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The Effect of of Biotin Deficiency in the Pre-Ruminant and Immediately Post-Ruminant Angora and Cashmere Kids

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Abstract: Responses of biotin in the pre-ruminant and immediately post-ruminant kids were studied in an experiment using 6Angora and 8 Scottish Cashmere male castrated kids. They were allocated into two equal groups and given a good quality biotin supplemented milk replacer up to 42 days of ages and then they allocated to continue to receive the biotin adequate diet or a diet without supplemented biotin and containing the biotin-binding protein avidin in dried egg white up to 84 days of ages. Thereafter all kids were continued to receive a progressively reducing quantities of the milk diet and in addition a commercial creep feed and chopped hay *ad libitum*. After weaning at 18 weeks of age, they received chopped hay (0.75) and concentrate (0.25) *ad libitum* to the end of the study at 24 weeks. Responses in live weight gain, dry matter intake, fibre characteristics, hair loss by combing, Plasma biotin and level of glucose, urea and PCV were measured through the experimental periods. The result indicated that at the end of the pre-ruminant period, biotin deficiency had a significant effect on reducing live weight gain ($p<0.05$), feed intake ($p<0.01$) and increasing combed hair loss ($p<0.001$). Total hair loss of mohair due to biotin deficiency was significantly ($p<0.01$) higher than cashmere. Following the introduction to solid feed there were no differences in animal performance due to diet. Total clean fibre yield by Angora goat in all periods was significantly ($p<0.001$) higher that for the Cashmere goats. While biotin supplemented milk replacer diet increased plasma biotin concentration this did not result in any significant increase in glucose, urea and PCV in all periods of study. However, during solid feeding Cashmere kids had a significantly higher live weight gain ($p<0.05$) and dry matter intake ($p<0.01$) than Angora kids. Difference between two genotypes was noted during soild feeding in superior values of feed intake(0.01), live weight gain ($p<0.01$), glucose concentration ($p<0.001$) and PCV% ($p<0.001$) in Chashmer than Angora goats.

Key words: Kids, biotin, ruminant, fibre, cashmere kids, PCV

INTRODUCTION

Biotin is sulphur containing B-vitamin group and well established as an essential co-factor for normal function of carboxylation enzymes which occupy an important position in metabolic pathway such as gluconeogensis, fatty acid synthesis and the catabolism of amino acids and regulation of carbohydrate metabolism (Mock, 1996). This vitamin is generally considered to provide the basic needs for the animal. Although biotin deficiency is responsible for some disorders in farm animals (Whitehead, 1988) and human (Hommes, 1986) but requirement the ruminant animals for supplemental biotin have not yet been established, may be due to the abundant synthesis of this vitamin in the ruminal and intestinal microflora.

Although biotin is widely distributed in feedstuffs, but its nutritional bioavailability may vary considerably between feedstuffs. The glucoprotein avidin is found in raw avian egg white and binds to biotin with extraordinarily high affinity. The avidin-biotin complex, is the strongest known noncovalent biological binding and it is exceptionally stable both to heat denaturation and to proteolytic digestion. Biotin deficiency can be induced in non-ruminants (Kopinski and Leibholz, 1989) and pre-ruminant animals (Hurstel, 1982) by adding sources of avidin, such as raw egg white or spray dried egg white, to their diets. No detailed analysis of the effect of biotin deficiency on the pre-ruminant goat has yet been reported.

The importance of nutrition and hormones in regulation of hair development and growth have been summarised by several authors (Galbraith, 1998; Bathes *et al*., 1997). Many vitamins and trace elements are essential for the process of fibre growth (Rise and Sahlu, 1994). Although there is no evidence to show vitamin

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deficiency directly restricts fibre growth in adult goats (Shelton, 1984) and sheep (Reis and Sahlu, 1994), there is some evidence to indicate that folic acid and pyridoxine are important for fibre growth for pre-ruminant animals due to their important role in the metabolism of S-containing amino acids (Chapman and Black, 1981). It is generally assumed that mature ruminants do not require supplementation of dietary B-vitamins as a consequence of microbial synthesis in the rumen (Poe *et al*., 1972). The aim of this experiment was to investigate the importance of biotin supply in goats with potential for fibre production, the effect of biotin deficiency on milk-fed pre-ruminant animals and the contribution of ruminal microbial biotin supply to growth, metabolism and fibre production following provision of solid feed to stimulate rumination.

MATERIALS AND METHODS

Fourteen kids (6 Angora and 8 Scottish Cashmere kids) were removed from their dam at one to three days after birth, after having had colostrum, in the time period of March and April. The mean initial weights at three days of age of Angora and Cashmere kids were 3.50 kg±0.45 and 2.90 kg±0.18, respectively. The kids were housed indoors at an ambient temperature. Angora kids were housed in two groups of three kids, Cashmere kids were housed in two groups of four kids. During the pre-experimental period kids were kept warm by electrical heater suspended over their pens. The kids were trained to bottle feeding on good quality biotin-adequate warm (37°C) milk replacer (Table 1, 125 g milk powder per litre of water) supplied for a preliminary period to 42 days of age. Milk was offered in clean bottles fitted with rubber teats four times a day (7.30, 11.30, 16.30 and 21.30 h) up to one month of age. When all the kids had been trained to bottle feed and were drinking well (approximately 4 weeks old), feeding frequency was reduced to 3 times daily (7.30, 13.30 and 19.30 h). Fresh water was supplied *ad libitum*. At the end of 6 weeks, the liveweight of the Angora and Cashmere kids was 8.2 kg±0.61 and 7.07 kg±0.55, respectively.

Angora and Cashmere kids were randomly allocated to one of two biotin treatment groups; group 1, biotin deficient, (-B) and group 2, biotin-supplemented (+B). Each group contained 3 Angora and 4 Cashmere kids. Animals were individually fed with *ad libitum* access to water. The feeding trial lasted for 18 and was split into 3 experimental periods. Period 1- milk replacer, (age 6-12 weeks), Period 2- milk replacer + creep feeding (age 13-18 weeks), Period 3- only solid feeding (age 19-24 weeks).

During period 1 a biotin deficient diet was produced by addition of lyophilised egg white to milk replacer unsupplemented with biotin (52.5 g egg white per kg of milk replacer). Based on manufactures information, milk replacer (Table 1) used in this experiment was mostly made up with the skimmed milk powder (Table 1) which have approximately 160 μg biotin kg^{-1}. It was calculated that the milk replacer diet would need to contain 25.2 g of egg white to inactivate all of its biotin in one kg. In this experiment, about double the amount of egg white (52.5 g egg white per kg milk replacer) was used to insure all the biotin was rendered inactive. The biotin-supplemented diet was prepared by addition of 100 μg biotin per kg of milk replacer. The kids were offered 210 mL reconstituted milk replacer per kg of liveweight, split in 3 feeds at 7.00, 13.30 and 19.30h as described above.

At 13 weeks of age, kids were introduced to creep feeding. The quantity of milk supplied (either biotin deficient or biotin-supplemented) was gradually reduced and replaced with hay plus creep feed (Table 2) in the ratio 0.75:0.25, based on dry matter. Hay was available *ad libitum* and consumption was measured daily. According to the manufacturer no biotin was added to the creep feed.

At 18 weeks of age, kids were offered hay and creep feed only. Hay was offered to appetite and creep feed concentrate was offered at 25% of the weight of hay dry matter consumed, calculated daily. The feed was provided twice daily at 8.30 and 16.30 h. Access to water was *ad libitum*.

Animals were weighed weekly on the same day throughout the experimental period (before morning feed).

Table 1: Composition of milk replacer

Ingredient	g kg^{-1}
Skimmed milk powder	200.0
Fat filled skimmed milk	700.0
Fat filled whey powder	50.0
Dextrose	25.0
Starch	20.0
Premix	5.0
Chemical composition	
Oil	240.0
Protein	250.0
Fibre	0.0
Ash	61.0
Calcium	9.1
Phosphorus	7.2
Magnesium	0.9
Salt	10.9
Lysine	19.0
Methionine	5.9
Methionine and cystine	8.8
Threonine	11.5
Selenium	0.2
Iodine	2.00
Iron	100.0
Zinc	50.0
Manganese	50.0
Cobalt	1.0
Vitamin A	30.000
Vitamin D3	7.500
Vitamin E	100

Table 2: Composition of creep feed

Ingredient	g kg^{-1}
Wheat	64.2
Wheat feed	120.0
Barley	225.0
Maize germ meal	75.0
Fish meal Herring (capelin)	50.0
Hi Pro Soya	71.0
Sunflower pellets	25.0
Palm Kernel (expelled)	25.0
Malt culms	100.0
Fat mixer	8.7
Molasses	70.0
Wheat dark grains	50.0
Sugar beet pulp	50.0
Salt	7.0
Limestone	19.7
Grass pellets	25.0
Lamb Vit.	0.25
Deccox	0.01 mg
Chemical composition	g kg^{-1}
Oil	30.0
Protein	180.0
Fibre	60.0
Ash	80.6
Calcium	7.5
Total phosphorus	5.5
Selenium	5.6 mg kg^{-1}
Copper	11.2 mg kg^{-1}
Vitamin A	12000 IU kg^{-1}
Vitamin D	2000 IU kg^{-1}
Vitamin E	20 IU kg^{-1}

using digital weigh scale. During the experimental period, milk, hay and concentrate intakes were measured daily and refusals were collected prior to morning feeding and recorded for individual kids. During period 3 (solid feeding) animals ate all of the concentrate offered within 10 min.

Hair was clipped from measured mid-side patch (8×8 cm) before the experiment commenced. Every 6 weeks. The hair harvested from the patchs were weighed immediately and washed in detergent (Teepol), water, ethanol and 2×solvent. A representative subsample weight of approximately 200 mg was taken from the Cashmere kids hair sample and the guard hair was separated from cashmere fibre. Diameter and length of fibre growth of the fibre samples were measured. Hair loss by combing was measured during period 1 after 4 weeks and again after 2 weeks and during periods 2 and 3 at 6 weekly intervals.

Blood samples were collected from all kids in the pre-treatment period at 6 weeks of age and subsequently two weekly intervals, between 11.30 and 12 am until the end of experiment. Blood was withdrawn by jugular venepuncture into 2×7 mL vacutainer tubes containing potassium oxalate and sodium fluoride anticoagulant using disposal needles. Plasma was harvested from the whole blood by centrifugation at 2500×g for 25 min. Plasma was frozen at-20°C prior to chemical analysis. Before chemical analysis, plasma samples were removed from the freezer and allowed thaw slowly at room temperature.

In order to determine differences in biotin status in the blood of the goat kids, plasma samples from these kids were assayed for biotin concentration using our ELLSA method. Parallel to microbiological assay for biotin. Glucose concentration in plasma was analysed colorimetrically by an enzymatic test based. Urea in plasma was measured by using a diagnostic kit (Sigma Chemical Co.). Packed Cell Volume (PCV) was measured by microhaematocrit in capillary tubes.

Data from this study were subjected to Analysis of Variance (ANOVA) by the general linear model using Minitab 13 for Windows. The statistical models used were as follow:

$$Y_{ijk} = \mu + \alpha_i + \beta_j + \alpha\beta_{ij} + \epsilon_{ijk}$$

where, Y_{ijk} is the observation on kth animal of ith breed (i is Angora or Cashmere goats) treated with jth level of biotin (j is biotin deficient or supplemented), μ is overall mean; α_i is the effect of breed of goat (Angora and Cashmere); β_j is the effect of treatment (biotin), $\alpha\beta_{ij}$ is interaction between breed and biotin and ϵ_{ijk} is the error term. In the parameter of liveweight gain, initial liveweight was tested as a covariate.

RESULTS

Towards the end of period 1, all Angora goats receiving the biotin deficient diet showed symptoms associated with the biotin-inadequacy e.g., hair loss around the ear and 2 of 4 Cashmere kids in this group showed dermatitis around the ear, in the corner of the mouth and around the nose Diarrhoea was observed in deficient animals specially in Angora kids during milk feeding. All 3 Angora kids in the biotin deficient group were treated for scouring on average for 6 days, whereas only 2 of 3 biotin-supplemented Angora kids were treated for an average 3.5 days. Scouring rate in Cashmere was less than Angora kids. Two of 4 Cashmere kids in the biotin-deficient group were treated for an average 2 days, whereas 1 of 4 Cashmere kids in the biotin-supplemented group was treated for scouring for 2 days. Cashmere kids in the biotin deficiency group showed a tendency to coprophagy (2 of 4 kids). This was minimised by frequently cleaning of faeces from the floor surface. These external symptoms of biotin deficiency had disappeared within two to three weeks of the introduction of solid (creep) feeding corresponding to the time of development of a functional rumen. The biotin-supplemented group did not develop any of the above symptoms and were

Table 3: Effects of biotin and breed on liveweight gain (g day^{-1}), feed intake (g kg^{-1} LW $^{0.75}$/day) and organic matter feed conversion efficiency of Angora and cashmere kids during different phase of life[1]

	Breed							
	Angora		Cashmere		Level of significance[2,3]			
Feeding regime	- B	+ B	- B	+B	*s.e.m*	B	b	B×b
Milk feeding (6-12 weeks)								
Liveweight gain (g d^{-1})	70.2	93.2	79.8	92.5	1.23	**	ns	ns
Dry matter (g kg^{-1} LW $^{0.75}$/d)	0.21	0.23	0.24	0.25	0.01	ns	ns	ns
Organic matter feed conversion efficiency	0.43	0.50	0.51	0.51	0.03	ns	ns	ns
Creep feeding (13-18 weeks)								
Liveweight gain (g d^{-1})	40.6	28.4	77.3	103	16.2	**	ns	ng
Dry matter (g kg^{-1} LW $^{0.75}$/d)	0.23	0.23	0.30	0.30	0.04	ns	*	ns
Organic matter feed conversion efficiency	0.18	0.16	0.29	0.42	0.05	ns	ns	ns
Solid feeding (19-24 weeks)								
Liveweight gain (g d^{-1})	39.7	49.6	78.9	95.3	11.6	ns	**	ns
Dry matter (g kg^{-1} LW $^{0.75}$/d)	0.33	0.35	0.49	0.50	0.04	ns	*	ns

[1] +B: Biotin supplemented ; –B: unsupplemented biotin, [2]B: Effect of biotin; b: Effct of breed, B×b: Intraction between biotin and breed, [3]*: $p<0.05$; ** $p<0.01$; ns: Non significant

apparently normal in every respect. However, these symptoms may arise directly or indirectly as a consequence of biotin deficiency and are consistent with other reports (Kopinski and Leibholz, 1989; Hurstel, 1982; Chapman and Black, 1981).

As shown in this Table 3, liveweight gains during milk feeding were significantly ($p<0.05$) lower in the biotin deficient group. During creep and solid feeding, differences in liveweight gain due to biotin status were not significant. There was no significant difference in liveweight gains between the 2 genotypes during milk feeding. However, Cashmere kids gained significantly ($p<0.05$) more than Angora kids during creep feeding and solid feeding regime.

The effect of supplementary dietary biotin on dry matter intake based on metabolic weight showed that the interaction between breed and biotin in all periods, was not significant ($p>0.05$). During creep and solid feeding, cashmere ate more than Angora kids and significant differences were observed between the two genotypes ($p<0.05$). consumed significantly ($p<0.05$) more than Angora kids. During solid feeding, Cashmere kids However, biotin status during milk feeding had no effect on subsequent dry matter intake during all periods of study, although when dry matter intake was express based on g day^{-1}, only during milk feeding, biotin supplemented group consumed significantly more than others ($p<0.01$).

There were no significant effects of biotin on the efficiency of conversion of organic matter into liveweight gain during the experimental periods ($p>0.05$). During creep feeding, the Cashmere kids on average had significantly ($p<0.01$) greater efficiencies than Angora kids (Table 3). The interaction between breed and biotin in all periods, was not significant ($p>0.05$).

Plasma biotin concentration of Angora and Cashmere kids fed milk replacer unsupplemented or supplemented with biotin, during different periods of study was measured either by microbiological or ELLSA method and results are presented in Table 4. Quantification of biotin in plasma was made on a logarithmic equation based on the standard curve produced in parallel with plasma samples. Results of microbiological assays for these samples also have been reported by Haffman-La Roche company. Biotin concentration in plasma of kids given biotin unsupplemented diet during milk feeding was significantly ($p<0.001$) lower than control groups. In contrast, when they started solid feeding, biotin concentration in plasma increased. There were no significant differences due to the breed or interaction between breed and treatment ($p>0.05$).

Glucose and urea concentrations in blood plasma, which were sampled between 11-12 PM before feeding, are summarised in Table 4. Although the mean values for glucose tended to be inconsistent, there were no significant differences in mean concentration of glucose due to biotin status. The overall mean values for glucose showed a consistent reduction over the experimental periods. During solid feeding glucose concentrations in Cashmere kids were significantly ($p<0.01$) higher than in Angora kids. The concentration of glucose in plasma of Cashmere kids before the start of the experiment was significantly higher than in Angora kids ($p<0.05$). There was no significant ($p>0.05$) effect of biotin on urea concentrations during the study. Angora kids showed higher urea concentrations than Cashmere kids during milk feeding ($p<0.001$) and creep feeding ($p<0.05$) (Table 4). The interaction between breed and biotin in all periods, was not significant ($p>0.05$).

Mean values for PCV% of kids during different times are shown in Table 4. While there was no significant effect of biotin on the PCV volume in plasma. The volume in plasma of Cashmere kids were significantly higher than Angora goat kids during periods of milk and creep feeding

Table 4: Effects of biotin and breed on blood metabolite of Angora and cashmere kids during different phase of life[1]

	Breed							
	Angora		Cashmere		Level of significance			
Feeding regime	-B	+B	-B	+B	*s.e.m*	B	b	B×b
Pre experiment								
Biotin (pg mL^{-1})	213	224	250	270	31.5	ns	ns	ns
Glucose (mg L^{-1})	1130	1175	1305	1300	90.4	ns	*	ns
Urea (mg L^{-1})	110	93	98	103	6.5	ns	ns	ns
PVC (%)	26.6	27.7	26.4	26.3	1.32	ns	ns	ns
Milk feeding (6-12 weeks)								
Biotin (pg mL^{-1})	176	334	162	312	35.9	**	ns	ns
Glucose (mg L^{-1})	806	854	817	859	74	ns	ns	ns
Urea (mg L^{-1})	147	149	128	102	10.8	ns	***	ns
PVC (%)	25.7	26.1	26.3	26.8	1.37	ns	ns	ns
Creep feeding (13-18 weeks)								
Biotin pg mL^{-1}								
(ELLSA assay)	321	401	297	399	67.6	ns	ns	ns
Glucose (mg L^{-1})	715	759	742	911	46.0	ns	*	ns
Urea (mg L^{-1})	138	134	108	94	16.5	ns	**	ns
PVC (%)	24.7	26.6	29.4	29.3	1.25	ns	**	ns
Solid feeding (19-24 weeks)								
Biotin (pg mL^{-1})	371	497	535	720	200.3	ns	ns	ns
Glucose (mg L^{-1})	573	618	743	750	35.0	ns	***	ns
Urea (mg L^{-1})	97	121	93	112	14.0	ns	ns	ns
PVC (%)	21.9	21.9	27.1	26.3	0.92	ns	***	ns

[1] For key to abbreviation see Table 3

Table 5: Effects of biotin and breed on total fibre production, fibre growth and diameters and hair combed loss of Angora and cashmere kids during different phase of life[1]

	Breed							
	Angora		Cashmere		Level of significance			
Feeding regime	- B	+ B	- B	+B	*s.e.m*	B	b	B×b
Milk feeding (6-12 weeks)								
Total clean fibre (g d^{-1})	145	138	53	54	10.5	ns	***	ns
Fibre growth (mm d^{-1}) (mg L^{-1})	0.96	0.98	0.37	0.45	0.042	ns	***	ns
Fibre diameter (im)	26.1	26.5	16.3	16.5	0.18	ns	***	ns
Combed hair loss (mm d^{-1})	158	49	2.50	7.80	4.92	***	***	***
	25.7	26.1	26.3	26.8	1.37	ns	ns	ns
Creep feeding (13-18 weeks)								
Total clean fibre (g d^{-1})	321	401	297	399	67.6	ns	ns	ns
Fibre growth (mm d^{-1}) (mg L^{-1})	NA	NA	NA	NA	NA	NA	NA	NA
Fibre diameter (im)	26.9	27.0	18.3	17.7	0.27	ns	***	ns
Combed hair loss (mm d^{-1})	146	33	11.1	18.8	13.8	**	***	**
Solid feeding 19-24 weeks)								
Total clean fibre (g d^{-1})	371	497	535	720	200.3	ns	ns	ns
Fibre growth (mm d^{-1}) (mg L^{-1})	0.89	0.98	0.37	0.45	0.042	ns	***	ns
Fibre diameter (µm)	26.8	27.0	18.3	17.7	0.27	ns	***	ns
Combed hair loss (mm d^{-1})	51	51	12.7	29.1	9.93	ns	*	ns

[1] For key to abbreviation see Table 3

($p<0.05$ and $p<0.001$, respectively). The interaction between breed and biotin in all periods was not significant ($p>0.05$).

The result for the clean total fibre production of the Cashmere (with and without guard hair) and Angora goats given different biotin treatments are presented in Table 5. Measurement of total fibre yield in each period showed that there was significant ($p<0.001$) difference in clean hair production due to breed (mg/day/100 cm^2) throughout the study but no differences were observed due to biotin status. The presence of dietary biotin had no effect on estimated total fibre production of both genotypes during experimental periods. Nor was there a significant interaction between breed and biotin in all periods ($p>0.05$).

Values for fibre growth and diameter are presented in Table 5. Fibre elongation and diameter during and diameter during the experimental period were significantly ($p<0.001$) higher in mohair compared to cashmere fibre. As indicated in Table 5, biotin status had no effect on fibre

growth and diameter in all periods of study ($p>0.05$). Interaction effects due to biotin and genotype, in all periods, were not significant ($p>0.05$).

The data for weight of combed hair loss were subjected to analysis based on two way analysis of variance with interaction model. The interaction between biotin and breed was significant during milk and creep feeding ($p<0.01$ and $p<0.001$, respectively).

As it shown in the Table 5, total loss of mohair fibre due to biotin deficiency was significantly ($p<0.001$) higher than for cashmere fibre during milk feeding and creep feeding. Interaction between diet and breed was significant ($p<0.001$) showing different breed had different response to treatments in that the response by Angora goats was greater than that for Cashmere kid. During solid feeding biotin status had no significant effect on combed hair loss, while difference between the two genotypes was significant ($p<0.05$; Table 5).

DISCUSSION

The present experiment using Angora and Cashmere kids, reports the response of two high potential fibre production genotype goats to biotin deficiency during pre-ruminant and following development of rumination in growth performance and fibre characteristics.

During the 12 weeks treatment period, using biotin-deficient milk replacer, biotin deficiency symptoms developed in both genotypes. The response in biotin deficiency was initially evident on feed intake, liveweight gain, horn formation and hair loss. The early onset of biotin deficiency symptoms in kids during this experiment presumably reflect the importance of biotin requirement for growth at this stage of life.

Low weight gain and feed intake values of kids receiving the biotin deficient milk replacer, compared to the biotin-supplemented group, are consistent with observation in a trial with pre-ruminant calves, where biotin deficiency reduced live weight gain after 105 days milk feeding compared to the control group (Hurstel, 1982) calves receiving normal milk replacer weighed an average 51 kg more than the calves on biotin-deficient diet (4% lyophilised egg white in milk replacer). In their experiment, average daily weight gain was significantly poorer in biotin deficient groups compared to the biotin-supplemented group. However, in our study, the greater liveweight gain in the biotin-supplemented group suggested that differences may be largely accounted by food intake. Dry matter intake (g day^{-1}) during milk feeding was significantly ($p<0.01$) higher in biotin-supplemented animals in both genotypes. Low feed intake and appetite during milk feeding in the biotin deficiency group may have been attributable partially to a higher rate of diarrhoea and alteration in digestive tract function in this group. Similarly, Hurstel (1982) noted that biotin deficiency increased diarrhoea in pre-ruminant calves. Diarrhoea is a common symptom of Leiner's disease which is caused by biotin deficiency in the human infant (Nisenson, 1957).

In the present experiment, when considering the metabolic body size ($LW^{0.75}$), biotin status did not affect mean values of dry matter intake over the duration of the study. Both groups therefore consumed similar amounts of food presented to them as a function of liveweight.

In the present study, during creep feeding (13-18 weeks of age), weaning from liquid to the solid diet resulted a reduction in liveweight gain in some kids in both breeds and treatments. However, after 2-3 weeks they adjusted to solid feed and began to gain weight. Although biotin status had no effect on liveweight gain during this period, Cashmere kids gained significantly ($p<0.05$) more than Angora goats, indicating the greater potential for gain in this breed. An alternative or additional explanation is that increased liveweight gain in Cashmere kids was due to higher feed intake (g kg^{-1} $LW^{0.75}$/day') during this period, although differences were not statistically significant ($p>0.05$).

The results of this study show that biotin status in milk feeding did not have a residual effect on dry matter intake during solid feeding, although significant differences between two genotypes were noted. Cashmere kids consumed and consequently gained more than Angora goats during solid feeding.

Liveweight gain during solid feeding (19-24 weeks of age) in control Angora and Cashmere kids (49.6±15.05 and 95.3±13.03 g day^{-1}, respectively) were of a similar order to values recorded for Angora kids (26- 52 g day^{-1}) (Souri, 1998) and Cashmere (39-60 g day^{-1}) (Souri, 1998) and indicated that both genotypes had a normal growth rate during solid feeding.

The absence of an effect of biotin status on organic matter conversion is not consistent with the work of Hurstel (1982) who observed a significant improvement of feed conversion in calves in response to feeding 500 μg biotin kg^{-1} milk replacer compared to biotin deficient group. Since significant differences in the efficiency of dry matter and organic matter conversion were observed between two genotypes during creep feeding, it is concluded that Angora goats were markedly more sensitive to the pattern of feed changes than Cashmere kids. Generally, goats are considered to have a greater digestive efficiency than sheep or cattle, because of longer retention time and slower passage of digesta and higher fermentation rates due to higher microorganism per mL of rumen liquid and therefore total VFA in the rumen

(Devandra and Burn, 1983). In support of the results obtained in the present study and in contrast to those cited above, it appears that Cashmere kids may have a greater propensity to partition nutrients for liveweight gain and away from fibre production compared with Angora goats. It seems also that Cashmere goats have less responsiveness for fibre production to improvement in nutrition supply at above maintenance levels of nutrition.

The influence of biotin status on some blood composition of the kids was studied. Significant differences in biotin concentrations in biotin deficient kids, during milk feeding, (176 and 162 pg mL^{-1} in Angora and Cashmere kids, respectively) were observed in both genotypes, compared to the biotin-supplemented groups (334 and 312 pg mL^{-1} in Angora and Cashmere kids, respectively). This indicated that the biotin deficient groups received less biotin as Whitehead (1988) stated that plasma biotin concentration in monogastric animals reflects dietary intake of this vitamin.

Following introducing to solid feed, plasma biotin concentration in biotin deficient groups of Angora and Cashmere kids gradually increased to approximately 2.1 and 3.3 times, respectively than the values during milk feeding. Review of literature indicated that biotin concentration in animal and human plasma shows extremely wide variation, depending upon the assay method used. Only limited data are available on plasma concentrations of goats. Similar concentrations to those report here for control kids (Angora and Cashmere kids) during solid feeding (an average 607 and 700 pg mL^{-1} with ELLSA and microbiological assay, respectively) have been reported by Straub and Frigg (1992; 513.6 pg mL^{-1}) for Cashmere goats.

There was a non-significant difference in glucose concentration due to biotin status during milk feeding in all experimental periods. Several reports have indicated that biotin deficiency may result in an impairment of glucose utilisation in animals such as rats (Furukawa *et al*., 1993) by its role in carbohydrate metabolism. Omission of biotin from artificial rumen systems has been shown to produced a marked decrease in production of volatile fatty acids and especially propionate (Milligan *et al*., 1967). It is known that, in ruminants, absorbed propionate may be responsible for 30-60% of glucose production.

Glucose concentration in new-born ruminant is substantially higher than those in adult ruminants and are similar to those of monogastric animals. However, glucose concentration before experiment (1175 and 1300 mg L^{-1} in Angora and Cashmere kids, respectively) was similar order to the mean values of 800-1200 mg L^{-1} reported for milk-fed goat kids by Swenson and Reece (1993). Glucose values gradually fell after kids were introduced to solid feed to reach values of 618 and 750 mg L^{-1} in Angora and Cashmere kids, respectively. These results are in agreement with Cronje′ (1992; 610 and 640 mg L^{-1} in Angora and Boer goats, respectively) and Souri (1998; 545 and 650 mg L^{-1} in Angora and Cashmere yearling goats, respectively). During solid feeding, concentrations of glucose in Angora goat were 17.5% less than Cashmere kids. It is postulated that the lower glucose concentrations of Angora goats, during this period, could be due to lower feed intake and or, due to impaired gluconeogensis and adrenal function of this breed, as described by Cronje′ (1992). In this respect, Cronje′ (1992) noted that, selection of Angora goats for only hair production has result in a shift in the partitioning of amino acids towards hair-protein synthesis and away from gluconeogenesis to the extent that animal may unable to mobilised sufficient protein reserves for glucose production. Other studies have also shown that glucose concentrations in Angora were lower than Cashmere goats (Souri, 1998).

The lack of effect of biotin status, during milk feeding, on plasma urea concentration in both genotypes indicated that plasma urea concentrations were not sensitive to biotin status in the diet. Although there are no other studies to evaluate the effect of biotin on the blood metabolite of goats, there is some evidence to show that biotin deficiency is responsible for some abnormalities in urea metabolism such as hyperammonia, in humans (Wolf and Raetz, 1983) and rat (Maeda *et al*., 1996) by mechanisms which are currently unclear.

The higher concentration of plasma urea in Angora, compared to Cashmere kids, during milk feeding and creep feeding could be due to higher rate of recycled urea of this breed (Cronje′, 1992). An alternative explanation for higher concentration of plasma urea in Angora goats during milk and creep feeding, could be due to higher rate of tissue protein breakdown, associated with low weight gain of this breed, especially during creep feeding. In contrast, lower plasma urea concentrations in Cashmere kids could be associated with greater liveweight gain of Cashmere kids during creep and solid feeding.

Packed Cell Volume (PCV) did not change consistently between treatments during the experimental periods. PCV is directly related to the erythrocyte concentration and haemoglobin content and it is known to increase due to factors such as dehydration and excitement (Swenson and Reece, 1993). Significantly higher PCV values in Cashmere kids than Angora goats during creep and solid feeding may contribute to the high demand for oxygen requirement of this breed possibly due to greater activity and or for higher liveweight gain of this breed.

Total fibre production per day in the patch samples collected in each period of study was not affected by biotin status. However, the difference between the two genotypes, for total fibre production, in all periods of the experiment was significant ($p<0.001$). Fibre production in Angora goats was significanty higher than Cashmere goats during the experiment. This data indicates that Angora goats may be the most efficient fibre producer and have been selected almost exclusively for mohair production. In the present study clean fibre production of Angora (7-11 g day^{-1}) and Cashmere kids (3-4.6 g day^{-1}) are consistent with those reported for well-fed Angora goats (10.1-16.7 g day^{-1}; Sahlu *et al.*, 1992) and for Cashmere kids (5.0 g day^{-1}; Souri, 1998). Down production (fibre without guard hair) in Cashmere kids in this experiment was in the range of 39-52% of total clean hair and is consistent with the results of Ryder (1987) who showed that in different groups of feral animals the percentage weight of down ranged between 6 to 20% in adult with values of over 30% in kids.

Biotin deficiency during milk feeding, significantly ($p<0.001$) increased weight of combed hair loss (mg day^{-1}) during milk and creep feeding particularly in Angora kids. Hair loss typically appeared on ears and around the nose and mouth of Angora kids and is characteristic of biotin deficiency whereas there was no apparent hair loss in Cashmere kids. However, some symptoms of dermatitis were observed around the nose and month in two of four Cashmere goats, indicating biotin deficiency. None of the biotin-supplemented kids showed such signs of dermatitis during any period of study.

Significant interactions between biotin and breed for combed hair loss indicated that different breed had different response to biotin deficiency. Evaluation of data in Table 5 shows that combed hair loss between the two treatments in the Cashmere kids was small and not significantly different. In contrast combed hair losses in Angora goats were significantly affected by treatment with apparent biotin deficiency produces a greater hair loss. Angora goats have been selected for fibre production and the results shown in Table 5 appear to suggest biotin deficiency. In other hand Angora goats have a higher mass of fibre compare to Cashmere goats and as a result a higher mass of hair was harvested in Angora compared to Cashmere kids (Table 5). An alternative explanation is that hair growth and moulting in Cashmere goats are seasonally dependent. Cashmere grows in the late summer and autumn and is moulted in late winter and spring. However, the milk feeding and creep feeding period in this experiment was conducted around spring and early summer, (during this time cashmere were shed), so biotin deficiency apparently had no affect on the combed hair loss in Cashmere kids. Indeed, hair loss and dermatitis in some species, are a common signal for biotin deficiency e.g. in humans (Innis and Allardyce, 1983), pigs (Kopinski and Leibholz 1989), calf (Hurstel, 1982) and mice (Mock and Mock, 1992).

Apparent hair loss in biotin-deficient Angora kids in this experiment is consistent with results of Chapman and Black (1981) in artificially reared lambs. They observed that during the course of administering the liquid diet, wool lacked crimp and in several instances were shed, even though body weights were increasing rapidly. In the lambs which had suffered alopecia, wool growth was restored by a diet with increased provision of B-group vitamins. However, in their study, crimp formation remained abnormal in all the lambs until shortly after they were weaned on to a roughage diet. These authors suggested that there might have been insufficient biotin in the milk diets to sustain wool growth in their rapidly growing lambs.

There are several possible explanations for hair loss to response of biotin deficiency in human and animals. For example, several authors suggested that hair loss, alopecia and skin rash in human due to biotin deficiency were related to abnormalities in metabolism of fatty acids (Mock, 1996). Human scalp hair contains constitutive lipids which represent 0.7-1.3% of the weight of hair (Wertz and Downing, 1988). Histology of human patient skins with biotin deficiency showed an absence of sebaceous glands and atrophic hair follicles (Proud *et al.*, 1990). It is known that the deficiency of biotin dependent enzyme, acetyl-CoA carboxylase, leads to impaired synthesis of malonyl-CoA which is critical for synthesis or metabolism of fatty acid especially long-chain polyunsaturated fatty acids Cooper *et al.* (1994) have suggested that tissues of biotin-deficient pigs may be more susceptible to lipid peroxidation, with possible adverse effects on animal health. Beemer *et al.* (1982) presented data showing an excessive hair loss, but no skin rash, in a human patient suffering biotin deficiency. These investigators speculated that alopecia was due to the accumulation of abnormal metabolites associated with the biotin dependent enzyme 3- methylcortonyl-CoA carboxylase (enzyme responsible for catabolism of leucine). An alternative explanation is that hair loss is the result of faulty hair retention that appears to be due to incomplete keratinization of hair shaft to the club as described by Ikeda in mice hair follicles. These authors reported that medulla and cortex of hair follicles were degenerated in the alopecia skin of germ-free mice fed the biotin-deficient diet. However, this interpretation does not explain why biotin deficiency inhibited viability of hair follicles, unless toxic intermediates accumulated or vital intermediates were depleted in a biotin-blocked system.

In the present experiment, it appeared that the effects of biotin deficiency were reversed following the introduction of kids to solid feed and development of

rumination. It may be concluded that the probable cause of increased biotin concentration in the digestive tract was due to microbial synthesis of biotin and or their present in feed. It is known that biotin can be synthesised by rumen and gastrointestinal microorganismes such as *Ruminococcus, Bacteroides succinogenes, Bacteroides ruminicola* and *Butyrivibrio* (Hungate, 1966). The amount of microbial vitamin synthesis may be related to the nature of diet, quantity of food ingested and availability of necessary precursors as suggested by Miller *et al.* (1986). Poe *et al.* (1972) have shown that creep-fed lambs exhibited higher ruminal B-vitamins concentration than milk-fed lambs at 28 days of age. Miller *et al.* (1986) reported that 0.1 to 1.3 mg biotin was synthesised daily by fermentation in the rumen of steers and 1.3 to 4.2 mg in other parts of gastrointestinal tract. Therefore, in the present study, it is possible to conclude that switching animal from pre-ruminant to ruminant state resulted in increased biotin synthesis and hence decreased biotin deficiency symptom.

The rate of fibre elongation, in both genotypes, did not respond to biotin treatment during experimental periods. The average of mohair and cashmere elongation of the biotin-supplemented groups were 0.98 and 0.45 mm day^{-1}, respectively and relate well with results from other studies with Angora ((0.67-1.00 mm day^{-1} (Devendra and Burns, 1983), 0.875 mm day^{-1} (Reis and Sahlu, 1994)) and Cashmere goats ((0.43-0.51 mm day^{-1}) The result of this experiment indicated that mohair growth (mm day^{-1}) during solid feeding was approximately 50% faster than for cashmere and the differences were significant ($p<0.001$).

Biotin status during milk feeding did not effect fibre diameter in both genotypes during all periods. The average mohair diameter of biotin-supplemented group was 27 μm. These values are in agreement with values published by Allain (1993, 22-45 μm) and Souri (1998, 27.6 μm). However, the present data suggest that mohair diameter increased with age from 26.7 to 28.1 μm throughout the experiment. The overall mean values of cashmere fibre diameter of kids in both treatments were 17.4 μm. Allain (1993) reported similar results for cashmere fibre (12-18 μm). However, similar to mohair fibre, average cashmere fibre diameter increased with age from 16.6 μm during milk feeding to 18.2 μm at the end of solid feeding.

CONCLUSION

The present study has confirmed that biotin is essential for health and performance of pre-ruminant animals. Biotin deficiency symptoms such as combeded hair loss, abnormality of head horn development and dermatitis occurred in kids 6-10 week after feeding milk replacer containing egg white. These results were confirmed following evaluation of biotin concentration in kid plasma in the present experiment. Biotin concentration in plasma of deficient kids was significantly lower than in the biotin-supplemented group. However, after introduction to solid feeding most of symptoms which may related to biotin deficiency disappeared. This may be due to microbial synthesis of biotin in the rumen or elsewhere in the digestive tract.

The results of this experiment show a clear effect of biotin deficiency and confirm the importance of the dietary supply of biotin as a constituent in the micronutrient supplementation of artificially reared goat kids. A future study could usefully investigate the biochemical role of biotin in animal health, hair viability and keratin synthesis.

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