Guar gum does not impair the absorption and utilization of dietary nitrogen but affects early endogenous urea kinetics in humans[1,2]

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ABSTRACT

Background: Viscous gums enhance viscosity in the upper gastrointestinal lumen, quickly disturbing motility and promoting fluid secretion.

Objective: We sought to determine whether guar gum could acutely affect the absorption and utilization of dietary nitrogen and whether these luminal effects could also perturb the kinetics of urea.

Design: We studied the short-term effect of adding 1% of highly viscous guar gum to a ^{15}N-labeled protein meal (30 g soy protein isolate in 500 mL water) during the postprandial phase in humans. The effects on bioavailability were studied by using the [^{13}C]glycine breath test (to assess gastric emptying) and ^{15}N enrichment in plasma amino acids (for systemic amino acid bioavailability). The kinetics of dietary and endogenous urea were assessed in plasma and urine.

Results: Guar gum modulated the gastric emptying kinetics of the liquid phase of the meal slightly ($P < 0.05$), but had no significant effect on either the systemic appearance of dietary amino acids or plasma and urinary dietary urea kinetics. Without significantly affecting plasma urea concentrations, guar gum reduced by ≈40% the urinary excretion of endogenous urea for the first 2-h period after the meal ($P < 0.01$), although endogenous urinary excretion was similar at later stages.

Conclusions: Guar gum did not significantly affect the bioavailability or utilization of dietary protein. We showed an early effect of guar gum on endogenous urea kinetics, which most probably arose from very early, short-term stimulation of the intestinal disposal of endogenous urea, at the expense of its urinary excretion. *Am J Clin Nutr* 2001;74:487–93.

KEY WORDS Guar gum, dietary nitrogen, urea kinetics, amino acids, intestinal absorption, dietary protein, protein metabolism, dietary fiber, viscosity

INTRODUCTION

The recycling of urea nitrogen from the intestine is recognized as an important pathway in nitrogen homeostasis, but the nutritional modulation of this recycling is not well understood (1–5). Intake of soluble fiber can influence different processes such as gastric and intestinal motility and secretion, distal bacterial activity, and postprandial glycemia and insulinemia, which may affect digestive and absorptive processes and subsequent utilization of nutrients (5–10). The effect of fiber on gastrointestinal viscosity is a mechanism by which soluble fiber, particularly viscous gums, could directly and acutely influence the gastrointestinal kinetics of nutrients and thus their availability and utilization (10–17). However, the direct effect of fiber on gastrointestinal viscosity may, at least in part, be counteracted by the rapid gastric and intestinal secretion of fluids diluting chyme (18–22). The consequences of this fluid secretion on the extent of intestinal nitrogen recycling have not been evaluated. However, through a solvent drag effect on urea, this fluid secretion may contribute to the shift of nitrogen excretion from the renal to the intestinal route. This shift of nitrogen excretion routes was associated with the addition of guar gum to the diet of rats (1, 8, 23). In this context, guar gum may have a particular, complex effect on nitrogen flux during the postprandial period. However, little information is available about the consequences of such luminal perturbations with regard to protein digestion (24, 25) or urea kinetics. Thus, in the present study we sought to investigate the precise effects of dietary fiber on the postprandial availability and utilization of dietary nitrogen and to concomitantly examine its effects on the kinetics of endogenous and dietary urea.

SUBJECTS AND METHODS

Subjects

Seven healthy volunteers (5 men and 2 women) aged 20–36 y ($\bar{x}$ ± SD: 23 ± 5 y) participated in the present study. The subjects were in good health as determined by a thorough medical examination. All subjects were studied after they ingested each of the meals and thus served as their own controls. The protocol was approved previously by the Institutional Review Board for Saint-

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Germain-en-Laye Hospital, France. All subjects gave their full consent to participate in the study after the experimental protocol had been explained to them in detail.

Test meals

Uniformly ^{15}N-labeled soy seeds (var. *Chandor*) were processed in a soy protein isolate by the Nestec Research Centre (Lausanne, Switzerland), as described previously (26). Two experimental meals were prepared. The first meal, mixed with 500 mL water, contained 30 g (316 mmol N) soy isolate (protein meal, P). The second experimental meal, similarly mixed with 500 mL water, contained 30 g soy isolate combined with 5 g powdered guar gum (protein plus guar gum, PG). The guar gum (VISCOGUM HV 3000A; SKW Biosystems, Boulogne Billancourt, France) included small quantities of protein (0.05g/g). Because this small amount of protein raised the nitrogen content of the PG meal only from 316 to 319 mmol N (N × 6.25), the meals were considered to be isonitrogenous. Both meals contained 75 mg [^{13}C]glycine (L-[1-^{13}C]glycine, 99% enrichment; Euriso-Top, Gif-sur-Yvette, France) to evaluate the gastric emptying kinetics of the liquid phase of the meals (27).

The viscosity of the P and PG meals was measured at shear rates of 1, 10, and 100 s^{-1} by using a rheometer (Carri-med CSL-100; TA Instruments, New Castle, DE) with a 4-cm steel conical probe. Viscosity was 0.01 Pa/s for the P meal at each shear rate, whereas for the non-Newtonian PG meal, the apparent viscosity was 0.01, 0.017, and 1 Pa/s at shear rates of 100, 10, and 1 s^{-1}, respectively.

Clinical protocol

Subjects were admitted to the hospital in the afternoon preceding the day of the study. They ate dinner at 2000 and then fasted overnight. On the morning of the study day, a catheter was inserted into a superficial forearm vein for blood sampling and subjects then ingested either the P or PG meal, after which the postprandial sampling period lasted for 8 h. Postprandial blood sampling was performed while the subjects rested. Subjects did not ingest food or fluid until the end. Blood samples were collected hourly during the 8-h period after ingestion of the meal, except for the first 2 h postprandially when additional samples were taken. Plasma was immediately separated from whole blood by centrifugation (1500 × *g* for 15 min at 4 °C) and then frozen at −20 °C until analyzed. Breath samples were collected every 30 min for 8 h and stored until later determination of $^{13}CO_2$ isotopic enrichment. Urine was collected over an 8-h period (0–2, 2–4, 4–6, and 6–8 h) after meal ingestion, treated with thymol crystals and liquid paraffin as preservatives, and stored at 4 °C until analyzed.

Extraction of amino acids, urea, and ammonia from plasma and urine

Urea and ammonia were isolated by batch method, as described previously (28). Briefly, for the extraction of amino acids and urea, plasma proteins were pelleted by adding solid 5-sulfosalicylic acid. Supernatant fluid was collected after centrifugation at 2400 × *g* for 25 min at 4 °C. Ammonia was first extracted from urine by using the sodium-potassium form of cation exchange resin (Dowex AG-50X8, mesh 100–200; BioRad, Montlugon France). The supernatant fluid fraction was collected for further extraction of the urea. Urea was extracted from both the plasma supernatant fluid fraction and the ammonia-free urine fraction by converting it to ammonium through hydrolysis with urease (Sigma, Saint-Quentin-Fallavier, France) for 2 h at 30 °C with cation exchange resin. The part of the plasma fraction not retained in the resin was considered to be the plasma amino acids fraction. Ammonia and urea-derived ammonia were eluted from the resins with the addition of 2.5 mol KH_2SO_4/L.

Analytic methods

The total nitrogen content of the samples was determined by using an elemental nitrogen analyzer (NA 1500 series 2; Micromass, Manchester, United Kingdom) with atropine as the standard. Urea was assayed in both plasma and urine by using an enzymatic method on a clinical analyzer (Dimension automate; Dupont de Nemours, Les Ulis, France). Ammonia was measured in urine by using an enzymatic method on a clinical analyzer (Kone automate; Kone, Evry, France). Creatinine was measured by using a direct colorimetric method on a clinical analyzer (Dimension automate; Dupont de Nemours). Glucose was measured in plasma by using a glucose oxidase method (glucose kit GOD-DP; Kone) and plasma insulin concentrations were measured by using a radioimmunoassay method (INSIK-5; Diasorin, Antony, France).

Isotopic nitrogen enrichments (^{15}N:^{14}N) were measured by isotope ratio mass spectrometry. An aliquot was burned in an elemental analyzer (NA 1500 series 2; Micromass) at 1020 °C and interfaced with an isotope ratio mass spectrometer (Optima; Micromass). The ^{15}N-^{14}N ratios [mass-to-charge ratio (*m/z*) 28:29:30] were measured with reference to a calibrated ^{15}N-^{14}N tank.

Isotopic carbon dioxide enrichments ($^{13}CO_2$:$^{12}CO_2$) were measured by using a gas chromatograph (HP 5890, series II; Hewlett Packard, Les Ulis, France) coupled with an isotope ratio mass spectrometer (Optima). Samples were separated by gas chromatography on a 2.5 m × 3 mm Haysep Q column (Chrompak, Les Ulis, France) at 80 °C and isotopic ratios (44:45:46) were determined by isotope ratio mass spectrometry with reference to a calibrated $^{13}CO_2$-$^{12}CO_2$ tank.

Calculations

$^{13}CO_2$ excretion

Excretion of ^{13}C in breath carbon dioxide, as a percentage of ingested ^{13}C, was calculated from ^{13}C enrichments in the breath according to a technique developed by Maes et al (27). Briefly, when estimating the parameters related to $^{13}CO_2$ excretion, the cumulative dose of $^{13}CO_2$ recovered in breath as a function of time was fitted onto a curve of the equation $y = m(1 - e^{-kt})^{\beta}$ where t is time and m, k, and β are the regression-estimated constants. From these regression-estimated constants, 2 parameters characterizing the gastric emptying rate were obtained according to the following formulas:

$$T_{1/2} = (-1/k)\ln(1 - 2^{-1/\beta}) \quad (1)$$

$$T_{lag} = (\ln\beta)/k \quad (2)$$

where $T_{1/2}$ is the recovery half-time (the time when half of the asymptotic recovery of ^{13}C is reached on the cumulative fitted curve) and T_{lag} is the excretory lag phase (the time at the point of inflexion on the cumulative fitted curve).

Systemic availability of dietary amino acids

The proportions of plasma amino acids containing dietary nitrogen (AA_{diet}:AA_{tot}) were calculated by using the following equation:

$$(AA_{diet}:AA_{tot})(t) = [E(t) - E(0)]/[E_{meal} - E(0)] \quad (3)$$

where $E(t)$ is the ^{15}N enrichment (expressed as ^{15}N atom%) in the amino acids fraction sampled at time t and E_{meal} is the ^{15}N enrichment (expressed as ^{15}N atom%) in the meal.

Dietary and endogenous plasma urea

Dietary and endogenous urea concentrations in the plasma ($Urea_{diet}$ and $Urea_{endo}$, in mmol/L) were calculated by using the following formulas:

$$Urea_{diet}(t) = Urea_{tot}(t) \times [E_{urea}(t) - E_{urea}(0)]/[E_{meal} - E_{urea}(0)] \quad (4)$$

$$Urea_{endo}(t) = Urea_{tot}(t) \times [E_{meal} - E_{urea}(t)]/[E_{meal} - E_{urea}(0)] \quad (5)$$

where $Urea_{tot}(t)$ and $E_{urea}(t)$ are the plasma urea concentration and the ^{15}N enrichment (expressed as ^{15}N atom%) in the plasma urea at time t, respectively.

Urinary dietary and endogenous urea

Dietary and endogenous urea nitrogen excreted in the urine ($Urea_{diet\text{-}urin}$ and $Urea_{endo-urin}$, mmol) were calculated by using the formulas:

$$Urea_{diet\text{-}urin}(t) = Urea_{tot\text{-}urin}(t) \times [E_{urin}(t) - E_{urin}(0)]/[E_{meal} - E_{urin}(0)] \quad (6)$$

$$Urea_{endo\text{-}urin}(t) = Urea_{tot\text{-}urin}(t) \times [E_{meal} - E_{urin}(t)]/[E_{meal} - E_{urin}(0)] \quad (7)$$

where $Urea_{tot\text{-}urin}(t)$ is the quantity of urinary urea at time t and $E_{urin}(t)$ is ^{15}N enrichment (expressed as ^{15}N atom%) in urinary urea at time t. These equations assumed that the amounts of [^{15}N-^{15}N]urea were negligible compared with the concentration of [^{15}N-^{14}N]urea.

Curve fittings and other curve estimates

Different model curves were used during the postprandial period to fit the experimental quantity of *1*) $^{13}CO_2$ excretion as a percentage of ^{13}C ingested, *2*) cumulative dietary urea nitrogen excreted in the urine, and *3*) plasma dietary urea. For *1* and *3*, the curve took the form of $y = \alpha \times e^{-1/2\,[\ln(t/t_0)/\beta]2}$, where t is time, and α, β, and t_0 are regression estimated constants. For *2*, the curve took the form $y = a(1-e^{-b \times t})^c$, where t is time and a, b, and c are regression-estimated constants. Curve fitting of experimental data was performed by using SIGMA PLOT 5.0 (SPSS Inc, Erkrath, Germany).

Statistical analyses

Results are expressed as means ± SDs. Comparisons between meals were assessed by using an analysis of variance procedure with all factors (ie, both time and meal) as repeated factors (SAS/STAT, version 6.03; SAS Institute, Cary, NC). For those measures in which there was a significant interaction, posthoc testing of differences between meals at each time point was performed with paired t tests and P values were adjusted by Bonferroni correction (SAS/STAT). A probability of $P < 0.05$ was considered significant. The correlation coefficients between time series were computed by using statistical software for time series analyses (tsMETRIX; RER Inc, San Diego).

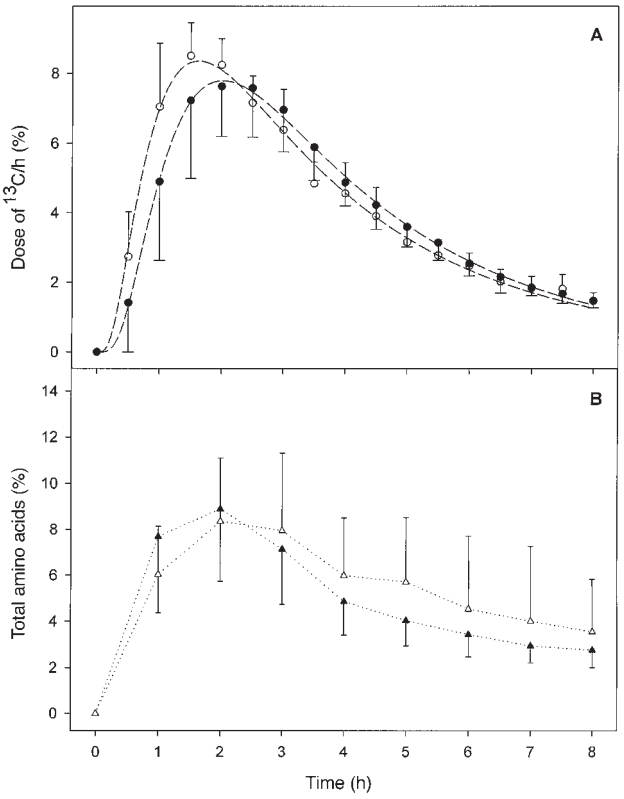

FIGURE 1. A: Mean (±SD) $^{13}CO_2$ excretion in breath, as percentage per hour of the ^{13}C dose ingested as [^{13}C]glycine after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. The meal-by-time interaction was significant, $P < 0.05$, but the meal effect and the difference between meals at each time point were not. B: Mean (±SD) dietary plasma amino acids as a percentage of the total plasma amino acid pool after the ingestion of a liquid protein meal without (△) and with (▲) guar gum, $n = 7$. Neither the meal effect nor the meal-by-time interaction was significant.

RESULTS

Gastric emptying and the appearance of dietary plasma amino acids

The course of ^{13}C excretion in breath carbon dioxide as a percentage of ingested ^{13}C is shown in **Figure 1**A. The type of meal had no significant effect on excretion rates of ^{13}C; however, the meal-by-time interaction was highly significant ($P < 0.01$), ie, the shape of the excretion rate was modulated by the meal. Similarly, the meal-by-time interaction was significant for cumulative ^{13}C recovery, whereas there was no significant difference between meals. Times of maximum ^{13}C excretion for P and PG meals were 110 ± 17 and 136 ± 33 min, respectively. This difference was not significant. The times of half excretion for P and PG meals were 179 ± 16 and 195 ± 30 min, respectively, and did not differ significantly.

The time course of changes in the ratio of dietary to total amino acids in plasma is shown in Figure 1B. This ratio, indicative of the systemic dietary amino acid inflow, was not signifi-

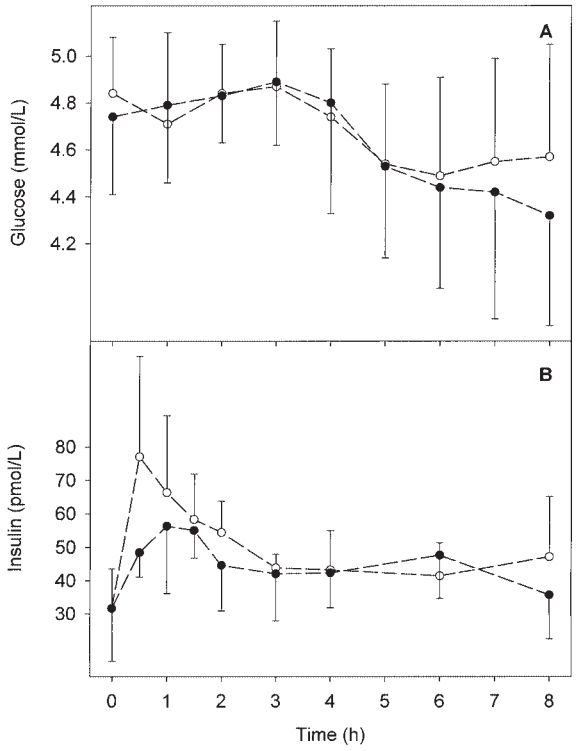

FIGURE 2. A: Mean (±SD) plasma glucose concentrations after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. Neither the meal effect nor the meal-by-time interaction was significant. B: Mean (±SD) plasma insulin concentrations after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. The meal-by-time interaction was significant, $P < 0.05$, but the meal effect and the difference between meals at each time point were not.

cantly different between meals. In addition, there was no meal-by-time interaction. The time course of dietary amino acid appearance correlated well with the appearance of ^{13}C ($r > 0.9$). Assuming that the mean total plasma amino acid concentration did not differ between meals, the ratio F, calculated as the ratio of the area under the (AA_{diet}:AA_{tot}) curve after the PG meal compared with that after the P meal, gives an idea of the relative systemic availability of dietary amino acids after the PG meal. F was ≥0.99 for the first 5 h postprandially, indicating that the systemic availability of dietary amino acids was not altered by adding guar gum to the test meal.

Plasma glucose and insulin

Plasma glucose concentrations after the P and PG meals are shown in **Figure 2**. Intake of guar gum had no significant effect on the concentrations or kinetics of plasma glucose. There were modest variations in plasma insulin concentrations after both meals. Guar gum had no influence on plasma insulin concentrations, but did modulate the global kinetics of insulin significantly, albeit slightly. There was no significant difference between meals at any time point.

Total, dietary, and endogenous plasma urea kinetics

Dietary plasma urea concentrations (**Figure 3**) rose rapidly and similarly after subjects ingested P and PG meals. The addition of guar gum had no significant effect on plasma dietary urea concentrations.

Baseline plasma urea concentrations did not differ significantly between the groups. Total plasma urea concentrations varied with time but guar gum had no significant effect on total plasma urea concentrations and kinetics. Plasma urea concentrations from endogenous sources were not significantly affected by guar gum and did not vary with time.

Urinary excretion of total, dietary, and endogenous nitrogen

Significant amounts of dietary urea were excreted in urine 2 h after both meals (**Figure 4**A). Cumulative excretion rates did not differ significantly between the meals. The excretion of dietary nitrogen ammonia in urine was also similar, but more rapid (data not shown). The type of meal had no significant effect on dietary ammonia excretion.

The type of meal had significant effects on both the magnitude and the shape of the cumulative excretion curves of total urea (Figure 4B). The excretion of total urea in urine was significantly different between meals at the first urine collection; urea

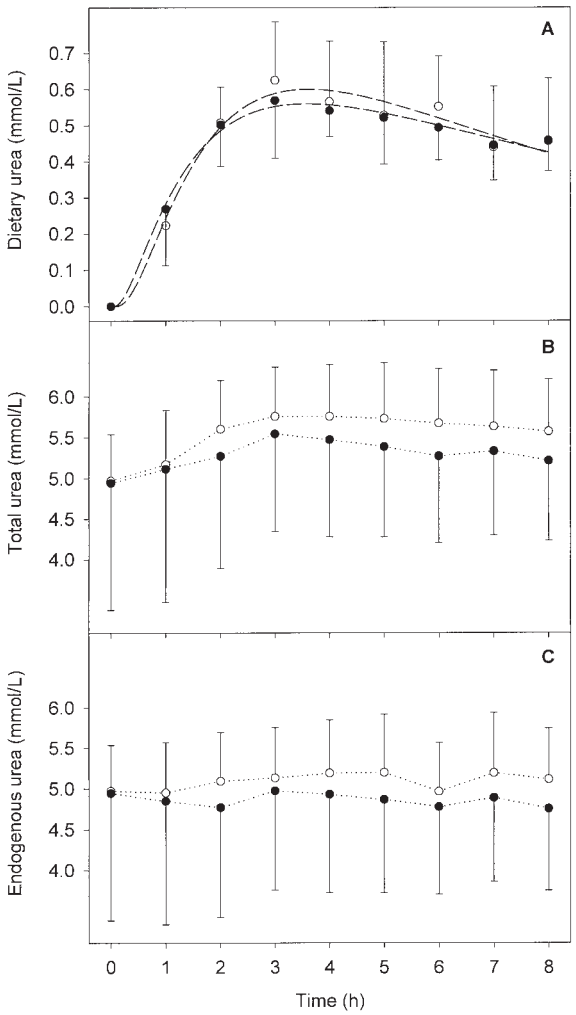

FIGURE 3. A: Mean (±SD) dietary plasma urea concentrations after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. B: Mean (±SD) total plasma urea concentrations after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. C: Mean (±SD) endogenous plasma urea concentrations after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. In all panels, neither the meal effect nor the meal-by-time interaction was significant.

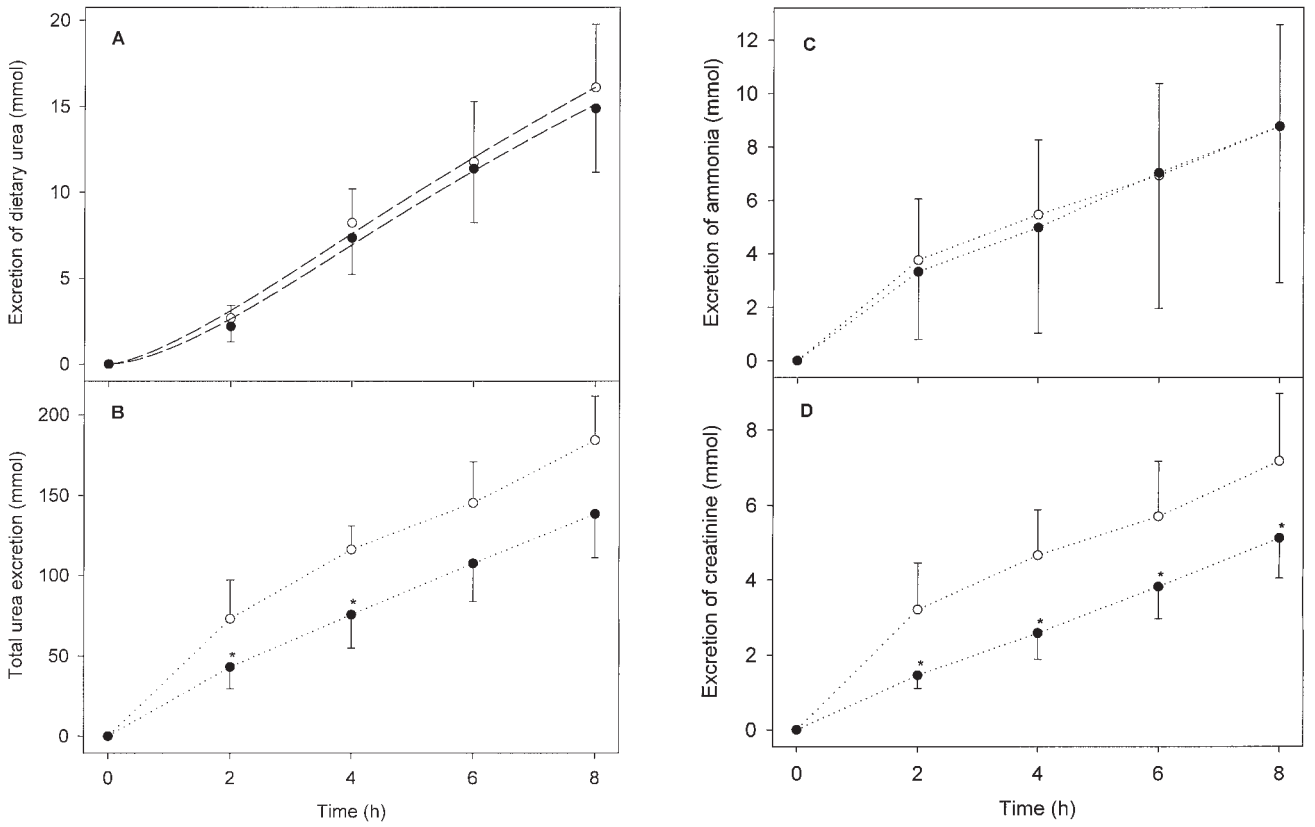

FIGURE 4. A. Mean (±SD) cumulative urinary excretion of dietary urea nitrogen after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. Neither the meal effect nor the meal-by-time interaction was significant. B. Mean (±SD) cumulative urinary excretion of total urea after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. Both the meal effect and the meal-by-time interactions were significant, $P < 0.05$. *Significantly different from the protein meal, $P < 0.0125$ (0.05/4; Bonferroni adjustment for multiple comparisons). C: Mean (±SD) cumulative urinary excretion of total ammonia after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. Neither the meal effect nor the meal-by-time interaction was significant. D: Mean (±SD) cumulative urinary excretion of creatinine after the ingestion of a liquid protein meal without (○) and with (●) guar gum, $n = 7$. Both the meal and the meal-by-time interaction were significant, $P < 0.05$. *Significantly different from the protein meal, $P < 0.0125$ (0.05/4; Bonferroni adjustment for multiple comparisons).

excretion was 41% lower 2 h after the PG than after the P meal. Thereafter, urea excretion appeared similar between meals. This difference in total urea excretion between meals came from the 42% reduction in endogenous urea excretion at 2 h. Four hours after the meals, but not later, cumulative total urea excretion continued to be significantly different. The same profile was seen with the cumulative excretion of creatinine (Figure 4D). The type of meal consumed also had a significant effect on both the extent and profile of excretion. The cumulative excretion of creatinine after subjects consumed the PG meal was always lower than it was after the subjects consumed the P meal. There was a reduction of 54% in urinary excretion for the 0–2-h period; however, the cumulative urinary excretion of ammonia (Figure 4C) did not differ between meals and was still very similar ($P > 0.99$) 8 h after ingestion.

DISCUSSION

The aim of the present study was to investigate the acute effects of guar gum on the bioavailability and utilization of dietary protein and to consider the possibility that guar gum might acutely modulate urea kinetics in humans. The viscosity of the meal was increased 100-fold, probably even more after ingestion because guar-gum-induced viscosity may develop in the stomach and may resist dilution and reneutralization more than viscosity induced by other gums (29). Moreover, although the inclusion of moderate amounts of guar gum in a meal often failed to markedly modify gastric emptying (30–32), it does modify postprandial glucose concentrations after a carbohydrate meal. For instance, identical or even small amounts of guar (≤1% wt:vol) markedly lowered plasma insulin and glucose concentrations and strongly affected single amino acid tolerance tests (24, 25, 29).

An important result was that the absorption, systemic availability, and utilization of dietary nitrogen remained unchanged after adding guar gum to a protein meal. First, there was no delay in the systemic availability of dietary amino acids after the PG meal, as assessed from ^{15}N amino acid enrichments in plasma. Thus, neither the intestinal digestion nor the absorption of dietary nitrogen was delayed by the addition of guar gum. Second, both plasma and urinary dietary urea remained remarkably unaffected by the consumption of guar gum, showing that dietary nitrogen was deaminated to the same extent and at the same speed. This result confirmed that the similarity between systemic dietary amino acid availability after P and PG meals did not result from changes in absorption, splanchnic extraction, or

peripheral utilization. Our results contrast with those of previous studies in which guar gum blunted the postprandial appearance of a single amino acid added to the meal (24, 25) and with some nitrogen balance studies in animals that reported changes in nitrogen utilization (33). In a mixed meal, the effect of guar gum on starch digestibility, insulin release, and portal or systemic glucose availability may modify the utilization of dietary nitrogen. However, mixing protein with other nutrients is unlikely to affect the absorptive balance of dietary nitrogen (34). Furthermore, the nitrogen balance method is not very sensitive to changes in bioavailability or utilization because it is often obscured by adaptive phenomena (35). This is particularly true of soluble fiber intake, which leads to profound changes in the nitrogen excretion pattern and can hamper the interpretation of dietary nitrogen bioavailability, efficiency of utilization, or nitrogen balance (8, 23, 33). The present study therefore enabled us to reach a clearer conclusion than what was known before, showing that guar-gum-based viscosity did not impair the digestibility and efficiency of postprandial dietary nitrogen utilization and could only indirectly affect the efficiency of dietary nitrogen utilization, ie, through its effect on postprandial glucose.

Although dietary nitrogen availability was unaffected and its utilization appeared to be remarkably unchanged, the urinary excretion of endogenous urea was dramatically reduced during the first 2 h postprandially, the difference being even greater when corrected for endogenous body urea variations. This major discrepancy in urinary urea was seen only for the endogenous urea and for the first 2-h period after the meal, whereas striking similarities were observed in all other respects. The possibility that this effect could originate from alternative endogenous urea production can be ruled out because postprandial endogenous and dietary amino acids metabolism, which are physiologically linked, are unlikely to exhibit highly dissimilar trends (34). Any increased utilization of endogenous amino acids for protein synthesis or modulation of endogenous protein catabolism would certainly have affected the utilization of dietary amino acids, which are at maximal absorption during this period. The only remaining alternative to explain this discrepancy is that some endogenous urea was lost by extra urinary routes soon after meal ingestion, when hardly any urea had been produced from dietary sources. Although many uncertainties exist concerning the site of urea disposal (hydrolysis of which is ≈20–25% of urea production in humans), many authors have suggested that the upper digestive tract (ie, the stomach and principally the small intestine) may be the major site for urea secretion (1, 36–40). Some studies reported that viscous fiber increases the gastric or intestinal secretion of fluids, specifically nitrogen, which indicates that guar gum may activate the upper intestinal disposal of urea (18, 19, 22, 41). Consistent with this idea, the present results suggest that the principal difference in $^{13}CO_2$ breath excretion occurred during the 0–2-h burst of ^{13}C excretion. This means that the minor difference in ^{13}C excretion between meals arose from a short delay in the initial rapid emptying phase of the stomach, probably because the effect of viscosity initially produced by the guar gum was reduced shortly after gastric secretion was activated (18, 19).

Viscous polysaccharides are known to increase intestinal water content (11); specifically, it was observed that the addition of 5 g guar gum to a meal doubled the water output in persons with ileostomies (20). This water-holding capacity of many such fibers is understood to improve fermentability in the large intestine by increasing microorganism access to food residues (6, 42). Morgan et al (43) reported that the addition of guar gum to a protein meal reduces gastric inhibitory polypeptide secretion and enhances protein-simulated gastrin secretion without modifying gastric emptying of the liquid phase. It is therefore probable that the presence of guar gum in the lumen of the upper gastrointestinal tract acutely promoted urea disposal in the proximal intestine. During long-term supplementation with nonstarch polysaccharides, augmentations in urea recycling are thought to be caused by changes in colonic urea removal after energy-yielding fermentation of nonstarch polysaccharides in the colon (5). This is a long-term mechanism, but it is possible that direct urea transport to the colon might also be increased (21). Despite this marked dissimilarity in urinary excretion, we note that no effects occurred at the plasma level. It is well established that a protein load induces a transient increase in renal hemodynamics and filtration rate (44), although the mechanism is yet to be identified (45). In the present study, ingestion of a liquid protein meal rapidly and transiently activated the excretion of urea and creatinine. On the other hand, the effect appeared to be blunted by guar gum. The reason for this is unknown, but it is consistent with the potential effects of guar gum on the intestinal removal of water, urea, or other solutes. Therefore, low urinary urea excretion appeared to approximate the diversion of urea to the intestine, whereas the endogenous plasma urea concentrations remained steady.

Finally, if substantial amounts of urea are directed to the intestine as an immediate response to guar gum, as suggested by the present results, the involvement of guar gum in enhanced urea disposal and salvage seen with long-term rises in nonstarch polysaccharide intake should be examined further. It has been suggested that consuming fermentable fiber may considerably benefit individuals with established or developing renal failure because the fiber reduces the amount of work required by the kidneys to excrete nitrogen and may enhance the salvage of urea nitrogen in persons who are usually placed on low-protein diets (46). The present findings show similar implications, yet are based on an acute effect of guar gum. We showed that the addition of a viscous gum can lower endogenous urinary excretion immediately after a protein meal, probably through an early rise in the intestinal disposal of endogenous urea. Furthermore, this occurred without affecting the bioavailability and postprandial utilization of dietary nitrogen and therefore may be of significant value for individuals consuming diets with only marginally adequate protein content, either spontaneously or for therapeutic reasons. These data should encourage further studies to determine the influence of fiber-induced luminal perturbations on the peripheral kinetics of both urea and other metabolites of clinical importance.

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