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## Plasma cholesterol and triglyceride concentrations in yearling Angus cattle

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### ABSTRACT

The concentrations of cholesterol and triglyceride in blood plasma were compared in two genetically distinct herds of Angus cattle. Any concentration differences between the herds should indicate a realised genetic response to selection for yearling weight.

Seventy-four yearling Angus cattle from the Waikite weight-selection and control herds were blood sampled after a 24-h fast in July, September and November 1993 (heifers twice, bulls three times;  $n = 178$  samples). Plasma cholesterol concentrations averaged 2.73 and 2.91 (s.e.d. 0.08) mmol/l in the two herds, respectively ( $P < 0.05$ ), and triglyceride concentrations averaged 0.296 and 0.316 (s.e.d. 0.015) mmol/l, respectively. There was a 30 kg (14%) difference in yearling live weight between the two herds ( $P < 0.001$ ). Both metabolites showed moderate between-animal repeatabilities,  $0.52 \pm 0.11$  and  $0.37 \pm 0.16$  respectively, and there was a positive phenotypic correlation of 0.40 between cholesterol and triglyceride concentrations within herds.

These results show that a 14% higher weight-for-age as a result of selection was associated with a 6% lower plasma cholesterol concentration. This was consistent with our previously observed cholesterol difference in cooked beef from the same herds.

**Keywords:** cholesterol; triglyceride; plasma; cattle; Angus; weight; selection lines.

### INTRODUCTION

Variation in blood cholesterol concentration is inherited in beef cattle (Stufflebean and Lasley, 1969). Selection studies to alter cholesterol concentration have been successful in mice (e.g. Weibust, 1973) and in pigs (e.g. Rothschild and Chapman, 1976), and cholesterol concentrations would also probably respond to direct selection in cattle. We monitored blood plasma cholesterol and triglyceride levels in beef cattle from herds selected on the basis of yearling weight, in order to determine if any changes in plasma concentration of these lipid moieties had occurred as a result of over 20 years of direct selection.

Cholesterol and triglycerides in blood plasma are important in human diseases associated with arterial blockages (e.g. heart attack, stroke, etc). Human serum cholesterol levels are influenced by *in vivo* cholesterol synthesis in the liver, by rates of biliary excretion, genetic factors and dietary consumption of lipids including triglycerides and cholesterol (Rose, 1990). For example, consuming 100 mg of dietary cholesterol (= one serving of steak, representing about a third of a normal day's intake) raises human serum cholesterol by 0.13 mmol/l, a 2.5% rise (Harper *et al.*, 1975). It is well known that arterial blockages are associated with (i) elevated serum levels of very low density lipoproteins enriched with triglycerides, or (ii) elevated serum levels of low density lipoproteins enriched with cholesterol, or (iii) elevated serum levels of both cholesterol and triglycerides. It is therefore pertinent to monitor the content of both in animals which are human food sources.

### MATERIALS AND METHODS

#### Animals

Angus yearling bulls and heifers in this study were born in 1992, the 21st calf crop of a weight-selection experiment

begun at Waikite (Rotorua) in 1971/72. The main selection experiment was described in detail by Baker *et al.* (1991). Briefly, two herds from the same genetic origin were established in 1971/72, with selection among the bulls and heifers of one herd being for increased adjusted yearling weight (AS1 herd), whilst selection in the other was at random (ACO herd). Over 100 cows per herd were wintered to calve each year until 1991, after which numbers were reduced. The 1992 calf crop was born in September/early October mainly at Whatawhata Research Centre, but with a sample of each cow herd calving off-station near Raglan. Animals from both sites were merged into one group from late October 1992. All calves were weaned in early March 1993, at which time the bull calves ( $n = 33$ ) were transferred to Tokanui Station whilst the heifers remained at Whatawhata ( $n = 41$ ). Calves grazed on predominantly ryegrass/white clover pastures through to November 1993, with some supplementation for bulls with hay and silage in the winter at Tokanui.

#### Blood samples

In July, September and November 1993, bulls at Tokanui were weighed immediately off pasture, and fasted in the yards for 24 hours, after which a blood sample was collected from the tail of each animal into a vacutainer tube containing EDTA. The same procedure was followed for heifers at Whatawhata in September and November 1993. In total, 178 blood samples resulting from 5 combinations of sex x month were processed.

Blood was centrifuged at 2400 g for 20 minutes. The clear supernatant was carefully aspirated, and plasma cholesterol and triglyceride levels were determined by using kits supplied by Boehringer Mannheim GmbH, and analysed on a Hitachi 717 autoanalyser (respectively, Kattermann *et al.*, 1984 and Wahlefeld, 1974).

## Data analyses

Least squares analyses were applied to the live weight data, using the Genstat (1990) computer programme, whilst restricted maximum likelihood procedures (Patterson and Thompson, 1971) were applied to the blood sample data to take account of repeated records. The final model for live weights included fixed effects for herd (AS1 or ACO) and sex of calf, with the remaining factors (site of birth, age of dam, the 2-way interactions and a covariate for date of birth) being excluded from the model as non-significant. For the blood traits, the final model included fixed effects for herd, sex x month combinations, their interaction (cholesterol only), and site of birth, with a random effect for animal. There were insufficient sires and progeny per sire to carry out heritability or genetic correlation analyses.

## RESULTS

Table 1 shows the herd means for cholesterol and triglyceride concentrations for each month separately, and overall, along with estimates of variability. Coefficients of variation were 0.14 for cholesterol and 0.25 for triglyceride, indicating that sampling variation was a less important factor for cholesterol. Overall, plasma cholesterol concentration was 6% lower in the AS1 herd than in the ACO herd ( $P < 0.05$ ). The herd mean difference in triglyceride concentration was in the same direction and of the same percentage magnitude as for cholesterol, but with a difference only significant at  $P < 0.10$ . The correlation between cholesterol and triglyceride concentrations within herds was 0.40.

**TABLE 1:** Plasma concentrations (mmol/l) of cholesterol and triglyceride after a 24 h fast in yearling bulls ( $n=33$ ) and heifers ( $n=41$ ) from the weight-selection (AS1) and control (ACO) herd.

Sex	Month	AS1 herd		ACO herd	
		Cholesterol	Triglyceride	Cholesterol	Triglyceride
Male	July	2.72	-	2.89	-
	September	2.74	0.40	2.84	0.41
	November	2.60	0.23	2.69	0.23
	s.e.d. (months)	0.07	0.015		
Female	September	2.32	0.29	2.89	0.33
	November	3.28	0.31	3.24	0.34
	s.e.d. (months)	0.06	0.013		
	Overall <sup>a</sup>				
	: mean	2.73	0.296	2.91	0.316
	: s.e.d. (herds)	0.08	0.015		
	Phenotypic s.d. <sup>b</sup>	0.39	0.076		

<sup>a</sup> REML estimate from a repeatability analysis, after adjustment for sex, month and herd.

<sup>b</sup> The phenotypic s.d. was after adjustment for sex, month and herd; standard errors for main effects (pooled over the other effects) were from the full REML analysis.

Herd differences in cholesterol were larger in July and September, but not significant in November (i.e. a significant interaction,  $P < 0.05$ ). The site of calving effect, where yearling weights tended to be lower at the off-station site, was as great as the selection herd effect, but opposite in direction (2.71 mmol/l off station where weights were lower *versus* 2.93 mmol/l at Whatawhata;  $P < 0.001$ ). For plasma triglyceride,

there was no interaction between selection herd and calving site. The selected herd had a 6.3% lower concentration than the Control herd, whilst the off-station site (with lower yearling weights) led to a 17% lower triglyceride concentration than the Whatawhata site ( $P < 0.001$ ): 0.277 *versus* 0.335 mmol/l, respectively. Between-animal repeatabilities over months were  $0.52 \pm 0.11$  and  $0.37 \pm 0.16$  for cholesterol and triglyceride respectively.

Yearling (September) live weights, adjusted for known fixed effects, were 246 and 216 kg for the AS1 and ACO herds respectively, a 30 kg or 14% difference ( $P < 0.001$ ). After adjusting for fixed effects, the regression of cholesterol concentration on the live weight at sampling time was  $0.0026 \pm 0.0013$  mmol l<sup>-1</sup> per kg ( $P < 0.05$ ; correlation = 0.16), and the corresponding regressions for individual herds were  $0.0006 \pm 0.0017$  mmol l<sup>-1</sup> per kg (AS1 herd; not significant) and  $0.0059 \pm 0.0020$  mmol l<sup>-1</sup> per kg (ACO herd;  $P < 0.01$ ).

## DISCUSSION

We have focused on plasma cholesterol and triglyceride levels in beef cattle because of the importance of these moieties in the human diet. Traditionally, it has been difficult to manipulate lipid components in ruminants. Only limited success has been achieved in this area through endocrinological or immunological approaches. The obstacle arises due to the microbial fermentation of grasses in the rumen, resulting in the formation of short chain fatty acids (acetate, a major by-product, and propionate and butyrate). These by-products are the precursors for cholesterol and triglycerides in cattle (Vernon and Flint, 1988). In comparison, monogastrics use glucose and dietary fatty acids as precursors. Our results show that genetic manipulation of growth and its associated changes in lipid components could provide an alternative means for modulating the cholesterol concentration of ruminant products. This approach could be effective in the long run and may have an easy consumer acceptance.

Cholesterol concentrations were significantly lower in the AS1 than in the ACO herd by 6%, and triglycerides were also correspondingly lower (although, perhaps as a result of a higher coefficient of variation, this contrast was not significant). The cholesterol results were consistent with previous work carried out on these herds (Morris *et al.*, 1995) where we observed a 7% lower cholesterol concentration in cooked meat from AS1 cattle than from ACO cattle ( $P < 0.10$ ), although not in raw meat. The reason for the disparity between results from cooked and raw meat is unclear. The positive correlation (0.40) between cholesterol and triglyceride was of interest because acetate is the precursor to both in ruminants, although independent pathways are utilised in generating these two lipids (Baldwin and Smith, 1971).

The within-subclass regressions of cholesterol concentration on live weight were small and positive, but inconsistent in size between herds. The fact that cholesterol concentration was lower in AS1 than ACO cattle, however, implies that the genetic correlation and regression are negative in sign. They would need to have respective values of -0.37 and -0.006 mmol l<sup>-1</sup> per kg to give the observed realised response in cholesterol concentration to a 30 kg increase in yearling weight (assuming an 0.3 heritability for cholesterol).

There was a 14% difference in yearling weight between the herds. This implies that a 6% reduction in plasma cholesterol was achieved as a correlated response to a 14% increase in live weight, or a -1:2 change in cholesterol relative to live weight. We know of no other weight-selection experiment where plasma cholesterol concentrations have been examined.

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