

Overview

Useful For

- Diagnosing vitamin A deficiency and toxicity
- Evaluating persons with intestinal malabsorption of lipids
- Evaluating individuals with motor and sensory neuropathies for vitamin E deficiency
- Monitoring vitamin E status of premature infants requiring oxygenation

Profile Information

Test Id	Reporting Name	Available Separately	Always Performed
VITAP	Vitamin A, S	Yes, (Order VITA)	Yes
VITE	Vitamin E, S	Yes	Yes

Method Name

Liquid Chromatography-Tandem Mass Spectrometry (LC-MS/MS)

NY State Available

Yes

Specimen

Specimen Type

Serum

Shipping Instructions

Ship specimen in amber vial to protect from light.

Specimen Required

- Patient Preparation:** Fasting overnight (12-14 hours) (infants-collect specimen prior to next feeding)
- Supplies:** Amber Frosted Tube, 5 mL(T915)
- Collection Container/Tube:**
- Preferred:** Red top
- Acceptable:** Serum gel
- Submission Container/Tube:** Amber vial
- Specimen Volume:** 1 mL
- Collection Instructions:** Within 24 hours of collection, centrifuge, and aliquot serum into an amber vial to protect from light.

Specimen Minimum Volume

0.5 mL

Reject Due To

Gross hemolysis	Reject
Gross lipemia	Reject
Gross icterus	OK

Specimen Stability Information

Specimen Type	Temperature	Time	Special Container
Serum	Refrigerated (preferred)	28 days	LIGHT PROTECTED
	Frozen	28 days	LIGHT PROTECTED
	Ambient	7 days	LIGHT PROTECTED

Clinical & Interpretive

Clinical Information

Vitamin A:

The level of vitamin A in the plasma or serum is a reflection of the quantities of vitamin A and carotene ingested and absorbed by the intestine (carotene is converted to vitamin A by intestine absorptive cells and hepatocytes).

Vitamin A plays an essential role in the function of the retina (adaptation to dim light), is necessary for growth and differentiation of epithelial tissue, and is required for growth of bone, reproduction, and embryonic development. Together with certain carotenoids, vitamin A enhances immune function, reducing the consequences of some infectious diseases.

Degenerative changes in eyes and skin are commonly observed in vitamin A deficiency. Poor adaptation of vision to darkness (night blindness) is an early symptom that may be followed by degenerative changes in the retina. In developing countries, vitamin A deficiency is the principal preventable cause of blindness. Severe or prolonged deficiency leads to dry eye (xerophthalmia), which can result in corneal ulcers, scarring, and blindness. Another important consequence of inadequate intake is acquired immunodeficiency disease, with an increased incidence of death related to infectious diseases. In patients with HIV, vitamin A deficiency is associated with increased disease progression and mortality.

Vitamin A in excess can be toxic. In particular, chronic vitamin A intoxication is a concern in normal adults who ingest more than 15 mg per day and in children who ingest more than 6 mg per day of vitamin A over a period of several months. Manifestations are various and include dry skin, cheilosis, glossitis, vomiting, alopecia, bone demineralization and pain, hypercalcemia, lymph node enlargement, hyperlipidemia, amenorrhea, and features of pseudotumor cerebri with increased intracranial pressure and papilledema. Liver fibrosis with portal hypertension and bone demineralization may also result. Congenital malformations, like spontaneous abortions, craniofacial abnormalities, and valvular heart

disease have been described in pregnant women taking vitamin A in excess. Consequently, in pregnancy, the daily dose of vitamin A should not exceed 3 mg.

Vitamin E (alpha-tocopherol):

Vitamin E contributes to the normal maintenance of biomembranes, the vascular and nervous systems, and provides antioxidant protection for vitamin A. The level of vitamin E in the plasma or serum after a 12- to 14-hour fast reflects the individual's reserve status.

The current understanding of the specific actions of vitamin E is very incomplete. The tocopherols (vitamin E and related fat-soluble compounds) function as antioxidants and free-radical scavengers, protecting the integrity of unsaturated lipids in the biomembranes of all cells and preserving retinol from oxidative destruction. Vitamin E is known to promote the formation of prostacyclin in endothelial cells and to inhibit the formation of thromboxanes in thrombocytes, thereby minimizing the aggregation of thrombocytes at the surface of the endothelium. Those influences on thrombocyte aggregation may be of significance in relation to risks for coronary atherosclerosis and thrombosis.

Deficiency of vitamin E in children leads to reversible motor and sensory neuropathies; this problem has also been suspected in adults. Premature infants who require an oxygen-enriched atmosphere are at increased risk for bronchopulmonary dysplasia and retrolental fibroplasia. Supplementation with vitamin E has been shown to lessen the severity of, and may even prevent, those problems.

Deficiencies of vitamin E may arise from poor nutrition or from intestinal malabsorption. At-risk persons, especially children, include those with bowel disease, pancreatic disease, chronic cholestasis, celiac disease, cystic fibrosis, and intestinal lymphangiectasia. Infantile cholangiopathies that may lead to malabsorption of vitamin E include intrahepatic and extrahepatic biliary atresia, paucity of intrahepatic bile ducts, arteriohepatic dysplasia, and rubella-related embryopathy. In addition, low blood levels of vitamin E may be associated with abetalipoproteinemia, presumably due to a lack of the ability to form very low-density lipoproteins and chylomicrons in the intestinal absorptive cells of affected persons.

Vitamin E toxicity has not been established clearly. Chronically excessive ingestion has been suspected as a cause of thrombophlebitis, although this has not been definitively verified.

Deficiencies of vitamins A and E may arise from poor nutrition or from intestinal malabsorption. Persons at risk, especially children, include those with bowel disease, pancreatic disease, chronic cholestasis, celiac disease, cystic fibrosis, and intestinal lymphangiectasia. Infantile cholangiopathies that may lead to malabsorption of vitamins A and E include intrahepatic dysplasia and rubella-related embryopathy.

Reference Values

VITAMIN A (RETINOL)

0-6 years: 11.3-64.7 mcg/dL

7-12 years: 12.8-81.2 mcg/dL

13-17 years: 14.4-97.7 mcg/dL

> or =18 years: 32.5-78.0 mcg/dL

VITAMIN E (ALPHA-TOCOPHEROL)

0-17 years: 3.8-18.4 mg/L

> or =18 years: 5.5-17.0 mg/L

Interpretation

Vitamin A:

The World Health Organization recommends supplementation when vitamin A levels fall below 20.0 mcg/dL. Severe deficiency is indicated at levels less than 10.0 mcg/dL. Vitamin A values above 120.0 mcg/dL suggest hypervitaminosis A and associated toxicity.

Vitamin E (alpha-tocopherol):

Therapeutic Ranges:

0-17 years: 3.8 - 18.4 mg/L

> or =18 years: 5.5 - 17.0 mg/L

Significant deficiency: <3.0 mg/L

Cautions

Testing of nonfasting specimens or the use of vitamin supplementation can result in elevated serum vitamin concentrations. Reference values were established using specimens from individuals who were fasting.

Acute ethanol ingestion may result in increased serum vitamin A levels.

Clinical Reference

1. Ball GFM: Vitamins: Their role in the human body. Blackwell Publishing; 2004:234-255
2. Ross AC: Vitamin A and carotenoids. In: Shils ME, Shike M, Ross MC, et al: Modern Nutrition in Health and Disease. 10th ed. Lippincott Williams and Wilkins; 2006:351-375
3. Traber MG: Vitamin E. In: Shils ME, Shike M, Ross AC, et al, eds. Modern Nutrition in Health and Disease. 10th ed. Lippincott Williams and Wilkins; 2006:434-441
4. Roberts NB, Taylor A, Sodi R: Vitamins and trace elements. In: Rifai N, Horvath AR, Wittwer CT, eds. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics. 6th ed. Elsevier; 2018:chap37

Performance**Method Description**

Deuterated vitamin A (d6-all-trans retinol) is added to serum as an internal standard. Vitamin A (all-trans retinol) and the deuterated internal standard are extracted from the specimens using on-line turbulent flow high performance liquid chromatography and analyzed by liquid chromatography-tandem mass spectrometry using multiple reaction monitoring in positive mode.(Unpublished Mayo method)

Deuterated vitamin E (d6-alpha-tocopherol) is added to serum as an internal standard. Vitamin E (alpha-tocopherol) and the deuterated internal standard are extracted from the specimens and analyzed by liquid chromatography-tandem mass spectrometry.(Unpublished Mayo method)

PDF Report

No

Day(s) Performed

Monday through Friday

Report Available

2 to 5 days

Specimen Retention Time

14 days

Performing Laboratory Location

Rochester

Fees & Codes

Fees

- Authorized users can sign in to [Test Prices](#) for detailed fee information.
- Clients without access to Test Prices can contact [Customer Service](#) 24 hours a day, seven days a week.
- Prospective clients should contact their account representative. For assistance, contact [Customer Service](#).

Test Classification

This test was developed and its performance characteristics determined by Mayo Clinic in a manner consistent with CLIA requirements. It has not been cleared or approved by the US Food and Drug Administration.

CPT Code Information

84446

84590

LOINC® Information

Test ID	Test Order Name	Order LOINC® Value
VITAE	Vitamin A and Vitamin E, S	96600-2

Result ID	Test Result Name	Result LOINC® Value
2350	A-Tocopherol, Vitamin E	1823-4
605124	Vitamin A	2923-1