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Nutrient Intake From Habitual Oral Diet in Patients With Severe Short Bowel Syndrome Living in the Southeastern United States

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Abstract

Background and Aims—Little data are published on habitual home oral diet of short bowel syndrome (SBS) patients living in the United States.

Methods—We assessed habitual macro-and micronutrient intake from oral food and beverages in 19 stable patients with severe SBS who live in the Southeastern United States. Intestinal absorption of energy, fat, nitrogen (N) and carbohydrate (CHO) was determined in a metabolic ward setting.

Results—We studied 12 women and 7 men, age 48±3 years (mean±SE) receiving chronic PN for 31±8 months following massive small bowel resection (118±25 cm residual small bowel). Patients had intact (N=5), partial (N=9), or no residual colon (N=5). The subjects demonstrated severe malabsorption of energy (59±3% of oral intake), fat (41±5%), N (42±5%) and CHO (76±3%). Average oral energy intake was 2656±242 kcal/day (39±3 kcal/kg/day) and oral protein intake was 1.4 ±0.1 g/kg/d. Oral food/beverage intake constituted 49±4% of total (enteral + parenteral) daily fluid intake, 66±4% of total daily kcal and 58±5% of total daily N intake. Oral fat intake averaged 92±11 g/day (≈ 35% of total oral energy). Oral fluid intake averaged 2712±240 ml/d, primarily from water, soft drinks, sweet tea and coffee. Simple sugars comprised 42±3% of oral CHO intake. Usual dietary intake of multiple micronutrients were below the Recommended Dietary Allowances (RDA) in a large percentage of patients: vitamin A (47%), vitamin D (79%), vitamin E (79%), vitamin K (63%), thiamine (42%), vitamin B6 (68%), vitamin B12 (11%), vitamin C (58%), folate (37%), iron (37%), calcium (63%), magnesium (79%) and zinc (68%). Only 7 patients (37%) were taking oral multivitamin-mineral supplements and only 6 subjects (37%) were taking oral iron and calcium supplements, respectively.

Conclusions—In these SBS patients living in the Southeastern United States, oral diet provides a significant proportion of daily nutrient intake. However, the types of foods and fluids consumed are

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likely to worsen malabsorption and increase PN requirements. Oral intake of essential micronutrients was very low in a significant proportion of this cohort of SBS patients.

Keywords

Short bowel syndrome; diet; vitamins; minerals; intestinal rehabilitation

Short bowel syndrome (SBS) after massive small intestinal resection is characterized by significant diarrhea, malabsorption and weight loss. Such individuals typically continue to eat regular meals, but often require parenteral nutrition (PN) to maintain adequate nutritional and hydration status. Unfortunately, PN is associated with serious complications, including catheter infections and sepsis, venous thrombosis and liver failure (1–5). The etiology and severity of SBS and the presence or absence of residual colon also appears to be important factors for prognosis (1). In recent years, methods of intestinal rehabilitation designed to achieve intestinal autonomy and decrease the need for PN in SBS patients have been studied, in addition to work in translational animal models of SBS (6–17). The approaches used in human SBS include intensive and individualized dietary modifications (6–8), administration of specific nutrients and growth factors [e.g. glutamine, glucagon-like peptide-2 (GLP-2) and growth hormone (GH)] (9,15,16) and combinations of diet, glutamine and GH therapy (6,13,14,17). Intestinal adaptation after massive small bowel resection clinically occurs in humans, as evidenced by clinical observations of decreased diarrhea and apparent improvement in nutrient absorption in individual patients during the first few years after bowel resection (2,7). Animal models of SBS clearly show that composition of the diet plays an important role in facilitating adaptive growth (crypt depth, villus height, cell proliferation) of the residual small bowel mucosa after massive small bowel resection (10–11); however, there is little or no evidence for structural small bowel adaptation in humans with SBS (10).

The presence of luminal nutrients also upregulates specific nutrient transporters and digestive enzymes, increases splanchnic blood flow, pancreatic-biliary secretions, gut neuronal activity and peristalsis and stimulates local production and release of gut-trophic growth factors, such as insulin-like growth factor-I (IGF-I) (10). These and other as yet unrecognized effects of luminal food may enhance adaptive growth of residual bowel mucosa and ultimately decrease the need for PN in human SBS. Unfortunately, little data are available on the effects of diet on adaptive intestinal mucosal growth in human SBS (10,13,16,20,21). With regard to the effects of PN, one study showed that use of PN without enteral food in healthy adults induced small bowel mucosal atrophy that was reversed by enteral tube feeding (19). A small case series in PN-dependent patients demonstrated small bowel villus atrophy during PN alone that was also reversed after a period of enteral refeeding (20). Levy et al showed that continuous in-patient enteral feeding (by tube and oral food) for several weeks immediately after massive small bowel resection in children and adults clinically enhanced gut adaptation as manifested by PN dependence (21). These studies demonstrate that small bowel mucosal growth and/or function in the human intestine, as in animal models, is dependent upon food in the intestinal lumen.

Intestinal rehabilitation programs typically incorporate individualized modification of oral diet is a cornerstone of therapy, in addition to optimization of anti-diarrheal medications, particularly as use of PN is weaned (6,17). Dietary modifications typically involve smaller, more frequent feedings, use of oral rehydration solutions and soluble fiber, avoidance of simple sugars, oxalate, and, in some cases, lactose and modification of fat intake and consumption of specific foods based on individual tolerance (2,6–8). Based on clinical observations and limited experimental evidence, the type and pattern of oral food/fluid intake can dramatically affect the overall nutritional status and stool output volume of SBS subjects (7–8). For example, consumption of high amounts of simple sugars, hyperosmotic fluids and sometimes water worsens fluid losses and nutrient absorption (7–8). Initial small studies in patients with SBS

found no difference in intestinal losses and nutrient absorption with modifications in diet composition (22,23). Recent studies with larger groups of patients clearly show that patients with residual colon require a different diet composition than those without colon, in that these individuals benefit from diets generally lower in fat content and high in complex carbohydrates via bacterial generation of short chain fatty acids (SCFA), whereas those with ileo- or jejunostomies do not generally respond to fat restriction (7–8,24,25,26,27,28,29).

Generalized malnutrition and certain micronutrient deficiencies such as vitamin A, vitamin E or zinc, may also impair intestinal adaptation and thus worsen diarrhea in SBS (10–11). Micronutrient deficiencies in SBS population may exist even if patients are able to maintain energy and protein balance, and hyperphagia may also contribute to excessive losses of micronutrients and fluids via the worsening diarrhea that may ensue (30). In addition, fluid, sodium and magnesium balance are important challenges in managing the dietary intake of SBS patients without a colon (27–29). Therefore, intensive and adequate nutrition counseling is essential for intestinal rehabilitation and PN independence in patients with SBS. Unfortunately, little data are available on *habitual* nutrient intake and supplement use in SBS subjects, and there are no comprehensive data reported on SBS patients in regions of the United States. The aim of this study was to assess usual micro- and macronutrient intake from food sources of patients living in the Southeastern United States with severe SBS prior to entry into a bowel rehabilitation program.

METHODS

Patients

All patients gave written informed consent for participation in the study approved by the Human Investigations Committee of Emory University, Atlanta. The patients in this report were referred from outside primary care physicians and admitted to the General Clinical Research Center (GCRC) at Emory University Hospital, Atlanta for a randomized, double-blind study of diet modification $\pm$ recombinant GH. For this report, we studied 19 patients (7 males, 12 females) with clinically and metabolically stable SBS requiring intravenous hydration and/or PN, with a mean age of 48 ± 3 years (range, 29 to 72 years) currently living in the Southeastern United States (Table 1). The 19 subjects lived in Alabama, Arkansas, Georgia, North Carolina, South Carolina and Tennessee, respectively. Their mean body mass index (BMI) was 23.8 ± 0.6 (range, 19.2 to 27.8). Twelve patients were receiving PN 7 days a week and seven patients were on chronic PN $\pm$ intravenous hydration fluids from 2 to 5 days weekly. The average duration of PN in the 19 subjects was 31 ± 8 months (range, 3 to 136 months).

The indications leading to massive intestinal resection are given in Table 1. The major causes of SBS were massive net small bowel $\pm$ colonic resection due to mesenteric ischemia ($n=6$), Crohn's disease ($n=5$), bowel obstruction and fistula ($n=5$), and abdominal trauma ($n=3$). All subjects had an intact stomach and duodenum, and 14 had in-continuity complete or partial residual colon. The other 5 patients studied had ileo- or jejunostomies and no residual colon in continuity. The residual bowel length was estimated from operative reports and small bowel barium-contrast studies. The average length of the remnant jejunum-ileum was 118 ± 25 cm. Patients with colon had a shorter remaining small bowel (71 ± 16 cm) compared to patients without any residual colon (248 ± 53 cm; $P<0.05$). The average time interval from the last intestinal surgery to study entry was 31.2 ± 8.8 months (range, 3 to 136 months). All patients were clinically stable, ambulatory, well nourished and on a stable PN/hydration and oral diet regimen for at least two months prior to study. PN was cycled overnight in all of the chronic PN-requiring patients.

Patients with Crohn's disease had no evidence of active disease or disease flare for at least 6 months before entry. Patients with uncontrolled diabetes, renal or hepatic dysfunction, history of cancer, AIDS, congestive heart failure, severe ischemic heart disease, active infectious illness, steroid-dependent chronic obstructive lung disease, colonic polyps or adenomas, rheumatoid arthritis, alcoholism, drug abuse or pregnancy were excluded from the study.

Study Protocol

A complete medical history and physical examination was performed on the admission day. Blood was drawn for fasting baseline values the morning after admission. Daily body weight, total parenteral and oral fluid intake and 24-hour output of urine, stool and emesis were recorded during the entire 28-day GCRC admission. The data reported here are derived from the initial 7-day baseline period in the GCRC, during which the patients received their usual home PN regimen and oral dietary and fluid intake and feeding pattern. The composition of the habitual oral diet was based upon the dietary analysis of a 7-day food recall instrument mailed to and completed by the patients in the week prior to admission. During the initial 3-day equilibration period on the GCRC, the registered dietitian Director of the GCRC Bionutrition Unit (KU) reviewed and adjusted the diet record according to detailed one-on-one discussions with each patient to determine actual intake as closely as possible. Analysis of the food records was performed using the GCRC Nutritionist IV software program.

Patients were maintained on their usual anti-diarrheal agents (3 of the 19 subjects were not taking anti-diarrheal agents due to perceived lack of efficacy), vitamin-mineral supplements and other medications. Patients were given intravenous electrolytes as indicated to maintain serum levels within the normal range and monitored as clinically indicated.

Nutrient absorption studies

Intestinal absorption of energy, macronutrients and minerals was studied in all patients. After the 3-day equilibration period, a 4-day intestinal nutrient absorption study was performed. Meals were prepared in duplicate and the second 24-hour diet was blended and aliquoted for later analysis. Refused food, stool, emesis and ostomy outputs were collected, pooled and maintained refrigerated for four days. At the end of the collection period, the pooled stool and refused food samples were individually homogenized and analyzed for nitrogen, sodium, potassium, calcium, phosphorus and magnesium concentrations using established methods of the GCRC Metabolic Core Laboratory. Aliquots were also saved at -70°C for future analysis of fat and energy. Mineral and electrolyte content of any ingested oral supplements was added to the intake amounts for these substances. Nitrogen content was determined by micro/Kjedahl digestion. Sodium and potassium content were analyzed by flame photometry after mixing the samples with acid using the wet ash method; calcium and magnesium content were estimated by atomic absorption in acidified, ashed samples and phosphorus content was determined by the Fiske method, respectively. Fat and energy measurement were performed by acid hydrolysis and bomb calorimetry, respectively, by Covance laboratories (Madison, WI). The total amount of carbohydrate in the diet and stool samples was calculated by subtracting fat and nitrogen calories from total calories and dividing by four (~ 4 kcal/g). The percent of nutrient absorption was calculated as $[(\text{oral intake} - \text{intestinal output})/\text{oral intake}] \times 100$.

D-Xylose absorption test

A D-xylose absorption test was performed on the morning following the 4-day stool collection to further assess intestinal malabsorption in these patients. After an overnight fast, patients ingested 25 grams of D-xylose in 250 ml of water orally. D-xylose was measured in serum at baseline and 2 hours after ingestion and in a 5-hour urine collection. D-xylose concentration in serum and urine was determined by a standard photometric method.

Statistical analysis

All data are presented as mean $\pm$ standard error of the mean (SEM). The paired student t-test or the Mann Whitney U-test was used as appropriate to determine associations between categorical and continuous variables. Regression analysis was utilized to correlate continuous variables. Data were also analyzed as a function of gender and presence or absence of residual colon. P values < 0.05 were considered statistically significant.

RESULTS

Intestinal absorption capacity

The study subjects in general demonstrated significant stool losses of energy, fat, nitrogen, carbohydrate, fluid and electrolytes, documenting SBS-induced malabsorption in all individuals (Table 2). Malabsorption was especially severe for fluids, although this endpoint demonstrated considerable variability among patients ($18 \pm 10\%$, range, -104 to $+82\%$). Nitrogen and fat absorption were very similar, $42 \pm 5\%$ and $41 \pm 5\%$, respectively, while carbohydrates were better absorbed in this SBS cohort $76 \pm 3\%$. The percentage of fluid absorption was positively correlated with energy, fat and CHO absorption (all $P < 0.05$). Nitrogen and potassium absorption were positively correlated with length of residual small bowel ($R = 0.55$ and 0.718 , respectively, $P < 0.02$). Patients without residual colon had better potassium absorption than those with residual colon (62 ± 8 versus $29 \pm 7\%$, $P < 0.02$). Most patients demonstrated severe malabsorption of calcium and magnesium and some subjects exhibited a secretory component. Average calcium absorption was $-7 \pm 8\%$ (range, -72 to $+39\%$) and magnesium absorption was $2 \pm 8\%$ (range, -111 to $+45\%$). The presence of residual colon did not influence the percentage of calcium and magnesium absorption.

The mean 2-hour serum D-xylose concentration was 21 ± 3 mg/dL, clearly below the normal range of between 32 and 58 mg/dL. Only three patients had plasma D-xylose values within normal parameters, although these individuals also had decreased urinary D-xylose excretion. In healthy individuals younger than age 65 with normal intestinal transit and absorptive function, D-xylose content in a 5-hour urine collection ranges between 4 and 10 g. In our SBS population, urinary D-xylose excretion averaged 4.2 ± 1.3 g, and was below normal in 16 patients (84%). There was no significant correlation, however, between the D-xylose indices and small bowel length or the absorption of specific macro- or micronutrients.

Dietary macronutrient intake

Based upon data derived from the 7-day dietary recall, our SBS patients reported an average habitual food intake of 2193 ± 216 kcal per day, although there was considerable variability among individuals, ranging between 721 and 4126 kcal daily (Table 3). Most patients (14/19 or 74%) underreported their average 7-day energy intake on the 7-day diet recall form; after detailed one-on-one review with the GCRC nutritionist co-investigator on the GCRC, actual energy intake from food was determined to be 2656 ± 242 kcal/day or 39 ± 3 kcal/kg/day (Table 2). The 14 patients with residual in-continuity colon had energy intake of 2501 ± 304 kcal/day compared to intake of 2942 ± 195 kcal/day in 5 subjects without residual colon (endjejunostomies or ileostomies); NS between these SBS subgroups. We did not observe any difference between genders and no correlation between the difference in reported and actual intake with BMI or energy intake. Underreporting in energy intake was due to a lower report of carbohydrate intake (diet recall form 276 ± 28 versus one-to-one interview 346 ± 31 g/day; $P < 0.05$). There was no statistically significant difference between daily protein and fat consumption reported with the diet recall instrument and the revised data obtained after the personal interview by the dietitian (nitrogen 14 ± 6 versus 17 ± 2 g/day and fat 83 ± 10 versus 92 ± 11 g/day, respectively; NS). Gender, BMI or total energy intake had no influence on dietary under- or over-reporting of macronutrient intake. Similarly, there was no difference

in daily nitrogen or fat intake between subjects with or without residual colon, respectively [nitrogen intake 15.5 ± 2.1 versus 17.9 ± 2.5 g/day; (NS); fat intake 90 ± 14 versus 95 ± 18 g/day; (NS)].

Based upon the 7-day dietary record, protein provided $17 \pm 1\%$, carbohydrates $51 \pm 2\%$ and fat $32 \pm 2\%$ of the total enteral calorie intake. Despite the differences in total energy and carbohydrate consumption between dietary record data and actual intake determined after review with the nutritionist, the percentages of energy provided by macronutrients were similar. During the nutrient absorption studies, protein provided 15% of total energy intake whereas carbohydrates and fat supplied 53% and 31% respectively. There were no significant differences in the reported sources of energy intake between genders or with the presence of colon (not shown).

Mean daily protein intake was 1.4 ± 0.1 g/kg/day (0.22 ± 0.02 g N/kg/day). Reported oral protein consumption was higher in males than in females, 112 ± 12 versus 75 ± 12 g/day, $P < 0.05$, but the difference was not significant when intake was corrected for body weight (males, 1.6 ± 0.2 versus females, 1.2 ± 0.1 g/kg/day; $P = \text{NS}$). The major sources of protein in the oral diet were red meat and meat products; consumption of fish and dairy products was qualitatively low. Total carbohydrate intake reported by our SBS patients was 276 ± 28 g/day (range, 76 to 500 g/day). Approximately 43% of oral carbohydrates were deemed to be simple carbohydrates by the nutritionist co-investigator. There were no differences in % carbohydrate intake as simple carbohydrates between subjects with or without residual colon was 43 ± 4 versus $37 \pm 6\%$, respectively (NS). The major food sources of simple carbohydrates were sugar, candies, juices and soft drinks. Because of low consumption of fruits and vegetables, fiber intake averaged 14 ± 1.7 grams per day, well below general recommendations for non-SBS patients. Reported fat intake averaged 83 ± 10 g/day (range, 22 to 170 g/day), adjusted to 92 ± 11 g/day after review by the research nutritionist; this provided $35 \pm 2\%$ of total oral food energy intake. The major reported sources of dietary fat were fried foods, butter, margarine, high fat meats and baked goods—all items common in the typical American diet.

Patients reported lower mean daily fluid consumption with the 7-day diet recall instrument (1766 ± 220 ml/day; range 457 to 4170 ml/day) than determined after personal interview with the research nutritionist (2712 ± 240 ml/day; range 1151 to 5748 ml/day; $P < 0.05$). The major sources of oral fluid intake were water (28%) and coffee (26%) followed by sodas or fruit juices (20%) and sport drinks (18%). Eleven patients reported drinking milk daily providing 8% of total oral fluid intake. Only one of our subjects was following a systematic program of oral re-hydration fluid therapy. A significant proportion of total macronutrient intake was provided by oral diet in these individuals with generally chronic PN-dependent SBS. Oral diet and fluid intake provided $49 \pm 4\%$ of total (enteral + parenteral) fluid intake, $66 \pm 4\%$ of total kcal intake and $58 \pm 5\%$ of total N intake (Table 3).

Oral protein and kcal intake negatively correlated with age ($P < 0.05$) but did not correlate with PN days/week, intravenous kcal or N intake, or macronutrient or fluid absorption. Thus, intravenous kcal or nitrogen intake did not appear to influence appetite, as estimated by habitual oral kcal or protein intake in our SBS patient cohort.

Dietary micronutrient intake

Based upon the 7-day dietary record data as revised by the GCRC nutritionist, we detected a high proportion of patients with intake of fat soluble vitamins for age and sex below the Recommended Dietary Allowances (RDA) of the Food and Nutrition Board, National Research Council, in patients with or without colon in continuity (Figure 1) (31). None of the 5 patients with end-jejunostomies or ileostomies and no residual colon reported below-RDA intake for vitamin B12, folate or iron (Figure 1). The low overall intake of food-derived fat-soluble

vitamins was probably related to low intake of dairy products (vitamin D intake was below RDA in 79%), low intake of fat (vitamin A, below RDA intake in 47% and vitamin E, below RDA intake in 79%) in some patients and low consumption of green leafy vegetables (vitamin K, below RDA intake in 63%). We also observed a high percentage of SBS patients overall with habitual dietary intakes below the RDA for thiamine (vitamin B1; 42%), vitamin B6 (68%), vitamin C (58%) and folate (37%) (Figure 1). Dietary consumption of vitamin A, B1, B6 and vitamin K was significantly higher in men than in women ($P<0.005$), probably because of higher total energy and fat intake. Only 7 of our patients (37%) were taking oral multivitamin or multivitamin-mineral supplements, and 4 of the patients who received PN less than 7 days a week did not take such oral multivitamin-mineral preparations. Low dietary iron intake was observed in 7 of 19 patients (37%); all in individuals with in-continuity colon. Only six patients (32%) were taking oral iron supplements and calcium supplements, respectively.

Reported dietary intake of sodium and potassium varied markedly among the study patients as a group. Mean sodium intake was 3563 ± 355 mg/day, but ranged between 301 and 6087 mg/day. Oral sodium intake was positively correlated with total oral energy, protein and potassium intake (each $P<0.05$). Reported daily potassium intake was 2404 ± 206 mg/day (range, 882 to 4088 mg/day). Oral potassium intake was positively correlated with total oral energy, protein and fat intake (each $P<0.05$).

We observed below-RDA dietary calcium intakes in 12/19 (63% of SBS patients overall) of patients and, as noted above, 79% of patients were consuming vitamin D at below-RDA levels (Figure 1). In general, the patients had very low intake of milk and other dairy products. In many patients, lactose intolerance appeared to be present by detailed questioning, but patients were typically tolerant to cheese and lactose-free dairy products. Only five patients were taking oral calcium supplements. In 7 individuals, oral multivitamins provided a small amount (162–324 mg/day) of additional calcium.

Dietary magnesium and zinc intakes were below recommendations in 79% and 68% of patients, respectively, related to poor consumption of green vegetables, dairy products, nuts and red meats, which, in addition to cereals, are major dietary sources. Only one patient was taking oral magnesium supplementation and none was taking oral zinc; oral multivitamin preparations provided a very small amount of magnesium and zinc in 6 patients.

DISCUSSION

Previous studies indicate that intensive and individualized nutrition and diet counseling can facilitate intestinal rehabilitation and PN independence in many patients with severe SBS (6–8). This small group of SBS patients who live in the Southeastern United States habitually follow diets that are deficient in many essential micro- and macronutrients, including protein, calcium, iron and lipid soluble vitamins. In addition, patients make food and beverage choices that are likely to worsen their diarrhea and increase PN requirements. Personal communication revealed that each subject had received little previous dietary instruction from their primary physicians. Further, most patients had not received specific instructions from their primary physicians on appropriate timing of use of anti-diarrheal medications (typically loperamide or diphenoxylate hydrochloride/atropine sulfate) in relation to meals and also were taking low doses of these agents.

No patient exhibited classical dermatologic or other clinical evidence of micronutrient deficiency. We did not measure levels of water-soluble vitamins in this SBS patient cohort and assume that intake of micronutrients on PN days with infusion of conventional vitamins and minerals, combined with habitual food intake was probably adequate. The purpose of this study was to examine habitual nutrient intake from food sources in subjects entering a bowel

rehabilitation program. We feel that the data presented here will be useful for clinicians caring for SBS patients and for investigators interested in intestinal rehabilitation research. The information on dietary habits in SBS patients, at least in this part of the U.S., can help guide such programs as patients are weaned from PN and must rely on diet and oral supplements alone.

Our study group's poor intestinal absorption reflects the severity of their underlying malabsorptive syndrome and their PN dependency, especially for fluids and electrolytes. The net intestinal macronutrient absorption values in our subjects are within the range of those reported in other studies of PN-dependent SBS patients (12–16). This broad range of nutrient absorption is related to the remaining small bowel length but also to the underlying disease and the anatomy of remnant intestine. These data also illustrates the need for individualized assessment of intestinal function, diet, and enteral and parenteral nutritional support in patients with SBS. In addition, while most

In our population, the presence of residual colon did not appear to significantly influence gastrointestinal absorption of fluids, macronutrients or minerals, except for lower potassium intestinal absorption in patients with colon. We confirmed, as expected, a negative correlation between PN requirements and length of remaining small bowel. It is possible that the small number of patients without residual colon in our subjects, did not allow statistical significance, but the lack of influence of colon on absorption of most oral nutrients in our study may also be because increased stool losses related to improper habitual diet interferes with nutrient absorption. As has been previously suggested, consumption of a non-physiologic diet pattern or habitual use of specific food items such as simple sugars, juices etc. may worsen patient's malabsorption and thus interfere with potential benefits of anti-diarrheal medications and other strategies of intestinal rehabilitation (8).

The D-xylose test is a standard test to detect malabsorption caused by multiple diseases (32). Low plasma D-xylose concentrations have been described in patients with short bowel syndrome (13,32–33). Gouttebel et al found a negative correlation between 2-hour serum Dxylose levels and length of remaining small bowel length in patients with SBS for less than 2 year, but not in the group of more chronic patients more than 2 years after massive small bowel resection (33). In our study, we did not find a significant correlation between plasma or urine D-xylose values and remaining small bowel length or the absorption of specific nutrients. Data from D-xylose testing are also influenced by intestinal transit time or bacterial overgrowth, but the test cannot differentiate these factors (32). Thus, the D-xylose test appears to offer poor specificity to assess the degree of intestinal failure in individual SBS patients and therefore, to estimate nutritional requirements and potential to achieve intestinal autonomy.

The high usual food intake of many of our subjects demonstrates that spontaneous hyperphagia occurs in SBS, whereas other patients demonstrated a low energy and protein intakes that would be inadequate to maintain their body weight even in the absence of severe intestinal failure. This variability in intake in SBS patients has been previously observed. Woolf et al reported spontaneous intake of 31 ± 3 kcal/kg/day (range, 16 to 38 kcal/kg/day) in 10 Canadian patients with SBS, only 2 of whom were PN-dependent (34). Ovesen reported habitual oral calorie intake of 5 PN-dependent SBS patients with a jejunostomy living in the United States to average 1900 kcal/day (range 600 to 3300 kcal/day), intake similar to our study subjects (Table 3) (30). In contrast, Messing et al. recorded hyperphagic spontaneous oral intake of 57 ± 15 kcal/kg/day in 10 French patients, 5 of them on chronic PN (35). Crenn et al. reported a similar habitual oral intake of 47 ± 15 (SD) kcal/kg/day in 90 French SBS patients, 39 of who received chronic PN (36). Ellegard reported usual oral caloric intake to be 63 kcal/kg/d (range, 38 to 103 kcal/kg/day) in 10 Swedish SBS patients, 2 of whom received chronic PN and the others only intravenous fluids (12). Whereas Messing et al and Crenn et al (35–36) did not find any

difference in oral intake between patients with or without PN, Jeppesen observed higher oral energy intake in 44 Danish SBS patients not dependent on PN (median ~ 2780 kcal/day) compared to that of 45 PN-dependent patients (~ 1800 kcal/day) (37). Our PN-dependent subjects spontaneous oral intake averaged 2656 ± 242 kcal/day. The study subjects were not previously followed by the investigators in our intestinal rehabilitation unit and had not, in general, received detailed or follow-up dietary instruction. Differences in habitual oral energy intake reported between these studies likely relate to the dietary instruction received by the patients and their underlying clinical conditions and residual bowel anatomy. None of the 5 patients with no residual colon reported below-RDA intake for vitamin B12, folate or iron, possibly due to the numerically increased daily oral kcal reported (NS; see above).

Hyperphagia is necessary for many SBS patients to avoid PN-dependency (35–37). PN also appears to induce satiety in some individuals. However, gastrointestinal symptoms, including exacerbation of diarrhea, anal burning associated with diarrhea, bacterial overgrowth and/or intermittent partial small bowel obstruction make it difficult for many patients to consume the enormous amount of calories necessary to maintain body weight without PN. In addition, there is large individual variation in tolerance to oral lactose, hypotonic fluids and individual food groups and specific food items (2,8,38). Because of the great variability in nutrient absorptive function in SBS patients, dietary counseling must be approached individually and with close follow up of intestinal function changes, diet tolerance and response to optimization of antidiarrhea medications.

In patients with residual in-continuity colon, diets high in complex carbohydrates may help to salvage extra calories (≈ 800 – 1100 kcal/day in some studies) derived from the SCFA acetate, butyrate, propionate that are produced by bacterial fermentation of malabsorbed dietary carbohydrate and fiber (24–26,38). High-fat diets in these patients increase losses of fluid and nitrogen, and result in higher fecal loss of energy (22,24). SBS patients without residual colon do not generally respond to a fat-restricted diet with decreased diarrhea volume; a more liberal use of dietary fat is important in these individual for adequate enteral caloric intake during PN weaning (7–8,21–22). In patients with jejunostomy or ileostomies high carbohydrate intake, especially simple sugars, worsen stomal effluents, whereas high-fat diets may increase ostomy fat loss but improve total energy absorption (21,25). Sipping of near-isotonic oral re-hydration fluids with a high content of sodium is essential in patients with jejunostomies, whereas patients with colon and ileostomies may tolerate hypotonic solutions as and additional sodium chloride added to food (2,8).

We observed that the individuals in our study frequently follow a home diet that is incorrect for their intestinal function and clinical needs. For example, there was no difference in fat and carbohydrate consumption whether colon was present or not. In many cases, the home diet was high in fat in patients with colon and low in fat for patients with jejunostomy-ileostomies. Patients' spontaneous intake of isotonic fluids was also very low, and only one subject had received specific instructions regarding consumption of oral re-hydration solutions. Many of our study patients consumed significant daily volumes of beverages with high osmolality and simple sugar content, such as sodas and juices, as well as caffeine, which stimulate diarrhea. In addition, many subjects with jejunostomy-ileostomies consumed large amounts of water, which can worsen diarrhea in these individuals due to electrolyte shifts into the proximal small bowel lumen (7–8). Also, most patients were taking anti-diarrheal medications.

Generalized malnutrition and specific nutrient deficiencies (e.g. zinc, vitamin A, essential fatty acids) blunt adaptive intestinal mucosal growth after massive small bowel resection in animal models of SBS (10–11). Although adaptive gut mucosal growth responses after bowel resection have been very little studied in human SBS (18), such nutrient deficiencies are not uncommon in subjects with SBS, particularly those not receiving PN, and may inhibit the adaptive response

(39). Electrolyte and micronutrient depletion may also contribute to symptoms including muscle weakness and cramping (e.g. selenium, magnesium, potassium) osteoporosis (calcium and vitamin D) and increased diarrhea (e.g. adequate zinc status is important for normal gut mucosal cell regeneration). In this study, we observed that habitual dietary intake of essential micronutrients in SBS patients receiving daily or intermittent home PN is very often below recommendations for general population. A high percentage of the subjects we studied had inadequate intake of fat-soluble vitamins, B-vitamins, vitamin C and folate, and minerals such as iron, calcium, and especially magnesium and zinc. Coupled with increased losses of these nutrients via large stool outputs, these subjects are thus at high risk for micronutrient depletion unless provided parenterally and/or via oral supplementation. We found that avoidance of certain foods that commonly increase diarrhea in SBS, such as dairy products or green vegetables, low general dietary intake and lack of variety in the diet likely accounted for low micronutrient consumption derived from food. It is possible that we have overestimated deficient intake of some nutrients in some patients because of underreporting of energy intake. However, patients appeared to underestimate primarily carbohydrate intake, and therefore, estimated dietary intake of vitamins and minerals in this study are probably very close to actual intake.

Patients (and physicians) often assume that PN covers the daily requirements for minerals and vitamins without taking into account that standard PN micronutrient doses may not cover specific needs, especially when PN is not infused daily. We found that a surprisingly low percentage of our patients were taking oral micronutrient supplements, even on non-PN infusion days. Furthermore, conventional PN may provide inadequate amounts of certain nutrients for the needs of some individuals, such as essential fatty acids (39), vitamin E (40), vitamin D or calcium (2,41). Patients with SBS require high doses of oral calcium, not only for total body function and bone metabolism but also to prevent oxalate absorption and oxalate renal stone formation, and as others and we have observed there is also active secretion of calcium in stools (14,37). Doses of vitamin D and calcium in PN may also be inadequate to prevent vitamin D deficiency and progressive osteopenia, especially in certain groups such as black patients with poor sun exposure. We previously reported a high incidence of vitamin D depletion (low 25-hydroxyvitamin D levels in blood) and bone mineral loss in this patient population (41). Furthermore, magnesium losses with secretory diarrhea may interfere with parathyroid hormone secretion or action and thus may complicate calcium metabolism in these patients. Therefore, it is important to ensure adequate oral supplementation in addition to parenteral supplementation of vitamins and minerals with frequent follow up of biochemical and clinical nutritional markers in SBS patients, particularly during the process of PN weaning and even in patients who are chronically PN-dependent. It is also important for physicians to prescribe the newer multivitamin preparations designed for subjects with malabsorption that contain water-solubilized vitamins A, D, E and K.

Limitations of this study include the small sample size, heterogeneous (but typical) causes of SBS among the subjects, and the lack of data from a control Southeastern U.S. population to enable us to test whether our SBS subject's habitual dietary intake was similar people without SBS inhabiting the same area of the country.

In conclusion, this small group of SBS patients receiving chronic PN followed dietary patterns that are likely to worsen diarrhea, cause malnutrition (or prevent nutritional repletion) and increase parenteral fluid and nutrient requirements. In addition, dietary intake and oral/intravenous supplementation of multiple essential micronutrients was often inadequate, and may blunt the intestinal adaptation process. The patients we studied would clearly benefit from specialized intestinal rehabilitation, which includes thorough and individualized nutritional assessment and counseling. Such rehabilitation includes a PN weaning process and close serial follow-up of plasma nutrient concentrations, reinforcement of appropriate dietary habits and

food choices, assessment of tolerance to specific food and beverage items and use of appropriate vitamin and mineral supplements.

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References

1. Messing B, Crenn P, Beau P, Boutron-Ruault MC, Rambaud JC, Matuchansky C. Long-term survival and parenteral nutrition dependence in adult patients with the short bowel syndrome. *Gastroenterology* 1999;117:1043–1050. [PubMed: 10535866]
2. Buchman AL, Scolapio J, Fryer J. AGA Technical review on short bowel syndrome and intestinal transplantation. *Gastroenterology* 2003;124:1111–1134. [PubMed: 12671904]
3. Howard L, Ashley C. Management of complications in patients receiving home parenteral nutrition. *Gastroenterology* 2003;124:1651–1661. [PubMed: 12761723]
4. O'Keefe SJ, Burnes JU, Thompson RL. Recurrent sepsis in home parenteral nutrition patients: an analysis of risk factors. *Jpen: J Parent Enteral Nutr* 1994;18:256–263.
5. Cavicchi M, Beau P, Crenn P, Degott C, Messing B. Prevalence of liver disease and contributing factors in patients receiving home parenteral nutrition for permanent intestinal failure. *Ann Intern Med* 2000;132:525–532. [PubMed: 10744588]
6. Byrne TA, Persinger RL, Young LS, Ziegler TR, Wilmore DW. A new treatment for patients with short-bowel syndrome. Growth hormone, glutamine, and a modified diet. *Ann Surg* 1995;222:243–254. [PubMed: 7677455]
7. Jeppesen PB, Mortensen PB. Enhancing bowel adaptation in short bowel syndrome. *Curr Gastroenterol Rep* 2002;4:338–347. [PubMed: 12149180]
8. Byrne TA, Cox S, Karimbakas M, Veglia LM, Bennett HM, Lautz DB, Robinson MK, Wilmore DW. Bowel rehabilitation: an alternative to long-term parenteral nutrition and intestinal transplantation for some patients with short bowel syndrome. *Transplant Proc* 2002;34:887–890. [PubMed: 12034223]
9. Scolapio JS, McGreevy K, Tennyson GS, Burnett OL. Effect of glutamine in short-bowel syndrome. *Clin Nutr* 2001;20:319–323. [PubMed: 11478829]
10. Ziegler TR, Evans ME, Fernandez-Estivariz C, Jones DP. Trophic and cytoprotective nutrition for intestinal adaptation, mucosal repair and barrier function. *Annu Rev Nutr* 2003;23:229–261. [PubMed: 12626687]
11. Swartz-Basile DA, Rubin DC, Levin MS. Vitamin A status modulates intestinal adaptation after partial small bowel resection. *JPEN: J Parent Enteral Nutr* 2000;24:81–88.
12. Ellegard L, Bosaeus I, Nordgren S, Bengtsson BA. Low-dose recombinant human growth hormone increases body weight and lean body mass in patients with short bowel syndrome. *Ann Surg* 1997;225:88–96. [PubMed: 8998124]
13. Scolapio JS, Camilleri M, Fleming CR, Oenning LV, Burton DD, Sebo TJ, Batts KP, Kelly DG. Effect of growth hormone, glutamine, and diet on adaptation in short-bowel syndrome: a randomized, controlled study. *Gastroenterology* 1997;113:1074–1081. [PubMed: 9322500]
14. Szkudlarek J, Jeppesen PB, Mortensen PB. Effect of high dose growth hormone with glutamine and no change in diet on intestinal absorption in short bowel patients: a randomised, double blind, crossover, placebo controlled study. *Gut* 2000;47:199–205. [PubMed: 10896910]
15. Jeppesen PB, Hartmann B, Thulesen J, Graff J, Lohmann J, Hansen BS, Tofteng F, Poulsen SS, Madsen JL, Holst JJ, Mortensen PB. Glucagon-like peptide 2 improves nutrient absorption and nutritional status in short-bowel patients with no colon. *Gastroenterology* 2001;120:806–815. [PubMed: 11231933]

16. Seguy D, Vahedi K, Kapel N, Souberbielle JC, Messing B. Low-dose growth hormone in adult home parenteral nutrition-dependent short bowel syndrome patients: a positive study. *Gastroenterology* 2003;124:293–302. [PubMed: 12557135]
17. Byrne TA, Wilmore DW, Iyer K, Dibaise J, Clancy K, Robinson MK, Chang P, Gertner JM, Lautz D. Growth hormone, glutamine, and an optimal diet reduces parenteral nutrition in patients with short bowel syndrome: a prospective, randomized, placebo-controlled, double-blind clinical trial. *Ann Surg* 2005;242:655–661. [PubMed: 16244538]
18. Ziegler TR, Estívariz CF, Gu LH, Wallace TM, Díaz EE, Pascal RR, Bazargan N, Galloway JR, Wilcox JN, Leader LM. Distribution of H⁺/peptide transporter PepT1 in human intestine: up-regulated expression in the colonic mucosa of patients with short bowel syndrome. *Am J Clin Nutr* 2002;75:922–930. [PubMed: 11976168]
19. Buchman AL, Moukarzel AA, Bhuta S, Belle M, Ament ME, Eckhart CD, Hollander D, Gornbein J, Kopple JD, Vijayaraghavan SR. Parenteral nutrition is associated with intestinal morphologic and functional changes in humans. *JPEN J Parenter Enteral Nutr* 1995;19:453–460. [PubMed: 8748359]
20. Pironi L, Paganelli GM, Miglioli M, Biasco G, Santucci R, Ruggeri E, DiFebo G, Barbara L. Morphologic and cytoproliferative patterns of duodenal mucosa in two patients after long-term total parenteral nutrition: Changes with oral refeeding and relation to intestinal resection. *JPEN J Parenter Enteral Nutr* 1994;18:351–354.
21. Levy E, Frileux P, Sandrucci S, Ollivier JM, Masini JP, Cosnes J, Hannoun L, Parc R. Continuous enteral nutrition during the early adaptive stage of the short bowel syndrome. *Br J Surg* 1988;75:549–553. [PubMed: 3134973]
22. McIntyre PB, Fitchew M, Lennard-Jones JE. Patients with a high jejunostomy do not need a special diet. *Gastroenterology* 1986;91:25–33. [PubMed: 3011582]
23. Woolf GM, Miller C, Kurian R, Jeejeebhoy KN. Diet for patients with a short bowel: high fat or high carbohydrate. *Gastroenterology* 1983;84:823–828. [PubMed: 6825993]
24. Nordgaard I, Hansen BS, Mortensen PB. Colon as a digestive organ in patients with short bowel. *Lancet* 1994;343:373–376. [PubMed: 7905549]
25. Nordgaard I, Hansen BS, Mortensen PB. Importance of colonic support for energy absorption as small-bowel failure proceeds. *Am J Clin Nutr* 1996;64:222–231. [PubMed: 8694024]
26. Jeppesen PB, Mortensen PB. Significance of a preserved colon for parenteral energy requirements in patients receiving home parenteral nutrition. *Scand J Gastroenterol* 1998;33:1175–1179. [PubMed: 9867096]
27. Ladefoged K, Olgaard K. Fluid and electrolyte absorption and renin-angiotensinaldosterone axis in patients with severe short-bowel syndrome. *Scand J Gastroenterol* 1979;14:729–735. [PubMed: 119306]
28. Nightingale JM, Lennard-Jones JE, Gertner DJ, Wood SR, Bartram CI. Colonic preservation reduces need for parenteral therapy, increases incidence of renal stones, but does not change high prevalence of gall stones in patients with a short bowel. *Gut* 1992;33:1493–1497. [PubMed: 1452074]
29. Gouttebel MC, Saint-Aubert B, Astre C, Joyeux H. Total parenteral nutrition needs in different types of short bowel syndrome. *Dig Dis Sci* 1986;31:718–723. [PubMed: 3087714]
30. Ovesen L, Chu R, Howard L. The influence of dietary fat on jejunostomy output in patients with severe short bowel syndrome. *Am J Clin Nutr* 1983;38:270–277. [PubMed: 6410899]
31. Food and Nutrition Board, National Research Council. Recommended Dietary Allowances. 10th. Washington DC: The National Academies Press; 1989.
32. Craig RM, Atkinson AJ. D-xylose testing: a review. *Gastroenterology* 1988;95:223–231. [PubMed: 3286361]
33. Gouttebel MC, Saint Aubert B, Colette C, Astre C, Monnier LH, Joyeux H. Intestinal adaptation in patients with short bowel syndrome. Measurement by calcium absorption. *Dig Dis Sci* 1989;34:709–715. [PubMed: 2496959]
34. Woolf GM, Miller C, Kurian R, Jeejeebhoy KN. Nutritional absorption in short bowel syndrome. Evaluation of fluid, calorie, and divalent cation requirements. *Dig Dis Sci* 1987;32:8–15. [PubMed: 3792183]

35. Messing B, Pigot F, Rongier M, Morin MC, Ndeindoum U, Rambaud JC. Intestinal absorption of free oral hyperalimentation in the very short bowel syndrome. *Gastroenterology* 1991;100:1502–1508. [PubMed: 1850371]
36. Crenn P, Matuchansky C, Messing B. Clinical and biochemical modelization of postsurgical intestinal failure in human adults. *Clin Nutr* 1997;16:133–135. [PubMed: 16844586]
37. Jeppesen PB, Mortensen PB. Intestinal failure defined by measurements of intestinal energy and wet weight absorption. *Gut* 2000;46:701–706. [PubMed: 10764715]
38. Nordgaard I, Hansen BS, Mortensen PB. Colonic fermentation of complex dietary carbohydrates in short-bowel patients. No association with hydrogen excretion and fecal and plasma short-chain fatty acids. *Scand J Gastroenterol* 1995;30:897–904. [PubMed: 8578190]
39. Jeppesen PB, Hoy CE, Mortensen PB. Deficiencies of essential fatty acids, vitamin A and E and changes in plasma lipoproteins in patients with reduced fat absorption or intestinal failure. *Eur J Clin Nutr* 2000;54:632–642. [PubMed: 10951512]
40. Jonas CR, Puckett AB, Jones DP, Griffith DP, Szeszycki EE, Bergman GF, Carlson JL, Newman MR, Galloway JR, Blumberg JB, Ziegler TR. Plasma antioxidant status after high-dose chemotherapy: A randomized trial of parenteral nutrition in bone marrow transplantation patients. *Am J Clin Nutr* 2000;72:181–189. [PubMed: 10871578]
41. Tangpricha V, Luo M, Fernández-Estívariz C, Bazargan N, Sitaraman SV, Gu LH, Klapproth J-M, Galloway JR, Leader LM, Ziegler TR. Growth hormone favorably affects bone turnover and bone mineral density in patients with short bowel syndrome undergoing intestinal rehabilitation. *JPEN J Parent Enteral Nutr* 2006;30:480–486.

Figure 1. A high percentage of SBS patients exhibited habitual oral dietary vitamin and mineral intake below the United States Recommended Dietary Allowances (RDA)

A total of 19 patients with short bowel syndrome (SBS) receiving chronic home parenteral nutrition and living in the Southeastern United States were investigated. Of these subjects, 14 SBS patients had incontinuity colon and 5 SBS patients were without residual in-continuity colon. Data (% below adequate RDA level intake) are shown separately for these two SBS subgroups. A 7-day food record of habitual oral intake was completed by each subject prior to admission to the metabolic unit and analyzed using food compositional computer software. Food intake data were analyzed by the General Clinical Research Center nutritionist using the Nutritionist IV database and modified as appropriate after detailed discussion with each subject. The following abbreviations for specific micronutrients are used : vitamin A (Vit A), vitamin D (Vit D), vitamin E (Vit E), vitamin K (Vit K), thiamine (B1), pyridoxine (B6), vitamin B12 (B12), vitamin C (Vit C), calcium (Ca), magnesium (Mg) and zinc (Zn).

Table 1

Clinical characteristics of SBS patients

Age	Sex	Diagnosis	Residual small bowel	Residual colon	Time on PN
32	M	Abdominal injury	70 cm jejunum	Left	3 mo
37	M	Abdominal injury	50 cm jejunum	Left	11 mo
55	F	Multiple surgery for SBO/fistula	105 cm jejunum	No	11 mo
52	F	Crohn's disease	350 cm jejunum-ileum	No	118 mo
35	F	Crohn's disease	180 cm jejunum-ileum	Entire	36 mo
57	F	Multiple surgery for SBO/fistula	75 cm jejunum-ileum	Entire	18 mo
37	F	Multiple surgery for SBO/fistula	36 cm jejunum	Left	18 mo
63	F	Multiple surgery for SBO/fistula	35 cm jejunum-ileum	Entire	21 mo
72	F	Mesenteric ischemia	61 cm jejunum	Entire	20 mo
67	M	Mesenteric ischemia	75 cm jejunum	Left	26 mo
32	M	Crohn's disease	183 cm jejunum-ileum	Left	7 mo
29	M	Crohn's disease	137 cm jejunum-ileum	No	6 mo
57	F	Mesenteric ischemia	15 cm ileum	Entire	19 mo
43	F	Multiple surgery for SBO/adhesions	350 cm jejunum-ileum	No	7 mo
32	F	Mesenteric ischemia	10 cm jejunum	Left	34 mo
52	F	Mesenteric ischemia	Duodenum only	Left	24 mo
35	M	Abdominal injury	75 cm ileum	Left	136 mo
58	F	Crohn's disease	300 cm jejunum-ileum	No	48 mo
61	M	Mesenteric ischemia	135 cm jejunum	Left	34 mo
48±3			118±25		31±8

BMI= Body mass index; PN= parenteral nutrition.

Table 2

Severity of intestinal failure by intestinal nutrient absorption in SBS patients

	Oral intake	GI output	% GI Absorption
Energy (Kcal)	2656 ± 242	1143 ± 149	59 ± 3
Fat (g)	92 ± 11	58 ± 9	41 ± 5
Nitrogen (g)	17 ± 2	9 ± 1	42 ± 5
Carbohydrate (g)	346 ± 31	90 ± 15	76 ± 3
Fluid (ml)	2712 ± 240	2192 ± 282	18 ± 10
Sodium (mEq)	207 ± 17	202 ± 32	6 ± 12
Potassium (mEq)	100 ± 11	58 ± 8	38 ± 7
Calcium (mg)	1190 ± 158	1165 ± 118	-7 ± 8
Phosphorus (mg)	1441 ± 132	948 ± 390	30 ± 6
Magnesium (mEq)	26 ± 3	25 ± 4	2 ± 8

Data as mean ± SEM; N=19. The % GI absorption was calculated with the formula: [(oral intake – intestinal output)/oral intake] × 100. SBS= short bowel syndrome

Table 3
Fluid and macronutrients reported by dietary recall and provided in parenteral nutrition

	Total intake	Reported oral	Reported oral intake as % of total intake	PN intake	PN intake as % of total intake
Fluid (ml/day) range	3498 ± 251 (1485 – 5820)	1766 ± 219 (457 – 4170)	49 ± 4	1732 ± 164 (286 – 3000)	51 ± 4 (14 – 77)
Energy (kcal/day) range	3281 ± 206 (1894 – 5534)	2193 ± 216 (721 – 4126)	66 ± 5 (29 – 92)	1088 ± 136 (244 – 1981)	34 ± 5 (8 – 71)
Energy (kcal/kg/day) range	49 ± 3 (33 – 75)	33 ± 3 (12 – 68)		16 ± 2 (4 – 35)	
Nitrogen (g/day) range	25 ± 2 (13 – 37)	14 ± 1 (5 – 27)	58 ± 5 (27 – 95)	10 ± 1 (1 – 18)	42 ± 5 (5 – 72)
Nitrogen (g/kg/day) range	0.36 ± 0.02 (0.22 – 0.54)	0.21 ± 0.02 (0.07 – 0.40)		0.15 ± 0.02 (0.02 – 0.26)	

Data as mean ± SEM; N=19. PN = parenteral nutrition