Dietary Carboxymethylcellulose with High Instead of Low Viscosity Reduces Macronutrient Digestion in Broiler Chickens[1]

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ABSTRACT The question addressed was whether the viscosity per se of dietary non-starch polysaccharides influences macronutrient digestion in broiler chickens. Water-soluble carboxymethylcellulose preparations of low (LCMC) or high viscosity (HCMC) were fed to broiler chickens (n = 10/group) from 21 to 35 d of age. The HCMC preparations reduced weight gain and raised water intake compared with LCMC. After the HCMC diet was fed, viscosity of the supernatant of small intestinal contents was significantly raised. The HCMC preparations raised the group mean ATP concentration in the digesta of duodenum plus jejunum, indicating that bacterial activity was increased. Consumption of HCMC depressed apparent fecal digestibility of lipids and nitrogen and also apparent ileal digestibility of starch. The dietary HCMC tended (P = 0.077) to reduce plasma triglyceride concentrations. After HCMC consumption, the weights of the small intestine and colon, without or with contents, were elevated. The data indicate that high viscosity of digesta in broiler chickens is associated with a reduced macronutrient digestion and impaired growth performance. Because the carboxymethylcellulose preparations were nonfermentable by fresh feces, we suggest that HCMC reduces macronutrient digestion by raising the viscosity of small intestinal contents, which is associated with enhanced bacterial fermentation due to accumulation of undigested material. J. Nutr. 127: 483–487, 1997.

KEY WORDS: • *dietary fiber* • *carboxymethylcellulose* • *broiler chickens* • *viscosity*

The water-soluble, non-starch polysaccharides (NSP) occurring in rye, wheat and barley are believed responsible for the reduction of growth performance and the digestibility of lipids, protein and starch in broiler chickens fed these feedstuffs (Choct and Annison 1992a, Fengler and Marquardt 1988, White et al. 1981). On the basis of a literature review, Annison (1993) proposed that dietary soluble NSP inhibit nutrient absorption in broiler chickens not only by raising the viscosity of the digesta but also by enhancing bacterial fermentation. It is not known how the effect of soluble NSP is mediated by enhanced bacterial fermentation, but the addition of antibiotics to the diet of broiler chickens can diminish the inhibitory effect of NSP on nutrient absorption (Misir and Marquardt 1978).

Viscous plant NSP generally are readily fermentable (Roberfroid 1993), and thus it is difficult to assess separately the antinutritive effects of viscosity and fermentability. The effect of viscosity per se can be studied by the use of non-fermentable carboxymethylcellulose (CMC) preparations with different degrees of viscosity. Thus, in the present study water-soluble CMC with various polymer lengths were fed to broiler chickens to investigate their effect on growth performance and on digestibility of macronutrients.

MATERIALS AND METHODS

Animals and diets. The experimental protocol was approved and supervised by the animal welfare officer of the DLO-Institute for Animal Science and Health, Lelystad, The Netherlands. One-day-old female broiler chickens (Ross, Cobroed, Lievelde, The Netherlands) were housed in wire-bottomed, suspended cages, exposed to constant light and given free access to food and water throughout. During the first 3 wk of age, all birds were fed the pelleted diet with CMC of low viscosity (**Table 1**). The diet fed during the run-in period contained cellulose type BWW40 instead of type BC1000 (Rettenmaier und Söhne, Ellwangen/Jagst, Germany), which was used in the experimental diets. Then, eight groups of 10 birds each were composed so that the groups had similar body weight distributions. Four groups received the diet with CMC of low viscosity (LCMC diet, Table 1), and the other four groups were fed the diet with CMC of high viscosity (HCMC diet, Table 1). The birds were randomly allocated to pens so that there were four pens per dietary treatment. The experimental pelleted diets were fed to the broiler chickens from 21 to 35 d of age. Body weights were determined at the beginning and end of the experiment. Food and water intakes were recorded.

Collection of samples. During the last 3 d of the experimental period, the excreta in each cage were collected daily and stored at −20°C. The excreta were pooled per cage, freeze-dried and milled using a 1-mm sieve. At the end of the experiment, all birds were killed in a nonfed state by intravenous injection with 2 mL of T61 containing 0.4 mg of butramide, 0.1 mg of mebezonicumiodide and 0.01 mg of tetracainehydrochloride (Hoechst Veterinär, GmbH, München, Germany). Immediately after injection, blood samples

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TABLE 1

Composition of the experimental diets

Ingredient	Amount
	g/kg
Corn	340
Cornstarch	300
Soybean protein isolate	200
Animal fat	50
Ground limestone	10
Monocalcium phosphate	14
Potassium bicarbonate	10
DL-Methionine	2
Vitamin-mineral premix[1]	12.5
LCMC[2] or HCMC[3]	10
Cellulose[4]	50
Chromium trioxide	1.5

[1] The vitamin-mineral premix supplied the following (mg/kg diet): thiamin, 1; riboflavin, 6; calcium pantothenate, 12; niacin amide, 40; pyridoxine, 2; cyanocobalamin, 0.0225; choline chloride, 369; folic acid, 1; biotin, 0.065; retinyl acetate, 25; cholecalciferol, 0.05; DL-α-tocopheryl acetate, 32.5; menadione, 1.8; ascorbic acid, 20; MgO, 995; MnO_2, 134; $ZnSO_4 \cdot H_2O$, 155; $FeSO_4 \cdot H_2O$, 233; $CuSO_4 \cdot 5H_2O$, 50; $Na_2SeO_3 \cdot 5H_2O$, 0.6; $CoSO_4 \cdot 7H_2O$, 0.6; KI, 4; ethoxyquin, 100.

[2] Carboxymethylcellulose of low viscosity (AF1985, Akzo Chemicals, Arnhem, The Netherlands).

[3] Carboxymethylcellulose of high viscosity (AF2805 Akzo Chemicals, Arnhem, The Netherlands).

[4] Cellulose type BC1000 (Rettenmaier und Söhne, Ellwangen/Jagst, Germany).

were taken by heart puncture. Plasma was collected by low-speed centrifugation and stored at −20°C until lipid analyses. The digestive tract between the gizzard and Meckel's diverticulum was removed to obtain the duodenum plus jejunum. The ileum was isolated as the intestine between Meckel's diverticulum and the ileo-cecal junction. The caeca and colon were removed as intestine distal to the ileo-cecal junction. Intestinal contents were collected by gently finger-stripping the intestinal segments. The weights of pancreas, liver and intestinal segments with or without contents were recorded. After collection, the intestinal contents were immediately placed on ice and pooled per segment per cage. Portions of the segments were stored at −80°C for ATP analysis. Portions of the ileal content pools were stored at −20°C and subsequently freeze-dried. Fresh samples of pooled duodenal plus jejunal and ileal contents were immediately centrifuged at 5000 × *g* for 15 min at 4°C in a MSE mistral 3000i centrifuge (MSE, Leicester, U.K.) using a Windshield rotor (type 43124-708 BS 4402, Beun de Ronde BV, Abcoude, The Netherlands). The supernatant was collected for viscosity measurement.

Analyses and measurements. Suspensions of the two CMC types (1 g/100 g) and of the experimental diets (10 g/100 g), milled at a 1-mm sieve, were prepared in distilled water and incubated for 30 min at 38°C for in vitro viscosity measurements. The diet suspensions were centrifuged at 6000 × *g* for 15 min, and the supernatant was isolated for the viscosity measurements. The viscosity of the CMC suspensions and the supernatants of diet suspensions was measured at a shear rate of 0.0623 s^{-1} and a temperature of 38°C using a Bohlin VOR Rheometer (Bohlin, Reologi, Mühlacker, Germany).

For the determination of the in vitro fermentability of the CMC preparations, a 32 g/100 g fecal slurry was made by using fresh human feces and sodium phosphate buffer (0.1 mol/L, pH 6.5). The slurry was kept under a continuous flow of nitrogen gas. Five milliliters of the slurry was mixed with 5 mL of a sodium phosphate buffer (0.1 mol/L) without addition or containing 2 g/100 mL citrus pectin, LCMC or HCMC. The citrus pectin served as a positive control and the buffer without addition as a negative control. For each condition, five plastic tubes were used to determine the fermentation products after 0, 1, 3, 6 and 24 h. The incubation tubes were placed in a water bath at 37°C. At each time interval, one tube was removed and centrifuged for 10 min at 4260 × *g* and 4°C, and the supernatant was collected and stored at −20°C. The amounts of acetic, propionic, butyric and valeric acids in the supernatant were determined by the method of Tangerman et al. (1983).

The viscosity of nondiluted supernatants prepared from the contents of duodenum plus jejunum and ileum was measured using a cone-plate viscometer (Brookfield digital DV-II+, Brookfield Engineering Labs, Stoughton, U.K.) maintained at 40°C. The values were recorded at a shear rate of 45 s^{-1}. Viscosity was expressed as milli-Pascal seconds (mPa·s). The pH of intestinal contents was determined immediately after collection (PHM 82 standard pH meter, Radiometer Nederland, Zoetermeer, The Netherlands). Plasma triglyceride and cholesterol concentrations were determined with the use of commercial test kits (CHOD-PAP and GPO kits, Boehringer-Mannheim GmbH, Mannheim, Germany). Chromium in 1-g samples of food and freeze-dried chyme and excreta was determined by atomic absorption spectrophotometry (SpectrAA-10, Varian Nederland BV, Houten, The Netherlands) after the samples had been ashed at 550°C and oxidized by the addition of 6 mL of potassium bromate (30 g/L) and 3 mL of a solution containing magnesium sulfate (0.67 g/L) and orthophosphoric acid (824.5 g/L). Total lipid contents of food and excreta were determined by extraction of the samples with hexane in a Sohxlet tube after they had been boiled in hydrochloric acid (3 mol/L) for 30 min. The determination of nitrogen in food, ileal digesta and excreta was performed with the Kjeldahl method. Fecal nitrogen in the excreta was calculated as total nitrogen minus nitrogen in uric acid. Uric acid was analyzed by the method of Terpstra and De Hart (1974). Starch in food and ileal digesta was analyzed according to the NEN 3574-procedure (NNI 1974). The concentration of ATP in chyme was estimated by the luciferin-luciferase (EC 1.13.12.7) method as described by Bach-Knudsen et al. (1991), with the use of ATP monitoring kits (Microbial Biomass Testkit Lumac 93221-1, ATP Standard Lumac 9263-8 Perstorp Analytical, Oud Beijerland, The Netherlands) and a luminometer (Lumac Biocounter M1500, Perstorp Analytical, Oud Beijerland, The Netherlands).

Statistical analyses. The level of statistical significance was preset at $P < 0.05$. The two-tailed Student's *t* test was used to identify significant differences between chickens fed the HCMC and LCMC diets. Each pen with 10 birds was considered to be one experimental unit.

RESULTS

The addition of pectin to fresh feces resulted in greater production of short-chain fatty acids, but the addition of either LCMC or HCMC did not have a stimulatory effect (**Fig. 1**).

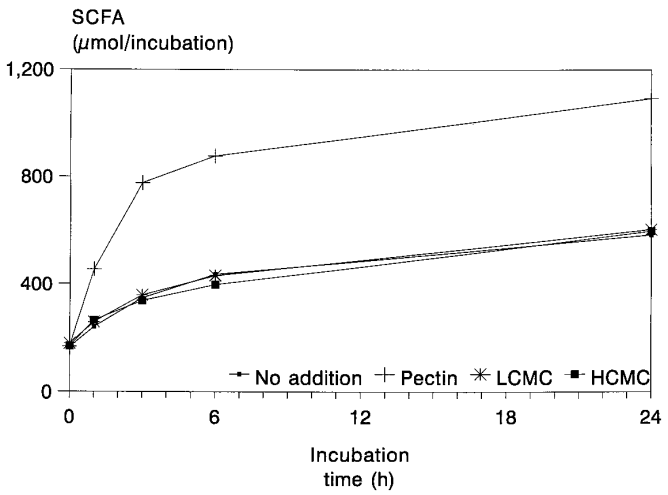

FIGURE 1 Time course of in vitro fermentation in fresh feces incubated with buffer alone or with buffer containing identical amount of either pectin, low (LCMC) or high (HCMC) viscous carboxymethylcellulose. Fermentation in single incubations per time interval was monitored by the formation of short-chain fatty acids (SCFA).

TABLE 2

Viscosity of supernatant, pH and ATP concentrations of digesta in broiler chickens fed diets with low (LCMC) or high (HCMC) viscous carboxymethylcellulose[1]

	Dietary group		
	LCMC	HCMC	Level of significance
Viscosity of supernatant, *mPa·s*			
Duodenum plus jejunum	7.90 ± 1.75	17.38 ± 1.05	$P = 0.001$
Ileum	17.05 ± 2.06	28.65 ± 7.59	$P = 0.062$
pH			
Duodenum plus jejunum	5.24 ± 0.53	5.40 ± 0.21	$P > 0.500$
Ileum	5.38 ± 0.19	5.21 ± 0.22	$P = 0.369$
Caeca	6.38 ± 0.10	6.22 ± 0.16	$P = 0.144$
ATP, *mmol/L*			
Duodenum plus jejunum	1.87 ± 0.52	2.65 ± 0.46	$P = 0.058$
Ileum	4.33 ± 1.33	4.59 ± 1.27	$P = 0.500$
Caeca	21.50 ± 6.74	27.47 ± 3.23	$P = 0.213$

[1] Values are means ± SD of four cage means per dietary group; each cage had 10 birds.

The HCMC preparation had a 59% higher viscosity than the LCMC preparation (207 compared with 130 mPa·s). The supernatant of the diet with HCMC had a 70% higher viscosity than that of the diet with LCMC (4.6 compared with 2.7 mPa·s). Feeding the diet with HCMC significantly raised the viscosity of the supernatant of chyme from the duodenum plus jejunum and from the ileum compared with feeding LCMC diet (**Table 2**).

The pH of the intestinal contents was not affected by the type of CMC. Consumption of HCMC vs. LCMC produced a rise in ATP levels, but the difference only approached significance for the chyme of duodenum plus jejunum ($P = 0.062$).

The feeding of HCMC instead of LCMC raised water intake and reduced weight gain (**Table 3**). The feed to gain ratio was significantly elevated after consumption of the HCMC diet compared with the LCMC diet.

The full and empty weights of small intestine and colon, expressed relative to body weight, were significantly raised by HCMC vs. LCMC in the diet (**Table 4**). Moreover, HCMC consumption, when compared with LCMC, produced an increase in length of the small intestine, caeca and colon. The full weight of the caeca was not significantly influenced by the type of CMC in the diet, but the empty weight was significantly elevated. Pancreas and liver weight were not different for broilers fed the diets with either HCMC or LCMC.

TABLE 3

Weight gain, food intake, water intake and feed conversion ratio of broiler chickens fed diets with low (LCMC) or high (HCMC) viscous carboxymetylcellulose from 21 to 35 d of age[1]

Variable	Dietary group		Level of significance
	LCMC	HCMC	
Weight gain, *g*	583 ± 16	480 ± 15	$P = 0.004$
Food intake, *g*	1059 ± 40	1115 ± 34	$P = 0.096$
Water intake, *g*	1998 ± 166	2475 ± 147	$P = 0.009$
Water:feed ratio, *g/g*	1.886 ± 0.102	2.218 ± 0.092	$P = 0.014$
Feed:gain ratio, *g/g*	1.815 ± 0.029	2.324 ± 0.063	$P = 0.001$

[1] Values are means ± SD of four cage means per dietary group; each cage had 10 birds.

Consumption of HCMC instead of LCMC resulted in lower apparent ileal digestibility of starch and lower apparent fecal digestibility of nitrogen and lipids (**Table 5**). Ingestion of HCMC rather than LCMC tended ($P = 0.077$) to result in lower group mean plasma concentrations of triglycerides (0.44 ± 0.10 compared with 0.58 ± 0.11 mmol/L). Plasma cholesterol concentrations for the HCMC and LCMC groups were 2.45 ± 0.42 and 2.93 ± 0.18 mmol/L, respectively ($P = 0.183$).

DISCUSSION

The objective of this study was to determine whether a higher viscosity of intestinal contents by itself causes depressed growth performance and reduced macronutrient digestibility in broiler chickens. To meet the objective, broilers were fed diets with either HCMC or LCMC. The two types of CMC were verified to be nonfermentable by fecal bacteria but showed different degrees of viscosity after consumption by the broiler chickens. Ingestion of HCMC produced a markedly higher viscosity of small intestinal contents than did the ingestion of LCMC.

When compared with LCMC, HCMC had an antinutritive effect in broiler chickens. Consumption of HCMC depressed growth performance, raised water intake and inhibited macronutrient digestion. Similar results were obtained earlier in broiler chickens after consumption of isolated viscous NSP of plant origin, such as water-soluble wheat pentosans (Choct and Annison 1992a), water-soluble rye pentosans (Antoniou and Marquardt 1981, Fengler and Marquardt 1988) and barley β-glucans (White et al. 1981). Our data indicate that a raised viscosity per se of intestinal contents may be associated with the antinutritive effects.

Consumption of HCMC compared with LCMC depressed the apparent digestibilities of starch, nitrogen and lipids. The mechanism by which an increase in digesta viscosity may affect macronutrient digestion is not clear. Possible processes involved are a reduction of the diffusion rate of digestive enzymes and bile acids (Ebihara and Schneeman 1989, Isaksson et al. 1982) and less mixing of chyme (Edwards et al. 1988). The latter may result in less contact of digesta with the absorptive surface. In the contents of duodenum plus jejunum from chickens fed HCMC, the ATP concentrations were elevated, pointing to increased microbial populations. Because HCMC itself is not fermented, the rise in microbial activity could be due to accumulation of nondigested material. In addition, an in-

TABLE 4

Weights of the intestinal segments, pancreas and liver in broiler chickens fed diets with low (LCMC) or high (HCMC) viscous caroxymethylcellulose[1]

	Dietary group		
	LCMC	HCMC	Level of significance
Small intestine			
Full weight, *g/100 g body wt*	5.10 ± 0.49	6.83 ± 0.62	$P = 0.026$
Empty weight, *g/100 body wt*	3.28 ± 0.23	4.52 ± 0.30	$P = 0.009$
Length, *cm/100 g body wt*	10.78 ± 0.23	13.72 ± 0.86	$P = 0.008$
Caeca			
Full weight, *g/100 g body wt*	0.74 ± 0.09	0.69 ± 0.04	$P = 0.378$
Empty weight, *g/100 g body wt*	0.42 ± 0.02	0.49 ± 0.03	$P = 0.009$
Length, *cm/100 g body wt*	2.16 ± 0.08	2.52 ± 0.16	$P = 0.030$
Colon			
Full weight, *g/100 g body wt*	0.21 ± 0.04	0.27 ± 0.01	$P = 0.023$
Empty weight, *g/100 g body wt*	0.17 ± 0.03	0.24 ± 0.01	$P = 0.009$
Length, *cm/100 g body wt*	0.51 ± 0.04	0.61 ± 0.07	$P = 0.027$
Total intestinal tract			
Full weight, *g/100 g body wt*	6.04 ± 0.44	7.79 ± 0.67	$P = 0.027$
Empty weight, *g/100 g body wt*	3.87 ± 0.24	5.26 ± 0.33	$P = 0.007$
Length, *cm/100 g body wt*	13.44 ± 0.20	16.84 ± 1.04	$P = 0.009$
Pancreas, *g/100 g body wt*	0.24 ± 0.02	0.25 ± 0.01	$P = 0.107$
Liver, *g/100 g body wt*	3.40 ± 0.42	3.48 ± 0.20	$P = 0.500$

[1] Values are means ± SD of four cage means per dietary group; each cage had 10 birds.

crease in viscosity of digesta may raise retention time (van der Klis et al. 1993), which would also stimulate fermentation. The primary effect of HCMC may be the elevation of the viscosity of intestinal digesta, causing depressed macronutrient digestion and enhanced bacterial fermentation. The material sustaining fermentation consists at least of carbohydrates and/or protein rather than lipids, because the latter compounds will not serve as bacterial substrates.

Lipid digestibility was more depressed by HCMC than was the digestibility of protein or starch. A similar pattern was noted in birds fed diets containing isolated water-soluble pentosans (Choct and Annison 1992a and 1992b, Fengler and Marguardt 1988). In young birds, the low bile acid concentration in the digesta may limit lipid absorption (Iñarrea et al. 1989, Polin et al. 1980). Various authors have suggested that the low lipid digestibility in broiler chickens fed diets with a high content of gelling NSP may be due to bacterial overgrowth in the small intestine and subsequent excessive deconjugation of bile acids, which reduces their efficacy in solubilizing lipids (Huhtanen and Pensack 1965, Salih et al. 1991). A marked increase in microbial cholytauryl hydrolase activity was observed in chicks fed rye (Feighner and Dashkevicz 1988). This observation could imply that a high viscosity of intestinal digesta may not be the primary cause for the lowered lipid digestion seen after HCMC consumption. However, it cannot be excluded that a viscosity-induced lowering of diffusion of bile acids through the digesta had a major effect on lipid digestion in birds fed HCMC. Addition of sodium taurocholate to a rye-based diet improved apparent lipid digestibility in broiler chickens (Campbell et al. 1983). In HCMC-fed birds there might be a lowered bile acid efficacy in the digesta, which could then explain not only the low lipid digestibility but also the low plasma triglyceride and cholesterol concentrations as secondary features. Increased viscosity of intestinal contents reduced cholesterol absorption and plasma cholesterol concentration in hamsters fed hydroxypropyl methylcellulose with varying viscosity (Carr et al. 1996, Gallaher et al. 1993).

Volatile fatty acids and polyamins produced by gut bacteria have stimulatory effects on the proliferation rate and secretory activity of intestinal mucosa (Furuse et al. 1991, Osborne and Seidel 1989, Sakata 1987). In HCMC-fed birds, with an increase in microbial fermentation there would be more loss of endogenous nitrogen, leading to a reduction of apparent nitrogen digestibility, as was seen. Angkanaporn et al. (1994) demonstrated that water-soluble pentosans in the diet significantly raised the endogenous losses in broiler birds. Larsen et al. (1993) found an increase in endogenous nitrogen loss when highly viscous CMC was added to the diets of rats, whereas

TABLE 5

Digestibility of nitrogen, starch and lipids in broiler chickens fed diets with low (LCMC) or high (HCMC) viscous carboxymethylcellulose[1]

	Dietary group		Level of significance
	LCMC	HCMC	
	%		
Nitrogen			
Ileal digestibility[2]	71.33 ± 3.58	57.31 ± 11.93	$P = 0.162$
Fecal digestibility[3]	81.63 ± 2.34	65.82 ± 2.53	$P = 0.004$
Starch			
Ileal digestibility[2]	95.38 ± 1.58	87.34 ± 3.64	$P = 0.026$
Lipids			
Fecal digestibility[2]	62.14 ± 9.78	34.34 ± 6.54	$P = 0.002$

[1] Values are means ± SD of four cage means per dietary group; each cage had 10 birds.

[2] Calculated as: $DC_{feed} = (1 - [(M_{feed}/M_{e,c}) \times (_{e,c}/C_{feed})]) \times 100$, where DC_{feed} = digestibility of a nutrient in the feed; M_{feed} = marker (chromium) concentration in feed; $M_{e,c}$ = marker concentration in excreta (e) or chyme (c); C_{feed} = concentration of nutrient in feed; $C_{e,c}$ = concentration of nutrient in excreta (e) or chyme (c).

[3] Corrected for the concentration of uric acid in the excreta (Terpstra and De Hart 1974).

ileal true nitrogen digestibility was not affected. Thus, viscous CMC did not influence nitrogen digestibility in those rats, at least not at the level of the terminal ileum. The increase in bacterial fermentation in the broilers fed HCMC may have been relatively small. There was a rise in ATP concentration only in the duodenum-jejunum, with no lowering of the pH of the digesta. Although part of the observed hypertrophic effect of HCMC on the small intestine, caeca and colon could have been the result of the increase in microbial fermentation, HCMC itself could also have caused the effect. Recently, Pell et al. (1995) observed a trophic effect on the small intestinal length and cecal weight in germ-free mice fed a diet with guar gum. Thus, the trophic effect of HCMC on the length of the small intestine may have been unrelated to the enhanced microbial fermentation. Extra losses of endogenous nitrogen could also be of pancreatic origin, because diets containing viscous fibers have been shown to raise the secretion of pancreatic juice in rats (Ikegami et al. 1991).

In conclusion, the feeding of HCMC instead of LCMC to broiler chickens lowered the apparent digestibility of starch, protein and lipids. The HCMC raised the viscosity of digesta, which may interfere with starch and lipid digestion. The accumulation of undigested carbohydrates would stimulate microbial fermentation. The observed reduction in apparent protein digestibility could have been the result of greater losses of endogenous nitrogen.

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