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EFFECTS OF DIETARY CRUDE PROTEIN LEVEL ON THE DIGESTIBILITY OF NUTRIENTS, EXCRETION OF FECAL N AND URINARY N-FRACTIONS AND THE KINETICS OF ^{15}N LABELED UREA IN GROWING MALE GOAT KIDS

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ABSTRACT: A feeding trial was conducted with 16 male Saanen kids (4 kids/treatment) weighing 19.0 ± 1.8 kg in the middle of which they were subject to a metabolism trial and kept in cages for 14 days for a quantitative 10 days collection of excreta. Kids were fed a constant amount of the four diets formulated out of molassessed wheat straw [55 g crude protein (CP) per kg dry matter (DM)] and pelleted concentrates having 87, 117, 144 and 176 g CP/kg DM. They were injected about 100 mg of ^{15}N-labeled urea into the jugular vein at the beginning of the collection period. Nutrient digestibility, fecal N and urinary N-fractions, N and ^{15}N balances were measured; irreversible loss (IL) of urea from the body urea pool (BUP) and the kinetics of N in the body calculated. Increasing dietary CP increased digestibility of organic matter (OM) from 71% to 80% and of crude fiber (CF) from 28% to 58%; excretion of fecal N by 56%; of urea N by 93%; proportion of N of urea, allantoin, creatinine, uric acid plus hypoxanthine in urinary N from 31% to 66%, 12% to 22%, 3% to 12%, and by 1.7%, respectively; irreversible iron (IL) of N from 1.98 to 12.24 g/d; transfer of urea to gastro-intestinal tract (GIT) from 1.58 to 6.69 g/d (*i.e.*, equivalent to 80% to 55% of IL); recycling of the degraded urea N to metabolic pool from 1.22 to 6.12 g/d; but it has decreased the proportion of retained N of BUP origin from 38 to 30% and excretion of urinary non-urea N from source other than BUP was constant. Excretion of urea is the mechanism by which goat kids adapt to variable supply of dietary protein. Excretion of fecal N was more closely related to DM intake, while that of urea N to dietary CP level and of purine derivatives (PD) to digestible OM intake.

Key words/phrases: Goat kids, ^{15}N kinetics, N-metabolism, protein, urea

INTRODUCTION

In order to evaluate protein utilization it is important to know how much of the nitrogen consumed is retained in the body as well as its rate of

elimination. Urea is quantitatively the most important end product of N metabolism in ruminants, and it is not only a simple waste product of N-metabolism but also an important precursor of protein biosynthesis through the circulation of urea into the gastro-intestinal tract (GIT) (Houpt, 1959; Boda, 1980).

A portion of the synthesized urea enters the GIT where it is hydrolyzed to NH_3, which can then be either reabsorbed or used as N source for microbial protein synthesis which can be digested and yield amino acids to the animal. Ruminants conserve N when dietary intakes are low by utilizing endogenous urea (Houpt, 1970). Thus any increase in urea excretion by ruminants would mean loss of potential protein and reduced efficiency of N-utilization.

Urea recycling into the rumen should be of greatest consequence when protein intake limits growth, because protein intake and recycling urea into the rumen are inversely related (Kennedy and Milligan, 1980). Net input of body urea N into the rumen cannot be adequately predicted without tracer methods. Urea production rate and degradation in sheep have been investigated using isotope labeled urea (Nolan and Leng, 1972). Earlier different balance trials, tracer studies and total body analysis were conducted by different researchers to determine the fate of different N-compounds in N metabolism and their influence on N utilization in ruminants (Petri, 1978; Schlieper, 1991; Bornemann, 1995; Tegene Negesse *et al.*, 2001). In contrast to sheep for which a large amount of data on N metabolism and rate of excretion of N are available, corresponding information on goats is more limited. Moreover information that integrates the extent of body urea degradation in the rumen, microbial protein synthesis and utilization with the dynamic state of N metabolism in growing goats is sparse. The objective of this experiment was to assess if the level of dietary crude protein has an effect on nutrient digestibility, excretion of fecal N and urinary N-fractions, the kinetics of urea and irreversible loss of N from the BUP in growing male goat kids. To this effect data were generated from a metabolism trial involving a tracer study using ^{15}N - labeled urea on male Saanen kids.

Abbreviation: BUP, body urea pool; CF, crude fiber; CP, crude protein; DM, dry matter; DOM, digestible organic matter; GIT, gastro-intestinal tract; IDOM, indigestible organic matter; IL, irreversible loss; MP, metabolic pool; N, nitrogen; ^{15}N, stable isotope of nitrogen; OM, organic matter; PD, purine derivatives.

MATERIALS AND METHODS

Formulation of the rations

Four concentrates were formulated. They were the source of variation in CP supply to the kids and the difference in their CP content was realized by alternate variation in the proportion of potato starch and soy protein. Ration 1 and ration 4 had the lowest and the highest CP concentration, respectively. The composition and nutrient contents of the concentrates and of the straw-molasses mixture are shown in Table 1.

Table 1. The proportion of the feedstuffs in the concentrates and nutrient content of the concentrates and straw-molasses mixture.

	Concentrate				Straw + Molasses
	1	2	3	4	
Ingredients (g/kg)					
Potato starch	480	439	398	357	
Dried beet pulp	456	456	456	456	
Soy protein concentrate	35	76	117	158	
Dicalciumphosphate	15	15	15	15	
Limestone	4	4	4	4	
Premix (mineral & vitamin)	10	10	10	10	
Nutrient contents (% DM basis)					
Crude protein	8.7	11.7	14.4	17.6	5.5
Crude fiber	7.6	7.9	7.9	7.8	41.5
Ether extract	0.6	0.7	1.7	1.7	1.0
Ash	5.7	5.9	6.1	6.4	7.9
Ca	1.1	1.2	1.2	1.2	
P	0.4	0.5	0.5	0.6	

Animal management and feeding

The experiment was conducted in the Institute of Animal Nutrition of Bonn University, in Bonn, Germany. The kids used in this experiment were on a feeding trial (4 kids/treatment) with the same treatment diets used in this experiment. When they attained 19.0 ± 1.98 kg of body weight they were transferred to metabolic cages. To orient the kids with the metabolic cages they were put in the cages for about 2 hours daily for 3 days before the metabolism trial began. They were then put in the cages for 14 days for a quantitative 10 days collection of feces and urine. Rations 1, 2, 3 and 4 were fed to kids of groups 1, 2, 3 and 4, respectively. Molassesed straw was fed to all groups. Each animal was offered a constant amount of feed that was 90% of its feed intake a week ahead of the beginning of the metabolism trial. Concentrate to straw ratio was 5:1. Feed was offered in 5 portions at about 8:00, 10:00, 12:00, 15:00 and 17:00h. Kids received water ad libitum.

Feed-stuffs

Wheat straw and molasses (10:1) were mixed daily and were fed as such. There were only four cages and therefore the metabolism trial was repeated four times. The trial had thus four periods of 14 days. Samples of straw were pooled for each period as they were common to all treatments but the pellets were sampled for each kid and used in the calculation of digestibility of nutrients. The feed-stuffs were then ground in a centrifugal mill using a 1 mm sieve. After thorough mixing representative samples were taken and put in plastic beakers with lids until they were chemically analyzed.

Preparation of urea solution for injection and its administration

The kids were injected with the urea solution into the jugular vein at the first day of the collection period of the metabolism trial. It was planned to inject into the left jugular vein of each kid about 15 ml of the urea solution produced by dissolving 100 mg of urea [$(^{15}NH_2)_2CO$ with 99 atom-% ^{15}N-excess] in 15 ml of physiological saline which thus contains 48 mg of ^{15}N. The injection was done with a syringe and a canula that was attached to a rubber tube with a closet. The hair on the neck around the jugular vein of the kids was shaved with a razor blade to facilitate disinfection and injection. The jugular vein was first punctured with the canula that was attached to the 15 cm long rubber tube. When the canula was in the vein, as determined with the flow of blood into the transparent rubber tube, the syringe that contained the urea solution was attached to the rubber tube and was injected slowly and uniformly through the rubber tube. In order to insure that all the urea solution entered the blood system, a syringe that was filled with physiological saline was attached to the rubber tube and the urea solution that remained in the rubber tube was washed and further injected. The amount of urea solution *vis-a-vis* ^{15}N injected was calculated by difference in weight of the syringe with the urea solution before injection and its weight after injection.

Feces and urine collection

The floor of the metabolic cage was made of sheet metal and had 0.5 m x 1.1 m floor space with square holes of 1.7 cm. The cage was standing on four angle irons, which were attached to the edges of the cage. The sides of the cages were made out of non-transparent hard plastic sheets. On the front of the cage there were two boxes, for presenting feed and water. The boxes had small windows. Flexible pieces of rubbers were attached on either side of the windows, to prevent contamination of the feed and water with urine and feces. Under the sheet metal was a slanting hard plastic sheet over which the urine flowed. In order to prevent the loss of moisture and nitrogen, the fecal sample collected during the day time was

transferred to an airtight bucket at about 16:00 h and was put in a refrigerator. It was then mixed with the fecal samples collected overnight and weighed together in the morning and stored at - 18° C. At the end of the collection period the fecal samples were thawed at room temperature. After thorough mixing a representative sample was taken. Dry matter was determined immediately from the representative sample. The rest of it was freeze-dried and ground in a centrifugal mill through 1 mm sieve. From the ground material nutrients including ^{15}N were analyzed.

To the urine collection bottle 30 ml of 25% H_2SO_4 was added to block the action of bacteria and thereby prevent loss of ammonia. The sloping hard plastic sheet of the cage over which the urine flowed was automatically sprayed with distilled water at an interval of 2 to 3 h. The pH of the urine-water-acid mixture was constantly monitored with litmus paper to be under 3. Urine was collected twice daily at 8:00 h and 17:00 h, filtered with a sieve and weighed in the morning. After thorough mixing with a powerful syringe a representative aliquot of 1.5% was taken and mixed with the respective sample pool of each kid. Urine samples were kept in plastic bottles with screw caps and stored at -18° C.

Nutrient analyses

Dry matter, ash, crude fiber and lipids in feed-stuffs and feces were analyzed according to VDLUFA (1976). Nitrogen was determined by DUMAS with the oxidizing equipment FP 128 of the company Leco (Foss Heraeus Macro N, York, North Yorkshire, UK). CP content was then calculated by multiplying with the factor 6.25. Total N in urine was measured using the principles of Kjeldhal.

Analysis of urea, purine derivatives and creatinine

Urea in urine was measured with the Urease-GLDH-Method (Talke and Shubert, 1965) in the laboratory of the Society of Medical Doctors of Bonn (Poppelsdorfer Allee 65, 53115 Bonn). Purine derivatives and creatinine were determined using reversed phase high performance liquid chromatography (HPLC) as of Resines *et al.* (1992) in the Institute of Food Science Technology of Bonn University.

^{15}N analysis

After intravenous injection of ^{15}N-labeled urea, the cumulative excretion of the isotope in urine as well as in feces was recorded during the 10 days collection period of the metabolism trial. ^{15}N in the feces of one kid from group 4 was not analyzed. Therefore the calculations of ^{15}N-balance, irreversible loss and all other associated parameters in the kinetics of urea of group 4 were done only out of 3 animals (Table 4 and Fig. 1).

For the determination of ^{15}N in the urea of urine, the urine was first treated with Urease - S that dissociates urea into ammonium and CO_2 as described by Bornemann (1995). The ammonium was distillated using the boratbuffer method described by Rodehutscord (1992). For this distillation 1% boric acid instead of the customarily used 3% boric acid was used. For the determination of ^{15}N in feces and urine samples, a distillate was collected according to the principles of Kjeldhal. ^{15}N excess was measured from the distillates using emission-spectrometer (FAN, NOI-6PC). Nitrogen that exists as NH_4CL reacts with Na-hypobromide and is converted to molecular N_2 that is then excited at high frequency to emit light. This light was split into its spectrum in a special monochromator and projected through an outlet. The peaks of $^{14}N_2$, $^{14}N^{15}N$, $^{15}N_2$ were then determined and the ratio of ^{15}N to total N (in atom-% ^{15}N) was calculated with a computer. For the calculation of ^{15}N - excess, the corresponding natural enrichment was subtracted from the absolute value measured.

Calculation of kinetics of urea

The question of effects of a variable supply of N on irreversible loss of urea from the BUP was handled with the help of the stable isotope ^{15}N. It was therefore planned to work out balances by quantitative recording of N and ^{15}N excreted in urine and feces after marking the BUP by a single intravenous injection of ^{15}N labeled urea into the blood of kids.

Parameters were calculated as follows:

$$\text{Irreversible Loss (g N/d)} = \frac{\text{urinary urea N (g/d)}}{\text{Recovery of } ^{15}\text{N in urinary urea (\%)}} \times 100$$

Multiplication of irreversible loss of N by percent ^{15}N recovered in feces, urinary urea and non-urea-N gives the corresponding amount of N leaving the BUP via feces, urinary urea and urinary non-urea-N compounds.

Net N degraded in the GIT (g N/d) = Irreversible Loss (N/d) - urinary urea (g N/d)

N recycled to MP from urea degraded in GIT = urea N degraded in GIT - fecal N from BUP.

The difference between total injected dose of ^{15}N into the BUP and ^{15}N recovered in feces and urine gives what is retained in the body. Multiplication of the percentage of ^{15}N retained in the body by irreversible loss gives the portion of N from the BUP that is retained in the body.

Statistical analysis

One-way analysis of variance was followed in variance analysis of variables (SAS, 1985). Differences between least-square means were examined using the Scheffe' Test. Means were considered to be significantly different at $P < 0.05$. Correlations and regression analyses were done with SPSS (1988).

RESULTS

Organic matter and nutrient digestibility

As shown in Table 2 the digestibility of OM, CF and lipid tend to increase with increasing CP level in the ration. However, only ration 1 had significantly lower digestibility of OM than the rest of the rations, with no significant differences among rations 2, 3 and 4. The digestibility of CF in ration 1 was significantly lower than that of rations 3 and 4. The rations showed no significant difference in their lipid digestibility.

Table 2. Digestibility of organic matter, lipid and crude fiber of the rations fed to kids (mean ± SD, n = 4).

		Ration 1	Ration 2	Ration 3	Ration 4
Body weight	[kg]	17.3 ± 0.9	20.0 ± 2.2	19.1 ± 1.4	19.8 ± 1.7
N intake	[g/d]	4.79^{a} ± 0.5	9.21^{b} ± 1.0	11.5^{b} ± 0.8	13.9^{c} ± 1.9
DM intake	[g/d]	364^{a} ± 41	538^{b} ± 76	551^{b} ± 51	568^{b} ± 69
Digestibility	[%]				
Organic matter		70.9^{a} ± 3.87	76.9^{b} ± 1.44	80.3^{b} ± 1.80	79.3^{b} ± 0.68
Lipid		38.6 ± 11.1	44.4 ± 23.1	41.2 ± 12.1	40.3 ± 12.3
Crude fiber		28.1^{a} ± 14.3	44.0^{ab} ± 5.8	58.4^{b} ± 5.0	56.2^{b} ± 1.8

[a, b] Means within a row with the same or without any superscript at all are not significantly different ($P < 0.05$).

Nitrogen excretion

A tendency of increment in daily urinary excretion of N and N-fractions occurred with increasing N-intake (Table 3). These increments were distinct at higher dietary CP level. Kids fed rations 1 and 2 excreted significantly lower urinary N than those fed rations 3 and 4 but differences were not significant between kids fed rations 1 and 2; and 3 and 4. Kids fed rations 1 and 2 had excreted daily larger amounts of non-urea-N than urea-N but the reverse happened in kids fed rations 3 and 4. The ratio of non-urea-N to urea-N decreased (0.70:0.34) but urea-N to non-urea-N increased (0.30:0.66) with dietary CP. Significantly larger amounts of urea N (13 fold) was excreted by kids fed ration 4 than ration 1 which shows that excretion of urea was strongly affected by dietary CP level.

Table 3. Excretion of urinary-N, urinary-N-fractions and fecal N of the kids (g/head and day, mean ± SD, n = 4) and their relation to daily N intake (%, mean ± SD, n=4).

		Ration 1	Ration 2	Ration 3	Ration 4
N intake	[g N/d]	$4.79^{a} \pm 0.50$	$9.21^{b} \pm 1.03$	$11.5^{b} \pm 0.79$	$14.3^{c} \pm 1.77$
Urinary :	[g N/d]	$1.34^{a} \pm 0.16$	$2.73^{a} \pm 0.35$	$4.86^{b} \pm 0.74$	$7.76^{c} \pm 1.26$
Urea	[g N/d]	$0.40^{a} \pm 0.06$	$1.16^{a} \pm 0.55$	$2.84^{b} \pm 0.64$	$5.15^{c} \pm 0.85$
	[% of N intake]	$8.50^{a} \pm 1.96$	$12.6^{ab} \pm 5.89$	$24.5^{bc} \pm 4.83$	$36.4^{c} \pm 7.70$
Non-urea	[g N/d]	$0.94^{a} \pm 0.21$	$1.57^{ab} \pm 0.42$	$2.02^{bc} \pm 0.17$	$2.61^{c} \pm 0.49$
	[% of N intake]	19.5 ± 3.79	17.0 ± 4.29	17.5 ± 1.02	18.4 ± 3.54
PD:	[g N/d]	$0.32^{a} \pm 0.03$	$0.65^{b} \pm 0.11$	$0.77^{bc} \pm 0.11$	$0.96^{c} \pm 0.12$
Allantoin	[g N/d]	$0.30^{a} \pm 0.03$	$0.61^{b} \pm 0.10$	$0.72^{bc} \pm 0.11$	$0.90^{c} \pm 0.11$
	[% of N intake]	6.24 ± 0.51	6.62 ± 0.77	6.26 ± 1.00	6.25 ± 0.19
Uric acid	[g N/d]	$0.01^{a} \pm 0.01$	$0.02^{ab} \pm 0.00$	$0.03^{b} \pm 0.01$	$0.03^{b} \pm 0.01$
Hypoxanthine	[g N/d]	0.01 ± 0.00	0.02 ± 0.01	0.03 ± 0.02	0.04 ± 0.01
Creatinine	[g N/d]	$0.17^{a} \pm 0.01$	$0.23^{b} \pm 0.02$	$0.23^{b} \pm 0.04$	$0.25^{b} \pm 0.01$
	[% of N intake]	$3.49^{a} \pm 0.56$	$2.54^{b} \pm 0.22$	$1.98^{bc} \pm 0.25$	$1.75^{c} \pm 0.20$
Fecal	[g N/d]	2.64 ± 0.62	3.65 ± 0.69	3.66 ± 0.34	4.13 ± 0.92
	[% of N intake]	$54.7^{a} \pm 8.29$	$39.4^{b} \pm 3.67$	$31.7^{b} \pm 2.67$	$28.7^{b} \pm 3.69$

[a, b, c] Means within a row with the same or without any superscript at all are not significantly different ($P < 0.05$).

Urea was the first and allantoin the second largest N-compound excreted in the urine of kids. Kids fed ration 4 excreted three times as much allantoin and uric acid and four times as much hypoxanthine as those of ration 1. The overall daily allantoin, uric acid and hypoxanthine excretion was 93%, 3% and 4% of PD excreted, respectively, and the same proportion was found in each of the groups. Thus allantoin showed similar tendency of increment like non-urea-N and PD with increasing dietary CP level. Differences in urinary N excretion were significant among the treatment rations except between rations 1 and 2. Differences in fecal N among treatment rations were not statistically significant. Kids fed ration 4 excreted 4 time as much urinary N but only 1.6 time as much fecal N as those of ration 1 whereas their N intake difference was only 3 fold.

^{15}N-balance

The results of ^{15}N-balance, IL and kinetics of urea are shown in Table 4 and Figure 1.The amount of ^{15}N injected varied on the average from 42 to 48 mg. When the mass of ^{15}N in the respective routes of excretion is related to the injected dose, it gives the recovery of the injected dose in the corresponding routes in 10 days (Table 4). Of the injected doses 20.2 and 22.3% were recovered in urinary urea of group 1 and 2, respectively, which were significantly lower than that of group 3 (38.8%) and group 4 (46.1%). Differences in recovery of the dose in urinary non-urea followed the same trend as in urinary urea but were not statistically significant. Percent recovery of the injected dose in feces of group 4 was the lowest (4.36%) and

of group 1 the highest (18.3%), percent recovery of groups 2 and 3 lying in between, and differences amongst groups were significant except between adjacent groups. Group 1 had significantly higher percentage of the dose not recovered in feces and urine, or retained in the body, (54.2%) than that of group 4 (33.1%). The lowest percentage of the injected dose was excreted in urine, more of it in feces but the highest percentage of it retained in the kids of group 1 than the rest of the groups. In kids of group 4 the opposite trend has happened.

Table 4. Injected dose, balance and recovery of the injected dose of ^{15}N in the goat kids during the 10 days collection period (mean ± SD)

	Ration 1 n = 4	Ration 2 n = 4	Ration 3 n = 4	Ration 4 n = 3
Injected dose [mg]	43.1 ± 7.4	47.1 ± 1.6	47.8 ± 2.2	42.2 ± 2.3
Recovered in (mg):				
urinary urea	8.6^{a} ± 0.6	10.5^{a} ± 2.0	18.6^{b} ± 2.6	19.3^{b} ± 1.7
urinary non-urea	3.4 ± 3.3	5.9 ± 3.8	6.3 ± 0.9	6.8 ± 2.6
feces	8.0^{a} ± 3.2	7.5^{a} ± 1.5	3.2^{ab} ± 1.0	1.9^{b} ± 1.3
not recovered	23.2 ± 2.9	23.3 ± 5.4	19.8 ± 2.5	14.2 ± 5.7
Recovery of dose (%)				
urinary urea	20.2^{a} ± 2.7	22.3^{a} ± 4.5	38.8^{b} ± 5.1	46.1^{b} ± 6.7
urinary non-urea	7.3 ± 6.8	12.6 ± 8.3	13.2 ± 2.1	16.4 ± 7.4
feces	18.3^{a} ± 5.8	16.0^{ab} ± 3.0	6.8^{bc} ± 2.4	4.4^{c} ± 2.9
not recovered	54.2^{a} ± 4.4	49.2^{ab} ± 10.6	41.3^{ab} ± 3.9	33.1^{b} ± 11.2

[a, b, c] Means within a row with the same or without any superscript at all are not significantly different ($p < 0.05$).

DISCUSSION

Digestibility of organic matter (OM)

Rations 2, 3 and 4 had higher OM digestibility than that of ration 1, which could be attributed to differences in their CF and lipid digestibility. Comparable results were reported where captive black bucks were fed oats (13% CP) and berseem (20% CP) and found increased OM digestibility with berseem than with oats (Pathak *et al.*, 1992). According to Jia *et al.* (1995) CF digestibility was significantly greater for Angora and cashmere-producing Spanish goats that consumed a high protein diet (16%) than those fed on a low protein diet (8%). This was attributed to increased NDF digestibility for the high CP diet. Tilahun Sahlu *et al.* (1993) fed wethers with 3 levels of CP (9, 15 and 21%) and found higher apparent digestibility of NDF with increasing CP level. Rations 2, 3, and 4 had higher amounts of N per unit of DM intake than ration 1. Increased N intake could have favored multiplication of rumen microbes, fastened fermentation of OM, facilitated production and absorption of VFA across the rumen wall and increased availability of absorbable microbial N in the small intestine. These could have in the final analysis increased OM digestibility.

Fecal N excretion

Kids fed different rations consumed different amounts of DM and DOM. However, the excretion of fecal N per kg of DM and DOM consumed were relatively constant but it was variable per kg of IDOM intake between treatment rations (Table 5).

Table 5. The influence of dry matter, digestible organic matter and indigestible organic matter intakes on excretion of fecal N.

	Ration 1	Ration 2	Ration 3	Ration 4
Fecal N:				
[g/kg DM intake]	7.2 ± 1.2	6.8 ± 0.5	6.6 ± 0.4	7.3 ± 1.1
[g/kg DOM intake]	10.9 ± 2.2	9.4 ± 0.9	8.9 ± 0.8	9.8 ± 1.5
[g/kg IDOM intake]	26.4^{a} ± 2.9	31.2^{ab} ± 1.4	36.2^{b} ± 2.7	37.6^{b} ± 6.3

[a, b] Means within a row with the same or without any superscript at all are not significantly different ($P < 0.05$).

Fecal N tended to be 0.7% of DM intake and 1.0% of DOM intake. And because DM intake, OM digestibility and dietary CP concentration were positively correlated with one another, the data were fitted into the following multiple regression model in order to distinguish which of the latter 3 had the greatest impact on fecal N excretion:

$$Y = a + b_1x_1 + b_2x_2 + \varepsilon \quad (1)$$

where y = fecal N excreted (g/d); x_1 = DM intake (g/d); x_2 = CP concentration (g/kg DM) or OM digestibility (%); b_1 and b_2=regression coefficients; a=intercept; and ε is the error term.

The data were tested at 5 and 10% significance levels. It was found that DM intake was highly significantly correlated with fecal N excretion even at a significance level of 5% ($r = 0.74$, $p < 0.001$). Multiple regression analysis of fecal N on DM intake and OM digestibility showed that OM digestibility was significantly correlated ($r = 0.79$) only at 10% significance level (Table 6). When fecal N was regressed on DM intake and CP concentration it was found that the correlation between CP concentration and fecal N was extremely low and nonsignificant even at 10% probability ($r = 0.35$, $p = 0.2$). It can thus be concluded that DM intake influenced fecal N excretion much more than dietary CP concentration.

The excretion of fecal N per unit of N intake declined from 55% to 29% with increasing N intake (Table 3). Even if DM intake and CP concentration had a cumulative effect on N intake, the progressive decline in the excretion of fecal N per unit of N intake with increasing N-intake partly explains why

CP level in the diet was less important in fecal N excretion. Large increment in the ratio of urinary N to fecal N from 0.5:1.0 to 1.9:1.0 with increasing CP level (Table 3) also indicates that excretion of urinary N rather than fecal N was more closely related to N intake.

Table 6. Parameters estimated from fitting data into a multiple regression model ($y = a + b_1x_1 + b_2x_2 + \varepsilon$) for daily fecal N excretion of goat kids from 1.92–5.33 g.

Parameter	Coefficient	SE	r^2	Significance
x_1, DM intake (kg/d)	9.15	1.55	0.74	$p < 0.001$
x_2, OM digestibility (%)	-0.07	0.04	0.79	$p < 0.1$
a, constant	4.11	2.27		

Tilahun Sahlu *et al.* (1993) conducted studies on goats fed with three levels of protein (9, 15 and 21%); and Jia *et al.* (1995) on Angora and cashmere-producing Spanish goats fed with two levels of protein (8 and 16%) and reported that fecal N remained unaffected with CP level in the diet. Qi *et al.* (1994) fed Alpine and Angora kids 22 and 16 g N/d, respectively and found similar fecal N output as percent of N intake.

Urinary N excretion, which is the reflection of N absorption, was higher in the kids fed higher levels of CP. At the same time the amount of fecal N per unit of DM intake remained constant whatever the CP level was. It could then be deduced that some of the fecal N was of endogenous origin. And higher DM intake could have increased the contribution of endogenous or metabolic N to feces. Higher endogenous N contribution at higher levels of feed intake could be associated with increased abrasion and sloughing of the epithelium of the gut (Kennedy and Milligan, 1978). More DOM and N were consumed and higher amounts of allantoin excreted in urine of kids fed ration 4 than the rest of the rations. Thus kids fed ration 4 could have produced more microbial protein and could have increased the contribution of microbial N in feces because the microbes are not absorbed intact and entirely. Earlier reports from the analysis of the materials excreted in feces indicated that microbial yield and its digestibility have a greater influence on fecal N loss (Mason, 1979; Van Soest, 1994).

Daily excretion of urinary N-fractions

Urea was the largest urinary N compound in the kids fed higher levels of CP. Percent urea-N of urinary N in kids fed rations 1, 2, 3 and 4 were 31, 42, 58 and 66%, respectively. Similarly percentage of urea N per unit of N intake increased from 9 to 36% with increasing dietary CP level (Table 3). This shows that excretion of urea was strongly affected by the level of dietary CP and N intake. Lindberg and Jacobson (1990) reported reduced excretion of total N and urea N with decreasing N-intake in sheep.

Allantoin was the major N-compound in PD and its daily excretion tend to increase with increasing dietary CP level. However, the percentage of allantoin per unit of N intake was constant, but percent urea-N increased and percent creatinine-N and fecal N decreased consistently with increasing N intake (Table 3). Kids fed ration 4 excreted 3 times as much allantoin and uric acid and 4 times as much hypoxanthine as those of ration 1. Excretion of urea N was very much influenced by N intake but PD-N was less affected and creatinine and fecal N were in fact negatively influenced by it, *i.e.*, as the amount of N intake increased beyond 10 g/d more of it was excreted as urinary urea and relatively less of it as fecal N and still much less as urinary non-urea N. Allantoin, hypoxanthine and and uric acid were 93, 4 and 3% of PD, respectively. This proportion was different from those reported by Chen *et al.* (1990) for sheep (55, 32 and 14%: for allantoin, uric acid and xanthine plus hypoxanthine, respectively) and cattle (82 and 18%: for allantoin and uric acid, respectively). The higher proportion of hypoxanthine than uric acid and the absence of xanthine in the kids indicate that the amount of xanthineoxidase in the plasma and tissues of the kids was most probably very limited. It could thus be assumed that the contribution of endogenous purines to the PD excreted in urine was minimal. Because the resulting hypoxanthine from tissue turnover of purines would have been reused, as of the well-known pathway of reactions in the catabolism of purine up to hypoxanthine to be reversible. If the endogenous hypoxanthine could have been reused, then the hypoxanthine found in urine was most likely of microbial origin that was not salvaged for the resynthesis of tissue purine by the kids. This is an indication of low N utilization of microbial nucleic acid by kids.

The influence of digestible OM intake (DOMI) on urinary urea and PD excretion

The results of the linear regression analysis indicated that DOMI was more closely related to excretion of PD than urea (Table 7). But larger DOMI was associated with higher dietary CP concentration. In order to tell which of the two factors had a greater influence on PD-N excretion, multiple regression analyses were run using model (2):

$$Y = a + b_1x_1 + b_2x_2 + \varepsilon \qquad (2)$$

where y=PD-N excretion (g/d); x_1=CP concentration in the ration (g/kg DM); x_2=DM intake (kg/d) or DOM intake (kg/d); b_1 and b_2=regression coefficients; a=intercept; and ε is the error term.

From the results of the regression analyses shown in Table 8, it could be generalized that PD excretion was strongly correlated with DOMI (r^2 = 0.95,

p<0.001) than with dietary CP concentration. According to Lindberg and Jacobson (1990), sheep were nourished with 3 levels of energy and 6 levels of protein by intragastric infusion. The proportion of urea-N in the urine of sheep was the highest at low energy intake and decreased in order of energy supply but urinary excretion of PD was largely unaffected by moderate changes in energy intake and by large changes in protein intake. Such a relationship was also found in goat kids (Lindberg, 1989).

Table 7. Parameters estimated from fitting data into a linear regression model ($y = ax + b + \varepsilon$) for the daily DOMI (variable, x) of goat kids from 201 - 483 g.

variable, y	a	b	SE	r^2	significance
Urea-N	15.1	-3.16	1.52	0.44	$p < 0.01$
Non-urea-N	6.49	-0.60	0.45	0.61	$p < 0.001$
PD-N	2.72	-0.32	0.12	0.81	$p < 0.001$
Allantoin-N	2.53	-0.30	0.11	0.81	$p < 0.001$

Table 8. Parameters estimated from fitting data into a multiple regression model for PD-N excretion (variable, y) of goat kids from 0.28 to 1.09 g/head/d.

	coefficient	SE	r^2	significance
CP-concentration	0.005	0.001	0.84	$p < 0.001$
DOM intake	1.477	0.296	0.95	$p < 0.001$
Constant	-0.441			

Irreversible loss and the kinetics of urea

The amount of IL of N from the BUP through individual routes within a day urea-N from BUP degraded in the GIT and the portion of it recycled to the MP are displayed in Figure 1. IL of N from BUP increased with increasing CP level in the ration. IL of group 1 was significantly lower than that of groups 3 and 4; and groups 2 and 3 than that of group 4. All other differences were not significant. In kids of groups 1, 2 and 3 the major portion of IL was retained in the body. This portion showed significant differences only between groups 1 and 4. IL of N via urinary urea depicted significant differences among all the groups except between groups 1 and 2. IL of N via urinary non-urea showed significant differences only between groups 1 and 4. As shown in Figure 1, an average of 1.98 and 12.24 g of urea-N were irreversibly lost daily from the urea pool of groups 1 and 4, respectively. Also 20% and 45% of IL were excreted in urinary urea whereas 80% and 55% of it were directed to and degraded in the GIT of groups 1 and 4, respectively. Groups 2 and 3 had values, which lie between the values of groups 1 and 4.

In lactating Saanen goats fed with low (LP) and high protein (HP) diets, IL of N from the BUP was 13.5 and 22.2 g N/d, respectively (Bornemann, 1995)

which were higher than IL found in the goat kids. The percentage of urea N degraded in the GIT was also higher in the lactating goats. Lower percentage of N leaving the BUP was directed to and degraded in the GIT of both lactating and growing kids fed higher level of protein and the vice versa.

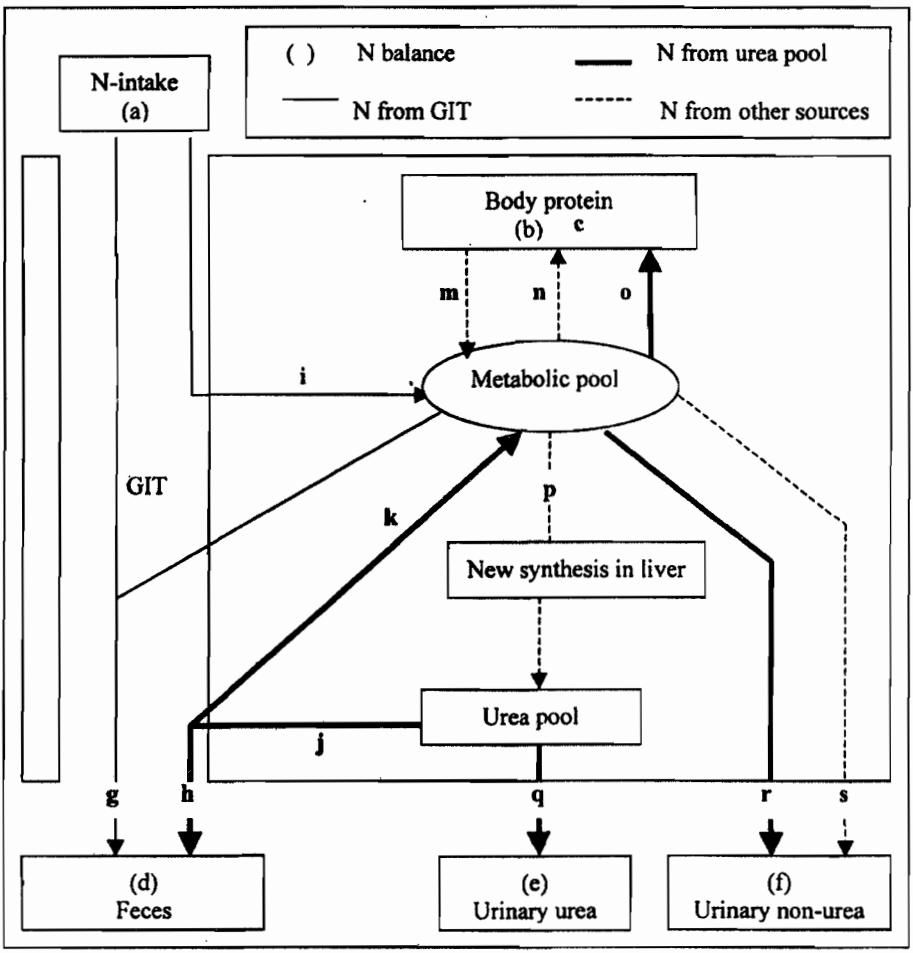

Fig. 1. Model of turnover of N of the kids (g N/d). The body fluid with dissolved urea is designated as urea pool. Influx of urea into this pool (p) is possible only through the liver. In growing male kids urea that leaves this pool flows into the kidney (q) and excreted as such or into GIT (j). After hydrolysis of the urea coming into the GIT, a portion of it is excreted in feces (h) and the rest (k) is transferred to the metabolic pool; into this pool also come end products of tissue protein breakdown (m) and absorbed feed nutrients (i). From this pool the following are removed: amino acids for body protein synthesis (n and o); ammonia for synthesis of urea by liver (p), non-urea N compounds to be excreted by the kidney (r and s). The following table (Table 9) is the summary of the turnover of N in the kids fed diets 1, 2, 3 and 4.

Table 9. The summary of the turnover of N in the kids fed diets 1, 2, 3 and 4.

Ration	1	2	3	4
N-intake (a)	(4.79)	(9.21)	(11.53)	(13.90)
Body protein (b)	(0.81)	(2.83)	(3.01)	(1.86)
	2.79	7.90	10.52	14.10
Feces (d)	(2.64)	(3.65)	(3.66)	(3.73)
Urinary urea (e)	(0.40)	(1.16)	(2.84)	(5.55)
Urinary non-urea (f)	(0.94)	(1.57)	(2.02)	(2.76)
g	2.28	2.84	3.13	3.16
h	0.36	0.81	0.53	0.57
i	2.51	6.37	8.40	10.74
j	1.58	3.91	4.67	6.69
k	1.22	3.10	4.14	6.12
m	1.98	5.07	7.51	12.24
n	1.72	5.35	7.37	9.88
o	1.07	2.55	3.15	4.22
p	1.98	5.07	7.51	12.24
q	0.40	1.16	2.84	5.55
r	0.15	0.55	0.99	1.90
s	0.79	1.02	1.03	0.86

Of the total IL of urea 55, 62, 77 and 80% were degraded in the GIT of kids of groups 4, 3, 2 and 1, respectively. Comparable results were reported where 25 to 80% of IL was degraded in the GIT of calves fed adequate to low protein diets, respectively (Dhiman and Arora, 1987; Bunting *et al.*, 1989). The rate of transfer of urea to the GIT of the kids increased with increasing ratio of g N intake/d: OM digestibility (Table 2 and Fig. 1). Comparable tendency was reported with cattle (Kennedy, 1980). A higher percentage of IL was degraded in the GIT of the kids fed less amount of N. Cocimano and Leng (1967) and Ford and Milligan (1970) reported that 23–92% of the plasma urea production in sheep was degraded, with the higher values associated with low intakes of dietary N. Of the urea-N degraded in the digestive system 23% and 9% of it left the animal system in feces of group 1 and 4, respectively. However, the absolute amount of it excreted in feces showed no trend and group differences were not statistically significant, indicating that IL of N through feces was almost constant. Variably larger proportion of the degraded urea-N was therefore reabsorbed into the MP. A larger proportion of IL was reabsorbed into the MP of group 1 (62%) than group 4 (50%). However, group 4 had reabsorbed significantly larger percentage of degraded urea-N (91%) than group 1 (77%), but differences between adjacent groups were not significant. Bornemann (1995) estimated the amount of N reabsorbed from the urea-N degraded in the GIT of lactating goats to be 17.3 and 13.5 g N per day which were 78 and 80% of degraded urea-N for the high and low protein groups, respectively and showed no distinct trend as in the kids.

Group 4 excreted the highest and group 1 the lowest amount of urinary non-urea-N of BUP origin but these values were almost constant in lactating

goats (Bornemann, 1995). The portion of urinary non-urea-N from sources other than BUP remained almost constant among groups. For body protein synthesis groups 2, 3 and 4 used smaller proportion of reabsorbed N of BUP origin than of dietary origin. The proportion of N of dietary origin used for body protein synthesis increased from 62 to 70% but that of absorbed N of BUP origin on the other hand decreased from 38 to 30% with increasing CP level in the ration. Previous report showed that sheep retained smaller percentage of the N of a dose of urea (22–52%) given intravenously (Houpt; 1959). Body protein synthesis by groups 1, 2, 3 and 4 were equivalent to 2.79, 7.90, 10.52 and 14.01 g N/d, respectively. From the body protein synthesized only 0.81, 2,83, 3.01 and 1.86 g N were retained in the body daily by groups 1, 2, 3 and 4, respectively. This indicates that kids of group 2 had maximum protein utilization (35%) but kids fed with the two extreme CP levels in the ration had lower protein utilization (Tegene Negesse *et al.*, 2001).

The gut microbes could have probably utilized urea N coming from BUP more efficiently than the dietary protein. Thus, a larger proportion of urea-N degraded in the GIT might have escaped the rumino-hepatic pathway, appeared as microbial N and reabsorbed into the MP whose end-products of metabolism could only be non-urea N and were eventually excreted as such. It can thus be said that the amount of urea recycled to the GIT explains most of the group differences in non-urea N excretion.

CONCLUSION

The excretion of urea is the mechanism by which growing goat kids adapt to variable supply of dietary protein. Excretion of fecal N was mainly influenced by DM intake and of urinary purine derivatives by digestible OM intake. Irreversible loss of N from BUP, transfer of urea to the digestive tract and recycling of urea degrade in GIT increased with increasing dietary CP level.

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