oligosaccharides from starch. The final hydrolysis to glucose monomers occurs at the brush border and includes disaccharide hydrolysis by sucrase and lactase. Glucose and galactose are actively transported in conjunction with sodium, whereas fructose absorption occurs by facilitated diffusion. About one half of dietary energy is derived from carbohydrate, with a nutritional goal of 55% and an increased component of insoluble fiber (i.e., that which is indigestible by mammalian enzymes but variably broken down by colonic bacteria.)

DIGESTION AND ABSORPTION OF PROTEINS

Dietary proteins are the major source for amino acids and the only source for the essential amino acids. Digestion starts in the stomach with pepsins secreted by the gastric mucosa, but most of the hydrolysis is accomplished by pancreatic enzymes in the proximal small bowel. The pancreas secretes the proteases trypsin, elastase, chymotrypsin, and carboxypeptidase as inactive proenzymes. Enterokinase (more properly, enteropeptidase) is secreted by the intestinal brush border; it splits trypsinogen to its active form, trypsin, which in turn converts the other proenzymes to their active forms. The products of luminal brush border peptidase digestion consist of amino acids and oligopeptides, which are transported across the epithelial cell. The transfer of most amino acids is sodium dependent and takes place in the proximal small bowel. Dietary requirements for amino acid nitrogen are met with about 15% of calories from protein.

MECHANISMS OF MALABSORPTION

The term *maldigestion* refers to defective hydrolysis of nutrients, whereas *malabsorption* refers to impaired mucosal absorption. In clinical practice, however, *malabsorption* refers to all aspects of impaired nutrient assimilation. Malabsorption can involve multiple nutrients, or it can be more selective. Therefore, the clinical manifestations of malabsorption are highly variable. The complete process of absorption consists of a *luminal phase*, in which various nutrients are hydrolyzed and solubilized; a *mucosal phase*, in which further processing takes place at the brush border of the epithelial cell with subsequent transfer into the cell; and a *transport phase*, in which nutrients are moved from the epithelium to the portal venous or lymphatic circulation. Impairment in any of these phases can result in malabsorption (Table 33-3).

Luminal Phase

Digestion is accomplished for the most part by pancreatic enzymes, particularly lipase, colipase, and trypsin; the gastric digestive enzymes do not play a major role. As a consequence, chronic pancreatitis can result in malabsorption, particularly for fat and protein. Deficiency in bile salts also contributes to fat malabsorption and may result from cholestatic liver disorders (impaired secretion of bile), bacterial overgrowth (resulting in luminal bile salt deconjugation), or ileal disease or resection with loss of effective enterohepatic circulation of the bile acids. The major part of the luminal phase of digestion occurs in the duodenum and the proximal jejunum.

Mucosal Phase

Mucosal disease is a more common cause of malabsorption. It can result from diffuse small intestinal diseases such as celiac disease or Crohn's disease or from a decrease in surface area (e.g., after surgical resection for small bowel infarction). The net effect is a smaller effective mucosal surface and a relative loss of mucosal absorption. Selective defects in an otherwise normal intestine may result in specific entities such as lactase deficiency or abetalipoproteinemia.

Transport Phase

After absorption, nutrients leave the cells through venous or lymphatic channels. Consequently, malabsorption may be associated with mesenteric venous obstruction, lymphangiectasia, or lymphatic obstruction due to malignancy or infiltrative processes such as Whipple's disease.

TABLE 33-3 PATHOPHYSIOLOGIC MECHANISMS IN MALABSORPTION

LUMINAL PHASE	MUCOSAL PHASE	TRANSPORT PHASE
Reduced nutrient availability	Extensive mucosal loss (resection or infarction)	Vascular conditions (vasculitis; atheroma)
Cofactor deficiency (pernicious anemia; gastric surgery)	Diffuse mucosal disease (celiac disease)	Lymphatic conditions (lymphangiectasia; irradiation; nodal tumor, cavitation, or infiltrations)
Nutrient consumption (bacterial overgrowth)	Crohn's disease; irradiation; infection; infiltrations; drugs: alcohol, colchicine, neomycin, iron salts	
Impaired fat solubilization	Brush border hydrolase deficiency (lactase deficiency)	
Reduced bile salt synthesis (hepatocellular disease)	Transport defects (Hartnup cystinuria; vitamin B_{12} and folate uptake)	
Impaired bile salt secretion (chronic cholestasis)	Epithelial processing (abetalipoproteinemia)	
Bile salt inactivation (bacterial overgrowth)		
Impaired cholecystokinin release (mucosal disease)		
Increased bile salt losses (terminal ileal disease or resection)		
Defective nutrient hydrolysis		
Lipase inactivation (Zollinger-Ellison syndrome)		
Enzyme deficiency (pancreatic insufficiency or cancer)		
Improper mixing or rapid transit (resection; bypass; hyperthyroidism)		

Modified from Riley SA, Marsh MN: Maldigestion and malabsorption. In Feldman M, Scharschmidt BF, Sleisenger MH, editors: Sleisenger and Fordtran's gastrointestinal and liver disease: pathophysiology/diagnosis/management, ed 6, Philadelphia, 1998, WB Saunders, pp 1501–1522.