NUTRIENT ABSORPTION IN MALNUTRITION

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The absorption of some nutrients is impaired by the gastrointestinal alterations commonly found in protein-energy malnutrition (PEM), especially in its severe edematous and marasmic forms (Table 1). Since severely malnourished persons usually live under poor hygienic conditions, it is often difficult to determine whether the gastrointestinal alterations are due to the malnutrition itself or to concomitant conditions, such as bacterial overgrowth in the small intestine [1-3] and gastrointestinal or systemic infections.

Regardless of their cause, these alterations may affect the malnourished individual because the impaired absorption interferes with the satisfaction of his nutritional requirements and/or because malabsorption of some nutrients may cause gastrointestinal dysfunction that further diminishes the absorption of those or other nutrients. For example, fat malabsorption will impair the absorption of liposoluble vitamins and lactose malabsorption may produce diarrhea that will reduce the absorption of various nutrients.

The nutritional implications of malabsorption of some nutrients are of little or no consequence under optimal therapeutic conditions, where the abundance of nutrients overwhelms the deficits in intestinal absorptive capacity. On the other hand, the provision of large amounts of certain nutrients may have undesirable effects when the gut is incapable of handling even smaller amounts of the nutrient and responds with the appearance or enhancement of diarrhea and more severe malabsorption.

It is, therefore, necessary to make a critical analysis of the relationship between malnutrition and malabsorption and of the functional and

TABLE 1 - *Gastrointestinal Alterations that Affect Nutrient Absorption in Severe Protein-Energy Malnutrition.*

Morphological abnormalities (e.g., reduction of brush border, villous atrophy) [18, 29, 62-67].
Mucosal enzyme deficiencies (e.g., lactase and other disaccharidases) [68-70].
Impaired absorptive capacity of the intestinal mucosa (e.g., glucose, vitamin B12) [4, 30, 48].
Reduced intestinal motility [64, 71].
Pancreatic and bile salt insufficiency [4, 16, 17, 20, 21].
Bacterial overgrowth, especially in the small bowel [1-3].

therapeutic implications of the latter. This overview refers to investigations done in patients with severe PEM and with milder forms of malnutrition.

ABSORPTION OF SPECIFIC NUTRIENTS

Proteins

Studies using milk diets to treat children with severe PEM [4-6] showed that "true" nitrogen absorption (i.e., corrected for obligatory fecal nitrogen losses) is of the order of 90%, regardless of the dietary lactose content [5]. As Table 2 shows, absorption was slightly lower at the beginning of treatment, especially among children with diarrhea on admission, who only absorbed between 65 and 89% of the dietary nitrogen (mean $\pm$ s.d.: 80 ± 9) [5]. Absorption rapidly improved as nutritional treatment progressed and, on the average, fully recovered children absorbed 94% of dietary protein [5], which is similar to the absorptive capacity of healthy, well-nourished children [7].

Children 9-40 months old with mild and moderate PEM (weight-for-height deficits ranging from 5 to 18%) or who had recently attained adequate weight-for-height, were fed between 1.6 and 2.5 g protein/kg/day using Thai [8], Filipino [9], and Guatemalan [10] mixed diets, mostly with vegetable proteins. As Table 3 shows, the average "true"

TABLE 2 - *Dietary Nitrogen and Energy Absorption in Children 2-4 Years Old, at Various Stages of Treatment for Severe Protein-Energy Malnutrition (Kwashiorkor and Marasmic Kwashiorkor) Using Milk-Based Formulas.**

	Milk protein intake, g/kg/day	«True» nitrogen absorption, ** % of intake	Apparent energy absorption, % of intake
Beginning of treatment (days 2-5)	1.0 - 2.0	88 ± 9 ***	90 ± 6
Early treatment (days 12-14)	3.5 - 4.0	91 ± 5	93 ± 3
Advanced treatment (days 42-44)	3.5 - 4.0	91 ± 4	94 ± 3
Fully recovered	1.25	94 ± 4	—

* From Torún *et al.* [5, 7].
** Corrected for obligatory fecal N loss [7].
*** Mean ± standard deviation.

nitrogen absorptions ranged from 67 to 77%. These results were similar to the average "true" absorptions (ranging from 69 to 81%) of well-nousished children of the same age groups who consumed similar diets [8, 11-13].

Fats

Absorption of lipids is abnormal in severe PEM, resulting in microscopic or visible steatorrhea [4, 14-16]. The latter is not a frequent complaint, as the populations among whom PEM is highly prevalent usually eat foods with low fat contents, which generally account for only 10 or 12% of dietary energy. In children who weigh 10 kg this corresponds to a daily intake of approximately 6 to 10 g of fat.

Bile in severe PEM has a decreased concentration of conjugated bile salts and a relative increase of free bile acids [17]. There is also an impaired ileal reabsorption of bile salts with formation of more free bile acids as a consequence of bacterial overgrowth [4, 16, 17]. The increased ratio of free-to-conjugated bile acids impairs micellar formation and fat

TABLE 3 - *Nitrogen Absorption of Well-Nourished or Moderately Malnourished Children Eating Mixed Local Diets Predominantly Made With Vegetable Foods.*

Food	Number of children	Age, months	Nutrional status *	Protein intake g/kg/day	True nitrogen absorption	Ref.
Thai	9	9-36	mild PEM or normal	1.7	67 ± 4**	8
Filipino	7	18-26	mild PEM or normal	1.7	77	9
Guatemalan	13	15-40	mild or moderate PEM	1.6-2.5	74 ± 7	10
Thai	6	8-12	normal	1.7	74 ± 2	8
Filipino	5	18-24	normal	1.2-1.5	81	11
Filipino	5	22-29	normal	1.2-1.5	69	12
Guatemalan	11	29-46	normal	1.5-2.3	79 ± 5	13

* Based on weight-for-height and, in the Filipino children, weight-for-age.
** Mean ± standard deviation.

absorption. Furthermore, the free bile acids damage the intestinal mucosa, impair cell function, increase the abnormality in ileal reabsorption of bile acids, and can cause or enhance diarrhea [17-19]. Pancreatic lipase secretion is decreased [20, 21], but this does not seem to be very important since small quantities of the enzyme are sufficient to hydrolyze triglycerides and the malnourished child is able to respond with lipase secretion when challenged with oral fat [21].

In spite of those abnormalities, fat absorption in kwashiorkor is around 95% when fed at a level of about 4 g/kg/day or 25% of dietary energy [22]. Vegetable fats appear to be better tolerated than animal lipids [23] and children with severe PEM tolerate high quantities of unsaturated fatty acids [24]. High-fat diets providing 50-70% of dietary energy as vegetable oil (80-120 g fat/day) have been successfully used to treat children with severe PEM in Jamaica [25], Costa Rica [26] and Guatemala [27, 28]. Even though there was visible fat in feces, there was no profuse diarrhea, 85 to 92% of the dietary fat was absorbed [27, 28] and the children grew well. Fat absorption improves further with protein repletion and reaches normal levels when diarrhea is absent and nutritional recovery is about 80% of the final expected value [4]. The free bile acids decrease and conjugated acids increase to normal levels as nutritional recovery proceeds [17, 29].

Carbohydrates

Pancreatic amylase secretion is reduced [20] and absorption of glucose and D-xylose is impaired in severe PEM [4, 29, 30]. However, the carbohydrate malabsorption problem most frequently associated with diarrhea is that of disaccharides, and especially lactose malabsorption.

The effects of lactose ingestion on malnourished children have caused many apparent contradictions, most of which can be explained based on the methods used to diagnose malabsorption and assess changes in nutritional status or on the composition of the therapeutic diets (reviewed in reference 31). Although malnourished children frequently have lactase deficiency, lactose malabsorption and/or milk lactose intolerance, many investigators have reported adequate clinical and nutritional responses with milk-based therapeutic diets that provide 4-8 g lactose/kg/day or 1-1.7 g lactose/kg/meal [31].

Diarrhea has been associated with milk intakes by severely malnourished children [32-34]. On the other hand, milk has been successfully used for many years to treat children with severe PEM in countries like Jamaica [25] and Guatemala [31, 35], and diarrhea has seldom been a problem, even though in some series of patients as many as 24% passed loose stools or had a diarrheal episode without important clinical nutritional consequences [36]. In some of those patients the diarrhea could have been due to causes other than dietary lactose. It should be pointed out that in addition to being severely malnourished, all Jamaican children were black and many Guatemalan children were Mayan indians, and both these races are known to develop a genetically determined primary lactase deficiency.

Carefully controlled studies were recently done to compare the effects of intact and lactose-free milk [5, 37], and of milk- and soy-based diets (Torún, Solomons *et al.*, unpublished) in the treatment of children with severe PEM. Among the 40 children studied, the diets that contained lactose did not produce important diarrhea, and did not impair absorption of total dietary energy, nitrogen and calcium nor retard nutritional recovery.

Total Dietary Energy

The small influence of malnutrition on nitrogen, fat and carbohydrate absorption is reflected by the high levels of total dietary energy absorption. As Table 2 shows, using milk-based therapeutic diets the apparent energy absorption (i.e., without corrections for endogenous fecal energy) was about 93% in severe PEM [5]. The slightly lower degree of absorption at the beginning of treatment was probably due to the diarrhea that some patients had when they arrived at the hospital; six severely malnourished children admitted with diarrhea absorbed only 84 ± 5% (range: 77-90%) of the dietary energy at the beginning of treatment.

Using mixed Guatemalan diets, mainly with foods of vegetable origin that provided 98 ± 4 kcal/kg/day to children with mild and moderate PEM [10] and 92 ± 5 kcal/kg/day to well-nourished children [13], the corresponding apparent energy absorptions were 91 ± 2% and 92 ± 2%, respectively. These results coincide with those of studies using a Thai mixed diet with 100 kcal/kg/day [8] or Jamaican milk-based formulas with 84-108 kcal/kg/day [6] , which showed apparent energy absorptions of 94% when fed to children with weights-for-height ranging from 78 to 100% of the standard.

Vitamins

Fat-soluble vitamins. The absorption of fat-soluble vitamins is impaired in children with severe PEM [4, 29, 38]. This is probably related to the impairment of micellar formation due to the low output of conjugated bile acids [4, 16, 17]. Another mechanism that may affect the absorption of certain vitamins is the impaired activity of some enzymes. This has not yet been demonstrated in humans, but defective intestinal hydrolysis and impaired absorption of vitamin A have been shown in protein-deficient rats [39] and chicks [40].

The impaired absorption of vitamin A in severely malnourished children who are otherwise healthy is not of great magnitude: using a small dose of 3,000 I.U. of vitamin A, the average absorption in children with kwashiorkor was 90%, compared with 95% in well-nourished children [41]. Using pharmacological doses of 100,000 I.U., significant increases in serum retinol levels can be seen within 4 hours [42]. However, many malnourished children have associated infections and there is evidence that vitamin A absorption is further affected by intestinal infections [43, 44] and parasites [45].

Children with mild and moderate PEM absorb about 70% of beta-carotene from green leafy vegetables, similar to well nourished children [46]. This absorption can be increased when vegetable oil is fed with the leafy vegetables [47].

Water soluble vitamins. The absorption of several water soluble vitamins, such as folic acid and vitamin B12, is also impaired in severe PEM [29]. In the case of vitamin B12, the defect is in the small intestine [48] and not necessarily associated with absence of intrinsic factor, in spite of the atrophy of gastric mucosa and hypochlorhydria seen in children with severe PEM [49]. At least for vitamin B12, absorption appears to be normal in mild and moderate forms of PEM [50].

Minerals

Iron. Absorption of iron is reduced in many, but not all, children with severe PEM [51, 52]. This led to the use of parenteral iron for the treatment of iron-deficiency anemia in kwashiorkor [53, 54]. It has been proven, however, that iron deficiency can be treated orally in a great majority of patients. The low absorptive or hematologic responses to oral iron are mainly due to an adaptation to low oxygen demands, which is secondary to reduced lean-body mass and physical activity [55, 56], or to the existence of adequate body iron stores [51, 57].

The intestinal regulation of iron absorption is preserved in severe PEM. Using a tracer technique with 59-Fe to measure absorption and stainable iron in bone marrow to assess body stores [51], it was shown that there is greater absorption when body iron stores are low than when body iron is high, and that absorption increases when body iron is depleted. The same conclusion was reached in another study that combined metabolic balance techniques to assess iron absorption and retention, and serum ferritin and blood hemoglobin levels to assess changes in circulating and total body iron [57].

Iron absorption is adequate enough to allow improvement of hematological and body iron indices of children with mild and moderate PEM fed a mixed, predominantly vegetable, diet and sugar fortified with NaFeEDTA [10].

Calcium. Its absorption seems to be unimpaired in severe PEM. Patients treated with milk diets absorbed throughout six weeks between 35 and 47% of 1.3-1.5 g of calcium provided by the daily diet [5, 58].

This was significantly more than the absorption of 20% expected in normal children with the recommended daily intake of 400 mg Ca/day [59]. The high absorption of calcium during recovery from severe PEM may be related to increased needs for calcium accretion in bone.

Other minerals and electrolytes. The loss of these elements usually seen in severe PEM seems to be related to the chronic or recurrent diarrhea that often accompanies malnutrition. However, other routes for electrolyte losses, such as urine and sweat, cannot be ruled out to explain the decrease in body potassium and magnesium often seen in severe PEM. Information about the absorption of these elements and trace minerals is scant. There are some studies, such as investigations on the role of dietary zinc to treat malnourished children [60], that suggest that the intestine is capable of absorbing these minerals, but it is not known whether the absorptive capacity is normal.

Conclusions

Gastrointestinal abnormalities impair the absorption of many nutrients in severe PEM. Furthermore, when dietary intake is only marginally adequate, chronic malabsorption may be an important limiting factor to achieve good nutritional status. These gastrointestinal alterations are not due to malnutrition alone, but are the result of an interaction between malnutrition and enteric infections or nonspecific bacterial proliferation in the small bowel.

In the absence of profuse diarrhea and intestinal infections, the ingestion of nutrients in high, therapeutic, amounts allows their absorption in sufficient quantity to permit nutritional recovery. In most cases this reduces the practical importance of malabsorption during treatment for malnutrition. Notable exceptions are food — or nutrient — intolerances that produce diarrhea and interfere with nutritional recovery. The causes of profuse or persistent diarrhea, whether of infectious or food origin, must be controlled as they may impair intestinal absorption to the extent of retarding or not allowing nutritional recovery. Since intestinal infections produce further damage and malabsorption, they should be adequately treated to facilitate nutritional rehabilitation. Whether drug treatment with antibiotics should be used in all enteric infections in children with severe PEM, is still a matter of discussion. Many such infections disappear as time and nutritional treatment progress. The critical issue is whether those infections threaten the malnourished child's life or overall health.

The clinical and experimental evidence that is currently available in humans suggests that malabsorption disappears with nutritional recovery and that there are no important gastrointestinal sequels due to malnutrition. This must be ascertained in a definitive way since most children treated for severe PEM continue living in highly contaminated environments and resume eating a deficient or marginally adequate diet after discharge from therapy. Consequently, if their gastrointestinal functions are still impaired, they will be more prone to develop malabsorption of an important degree or diarrhea that may lead to a new episode of severe malnutrition.

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