Effects of Mild Heat Exposure and Suppression of Prolactin Secretion on Gastro-intestinal Tract Function and Temperature Regulation in Sheep

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Abstract
The effects of mild heat exposure (30°C; 30% relative humidity), and of the suppression of prolactin secretion under such conditions, were studied in anoestrous ewes given daily 791 g dry matter of a pelleted mixture of lucerne hay and oat grain (3 : 2). 51Cr-EDTA, 109Ru-phenanthroline and lignin were used to determine mean retention times (MRT) in the gastro-intestinal (GI) tract by a continuous infusion–total sampling procedure.

Mild heat exposure reduced the digesta-free tissue weight of all GI tract segments distal to the omasum. Increases in the amounts of digesta in the stomach compartments were largely due to increases in water content, although the solids content of the abomasum (P < 0·10) and omasum also increased. The treatment caused an increase in water intake (P < 0·10), increased the MRT of 51Cr-EDTA in the reticulo-rumen (P < 0·05), and tended to increase the MRT of all three markers in the omasum and abomasum. However, MRT in the whole GI tract was unaffected because of a compensating decrease (P < 0·01) in digesta MRT in the distal large intestine.

Suppression of prolactin secretion with 2-bromo-α-ergocryptine impaired the ewes’ ability to maintain their body temperature under the warm conditions imposed. This was associated with a consistent reduction in the degree to which water intake and its transactions in the GI tract changed in response to mild heat exposure. The treatment also caused decreases in omasal MRT and increases in abomasal MRT. Increased plasma concentrations of somatostatin and gastrin were associated with reduced plasma concentrations of prolactin, and it was postulated that some of the effects of prolactin on the GI tract may be mediated via somatostatin.

Introduction
The growth of young grazing animals is usually greatest during spring and early to mid summer, a period during which pastures reach their highest availability and digestibility (Scott et al. 1976). From a review of the literature, Peters and Tucker (1978) concluded that prolactin concentrations in sera of sheep, cattle and goats were highest during the summer and lowest during the winter, corresponding to the seasonal pattern of temperature and photoperiod. Pelletier (1973) showed that increased photoperiod stimulates prolactin release, and mild heat exposure has been reported to increase prolactin concentrations (Wetteman and Tucker 1974). Extending natural day length to 16 h using fluorescent lighting has increased blood serum concentrations of prolactin in dairy cows as well as increasing feed intake and milk yield (Peters et al. 1981). Similarly, increased photoperiod has been associated with both increased prolactin levels and increased feed intake and growth rate in lambs (Schanbacher and Crouse 1980); suppression of prolactin secretion under these conditions reduced liveweight gain (Eisemann et al. 1984a). However, it is possible that prolactin...
concentrations were correlated with but did not cause the changes in intake and performance.

Nevertheless, Mainoya (1978) showed in rats that prolactin was involved in the hypertrophy of the gut that occurs during pregnancy and, especially, lactation. Thus, if prolactin similarly affects the gastro-intestinal (GI) tract of grazing animals in early summer, it may be involved in the production responses observed. This paper reports a study of some aspects of GI tract function and temperature regulation in sheep exposed to continuous lighting and a moderately elevated environmental temperature, with or without treatment with bromocryptine, a compound which suppresses pituitary prolactin secretion and plasma concentrations in sheep (Ravault et al. 1977; Hill et al. 1980), cattle (Akers et al. 1981) and goats (Hart 1973).

Materials and Methods

Animals and Diet

Three groups of anoestrous Corriedale ewes aged 5–6 years were used. Group 1, the untreated ewes, were the five control ewes described by Barry et al. (1985); groups 2 and 3, the treated ewes, each consisted of six similar ewes. The mean liveweights (± s.e.) for groups 1, 2 and 3 were, respectively, 38·4 ± 0·94, 42·3 ± 1·50 and 42·8 ± 2·09 kg. The ewes were shorn 57–58 days prior to slaughter. They were held in individual pens for 4 weeks and were then transferred to metabolism cages for the 4 weeks prior to slaughter. Group 1 was held in a room with normal windows but with continuous artificial lighting and maintained between 19 and 22°C; groups 2 and 3 were held from mid-September in a controlled-climate room with continuous artificial lighting and maintained at 22°C. The diet was a ground and pelleted mixture of 60% lucerne hay and 40% oat grain as described by Barry et al. (1985) and was given at 791 g dry matter per day from the day of shearing; during the 4 weeks prior to slaughter, it was presented continuously from a conveyor belt. There were no feed refusals. Voluntary water consumption, corrected for evaporation, was recorded daily. Total water consumption included that in the pellets eaten.

Experimental Methods

Measurements were made in group 1 in the first period (mid-August) and on three ewes from groups 2 and 3 in each of the second (mid-October) and third (early November) periods. In each period, 3 weeks before slaughter, each sheep was fitted with a self-retaining rumen catheter (Faichney and Colebrook 1979). In the second and third periods, 2 weeks before slaughter, the temperature in the climate room was raised to 30°C and the relative humidity lowered to 30%; these conditions were maintained until slaughter. For the 10 days before slaughter, the heated ewes were given a subcutaneous injection at 0900 and 2100 h of 1 ml of either a placebo solution (group 2) or a bromocryptine solution (1 mg/ml; group 3). 2-Bromo-α-ergocryptine (CB154; Sandoz Australia Pty. Ltd; 100 mg), together with 100 mg of tartaric acid, were dissolved in 40 ml of 70% (v/v) ethanol, made up to 100 ml with sterile saline (9 g NaCl/l) and stored in a light-proof bottle at 4°C. The placebo solution was identical, excluding only the bromocryptine. During each period, faecal output was collected for 7 days prior to slaughter and indigestible markers were infused into the rumen for at least 5 days; the infusions were maintained until the moment of slaughter. Urine output was collected during the 24 h prior to slaughter. Rectal temperature and respiration rate were recorded for each ewe at 0800 and 1100 h during the 5 days before slaughter; there were no differences between times so only the mean daily values are reported.

Infusion Procedure

The markers infused were the 51Cr complex of EDTA (51CrEDTA) (Downes and McDonald 1964) and 108Ru-labelled tris-(1,10-phenanthroline)-ruthenium(II)chloride(108Ru-phen) (Tan et al. 1971). The infusate was made up to contain 37 kBq 51Cr and 7·4 kBq 108Ru per millilitre, with 5·4 μg inert CrEDTA/kBq 51Cr added as carrier. Following a priming dose of 30 ml into the rumen, the infusion was begun and maintained at 47 ml/day for at least 5 days before slaughter.

Sampling Procedures

A composite sample of faeces was prepared for each sheep by combining 20% of the daily output; this and a urine sample were stored at −10°C. Blood samples were drawn into heparinized syringes by
jugular venipuncture of all animals at 10·00 h for three consecutive days; the blood was transferred to tubes kept in an ice slurry and 0·33 ml aliquots of plasma prepared by centrifugation were pooled each day to give 1 ml samples for individual hormone analyses. The proteinase inhibitor Trasylol (Bayer, Leverkusen, W. Germany) was added to those tubes (1000 k.i.u./ml plasma) that were to be assayed for somatostatin, glucagon and gastrin, to prevent proteolysis of these hormones. Blood samples were taken after the infusion of the indigestible markers had been running for 48, 72 and 96 h.

The animals were killed by the intravenous administration of concentrated sodium pentobarbitone, after which the abdomen was opened and the oesophagus, the omasal-abomasal junction and the abomasal–duodenal junction were quickly ligated and the entire GI tract removed. The small intestine was then divided into five approximately equal sections and the large intestine into the caecum–proximal colon (caecum), the cœntrietal (spiral–colon 1), centrifugal (spiral–colon 2) and terminal colon and the rectum. Total digesta in each segment was removed and weighed and subsamples taken for the determination of radioactivity, dry matter and lignin. Visible fat was then trimmed from all gut tissues, which were then washed, blotted dry and weighed. The reticulo-rumen will be referred to in this paper as the rumen.

**Analyses**

Samples of digesta, faeces and urine were assayed for $^{51}$Cr and $^{103}$Ru simultaneously in duplicate tared vials using a model 5320 Auto Gamma Spectrometer (Packard Instrument Co., Illinois). Hormones were determined by the standard radioimmunoassay procedures described by Barry et al. (1982). Total glucagon was measured using antibody K4023, which recognizes both pancreatic glucagon and enteroglucagon; pancreatic glucagon was also measured with a specific antibody and enteroglucagon estimated by difference. Gastrin was determined as described by Reynolds et al. (1984) and thyroid stimulating hormone as described by Hopkins et al. (1975). The interassay variation and assay detection limits for the hormone assays were: growth hormone 8·6%, 1·2 μg/l; prolactin 7·4%, 2·4 μg/l; somatostatin 14·6%, 7·0 ng/l; insulin 4·0%, 1·5 μl.u./l; glucagon 6·3%, 15·3 ng/l; gastrin 10%, 1·3 pmol/l; thyroid stimulating hormone 13·8%, 0·5 μg/l; thyroxine 6%, 2·0 nmol/l; tri-iodothyronine 6%, 0·04 nmol/l. Plasma glucose was determined using glucose oxidase (Trinder 1969) and plasma non-esterified fatty acids (NEFA) were determined as described by Patterson (1963). Feed samples were analysed as described by Faichney and White (1983). Lignin in digesta and faeces was determined as the organic matter remaining after extraction as described by Bailey (1967).

**Calculations**

Marker concentrations were expressed as a fraction of the infusion rate, the $^{51}$Cr-EDTA values being corrected for absorption as described earlier (Faichney 1980) assuming that $^{51}$Cr-EDTA absorption from any segment of the GI tract was proportional to its retention time in that segment (Faichney 1975b). Mean $^{51}$Cr urinary excretion was 5·28 ± s.e. 0·53% of that infused. Lignin concentrations were expressed as a fraction of the daily faecal output. Mean retention times (MRT) of the markers and of lignin in each segment of the GI tract were calculated using the continuous infusion-total sampling procedure (equation 7, Faichney 1975a). Because the markers do not behave independently in the small and large intestines (Faichney 1975b; Faichney and Boston 1983; Barry et al. 1985) the MRT values reported for the segments of the GI tract distal to the stomach are the mean of those determined for $^{51}$Cr-EDTA and $^{103}$Ru-phen.

Fractional outflow rates (FOR) of water from the rumen and abomasum were calculated as the reciprocal of the MRT for $^{51}$Cr-EDTA in these organs (Faichney 1975a) and, for the caecum, as the reciprocal of the mean of $^{51}$Cr-EDTA and $^{103}$Ru-phen MRT values. Water outflow from all three organs was calculated as the product of FOR and pool size. Water flow rate (l/d) through other organs was calculated as the reciprocal of marker concentration, using $^{51}$Cr-EDTA only for the omasum, and the mean of $^{51}$Cr-EDTA and $^{103}$Ru-phen for all segments distal to the stomach.

During the second period, the catheter became dislodged from the rumen of one of the ewes in group 3 so the MRT and flow data reported for this group are means for 5 ewes.

**Statistical Analysis**

The data were subjected to one way analysis of variance. The effects of bromocryptine injection were assessed by comparing groups 2 and 3. The effects of mild heat exposure were assessed by comparing groups 1 and 2 but, where the effect of bromocryptine injection was not significant, the value for group 1 was compared with the pooled value for groups 2 and 3.
Results

There were no differences between groups in overall digestion of the diet; apparent digestibilities of DM (± s.e.) were, respectively, 0.647 ± 0.008, 0.657 ± 0.005 and 0.641 ± 0.007 for groups 1, 2 and 3.

Mild heat exposure caused a fourfold increase in the respiration rate of the ewes but did not affect their rectal temperature (Table 1). Bromocryptine treatment significantly impaired the ewes' thermoregulatory ability, being responsible for a further significant increase in respiration rate and a significant rise in rectal temperature.

Table 1. Effect of mild heat exposure and bromocryptine injection on rectal temperature and respiration rate

In this and subsequent tables, bromocryptine injected at the rate of 1 mg CB154/12 h for each ewe.

Means for five untreated ewes and six ewes in each heat-exposed group

| Parameter                     | Untreated ewes | Heated ewes -CB154 | +CB154 | s.e. | Significance of effect |
|-------------------------------|----------------|--------------------|--------|------|------------------------|
| Environmental temperature (°C) | 19-22          | 30                 | 30     |      |                        |
| Rectal temperature (°C)       | 38.9           | 39.0               | 39.4   | 0.06 | P<0.01                 |
| Respiration rate (breaths/min) | 18.3           | 81.5               | 111.3  | 4.49 | P<0.01                 |

* A Pooled mean standard error if n = 5.

Table 2. Effect of mild heat exposure and bromocryptine injection on plasma concentrations of hormones and metabolites in sheep

Means for five ewes in the untreated group and six ewes in the heated groups

| Parameter                     | Untreated ewes | Heated ewes -CB154 | +CB154 | s.e. | Significance of effect |
|-------------------------------|----------------|--------------------|--------|------|------------------------|
| Growth hormone (μg/l)         | 5.8            | 4.0                | 5.6    | 1.07 |                        |
| Prolactin (μg/l)              | 162.4          | 133.9              | 13.8   | 36.08|                        |
| Somatostatin (ng/l)           | 9.5            | 12.2               | 27.1   | 3.35 |                        |
| Insulin (mi.u./l)             | 14.9           | 14.3               | 14.5   | 3.06 |                        |
| Pancreatic glucagon (ng/l)    | 103.5          | 145.0              | 129.7  | 14.34|                        |
| Enteroglucagon (ng/l)         | 291.1          | 288.0              | 340.1  | 47.31|                        |
| Gastrin (pmol/l)              | 13.3           | 10.9               | 13.9   | 1.27 |                        |
| Thyroid stimulating hormone (μg/l) | 1.74         | 2.32               | 2.00   | 0.453|                        |
| Thyroxine (nmol/l)            | 122.2          | 95.8               | 96.2   | 9.24 |                        |
| Tri-iodothyronine (nmol/l)    | 1.27           | 1.42               | 1.54   | 0.228|                        |
| T₃ : T₄                      | 0.010          | 0.015              | 0.016  | 0.0011|                        |
| Glucose (mg/l)                | 671.6          | 636.5              | 631.0  | 16.08|                        |
| NEFA (μmol/l)                 | 142.2          | 188.8              | 133.7  | 22.74|                        |

* A Pooled mean standard error if n = 5.
**Plasma Hormone and Metabolite Concentrations**

Mild heat exposure increased plasma concentrations of pancreatic glucagon and depressed those of thyroxine (Table 2); it also tended to increase plasma concentrations of non-esterified fatty acids (NEFA) and tri-iodothyronine, but these effects were not statistically significant \((P > 0.10)\). Bromocryptine treatment depressed plasma concentrations of prolactin and NEFA but increased somatostatin and gastrin concentrations; plasma concentrations of the other hormones measured were not affected.

**Table 3. Effect of mild heat exposure and bromocryptine injection on the weight (g) of digesta-free organs of the gastro-intestinal (GI) tract of sheep**

| Parameter | Untreated ewes | Heated ewes -CB154 | +CB154 | s.e. | Significance of effect |
|-----------|----------------|---------------------|--------|------|------------------------|
| Total GI tract | 1971 | 1853 | 1843 | 64.6 | Heat CB154 |
| Rumen + omasum | 895 | 926 | 885 | 29.6 | |
| Abomasum (A) | 202 | 192 | 181 | 18.3 | |
| Small intestine (SI) | 436 | 377 | 402 | 36.7 | |
| Caecum–proximal colon (CPC) | 188 | 151 | 162 | 9.9 | \(P < 0.05\) |
| Distal large intestine (DLI) | 250 | 207 | 212 | 15.5 | \(P < 0.10\) |
| A + SI + CPC + DLI | 1076 | 927 | 957 | 57.4 | \(P < 0.10\) |

\(^{A}\) Pooled mean standard error if \(n = 5\).

**Weight of GI Tract Tissues and Contents**

Bromocryptine treatment did not affect the weight of individual GI tract segments. However, mild heat exposure consistently decreased the weight of all segments distal to the omasum (Table 3), the reduction being particularly large for the caecum–proximal colon \((P < 0.05)\) and distal large intestine \((P < 0.10)\). Mild heat exposure tended to increase the amounts of digesta found in the rumen and omasum; it increased the digesta content of the abomasum and caecum but decreased that of the distal large intestine (Table 4). The pH of abomasal digesta was significantly reduced \((P < 0.05)\) by mild heat exposure; the mean values (± s.e.) for groups 1, 2 and 3 were, respectively, 3.06 ± 0.09, 2.52 ± 0.09 and 2.28 ± 0.05. Bromocryptine treatment decreased omasal digesta content \((P < 0.05)\) but tended to increase abomasal digesta.

**Marker Mean Retention Times**

Mild heat exposure had no effect on the MRT of any of the markers in the entire GI tract (Table 5). However, it increased the rumen MRT of \(^{51}\)Cr-EDTA and tended to increase the MRT's of all three markers in the abomasum \((P < 0.10)\) and omasum. MRT's in the small intestine and the caecum were not affected but were significantly decreased in the distal large intestine. Thus the treatment increased the proportion of the MRT spent in the stomach (rumen, omasum and abomasum) and decreased the proportion spent in the distal large intestine by a corresponding amount.
These changes were largest for $^{57}$Cr-EDTA ($P < 0.05$), intermediate for $^{103}$Ru-phen and smallest for lignin. Bromocryptine treatment tended to reduce the omasal MRT and increase the abomasal MRT of all three markers; it had no effect on MRT in other segments of the GI tract.

Table 4. Effect of mild heat exposure and bromocryptine injection on the amount of total digesta, dry matter and lignin in the GI tract of sheep

| Parameter                        | Untreated ewes | Heated ewes - CB154 + CB154 | s.e. | Significance of effect |
|----------------------------------|----------------|-------------------------------|------|------------------------|
| Total GI tract                   | 5820           | 6311                          | 6136 | 318·8                  | Heat CB154 |
| Rumen                            | 3847           | 4271                          | 4065 | 269·0                  | $P<0.05$  |
| Omasum                           | 165            | 197                           | 141  | 19·3                   | $P<0.10$  |
| Abomasum (A)                     | 295            | 415                           | 470  | 60·0                   | $P<0.05$  |
| Small intestine (SI)             | 553            | 493                           | 548  | 45·8                   | $P<0.05$  |
| Caecum–proximal colon (CPC)      | 630            | 684                           | 673  | 63·6                   | $P<0.05$  |
| Distal large intestine           | 330            | 246                           | 239  | 18·5                   | $P<0.01$  |
| A + SI + CPC                     | 1478           | 1592                          | 1691 | 32·1                   | $P<0.05$  |

**Dry matter**

| Parameter                        | Untreated ewes | Heated ewes - CB154 + CB154 | s.e. | Significance of effect |
|----------------------------------|----------------|-------------------------------|------|------------------------|
| Rumen                            | 668            | 652                           | 649  | 49·8                   | $P<0.01$  |
| Omasum                           | 39             | 45                            | 33   | 4·7                    | $P<0.05$  |
| Abomasum                         | 40             | 61                            | 67   | 9·5                    | $P<0.01$  |
| Caecum–proximal colon            | 113            | 115                           | 113  | 12·5                   | $P<0.05$  |

**Lignin**

| Parameter                        | Untreated ewes | Heated ewes - CB154 + CB154 | s.e. | Significance of effect |
|----------------------------------|----------------|-------------------------------|------|------------------------|
| Rumen                            | 167·0          | 163·0                         | 159·8| 13·3                   | $P<0.01$  |
| Omasum                           | 11·4           | 14·6                          | 10·3 | 1·7                    | $P<0.05$  |
| Abomasum                         | 10·4           | 18·8                          | 20·6 | 3·2                    | $P<0.05$  |

$^A$ Pooled mean standard error if $n=5$.

**Net Water Exchanges along the GI Tract**

Mild heat exposure increased water intake and decreased rumen water balance. There were small ($P > 0.10$) increases in rumen and abomasal pool sizes, decreases in outflow from the abomasum and decreases in apparent abomasal secretion and apparent absorption from the small intestine. Treatment with bromocryptine tended to reverse these effects, except for abomasal pool size which increased further.

**Discussion**

The results of this experiment show clearly that the injection of bromocryptine not only reduced prolactin secretion but also was associated with increased somatostatin and gastrin levels and with an impairment of the thermoregulatory ability of the sheep. Thus ewes exposed to moderately elevated temperatures responded by increasing respiration rate and were able to maintain their body temperature.
However, the body temperature of ewes treated with bromocryptine increased significantly despite the fact that they increased their respiration rates more than did the untreated ewes.

Table 5. Effect of mild heat exposure and bromocryptine injection on the mean retention times (h) of $^{51}$Cr-EDTA, $^{103}$Ru-phen and lignin in the GI tract of sheep

Means for five ewes in the untreated and +CB154 groups and six ewes in the –CB154 group

| Parameter           | Untreated ewes | Heated ewes | s.e. | Significance of effect |
|---------------------|----------------|-------------|------|------------------------|
|                     | −CB154 | +CB154      |      |                        |
| **51Cr-EDTA**       |         |             |      |                        |
| Total GI tract      | 34·0    | 34·3        | 32·8 | 1·74                   |
| Rumen (R)           | 11·3    | 13·4        | 12·1 | 0·88                   |
| Omasum (O)          | 0·7     | 0·8         | 0·6  | 0·09                   |
| Abomasum (A)        | 0·8     | 1·3         | 1·4  | 0·19                   |
| R + O + A           | 12·7    | 15·5        | 14·1 | 0·96                   |
| **103Ru-phen**      |         |             |      |                        |
| Total GI tract      | 44·0    | 44·3        | 43·5 | 2·49                   |
| Rumen (R)           | 18·5    | 19·7        | 18·9 | 1·54                   |
| Omasum (O)          | 2·3     | 2·7         | 2·0  | 0·32                   |
| Abomasum (A)        | 1·9     | 3·1         | 3·9  | 0·62                   |
| R + O + A           | 22·7    | 25·5        | 24·8 | 1·77                   |
| **Lignin**          |         |             |      |                        |
| Total GI tract      | 74·6    | 73·9        | 72·7 | 4·36                   |
| Rumen (R)           | 47·1    | 45·9        | 45·0 | 3·91                   |
| Omasum (O)          | 3·2     | 4·1         | 2·9  | 0·45                   |
| Abomasum (A)        | 3·0     | 5·2         | 6·2  | 0·80                   |
| R + O + A           | 53·3    | 55·2        | 54·0 | 4·21                   |

Mean retention times (mean of $^{51}$Cr-EDTA and $^{103}$Ru-phen)

- Small intestine: 2·9, 2·7, 2·8, 0·27
- Caecum–proximal colon: 9·8, 10·0, 9·7, 1·28
- Distal large intestine: 8·6, 6·1, 6·2, 0·57

A Pooled mean standard error if n = 5.

Mild Heat Exposure

The responses of the GI tract to mild heat exposure were generally small but showed a consistent pattern. Thus the digesta content of the stomach compartments and of the region from the abomasum to the caecum–proximal colon increased and that of the distal large intestine decreased. These changes were largely the result of changes in water content, although the solids content of the omasum and abomasum also increased, and were reflected in increased solute MRT in the stomach and decreased MRT in the distal large intestine. These findings are consistent with the known decrease in rumen motility after heat exposure (Christopherson 1985) and the reductions observed by Hales (1973) in blood flow to the rumen and small intestine, but not the large intestine, during mild heat exposure. There does not appear to be any information on the effect of temperature on the motility of the post-ruminal GI tract (Christopherson 1985) but the changes observed here suggest that heat exposure may
decrease motility in the abomasum and increase it in the distal large intestine. The reduction in MRT in the distal large intestine was similar in magnitude to the increase observed for the marker MRT's in the stomach. Barry et al. (1985) observed a similar effect and suggested that it could represent some compensatory mechanism within the GI tract which tended to keep total MRT constant.

Table 6. Effect of mild heat exposure and bromocryptine injection on the mean retention times of $^{51}$Cr-EDTA, $^{103}$Ru-phen and lignin as a percentage of the total mean retention times for each marker in sheep

Means for five ewes in the untreated and +CB154 groups and six ewes in the −CB154 group

| Parameter                | Untreated ewes | Heated ewes -CB154 | Heated ewes +CB154 | s.e. | Significance of effect |
|--------------------------|----------------|--------------------|--------------------|------|------------------------|
| Rumen (R)                | 33.2           | 39.0               | 36.8               | 1.97 | $P<0.05$               |
| Omasum (O)               | 2.2            | 2.5                | 2.0                | 0.32 | $P<0.10$               |
| Abomasum (A)             | 2.3            | 3.8                | 4.4                | 0.53 | $P<0.05$               |
| Small intestine (SI)     | 8.4            | 7.7                | 8.5                | 0.72 |                        |
| Caecum–proximal colon (CPC) | 28.7         | 29.1               | 29.3               | 2.92 |                        |
| Distal large intestine   | 25.3           | 17.9               | 19.0               | 1.66 | $P<0.01$               |
| R + O + A                | 37.7           | 45.3               | 43.2               | 2.36 | $P<0.05$               |
| A + SI + CPC             | 39.4           | 40.6               | 42.2               | 2.85 |                        |

$^{51}$Cr-EDTA

| Parameter                | Untreated ewes | Heated ewes -CB154 | Heated ewes +CB154 | s.e. | Significance of effect |
|--------------------------|----------------|--------------------|--------------------|------|------------------------|
| Rumen (R)                | 42.0           | 44.3               | 43.7               | 2.27 |                        |
| Omasum (O)               | 5.3            | 6.2                | 4.5                | 0.70 | $P<0.10$               |
| Abomasum (A)             | 4.3            | 6.9                | 8.9                | 1.10 | $P<0.05$               |
| Small intestine (SI)     | 6.5            | 6.0                | 6.5                | 0.64 |                        |
| Caecum–proximal colon (CPC) | 22.2         | 22.7               | 22.3               | 2.47 |                        |
| Distal large intestine   | 19.6           | 13.9               | 14.2               | 1.27 | $P<0.01$               |
| R + O + A                | 51.6           | 57.4               | 57.1               | 2.65 |                        |
| A + SI + C               | 33.0           | 35.7               | 37.6               | 2.69 |                        |

$^{103}$Ru-phen

| Parameter                | Untreated ewes | Heated ewes -CB154 | Heated ewes +CB154 | s.e. | Significance of effect |
|--------------------------|----------------|--------------------|--------------------|------|------------------------|
| Rumen (R)                | 62.9           | 61.9               | 61.4               | 2.42 | $P<0.10$               |
| Omasum (O)               | 4.3            | 5.6                | 4.0                | 0.71 | $P<0.05$               |
| Abomasum (A)             | 3.9            | 6.9                | 8.5                | 0.90 | $P<0.05$               |
| Small intestine (SI)     | 3.8            | 3.6                | 4.0                | 0.42 |                        |
| Caecum–proximal colon (CPC) | 13.4         | 13.6               | 13.7               | 1.89 |                        |
| Distal large intestine   | 11.7           | 8.3                | 8.5                | 0.84 | $P<0.05$               |
| R + O + A                | 71.1           | 74.4               | 73.8               | 2.32 |                        |
| A + SI + CPC             | 21.1           | 24.2               | 26.1               | 2.10 |                        |

A Pooled mean standard error if $n = 5$.

The reduced plasma thyroxine levels observed here in response to mild heat exposure are consistent with reports that thyroid hormone secretion declines in response to chronic heat exposure in rats (Purves 1964; Yousef 1976), pigs (Ingram and Ślebodziński 1965) and ruminants (Thompson 1973; Christopherson 1985). Yousef and Johnson (1985) suggested that the effect of temperature on thyroid activity is.
probably initiated at the hypothalamic level. However, in this experiment, thyroid stimulating hormone levels were not reduced; they were, if anything, slightly higher in the treated ewes. It may be that the conditions to which the ewes were exposed were not sufficient to affect the hypothalamus, and hence thyroxine secretion, but were sufficient to reduce blood flow to the thyroid gland (Hales 1973). A reduction in plasma thyroxine levels from such a cause might be expected to result in an increase in thyroid stimulating hormone levels and T₃ : T₄ ratios as a means of maintaining tissue levels of thyroid hormones; such trends were apparent here.

Table 7. Effect of mild heat exposure and bromocryptine injection on the net movement of water into and out of the organs of the GI tract of sheep

| Parameter                          | Untreated ewes | Heated ewes -CB154 | +CB154 | s.e.¹ | Significance of effects |
|------------------------------------|----------------|--------------------|--------|-------|------------------------|
| Rumen                              |                |                    |        |       |                        |
| Total intake (l/d)                 | 3·06           | 5·95               | 4·89   | 1·044 | P<0.10                 |
| Pool size (l)                      | 3·18           | 3·62               | 3·53   | 0·227 |                        |
| Outflow (l/d)                      | 6·79           | 6·59               | 7·06   | 0·407 |                        |
| Net balance (l/d)⁺                 | 3·73           | 0·64               | 2·17   | 1·137 |                        |
| Omasum                             |                |                    |        |       |                        |
| Outflow (l/d)                      | 4·20           | 4·49               | 4·09   | 0·260 |                        |
| Apparent absorption (l/d)         | 2·59           | 2·09               | 2·97   | 0·330 |                        |
| Abomasum                           |                |                    |        |       |                        |
| Pool size (l)                      | 0·26           | 0·36               | 0·43   | 0·048 | P<0.05                 |
| Outflow (l/d)                      | 8·24           | 6·85               | 7·33   | 0·861 |                        |
| Apparent endogenous secretion (l/d)| 4·04           | 2·36               | 3·24   | 0·789 |                        |
| Small intestine                    |                |                    |        |       |                        |
| Outflow (l/d)                      | 2·73           | 2·61               | 3·10   | 0·223 |                        |
| Apparent absorption (l/d)         | 5·51           | 4·24               | 4·23   | 0·870 |                        |
| Caecum–proximal colon              |                |                    |        |       |                        |
| Pool size (l)                      | 0·52           | 0·57               | 0·55   | 0·053 |                        |
| Outflow (l/d)                      | 1·31           | 1·40               | 1·36   | 0·081 |                        |
| Apparent absorption (l/d)         | 1·42           | 1·21               | 1·74   | 0·234 |                        |
| Distal large intestine             |                |                    |        |       |                        |
| Outflow (l/d)                      | 0·44           | 0·50               | 0·43   | 0·032 |                        |
| Apparent absorption (l/d)         | 0·86           | 0·90               | 0·93   | 0·094 |                        |

¹ Pooled mean standard error if n=5.
⁺ Net balance = saliva secretion + net influx by diffusion.

Heat exposure did not cause an increase in prolactin concentrations beyond that already achieved by continuous lighting. The observed concentrations were within the range (100–280 µg/l) that has been reported for lactating ewes (Barry 1980; Gow et al. 1983).

Suppression of Prolactin Secretion

The decrease in plasma prolactin concentration induced by the bromocryptine treatment was associated with an increase in plasma somatostatin concentration, confirming the inverse relationship observed between these hormones during
intravenous infusion of somatostatin (Barry et al. 1985). Plasma NEFA concentrations were also reduced, suggesting a decrease in lipolysis, a finding consistent with the decrease in glycerol release observed by Eisemann et al. (1984b) when adipose tissue from lambs treated with bromocryptine was incubated in vitro. It has also been reported that prolactin administration increased lipolysis in dogs (Winkler et al. 1971).

The bromocryptine treatment consistently reduced the degree to which water intake and many water transactions in the GI tract changed in response to heat. If these effects of bromocryptine are due to a reduced ability of ewes with low plasma prolactin concentrations to divert water from the GI tract, particularly the stomach, for evaporative cooling, they could explain the reduced ability of the ewes to maintain their body temperatures during heat exposure. Such an explanation would be consistent with the suggested role of prolactin in electrolyte and fluid balance through its effects on Na+/K+ ATPase activity (Falconer 1980; Nicoll 1981). The treatment also reduced the MRT of all markers in the omasum, and tended to increase the quantity of digesta and the MRT of all markers in the abomasum. These effects are in the same direction as those produced by intravenous somatostatin infusion (Barry et al. 1985), but were of smaller magnitude, perhaps because the increase (twofold) in somatostatin concentration was smaller than the sevenfold increase observed during somatostatin infusion. These findings offer indirect evidence that some of the effects of prolactin on gut function may be mediated via somatostatin, a suggestion supported by the associated rise in plasma gastrin concentration, which also increased during somatostatin infusion (Barry et al. 1985).

Measurement of Mean Retention Time

Warren et al. (1974) reported an increase in the MRT of a particle-adsorbed marker (144Ce) in the GI tract of cattle when the temperature was increased from 18 to 32°C. Although in the present work the rumen MRT of solutes was higher in the heated ewes, the total MRT did not differ from that in the control ewes because of the compensatory decrease in MRT in the distal large intestine. Faichney (1975b) noted increased MRT's in the abomasum and small intestine associated with decreases in both the rumen and large intestine and, during pregnancy, decreases in solute MRT in the rumen were compensated for by increases in the small intestine and caecum–proximal colon (G. J. Faichney and G. A. White, unpublished data). These results indicate not only that measurement of total MRT alone may miss significant changes occurring within the tract but also that techniques which estimate rumen and intestinal MRT's from the analysis of faecal marker excretion curves (Grosvum and Williams 1973; Ellis et al. 1979) could be misleading. Such methods are usually based on a simplified two-pool plus time-delay model and commonly assume that the pools represent the rumen and caecum–proximal colon. These approaches ignore retention in the abomasum (Faichney and Boston 1983) and lump together transit through the omasum and the small and distal large intestines, which can vary in opposite directions (Barry et al. 1985). If MRT is to be assessed adequately from faecal excretion patterns, more realistic models must be developed (Dhanoa et al. 1985; Faichney and Boston 1983; France et al. 1985; Faichney 1986).

Conclusion

Apart from its effects on lactogenesis (Falconer 1980; Akers et al. 1981), the functional role of prolactin in ruminants remains unclear (Fennessy and Suttie 1984;
Bauman and McCutcheon 1986). The results of this experiment suggest that the seasonal correlation between ambient temperature and plasma prolactin concentration may reflect a role for prolactin in thermoregulation, probably through its effect on fluid balance. They do not support the suggestion that there may be effects of prolactin on the GI tract which could be involved in the production responses observed in young ruminants in summer, except to the extent that they are involved in the animals' ability to adapt to the effects of heat exposure.

Acknowledgments

The authors thank Mr J. Rawlinson for skilled technical assistance, Miss G. Caughey for performing the digesta and faecal lignin analyses, Dr G. Reynolds for arranging the gastrin assays, Mr A. L. Wallace for the thyroid stimulating hormone assays, Mrs C. Redekopp for the remaining hormone assays and Ms S. Genge of Sandoz Australia Pty Ltd for the bromocryptine.

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Manuscript received 2 September 1985, accepted 3 February 1986
