

Lysozyme, Blood or Urine or CSF

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Synonyms: Muramidase

Background: Lysozyme is present in neutrophil granules, in leukemic and normal eosinophils. The test is used in the differentiation of leukemia. Lysozyme is present in the M4 and M4 type of acute myeloid leukemia, occasionally in the M1, M2, and M6 type. It has been found to correlate with the degree of differentiation of monocytes in leukemia.

Also used as a marker in monitoring sarcoidosis, and it may be elevated in tuberculosis.

Sampling: Serum: 1 mL, separate serum or plasma and freeze immediately.

Urine: 5 mL aliquot of a 24 h collected urine shipped frozen and collected on ice.

A random urine sample is suitable, too.

CSF (Cerebrospinal fluid): 0.5 mL

Reference Interval:

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|--------|----------------|
| Serum: | 4 - 15.6 µg/mL |
| Urine: | 0 - 1.4µg/mL |
| CSF: | < 1.5 µg/mL |

M2 – PK, Feces

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Related Information: CA 19-9, Serum (Gastrointestinal)

Background: M2-PK is an isoenzyme of pyruvate kinase (PK), expressed in proliferating and in tumor cells. PK occurs in a tetrameric form and in a dimeric form. In tumor cells, the dimeric form (tumor M2-PK) is predominant. Since tumors of the gastrointestinal tract grow into the lumen, tumor M2-PK is detectable in the feces of patients with GI malignancies.

Colorectal cancer: Sensitivity for detection of colorectal cancer or polyps was shown to be 27% and 10% for the occult blood (Guajak), 91% and 19% for the immunological test for occult blood and 73% - 77% and 48% for the M2-PK-test, respectively. Specificity was 89%, 94% and 72%, respectively, indicating that M2-PK display a lower specificity in diagnosing cancer.

TNM and Dukes' classification of the tumors correlates strongly with faecale M2-PK levels.

Gastric cancer: Compared to controls, samples of patients with inflammatory bowel disease or different types of gastrointestinal tumors did not show significant differences, but up to 80% of patients with gastric cancer present elevated M2-PK.

Sampling: approx. 2 g stool

Reference Interval: < 4 U/mL

M-N

Magnesium (Mg), Serum

a

Related Information:

- Calcium (Ca), Total, Serum or Urine
- Digoxin, Serum
- Magnesium, Urine
- Potassium, Urine
- Vancomycin, Serum

Background: Magnesium is the fourth most abundant cation in the body with an amount of approx. 22 g behind sodium, potassium and calcium and the second most prevalent intracellular cation. Half of the amount is located in the bone, the other half in soft tissue. Extracellular Mg accounts only for 1% of total body Mg (TBMg). In the serum, approx 55% is unbound, 30% is albumin associated and 15% is complexed with phosphate, citrate or other anions.

Magnesium is essential for the function of approx. 300 enzymes, in DNA replication, mRNA translation. Mg is necessary in membrane stabilization, energy metabolism (ATP), and maintaining potassium balance.

TBMg depends on gastrointestinal absorption and renal excretion. Dietary intake is estimated to 300 - 350 mg/day, the renal excretion 120 - 140 mg/day by glomerular filtration of 70% - 80%. Useful in: Acute myocardial infarct, cardiac arrhythmias, hypokalemia, hyponatremia, during diuretic therapy, digoxin therapy, diarrhea, in the diagnosis of neuromuscular symptoms such as spasm, fasciculations, weakness, dizziness, tetany, convulsions.

Sampling: 1 mL serum or plasma, EDTA plasma is not acceptable. Avoid hemolysis, since erythrocytes contain threefold the concentration as compared to serum.

Reference Interval: 1.8 - 2.5 mg/dL

Since a fraction of Mg is bound to albumin, patients with hypalbuminemia may have values at the lower reference limit.

Magnesium (Mg), Urine

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Related Information: Calcium (Ca), Total, Serum, Urine
Cyclosporine A (monoclonal)
Digoxin, Serum
Magnesium, Serum
Oxalate, Urine
Potassium, Serum or Plasma
Vitamin D, Serum

Background: Urinary magnesium may serve as an early indicator for developing serum magnesium deficiency leading to hypocalcemia with cardiac arrhythmias.

Useful in the assessment of Mg deficiency in patients with gastrointestinal disorders such as Crohn disease or gut failure, in patients suffering from calcium oxalate kidney stones, since oxalate stone formation is related to urinary concentrations of oxalate and calcium but inversely related to urinary citrate and magnesium.

Elevated urinary excretion occurs in patients with elevated blood alcohol levels, diuretics, Bartter syndrome, Gitelman syndrome, aldosterone therapy or corticosteroids; in renal transplant patients on cyclosporine and prednisone.

Drugs reducing magnesium storage with high magnesium excretion: aminoglycosides, cyclosporine, pentamidine, foscarnet, amphotericin B.

Sampling: 5 mL aliquot of a 24 h urine collection recommended, due to circadian rhythm. Use

a clean plastic container for collection, avoid strictly contact of the urine with metals. Keep cool.
Note total quantity.

Reference Interval: 2.5 - 8.5 mmol/24 h

Malaria

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Related Information: Myoglobin Qualitative, Urine
Blood Count Complete

Test includes: Microscopic examination of thick and thin smears; antibodies to *P. falciparum* and *P. vivax*.

Background: Malaria is the most common infectious disease now due international travel frequently coming to attendance in non endemic areas.

Malaria is caused by four members of the species Plasmodium: *P. vivax*, *P. falciparum*, *P. malariae*, and *P. ovale*, transmitted by the insect vector of the genus Anopheles.

P. vivax causes tertian malaria with an incubation time of 10 - 14 days and a cycle length of 45 h.

P. falciparum causes malignant tertian, tropical malaria with an incubation time of 10 - 14 days and a cycle length of 48 h.

P. ovale causes malaria ovale with a cycle length of 48 h.

P. malariae causes quartan malaria with an incubation time of 18 - 42 days and a cycle length of 72 h.

Sampling:

Microscopy: 2 to 3 thin and 2 - 3 thick air dried smears made at bedside on oil free clean slides. To prepare a thick smear, spread a drop of blood without anticoagulants on 2 - 5 fold the area of the original drop on a slide using a corner of another slide. To prepare a thin slide, smear the drop over the entire area of the slide. Specimens should be prepared immediately before or during the start of a fever spike. Thick films have an increased sensitivity, but are more difficult to read. Optimal circumstances allow a sensitivity of 10 parasites per ul blood.

Serology: 3 mL serum for antibody test available for Plasmodium falciparum and *P. vivax*.

Important: Please note travel history and countries visited by the patient.

Reference Interval: Microscopy: Plasmodium not detectable
Limitations: Negative result does not rule out malaria.
Serology: Antibody negative for Plasmodium falciparum and *P. vivax*.

M-N

Marijuana see Cannabinoids (Marijuana Metabolites) Immunological Drug Screen, Urine

Measles (Morbilli), Serology

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Background: Measles, a paramyxovirus, causes infection of the lymphoreticular system and the respiratory tract. Clinically measles infection presents with maculopapular eruption, starting on the face and descending to the extremities including palms and soles, Koplik's spots, lymphadenopathy and cough. Measles are near elimination in countries with childhood vaccination programs. Due to maternal neutralizing antibodies vaccination not is performed after 15 month of age. Booster vaccination is strongly recommended.

Measles are transmitted by respiratory droplets, produced during the prodromal period and some days after the rash appears. The measles virus is highly infectious with an incubation time of 10 - 14 days. The rash is caused by cytotoxic T cells targeted to virus infected vascular endothelial cells in the skin. There is a 0.1% incidence of encephalitis with 40% permanent sequelae. Also measles and bacterial pneumonia and bacterial otitis media may occur.

Measles in pregnant women increases the risk of stillbirth and malformation of the baby. Immunity lasts life long, is mainly cell-mediated, but IgG antibody levels indicate immunity. Maternal immunity is transferred to the fetus and lasts for the first 6 month of life. Measles infection may depress cell mediated immunity, particularly against *Mycobacteria* sp.

Sampling: 2 mL serum

Reference Interval:

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|-----------------------|--------------|
| IgG antibody negative | < 250 mIE/mL |
| IgM antibody negative | < 1.0 COI |

Melanin, Urine

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Background: Melanocytes metabolize tyrosine by tyrosinase to dihydroxyphenylalanine (DOPA) further to dopaquinone and by oxidation to melanin. The first step catalyzed by tyrosinase takes place in melanosomes and under the control of melanin stimulating hormone. Melanosomes are transferred to skin or mucosa cells.

Urinary increase of melanin metabolites such as indole, catechols and catecholamines occur in malignant melanoma in the stage of metastasis.

Sampling: 10 mL of random urine; transport to laboratory soon.

Reference Interval: Not detectable

Melatonin, Serum

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Background: Melatonin is produced by the pineal gland and is derived from serotonin. It is involved in regulating sleep cycles and is released with darkness and suppressed by daylight. Melatonin is used to soften jet lag symptoms. Melatonin has been shown to be effective in sleep disorders, improving onset of sleep, duration and quality of sleep and increasing REM phases. Particularly patients older than 65 years with low melatonin levels show improvement of the sleep onset time but not an improved sleep or total sleeping time. Melatonin may be associated with midcycle suppression of luteinizing hormone, resulting in inhibition of ovulation. It also may suppress prolactin release.

Sampling: 1 mL serum

Reference Interval: Daylight values < 30 pg/mL
Night values at approx. 9 pm to 4 am < 150 pg/mL

Metanephrines, Urine

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Related Information: Catecholamines, Fractionation, Plasma
Catecholamines, Fractionation, Urine
Homovanillic Acid (HVA), Urine
Vanillylmandelic Acid, Urine

Test includes: Normetanephrine and Metanephrine

Background: The catecholamines epinephrine and norepinephrine are secreted by the adrenal medulla and small amounts are secreted unchanged in the urine, most are metabolized by monamine oxidase and catechol-o-methyltransferase (COMT) producing the major urinary metabolites vanillylmandelic acid (VMA), metanephrine, and normetanephrine. Homovanillic acid, the final product of dopamine metabolism, is excreted in the urine too.

Useful to assay in the diagnosis of catecholamine secreting neoplasms from the adrenal medulla such as pheochromocytomas, paragangliomas and neuroblastomas.

Limitations: Drugs or substances which increase values: Benzodiazepines, diuretics, exogenic catecholamines (nose drops, appetite suppressants), amphetamines, methyl dopa, cigarette smoking, caffeine ingestion, alcohol, nitrates, phenothiazine, and tricyclic antidepressants.

Drugs decreasing values: Bromocriptine, clonidine, dexamethasone, monoamine oxidase inhibitors.

Sampling: A 10 mL aliquot of a 24 h urine, collected in a pre-filled container with 10 mL of 20% hydrochloric acid (not boric acid), note total quantity. Keep cool.

Reference Interval: Total: < 850 µg/24 h
Normetanephrine: < 450 µg/24 h
Metanephrine: < 400 µg/24 h
(Conversion into SI: µmol/ 24h = µg/24h x 5.46)

M-N

Methadone, Urine

f

Related information: Antidiuretic Hormone, Plasma
Opiates, Quantitative, Urine

Synonyms: Dolophine®; Eptadone®; Metasedin®; Methadose®;
Physeptone®; Symoron®

Background: A modified diphenylheptane analgesic, similar properties as morphine, but orally more effective and with a longer half life time. Methadone is metabolized by the liver and excreted in bile and urine.

As a morphine substitute, it does not cause euphoria or serve somnolence. It blocks the action of other co-administered opiates.

Bioavailability 80% - 100%; urinary excretion 14% - 34%; plasma binding 86% - 92% with a blood to plasma concentration ratio of 0.75; volume of distribution 2.4 - 4.8 L/kg; serum half life 15 - 39 h correlates to urine pH and decreased in burn patients and children; serum peak time 3 h, serum peak concentration IV: 450 - 550 ng/mL after a 10 mg dose or oral: 70 - 980 ng/mL after 0.12 - 1.9 mg/kg for 2 month.

Onset of action 0.5 - 1 h after oral dose.

Adverse effects: sedation, CNS and respiratory depression, nausea, vomiting, bradycardia, hypotension, miosis, antidiuretic hormone release. Overdose treatment with Naloxone.

Sampling: 10 mL of random urine, refrigerate if not transported to the laboratory within 8h.

Reference Interval: Negative for urine < 250 ng/mL
Serum levels >100 ng/mL prevent withdrawal symptoms, pain relief in cancer patients requires serum levels of 170 - 530 ng/mL.
Confirmation by GC-MS method.

Methemoglobin (MetHb), Whole Blood

f

Related Information: Carboxyhemoglobin, Blood

Background: A form of hemoglobin with oxidized Fe⁺⁺⁺ state, unable to bind oxygen.

Slight cyanosis occur if values exceed 15% to 30% of total hemoglobin, symptoms such as fatigue, dizziness, dyspnea, headache, tachycardia and severe cyanosis above 40% - 50%.

Lethal > 70%.

Most common causes are drug or chemically induced, particularly by aniline, nitrites, nitroglycerin, nitrate, flutamide, metoclopramide, phenazopyridine, dapsone, phenacetin, acetophenetidin, prilocaine, sulfonamides, sulfones, chlorates, primaquine, quinones, local anesthetics such as benzocaine, lidocaine, and procaine.

A rare form of hereditary metHb is a deficiency of cytochrome b5 reductase (or red cell NADH-methemoglobin reductase), as a recessive autosomal trait. Met Hb levels in homozygotes are 15% - 20%, in heterozygotes MetHb is not increased.

Newborns may react more sensitive to metHb inducing drugs or nitrate in drinking water, since cytochrome b5 reductase is less active.

Sampling: 5 mL of EDTA whole blood. After sample drawing, keep on ice. Process soon. 10% decrease within 4 h, 16% within 8 h if kept on ice.

Reference Interval: < 1% of total hemoglobin
Clinical symptoms > 15%, lethal > 70%

Methylmalonic Acid, Serum or Plasma or Urine

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Related Information: Vitamin B 12, Plasma or Serum
Folic Acid, Serum
Homocysteine, Plasma

Background: Cobalamin is required for isomerization of methylmalonic acid to succinic acid. An increase in methylmalonic acid in urine or serum indicates a cobalamin deficiency, and may be used for vitamin B12 therapy monitoring. Folate deficiency does not influence Methylmalonic acid values. (Serum homocysteine concentrations are increased in folate and Cobalamin deficiency).

Methylmalonic acid is a diagnostic marker in rare inherent disorders presenting with metabolic ketoacidosis, methylmalonic acidemia and aciduria.

Limitation: Renal failure and decreased plasma volume may increase methylmalonic acid and folate levels.

Patients with impaired gut flora due to antibiotic treatment may have decreased Cobalamin levels and subsequently elevated methylmalonic acid values.

Sampling: 2 mL serum; 10 mL urine

Reference Interval: Serum: 9 - 32 µg/L
 Urine: < 10 mg/g of creatinine
 (Conversion into SI: µmol/L = µg/L x 0.0085)

Metrapone Stimulation Test, Serum

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Related Information: Adrenocorticotrophic Hormone, (ACTH), Plasma
 Cortisol, Serum or Plasma
 Cortisol free, Urine
 Applies to: 11-Desoxycortisol, Serum

Background: The metrapone stimulation test (MST) is used to assess the adrenal function in adrenal insufficiency (AI) or Cushing syndrome (CS). Metrapone inhibits the conversion of 11-deoxycortisol to cortisol and thereby produces decrease in serum cortisol which increases the pituitary release and serum levels of ACTH. In the overnight MST metrapone is administered at midnight and cortisol, 11-deoxycortisol and ACTH is measured at 8 AM the following morning. The MST is useful in patients suspected for secondary AI (pituitary or hypothalamic), which display decreased 11-deoxycortisol and ACTH levels in the MST. In Cushing syndrome, the MST is used to distinguish pituitary caused CS from adrenal based CS. In CS patients meeting or exceeding the criteria, the CS is of pituitary origin; patients not meeting the criteria, diagnosis are either an adrenal tumor or ectopic corticotrophin syndrome. Adrenal tumor usually leads to low ACTH levels; ECS patients have normal or elevated ACTH levels.

Dosage of metrapone (overnight protocol): Patients < 70 kg: 2 g orally
 70 - 90 kg: 2.5 g orally
 > 90 kg: 3 g orally

Drugs inducing metabolism of metrapone such as phenytoin, rifampin, phenobarbital, mitotane, corticosteroids should be discontinued before the test.

Sampling: 2 mL heparin plasma

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| Reference Interval: | 11-desoxycortisol, unstimulated, basal |
| | Premature newborn < 1.4 µg/dL |
| | Children 0–12 years 0.02 - 0.25 µg/dL |
| | Adult 0.05 - 0.3 µg/dL |

Overnight MST protocol: Morning (8 am) levels should be < 3 µg/dL. If levels are higher, results can not be interpreted and may be due to failure to take the metyrapone or a rapid clearance due to enzyme induced by other drugs. Approx 5% of normal population exceed 3 µg/dL.

If the cortisol level is < 3 µg/dL the thresholds are:

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|-----------------------------|------------|
| for 11-deoxycortisol serum: | > 7 µg/dL |
| for ACTH serum: | > 75 pg/mL |

Microglobulin β-2- , Serum or Urine

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Related Information: HIV-1/HIV2 Serology
Vancomycin, Serum

Background: Beta-2-Microglobulin is a low molecular weight, membrane derived light chain component of class I human leucocyte antigen (HLA). It is filtered in the glomerulus and reabsorbed in the tubules.

A broad range of diseases lead to elevated levels such as renal failure, lymphomas, neoplasms, inflammatory state (Crohn's disease, hepatitis, sarcoidosis, vasculitis), amyloidosis, immunodeficiency states, hyperthyroidism, viral infections and multiple myeloma.

It correlates with size, growth rate and renal function in multiple myeloma and prognosis.

Used as a predictive marker in therapy of patients with low grade lymphomas. Early marker in aminoglycoside renal toxicity. Progression marker in HIV patients.

Sampling: 1 mL of serum. A 5 mL aliquot of a 24 urine; urine pH should not decrease below < 5.5 since stability of the compound is lost. Note total quantity.

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|----------------------------|--------|-----------|---------------|---------------|
| Reference Interval: | Serum: | Averages: | Neonates | 0.30 mg/dL |
| | | | 0 - 59 years | 0.19 mg/dL |
| | | | 60 - 69 years | 0.21 mg/dL |
| | | | > 69 years | 0.24 mg/dL |
| | Urine: | | | < 120 µg/24 h |

Molybdenum (Mo), Serum or Urine

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Related Information: Uric Acid, Serum

Background: Mo is essential for at last 3 enzymes: xanthine oxidase (purine metabolism), aldehyde oxidase (ethanol metabolism) and sulfite oxidase (amino acid metabolism). A body-total of 8 - 10 mg of Mo is distributed in the skeletal muscle (60%), the liver (20%) and other organs. The recommended daily intake is around 75 - 250 µg, in children 2 µg/kg body weight. Rich in Mo are dairy products, liver, coconuts, and vegetables. Deficiency may develop after

bowel resection. Mo is bound to alpha₂ globulin during circulation.

Dietary sulfides interact with copper and Mo to form insoluble copperthiomolybdenates in the gut, influencing the resorption.

Diseases associated with Mo:

Dysfunction of xanthine oxidase causes xanthinuria, a hereditary disease.

During the initial phase of acute virus hepatitis, circulating Mo is increased. Increased Mo values occur in cirrhosis, alcohol abuses, drug induced liver toxicity, liver metastasis, occlusion of the bile tract, which is due to release of Mo from hepatocytes or impaired uptake into the liver.

Failure to synthesize molybdopterin, a recessive inherent error of metabolism, is characterized by xanthine oxidase deficiency (serum uric acid < 1 mg/dL with increased urine hypoxanthine and xanthine) in combination with sulfite oxidase deficiency (increased urine sulfite, absent inorganic urinary sulfate, increased urinary S-sulfo cysteine) and presents clinically with neurologic abnormalities, (seizures, opisthotonos, and impaired myelin synthesis resulting in early death). Histopathology shows the absence of Mo in the liver, indicating molybdopterin as an essential tissue storage factor.

Patients with chronic renal failure may accumulate Mo up to toxic levels.

Sampling: 2 mL serum. 10 mL random urine

Reference interval: Serum: 0.5 - 3 ng/mL
70% of US population have < 5 ng/mL
toxicity may start at 170 ng/mL
Urine: 10 - 16 µg/L

M-N

Mononucleosis, infectious see Epstein Barr virus

Morphine see Opiates, Quantitative, Urine

Mumps Virus, Serology

f

Background: Mumps virus belong to the paramyxoviruses typically with a RNA genome and two types of envelope spikes, one with hemagglutinin and neuroamidase activities and the other with cell-fusing and hemolytic activities. There is only one serotype. Neutralizing antibodies are directed against the hemagglutinin. The internal nucleocapsid protein S antigen is used in the complement fixation test.

Mumps virus is limited to humans as host. It is transmitted by respiratory droplets. It occurs worldwide with a peak in winter. 30% are inapparent. Virus is excreted approx. between 10 days before and 1 week after onset. Following the recommendations of vaccinating at the age of 12 - 15 month (measles, mumps, and rubella) and a second vaccination at 4 - 6 years or 11 - 12 years, the incidence of mumps has fallen to 308 cases in 1999 in the US. Immunity lasts

life long. During the first 6 month maternal antibodies are protective.

Clinic: after an incubation of 18 - 21 days a prodromal stage with fever malaise anorexia is followed by the tender swelling of the parotid glands unilateral or bilateral, resolving after 1 week. The disease may be complicated by orchitis in postpubertal males, and meningitis, which is usually benign.

Sampling: 1 mL serum, draw acute and convalescent sera 10 - 20 days apart.

Reference Interval: Differentiation of immunoglobulin classes

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|--------------|-----------|------------|
| IgG antibody | negative: | < 20 RE/mL |
| IgM antibody | negative: | < 1.0 COI |

Mycobacteria

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Background: The major pathogens are *Mycobacterium tuberculosis* and to a minor extend *M. bovis*, the cause of tuberculosis and *M. leprae*, the cause of leprosy. Atypical mycobacteria (*M. avium-intracellulare* complex and *M. kansasii*) cause tuberculosis-like disease. Members of the rapid growing *Mycobacterium fortuitum-chelonae* complex cause disease in immunocompromised patients or are associated with implants.

Transmission occurs in *M. tuberculosis* by respiratory droplets. In *M. bovis* by non-pasteurized milk from infected animals, mainly cows. In *M. leprae* by long term close contact to patients. In atypical mycobacteria (*M. kansasii*, *M. marinum*, *M. avium-intracellulare* complex, *M. fortuitum-chelonae* complex) by contaminated soil and water. Patients with smear negative sputum sample may transmit *M. tuberculosis* in up to 20%. On multidrug therapy, which lasts for 6 - 12 month, patient's sputum becomes non-infectious after 3 - 4 weeks.

Sampling: 3 - 5 mL sputum
50 mL of morning urine
20 mL of gastric juice

Reference Interval: Culture: Report on diagnostic finding
For *M. tuberculosis*: direct detection by PCR assay for DNA:
negative: *M. tuberculosis* DNA not detectable

Mycoplasma hominis

f

Background: The small organism lacking a cell wall has been implicated in an infrequent cause of pelvic inflammatory disease. *Ureaplasma urealyticum* however causes approx. 20% of non-gonococcal urethritis.

Sampling: urethral, vaginal or cervical swab; urine.

Reference Interval: Culture result: not detectable

Mycoplasma pneumoniae, Serology

f

Background: The fastidious free-living organism is known to account for up to 20% of hospitalized adults with community acquired pneumonia.

Mycoplasma pneumoniae causes primary atypical pneumonia. Infections are transmitted by respiratory droplets, occurs worldwide with a peak in winter. Outbreaks are reported in young adults and in groups with close contact. Approx. 10% of infected develop pneumonia. Immunity is incomplete, further episodes may occur.

Classification: *Mycoplasma* organisms belong to the genus *Mycoplasma* (class Mollicutes) and are described as the simplest and smallest self-replicating bacteria because of their total lack of cell wall, the paucity of their metabolic pathways, and the small size of their genome. In the 1980s, they were shown to have evolved from more classical bacteria of the firmicutes taxon by a so-called regressive evolution that resulted in massive genome reduction but are considered successful pathogens of man and animal.

Sampling: 1 mL serum, convalescent serum 2 - 4 weeks apart recommended for definitive diagnosis.

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| Reference Interval: | Differentiation of immunoglobulin class |
| | IgA antibody negative: < 9 RE/mL |
| | borderline: 9 - 11 RE/mL |
| | positive: > 11 RE/mL |
| | IgG antibody negative: < 9 RE/mL |
| | borderline: 9 - 11 RE/mL |
| | positive: > 11 RE/mL |
| | IgM antibody negative: < 9 RE/mL |
| | borderline: 9 - 11 RE/mL |
| | positive: > 11 RE/mL |

M-N

Myeloperoxidase Antibody (MPO) see Antineutrophil Cytoplasmatic Antibody (ANCA)

Myoglobin, Blood or Serum or Plasma

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|-----------------------------|-------------------------------------|
| Related Information: | Carboxyhemoglobin, Blood |
| | Creatine Kinase (CK, NAC-activated) |
| | Creatinine Kinase Isoenzymes, Serum |
| | Haptoglobin (Hp), Serum |
| | Lactate Dehydrogenase (LDH), Serum |
| | Myoglobin, Qualitative, Urine |
| | Troponin T, Serum |

Background: Myoglobin is a small 17.8 kDa oxygen binding protein of the cytoplasm of the skeletal and heart muscle cells, released through traumatization with a plasma half life of

10 - 20 min (CK: 15 h, CKMB: 12 h). Myoglobin is not specific for a distinct tissue. Most of urine myoglobin is derived from skeletal muscle.

Myoglobin is an early and highly sensitive marker to confirm acute myocardial infarction.

Indicated: Acute myocardial infarction (AMI): serum myoglobin increases 2 - 4 h after onset of pain, whereas CK starts to increase after 4 - 6 h. Predictive marker for AMI: to rule out: negative predictive value 98%; to confirm: positive predictive value 64%. Myoglobin peaks within 4 - 12 h and decreases into reference interval with in 24 - 36 h.

Monitoring thrombolytic therapy of AMI: An initially rapid myoglobin increases > 150 µg/L/h, and a decrease into reference interval within 10 - 20 h is correlated with successful thrombolysis.

Monitoring diseases of the skeletal muscle

Sampling: 2 mL serum, plasma, blood;

Urine: 5 mL

Reference Interval: Serum < 64 ng/mL
Urine < 30 ng/mL

Myoglobin, Qualitative, Urine

f

Related Information: Carboxyhemoglobin, Blood
Creatine Kinase (CK, NAC-activated)
Creatinine Kinase Isoenzymes, Serum
Haptoglobin (Hp), Serum
Lactate Dehydrogenase (LDH), Serum
Myoglobin, Blood or Serum or Plasma
Troponin T, Serum

Background: Please see also Myoglobin Blood, Serum or Plasma. Most of the myoglobin detected in urine is of skeletal muscle origin. The assay is used in the investigation of myositis, rhabdomyolysis and muscle traumatization. Elevated accompanying parameters are serum creatine kinase, serum myoglobin.

Myoglobinuria may occur after viral diseases caused by Influenza-, Herpes simplex-, Epstein-Barr, -Enteroviruses; during bacterial diseases; in primary muscle diseases such as muscle dystrophy, polymyositis, dermatomyositis, particularly under steroid therapy; by drug toxicity (alcohol, carbon monoxide, amphetamine abuse, by animal poisons; during ischemia (compression, thromboembolism, myocardial infarction) and by trauma of the muscles (caused by epilepsy, injuries, high voltage shock).

Sampling: 5 mL random urine

Reference Interval: < 17 µg/g of creatinine

Myositis Antibody see Jo-1 Antibody