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Summary

A study was conducted to determine the effects of implants and transportation on the metabolic status of feedlot steers. Steers (n = 28) were sorted by body weight, allocated into light or heavy blocks, and randomly assigned to one of two treatments. Treatments included non-implanted controls (CON) and steers implanted with Synovex Plus 70 d prior to harvest (IMP). Jugular blood and muscle biopsy samples (longissimus dorsi (LD) and semimembranosis (SM)) were collected 70 d post-implant, prior to transit. Steers were transported to Schuyler, NE, where blood and biopsy sampling was repeated. After harvest, carcass data were collected and muscle samples were taken from the LD, SM, Psoas Major (PM), and Illiacus (IL) muscles. Implanting increased ($P < 0.05$) estradiol levels and improved live animal performance. Carcass weight and rib eye area were increased ($P < 0.05$) in implanted steers. No dark cutters were found in either treatment. Pre-transit insulin/glucagon ratio and muscle glycogen levels did not differ ($P > 0.10$) between treatments. Non-esterified fatty acid (NEFA) levels were reduced ($P < 0.05$) in implanted steers pre-transit. Transit increased ($P < 0.05$) NEFA levels, but had no effect ($P > 0.10$) on insulin/glucagon ratio or muscle glycogen levels. Implanting did not affect ($P > 0.10$) insulin/glucagon ratio, NEFA, or LD glycogen levels post-transit. Implanted steers had lower ($P < 0.05$) glycogen levels in the SM than did non-implanted steers post-transit. Weight block affected ($P < 0.05$) insulin and insulin/glucagon ratio levels, with steers in the light block having greater levels of each. Muscle pH and objective color (L*, a*, b*) of the LD were not biologically different between treatments. Implanted steers had greater ($P < 0.05$) glycolytic potential values in the LD, and tended ($P < 0.10$) to have higher L* values in the PM. Implanting increased ($P < 0.05$) shear force of the LD. These data indicate that although implants affect bovine metabolism, other factors are necessary to cause a sufficient reduction in muscle glycogen and to produce a dark cutting carcass.

Introduction

Dry, firm, and dark beef (dark cutters) is thought to be caused by a lack of muscle glycogen prior to harvest. Glycogen is the storage form of glucose in hepatic and muscular tissues, and is used by an animal to generate energy. When an animal is harvested, glycogen is utilized anaerobically in muscular tissue. Muscle glycogen consumption postmortem generates lactic acid, which lowers muscle pH from approximately 7.2 pre-harvest to 5.4 at 24 h post-harvest. Low levels of glycogen in muscular tissue postmortem can lead to the dark cutting condition if pH reduction is severely limited. If muscle pH is unusually high, i.e. above 5.8, the cut surface is at risk to become dryer and darker in color.

Pre-harvest stressors are thought to reduce muscle glycogen levels and cause the dark cutting condition. These include psychological, physiological, and genetic factors. The use of implants as a physiological factor may play a role in the dark cutting condition, but definitive cause and effect relationships have not been established.

Metabolic factors that play a role in glycogen metabolism include blood levels of insulin and glucagon in the animal. Insulin is an inducer of enzymes that promote glucose uptake into muscle tissue and is released when blood glucose levels are elevated. Glucagon promotes the release of glucose from hepatic tissue into systemic blood circulation, and is

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usually released when circulating glucose levels are low. It is believed the insulin/glucagon ratio determines the metabolic state of an animal rather than the absolute levels of either hormone. Besides glucose, other compounds, namely non-esterified fatty acids (NEFA) and amino acids, can be used by the animal for energy. Increased circulating NEFA levels indicate the animal is mobilizing triglyceride stores and may be using fatty acids for energy substrate.

The objective of this study was to determine the effect of implants on muscle glycogen status and consequently dark cutting in finishing steers. Additionally, this study intended to examine the effect of transportation on muscle glycogen and blood parameters in finishing steers.

Materials and Methods

Steers (n = 28) of predominantly Angus background were sorted by body weight and blocked into light and heavy weight groups. They were then randomly assigned to one of two treatments, and one of two pens within each treatment. Steers were fed a typical finishing diet (Table 1) twice daily for the duration of the study. Bunk scores and pen intake were recorded daily prior to morning feeding.

Steers that did not receive an implant (CON) were designated as control animals. Implanted steers (IMP) were given Synovex Plus 70 d prior to harvest. Implants were checked at 21 d, with none found to be defective or missing. Live weights were taken prior to morning feeding at 1, 21, 42, and 70 d.

At 70 d, blood and muscle biopsy samples were taken on the left side of each animal prior to morning feeding. Blood was collected via jugular venipuncture into vacuum-sealed tubes to yield plasma and serum samples. Plasma was obtained after blood collection by centrifugation at 3000 x g for 30 min and stored at -20^{o}C until further analysis. Serum was obtained over 24 h after initial blood collection by centrifugation at 3000 x g for 30 min and was also stored at -20^{o}C until further analysis. A single biopsy sample of approximately 50 mg was taken from the Longissimus dorsi (LD) and Semimembranosis (SM) of each steer using the Bergstrom needle technique. Samples were collected from the LD approximately 12 in dorsal to the hook bone. Samples collected from the SM were taken from the left side of the animal at approximately the midpoint of the muscle.

On the afternoon of sampling, steers were transported 261 mi to Schuyler, NE, for harvest. Prior to harvest, steers were unloaded near (< 4 mi) the packing plant where jugular blood and muscle biopsy collections were repeated. Steers were then transported to the packing plant and harvested the following morning.

Carcasses were chilled for 120 h prior to grading. Three carcasses were lost in the packing plant prior to data collection. All variables needed to determine carcass USDA Quality and Yield Grades were recorded by trained SDSU personnel. The presence of the dark cutting condition was determined by USDA grading personnel. Postmortem carcass pH and objective color (L*, a*, b*) measurements were collected at 5 d postmortem by plant personnel. Carcass pH was measured using a pH-Star probe (SFK Tech., Herelev, Denmark) that was inserted into the LD of each carcass. Objective color was taken on the cut surface of the LD using a Minolta CR-300 colorimeter (Minolta Corp., Ramsey, NJ). Samples of four muscles, the LD, SM, Psoas Major (PM), and Illiacus (IL) were obtained and returned to the university abattoir for determination of individual muscle pH, objective color, shear force, and glycolytic potential at 7 d postmortem. Muscle pH and glycolytic potential were determined on all four muscles. Objective color was determined on the LD, PM, and IL. Shear force was determined on the LD.

Muscle tissue (10 g; 7 d postmortem) was homogenized in 90 mL of deionized water using a Ultra-Turrax T25 homogenizer and S25N probe (Divitech Equipment Co., Cincinnati, OH). Muscle pH was then determined on the homogenate using an Orion 420A pH meter (Boston, MA). To eliminate the possibility of bacterial contamination, all muscle pH samples were analyzed within 48 h of collection from the packing plant. Objective color was determined on individual muscle samples using a Minolta CR-310 colorimeter (Minolta Corp., Ramsey, NJ). Samples were allowed to bloom a minimum of 40 min prior to objective color determination. Shear force was determined through the Warner-Bratzler method.

Glycolytic potential was determined through two separate assays. Residual glycogen in muscle tissue post-harvest and muscle lactate (salt form

of lactic acid) were determined through enzymatic analysis. Based on the values of these assays, the equation [(2 x glycogen) + lactate] was then used to determine the glycolytic potential of each muscle examined.

Plasma samples were used to determine circulating estradiol and glucagon concentrations in steers. Serum samples were used to determine insulin and NEFA concentrations. Estradiol, insulin, and glucagon were determined through radioimmunoassay analysis. Non-esterified fatty acid levels were determined through colorimetric analysis. Glycogen concentration was determined in biopsy samples by enzymatic analysis.

Production data were determined based on a 4% shrink applied to body weight values with pen being considered the experimental unit. All other data were analyzed with steer as the experimental unit. With the exception of performance data, all models used to analyze data contained weight block as a factor. Transportation was included in the model as a main effect in samples collected both pre- and post-transport. All data were analyzed using the GLM procedure of SAS. Chi-square analysis was conducted on shear force data.

Results and Discussion

Implants increased production rates and serum estradiol of steers (Table 2) confirming their biological activity. Hot carcass weight (HCW) and rib eye area were increased ($P < 0.05$) due to implants (Table 3). No carcasses in either treatment were classified as dark cutting.

There was a tendency ($P = 0.087$) for a treatment x group x transit interaction with insulin levels (Table 4). Transit tended to decrease ($P = 0.069$) insulin levels of implanted, light steers. No differences ($P > 0.10$), with respect to implant treatment and transit, were found on insulin levels in non-implanted steers and heavy implanted steers. Insulin levels varied greatly ($P < 0.01$) between weight block, with lighter steers having an insulin level of 555 pg/ml versus heavy steers having an insulin level of 332 pg/ml. There was also a tendency ($P = 0.057$) for glucagon levels to be lower in heavier cattle (109 pg/ml light group versus 91 pg/ml heavy group). Lighter steers had higher ($P < 0.05$) insulin/glucagon ratio values than heavier steers (5.48 light group versus 3.59 heavy group). It appeared weight block played a larger role in influencing the insulin/glucagon ratio in this experiment than did either implant treatment or transportation.

The NEFA levels were lower ($P < 0.05$) in implanted steers prior to transit (Table 5). Transit increased ($P < 0.05$) NEFA levels in each implant treatment. There were no post-transit differences ($P > 0.10$) in NEFA due to implants. Implants had no effect ($P > 0.10$) on glycogen levels in the LD and SM pre-transit. Transportation had no effect ($P > 0.10$) on glycogen levels in the LD and SM. Post-transit, implanted steers had lower ($P < 0.05$) glycogen values in the SM than did non-implanted steers. The LD had higher ($P < 0.01$) glycogen levels than the SM (104 µmol/g LD versus 83 µmol/g SM) in both implant treatments.

Longissimus dorsi muscle pH, determined 5 d postmortem, was lower ($P < 0.05$) in implanted steers (Table 6). It is of interest to note that the statistical difference in pH demonstrated here would indicate implanted steers are less likely to become dark cutters versus non-implanted steers. However, this difference is of little biological relevance as both treatments had carcass pH values well below 5.8. Muscle pH, determined 7 d postmortem, did not differ ($P > 0.10$) between treatments for the SM, PM, or IL muscles. There was a tendency ($P = 0.098$) for implanted steers to have higher postmortem L* values in the PM (43.34 CO versus 44.24 IMP) 7 d postmortem. However, a* and b* values were not different ($P > 0.10$) between treatments for the PM. Objective color values did not differ ($P > 0.10$) between treatments for the LD or IL muscles.

Implants increased ($P < 0.05$) shear force values of the LD (Table 6). Implanted steers had a tendency ($P = 0.087$) to produce lower percentage tender (shear force < 3.5 kg) and higher percentage tough (shear force > 5.0 kg) steaks versus non-implanted steers.

The LD from implanted steers had greater ($P < 0.05$) glycolytic potential values than non-implanted steers (Table 7). Their was a tendency ($P = 0.074$) for implanted steers to have lower glycolytic potential levels than non-implanted steers in the PM. The decrease in glycolytic potential is mainly due to a tendency for decreased ($P = 0.097$) glycogen levels in the

PM of implanted steers after carcass fabrication. Implants had no effect ($P > 0.1$) on the proportion of lactate produced as a percentage of the total glycolytic potential. The IL and PM were similar ($P > 0.10$) in lactate produced, and produced more ($P < 0.01$) lactate as a ratio of the total glycolytic potential than the LD and SM muscles (81.3 % IL, 78.4 % PM, 66.7 % LD, and 52.7 % SM, respectively). In addition the LD had a greater ($P < 0.01$) percentage of lactate produced than the SM.

Conclusions

Implants had a desirable effect on steer performance. In this study, implanted steers produced no dark cutting carcasses, and muscle glycogen levels in the Longissimus dorsi were unaffected by implants. Glycolytic potential values were increased in implanted steers. Muscle pH and objective color values did not exhibit biologically relevant differences between implanted and non-implanted animals. Some metabolic changes occurred, as implanted cattle had reduced NEFA levels and yielded steaks that were tougher on average. Although implanted cattle did show altered NEFA levels, transit had a much greater effect on this factor. Insulin/Glucagon ratio was not affected by implants, but was related to body weight.

Tables

Table 1. Finishing trial diet[a]

Ingredient	%
Whole Shell Corn	55.00
High Moisture Corn	19.75
Corn Silage	13.00
Liquid Supplement[b]	4.25
CP[c]	11.50
NE_m, Mcal/cwt[d]	91.35
NE_g, Mcal/cwt[d]	60.75

[a]DM basis.
[b]Provided monensin and tylosin to make final diet 28 g/T and 11 g/T, respectively; provided vitamins and minerals to meet or exceed nutrient requirements (NRC, 1996).
[c]Based on weekly sample analysis.
[d]Derived from tabular values for feeds used (NRC, 1996).

Table 2. Interim period and cumulative performance of steers by treatment[a]

	Control	Implanted	SEM
Initial BW, lb[b]	949	945	10.5
1 to 21 d			
BW, lb[b]	1027	1037	8.5
ADG, lb	3.85	4.57	0.23
DMI, lb	21.44	21.9	0.43
F/G	5.59	4.81	0.32
21 to 42 d			
BW, lb[b]	1087	1109	10.4
ADG, lb	3.00	3.62	0.21
DMI, lb	23.07	23.83	0.46
F/G	7.72	6.61	0.33
42 to 70 d			
BW, lb[b]	1162	1203	12.0
ADG, lb	2.82[c]	3.45[d]	0.11
DMI, lb	22.39	23.03	0.70
F/G	7.98	6.68	0.36
1 to 70 d			
ADG, lb	3.18[e]	3.83[f]	0.05
DMI, lb	22.31	22.93	0.45
F/G	7.02[c]	5.99[d]	0.18
Estradiol, pg/ml	19[e]	9[f]	1.1

[a]Least squares means.
[b]Adjusted with 4% shrink.
[cd]Means with uncommon superscripts differ ($P < 0.10$).
[ef] Means with uncommon superscripts differ ($P < 0.05$).

Table 3. Carcass characteristics by treatment[a]

	Control	Implanted	SEM
HCW, lb	738[d]	766[e]	6.96
Dress %[b]	62.15	62.18	0.004
Rib fat, in	0.43	0.44	0.03
Rib eye area, in^2	11.78[d]	12.18[e]	0.14
KPH, %	1.94	1.86	0.11
Marbling[c]	5.44	5.15	0.15
Yield Grade	3.04	3.03	0.08

[a]Least squares means.
[b]Adjusted with 4% shrink.
[c]5.0=small^0; 4.0=slight^0.
[de]Means with uncommon superscripts differ ($P < 0.05$).

Table 4. Insulin, glucagon, and insulin/glucagon ratio by treatment and weight block[ab]

	CON Light	CON Heavy	IMP Light	IMP Heavy	SEM
Pre-Transit					
Insulin, pg/ml[c]	475[de]	355[d]	665[e†]	287[d]	67.37
Glucagon, pg/ml	108[fg]	104[fg]	115[f]	81[g]	8.34
Insulin/Glucagon	4.76	3.59	6.46	3.59	0.88
Post Transit					
Insulin, pg/ml[c]	631[d]	324[e]	450[de†]	284[e]	63.08
Glucagon, pg/ml	107	88	105	90	9.49
Insulin/Glucagon	6.31[f]	3.96[g]	4.37[g]	3.22[g]	0.65

[a]Least squares means.
[b]Light and heavy denote weight block
[c]Implant treatment * transportation * weight block interaction ($P = 0.087$).
[de]Means with uncommon superscripts differ ($P < 0.05$).
[fg]Means with uncommon superscripts differ ($P < 0.10$).
[†]Corresponding values within a column differ ($P < 0.10$).

Table 5. Live animal muscle glycogen and NEFA by treatment[a]

	Control	Implanted	SEM
Pre-transit			
NEFA, meq/l	0.156[c*]	0.116[d*]	0.01
Longissimus Glycogen, µmol/g[b]	111	104	11.7
Semimembranosis Glycogen, µmol/g[b]	86	79	5.4
Post-transit			
NEFA, meq/l	0.228[*]	0.212[*]	0.02
Longissimus Glycogen, µmol/g[b]	97	103	11.5
Semimembranosis Glycogen, µmol/g[b]	97[c]	68[d]	9.7

[a]Least squares means.
[b]Fresh wt. basis, obtained from live animal.
[cd]Means with uncommon superscripts differ ($P < 0.05$).
[*]Corresponding values within a column differ ($P < 0.05$).

Table 6. Carcass pH, objective color (L*, a*, b*) and shear force of the longissimus dorsi by treatment

	Control[a]	Implanted[a]	SEM
pH[b]	5.48[g]	5.43[h]	0.01
L*[bc]	36.88	37.09	0.45
a*[bd]	24.38	24.05	0.45
b*[be]	11.13	10.83	0.19
Shear force, kg	3.58[g]	4.42[h]	0.21
Shear force distribution[f]			
Shear < 3.5 kg, %	46.15	9.09	
Shear >3.5, < 5.0 kg, %	53.85	72.73	
Shear > 5.0 kg, %	0	18.18	

[a]Least squares means.
[b]Determined at time of grading.
[c]0=black, 100=white.
[d]Negative values = green, positive values = red.
[e]Negative values = blue, positive values = yellow.
[f]Chi-Square probability = 0.06.
[gh]Means with uncommon superscripts differ ($P < 0.05$).

Table 7. Muscle residual glycogen and lactate concentrations[a]

	CO	IMP	SEM
Longissimus dorsi			
Glycogen, µmol/g	21	25	1.80
Lactate, µmol/g	87	91	1.30
Glycolytic potential, µmol/g[b]	128[d]	141[e]	3.99
Lactate, %[c]	68.4	65.0	1.71
Semimembranosis			
Glycogen, µmol/g	44	45	2.49
Lactate, µmol/g	97	99	1.78
Glycolytic potential, µmol/g[b]	186	189	5.56
Lactate[c]	52.8	52.7	1.42
Psoas major			
Glycogen, µmol/g	13[f]	10[g]	1.18
Lactate, µmol/g	81	79	1.33
Glycolytic potential, µmol/g[b]	106[f]	98[g]	3.04
Lactate[c]	76.4	80.5	1.79
Illiacus			
Glycogen, µmol/g	9	8	1.25
Lactate, µmol/g	69	72	1.37
Glycolytic potential, µmol/g[b]	87	88	2.54
Lactate[c]	79.9	82.7	2.31

[a]Least squares means
[b]Calculated by the equation (2 X glycogen) + lactate.
[c] µmol*g^{-1} lactate / µmol*g^{-1} glycolytic potential x 100.
[de]Means with uncommon superscripts differ ($P < 0.05$).
[fg]Means with uncommon superscripts differ ($P < 0.1$).