supply. Therapeutic large doses (5 - 10 mg) are applied to infants with seborrhea or genetic alteration of biotin dependent enzymes, no toxicity has been reported so far.

**Sampling:** 2 mL serum

**Reference Interval:** > 200 ng/L
< 100 ng/L interpreted as a biotin deficiency

## Vitamin K-1, Serum f

**Related Information:** Factor II Mutation (Prothrombin Mutation)
Factor V Mutation (Leiden Mutation)
Protein C
Protein S, Total

**Background:** Vitamin K is a fat soluble vitamin, essential for the synthesis of clotting factors by the liver as a co factor in carboxylation of glutamic acid residues to form gamma-carboxyglutamic acid. Since bile salts are necessary for absorption, an obstruction of the bile ducts may cause vitamin K deficiency. Besides dietary intake, the vitamin is also synthesized by intestinal bacteria; antibiotic treatment may cause a deficiency. Vitamin K deficiency is characterized by decrease of factor II, VII, IX, X, Protein C and Protein S. Prolongation of PT occurs.
Coumarin blocks vitamin K dependent carboxylation, therefore, according to the half life time of the clotting factors, factor VII, and Protein C in the serum decreases first, thereafter factor X, II and IX .
Cephalosporins interfere directly with vitamin K regeneration.

**Sampling:** 2 mL serum

**Reference Interval:** 50 - 900 ng/L

## Willebrand factor, Plasma f

**Related Information:** Coagulation factors
Bleeding disorders
Willebrand Disease

**Synonyms:** von Willebrand factor (vWF)

**Background:** Willebrand factor (WF) regulates important steps in primary and secondary hemostasis. Deficiency or functional defects of WF lead to bleeding disorders affecting both plasmatic coagulation as well as platelet function. In rare cases also increased risk of thrombosis may occur. Furthermore, WF deficiency is involved in thrombotic thrombocytopenic purpura or hemolytic-uremic syndrome. Deficiency of WF also leads to increased turnover rate of coagulation factor VIII resulting in low levels of this factor.
Both hereditary and acquired defects of WF are known, leading to bleeding disorders involving skin, mucosa and gingival bleedings, nosebleeds, menorrhagia or gastrointestinal and urinary tract bleedings. Please note that persons with blood group 0 reveal lower levels of WF without

signs of bleeding disorders.

Several test systems are available to measure WF concentration and function. MEDLAB offers you state of the art, step-by-step diagnostic tools to identify and classify the type of Willebrand Disease in your patient.

**Sampling:** 2 mL of citrate plasma

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|---|---|---|
| **Reference Interval:** | WF antigen | 50 - 160 % |
| | WF activity | 60 - 170 % |
| | WF activity (blood group 0) | 46 - 150 % |

## Xylose Absorption Test, Serum f

**Related Information:** Endomysial Antibodies
Gliadin IgG/IgA Antibodies

**Synonyms:** D-Xylose Absorption Test, Serum,

**Background:** D-xylose is absorbed in the duodenum and jejunum and excreted by the kidney. The test screens for carbohydrate malabsorption and differentiates from pancreatic insufficiency, since pancreatic enzymes are not necessary for xylose absorption. Diseases such as celiac disease, tropical sprue, M. Crohn, surgical bowl resection impair xylose resorption.

Sampling: Patient should be fasting at least for 4 h and remain in a supine position during the test. Patient should be withdrawn from interfering medications (aspirin, indomethacin, neomycin, glipizide, atropine). Draw first sample (1 mL serum) before administer 25 g xylose orally in water, 10% w/v in adults. In children use 0.5 g/kg body weight. Draw second (1 mL serum) sample after 60 minutes.

| | | |
|---|---|---|
| **Reference Interval:** | Adult,1 h, 25 g of xylose | > 25.mg/dL |
| | Adult,1 h, 25 g of xylose, renal insufficiency | > 20 mg/dL |
| | Adult,1 h, 5 g dose of xylose | 20 - 40 mg/dL |
| | Children < 12 years, 1 h , 5g dose | > 20 mg/dL |

## Yersinia enterocolitica and Yersinia pseudotuberculosis, Culture and Serology f

**Background:** Yersinia enterocolitica and Yersinia pseudotuberculosis are gram negative oval rods. Transmission occur by contamination of food (milk, water, meat) with excreta from the reservoir animals such as pigs, goats, sheep, dogs, cats. Y. enterocolitica causes enterocolitis that is clinically indistinguishable from that caused by Salmonella or Shigella. It is characterized by abdominal pain, gastroenteritis and possibly bloody diarrhea. Both Yersinia sp. can cause an acute appendicitis resembling mesenteric adenitis. Yersinia infection may be associated with reactive arthritis and Reiter's syndrome, but Salmonella spp., Shigella spp. and Campylobacter spp. may also trigger these autoimmune diseases.

Limitations: Low antibody titers of IgG class may persist for years.