

Table 31-1 Etiology of Vitamin and Nutrient Deficiency States

ETIOLOGY	DEFICIENCY
DIET	
Vegans (strict)	Protein, vitamins B ₁₂ , D, riboflavin, iron
Breastfed infant	Vitamins K, D
Cow's milk–fed infant	Iron
Bulimia, anorexia nervosa	Electrolytes, other deficiencies
Parenteral alimentation	Essential fatty acids, trace elements
Alcoholism	Calories, vitamin B ₁ , B ₆ , folate
MEDICAL PROBLEMS	
Malabsorption syndromes	Vitamins A, D, E, K, zinc, essential fatty acids
Cholestasis	Vitamins E, D, K, A, zinc, essential fatty acids
MEDICATIONS	
Sulfonamides	Folate
Phenytoin, phenobarbital	Vitamins D, K, folate
Mineral oil	Vitamins A, D, E, K
Antibiotics	Vitamin K
Isoniazid	Vitamin B ₆
Antacids	Iron, phosphate, calcium
Digitalis	Magnesium, calcium
Penicillamine	Vitamin B ₆
SPECIFIC MECHANISMS	
Transcobalamin II or intrinsic factor deficiency	Vitamin B ₁₂
Other digestive enzyme	Carbohydrate, fat, protein deficiencies
Menkes kinky hair syndrome	Copper
Acrodermatitis enteropathica	Zinc
Reduced exposure to direct sunlight	Vitamin D

and vegetables. Subperiosteal hemorrhage, bleeding gums and petechiae, hyperkeratosis of hair follicles, and a succession of mental changes characterize the progression of the illness. Anemia secondary to bleeding, decreased iron absorption, or abnormal folate metabolism is also seen in chronic scurvy. Treatment is noted in [Table 31-3](#).

B Vitamins



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Anemia
Neutropenia

The B vitamins thiamine, riboflavin, and niacin are routinely added to enriched grain products; deficiencies in normal hosts

are rare in the United States. Levels from human milk reflect maternal intake, and deficiency can develop in breastfed infants of deficient mothers.

Thiamine

Vitamin B₁ functions as a coenzyme in biochemical reactions related to carbohydrate metabolism, decarboxylation of α -ketoacids and pyruvate, and transketolase reactions of the pentose pathway. Thiamine also is involved in the decarboxylation of branched-chain amino acids. Thiamine is lost during milk pasteurization and sterilization.

Thiamine deficiency occurs in alcoholics and has been reported in adolescents who have undergone bariatric surgery for severe obesity. **Infantile beriberi** occurs between 1 and 4 months of age in breastfed infants whose mothers have a thiamine deficiency (alcoholism), in infants with protein-calorie malnutrition, in infants receiving unsupplemented hyperalimentation fluid, and in infants receiving boiled milk. Acute **wet beriberi** with cardiac symptoms and signs predominates in infantile beriberi. Anorexia, apathy, vomiting, restlessness, and pallor progress to dyspnea, cyanosis, and death from heart failure. Infants with beriberi have a characteristic aphonic cry; they appear to be crying, but no sound is uttered. Other signs include peripheral neuropathy and paresthesias. For treatment see [Table 31-3](#).

Riboflavin

Vitamin B₂ is a constituent of two coenzymes, riboflavin 5'-phosphate and flavin-adenine dinucleotide, essential components of glutathione reductase and xanthine oxidase, which are involved in electron transport. A deficiency of riboflavin affects glucose, fatty acid, and amino acid metabolism. Riboflavin and its phosphate are decomposed by exposure to light and by strong alkaline solutions.

Ariboflavinosis is characterized by an angular stomatitis; glossitis; cheilosis; seborrheic dermatitis around the nose and mouth; and eye changes that include reduced tearing, photophobia, corneal vascularization, and the formation of cataracts. Subclinical riboflavin deficiencies have been found in diabetic subjects, children in families with low socioeconomic status, children with chronic cardiac disease, and infants undergoing prolonged phototherapy for hyperbilirubinemia.

Niacin

Niacin consists of the compounds nicotinic acid and nicotinamide (niacinamide). Nicotinamide, the predominant form of the vitamin, functions as a component of the coenzymes nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP). Niacin is involved in multiple metabolic processes, including fat synthesis, intracellular respiratory metabolism, and glycolysis.

In determining the needs for niacin, the content of tryptophan in the diet must be considered because tryptophan is converted to niacin. Niacin is stable in foods and withstands heating and prolonged storage. Approximately 70% of the total niacin equivalents in human milk are derived from tryptophan. **Pellagra**, or niacin deficiency disease, is characterized by weakness, lassitude, dermatitis, photosensitivity, inflammation of mucous membranes, diarrhea, vomiting, dysphagia, and, in severe cases, dementia.