reductase are discussed in the text.

■ NUTRITIONAL

Dietary folate deficiency is common. Indeed, in most patients with folate deficiency, a nutritional element is present. Certain individuals are particularly prone to have diets containing inadequate amounts of folate (Table 99-5). In the United States and other countries where fortification of the diet with folic acid has been adopted, the prevalence of folate deficiency has dropped dramatically and is now almost restricted to high-risk groups with increased folate needs. Nutritional folate deficiency occurs in kwashiorkor and scurvy and in infants with repeated infections or those who are fed solely on goats' milk, which has a low folate content.

■ MALABSORPTION

Malabsorption of dietary folate occurs in tropical sprue and in gluten-induced enteropathy. In the rare congenital recessive syndrome of selective malabsorption of folate due to mutation of the PCFT, there is an associated defect of folate transport into the cerebrospinal fluid, and these patients show megaloblastic anemia, which responds to physiologic doses of folic acid given parenterally but not orally. They also show mental retardation, convulsions, and other central nervous system abnormalities. Minor degrees of malabsorption may also occur after jejunal resection or partial gastrectomy, in Crohn's disease, and in systemic infections, but in these conditions, if severe deficiency occurs, it is usually largely due to poor nutrition. Malabsorption of folate has been described in patients receiving sulfasalazine (Salazopyrin), cholestyramine, and triamterene.

■ EXCESS UTILIZATION OR LOSS

Pregnancy Folate requirements are increased by 200–300 μg to ~400 μg daily in a normal pregnancy, partly because of transfer of the vitamin to the fetus but mainly because of increased folate catabolism due to cleavage of folate coenzymes in rapidly proliferating tissues. Megaloblastic anemia due to this deficiency is prevented by prophylactic folic acid therapy. It occurred in 0.5% of