NORMAL VALUES

Qualitative Fecal Fat

Neutral fat	<60 fat globules per high powered field
Fatty Acids	<100 fat globules per high powered field

Quantitate Fecal Fat

Infant, breast fed	<1 g/ 24 hr and 10-40% of total solids
Infant, bottle fed	<1 g/ 24 hr and 30-50% total solids
Child	<2 g/ 24 hr
Adult	2-7 g/24 hr and < 20% total solids

Coefficient of fat absorption (%)=
(Fat ingested – Fat excreted/Fat ingested) X 100

NUTRITIONAL SIGNIFICANCE

Fecal fat is the standard test for diagnosing malabsorption and steatorrhea. The primary etiologies of steatorrhea are:

- Impairment in intestinal absorption
- Deficiency of pancreatic enzymes
- Deficiency of bile

Specimens collected over a 72 hour period are examined for excessive fecal fat. The test is used for patients who are suspected of having steatorrhea and for patients undergoing treatment for malabsorption disorders. Since fat is not released into the stool at a constant rate, a 72 hour collection provides a more accurate picture of average absorption and elimination than in a single fecal sample.

For the quantitative fat test, the patient is required to eat a moderately high amount of fat per day prior to and during the sample collection. A positive qualitative fecal fat test indicates an increased amount of fat in a 72 hr sample. A positive quantitative fecal fat test indicates that the fat is probably not being absorbed due to impaired or maldigestion.

STOOL FAT

RELATED TESTS: celiac disease antibody tests, fecal elastase, fecal occult blood test, trypsin and chymotrypsin, xylose absorption test

INTERFERING FACTORS

- Ingestion of castor oil or mineral oil may cause increased neutral fat in stool.
- Ingestion of dietetic low calorie mayonnaise or oily salad dressings may cause increased neutral fat in stool.
- Ingestion of diet containing > 100 g dietary fiber/day may cause increased neutral fat in stool.
- Stool must not be contaminated with urine.

Increased with:

- Atrophy of malnutrition
- Celiac disease
- Crohn's disease
- Cystic fibrosis
- Gallbladder cancer
- IBS
- Infections (parasitic, bacterial, or viral)
- Narrowing or blockage of common bile duct
- Pancreatic cancer
- Pancreatic diseases associated with lack of lipase
- Regional enteritis
- Resection of small bowel
- Shwachman-Diamond Syndrome
- Sprue

THIAMINE (B1) (THIAMINE DIPHOSPHATE TDP) Blood & Urine

NORMAL VALUES: Whole Blood/Erythrocyte
Adults: 2.5-7.5 mcg/dL; 74-222 nmol/L. (SI)

NORMAL VALUES: Plasma
Adults: 4-15 nmol/L. (SI)

NORMAL VALUES: Transketolase
Adults: >150 nmol/L (SI)

THIAMINE (B1) (THIAMINE DIPHOSPHATE TDP)Blood & Urine

NORMAL VALUES: Erythrocyte Transketolase Activity

Adults:	0-15%

NORMAL VALUES: Urinary thiamine

Adults:	>100 mcg/24 hr

CRITICAL VALUES

Whole Blood/Erythrocyte	<70 nmol/L. (SI)
Erythrocyte Transketolase Activity	>20%
Urinary thiamine	<40 mcg/24 hr
Urinary thiamine/creatinine ratio	<27 mcg/g creatinine

NUTRITIONAL SIGNIFICANCE

Thiamine is a water soluble vitamin absorbed in the proximal jejunum by active transport. Erythrocytes contain 80-90 percent of the total body thiamine stores, and are estimated to be about 30 mg. Healthy adults use about 1-2 mg daily. Thiamine has a half-life of 9-18 days. Depletion of thiamine stores can take place in 14-30 days.

Thiamine pyrophosphate is essential for the proper transfer of the aldehyde groups, and it is an essential coenzyme for glycolytic and pentose pathways of glucose metabolism. Enzymes that require thiamine include pyruvate dehydrogenase, α -ketoglutarate dehydrogenase, transketolase, and branched-chain α -ketoacid dehydrogenase. Deficiency leads to decreased transketolase activity in erythrocytes and increased levels of pyruvic acid in the blood. The pyruvic acid is not converted to acetyl-coA and cannot enter the Krebs cycle for aerobic oxidative metabolism. High levels of pyruvic acid are metabolized anaerobically to lactic acid.

There is an interrelationship between thiamine and folate status. Thiamine pyrophosphate (coenzyme form) is regenerated via the transfer of a proton from the reduced form of NADH. Folate as dihydrofolate reductase is required to regenerate NADH from its

oxidative form. This allows NADH to be present to regenerate thiamine pyrophosphate. If folate is deficient in cells, it causes an indirect thiamine deficiency because while thiamine is present it cannot be activated.

Persistent vomiting, a deficient diet, or excessive utilization can deplete thiamine stores rapidly. Glucose infusions given to a patient with depleted thiamine status will worsen symptoms and may cause permanent cognitive and neuromuscular impairments.

Worldwide, the primary cause of thiamine deficiency is due to inadequate intake (e.g. diet of highly refined carbohydrates). The prevalence of thiamine deficiency is higher in populations that consume milled rice that is not fortified with thiamine as well as foods containing thiaminases that inactivate thiamine. Raw fish, shell fish, coffee, and tea contain thiaminases. In developed nations, the primary cause of thiamine deficiency is alcoholism or chronic illness (e.g. cancer). Secondary thiamine deficiency is caused by increased demand, impaired absorption, or impaired metabolism. In alcoholics, thiamine deficiency may occur due to decreased intake, impaired absorption, increased demand, and possibly an apoenzyme defect.

Thiamine deficiency can cause wet beriberi or dry beriberi. Heart failure is the primary symptom of wet beriberi. The first effects are vasodilation, tachycardia, a wide pulse pressure, sweating, warm skin, and lactic acidosis. Later, heart failure develops, causing orthopnea and pulmonary and peripheral edema. Vasodilation can continue, sometimes resulting in shock.

Dry beriberi refers to peripheral neurologic deficits due to thiamine deficiency. These deficits affect predominantly the lower extremities, beginning with paresthesias in the toes, burning in the feet, heaviness in legs, muscle cramps, and tenderness in the