interactions. When significant ($P < .05$) main effects for weight groups were detected, Duncan's Multiple Range Test was used to separate means.

RESULTS AND DISCUSSION

Carcass fat deposition followed a different pattern for T-pigs compared to C-pigs (Table 1). Total carcass lipid increased (190%) in the C-pigs from 14 to 92 kg live wt, whereas in the T-pigs, total carcass lipid increased (32%) only from 14 to 48 kg live weight and then decreased (45%) from 48 to 92 kg (overall 27% decrease from 14 to 92 kg). At 14 kg slaughter weight, carcasses from T-pigs contained 38% less fat than C-pigs. At 28 kg, T-pigs had 38% less total carcass fat; at 48 kg, 48% less total carcass fat; at 68 kg, 78% less total carcass fat, and at 92 kg, T-pigs contained 85% less carcass fat than C-pigs. Total cholesterol content of the ground carcass tissue was not different between T- and C-pigs at any of the designated weights; however, with increasing live weight (14 to 92 kg), cholesterol content decreased for both C- and T-pigs (23% C-pigs; 28% T-pigs).

Carcass from T-pigs consistently contained less total saturated fatty acids (SFA) than C-pigs at each slaughter weight. At 14 kg slaughter weight, carcasses from T-pigs contained 44% less SFA compared to C-pigs. At 28 kg, T-pigs had 42% less SFA; at 48 kg, T-pigs had 42% less SFA; at 68 kg, T-pigs had 78% less SFA, and at 92 kg, T-pigs contained 85% less SFA than C-pigs. Carcass tissue from T-pigs reflects a much more favorable (lesser quantity) of SFA. A 137% increase in total SFA was observed in C-pigs with increasing live wt (14 to 92 kg) compared to a 40% decrease in total SFA for T-pigs. These differences in SFA were mostly a result of changes in palmitic acid, stearic acid, and myristic acid (not in tabular form). Margaric and arachidic acids were also present but in very small quantities; however, T-pigs did not contain any arachidic (not in tabular form). Palmitic acid accounted for 62% of the total detectable SFA in the C-pigs (92 kg weight group) compared to 66% in T-pigs. Stearic acid accounted for 34% of the total SFA in the C-pigs (92 kg weight group) compared to 30% in T-pigs. Both myristic acid and palmitic acid have been reported to be hyperlipidemic and hypercholesterolemic (KEYS et al, 1965). Human consumption of hypercholesterolemic fatty acids has come under attack by health professionals (NRC, 1988). BONAMONE and GRUNDY (1985)

onstrated that a diet high in stearic acid did not elevate plasma levels of low density lipoprotein (LDL) cholesterol, perhaps because it is poorly digested and can be easily saturated to oleic acid (KEYS et al, 1965).

Carcasses from T-pigs contained less total monounsaturated fatty acids (MUFA) than C-pigs at each slaughter weight (Table 1). At 14 kg slaughter weight, carcasses from T-pigs contained 48% less MUFA; at 28 kg, 46% less MUFA; at 48 kg, 59% less MUFA; at 68 kg, 79% less MUFA, and at 92 kg, 91% less MUFA than C-pigs. The fatty acids comprising the MUFA were palmitoleic, palmitelaidic, oleic, and cis-11-eicosenoic acids. Oleic acid was the major contributor of MUFA in the total lipid fraction (not in tabular form). The trend was for the quantity of MUFA to increase with increasing slaughter weight in C-pigs, while a dilution effect was observed for MUFA in T-pigs after 48 kg live weight. Oleic acid has been reported to be hypolipidemic, reducing cholesterol (GRUNDY, 1986), and it is therefore not considered to be an undesirable dietary fatty acid. MATTSON and GRUNDY (1985) demonstrated that oleic acid had the ability to reduce LDL cholesterol.

Significant differences in the percentage of polyunsaturated fatty acids (PUFA) were observed in the carcass tissue (Table 1) from T- and C- pigs. At 14 kg slaughter weight, carcasses from T-pigs contained 38% less PUFA; at 28 kg, 24% less PUFA; at 48 kg, 22% less PUFA; at 68 kg, 53% less PUFA, and at 92 kg, 66% less PUFA than C-pigs. The trend was for PUFA to increase with increasing live weight in C-pigs and to decrease with increasing live weight in T-pigs. The fatty acids comprising the PUFA were linoleic, linolenic and arachidonic acids.

CONCLUSIONS

In general, carcass tissue from T-pigs contained significantly less long chain fatty acids than C-pigs at each of the designated slaughtered weights. In fact, a dilution effect was observed for fatty acid deposition in T-pigs with increasing slaughter weight. On the other hand, fatty acid deposition either increased or remained the same with increasing slaughter weight in C-pigs. Carcass tissue from T-pigs reflects much more favorable levels of fatty acids compared to C-pigs when considering dietary health recommendations and concerns.

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TABLE 1. COMPARISON OF TOTAL CARCASS LIPID, CHOLESTEROL AND FATTY ACID^a CONTENT FOR TRANSGENIC^b AND CONTROL PIGS

Treatment	Weight Group, kg	Total Lipid g/100g	Cholesterol mg/100g	Total SFA	Total MUFA	Total PUFA
Controls	14	10.04	100.88	3.24	3.74	1.51
	28	12.32	94.97	3.69	4.69	2.38
	48	16.58	85.92	5.37	6.73	2.87
	68	26.78	74.15	8.22	8.74	3.95
	92	29.07	77.81	7.67	8.73	3.91
Transgenics	14	6.19	106.45	1.92	1.98	1.08
	28	7.62	99.97	2.20	2.47	1.82
	48	8.16	86.28	2.39	2.50	2.02
	68	5.97	74.55	1.60	1.57	1.63
	92	4.49	77.08	1.15	1.13	1.29
Significance of treatments, P<:						
Treatment (T)		.01	NS	.01	.01	.01
Weight Group (W)		.01	.01	.01	.01	.01
T X W		.01	NS	.01	.01	.01

^aGrams in 100g sample. SFA = saturated fatty acids; MUFA = monounsaturated fatty acids; PUFA = polyunsaturated fatty acids.

^bTransgenic pigs = pigs expressing bovine growth hormone gene.