Overview of the treatment of malabsorption in adults

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INTRODUCTION

Malabsorption refers to impaired absorption of nutrients, including water and electrolytes [1]. It can result from congenital defects in the membrane transport systems of the small intestinal epithelium (primary malabsorption) or, much more commonly, from acquired defects in the epithelial absorptive surface (acquired malabsorption). Maldigestion, which is the impaired digestion of nutrients within the intestinal lumen or at the terminal digestive site of the brush border membrane of mucosal epithelial cells, can also interfere with nutrient absorption since it is the necessary predecessor to many absorptive mechanisms.

Although malabsorption and maldigestion are pathophysiologically distinct, the processes underlying digestion and absorption are interdependent, so that in clinical practice the term malabsorption has come to denote derangements in either process. This topic will provide an overview of the principles of management of malabsorption. The clinical manifestations, diagnosis, and pathophysiology of malabsorption and the management of specific diseases associated with malabsorption are discussed separately. (See "Approach to the adult patient with suspected malabsorption" and "Overview of nutrient absorption and etiopathogenesis of malabsorption" and "Management of celiac disease in adults" and "Management of short bowel syndrome in adults" and "Small intestinal bacterial overgrowth: Management".)

Classification — Malabsorption may either be global or selective.

- **Global malabsorption** – Global malabsorption arises from diseases associated with either widespread mucosal involvement or a reduced absorptive surface. Typically, a broad array

of nutrients are not adequately absorbed. An example is celiac sprue, in which impaired absorption of almost all nutrients occurs.

- **Selective malabsorption** – Selective or isolated malabsorption results from diseases that interfere with the absorption of a single nutrient or a limited array of nutrients. An example is pernicious anemia, a disease that leads to defective cobalamin (vitamin B12) absorption.

GENERAL MANAGEMENT

Goals of management

- Treat the underlying disease – Although a diverse array of pathological conditions may result in global malabsorption, diarrhea, weight loss, and nutritional deficits are common manifestations regardless of the cause. Thus, while the clinical presentation of two patients with malabsorption may be similar, the underlying pathophysiology and appropriate treatment may be very different. Establishing a diagnosis of malabsorption is therefore only a first step since identifying and treating the underlying disease process must follow.
- Optimize control of the diarrhea that often accompanies malabsorption.
- Identify and treat nutritional deficits, and then monitor for re-occurrence.
- Optimize quality of life – Malabsorption, especially when severe and when the pathophysiology of the underlying disease cannot be fully corrected, creates major impositions on one's life due to issues such as long-term dietary constraints, the need for a multi-drug pharmacologic regimen, the need for immediate access to toilets, the social stigma attached to weight loss, etc. The optimal management strategies for the same malabsorptive condition in a jet-setting business executive may be very different than the optimal strategies for a college-aged teen: thus, person-specific factors need to be taken into consideration when designing a management plan.

Antidiarrheals in selected patients — In many cases, treating the underlying disease and applying appropriate dietary interventions is sufficient to control diarrhea. However, when such measures are insufficient, loperamide or diphenoxylate with atropine can be used to prolong transit time and enhance fluid and electrolyte absorption. In general, it is best to begin with loperamide rather than diphenoxylate with atropine because it is largely metabolized on first pass through the liver and, as it does not easily cross the blood-brain barrier, it rarely has

central nervous system side-effects. It is also generally less expensive than diphenoxylate with atropine. In patients with severely shortened small bowel or who otherwise have very rapid intestinal transit, using the liquid forms of these drugs is sometimes more effective since it avoids the issue of incomplete tablet dissolution. Deodorized tincture of opium (DTO) is a highly effective anti-diarrheal and is particularly useful in patients with short bowel syndrome or other causes of chronic, severe diarrhea that persists despite loperamide and diphenoxylate with atropine. In a small minority of patients, DTO can produce mild sedation. Further, it does have a very low, but real, addictive potential. However, in reliable patients without a history of drug abuse, it can be quite useful. (See "Management of short bowel syndrome in adults", section on 'Antidiarrheals' and "Management of acute chemotherapy-related diarrhea", section on 'Pharmacologic management'.)

Dietary modifications

General measures — Dietary measures are important adjuncts in the effective management of malabsorption. These dietary strategies can help address nutritional deficits and, importantly, can reduce diarrhea, especially in circumstances in which the underlying pathophysiology cannot be entirely corrected. We suggest the following general dietary measures:

- Avoidance of more than one serving a day of caffeine-containing beverages.
- In patients with very rapid transit or whose colons are absent or very shortened, full-strength sugar-sweetened beverages and fruit juices should be avoided as they can markedly increase the volume of diarrhea due to their osmotic load. If consumed, they should be diluted with water in a 1:1 ratio.
- Sorbitol-containing candies and gums should also be avoided. They can cause diarrhea due to the osmotic effects of this sugar alcohol.
- Patients with severe diarrhea resulting in dehydration or electrolyte abnormalities should receive an oral rehydration solution. Commercially-available products vary widely in their formulation, and some are designed more for commercial appeal than for adherence to scientific principles, so a formula that abides by World Health Organization guidelines should be used. (See "Approach to the adult with acute diarrhea in resource-abundant settings", section on 'Fluid repletion' and "Oral rehydration therapy".)

Specific dietary restriction in selected patients — Dietary restrictions of specific components of the diet in some diseases leading to malabsorption can result in full restoration of mucosal function and nutritional status. Examples include elimination of a particular

carbohydrate in patients with isolated disaccharidase deficiencies, or avoidance of gluten in patients with celiac disease [2]. (See "Lactose intolerance and malabsorption: Clinical manifestations, diagnosis, and management", section on 'Management' and "Management of celiac disease in adults", section on 'Components of the gluten-free diet'.)

When fat malabsorption cannot be corrected by treatment of the underlying disease, a restriction of dietary fat should be imposed. For an adult, restricting fat to <50 grams per day represents a modest and achievable fat restriction; achieving <40 grams per day is difficult. Restricting fat will reduce diarrhea since malabsorbed fatty acids stimulate a secretory diarrhea when they reach the colon. In patients whose colon is no longer in the fecal stream, this is not a consideration, so fat restriction in this setting does not diminish diarrhea and fluid losses. However, regardless of whether a colon is present or not, malabsorbed fatty acids bind calcium and magnesium, creating a malabsorption of those nutrients that is proportional to the degree of fat malabsorption, thereby exacerbating the metabolic bone disease that often accompanies longstanding malabsorption. This factor alone may therefore dictate a fat restriction in an individual whose colon is not in the fecal stream.

Medium-chain triglyceride supplements, either as a powder or as an oil, are a useful dietary supplement for those adhering to a fat restriction since the caloric density is the same as those for long-chain triglycerides, thereby compensating for the calories lost due to the dietary restriction. The oil format produces nausea and diarrhea in a minority of individuals.

Fructose intolerance is another condition that responds to a specific dietary restriction, albeit a diagnosis that remains somewhat controversial. A fructose breath test has been suggested as an objective means of identifying fructose intolerance, although additional studies are needed to validate the predictive value of this test [3]. Patients with suspected fructose intolerance should be advised to avoid foods that contain a high net amount of fructose (ie, more fructose than glucose, or more fructose and sorbitol than glucose). In particular, they should avoid intake of food or beverages that are sweetened with fructose, crystalline fructose, or high-fructose corn syrup [4]. Honey should also be avoided since it contains 35 g of fructose and 29 g of glucose per 100 g (3 tablespoons). Fruits with high-fructose concentration include apples, pears, sweet cherries, prunes, and dates. If foods containing fructose are consumed, they should be eaten with meals. Beverages sweetened with high- fructose corn syrup should be limited to 12 ounces. Long-term elimination of high-fructose fruits should only be entertained if there is unequivocal symptomatic improvement with their elimination. Similarly, sucrase-isomaltase deficiency has been reported to be more prevalent than previously thought, often masquerading as irritable bowel syndrome, and responds to a low-sucrose or sucrose-free diet,

sometimes with the addition of a low-starch or starch-free diet [5]. (See "Approach to the adult patient with suspected malabsorption", section on 'Malabsorption of specific carbohydrates'.)

Nutrient repletion and supplementation

Fat soluble vitamins — Patients with significant steatorrhea may require supplementation with special forms of fat-soluble vitamins that have a more water-soluble nature. Examples include the 25-hydroxy (calcifediol), 1-hydroxy (alfacalcidol), or 1,25 dihydroxy (calcitriol) forms of vitamin D. The serum calcium should be monitored for the first few weeks of therapy with these vitamin D analogues since they are more potent than vitamin D2 or D3, and can more easily produce hypercalcemia. This is especially true of the dihydroxy form. In assessing blood levels of vitamin D in patients on alfacalcidol or calcitriol, it is necessary to check serum 1,25 dihydroxyvitamin D levels rather than the more conventional 25-hydroxyvitamin D, since the 25-hydroxy stage is bypassed with these supplements.

Vitamin D deficiency diminishes intestinal absorption of calcium and phosphate, thereby promoting bone de-mineralization. Optimal reduction of bone fracture risk is best achieved when serum 25-hydroxyvitamin D levels are maintained at or slightly above the normative range (ie, 30 to 40 ng/mL) among those using vitamins D2, D3, or 25-hydroxy vitamin D as a supplement [6]. Among those taking the 1-hydroxylated or 1,25-dihydroxy forms of the vitamin, the optimal blood concentrations of vitamin D metabolites are less clear. It would probably be best in the latter situation to aim for a blood level within the normative range for 1,25 dihydroxy vitamin D. (See "Vitamin and mineral deficiencies in inflammatory bowel disease", section on 'Vitamin D'.)

In patients with fat malabsorption and vitamin E deficiency, a special form of vitamin E (d-alpha-tocopherol glycol 1000 succinate) is preferred as other vitamin E preparations are poorly absorbed. Some fat-soluble vitamins can also be obtained in capsules containing an emulsifying solution (eg, Aquasol A, Aquasol E). However, it is unproven whether these products actually enhance vitamin absorption. (See "Vitamin intake and disease prevention".)

Oral supplementation with the daily recommended intake is sometimes sufficient to correct existing nutritional deficiencies, although over the few weeks immediately following the identification of a vitamin or mineral deficiency, more rapid recovery is most readily achieved by supplementation with 5 to 10 times the recommended dietary allowance (table 1).

If the underlying etiology of malabsorption can be identified and corrected, patients may not require long-term supplementation. In patients with persistent malabsorption, long-term supplementation of fat-soluble vitamins may be required based on the degree of ongoing malabsorption. (See 'Laboratory studies for micronutrient deficiencies' below.)

Other micronutrients — Calcium and magnesium supplementation is often required in patients with fat malabsorption. In patients with fat malabsorption and a colon in continuity with the fecal stream, calcium supplementation also assists in the prevention of nephrolithiasis due to oxalate stones [7]. (See "Chronic complications of the short bowel syndrome in adults", section on 'Nephrolithiasis'.) When calcium supplementation is administered for this purpose, it should be taken with meals, thereby enabling the calcium to bind the oxalate contained in the meal [8]. Intermittent parenteral repletion of magnesium may be required in some patients since oral magnesium supplements can exacerbate their underlying diarrhea. (See "Treatment of hypocalcemia", section on 'Mildly symptomatic or chronic hypocalcemia' and "Hypomagnesemia: Evaluation and treatment".)

Other micronutrients that often require supplementation include iron, folate, vitamin B12, and zinc. Even in the absence of documented micronutrient deficiencies, it is prudent to administer a daily multivitamin/multimineral tablet to patients with chronic malabsorption due to the frequent and insidious evolution of deficiency states. (See "Treatment of iron deficiency anemia in adults", section on 'Oral iron' and "Treatment of vitamin B12 and folate deficiencies", section on 'Treatment of folate deficiency' and "Treatment of vitamin B12 and folate deficiencies", section on 'Treatment of vitamin B12 deficiency' and "Overview of dietary trace elements", section on 'Dietary reference intake'.)

Monitoring

Bone density — Chronic malabsorption, and particularly fat malabsorption, is a risk factor for metabolic bone disease and fractures [9,10]. Patients with malabsorption require screening for fracture risk with history and physical examination to assess for risk factors and measurement of bone mineral density. (See "Screening for osteoporosis in postmenopausal women and men", section on 'Fracture risk assessment'.)

Laboratory studies for micronutrient deficiencies — Patients whose malabsorption cannot be fully corrected require intermittent monitoring for early detection since many vitamin and trace mineral deficiencies evolve insidiously. In chronic fat malabsorption, it is worthwhile to measure all four fat-soluble vitamins, whereas checking vitamin B12 and just one other representative water-soluble vitamin will usually suffice. The assessment of plasma trace mineral (chromium, copper, manganese, molybdenum, selenium, and zinc) levels are problematic and they should be interpreted thoughtfully since: 1) the vanishingly low concentrations of these minerals in the blood are difficult to measure accurately, 2) because plasma levels do not necessarily reflect tissue concentrations, and 3) because minor deviations from normative ranges are often not clinically meaningful. Among the trace minerals, zinc is the most commonly observed deficiency among malabsorbers due its high concentrations in

gastrointestinal secretions. Consequently, this author routinely monitors zinc in chronic malabsorbers and only checks other trace mineral levels when the clinical scenario so dictates. The frequency of routine monitoring depends on the clinical situation but should not occur any less often than annually.

DIRECTED THERAPY BASED ON THE UNDERLYING ETIOLOGY

Specific interventions for malabsorption based on the underlying etiology include (table 2):

- **Bile acid malabsorption** – Minor degrees of bile acid re-absorption result in bile acid diarrhea (cholorrhea), which is treated very differently than those with marked degrees of bile acid malabsorption, which results in steatorrhea: this is an important distinction to make. (See "Overview of nutrient absorption and etiopathogenesis of malabsorption", section on 'Causes of fat malabsorption'.)

  In patients in whose bile acid deficiency is marked and therefore plays a role in fat malabsorption (eg, extensive ileal disease or resection, chronic cholestasis, end-stage cirrhosis), therapy with exogenous conjugated bile acids can decrease steatorrhea. Although such preparations are no longer available by prescription, there are some reliable companies that sell desiccated ox and cattle bile over the internet. However, natural bile acid preparations often increase the volume of diarrhea due to colonic secretion occurring after bacteria in the colon deconjugate the bile acids. This can occur even though they are decreasing the magnitude of steatorrhea. This is generally not of concern in patients who have undergone a colectomy [11]. For patients who have a colon in continuity with the fecal stream, however, a synthetic conjugated bile acid that does not undergo bacterial deconjugation (cholylsarcosine) may be beneficial [12,13]. Apart from reducing diarrhea, cholylsarcosine reduces urinary oxalate excretion in patients with short bowel syndrome [14]. Cholylsarcosine is presently not available in the United States.

- **Small intestinal bacterial overgrowth** – The mainstay of therapy for small intestinal bacterial overgrowth are antibiotics to reduce (rather than eradicate) small intestinal bacteria. (See "Small intestinal bacterial overgrowth: Management", section on 'Initial approach'.)

- **Post-bariatric surgery** – Although gross macronutrient malabsorption is very uncommon following bariatric surgery, micronutrient deficiencies often arise and have multiple causes, including malabsorption [15]. The likelihood and nature of deficiency states are partly determined by the type of surgery that is performed. The most common clinically

relevant micronutrient deficiencies after gastric bypass include thiamine, vitamin B12, vitamin D, iron, and copper. Malabsorption of certain drugs such as thyroid replacements may occur, necessitating monitoring and adjustment of dosages [16].

- **Exocrine pancreatic insufficiency** – In patients with chronic pancreatitis and exocrine pancreatic insufficiency, the mainstay of treatment is a balanced fat intake and administration of exogenous pancreatic enzymes. The management of exocrine pancreatic insufficiency is discussed in detail separately. (See "Exocrine pancreatic insufficiency", section on 'Management' and "Chronic pancreatitis: Management", section on 'Management of pancreatic insufficiency'.)

- **Short bowel syndrome** – The management of short bowel syndrome varies in the acute and chronic phases and is discussed in detail separately. (See "Management of short bowel syndrome in adults", section on 'Management of acute phase' and "Management of short bowel syndrome in adults", section on 'Management in adaptation phase'.)

- **Celiac disease** – A gluten-free diet remains the mainstay of treatment in patients with celiac disease. (See "Management of celiac disease in adults".)

- **Zollinger Ellison syndrome** – Patients with Zollinger-Ellison syndrome require high-dose proton-pump inhibitors to effectively block acid secretion as reduction in duodenal pH inhibits fat absorption. (See "Management and prognosis of the Zollinger-Ellison syndrome (gastrinoma)", section on 'Medical management' and "Overview of nutrient absorption and etiopathogenesis of malabsorption", section on 'Mechanism of fat digestion and absorption'.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Malabsorption".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more

sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Gas and bloating (The Basics)")
- Beyond the Basics topics (see "Patient education: Lactose intolerance (The Basics)" and "Patient education: Gas and bloating (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- The general management of patients with malabsorption and maldigestion consists of treating the underlying disease, managing the accompanying diarrhea, correcting nutritional deficits, and optimizing quality of life. (See 'General management' above and 'Directed therapy based on the underlying etiology' above.)
- General dietary measures are helpful adjuncts in the management of diarrhea due to malabsorption in all patients. More specific dietary restrictions may be necessary (eg, fat restrictions, elimination of a particular carbohydrate or avoidance of gluten) based on the underlying etiology. (See 'Dietary modifications' above.)
- More specific interventions for malabsorption are based on the underlying etiology (eg, antibiotics to treat small intestinal bacterial overgrowth, pancreatic enzyme supplementation in patients with pancreatic exocrine insufficiency). In patients with diarrhea, loperamide and diphenoxylate with atropine are each effective in reducing gastrointestinal motility and enhancing fluid resorption. In severe, refractory diarrhea, deodorized tincture of opium may be indicated. (See 'Antidiarrheals in selected patients' above.)
- Patients with significant steatorrhea often require supplementation with fat-soluble vitamins, calcium, and sometimes magnesium. Regardless of whether steatorrhea is present, supplementation with other micronutrients (including iron, folate, vitamin B12 and zinc) is often needed. (See 'Nutrient repletion and supplementation' above.)
- Patients with malabsorption require screening for fracture risk, measurement of bone mineral density, and intermittent laboratory monitoring for vitamin and other

micronutrient deficiencies. (See 'Monitoring' above.)

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GRAPHICS

Medications and nutrient supplements commonly used in malabsorption and maldigestion

Medication/supplement	Comments
Antidiarrheal agents	
Loperamide 2 to 4 mg as needed. Not to exceed 16 mg/day.	▪ Preferred initial antidiarrheal. ▪ Gradually titrate down to minimum required. ▪ Commercial liquids (1 mg/5 mL or 1 mg/7.5 mL) also available.
Diphenoxylate-atropine (2.5/0.025 mg). 1 to 2 tablets after each loose stool. Not to exceed 8 tablets/day.	▪ Each tablet contains 2.5 mg diphenoxylate (synthetic opioid). ▪ Atropine ingredient is to discourage abuse; causes anticholinergic effects with excess dosing. ▪ Commercial liquid (2.5/0.025 mg/5 mL) also available.
Deodorized tincture of opium 1% solution (contains 10 mg morphine per mL) 0.3 to 0.8 mL in water three times daily.	▪ Effective alternative in severe diarrhea with insufficient response to loperamide and diphenoxylate. ▪ US DEA C-II controlled substance; addictive potential. ▪ Avoid abrupt discontinuation, which may produce withdrawal syndrome. ▪ Not recommended for use in patients with a history of substance abuse.
Bile acid binding resins for bile acid malabsorption-associated diarrhea [cholorrhea], not fat malabsorption	
Cholestyramine 4 g once daily initially; increase gradually (eg, weekly) to 4 g three times daily.	▪ May reduce absorption of other drugs and supplements; administer either ≥1 hour before or 4 to 6 hours after other drugs and supplements. Refer to drug interactions database. ▪ Available as 4 g packets and 4 g/scoop bulk powder.
Colestipol: ▪ Granules: 5 g once or twice daily; increase gradually to 5 g three times daily ▪ Tablets: 2 g once or twice daily; increase gradually to 2 g three times daily	▪ May reduce absorption of other drugs and supplements; administer either ≥1 hour before or 4 to 6 hours after other drugs and supplements. Refer to drug interactions database.
Pancreatic enzymes for exocrine pancreatic insufficiency	

Pancrelipase microencapsulated, delayed-release (eg, Creon). Initially 30,000 USP units lipase (~500 USP units lipase/kg) with meals and half of that amount with snacks; adjust gradually to patient needs*.	▪ Use in patients with intact upper GI tract and intact gastric secretions.
Pancrelipase, non-microencapsulated (eg, Viokace). Initially 30,000 USP units lipase (~500 USP units lipase/kg) with meals and half of that amount with snacks; adjust gradually to patient needs*.	▪ Inactivated by stomach acid. Use in patients lacking acid-peptic gastric environment or administer with acid-suppressing drug.
Vitamins and minerals (doses for oral acute repletion cited; required maintenance doses are usually lower but vary widely)¶	
Vitamin A 40,000 to 50,000 units (12,000 to 15,000 mcg retinol activity equivalent) twice daily.	▪ Periconceptional exposure to a single dose of >25,000 units or >10,000 units/day has been reported to be teratogenic, so take caution in women of child-bearing age.
Vitamin D3 (cholecalciferol) 10,000 to 50,000 units (250 to 1250 mcg)/day individualized according to serum 25(OH)D level.	▪ Patients who remain deficient on such doses will need to be treated with hydroxylated vitamin D metabolites (eg, calcitriol) because they are more readily absorbed.
Vitamin K (phytonadione) 2.5 to 12.5 mg/day.	▪ Intravenous preparation available.
Folic acid 1 mg/day.	
Vitamin B12 (cyanocobalamin) 1 mg subcutaneously or intramuscular, repeat 3 times in first week. Acute oral repletion not recommended.	▪ Formulations are available for intramuscular/deep subcutaneous injection and oral, sublingual, and nasal administration. ▪ 1 mg/day orally often sufficient for maintenance of pernicious anemia.
Calcium carbonate 500 mg (200 mg elemental calcium) twice daily.	
Magnesium gluconate 1 to 4 g (54 to 216 mg elemental magnesium) four times daily.	▪ Often exacerbates diarrhea, necessitating parenteral replacement.
Ferrous sulfate 325 mg (65 mg elemental iron) three times daily.	▪ Available as oral liquids in multiple concentrations. ▪ Equivalent dose mixed in 8 to 12 ounces (240 to 360 mL) of orange juice or taken with a 250 mg ascorbic acid tablet enhances bioavailability.

All dosages oral unless indicated otherwise.

USP: United States pharmacopeia.

* In most adult patients, 90,000 USP units of lipase per meal is the amount that will be needed to abolish steatorrhea. Pancreatic enzyme replacement products available in various countries are not equivalent; consult local labeling before prescribing.

¶ Refer to UpToDate clinical topics and Lexicomp drug monographs (included with UpToDate) for details of dosing and treatment of acute deficiency in setting of malabsorption.

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Malabsorption: Conditions causing malabsorption, categorized by the phase of absorption that is impaired

Phase and nature of malabsorptive defect	Example
Luminal phase	
A. Substrate hydrolysis	
1. Digestive enzyme deficiency	Chronic pancreatitis
2. Digestive enzyme inactivation	Zollinger-Ellison syndrome
3. Dyssynchrony of enzyme release, inadequate mixing	Post Billroth II procedure
B. Fat solubilization	
1. Diminished bile salt synthesis	Cirrhosis
2. Impaired bile secretion	Chronic cholestasis
3. Bile salt de-conjugation	Bacterial overgrowth
4. Increased bile salt loss	Ileal disease or resection
C. Luminal availability of specific nutrients	
1. Diminished gastric acid	Atrophic gastritis - vitamin B12
2. Diminished intrinsic factor	Pernicious anemia - vitamin B12
3. Bacterial consumption of nutrients	Bacterial overgrowth - vitamin B12
Mucosal (absorptive) phase	
A. Brush border hydrolysis*	
1. Congenital disaccharidase defect	Sucrase-isomaltase deficiency
2. Acquired disaccharidase defect	Lactase deficiency
B. Epithelial transport	
1. Nutrient-specific defects in transport	Hartnup's disease
2. Global defects in transport	Celiac sprue
Postabsorptive, processing phase	
A. Enterocyte processing	Abetalipoproteinemia
B. Lymphatic	Intestinal lymphangiectasia

* This process is sometimes considered as part of the luminal phase.

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