Differences in Beef Strip Loin Steaks of Steers Due to the Inactive Myostatin Mutation

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Summary

Strip loins from steers with genotypes containing zero, one, or two copies of the inactive myostatin mutation (IM) (n = 20, 22, and 16, respectively) were obtained. Loins were cut into 1-inch steaks where total number of steaks and total numbers of steaks with gluteus medius (often called vein steaks) were noted. Loins from zero copy cattle had a higher total number of steaks, but no difference in proportion of steaks, both with and without the gluteus medius compared to one and two copy cattle. This study indicates that increasing copies of the IM mutation has no impact on the proportion of steaks containing the gluteus medius muscle within the strip loin.

Introduction

The inactive myostatin allele is a negative regulator of myogenesis and causes an increase in muscle fiber number (hyperplasia) caused by base pair deletions, which is the primary reason for approximately 20% increase in skeletal muscle mass in Piedmontese cattle (Kambadur et al *Genome Research*, 1997). Cattle with two copies of the inactive myostatin allele possess nearly twice the number of muscle fibers than other cattle and yield extremely lean and heavily muscled carcasses.

Toward the posterior end of the strip loin the GM increases in size while the *Longissimus lumborum* decreases in size and narrows. Steaks containing the GM also include a

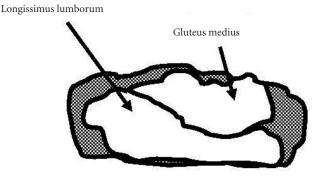


Figure 1. Illustration showing vein steak from posterior end of strip loin.

piece of connective tissue separating the GM and LD that will appear on both sides of the steak. Such steaks, called vein steaks, are lower in value than strip steaks without the GM. (Figure 1) This research was conducted to determine if cattle with two copies of the inactive myostatin gene would produce a greater number and greater percentage of strip loins containing the *Gluteus medius* (GM) when compared to cattle with zero and one copy of the inactive myostatin allele.

Procedure

All steers (n = 20, 22, and 16 carrying 0, 1, and 2-copies of the inactive myostatin mutation, respectively) were individually fed the same diet for 182 days, and then finished for 50 days, for a total of 232 days on feed. At three days postmortem strip loins were collected from the carcasses (n = 58). Each strip loin was then measured for loin weight, loin length, sirloin face width, rib face width, sirloin tail length, rib tail length, and fat thickness at the rib face. The loins were then cut into 1-inch thick steaks and the following information was gathered for each: total number of

steaks, total number of vein steaks, total number of non-vein steaks, and weight of each individual steak.

Data was analyzed using a completely randomized design in SAS (Version 9.1) with the fixed effects being the different inactive myostatin mutations and random effect of the animal was used. Analysis of Variance (ANOVA) was performed using the Proc Mixed procedure with mean separation determined using LS MEANS and DIFF LINES options of SAS, with significance determined at $P \le 0.05$.

Results

Despite having an increase in strip loin weight (Table 1), strip loins from cattle containing one or two copies of the inactive myostatin allele were shorter and wider, yielding fewer total steaks and fewer vein steaks (Table 2) than loins from cattle containing zero copies of the myostatin allele. This was reflected by greater mean steak weight (Table 2) for one and two copy samples. The two copy samples had a lower number of total steaks (P < 0.001) and number of veins steaks (P < 0.001) than zero and one copy samples. When comparing two copy to zero and one copy the number

Table 1. Dimensional measurements of strip loin from cattle with 0, 1, or 2 copies of the inactive myostatin allele.

	Number of Inactive Myostatin Alleles				
Measurements	0	1	2	SEM	P-Value
Fat Thickness (in)	0.56 ^a	0.27 ^b	0.13 ^c	0.031	<.01
Loin Weight (kg)	4.99 ^b	5.48 ^a	5.10 ^a	0.149	0.03
Loin Length (in)	15.31 ^a	14.80 ^a	13.61 ^b	0.231	<.01
Sirloin Face Width (in)	8.90 ^b	9.62 ^a	9.53 ^a	0.140	0.02
Rib Face Width (in)	7.47 ^b	8.39 ^a	8.66 ^a	0.135	<.0001
Sirloin Tail Length (in)	2.57 ^b	3.00 ^a	2.43 ^b	0.137	0.01
Rib Tail Length (in)	1.17 ^{ab}	1.25 ^a	1.03 ^b	0.059	0.02
Fat Thickness over Loin (in)	0.62 ^a	0.27 ^b	0.15 ^c	0.038	<.01

^{a,b,c}Means with different superscripts within the same row differ ($P \le 0.05$).

 Table 2.
 Number, weight, and proportion of vein steaks from strip loin from cattle with 0, 1, or 2 copies of the inactive myostatin allele.

Steak Trait	Number of Inactive Myostatin Alleles				
	0	1	2	SEM	P-value
Number of Loins Analyzed	20	22	16		
Total Steaks	14.7 ^a	13.77 ^b	12.38 ^c	0.233	<.01
Number Vein Steaks	4.6 ^a	4.18 ^b	3.56 ^c	0.151	<.01
Non-Vein Steaks	10.15 ^{ab}	9.5 ^a	8.87 ^b	0.238	0.09
% of Vein Steaks in Loin	31.37	30.39	28.82	1.068	0.21
Combined Weight of Steaks (g)	4993.37 ^b	5446.37 ^a	5093.29 ^{ab}	137.653	0.05
Total Weight of Vein Steaks (g)	1569.101	1704.30	1505.81	68.119	0.12
% Weight of Vein Steaks	31.42	31.35	29.35	1.109	0.30
Mean Steak Weight (g)	339.5 ^b	396.6 ^a	410.1 ^a	0.052	<.01

^{a,b,c}Means with different superscripts within the same row differ (P \leq 0.05).

of non-veins steaks (P = 0.0009) was greater. The numeric and weight percentages of vein steaks did not differ among genotypes (Table 2). Thus, increasing copies of the myostatin allele was not detrimental to the percentage of vein steaks derived from the strip loin.

These data indicate that increasing copies of the myostatin allele have no impact on the proportion of vein steaks in the strip loin. Strip loins from cattle with one copy and two copies yielded fewer total one inch steaks that were wider and heavier. This could impact the attractiveness of steaks to consumers because they would represent a larger portion size at equal thickness or a thinner steak if cut to constant weight.

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