# Metabolic changes in cattle due to the specific effect of the tick, Boophilus microplus

### By J. C. O'KELLY AND P. M. KENNEDY<sup>†</sup>

## CSIRO, Division of Animal Production, Tropical Cattle Research Centre, PO Box 542, Rockhampton, Queensland 4700, Australia

### (Received 5 December 1979 – Accepted 16 December 1980)

1. An experiment was designed to provide information on the alterations in body metabolism which would account for the loss of body-weight in cattle due to the specific effect (factors other than reduced food intake) of the tick *Boophilus microplus*.

2. Two groups of British (Shorthorn  $\times$  Hereford) and Africander  $\times$  British calves, each approximately 6 months old, were used: one group (treated) of each breed was tick-infested and the other (control) was tick-free. Within breeds, calves in the control group were pair-fed to calves in the treated group.

3. In both breeds, the effect of ticks: (a) depressed packed cell volume, serum alkaline phosphatase (EC 3.1.3.1) and amylase (EC 3.2.1.1) activities, plasma cholesterol and phospholipid levels, serum iron and albumin levels, (b) increased the plasma levels of urea-nitrogen and  $\gamma$ -globulin (c) increased rectal temperature, water intake, urine volume, urinary and faecal total N, urinary urea-N and  $\alpha$ -amino acids, the excretion of water, sodium and potassium in the faeces and (d) reduced N balance, N and dry-matter digestibilities.

4. In the British breed, ticks increased the excretion of K with a corresponding decrease in the excretion of Na in the urine and inreased the plasma clearance of bromsulphthalein.

5. A second experiment showed that the specific effect of tick infestation increased the flow of organic matter (OM) from the abomasum and the fractional turnover of rumen fluid of Hereford steers. It was also shown that the decrease in OM digestibility in the gastrointestinal tract was largely due to a decrease in OM digestibility in the rumen and that the increased urinary urea excretion and plasma urea concentration was caused by higher production rates of urea despite a tendency for lowered urea degradation in the gastroinestinal tract.

The tick *Boophilus microplus* causes large economic losses in cattle production in several countries. A study under carefully controlled nutritional conditions showed that 65% of the body-weight loss in Hereford steers resulting from tick infestation was due to an anorectic effect and 35% to a specific effect (Seebeck *et al.* 1971). There are no comparable studies in zebu breeds of cattle but it is known that these breeds are more resistant to the tick than British breeds.

In parts of tropical Australia, grazing cattle are exposed to natural field infestations of ticks but when taken into yards and confined for fattening or biological studies they are kept free of ecto-parasites by treating with acaricides. There is evidence that tick infestation has prolonged after-effects on metabolism (Seebeck *et al.* 1971; Vercoe & O'Kelly, 1972) and the normal relationships between body-water and body-composition measurements (Springell *et al.* 1971). Thus, assessment of animal performance or breed comparisons in metabolic studies on yarded animals may be confounded by a previous history of tick infestation. Nutritional rehabilitation aimed at alleviating the effects of ticks present or replacing any deficits in body tissues arising from previous tick infestation would be helped by knowing the changes in host metabolism which are induced by the specific effect of the parasite.

This paper reports the results of experiments designed to provide information on alterations in body metabolism which would account for the loss in body-weight due to the specific effect of tick infestation on British and Africander  $\times$  British cattle.

† Present address: Department of Animal Science, University of Alberta, Edmonton, Alberta T6G 2E3, Canada.

#### EXPERIMENTAL

#### Animals and treatments

*Expt* 1. Sixteen bull calves of two breeds, each approximately 6 months old, were used in a 28 d trial but because only eight metabolism cages were available the experiment was divided into two periods. The British calves were eight Shorthorn  $\times$  Hereford crosses; the animals referred to subsequently as AX were eight Africander  $\times$  British crosses. All animals received subcutaneous injections of trichlorphon (Neguvon injectable; Bayer Leverkusen Ltd) to control helminths.

In each period one group of four animals (the tick group) was infested with tick larvae and another group of four animals (the control group) was kept tick-free. Each calf in the control groups was paired with one of similar body-weight in the tick groups and offered the same amount of feed as consumed by the tick-infested calf during the previous day.

British calves only were used in the first period. Each calf in the tick group was infested with 40000 larvae followed by the same dose of larvae 2 and 4d later, using the methods of O'Kelly & Spiers (1976). With our system of artificial tick infestation it is usual that all females which mature from a single dose of larvae do so within 18–23 d with major peaks on days 19–20. After 17 d, the animals and their pair-fed controls were removed from pens to metabolism cages for the remaining 11 d. This design ensured that the time in metabolism cages corresponded to the maturing of all female ticks on treated animals from the three infestations of larvae. The second period started 13 d after the start of the first period and the same procedure was followed with the AX calves. However, in an attempt to achieve a similar number of female ticks maturing on both breeds, the AX calves in the tick group were infested with three doses of 60000 larvae, since preliminary studies showed that the proportion of larvae reaching maturity from equal doses was approximately 4% on the AX compared to approximately 7% on the British calves.

A diet of standard cattle cubes was offered in portions of 1.5 kg twice daily at 07.30 and 13.30 hours to each tick-infested calf. Water was freely available throughout. The cattle cubes were composed of (g/kg); 150 lucerne, 150 wheatmeal, 400 sorghum, 50 meatmeal, 50 cotton seed meal, 60 coconut meal, 50 bran, 10 salt, 60 lime and 20 molasses.

The mean  $(\pm sE)$  body-weight (kg) at the beginning of the treatment periods were: British control group  $152\pm7$ , British tick-infested group  $149\pm4$ , AX control group  $172\pm1$ , AX tick-infested group  $171\pm7$ .

Daily water and food intakes, rectal temperature and excretions of faeces and urine were measured for each animal during the time in the metabolism cage. The urine was collected in plastic vessels containing 200 ml copper sulphate-sulphuric acid preservative. Daily subsamples of urine and faeces from each animal were bulked over each collection period in proportion to the amounts daily excreted. The number of adult female ticks between 4.5 and 8 mm was counted daily on the left side of tick-infested calves in the metabolism cages. The daily tick counts multiplied by two was used as a measure of the daily tick load on each animal. Blood samples were obtained by jugular venepuncture at the beginning (day 1) and end (day 28) of the experimental period and each animal in the tick-infested groups and its pair-fed partner were bled at the same time after feeding. The clearance rate of a dose of BSP (Sulphobromophthalein sodium salt; Koch-Light Laboratories Ltd) was used as a test of liver function for each animal at the end of its experimental period. The animals were fitted with catheters in each jugular vein and infused with an aqueous solution of BSP (50 g/l) at a dose rate of  $2\cdot 2 \text{ mg/kg}$  body-weight according to the method of Hunt & McCosker (1967).

Methods for the analysis of plasma lipids, glucose,  $\alpha$ -amino-nitrogen, alkaline phosphatase (EC 3.1.3.1), packed cell volume, dry matter (DM) and total N in urine, faeces and feed

were those of O'Kelly (1973) and methods for the electrophoretic separation of serum proteins were those of O'Kelly & Spiers (1976). Urinary  $\alpha$ -amino acids were estimated by the method of Wells (1969). Serum amylase (EC 3.2.1.1) was assayed by the saccharogenic method of Henry & Chiamori (1960) and serum iron was determined by the method of Ness & Dickerson (1965). Urea-N and creatinine were determined by AutoAnalyzer procedures (Technicon Instruments Corp., 1963). Sodium and potassium were estimated by means of an EEL flame photometer on diluted samples of plasma and urine and in faecal samples after ashing at 700° and extraction with 0.1 M-nitric acid.

The results were subjected to analyses of variance using the method of least squares and breeds were analysed separately. The results for each blood response variable on day 28 (Tables 1 and 2) were analysed taking account of the day 1 differences between animals in that variable by analysis of covariance.

Expt 2. Six Hereford steers, each approximately 19 months old, were used. One group of three animals (the control group) was kept tick-free and the other group of three animals (the tick group) was infested with tick larvae as described for Expt 1, except that each steer received 10000 larvae on each of 2 d of every 3 d for the first 24 d, to ensure that female ticks matured every day for the final 3 weeks of the experiment. All animals had been surgically prepared 6 months before the experiment with cannulas into the rumen and, in addition, two animals from the control group and two from the tick group were fitted with simple cannulas in the abomasum.

All steers were given lucerne hay: cracked milo grain (2:1), 22.7 g N/kg organic matter (OM), at the rate of 4.5 kg/d. The ration was given twice daily in equal amounts for 28 d. Steers were then placed into metabolism cages for 14 d and offered 0.19 kg portions of feed at hourly intervals using a belt feeder. Water was freely available throughout. The methods of collection of urine and faeces were the same as those described in Expt 1. Rectal temperatures were measured at 10.00 hours each day. Estimates of production and degradation rates of plasma urea were made on the sixth day after animals had entered metabolism cages. Each steer was given an intravenous priming dose of [14C]urea (5 ml, 40  $\mu$ Ci/ml saline, 9 g sodium chloride/l) followed by a continuous infusion (800 ml/d,  $0.5 \,\mu$ Ci/ml saline) for 24 h. Blood (10 ml) was taken at intervals of 45 min during the last 8 h of tracer infusion, through a catheter previously established in a jugular vein. Degradation of plasma urea in the gastrointestinal tract was calculated as the difference between urinary urea excretion over 10 d and the plasma urea production rate calculated from the specific radioactivity at plateau and the infusion rate of tracer (Kennedy, 1980). The flow of OM was estimated by reference to two non-radioactive markers, chromium complexed with ethylenediaminetetraacetic acid (Cr-EDTA) and Tris (1,10-phenanthroline)ruthenium (II) chloride (Ru-P), prepared using the methods of Downes & McDonald (1964) and Tan et al. (1971) respectively, with the omission of the radionuclide. The markers were continuously infused (150 mg Cr, 50 mg Ru/d, 800 ml/d) into the rumen for 7 d after a priming dose (150 mg Cr, 50 mg Ru). Abomasal digesta (400 g) were collected at 4 h intervals during 48 h (on the eleventh and twelfth days after animals had entered metabolism cages) and bulked for each steer and stored at  $-15^{\circ}$ . [<sup>51</sup>Cr]EDTA (0.5 mCi in 100 ml) was injected into several sites in the rumen of each steer on the twelfth day after aninals had entered metabolism cages, and the fractional turnover rate of rumen fluid was measured by reference to <sup>51</sup>Cr in filtered rumen fluid samples taken at 2 h intervals for 24 h after injection of dose.

A portion of the bulked abomasal digesta sample was separated into a supernatant fraction by centrifugation at 750 g for 30 min, and analyses for OM, Cr and Ru were performed on both supernatant and whole digesta. OM was determined by incineration at 600° for 6 h. Ru and Cr were determined by atomic absorption spectroscopy on samples

treated according to the methods of Megarrity & Siebert (1977) and Christian & Coup (1954), respectively. Flow of OM from the abomasum was calculated by reference to Ru and Cr by mathematical reconstitution of 'true abomasal digesta' as described by Faichney (1975). <sup>51</sup>Cr was assayed using a  $\gamma$ -spectrometer (model 5320, Packard Instrument Co. Inc., Downers Grove, Ill, USA).

#### RESULTS

## Expt 1. The results are presented in Tables 1-5.

Tick resistance in cattle is expressed as the number of adult female ticks resulting from a given dose of larvae but does not necessarily relate to the effect of ticks on the animal. Clearly, a major difficulty in designing experiments comparing the effect of ticks on the metabolism of hosts of different genotypes (and hence resistance) is to predict the doses of larvae that will produce comparable variations in the numbers of adult ticks within those genotypes. In an attempt to overcome this problem the doses of larvae applied to the AX calves were 1.5 times greater than those applied to the British calves. Despite the differential doses of larvae, the number of mature ticks carried by the AX calves was lower (P < 0.05) than the number carried by British calves. Ticks matured on the British animals on days 1-10 and on the AX calves on all eleven days in the metabolism cages. The mean ( $\pm$ se) daily number of mature ticks were British  $370 \pm 42$  and AX  $177 \pm 7$ , the sE values are between animal variations (3 df) within groups.

Within breeds, there were no significant differences between the groups in any of the blood factors determined at the beginning of the experimental period (day 1) and so the mean values for all eight animals are shown in Tables 1-2; the composition of the blood of control and tick-infested groups at the end of the experimental period (day 28) is shown as absolute changes from the day 1 values. Packed cell volume and serum Fe levels were significantly lowered (P < 0.05) by ticks only in the British calves (Table 1). Calculations based on an average uptake of 0.3 ml blood/tick per day (Springell et al. 1971) showed that the daily blood loss during infestation was 111 ml in the British and 53 ml in the AX calves. Further calculations, assuming that blood volume was 5% of body-weight, indicated that for each breed the reduction in red cell volume of the host was approximately equal to the amount removed by the ticks and agreed with the findings from British cattle where blood volumes were determined isotopically (Springell et al. 1971). The plasma concentrations of esterified and free cholesterol and phospholipid were higher (P < 0.01) in the AX than in the British calves but ticks lowered the concentrations of these lipid components in both breeds (Table 1). Ticks were without effect on the plasma concentrations of non-esterified fatty acids, glucose, Na and K (Table 1). The plasma glucose concentration was higher (P < 0.01) in AX than in British calves.

The concentrations of serum nitrogenous components are given in Table 2. The serum  $\alpha$ -amino-N concentration was higher (P < 0.01) in AX than in British calves and was not influenced by ticks. On the other hand, ticks significantly increased urea-N in both breeds. Serum albumin concentrations in both breeds were depressed (P < 0.05) by ticks which were without effect on the concentrations of  $\alpha$ - and  $\beta$ -globulins. In both breeds,  $\gamma$ -globulin increased (P < 0.001) in tick-infested animals and decreased (P < 0.01) in control animals from days 1 to 28. Within both control and tick groups the  $\gamma$ -globulin concentrations were higher (P < 0.01) in the British than in the AX calves. The activities of serum alkaline phosphatase and amylase were lowered (P < 0.01) by ticks in both breeds (Table 2). Serum amylase activity also decreased in the British control group during the experimental period but the terminal concentrations were still higher (P < 0.01) than in the tick-infested group.

In both breeds, rectal temperature, water intake, urine volume and faecal water significantly increased with tick infestation (Table 3). Food refusal commenced 4 d after

ļ

Table 1. The mean absolute changes from day 1 to day 28 in the packed cell volume, serum
iron concentration, plasma lipid composition, glucose and electrolyte concentrations of control
(CON) and tick-infested (TICK) calves

	Sho	rthorn × Her	eford	Africander cross		
		Changes in			Changes in	
	Day 1	CON	TICK	Day 1	CON	TICK
Packed cell volume	0.330	+0.004	-0.096**	0.325	+0.007	-0·014
Serum Fe ( $\mu g/l$ )	1274.5	+83.0	-537.1*	1189-5	+164.3	-216·2
Esterified cholesterol (mg/1)	586-5	+22.1	- 186·4**	831·0	+10.0	-220.0**
Free cholesterol (mg/l)	143.0	+7.2	-41·6**	214.5	+1.0	-40.3**
Phospholipid (mg/l)	1096.0	+15.0	- 202·5**	1341.0	+ 39.1	-289.5**
Non-esterified fatty acid (µequiv/l)	443	-108	-122	357	-8	-18
Glucose (mmol/l)	3.61	-0.03	-0.12	4.20	0.27	-0·01
Sodium (mmol/l)	149	-1	-2	148	+1	-1
Potassium (mmol/l)	4.2	Ō	0	4.3	+0.1	+0.1

(Mean values for eight calves are given for day 1; the changes in CON and TICK groups are mean values for four calves)

For each breed, the changes from day 1 were significant: • P < 0.05. \*\* P < 0.01.

Table 2. The mean absolute changes from day 1 to day 28 in the concentrations of serum nitrogenous constituents and serum enzyme activities of control (CON) and tick-infested (TICK) calves

(Mean values for eight calves are given for day 1; the changes in CON and TICK groups are mean values for four calves)

	SI	horthorn × H	ereford	Africander cross		
	Day 1	Cha	nges in	Day 1	Changes in	
		CON	TICK		CON	TICK
a-amino-N (mg/l)	39.9	+0.1	+1.0	50.9	-0.9	-0.2
Urea-N (mg/l)	97.0	+0.7	+20.0*	100-1	+0.6	+21.0*
Albumin (g/l)	36-9	+0.8	- 5.7**	39.8	+0.6	-3-3**
a-globulin (g/l)	8.8	+0.1	+0.2	<b>8</b> ∙0	+0.1	+0.1
$\beta$ -globulin (g/l)	7.6	-0.3	-0.4	7.0	+0.4	-01
γ-globulin (g/l)	18-5	-2·2**	+ 3.2***	13.7	-3.2**	+ 3.2***
Alkaline phosphatase (King-Armstrong units/1)	90-4	-2.8	- 17- <b>9</b> *	94.8	<b>−0·7</b>	-17.9*
Amylase (units / 1)	2485	-728**	-1359***	1804	68	- 366**

For each breed, the changes from day 1 were significant: • P < 0.05, •• P < 0.01, ••• P < 0.001. † Henry & Chiamori (1960). Table 3. The daily mean rectal temperature, water balance and dry matter (DM) digestibility of control (CON) and tick-infested (TICK) calves during the period (days 18-28) in the metabolism cages

	Shorthorn × Hereford			Africander cross			
	CON	TICK	SED	CON	TICK	SED	
Rectal temperature (°)	38.6	39.4**	0.2	38.6	39.1**	0.1	
Water intake (1)	8.6	12.1*	0.9	10.5	13.9*	1.3	
Urine volume (1)	5-1	8.1*	1.0	5.9	9.7*	1.6	
Faecal water (l)	1.0	1.2*	0.02	1.4	1.7*	0.07	
Apparent water balance (1)	2.4	2.7	0.16	3.2	2.6	0.6	
DM intake (kg)	1.65	1.65		2.28	2.28		
Faecal DM (kg)	0.38	0.42*	0.04	0.57	0.65*	0.02	
DM digestibility	0.770	0.748*	0.017	0.750	0.715*	0.011	

(Each value represents a mean result for four calves. Comparisons were made between pair-fed animals and standard errors of differences (SED) are given (i.e. 3 df))

For each breed, the values were significantly different from control values: • P < 0.05, \*\* P < 0.01.

Table 4. The daily mean nitrogen balance and digestibility and daily urinary nitrogen constituents of control (CON) and tick-infested (TICK) calves during the period (days 18-28) in the metabolism cages

(Each value represents a mean result for four calves. Comparisons were made between pair-fed animals and standard errors of differences (SED) are given (i.e. 3 df))

	Shorthorn × Hereford			Africander cross			
	CON	TICK	SED	CON	TICK	SED	
N intake (g)	52.1	52·1		68·8	68·8		
Urinary N (g)	29.9	33.6**	0.9	29-4	35.9**	0.8	
Faecal N (g)	13.0	15.8*	0.8	19-4	23.3**	0·7	
N balance (g)	9.2	2.7*	1.6	19.9	9.6***	1.1	
N digestibility	0.752	0.699*	0.019	0.718	0.662**	0.013	
Urinary urea-N (g)	21.5	24·1•	1.0	19-4	23.2*	1.4	
Urinary creatinine (g)	5.3	5.0	0.8	5.7	<b>8</b> ⋅3	2.1	
Urinary α-amino acids (m-equiv)	23-2	27.5*	1.1	27.7	35-2*	2.0	
Faecal N concentration (g/kg dry matter)	34-2	38.1*	1.1	34-2	36-3*	0.7	

For each breed, the values were significantly different from control values: \* P < 0.05, \*\* P < 0.01, \*\*\* P < 0.001.

the initial dose of tick larvae and was greater in the British than in the AX calves. Ticks significantly increased faecal DM and decreased DM digestibility in both breeds (Table 3).

Urinary total N excretion and faecal N increased in all calves during tick infestation with a resultant significant decrease in N balance and apparent N digestibility (Table 4). Urea-N accounted for the major portion of the increased urinary N output (Table 4).

Ticks significantly increased urinary  $K^+$  and decreased urinary  $Na^+$  in British calves (Table 5). It is noteworthy that within breeds the sum of the positive ions  $(K^+ + Na^+)$  in

Table 5. The daily mean excretion of potassium and sodium ions during the period (days 18-28) in the metabolism cages and the bromsulphthalein (BSP) excretion on day 28 of control (CON) and tick-infested (TICK) calves

	Shorthorn × Hereford			Africander cross			
	CON	TICK	SED	CON	TICK	SED	
Urinary K <sup>+</sup> (mmol)	210.5	268.4*	15.5	341.7	338.9	11.9	
Urinary Na <sup>+</sup> (mmol)	241.2	189-3*	20.8	172.9	172-3	13-2	
Urinary Na++K+ (mmol)	451·7	457·7	4.1	514.5	512·9	1.8	
Urinary N <sup>+</sup> :K <sup>+</sup>	1.2	0·7*	0.16	0.5	0.5	0.06	
Faecal K <sup>+</sup> (mmol)	16.8	26.9**	1.6	34.9	48.6**	2.3	
Faecal Na <sup>+</sup> (mmol)	24.3	31.0*	2.1	29.7	31.6*	0.4	
BSP: Fractional clearance	0.082	0.225**	0.024	0.148	0.154	0.030	
Half-life (min)	8.57	3.21**	0.6	4.96	4.78	0.9	

(Each value represents a mean result for four calves. Comparisons were made between pair-fed animals and standard errors of differences (SED) are given (i.e. 3 df))

For each breed, the values were significantly different from control values: \* P < 0.05, \*\* P < 0.01.

Table 6. The rectal temperature, digestion of organic matter (OM) in the stomach and intestines, fractional turnover rate of rumen fluid, and urea metabolism in the plasma and in the gastrointestinal tract (GIT) in control and tick-infested Hereford steers

	Control		Tick-i	nfested	Statistical signficance of difference	
	Mean	SE	Mean	SE	between groups	
Body-wt (kg)	221	5	233	11	NS	
Rectal temperature (°)	39.0	0.1	39.8	0.5	*	
OM intake (g/d)	3734		3734			
OM digestibility in GIT	0.726	0.006	0.701	0.003	*	
OM leaving abomasum (g/d) <sup>†</sup>	1503	5	1707	24	*	
OM apparently digested in stomach $(g/d)^{\dagger}$	2232	5	2028	24	*	
Apparent OM digestibility in stomach (g/g OM intake)†	0.598	0.001	0.543	0.006	**	
Apparent OM digestibility in intestinal tract (g/g OM leaving abomasum)†	0-330	0.018	0.339	0-009	NS	
Fractional turnover rate of rumen fluid (h)	0.057	0.002	0.068	0.002	*	
Plasma urea concentration (mg N/l)	150	3	184	8	*	
Production rate of plasma urea (g N/d)	61-1	1.1	69.5	1.3	**	
Degradation of urea in GIT (g N/d)	32.0	1.9	27.2	2.0	NS	
Urinary area excretion (g N/d)	29.1	1.2	42·2	3.1	•	

(Mean values with their standard errors, n 3 unless other wise indicated)

† n 2. NS, not significant; • P < 0.05; \*\* P < 0.01.

the urine was similar in control and tick-infested animals. Faecal  $K^+$  and Na<sup>+</sup> excretion significantly increased in both breeds during tick infestation. The BSP clearance capacity was unaffected by ticks in the AX calves (Table 5). However, the BSP fractional clearance rate was significantly increased by ticks in British calves and was lower (P < 0.01) in British control than in AX animals.

The mean  $(\pm sE)$  body-weights (kg) at the end of the treatment periods were: British control group  $160\pm 8$ , British tick-infested group  $148\pm 4$ , AX control group  $191\pm 2$ , AX tick-infested group  $178\pm 7$ .

*Expt* 2. The results are presented in Table 6. The (mean  $\pm$  sE) daily number of mature ticks carried by calves of the tick group over the final 3 weeks of the experiment were  $188 \pm 21$ , the sE value is the between-animal variation with 2 df. Tick infestation increased (P < 0.05) rectal temperature and the mean daily water intake was higher (P < 0.05) in the tick-infested animals (15.9 l/d) than in the control animals (13.2 l/d).

OM digestibility in the gastrointestinal tract was lower (P < 0.05) in tick-infested steers than in control steers. The marker-based measurements show that, compared to controls, there was a 14% increase in the flow of OM from the abomasum with a corresponding decrease in the apparent digestibility of dietary OM in the stomach of infested steers. This lower OM digestibility was associated with a 17% increase in the fractional turnover rate of rumen fluid. On the other hand, tick infestation had no effect on the digestibility of OM entering the intestines. While production rate and plasma concentration of urea and urinary urea excretion were higher in tick-infested than in control steers the degradation of urea in the gastointestinal tract tended to be lower in the tick-infested animals.

#### DISCUSSION

The specific effect of ticks in British cattle is to cause a reduction in the concentration of plasma Fe, packed cell volume and red cell volume (O'Kelly *et al.* 1971; Springell *et al.* 1971). It could appear from the present results that breed differences in the effects of ticks on haematocrits were accounted for simply by differences in numbers of ticks removing blood. However, previous work from this laboratory presented evidence that the metabolic derangements in tick-infested British cattle may in part be due to the uptake of blood by the tick and in part due to toxin secreted by the tick (O'Kelly *et al.* 1971; Springell *et al.* 1971). Further, it is not known whether there are genetic differences in susceptibility to toxin produced by ticks or in the metabolism of such toxins. Therefore, it was not considered prudent in the present study to make quantitative comparisons of metabolic alterations between the breeds in terms of differences in tick numbers.

Alterations in the plasma lipid and protein composition are established features of the specific effect of ticks on host metabolism in British cattle (O'Kelly *et al.* 1971). In the present study, the measurements of these metabolites in the tick-infested AX calves (Tables 1 and 2) essentially recapitulated what has been described in the British animals. The plasma concentrations of cholesterol and phospholipid were significantly higher in AX than in British calves yet ticks depressed the concentrations of these components similarly in both breeds. While ticks remove protein from the blood of the host (Springell *et al.* 1971) the hypoalbuminaemia could be produced by altered metabolism as a result of either impaired absorption of nutrients or liver damage, since albumin is formed exclusively in this organ. The depressed N digestibility in tick-infested animals may implicate a reduced net uptake of nutrients for synthesis of this protein. On the other hand, an increased amion acid and energy requirement by other tissues for the synthesis of the increased amounts of circulating  $\gamma$ -globulin may be a factor diverting nutrients away from albumin and  $\gamma$ -globulin reflect a deranged protein metabolism which is limiting nutrient availability for muscle protein

synthesis it could partly explain the reduction in growth rate of tick-infested compared to pair-fed tick-free British steers reported by Seebeck *et al.* (1971).

The increased body temperatures, water intakes and urine volumes in both breeds due to ticks are physiological adjustments similar to those found during heat stress (O'Kelly, 1973). Unlike hyperthermia resulting from heat exposure, however, the increased body temperature in tick-infested calves was not associated with an increase in evaporative water loss. Nor was there any suggestion from the plasma concentrations of Na and K that the higher water turnover in tick-infested calves was associated with electrolyte imbalance. However, the tick-infested British calves showed an increased urinary excretion of K. This was not considered to be caused by an increased urine flow since the tick-infested AX calves showed a similar change in renal function and Scott (1969) found that for sheep receiving a fixed K intake the amount of K excreted was not affected by urine volume provided this was not very low. The increased content of sodium and potassium in the faeces of tick-infested calves of both breeds is similar to that reported for sheep infested with parasitic nematodes (Bawden, 1969) and was probably related to the increased amount of water excreted in the faeces as noted by Blaxter & Wood (1953).

There was a suggestion of liver damage in tick-infested calves from the reduced activities of serum amylase and alkaline phosphatase (Table 2). However, these enzymes are not synthesized exclusively in the liver and it is possible that their lowered activities in the serum resulted from the effect of ticks on other organs. We also used the clearance of BSP from the blood plasma as a measure of liver damage and loss of functional capacity. There was no impairment of BSP fractional clearance in the AX breed due to ticks and the observed values for control and tick-infested animals were similar to those reported by Hunt & McCosker (1967) for normal beef cattle. In contrast, the BSP clearance in British control calves was approximately 50% lower than in AX calves but this could be explained by differences in nutritional status, the British calves consuming less food. Further, the higher excretion of N in urine relative to intake in British control compared to AX control calves (55 v. 43% respectively) probably reflected a greater loss of labile protein as a result of inadequate energy and N intake (Blaxter & Wood, 1951). There is strong evidence that impaired intrahepatic conjugation of BSP is responsible for the decrease in the rate of dye excretion into bile when free BSP is administered intravenously into rats on a protein-free diet (Whelan et al. 1969). On the other hand, it is difficult to interpret the dramatically high plasma clearance of BSP in the tick-infested British calves, but it might be noted that significant amounts of the dye are lost in the urine of man in certain disease states (Ingelfinger et al. 1948).

By partitioning digestion between the stomach and intestines (Table 6), it is evident that the decrease in OM and DM digestibilities in the gastrointestinal tract of tick-infested animals was largely due to a decrease in OM digestibility in the rumen; the simultaneous increase in the fractional turnover rate of rumen fluid indicates that this result is at least partially attributable to increased rumen motility, with a consequent increase in the rate of propulsion of digesta from the abomasum.

The gastrointestinal disturbances in the digestion of DM in British and AX calves was accompanied by a reduction in N digestibility. However, increased urinary-N as well as faecal-N loss made significant contributions to the total negative N balance. Urea was the main component of the increased urinary-N excretion which coincided with a raised concentration of plasma urea in animals parasitized with ticks (present results) and internal parasites (Roseby, 1973; Parkins *et al.* 1973). The origin of the increased amounts of urea in the plasma has, however, not been previously investigated. The results from Expt 2 show that plasma urea production rate was related to concentration; therefore the increased urinary urea excretion and plasma urea concentration observed in tick-infested calves was probably caused by higher production rates of urea rather than changes in kidney function or in urea space. Despite the increased urea production rates in steers infested with ticks, urea degradation in the gastrointestinal tract was less than that for control steers and was perhaps associated with the depression in the amount of OM digested in the stomach of tick-infested steers (Kennedy, 1980). Since apparent N digestibility was lower in tick-infested animals, the higher urea production rate was probably due to catabolism of body protein.

This work was supported in part by the Australian Meat Research Committee; technical assistance by C. R. Holmes.

#### REFERENCES

Bawden, R. J. (1969). Aust. J. agric. Res. 20, 589.

- Blaxter, K. L. & Wood W. A. (1951). Br. J. Nutr. 5, 29.
- Blaxter, K. L. & Wood, W. A. (1953). Vet. Rec. 65, 889.
- Christian, K. R. & Coup, M. R. (1954). N. Z. J. Sci. Technol. 35A, 328.
- Downes, A. M. & McDonald, I. W. (1964). Br. J. Nutr. 18, 153.
- Faichney, G. J. (1975). In Digestion and Metabolism in the Ruminant, p. 277 [I. W. McDonald and A. C. I. Warner, editors]. Armidale, Australia: University of New England Publishing Unit.
- Henry R. J. & Chiamori, N. (1960). Clin. Chem. 6, 434. Hunt, S. E. & McCosker, P. J. (1967). Clinica chim. Acta 18, 133.
- Ingelfinger, F. J., Bradley, S. E., Mendeloff, A. I. & Kramer, P. (1948). Gastroenterology 11, 646.
- Kennedy, P. M. (1980). Br. J. Nutr. 43, 125.
- Megarrity, R. G. & Siebert, B. D. (1977). Analyst, Lond. 102, 95.
- Ness, A. T. & Dickerson, H. C. (1965). Clinica chim. Acta. 12, 579.
- O'Kelly, J. C. (1973). Br. J. Nutr. 30, 211.
- O'Kelly, J. C., Seebeck, R. M. & Springell, P. H. (1971). Aust. J. biol. Sci. 24, 381.
- O'Kelly, J. C. & Spiers, W. G. (1976). J. Parasitol. 62, 312.
- Parkins, J. J., Holmes, P. H. & Bremner, K. C. (1973). Res. vet. Sci. 14, 21.
- Roseby, F. B. (1973). Aust. J. agric. Res. 24, 947.
- Scott, D. (1969) Q. Jl exp. Physiol. 54, 16.
- Seebeck R. M., Springell, P. H. & O'Kelly, J. C. (1971). Aust. J. biol. Sci. 24, 373.
- Springell, P. H., O'Kelly, J. C. & Seebeck, R. M. (1971). Aust. J. biol. Sci. 24, 1033.
- Tan, T. N., Weston, R. H. & Hogan, J. P. (1971). Int. J. appl. Radiat. Isotop. 22, 301.
- Technicon Instruments Corp. (1963). Technicon Method Sheets, nos. N-1A, N-11A. Tarry Town, New York: Technicon Instruments Corp.
- Vercoe, J. E. & O'Kelly, J. C. (1972). Proc. Aust. Soc. Anim. Prod. 9, 356.
- Wells, M. G. (1969). Clinica chim. Acta. 25, 27.
- Whelan, G., Hoch, J. & Combes, B. (1969). Proc. Soc. exp. Biol. Med. 132, 704.