# MALABSORPTION SYNDROME

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# Definitions

- **Malabsorption:** inability to absorb dietary food. Mucosal barrier to absorption: *disease of small intestine.*
- **Maldigestion:** deficiency or inactivation of pancreatic enzymes and bile salt. *Primary pancreatic exocrine insufficiency.*

# Classification of the malabsorption syndromes

*Impaired digestion*
- Gastric surgery
- Gastrinoma

*Reduced bile salt concentration*
- Liver disease
- Small intestine bacterial overgrowth
- Ileal disease or resection

*Abnormalities of intestinal mucosa*
- Dissaccharide deficiency
- Impaired monosaccharide transport
- Folate or cobalamine deficiency
- Nontropical sprue
- Nongranulomatous ileojejunitis
- Amyloidosis
- Crohn's disease
- Eosinophilic enteritis
- Radiation enteritis
- Abetalipoproteinemia
- Cystinuria
- Hartnup disease

*Inadequte absorptive surface*
- Short bowel syndrome
- Jejunoileal bypass

*Infection*
- Tropical sprue
- Whipple's disease
- Acute infectious enteritis
- Parasitic: Giardia, AIDS, helminthiasis

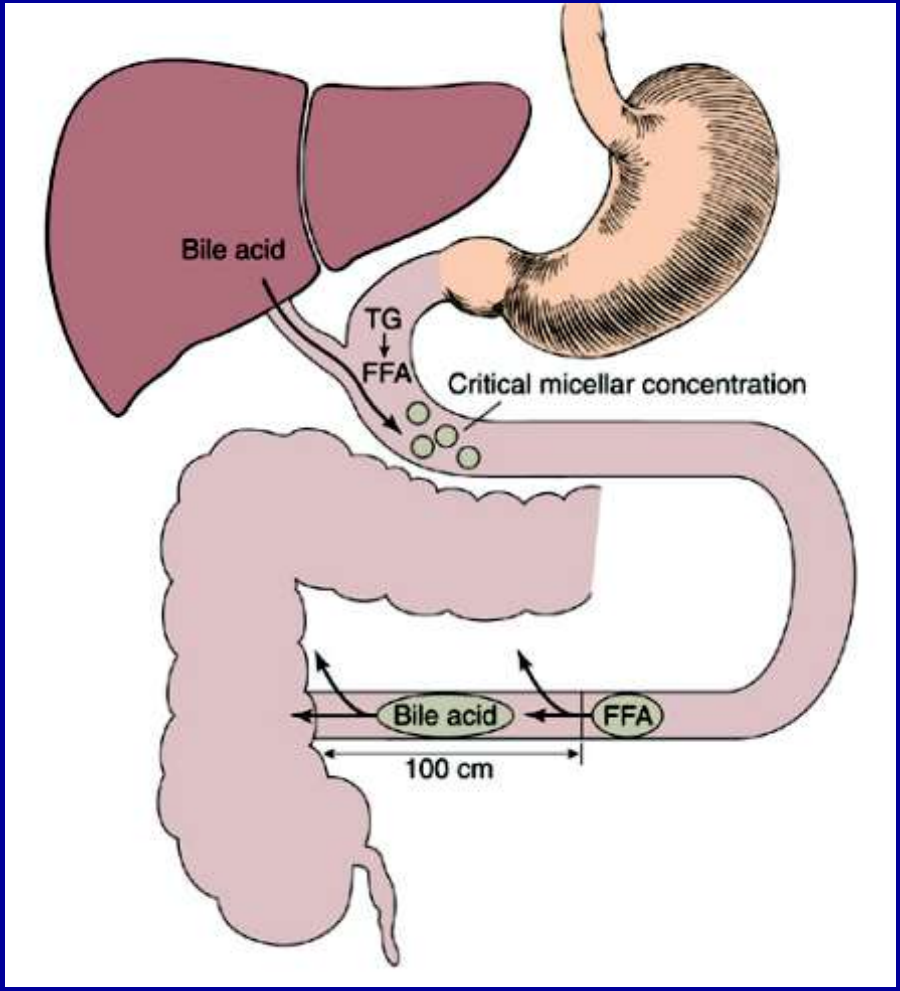
*Lymphatic obstruction*
- Intestinal lymphoma
- Tuberculosis
- Lymphangiectasia

*Cardiovascular disorders*
- Congestive heart failure
- Constrictive pericarditis
- Mesenteric vascular insufficiency

*Drug-induced*
- Cholestyramine, neomycin, colchicin, phenindione, irritant laxatives

*Unexplained*
- Carcinoid syndrome
- Diabetes mellitus
- Adrenal insufficiency
- Hyper- and hypothyroidism
- Mastocytosis
- Hypogammaglobulinemia, CVID

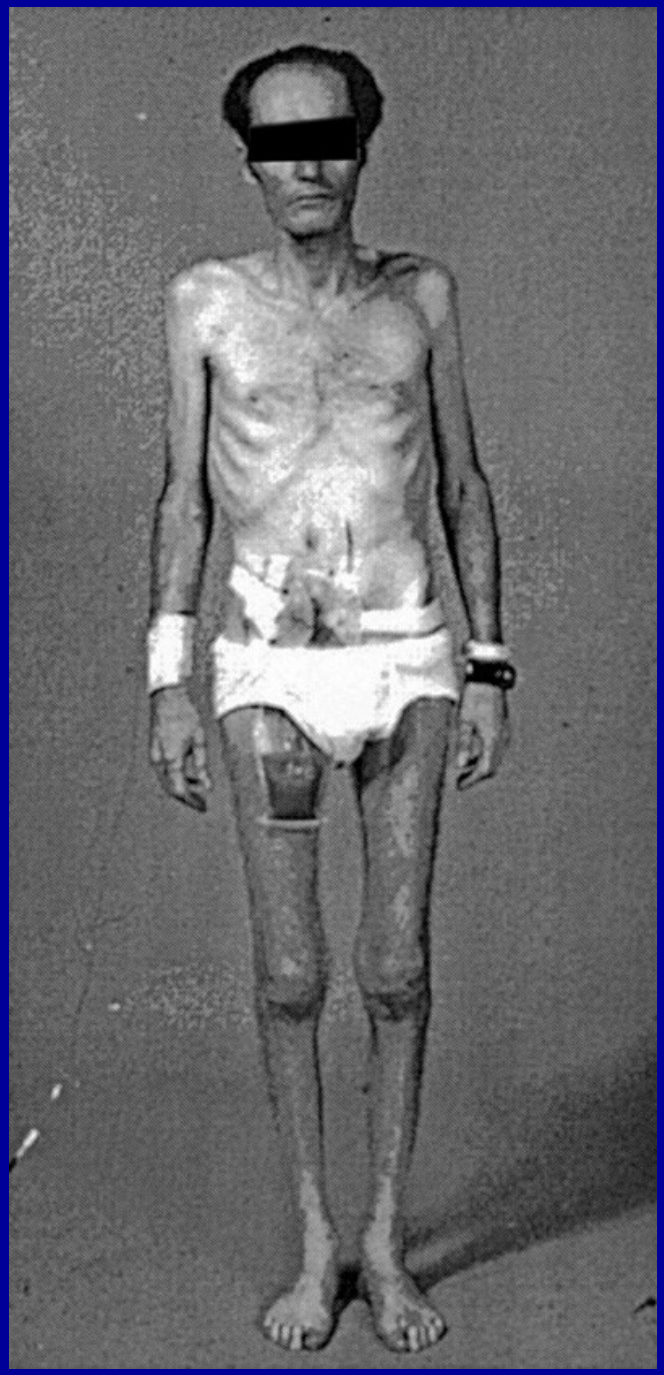
# Malabsorption of bile acids occur after resection of the terminal ileum, the only site of active bile acid absorption




- When resection is minimal (<100 cm), increased bile acids are delivered to the colon, where they inhibit colonic water and electrolyte absorption and stimulate colonic motility. The liver can compensate for minimal bile acid losses by increasing hepatic bile acid synthesis, thereby maintaining a normal bile acid-pool size and sufficient bile acids for micelle formation.
- When ileal resection is >100 cm, however, the liver is unable to maintain the bile acid pool and the critical micellar concentration cannot be reached, thus causing fat malabsorption and steatorrhea.
- Therefore, understanding the pathophysiology of diarrhea after ileal resection has important therapeutic implications. Minimal ileal resection causes bile acid diarrhea that can be treated with cholestyramine, a bile acid binder. More marked ileal resections cause steatorrhea, which can actually be made worse by cholestyramine therapy by further depleting of the remaining bile acid pool.

# Clinical and laboratory manifestations of malabsorption 1

| Manifestation | Laboratory findings | Malabsorbed nutrients |
|---|---|---|
| Steatorrhea (bulky, light color) | Increased fecal fat, decreased serum cholesterol | Fat |
| Diarrhea (increased fecal water) | Increased fecal fat or positive bile salt breath test | Fatty acids or bile salts |
| Weight loss, malnutrition (muscle wasting), weakness, fatigue | Increased fecal fat and nitrogen decreased glucose and xylose absorption | Calories (fat, protein, carbohydrate) |
| Abdominal distention | | |
| Iron deficiency anemia | Hypochromic anemia | Iron |
| Megaloblastic anemia | Macrocystosis, decreased vitamin $B_{12}$ absorption ($^{67}$Co-labelled $B_{12}$), decreased serum vitamin $B_{12}$ and folic acid activity (microbiologic culture) | Vitamin $B_{12}$ or folic acid |



# Malabsorption in Crohn's disease

## Clinical and laboratory manifestations of malabsorption 2

| Manifestation | Laboratory findings | Malabsorbed nutrients |
|---|---|---|
| Paresthesia, tetany, positive Trousseau and Chvostek signs | Decreased serum calcium, magnesium and potassium | Calcium, vitamin D, magnesium, potassium |
| Bone pain, pathologic fractures, skeletal deformities | Osteoporosis, osteomalatia on x-ray | Calcium, protein |
| Bleeding tendency (ecchimoses, melena, hematuria) | Prolonged prothrombin time | Vitamin K |
| Edema | Decreased serum albumin, increased fecal loss of $\alpha_1$-antitripsin | Protein (or protein-losing enteropathy) |
| Nocturia, abdominal distention | Increased small bowel fluid on x-ray | Water |
| Milk intolerance (cramp, bloating, diarrhea) | Flat lactose tolerance test, decreased mucosal lactase levels | Lactose |

# Gluten-sensitive enteropathy 1

## Sprue syndrome

- Disturbed small intestine function - impaired absorption, particularly of fats, and motor abnormalities.

## Pathology

- A flat intestinal mucosa without villi in the small intestine.
- Degenerative changes in the myenteric nerve plexuses.
- Loss of villi → loss of microvilli → disaccharidase deficiency (particularly, lactase).

## Pathophysiology

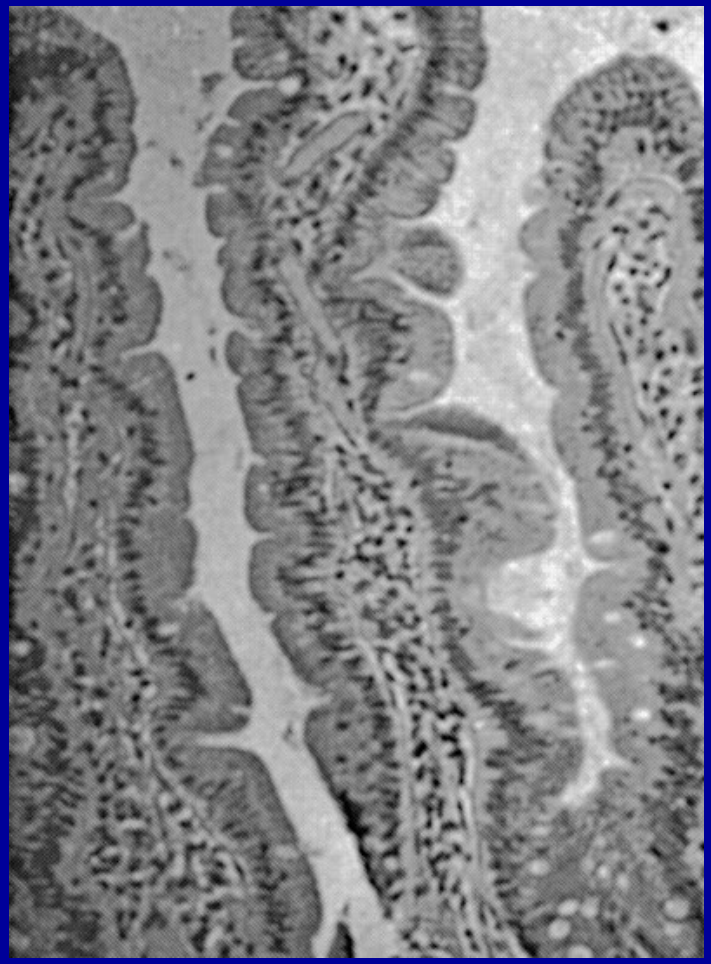
- Defective absorption of fat, protein, carbohydrates, iron and water. Impaired absorption of fat soluble vitamins A, D, K. Osteomalatia. Protein loss from small intestine.
- Gluten is found in wheat, barley, oats, rye; filler in many prepared foods and medications.

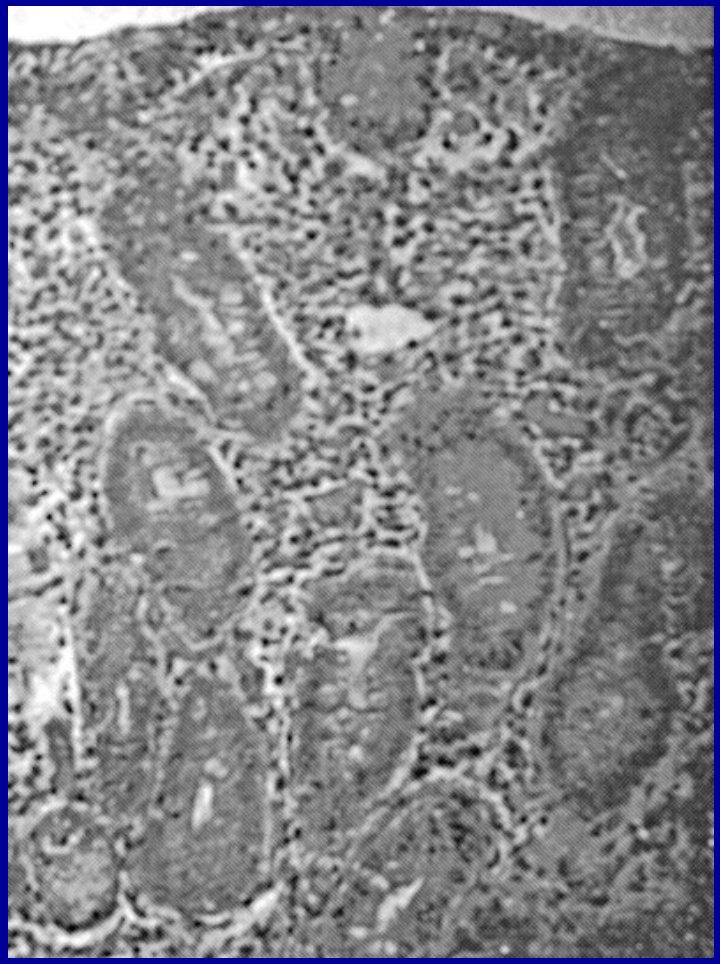
# Gluten-sensitive enteropathy 2

Clinical symptoms

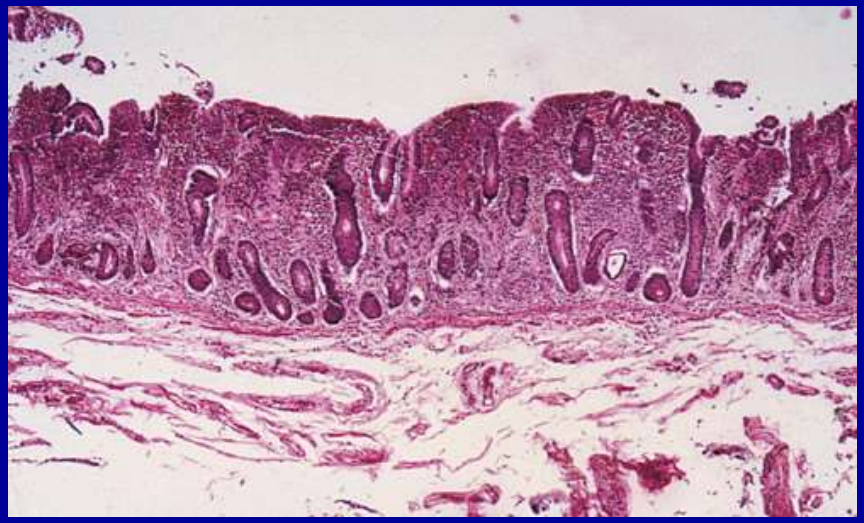
- In one third of patients with celiac sprue, symptoms begin in early childhood. Symptoms may persist into adult life, but there is usually a latent phase of apparent good health.
- Anemia: hypochromic, microcytic.
- Complications: infantilism, dwarfism, tetany, vitamin deficiency signs, rickets.
- Definitive diagnosis: quantitative measurements of fecal fat (preferably on a known fat intake), characteristic small bowel biopsy.
- Anti-endomysium, anti-transglutaminase antibodies
- Dermatitis herpetiformis: frequent association.

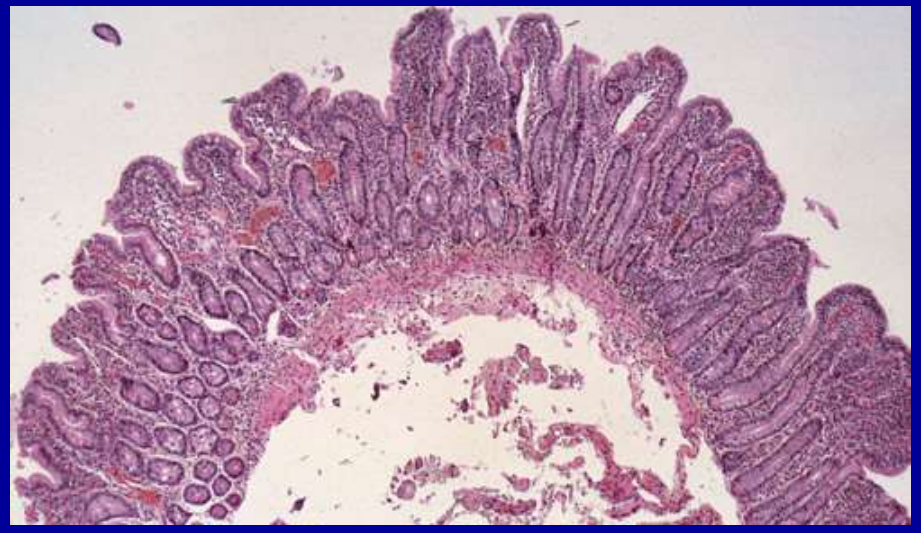
# Normal jejunal mucosa (left) and jejunal mucosa in celiac disease (right)





# Jejunal mucosal biopsy from patient with celiac sprue before and after treatment with a gluten-free diet





# Treatment of celiac sprue

- Strict elimination of gluten. If no response for gluten-free diet $\rightarrow$ ? collagenous sprue.
- Diet: gluten-free and initially lactose-free. High-calorie, high-protein, low-fat.
- Prothrombin deficiency: water-soluble vitamin K orally.
- Hypocalcemia or tetany: calcium phosphate or gluconate and vitamin D.
- Macrocytic anemia: vitamin B12
- Corticosteroids: in severe forms.