SOME MANIFESTATIONS OF MALABSORPTION IN DISEASE

James D. Jones, Ph.D.
Section of Clinical Chemistry, Department of Laboratory Medicine, Mayo Clinic

Mary A. Jones, Ph.D.
Department of Dietetics, Saint Marys Hospital Rochester, Minnesota

INTRODUCTION

The term malabsorption has come to include both defective digestion and absorption. All ingested nutrients, i.e., fat, carbohydrate, proteins, minerals, H2O, and vitamins, normally enter the body via the gastrointestinal tract. It must be remembered that the absorbing surface of the digestive tract is not a one-way street. Therefore, in addition to oral food intake, a significant load of 'endogenous nutrients' is added to the intestinal contents of the alimentary canal. Complex nutrient molecules of both exogenous and endogenous origin undergo stepwise degradation to absorbable form and are transported into the intestinal cell also in a stepwise manner. Consideration of these steps and appreciation that absorbed nutrients exit from the intestinal cell, via either the portal vein or the lymphatic system, are essential to understanding malabsorption.

Equally important, though not as well appreciated, are the nutrient interactions and discriminations which occur not only in the G.I. tract but in other tissues. It would be highly desirable from the laboratory point of view to be able to relate specific tests to specific problems. However, malabsorption problems frequently present as multiple nutrient deficiencies of chronic syndromes, seldom as deficiencies of single nutrients, which have been characterized so well by the nutritional biochemists of the 40's and 50's.
Ingested food has been subjected to maceration, acidification, denaturation, and dilution by the time it leaves the stomach. Little digestion occurs until it is exposed in the small intestine to pancreatic enzymes and, in the case of dietary fat, solubilized with conjugated bile acids. The final steps in digestion of carbohydrates and protein occur on and within intestinal epithelial cells yielding simple compounds to be removed via the portal system. The micelles formed within the intestinal lumen from bile salts, fatty acids and 3-monoglycerides disaggregate at the epithelial surface. The bile salts remain within the lumen, available for reuse, while lipid components readily pass the lipophilic brush border membrane. Products of fat digestion, long chain fatty acids (>C10) and their mono and diglycerides, must be modified to triglycerides, packed with other lipids within a lipoprotein envelope to form chylomicra before they can exit from the cell to be carried away via the lymph. Short chain fatty acids (called medium chain in medical literature) are absorbed directly without modification and removed via the portal vein.

Interference with any of these steps essential to digestion, absorption and transport usually results in malabsorption with accompanying steatorrhea and diarrhea. Dehydration due to electrolyte and water loss may become rapidly critical, particularly in infants and young children because of their high extracellular water. In addition there is evidence that the bowel of the infant is more permeable and susceptible to osmotic loads than the bowel of adults (Younoszai et al., 1978).

**ABNORMALITIES ASSOCIATED WITH MALABSORPTION**

Most abnormalities associated with malabsorption are readily classified into those of intraluminal phase, mucosal cell transport and intestinal lymphatic transport of fat. Each of these plus an unclassified category will be considered separately.

**Intraluminal Phase**

Hydrolysis of dietary triglyceride by pancreatic lipase must precede normal absorption. Lipase at pH 6 to 7 in the presence of conjugated bile salts solubilizes the lipid in the form of monoglyceride, fatty acids and fat soluble vitamins into micelles. A defect in any one of these steps leads to defective fat absorption which is evident by an increased excretion of fecal fat (steatorrhea).

Along with malabsorption of fat, one expects malabsorption of fat soluble vitamins which may be seen in increased prothrombin time where vitamin K is not absorbed, and in hypocalcemia and tetany.