Adrenaline sensitivity leads to leanness in high muscled cattle

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Abstract

Carcass weight and fat depth are used extensively within the beef industry as indicators of saleable meat yield, an important determinant of profitability. Estimated breeding values (EBV) for retail beef yield (a composite of eye muscle area and fatness) (ABRI, 2006) are increasingly used by Australian producers to select superior animals, typically showing increased expression of muscle and lower levels of fatness (Perry *et al*., 1993). The consequences of this selection on physiological mechanisms within the animal are not clear. In this paper we report the impact of selection for muscling in Angus steers on adipose tissue sensitivity to adrenaline with the hypothesis that selection for muscling will not alter adipose tissue sensitivity to adrenaline. Ten low muscled and 11 high muscled, 18 month old Angus steers were challenged with 7 adrenaline levels ranging from 0.2 to 3 µg/kg liveweight. Blood samples were taken at 16 time points between -30 to 130 minutes relative to administration of challenge. Peak Non-Esterified Fatty Acid (NEFA) concentration in response to challenges was analysed. The high muscled genotype showed a 20% higher ($P<0.05$) peak NEFA concentration at all levels of adrenalin challenge compared to the low muscled steers. Peak NEFA concentrations for the high and low muscled genotypes were 0.162mM ± 0.008 and 0.135mM ± 0.009. Given that adrenalin causes rapid lipolysis within adipose tissue, the greater sensitivity to adrenalin in high muscled steers may partly explain their leanness.

Introduction

Beef producers are generally paid by processors on a grid basis determined by carcass weight and fat depth, with heavy financial penalties incurred if optimal specifications are not met. In more recent times there has been increased movement towards yield and quality based payment to allow for a transparent trading environment in which the producer is rewarded according to their impact on both retailer (saleable beef yield) and consumer (meat quality) value (Polkinghorne, 2006). In order to increase yield in Australian beef cattle herds, producers have been selecting for muscling using genetic selection tools such as Estimated Breeding Values (EBV), visual muscling selection techniques, and gene markers for the non functional myostatin gene. Due to the strong negative correlation in beef cattle between muscling and subcutaneous fat (Perry *et al*., 1993), producers can reach target carcass weights without being penalized for excess fat cover. However, fat reduction in cattle will possibly reduce marbling which accounts for around 10 – 15% of variance in palatability (Dikeman, 1987) and attracts a premium in some export markets. With continued selection for muscling this correlation must be monitored, as the physiological mechanisms which are responsible for the decreased adipose tissue and increased beef yields in high muscled cattle are unknown.

More heavily muscled genotypes of cattle demonstrate increased whole body responsiveness to insulin (Bonny *et al*. 2007), while Gardner *et al*. (2005) showed that both high muscled cattle and sheep had reduced adrenaline sensitivity at the level of the muscle compared to lower muscling animals. Generically, this would support greater rates of anabolism and reduced amounts of catabolism in muscle, leading to greater lean deposition. However, if this was also the case in adipose tissue, there would be more lipogenesis and less lipolysis, creating a net increase in fatness, which contradicts the commonly observed phenotypic leanness of high muscled animals. Therefore we must assume that this decrease in adrenaline sensitivity seen in the high muscled animals does not extend to adipose. Thus we can propose the hypothesis that selection for muscling will have no effect on adrenaline sensitivity of adipose tissue.

Materials and methods

Adipose tissue sensitivity to adrenaline was examined in 21 Angus steers at 18 months of age (± 1 month). There were 10 low muscled and 11 high muscled steers from a herd selected for muscling (via a visual scoring system (Perry *et al*. 1993) since 1992. They were habituated in individual pens on an ad-libitum grain based diet for 2 weeks prior to the challenge period. At the end of habituation, an indwelling jugular catheter was inserted.

Each steer was then challenged with adrenaline at 7 different levels (0.2, 0.4, 0.6, 1.0, 1.6, 2.2 and 3.0 μg/kg liveweight). Two randomly allocated challenges were administered per day at either 10:00hr or 14:00hr. Blood samples were taken from the catheter at -30, -15, -10, -5, 0, 2.5, 5, 10, 15, 20, 30, 45, 60, 120, 125 and 130 minutes relative to challenge administration. Samples were collected in tubes containing EDTA, and following centrifugation plasma was decanted and stored at -80 ºC until analysis for NEFA concentration using a NEFA C kit (Wako®, Wako Pure Chemicals Industires, Osaka. Cat. No. 279.75401) was completed.

A derived function with multiple exponential components was fitted to each animal's plasma substrate response over time for each of the adrenalin challenges. This function had the following form:

$$y(t)= Int +(e^{[-\beta t]*[-\gamma/(\beta-\alpha)+\gamma/(\beta-\alpha-\Delta)-\varepsilon/\beta+\gamma/(\beta-\alpha)*e((\beta-\alpha)t)-\gamma/(\beta-\alpha-\Delta)*e((\beta-\alpha-\Delta)t)+\varepsilon/\beta*e(\beta t)]})$$

Where y(t) is substrate concentration (mM) at any given point in time, t is time (minutes), Int is basal substrate concentration (mM) prior to challenge, γ, β, α, Δ are exponential constants and ε is the adjustment from basal substrate concentrations after substrate levels have returned to a steady state. From this function, NEFA concentration and area under curve till maximum concentration was derived. These parameters were analysed using a linear mixed effects model, with liveweight and P8 fat depth as covariates, and animal within sire as the random term.

After completing the challenges, the steers were lotfed until export market specifications were met (280-380 kg carcass weight & 7 to 22 ml of P8 fat), after which they were slaughtered at a commercial abattoir. Half of each carcass was boned-out into primal cuts with subcutaneous fat trimmed to 10mm. All primals were weighed individually, along with lean trim, fat trim and bone to determine retail beef yield.

Results and discussion

When trimmed to export standards of 10mm subcutaneous fat, the high muscled cattle had 2.61 percent less trimeable fat ($P<0.05$), and had 2.95 percent more saleable meat per kilogram carcass weight than the low muscled steers ($P<0.01$). There was no difference in the percentage of bone ($P=0.52$). Thus, the increase in yield in the high muscled genotype steers was largely driven by these animals having less trimeable subcutaneous and intermuscular fat, and more muscle.

Table 1. The mean ± standard error for the weight of trimeable fat, saleable meat and bone as a percentage of carcass weight for the high & low muscling genotype steers

	High Muscled	Low Muscled	Significance Level
Trimeable Fat (%)	15.71 ± 0.75	18.32 ± 0.79	P<0.05
Saleable Meat (%)	65.34 ± 0.61	62.39 ± 0.67	P<0.01
Bone (%)	18.07 ± 0.4	18.45 ± 0.42	N.S.

As the level of adrenaline challenge increased, the peak NEFA concentration also increased ($P<0.01$) by around 40% across the range of adrenaline challenges (Figure 1). The peak NEFA response was 20% higher ($P<0.1$) for the high muscled genotype steers compared to the low muscled genotype steers across all adrenaline challenges. The results for the area under curve data mirrored this (data not shown). This suggests that high muscled cattle are more responsive to stress at all given levels, which is contrary to our initial hypothesis. This may indicate a catabolic/lipolytic mechanism explaining the decreased adiposity displayed in these animals.

The physiological mechanisms which underpin this difference in lipolytic response to adrenaline is unclear, however work in obese humans has shown reduced blood flow through adipose tissue, leading to decreased plasma triacylglycerol (TAG) extraction from this tissue compared to lean subjects (Goossens, 2008). Decreased blood flow may limit the distribution of adrenaline in times of stress to all adipose sites. Adrenaline stimulates hormone sensitive lipase (HSL; EC 3.1.1.3), the rate limiting enzyme of lipolysis (Holm *et al*., 2000). Therefore lower blood flow leads to less activation of HSL by adrenaline, resulting in lower levels of lipolysis in obese subjects than in their lean counterparts, reflected by a decrease in release of NEFA from the adipose tissue into plasma. Jocken and Blaak (2008) state that obese humans also have lower adipose adrenaline sensitivity due to reduced HSL expression. This is commonly coupled with obese subjects having a decreased number and function of β_2-adrenoreceptors (Reynisdottir *et al.* 1994).

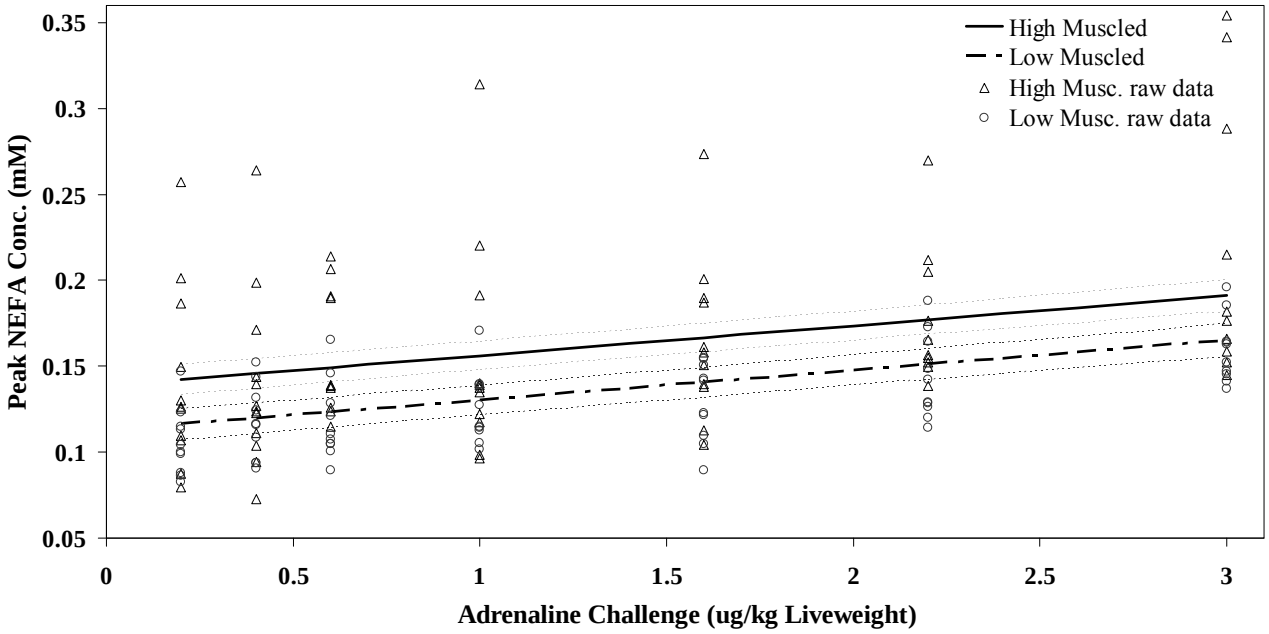

Figure 1. The effect of adrenaline challenge, within genotype, on the peak plasma NEFA concentration. The trend lines represent the least squared means estimates with standard error lines. The raw data is also displayed for both genotypes.

Conclusions

Animals selected for high muscling have less trimeable fat at slaughter than their low muscling counterparts substantially contributing to the increased saleable beef yield. The high muscled genotype steers also demonstrated higher adrenaline sensitivity in adipose and subsequently more lipolysis, thus we can conclude that selection for muscling does increase adrenaline sensitivity in adipose. This response may be delivered through numerous mechanisms which affect the rate of lipolysis.

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