SULTAN MEDICA

# SULTAN LAB BOOK

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(2015/2/454)

# SULTAN LAB BOOK

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*Welcome to Sultan's Lab Book test directory. We provide a list of tests performed in our specialized laboratories with valuable information for each test.*

*The tests and examinations are listed in alphabetical order.*

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Our aim at the **Sultan Medica™ Group** franchise is to be a name synonymous with trust, Quality and precision in the Medical Technology world.

To enable us to reach and maintain this goal we strive always to provide our laboratories with very latest technology available, use only the best and most credited materials, implement target quality control results on a daily basis and train all staff to the highest level.

In the near future we hope to see our franchise in every major city in the Arab world and with this in mind, we make this promise we will continue our programme of excellence in both technology and patient care and also push the boundaries of possibilities, always with the thought in mind that we try to make a difference whilst remembering that a happy, healthy patient is the ultimate goal.

***About Sultan Medica™ Group***

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- Branches & Agents
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  - Internationally: Oman • Syria • Kuwait • Libya • Iraq • SA

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## *Accreditations and Certifications*

**• ISO 15189 Laboratory Accreditation (Final stage)**

Medical laboratories - Particular requirements for quality and competence specifies the quality management system requirements particular to medical laboratories (Specific requirements to ensure Quality, Competence and Reliable Results).

**• ISO Certification 9001:2000**

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- Labs consultancy services and labs management services for the other laboratories in all departments.

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- Sultan Medica™ Group is recognized by the Ministry of Health for training medical Technologists to obtain the license of the Laboratory Director.
- Sultan Medica™ Group is recognized by the Ministry of Health for performing special tests.
- Sultan Medica™ Group labs were the first to be member (Pioneer members) in Laboratory - Based Surveillance Programme.

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  - MOH National Q.C. Programme
  - Qualiris by Stago
  - DAR Accreditation

## Table of contents

# Test Directory

# 5,10-Methylenetetrahydrofolate Reductase (MTHFR)

## Summary

MTHR is the Homocystein Gene

MTHFR C677T: homozygosity predisposes to arterial and venous thrombosis in the presence of additional risk factors; the thermolabile variant (T allele) is associated with reduced enzyme activity and elevated plasma homocysteine levels in conjunction with folate deficiency.

MTHFR A1298C: compound heterozygosity for C677T and A1298C is considered as cardiovascular disease risk factor; the C allele is also associated with reduced MTHFR enzyme activity.

| | |
|---|---|
| Method | PCR |
| TAT | 2d |
| Sample type | EWB/CWB |
| Expected value | Normal Genotype |
| Sample stability | 3 d at room temperature |
| | 1 wk at 2-8˚C |

# 17-Hydroxyprogesterone (17-OH Prog.)

## Summary

17-Hydroxyprogesterone is a natural progestin that increases in pregnancy during the third trimester, primarily due to fetal adrenal production.

Measuring the levels of 17-hydroxyprogesterone is useful in the evaluation of patients with suspected congenital adrenal hyperplasia. In the rare case of a patient with 17α-hydroxylase deficiency, very low or undetectable levels of 17-OHP will be recorded. Elevated serum 17-OHP levels at baseline and/or after ACTH stimulation have also been reported in other forms of adrenal hyperplasia.

| | |
|---|---|
| Method | ELISA |
| TAT | Dy/A |
| Sample type | S/EP/HP |
| Expected value | Children: 0.2-0.9 ng/mL |
| | Male (♂): 0.2-2.3 ng/mL |
| | Follicular: 0.2-1.5 ng/mL |
| | Leuteal: 1-4.5 ng/mL |
| | Postmenopause: 0.2-0.9 ng/mL |
| Conversion factors | ng/mL x 3.03 = nmol/L |
| | mmol/L x .0330 = ng/mL |
| Sample stability | 1 wk at 2-8˚C |
| | 6 mth at -20˚C |

# 17-Ketosteroids

## Summary

Urinary 17-ketosteroid excretion is used as a measure of adrenal cortical activity and male gonadal activity. It is therefore used to diagnose abnormal adrenal cortical activity in such conditions as hyperadrenocorticism (Cushing's disease), adrenal cortical tumors, hypoadrenocorticism (Addison's disease), male hypergonadism (Leydig cell tumors) and hypogonadism and panhypopituitarism. The determination of 17-ketosteroids in the urine has considerable clinical importance. Hypogonadism in males is reflected by low (female) values of 17-ketosteroid excretion, as seen in gynecomastia, cretinism and mongolism. Abnormalities in 17-ketosteroid excretion in certain psychoses, indicating a faulty adrenal cortical response to stress, have been observed, and low values in diabetes have been reported.

| | | |
|---|---|---|
| Method | Colorimetric | |
| TAT | 7d | |
| Sample type | 24U | |
| Expected value | Male (♂): 10-25 mg/d | |
| | Female (♀): 6-14 mg/d | |
| Sample stability | 4 h | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 mth | at -20˚C |

# 5-Hydroxyindoleacetic Acid (5-HIAA)

## Summary

5-HIAA is used to determine serotonin levels in the body. Since 5-HIAA is a metabolite of serotonin, testing is most frequently performed for the diagnosis of carcinoid tumors of the enterochromaffin (Kultschitzsky) cells of the small intestine, which release large amounts of serotonin.

Low levels of 5-HIAA in the cerebrospinal fluid have been associated with aggressive behavior and suicide by violent means, correlating with diminished serotonin levels.

Elevated serotonin (hyperserotonemia) is one of the most common biological findings in autism and 5-HIAA may be elevated in patients with autistic spectrum disorders.

| | | |
|---|---|---|
| Method | RIA | |
| TAT | 3 d | |
| Sample type | 24U | |
| Expected value | 6-10 mg/d | |
| Sample stability | 2 h | at room temperature |
| | 2 d | at 2-8˚C |
| | 2 d | at - 20˚C |

# 5-Nucleotidase

## Summary

The determination of 5'-nucleotidase enzyme is used to evaluate and manage suspected diseases of the liver, and also can be useful in differentiating the cause of Alkaline Phosphatase elevation. Elevated levels are found in hepatobiliary disease, malignant infiltration of the liver and billiary cirrhosis.

| | |
|---|---|
| Method | Enzymatic |
| TAT | Dy/A |
| Sample type | S |
| Expected value | <9.0 U/L |
| Sample stability | 4 h at room temperature |
| | 1 wk at 2-8˚C |
| | 2 wk at -20˚C |

# Acetaminophen (Paracetamol)

## Summary

Acetaminophen is a common drug used in many formulations due to its analgesic and antipyretic properties. Chronic excessive use of acetaminophen can result in hepatotoxicity and nephrotoxicity. Overdosage can lead to severe hepatic damage and hepatic failure if untreated.

| | |
|---|---|
| Remark | Neither hemolyzed nor lipemic sample. |
| Method | ECLIA |
| TAT | Dy/A |
| Sample type | S/HP |
| Expected value | 10-30 µg/mL |
| | Toxic level: >100 µg/ml |
| Conversion factors | µg/mL x 6.62 = µmol/L |
| Sample stability | 2 d at 2-8 °C |
| | 1 mth at -20 °C |

# Acetone

## Summary

Acetone is believed to exhibit only slight toxicity in normal use, and there is no strong evidence of chronic health effects if basic precautions are followed. At very high vapor concentrations, acetone is irritating and may depress the central nervous system. It is also a severe irritant on contact with eyes, and a potential pulmonary aspiration risk. In occupational health medicine, acetone in urine is an indicator of exposure to Isopropyl Alcohol (IPA).

| | |
|---|---|
| Method | Dry chemical |
| TAT | Dy/AB |
| Sample type | S/U |
| Expected value | Negative |
| Sample stability | 5d at room temperature |
| | 1 wk at 2-8˚C |
| | 1 mth at -20˚C |
| Sample stapility in U | undetermined |

# Acetylcholine Receptor Autoantibody (ACHA)

## Summary

ACHA are produced against the nicotinic acetylcholine receptor in myasthenia gravis. They cause postsynaptic interferences and reduce the amount of receptors. The levels of the antibody concentration correlate with the severness of the clinical picture.

| | | |
|---|---|---|
| Method | IF | |
| TAT | 2 d | |
| Sample type | S | |
| Expected value | Negative | |
| Sample stability | 2 h | at room temperature |
| | 2 d | at 2-8˚C |
| | 6 mth | at - 20˚C |

# Acid Fast Bacilli (AFB)

## Summary

AFB cultures are used to diagnose active M. tuberculosis infections, infections due to another member of the Mycobacterium family, or to determine whether TB-like symptoms are due to another cause. They are used to help determine whether the TB is confined to the lungs (pulmonary disease) or has spread to organs outside the lungs (extrapulmonary disease). AFB cultures can also be used to monitor the effectiveness of treatment and can help determine when a patient is no longer infectious.

| | | |
|---|---|---|
| Method | Ziehl–Neelsen stain | |
| TAT | Dy/A | |
| Sample type | D | |
| Expected value | Negative | |
| Sample stability | 1 d | at room temperature |
| | 1 wk | at 2-8˚C |
| | 1 wk | at -20˚C |

# Acid Phosphatase (ACP) ,Total

## Summary

Total acid phosphatase and prostatic acid phosphatase levels increase in the presence of progressive and metastasizing prostate carcinoma, the increase being dependent upon the disease stage in 80 % of patients with metastasizing prostate cancer. The percentage increases at each stage depends on the classification (pathological or clinical).

Increased acid phosphatase levels occur in Gaucher's disease, Niemann-Pick disease, 1-2 days after prostate surgery, biopsy, manipulation or catheterization, and in the presence of benign prostate hypertrophy, prostatitis and prostate infarction.

| | | |
|---|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. | |
| Method | Colorimetric | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Male (♂): | Up To 6.6 U/L |
| | Female (♀): | Up To 6.5 U/L |
| Conversion Factors | U/L x 0.0167 = µkat/L | |
| Sample stability | 8 d | at room temperature |
| | 8 d | at 2-8 °C |
| | 4 mth | at -20 °C |

# Acid Phosphatase, Prostatic

## Summary

Prostatic acid phosphates levels are greatly increased in cases of prostatic cancer, total acid Phosphatase activity can be elevated in Paget's disease, in hyperparthyroisdism with skeletal involvement, in the presence of malignant invasion of the bones by cancer, in Gaucher's, Niemann-Pick disease, and in leukemia.

| | | |
|---|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. | |
| Method | Enzymatic | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Up To 3.5 U/L | |
| Sample stability | Several hr | at room temperature |
| | 1 wk | at 2-8 °C |

# Adenovirus Abs (IgA,IgG,IgM)

## Summary

Most infections with adenovirus result in infections of the upper respiratory tract. Adenovirus infections often show up as conjunctivitis, tonsillitis, ear infection, or croup. Adenoviruses can also cause gastroenteritis, viral meningitis or encephalitis. Rarely, adenovirus can cause cystitis.

The presence of IgG abs to adenoviruses indicates past infection. Raising IgG titres suggest acute or current infection.

The presence of IgM/IgA abs to adenoviruses indicates current or recent infection with adenoviruses.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Negative | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8 °C |
| | 1 yr | at -20 °C |

# Adenovirus Ag

## Summary

Most infections with adenovirus result in infections of the upper respiratory tract. Adenovirus infections often show up as conjunctivitis, tonsillitis, ear infection, or croup. Adenoviruses can also cause gastroenteritis, viral meningitis or encephalitis. Rarely, adenovirus can cause cystitis.

Adenovirus Ag test is important in the diagnosis of adenovirus infection and to monitor the effectiveness of therapeutic treatment.

| | |
|---|---|
| Method | ICT |
| TAT | Dy/AB |
| Sample type | ST |
| Expected value | Negative |
| Sample stability | 2 h at room temperature |
| | 3 d at 2-8 °C |
| | 1 wk at -20 °C |

# Adrenocorticotropic Hormone, Corticotrophin (ACTH)

## Summary

ACTH concentrations show a diurnal variation with high levels in the morning and low levels in the evening. Plasma ACTH measurements are useful in the differential diagnosis of pituitary Cushing's disease (ACTH hypersecretion), autonomous ACTH producing pituitary tumors (e.g. Nelson's syndrome), hypopituitarism with ACTH deficiency and ectopic ACTH syndrome. In addition to cortisol measurements, ACTH determinations can be used together with functional or stimulation tests to diagnose the origin of glucocorticoid overproduction. Similarly, ACTH measurements can be employed to facilitate differential diagnosis of adrenocortical insufficiency (Addison's disease).

| | | |
|---|---|---|
| Remark | Keep sample cool. | |
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | EP | |
| Expected value | 5-60 pg/mL | |
| Conversion factors | pg/mL x 0.2202 = pmol/L | |
| | pmol/L x 4.541 = pg/mL | |
| Sample stability | 2 h | at room temperature |
| | 1mth | at -20 °C |

# Alanine Aminotransferase (ALT)

## Summary

The major source of ALT is the liver, which has led to the measurement of ALT activity for the diagnosis of hepatic diseases. Elevated serum ALT is found in hepatitis, cirrhosis, obstructive jaundice, carcinoma of the liver, and chronic alcohol abuse. ALT is only slightly elevated in patients who have an uncomplicated myocardial infarction. In patients with vitamin B6 deficiency, serum aminotransferase activity may be decreased.

| | | |
|---|---|---|
| Method | Enzymatic | |
| TAT | Dy/AB | |
| Sample type | S/EP/HP | |
| Expected value | Male (♂): | up to 41 U/L |
| | Female (♀): | up to 33 U/L |
| Conversion factors | U/L × 0.0167 = µkat/L | |
| Sample stability | 3 d | at room temperature |
| | 1 wk | at 2-8 °C |
| | > 1 wk | at -70 °C |

# Albumin (ALB)

## Summary

Hyperalbuminemia occurs in dehydration. Hypoalbuminemia occurs during many illnesses and is caused by several factors such as compromised synthesis due either to liver disease or as a consequence of reduced protein uptake, elevated catabolism due to tissue damage (severe burns) or inflammation, malabsorption of amino acids (Crohn's disease), proteinuria as a consequence of nephrotic syndrome and protein loss via the stool (neoplastic disease). The determination of albumin levels allows monitoring of controlled patient dietary supplementation and also serves as an excellent test of liver function.

| | | |
|---|---|---|
| Method | Colorimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Age | Value (g/dL) |
| | 0 d - 4 d | 2.8-4.4 |
| | 4 d -14 yr | 3.8-5.4 |
| | 14yr -18 yr | 3.2-4.5 |
| | 18 yr - 60 yr | 3.5-5.0 |
| | >60 yr | 3.4-4.8 |
| Conversion factors | g/dL x 10 = g/L | |
| | g/L x 0.1 = g/dL | |
| Sample stability | 2.5 mth | at room temperature |
| | 5 mth | at 2-8˚C |
| | 5 mth | at - 20˚C |

# Aldolase

## Summary

The determination of aldolase enzyme high level is useful to the diagnosis of progressive muscle dystrophy (Duchenne) and muscle disease with myoglobinuria.

| | | |
|---|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. | |
| Method | Enzymatic | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Up To: 7.6 U/L | |
| Sample stability | 8 h | at room temperature |
| | 5 d | at 2-8˚C |
| | 6 mth | at - 20˚C |

# Alkaline Phosphatase (ALP),Total

## Summary

A rise in the alkaline phosphatase occurs with all forms of cholestasis, particularly with obstructive jaundice. It is also elevated in diseases of the skeletal system such as behget's disease, hyperparathyroidism, rickets and osteomalacia, fractures and malignant tumors.

A considerable rise in the alkaline phosphatase activity is sometimes seen in children and juveniles. It caused by increased osteoblast activity following accelerated bone growth.

| | | | |
|---|---|---|---|
| Method | Colorimetric | | |
| TAT | Dy/AB | | |
| Sample type | S/HP | | |
| Expected value | Age | Female U/L | Male U/L |
| | 1 d | <250 | <250 |
| | 2 d – 5 d | <231 | <231 |
| | 6 d – 6 mth | <449 | <449 |
| | 7 mth – 1 yr | <462 | <462 |
| | 1 yr – 3 yr | <281 | <281 |
| | 4 yr – 6 yr | <269 | <269 |
| | 7 yr – 12 yr | <300 | <300 |
| | 13 yr - 17 yr | <187 | <390 |
| | + 17 yr | 35-104 | 40-129 |
| Conversion factors | U/L x 0.0167 = µkat/L | | |
| Sample stability | 1 wk | at room temperature | |
| | 1 wk | at 2-8˚C | |
| | 2mth | at - 20˚C | |

# Alkaline Phosphatase (ALP) Isoenzyemes

## Summary

ALP isoenzyemes is orderd when the Alkaline Phosphatase (ALP) test result is high. This test will help determine what part of the body is causing higher ALP levels.

This test may be used to diagnose bone disease, cause of pain in the abdomen, liver, gallbladder, or bile duct disease, Parathyroid gland disease, Vitamin D deficiency. It may also be done to check liver function and therapy monitoring.

| | | |
|---|---|---|
| Remarks | Fasting, Freezed. | |
| Method | Enzymatic | |
| TAT | 3 d | |
| Sample type | S | |
| Expected value | Total | <290 IU/L |
| | Liver Fraction | <160 IU/L |
| | Bone Fraction | <150 IU/L |
| | Intestine Fraction | <5 IU/L |
| | Macro hepatic Fraction | Negative |
| Macro hepatic Fraction | Negative | |
| Sample stability | 1 h | at room temperature |
| | 1 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Alpha-1- Antitrypsin (AAT)

## Summary

α1- Antitrypsin is an important positive phase reactant found in elevated concentrations in inflammatory ailments (e.g. infectious and rheumatoid diseases), tissue necrosis, malignancy and traumas. Inflammation of the liver parenchymal cells is often accompanied by elevated α1- Antitrypsin levels, it is also suspected when severe pulmonary emphysema occurs in adults.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP | |
| Expected value | 0.9-2.0 g/L | |
| Conversion factors | g/L x 18.4=μmol/L | |
| | g/L x 100 = mg/dL | |
| Sample stability | 3mth | at room temperature |
| | 5mth | at 2-8˚C |
| | 3mth | at -20˚C |

# Alpha-1-Antitrypsin Genotype

## Summary

α1-Antitrypsin Deficiency (AATD) is an autosomal hereditary disorder caused by several mutations in the AAT gene. Its major clinical manifestations include pulmonary emphysema in adults and liver disorders in children as well as in adults. The large majority of subjects affected by AATD carry the PIZZ or PISZ genotypes. Another pathologic allele, the M-Malton variant (also known as Mnichinan and Mcagliari), can mimic the Pi Z clinical phenotype, but this α1-antitrypsin deficiency variant is not easily recognizable and seems to be more under recognized than the Z or S alleles.

| | | |
|---|---|---|
| Method | PCR | |
| TAT | 4 d | |
| Sample type | EWB | |
| Expected value | Normal genotype | |
| Sample stability | 3 d | at room temperature |
| | 1 wk | at 2-8˚C |

# Alpha-1- Fetoprotein (AFP)

## Summary

AFP is formed in the yolk sac, non differentiated liver cells and the fetal gastro - intestinal track. 70-95% of patients with primary hepatocellular carcinoma have elevated AFP values. The later stage of non - seminomatous germ cells, the higher the AFP value. Measuring AFP levels contributes to the risk assessment for Trisomy 21 (Down syndrome) in the second trimester of pregnancy.

Furthermore, AFP is considered a tumor marker for primary liver cell carcinoma and tumors of the testes and ovaries.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/EP/CP. | |
| Expected value | Up to 7.0 ng/mL | |
| | Median 14wk: 27.9 ng/mL | |
| | Median 15wk: 30.9 ng/mL | |
| | Median 16wk: 36.1 ng/mL | |
| | Median 17wk: 40.4 ng/mL | |
| | Median 18 wk: 48.3 ng/mL | |
| | Median 19 wk:54.8 ng/mL | |
| Conversion factors | ng/L x 0.083 = IU/mL | |
| | IU/mL x 1.21 = ng/mL | |
| Sample stability | 3 d | at room temperature |
| | 1 wk | at 2-8˚C |
| | 3 mth | at - 20˚C |

# Alpha 2-Macroglobulin

## Summary

Alpha 2 macroglobulin is a major serum protein with diverse functions, including inhibition of protease activity and pending of growth factor, cytokines, and disease factors. Increased values have been suggested to be associated with multiple sclerosis, glomerular disease, and liver diseases. This test is an important factor in calculating Fibrotest (Formula that measures liver Fibrosis).

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | 1.49-1.79 g/L | |
| Sample stability | 8 h | at room temperature |
| | 8 d | at 2-8˚C |
| | 1 yr | at -20˚C (if frozen within 24 h ) |

# Aluminium (Al)

## Summary

Aluminium is not considered an essential trace element. It is found in relatively small concentrations in serum from our daily food intake. Increased lev¬els are found in increased intake or insufficient elimination. Aluminium has a different affinity to different organs; low concentrations can be toxic to the CNS (aluminum encephalopathy), whereas patients on dialysis need higher doses of aluminium hydroxide in order to prevent hyperphosphatemia. The diagnosis of Aluminium levels is used to monitor patients on dialysis with oral aluminium therapy (aluminium hydroxide to neutralize phosphate), workers in aluminium processing industries and patients with Alzheimer's disease.

| | |
|---|---|
| Remarks | Standard monovettes are not recommended as results are falsely increased, often more than 100 µg, collection of whole blood in special tubes (trace element free heparin-monovette) is highly recommended. |
| Method | AAS |
| TAT | 10 – 14 d |
| Sample type | HWB |
| Expected value | < 7.50 µg/L |
| Sample stability | 3 d at room temprature<br>2 wk at 2-8 °C<br>6 mth at -20 °C |

# Amikacin

## Summary

Amikacin is most often used for treating severe, hospital-acquired infections with multidrug resistant Gram negative bacteria such as Pseudomonas aeruginosa, Acinetobacter and Enterobacter. Serratia marcescens and Providencia stuartii are also included in the spectrum. Amikacin can also be used to treat non-tubercular mycobacterial infections and tuberculosis (if caused by sensitive strains) when first line drugs fail to control the infection.

The determination of serum or plasma drug levels is required to achieve optimal therapeutic efficacy and to minimize toxicity.

| | |
|---|---|
| Remarks | Just before dose. |
| Method | FP |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Peak Level: 20-25 µg/mL |
| | Trough Level: 5-10 µg/mL |
| Conversion factors | µg/mLx 1.71 =µmol/L |
| | µmol/L x 0.585 =µg/mL |
| Sample stability | 3 mth at room temperature |
| | 5 mth at 2-8 ˚C |

# Amino Acids (Quantitative)

## Summary

The common indications for amino acid testing include clinical situations such as acute life-threatening episode, failure to thrive, recurrent vomiting, neurological deterioration, hyperammonemia, lethargy, metabolic acidosis, and testing or following therapy for a specific inborn error of metabolism (PKU, MSUD, tyrosinemia, etc.). Listing of clinical information is particularly important for appropriate interpretation.

| | |
|---|---|
| Method | HPLC |
| TAT | 3 d |
| Sample type | HP/24U/US |
| Expected value | Refer To Table |

| Components | 0-11 months | 1 year and older | Components | 0-11 months | 1 year and older |
|---|---|---|---|---|---|
| Alanine | 200-600 µmol/L | 240-600 µmol/L | Isoleucine | 20-130 µmol/L | 30-130 µmol/L |
| Ilo-isoleucine | None Detected | None Detected | Leucine | 40-230 µmol/L | 60-230 µmol/L |
| Arginine | 20-160 µmol/L | 40-160 µmol/L | Lysine | 60-250 µmol/L | 80-250 µmol/L |
| Aspartic Acid | 0-40 µmol/L | 0-20 µmol/L | Methionine | 10-60 µmol/L | 17-53 µmol/L |
| Citrulline | 6-60 µmol/L | 10-60 µmol/L | Ornithine | 20-135 µmol/L | 20-135 µmol/L |
| Cystine | 7-70 µmol/L | 7-70 µmol/L | Phenylalanine | 30-100 µmol/L | 30-80 µmol/L |
| Glutamic Acid | 10-190 µmol/L | 10-120 µmol/L | Proline | 110-500 µmol/L | 110-500 µmol/L |
| Glutamine | 410-960 µmol/L | 410-700 µmol/L | Serine | 90-250 µmol/L | 60-200 µmol/L |
| Glycine | 220-520 µmol/L | 140-490 µmol/L | Taurine | 25-160 µmol/L | 25-80 µmol/L |
| Histidine | 40-120 µmol/L | 50-130 µmol/L | Threonine | 50-300 µmol/L | 60-220 µmol/L |
| Homocystine | None Detected | None Detected | Tyrosine | 30-140 µmol/L | 30-120 µmol/L |
| Hydroxyproline | 6-90 µmol/L | 6-50 µmol/L | Valine | 110-300 µmol/L | 140-350 µmol/L |

| | | |
|---|---|---|
| Sample stability | 2 d | at 2-8 ˚C |
| | 1 mth | at -20˚C |

# Ammonia (NH3L)

## Summary

An excess of ammonia can be toxic to the central nervous system. The Krebs-Henseleit urea cycle provides a means of disposal of ammonia by metabolizing ammonia to urea in the liver. Hyperammonemia in infants can be caused by inherited deficiencies of the urea cycle enzymes or acquired through acute (as in Reye's syndrome) or chronic (as in cirrhosis) liver disease. In adults, elevated ammonia levels can aid in diagnosis of liver failure or hepatic encephalopathy from advanced liver diseases such as viral hepatitis or cirrhosis.

| | |
|---|---|
| Remarks | Vaccutainer, On Ice, Fresh, Neither hemolyzed nor lipemic sample. |
| Method | Enzymatic |
| TAT | Dy/AB |
| Sample type | EWB |
| Expected value | Male (♂): 25-94 µg/dL<br>Female (♀): 19-82 µg/dL |
| Conversion factors | µmol/L x 1.703 = µg/dL |
| Sample stability | 2 h at 2-8˚C<br>3 wk at - 20˚C |

# Amphetamines (AMPS)

## Summary

Amphetamines are powerful psychostimulants, producing increased alertness, wakefulness, insomnia, energy and self-confidence in association with decreased fatigue and appetite as well as enhanced mood, well being and euphoria. Amphetamines and methamphetamines drug toxicity.

| | |
|---|---|
| Method | Kinetic |
| TAT | Dy/A |
| Sample type | US |
| Expected value | Not detected |
| Sample stability | 5 d 2-8˚C |

# Amylase (AMYL)

## Summary

Amylase testing is of considerable importance in the verification and exclusion of acute pancreatitis (acute upper abdominal symptoms), chronic pancreatitis (relapse), obstructive chronic pancreatitis parotitis, suspicion of macoamylasemia and the verification of renal insufficiency.

| | |
|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. |
| Method | Colorimetric |
| TAT | Dy/AB |
| Sample type | S/HP/EP/US |
| Expected value | Blood: 28-100 U/L |
| | Urine: Up To 460 U/L |
| Conversion factors | U/L x 0.0167 = μkat/L |
| Sample stability | 1 wk at room temperature |
| | 1 wk at 2-8˚C |
| | 1 yr at -20˚C |

# Androstenedione

## Summary

The measurement of serum androstenedione provides a useful marker of androgen biosynthesis. Elevated androstenedione levels have been demonstrated in virilizing congenital adrenal hyperplasia. Serum androstenedione levels are also increased in polycystic ovary syndrome, and in the case of hirsutism in women. Elevated serum androstenedione levels may also occur in adrenal and ovarian virilizing tumors.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S/EP/HP/CP | |
| Expected value | Male (♂) | 0.60 - 2.7 ng/mL |
| | Follicular Phase | 0.75 - 3.1 ng/mL |
| | Luteal Phase | 0.94 - 3.2 ng/mL |
| Sample stability | 1 d | at room temperature |
| | 1 wk | at 2-8˚C |
| | 6 mth | at -20˚C |

# Angiotensin Converting Enzyme (ACE)

## Summary

ACE, angiotensin I and angiotensin II are part of the Renin-Angiotensin System (RAS), which controls blood pressure by regulating the volume of fluids in the body. ACE is secreted in the lungs and kidneys by cells in the inner layer of blood vessels. Elevated levels of ACE are found in sarcoidosis and are used to diagnose and monitor the disease.

| | | |
|---|---|---|
| Method | Enzymatic | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | 8-65 U/L | |
| Sample stability | 1 wk | at room temperature |
| | 1 wk | at 2-8˚C |
| | 2 mth | at -20˚C |

# Anti Adrenal Gland Abs

## Summary

The determinations of adrenal antibodies are used to diagnose autoimmune adrenal insufficiency (Addison´s disease) which is the frequent consequence of various immunepathological reactions. The insufficiency of the adrenal glands is thought to be a consequence of a disorder of the physiological immune tolerance, especially with congenital immune defects.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:10 | |
| Sample stability | 2 wk | at 2-8˚C |
| | Long term storage | at -20˚C |

# Anti Annexin V Abs (IgG, IgM)

## Summary

The detection of IgG and IgM class autoantibodies against annexin V is used for the diagnosis of primary or Secondary Antiphospholipid syndrome (APS), which is a systemic autoimmune disease that causes thromboses, recurrent miscarriage and intrauterine fetal death. Clinical symptoms are accompanied by the occurrence of specific autoantibodies that are detectable in the blood of patients with APS. The determination of IgG antibodies is a valuable indicator in the diagnosis of beginning autoimmune diseases, whereas IgM antibodies are present in progressive stages of manifested autoimmune disorders.

| | |
|---|---|
| Method | ELISA |
| TAT | Dy/A |
| Sample type | S/EP/CP |
| Expected value | Negative: <5 IU/mL |
| Sample stability | 5 d at 2-8°C |
| | 6 mth at -20°C |

# Anti Beta 2 Glycoprotein I (β2 GPI IgG,IgM)

## Summary

Anti-Beta-2-Glycoprotein I antibodies are associated with diseases of antiphospholipid syndrome such as thrombosis, thrombocytopenia or fetal loss in the context of systemic lupus erythematosus. Autoantibodies against beta-2-Glycoprotein I are described for various autoimmune diseases. The presence of anti-beta-2GPI antibodies can be related to the development of arterial and venous thromboses, venous thromboembolism, thrombocytopenia and fetal loss.

The determination of IgG antibodies is a valuable indicator in the diagnosis of beginning autoimmune diseases, whereas IgM antibodies will be found in progressive stages of manifested autoimmune disorders. Anti-beta-2GPI IgG antibody titers correlate well with the clinical status of the patients in thrombosis, thromboembolism and repeated fetal loss, while anti-beta-2GPI IgM antibodies show a significant association with thrombosis and thrombocytopenia.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <5.0 U/mL | |
| Sample stability | 5 d | at 2-8 °C |
| | 6 mth | at -20 °C |

# Anti-Cardiolipin (IgG,IgM)

## Summary

The presence of anti-cardiolipin antibodies in Systemic Lupus Erythematosus (SLE) can be related to the development of thrombosis and thrombocytopenia. In gynaecology they are supposed to cause intrauterine death or recurrent abortion. Furthermore, anti-cardiolipin antibodies have been detected in neurological disorders like cerebrovascular insufficiency, cerebral ischemia, epilepsy or chorea. Anti-cardiolipin autoantibodies occur in the immunoglobulin classes IgG, IgM. The determination of IgM antibodies is a valuable indicator in the diagnosis of beginning autoimmune diseases, whereas IgG antibodies are present in progressive stages of manifested autoimmune disorders.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | IgG: Negative: <10 [GPL U/ml] | |
| | IgM: Negative: <7 [MPL U/ml] | |
| Sample stability | 5 d | at 2-8 °C |
| | 6 mth | at -20 °C |

# Anti-Centromere Abs (ACA)

## Summary

Anti-Centromere Antibodies (ACA) occur in auto immune disorders; frequently in limited systemic scleroderma, (formerly called CREST syndrome), and, occasionally are found in the diffuse form of scleroderma. They are rare in other rheumatic conditions and in healthy persons.

Anti-centromere antibodies are found in approximately 60% of patients with limited systemic scleroderma, and in 15% of those with diffuse form of scleroderma. The specificity of this test is >98%. Thus, a positive anticentromere antibody finding is strongly suggestive of limited systemic scleroderma. Anti-centromere antibodies present early in the course of disease, and are notably predictive of limited cutaneous involvement and a decreased likelihood of aggressive internal organ involvement, such as lung fibrosis.

When present in primary biliary cirrhosis, ACA are prognostic of portal hypertension.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:40 | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Anti Cyclic Citrulinated Peptide (Anti CCP)

## Summary

Anti CCP combines the many advantages of the detection of autoantibodies against the native autoantigen Mutated Citrullinated Vimentin (MCV). Anti-CCP detects autoantibodies very early on, sometimes even years before symptoms become evident. Patients without symptoms but with an increased Anti-CCP antibody titre are at high risk for future Rheumatoid Arthritis (RA) development. Furthermore, a positive result is predictive for a severe course of RA. Therefore, Anti-CCP is an effective tool for rapid and precise routine diagnosis and favours immediate implementation of treatment.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | < 20 Units | |
| Sample stability | 5 d | at 2-8 °C |
| | 6 mth | at -20 °C |

# Anti double stranded DNA (Anti dsDNA)

## Summary

Double stranded DNA (dsDNA) antibodies (1:10 or greater) are found in 50-60 percent of Systemic lupus Erthematosus (SLE), 20-30 percent in Sjögren syndrome, 20-25 percent in Mixed Connective Tissue Disease (MCTD), and less than 5 percent in Progressive Systemic Sclerosis (PSS). High titers of antibody to native (double stranded) DNA is specific for SLE.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:10 | |
| Sample stability | 2 d | at 2-8˚C |
| | 1 yr | at -20˚C |

# Anti Endomysium Abs (IgA, IgG, IgM)

## Summary

The determination of anti-endomysium antibodies is important in the diagnosis of gluten sensitive enteropathy (celiac disease, non-tropical sprue), Duhring's herpetiform dermatitis. IgG antibodies is a valuable indicator in the diagnosis of past infection, whereas IgM/IgA antibodies will be found in recent infection.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:20 | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Anti Gliadin Abs (IgA,IgG,IgM)

## Summary

The protein gliadin is found in gluten, a protein found in many grains . A permanent intolerance to gluten is the cause of celiac disease, which causes villous atrophy and thus decreased absorption of nutrients. The clinical manifestations of this include chronic diarrhoea, abdominal pain, flatulence, and weight loss. Children suffering from celiac disease demonstrate slow growth. In adults it is also possible that gluten sensitivity can develop without any classical symptoms.

Anti-Gliadin titres correlate with the condition of the mucous membrane of the small intestine. A gluten-free diet results in a decrease in the antibody titre and thus leads to a remission of the disease.

The determination of IgG/IgA antibodies is a valuable indicator in the diagnosis of beginning autoimmune diseases, whereas IgM antibodies will be found in progressive stages of manifested autoimmune disorders.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative | : <8 U/mL |
| | Equivocal | : 8-12 U/mL |
| | Positive | : >12 U/mL |
| Sample stability | 5 d | at 2-8 °C |
| | 6 mth | at -20 °C |

# Anti Glomerular Basement Membrane Abs (GBM)

## Summary

Anti-Glomerular Basement Membrane (GBM) antibodies are associated with an array of anti-GBM nephritis conditions characterized by rapidly progressive renal failure and involvement of the lungs. With lung hemorrhage, the condition is referred to as Goodpasture's syndrome.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:4 | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Anti Glutamic Acid Decarboxylase (Anti GAD)

## Summary

GAD autoantibodies are found in type 1 diabetes and in the rare neurological disorder Stiff-Man Syndrome (SMS). However, the GAD autoantibodies profile in the two diseases differs. Autoantibodies of SMS patients recognize a combination of linear and conformational epitopes of GAD, while GAD65 autoantibodies in patients with type 1 diabetes are predominantly directed to the conformational epitopes. GAD65 autoantibodies (GAD65 Abs) are present in 70-80% of newly diagnosed patients with type 1 diabetes. The combination of the autoantibodies to GAD65 and Insulinoma Antigen 2(IA2) is highly relevant for risk assessment of type 1 diabetes in children and adolescents. These tests in combination are more sensitive and predictive than Islet cell Antibodies (ICA) in risk groups, e.g. relatives of patients with type 1 diabetes. GAD65 Abs also occurs in a subset of adults with type 2 diabetes. These patients can have pronounced hyperglycemia, and after therapy with oral hypoglycemic agents for several months to years they may become insulin dependent. Therefore, these patients are thought to have a slowly progressive form of type 1 diabetes, often called latent diabetes or Latent Autoimmune Diabetes in Adults (LADA). The presence of GAD65 Abs in sera of such patients is a sensitive and specific marker for future insulin dependency.

| | |
|---|---|
| Method | ELISA |
| TAT | 2 d |
| Sample type | S |
| Expected value | Negative :<10 IU/mL |
| | Positive: ≥ 10 IU/mL |
| Sample stability | 2 wk at 2-8˚C |

# Anti Insulin Abs (Type I DM)

## Summary

Autoantibodies against insulin can appear in the prediabetic phase of type I diabetes mellitus or upon its manifestation. These autoantibodies are found in 50 to 70 % of children with diabetes mellitus and in 20 to 30 % of affected adults. In rare cases, insulin-dependent diabetes patients generate antibodies against exogenous insulin. These antibodies can give rise to insulin-resistance and should therefore be controlled when monitoring insulin therapy.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <12 U/mL | |
| | Positive: >18 U/mL | |
| Sample stability | 5 d | at 2-8°C |
| | 6 mth | at -20°C |

# Anti Intrinsic Factor (IgG, IgM)

## Summary

The detection of IgG and IgM class autoantibodies against intrinsic factor is used for the diagnosis of pernicious anemia (Biermer´s anemia), which causes damage to the stomach mucosa. This hinders the synthesis of intrinsic factor and results in a vitamin B12 deficiency. In addition, autoantibodies against parietal cells and intrinsic factor appear as they are detected in 50 to 70 % of affected patients and are highly specific to this disease. The determination of IgG antibodies is a valuable indicator in the diagnosis of beginning autoimmune diseases, whereas IgM antibodies are present in progressive stages of manifested autoimmune disorders.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <6.0 IU/mL | |
| Sample stability | 5 d | at 2-8°C |
| | 6 mth | at -20°C |

# Anti Islet Cells Abs

## Summary

Demonstration of Islet Cell antibodies enables serologic assessment or possible detection of pancreatic disease. The presence of a (histologically defined) circulating antibody to one or more of the islet cell antigens can aid in patient diagnosis and prognosis.

Islet Cell antibodies have been associated with a group of “autoimmune” endocrine disorders, more specifically with insulin dependent diabetes. Organ-specific autoimmunity is characterized by the presence of antibodies in patients that can be detected years before the onset of the clinical symptoms. These antibodies are useful monitors to detect, well before metabolic tests can detect, humoral deficiencies.

So far, islet-cell antibodies have only been detected in association with overt autoimmunity, almost exclusively in insulin dependent diabetes, sometimes before onset as well as after the patient has been diagnosed.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:5 |
| Sample stability | 3 d at 2-8˚C |

# Anti Keratin Abs

## Summary

The keratin antibodies react with the stratum corneum and are pointed to the protein filaggrin located in the squamosous epithelium. The antibodies have a high specifity but medium sensitivity for a rheumatoid arthritis and can be detected with rheuma factor-negative patients mainly at early stages.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:40 | |
| Sample stability | 1 wk | at 2-8˚C |
| | Long term storage | at -20˚C |

# Anti Mitochondrial Abs (AMA)

## Summary

The detection of AMA is the best indicator for the diagnosis of Primary Biliary Cirrhosis (PBC) which is an organ-specific autoimmune disease characterized by chronic progressive destruction of small intrahepatic bile ducts with portal inflammation and ultimately fibrosis.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:20 |
| Sample stability | 8 h at room temperature |
| | 1 wk at 2-8˚C |

# Anti Mullerian Hormone / Mullerian Inhibiting Substance (MIS/AMH)

## Summary

AMH inhibits the recruitment of primordial follicles and also decreases the responsiveness of growing follicles to FSH. In contrast to most hormonal biomarkers of the follicular status, AMH is exclusively produced by the granulosa cells of a wide range of follicles (primary to the early antral stages). It has been shown that AMH concentration in serum is directly related to the antral follicle count and is a better indicator of the ovarian reserve than FSH, inhibin B or estradiol on cycle day. Moreover, in contrast to the other cited markers, AMH level in serum does not vary significantly neither during a menstrual cycle nor between consecutive cycles . In assisted reproductive technology, levels of AMH are also predictive to the ovarian response and of the chance of successful pregnancy.

| | |
|---|---|
| Remarks | Fresh sample. |
| | 1 day max storage at 2-8°C |
| Method | ELISA |
| TAT | Dy/A |
| Sample type | S |
| Expected value | 0.7-5 ng/mL |
| | 2-3 folds higher in PCOS |
| Sample stability | 1 d at 2-8°C |

# Anti Nuclear Abs (ANA)

## Summary

The detection of antinuclear antibodies has an aids in determining Systemic Lupus Erythematosus (SLE) and differentiating clinically similar connective tissue disorders, in addition, ANA may be associated with numerous drug-induced lupus syndromes which mimic the spontaneous form of SLE clinically.

Different patterns have been associated with a variety of autoimmune disorders, although overlap may occur. Some of the more common patterns include:

• Homogenous (diffuse) pattern - associated with Systemic Lupus Erythematosus (SLE ) and mixed connective tissue disease

• Speckled pattern - associated with Systemic Lupus Erythematosus (SLE), Sjogren syndrome, scleroderma, polymyositis, rheumatoid arthritis, and mixed connective tissue disease

• Nucleolar pattern - associated with scleroderma and polymyositis

• Centromere pattern (peripheral) - associated with scleroderma and CREST (Calcinosis, Raynaud's syndrome, Esophogeal dysmotility, Sclerodactyly, Telangiectasia)

• Anti nuclear membrane, multiple nuclear dot patterns - associated with Primary Biliary cirrhosis (PBC).

• Mitotic Spindle pattern - is nonspecific and can be seen in a variety of disease states; has no known clinical significance.

• Centrosome (Centriole) pattern - is a rare pattern that may be seen in patients with nonspecific rheumatic diseases and some chronic post-viral syndromes.

• Golgi Apparatus pattern - is observed in patients with Systemic Lupus Erythematosus (SLE), Sjogren's syndrome, mixed connective tissue disease, and is seen in 30% to 80% of rheumatoid arthritis.

• Midbody pattern - is a rare pattern found to occur in scleroderma and Raynaud's phenomenon.

• Cytoplasmic Staining pattern - is associated with antibodies that may be derived from multiple cytoplasmic proteins including Jo-1 (20%-40% polymyositis), ribosome P (10%-15% Systemic Lupus Erythematosus [SLE]), and mitochondrial M2 (90% PBC and 40% scleroderma).

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:40 |
| Sample stability | 8 h at room temperature |
| | 2 d at 2-8˚C |

# Anti Ovarian Abs

## Summary

Anti ovarian antibodies are typically found in women with premature ovarian failure and unexplained infertility. Anti ovarian antibodies are also found in Addison's disease, thyroid disease and endometriosis.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | <1:5 | |
| Sample stability | 1 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Anti-Phospholipids

## (Anti-Phosphatidic acid , Anti-Phosphatidyl Serine, Anti-Phosphatidyl Inositol (IgG/IgM))

### Summary

Anti-Phospholipid test is used to detect autoantibodies (class IgG and IgM) against three other negatively charged phospholipids: phosphatidyl serine, posphatidyl inositol, and phosphatidic acid. This test is thus an outstanding tool for the serological detection of primary and secondary antiphospholipid syndrome.

Anti-Phosphatidic acid antibodies IgG/IgM almost exclusively appear together with cardiolipin antibodies and/ or phospatidyl serine antibodies. Their detection supports a diagnosis of Antiphospholipid Syndrome (APS) and helps in the assessment of the risk of thrombosis in patients with Systemic Lupus Erythematosus (SLE) and lupus-like diseases. Anti Phospholipid Syndrome (APS, Hughes Syndrome) is a systemic autoimmune disease that causes thrombosis, recurrent miscarriage, and intrauterine fetal death. The anti- phospholipids' antibodies family includes anti cardiolipin antibodies, anti β-2 Glycoprotein I antibodies and Lupus Anticoagulant.

Antibodies against phosphatidyl inositol, a negatively charged phospholipid, appear together with cardiolipin antibodies and antibodies against phospahtidyl serine. However, anti-phoshatidyl inositol has also been detected by itself in patients with Antiphospholipid Syndrome (APS). In the absence of anti-cardiolipin antibodies and anti-phospahtidyl serine, the detection of phosphatidyl inositol antibodies thus points a finger toward APS. In younger patients with cerebral ischaemia of unclear origins, anti-phosphatidyl inositols are the most numerous phospholipid antibodies. In addition, their appearance seems to be associated with a tendency toward miscarriage.

Phospatidyl serine autoantibodies are associated with Antiphospholipid Syndrome (APS). Anti-cardiolipin and antibodies against phosphatidyl serine do not always appear together. For the diagnosis of APS, some authors hold the detection of anti-phospahtidyl serine to be more meaningful than the detection of anti-cardiolipin. The detection of phosphatidyl serine autoantibodies also allows for the assessment of the risk of thrombosis or miscarriage in patients with SLE or collagenosis.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: < 10 IU/mL | |
| Sample stability | 5 d | at 2-8˚C |
| | 6 mth | at -20 ˚C |

# Anti Reticulin Abs (IgA, IgG ,IgM)

## Summary

Reticulin Abs are found in patients with gluten-sensitive enteropathy dermatis herpetiformis, and Crohn's disease. IgA class reticulin antibodies are found in dermatitis herpetiformis (25%) and celiac disease (60%), but not in other autoimmune problems. IgG class reticulin antibodies are found in other disease states, especially bullous dermatoses, and sometimes in normal patients.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:10 | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Anti Saccharomyces Cerevisiae (IgA,IgG)

## Summary

Anti-Saccharomyces Cerevisiae antibodies (ASCAs) have been proposed as serological markers, which may differentiate Crohn's disease (CD) from Ulcerative Colitis (UC) and predict disease phenotype. IgA Antibodies Against Saccharomyces Cerevisiae (ASCA) occur in patients with Crohn's disease in 60-75% but are rare in patients with ulcerative colitis. IgG ASCA also often occurs together with IgA ASCA but some patients have only one immunoglobulin class.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | 2 d | |
| Sample type | S | |
| Expected value | Negative: <10 U/mL | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Anti single stranded DNA (Anti ssDNA)

## Summary

The determination of single stranded DNA is used as an indicator for drug induced Lupus Eythematosus (LE), rheumatic arthritis and leukosis.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:10 |
| Sample stability | 2 d at 2-8˚C<br>1 yr at -20˚C |

# Anti Skin Abs (IgA, IgG,IgM)

## Summary

The detection of skin-specific antibody indicates autoimmune bullous dermatomes. The intercellular substance antibody has been associated with Pemphigus Vulgaris (PV). From 80 to 90% of PV Patients have intercellular antibodies. Low antibody levels are sometimes found in burn and lupus erythematosus patients as well as normal individuals. Circulating antibodies to basement membrane are found in greater than 70% of Bullies Pemphigoid (BP) patients.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:10 | |
| Sample stability | 4-7 d | at 2-8˚C |
| | 3-6 mth | at -20˚C |

# Anti Smooth Muscle Abs (ASMA)

## Summary

The detection of smooth antibody indicates chronic aggressive hepatitis and autoimmune hepatitis diseases.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:20 | |
| Sample stability | 1 d | at 2-8˚C |
| | Long term storage | at -20˚C |

# Anti Sperm Abs

## Summary

Anti sperm antibody test looks for special proteins (antibodies) that fight against a man's sperm in blood, vaginal fluids, or semen. The test uses a sample of sperm and adds a substance that binds only to affected sperm.

Semen can cause an immune system response in either the man's or woman's body. The antibodies can damage or kill sperm. If a high number of sperm antibodies come into contact with a man's sperm, it may be hard for the sperm to fertilize an egg. The couple has a hard time becoming pregnant. This is called immunologic infertility.

A woman can have an allergic reaction to her partner's semen and make sperm antibodies. This kind of immune response is not fully understood but may affect fertility. This is a rare cause of infertility.

Anti sperm antibodies prevent the fertilization of the oocyte into the female genital tract and are therefore one of the major reasons for an immunologically induced infertility.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | SF/S | |
| Expected value | Negative | |
| Sample stability | 1 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Anti Striated Muscle Abs

## Summary

Demonstration of striated muscle antibody enables serologic detection of muscular diseases. The presence of a histologically defined skeletal antibody in conjunction with a positive acetylcholine receptor site assay is helpful in the confirmatory diagnosis of Myasthenia Gravis (MG).

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:10 | |
| Sample stability | 4-7 d | at 2-8˚C |
| | 3-6 mth | at -20˚C |

# Anti Testicular Abs

## Summary

Antibodies against different structures of the testicle are reported in male sterility with azoospermia. Anti-Leydig cell antibodies are observed in steroid defects. Antibodies staining the interstitial cells of the testis have been detected in patients with Addison's disease.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:5 | |
| Sample stability | 3 d | at 2-8˚C |
| | 3-6 mth | at -20˚C |

# Anti-Thrombin III

## Summary

Hereditary antithrombin deficiency is inherited in an autosomally dominant fashion and is prevalent in both men and women in approximately the same amount.

- Type I deficiency: Antithrombin concentrations and activities are reduced to the same extent due to decreased synthesis in the liver.
- Type II deficiency: Antithrombin concentration remains normal, but its biological activity is reduced due to altered molecular structure.

Acquired antithrombin deficiency is much more common than hereditary antithrombin deficiency, but nevertheless much more rarely causes an increased risk of thrombosis. Antithrombin concentration and activity are both decreased to the same extent. Antithrombin deficiency can be caused by:

- Decreased synthesis due to restricted (hepatic disease) or immature functioning of the liver (newborn and premature babies). In general, all liver-dependent coagulation factors and inhibitors are decreased to the same extent. Because of the overall balanced hemostatic equilibrium, an increased risk of thrombosis does not arise.
- Intravasal antithrombin loss due to its relatively small molecular weight:
  - Renal loss in the course of a nephrotic syndrome.
  - Enteral loss in the course of protein-loss enteropathies.
  - Increased permeation into the extravasal space due to increased blood vessel permeability.

- Increased loss due to elevated activation of the coagulation process or activation of coagulation over a longer period of time, e.g.
  - Postoperatively.
  - Continuous intravenous heparin therapy.
  - Consumptive coagulopathy, Disseminated Intravascular Coagulation (DIC).
- In the course of septic infection there is a direct relationship between antithrombin activity loss and the severity of the infection or the course of the sepsis. When septic disease is clinically suspected, early determination of antithrombin activity is indicated to ensure early detection of DIC.

| | |
|---|---|
| Method | Kinetic colorimetric |
| TAT | Dy/AB |
| Sample type | CP |
| Expected value | 80-120 % |
| Sample stability | 5 d at 2-8˚C |

# Anti Thyroid Microsomal Abs (ATMA)

## Summary

ATMA are the hallmarks of human autoimmune thyroid diseases and commonly associated with the presence of Graves 'disease and Hashimoto's Thyroiditis disease.

| | |
|---|---|
| Method | IF |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Negative: <1/100 |
| Sample stability | 8 h at room temperature |
| | 1 wk at 2-8˚C |
| | 6 mth at -20˚C |

# Anti Thyroid Peroxidase (ATPO)

## Summary

ATPO antibodies are detectable in over 90 % of patients with Hashimoto's thyroiditis and over 70 % of those with Graves' disease (autoimmune hyperthyroidism), irrespective of the functional state of the thyroid gland. Moderately elevated TPOAb values are sometimes found in patients without other evidence of thyroid disease, especially in the elderly.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | <34 IU/mL | |
| Sample stability | 8 h | at room temperature |
| | 1 wk | at 2-8˚C |
| | 6 mth | at -20˚C |

# Anti Tissue-Transglutaminase (IgA,IgG)

## Summary

Anti-tTG IgA are a highly sensitive marker for celiac disease with 95-100%, and have a specificity of 90 to 97%. Celiac disease reported poor growth, abnormal stools and abdominal distension as common symptoms in children. Patients suffering from this disease showed a flat appearance of the mucosa, with villous atrophy and hypertrophy of the crypts, diarrhea, various gastrointestinal problems, anemia, fatigue, psychiatric problems or they may be asymptomatic.

The increased association of celiac disease with selective IgA deficiency is a potential source of false-negative IgA. Therefore testing for IgG class autoantibodies is recommended if celiac disease is suspected.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <10 U/ml | |
| Sample stability | 5 d | at 2-8 °C |
| | 6 mth | at -20 °C |

# Anti TSH Receptor Abs (TRAb)

## Summary

TRAb determination is used in the detection or exclusion of autoimmune hyperthyroidism and its differentiation from disseminated autonomy of the thyroid gland.

The presence of TRAb indicates that the patient's thyrotoxicosis is of autoimmune etiology rather than due to toxic nodular goiter.

Hyperthyroidism in Graves' disease (autoimmune hyperthyroidism) is caused by autoantibodies to the TSH receptor (TSHR), Monitoring the therapy of Graves' disease patients and prediction of relapse, thereby constituting an important decision-making aid in the management of the treatment, Low levels or the absence of TRAb after a course of drug treatment may indicate disease remission, and therefore the withdrawal of therapy can be considered.

TRAb measurement during the last trimester of pregnancy is important since they cross the placenta and can cause neonatal thyroid disease.

| | |
|---|---|
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Negative: <1.8 IU/L |
| | Borderline: 1.8-2.0 IU/L |
| | Positive: >2.0 IU/L |
| Sample stability | 3 d at 2-8 °C |
| | 1 mth at -20 °C |

# Anti Vasopressin (AVP)

## Summary

AVP has been shown to be released upon both osmotic and non-osmotic stimuli, and its release into Peripheral Blood Causes effects upon a number of factors, including emotional stress, posture, blood volume and temperature. Alcohol appears to inhibit AVP secretion. Serum AVP measurement is used clinically for studies involving diabetes insipidus, Syndrome of Inappropriate ADH secretion (SIADH), ectopic AVP production and psychogenic water intoxication.

| | |
|---|---|
| Remarks | Fresh sample. |
| Method | ELISA |
| TAT | 2 d |
| Sample type | EWB |
| Expected value | Up to 13 pmol/L |
| Sample stability | 2 d at 2-8 ˚C |

# Antineutrophil cytoplasmic Abs (ANCA - cANCA,pANCA)

## Summary

Antineutrophil Cytoplasmic Antibodies (ANCA) are a sensitive and specific marker for ANCA-associated systemic vasculitis. ANCA occur in patients with wegener's granulomatosis, microscopic polyarteritis, Necrotizing or crescentic glomerulonephritis, other vasculitides, inflammatory bowel disorders (primary ulcerative colitis) and primary sclerosing cholangitis. cANCA are found primarily in patient with wegener's granulomatosis, microscopic polyarteritis, where is pANCA occur in various vasculitic disorders, ulcerative colitis and primary sclerosing cholangitis.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:20 | |
| Sample stability | 5 d | at 2-8˚ C |
| | 6 mth | at -20˚C |

# Antistreptolysin O (ASOT)

## Summary

The measurement of Antistreptolysin O antibodies is used for the diagnosis of a streptococcal infection or to indicate a past exposure to streptococci and helps direct antimicrobial treatment and is used to assist in the diagnosis of scarlet fever, rheumatic fever and post infectious glomerulonephritis. In the case of streptococcal infection of the respiratory tract the ASOT titer rises after 4-8 weeks and then decreases slowly over weeks or months. Single titers do not allow any statement. Only double increased titers indicate a previous infection.

| | | |
|---|---|---|
| Method | Turbidometric | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Children : Up To 150 IU/mL | |
| | Adult : Up To 200 IU/mlL | |
| Sample stability | 2 d | at room temperature |
| | 2 d | at 2-8˚C |
| | 3 mth | at -20˚C |

# Apolipoprotien A-1 (APOAT)

## Summary

Apolipoprotien A-1 levels increase in liver disease, pregnancy and as a result of estrogen administration (e.g. oral contraceptive). It is decreased in inherited hypo-α-lipopoteinemia (e.g. tangier disease), cholestasis, sepsis and atherosclerosis. The calculation of Apoliporotien B: Apolipoprotien A-1 ratio can reflect a lipid metabolism disorder and the risk of developing atherosclerosis of coronary heart disease particularly well thus providing an excellent addition to the classical HDL/LDL cholesterol determination.

| | | |
|---|---|---|
| Remark | Fasting | |
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP. | |
| Expected value | Male (♂): | 104-202 mg/dL |
| | Female (♀): | 108-225 mg/dL |
| Conversion factors | g/L x 35.7 =µmol/L | |
| | mg/dL x 0.01 = g/Lg/L x 100 =mg/dL | |
| Sample stability | 1 d | at room temperature |
| | 3 d | at 2-8˚C |
| | 2 mth | at - 20˚C |

# Apolipoprotien B (APOBT)

## Summary

Apolipoprotien B levels increase in pregnancy, hypercholesterolemia, LDL receptor defects, bile obstruction, type II hyperlipidemia and nephritic syndrome. It is decreased during liver disease, α-β lipoprotienemia. The calculation of Apoliporotien B: Apolipoprotien A-1 ratio can reflect a lipid metabolism disorder and the risk of developing atherosclerosis of coronary heart disease particularly well thus providing an excellent addition to the classical HDL/LDL cholesterol determination.

| | | |
|---|---|---|
| Remark | Fasting | |
| Method | Turbidmetric | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP. | |
| Expected value | Male (♂): 66-133 mg/dL | |
| | Female (♀): 60-117 mg/dL | |
| Conversion factors | g/L x 1.95 =µmol/L | |
| | mg/dl x 0.01 = g/L | |
| | g/L x 100 =mg/dL | |
| Sample stability | 1 d | at room temperature |
| | 3 d | at 2-8˚C |
| | 2 mth | at - 20˚C |

# Aspartate Aminotransferase (AST)

## Summary

Elevated serum levels are found in diseases involving hepatic, cardiac, muscle and kidney tissue, myocardial infarction and hepatobiliary diseases such as cirrhosis, metastatic carcinoma and viral hepatitis. In patients undergoing renal dialysis or those with vitamin B6 deficiency, serum AST may be decreased.

| | |
|---|---|
| Method | Enzymatic |
| TAT | Dy/AB |
| Sample type | S/EP/HP |
| Expected value | Male (♂): Up To 37 U/L |
| | Female (♀): Up To 31 U/L |
| Conversion factors | U/L x 0.0167 = µkat/L |
| Sample stability | 1 d at room temperature |
| | 1 wk at 2-8 °C |

# Azathioprine (Immuran)

## Summary

Azathioprine is an immunosuppressive drug used in organ transplantation and autoimmune diseases such as rheumatoid arthritis and pemphigus , inflammatory bowel disease. It is also used in multiple sclerosis, autoimmune hepatitis and restrictive lung disease. Patients receiving immunosuppressants, including Azathioprine, are at increased risk of developing lymphoma and other malignancies. Physicians using this drug should be very familiar with this risk as well as with the mutagenic potential to both men and women and with possible hematologic toxicities.

| | | |
|---|---|---|
| Method | HPLC | |
| TAT | 4 d | |
| Sample type | S | |
| Expected value | 0.1-2.0 mg/L | |
| Sample stability | 5d | at room temperature |
| | 5 d | at 2-8˚C |
| | 4 mth | at -20˚C |

# Bence Jones Protein

## Summary

Bence Jones Protein (monoclonal free light chains of immunoglobulins) were first described in the urine of patients with multiple myeloma and also associated with other abnormalities of the immune system including leukaemia, lymphoma, malignant bone marrow cancer, renal failure, lytic bone disease or anemia and autoimmune and infectious diseases.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/AB | |
| Sample type | US | |
| Expected value | Negative | |
| Sample stability | 2h | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 1 mth | at - 20˚C |

# Bicarbonate (CO2)

## Summary

The bicarbonate content of serum or plasma is a significant indicator of electrolyte dispersion and anion deficit. Together with pH determination, bicarbonate measurements are used in the diagnosis and treatment of numerous potentially serious disorders associated with acid-base imbalance in the respiratory and metabolic systems .

| | | |
|---|---|---|
| Method | Enzymatic | |
| TAT | Dy/AB | |
| Sample type | S/HP | |
| Expected value | 22-29 mmol/L | |
| Sample stability | 4 h | at room temperature |
| | 1 wk | at 2-8˚C |
| | 1 mth | at -20˚C |

# Bilirubin direct

## Summary

The elevation of direct bilirubin typically results from obstruction either within the liver (intrahepatic) or a source outside the liver or a tumor blocking the bile ducts.

Bilirubin direct test indicates the suspicion of intrahepatic or extrahepatic icterus.

| | |
|---|---|
| Remarks | Dispatch in light protected vessel, no lipemic samples. |
| Method | Kinetic |
| TAT | Dy/AB |
| Sample type | S/HP |
| Expected value | Up to: 0.3 mg/dL |
| Conversion factors | µmol/L × 0.0585 = mg/dL |
| | mg/dL × 17.1 = µmol/L |
| Sample stability | 2 d at room temperature |
| | 1 wk at 2-8 °C |
| | 6 mth at -20 °C |

# Bilirubin Total

## Summary

Diseases or conditions which, through hemolytic processes, produce bilirubin faster than the liver can metabolize it, cause the levels of unconjugated (indirect) bilirubin to increase in the circulation. Liver immaturity and several other diseases in which the bilirubin conjugation mechanism is impaired cause similar elevations of circulating unconjugated bilirubin. Bile duct obstruction or damage to hepatocellular structure causes increases in the levels of both conjugated (direct) and unconjugated (indirect) bilirubin in the circulation.

| | | |
|---|---|---|
| Remark | Dispatch in light protected vessel,<br>no lipemic sample. | |
| Method | Kinetic | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Age | Value (mg/dL) |
| | 1 d | <6.0 |
| | 2 d | <8.0 |
| | 3 – 5 d | <12.0 |
| | Children & Adults | <1.1 |
| Conversion factors | µmol/L × 0.0585 = mg/dL | |
| | mg/dL × 17.1 = µmol/L | |
| Sample stability | 1 d | at room temperature |
| | 1 wk | at 2-8 °C |
| | 6 mth | at -20 °C |

# Bladder Tumor Marker (NMP22)

## Summary

The NMP22 is a bladder marker intended as an aid in the management of patients with Transitional Cell Carcinoma of the Urinary Tract (TCCT/UT). It is used after surgical treatment to identify patients with residual or rapidly recurring TCC/UT.

| | | |
|---|---|---|
| Method | EIA/ICT | |
| TAT | Dy/AB | |
| Sample type | US | |
| Expected value | Negative: < 10 U/mL | |
| Sample stability | 2 d | at room temperature |
| | 3 d | at 2-8˚C |
| | 1 mth | at -20˚C |

# Blood film

## Summary

The blood film is used to evaluate blood cell populations. It also indicates the presence of abnormal or immature cells, a deficiency, disease or disorder that is affecting blood cell production such as anaemia, decreased or abnormal production of cells in the bone marrow or increased cell destruction.

| | | |
|---|---|---|
| Method | Blood smear | |
| TAT | Dy/AB | |
| Sample type | EWB | |
| Expected value | Normal | |
| Sample stability | 2 h | at room temperature |
| | < 8 h | at 2-8˚C |

# Borrelia Burgdorferi (IgG,IgM)

## Summary

Borrelia burgdorferi is a gram negative bacteria which causes the disease syndrome known as Lyme-Borreliosis which is the most common tick-borne disease transmitted to human hosts by infected ticks.

The time course of Lyme disease can be divided into three separate stages.

- Stage I starts a few days up to several weeks after tick bite and subsequent infection with Borrelia burgdorferi. Patients suffer from non-specific symptoms such as fever, headache, muscle pain, joint pain, and exhaustion. Dermal manifestations with Erythema Migrans (EM) as a characteristic symptom of early disease are displayed by 30 to 60 % of infected persons. The prevalence of IgM is 20-50% after beginning of infection.
- Stage II occurs between a few weeks and several months post infection as a systemic disease. In addition to non-specific symptoms, in particular neurological disorders (Morbus Bannwarth), less frequently Lyme carditis and opthalmological disorders may be observed, IgG and IgM antibodies increased.
- stage III symptoms may occur up to several years after the tick bite and are characterized by dermatological diseases (Acrodermatitis Chronicum Athrophicans (ACA), diseases of the joints (Lyme-Arthritis), and neurological diseases (chronical encephalomyelitis), the prevalence of IgG antibodies is 90-100%, no IgM antibodies founded.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:10 |
| Sample stability | 2 wk at 2-8˚C |
| | 1 yr at -20˚C |

# Brucella Abs (IgG,IgM)

## Summary

Brucella is a gram negative bacterium which is not considered a contagious disease. Humans are infected by contact with fluids from infected animals (sheep, cows and pigs) or derived food products like unpasteurized milk and cheese. Malta fever is one of the most common diseases caused by Brucella.

Burcella's IgG and antibodies screening tests are important for the suspicion of past infection, fever of unknown origin and animal contact, while Burcella's IgM test is important for the suspicion of a recent infection.

| | |
|---|---|
| Method | ELISA |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1.0 |
| Sample stability | 2 d at room temperature |
| | 2 wk at 2-8˚C |
| | 6 mth at -20˚C |

# C1 Esterase Inhibitor

## Summary

C1-Esterase Inhibitor is formed predominantly in hepatocytes. Through initial activation of the complement system, a permanent decrease is observed. Hereditary deficiency leads to episodic disturbances of tissue permeability of the skin, gastrointestinal tracts and, rarely, respiratory tract.

Main symptoms are circumscribed edemata and convulsive, recurrent abdominal pain. Deficiency can lead to angioneurotic edema with acute swellings of lips and eyelids, gastrointestinal colics and particularly dangerous swellings of larynx and pharynx. An edema of glottis can lead to asphyxia.

| | | |
|---|---|---|
| Method | IF | |
| TAT | 2d | |
| Sample type | S | |
| Expected value | 0.048-0.725 g/L | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8˚C |

# C1q

## Summary

Clinical significance has deficiencies of the subunit of the first complement factors C1q, C1r and/or C1s. The most important defect is the C1q-deficiency, which often is associated with Systemic Lupus Erythematosus (SLE). Decreased levels are also detected with hypogammaglobulinemia, hypocomplementary urticaria-vasculitis and Severe Immune Deficiency (SCID).

| | |
|---|---|
| Method | RID |
| TAT | 10-14 d |
| Sample type | S |
| Expected value | 5.0 - 30.0 mg/dl |
| Sample stability | 2h at room temperature |
| | 2 wk at -20˚C |

# Calcitonine

## Summary

Calcitonin is a polypeptide, which is synthesised in the parafollicular cells (C-cells) of the thyroid gland. It is the antagonist of Parathyroid Hormone (PTH), which inhibits the activity of osteoclasts and causes a reduction of the calcium concentration in the blood. The clinical significance is the diagnosis and therapy monitoring of the medullar or C-cell carcinoma. Since C-cell carcinomas appear to have a genetic component with 20% familial accumulation, screening of calcitonin levels is recommended in such cases.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | 2 d | |
| Sample type | S | |
| Expected value | Female (♀): | < 11.5 pg/mL |
| | Male (♂): | < 18.2 pg/mL |
| Sample stability | 8 h | at room temperature |
| | 3 mth | at 2-8˚C |
| | 1 wk | at - 20˚C |

# Calcium (Ca)

## Summary

Serum calcium levels and hence the body content are believed to be controlled by Parathyroid Hormone (PTH), calcitonin, and vitamin D. An imbalance in any of these modulators leads to alterations of the body and serum calcium levels. Increases in serum PTH or vitamin D are usually associated with hypercalcemia. Increased serum calcium levels may also be observed in multiple myeloma and other neoplastic diseases. Hypocalcemia may be observed in hypoparathyroidism, steatorrhea, nephrosis, and pancreatitis.

| | | | |
|---|---|---|---|
| Method | Colorimetric | | |
| TAT | Dy/AB | | |
| Sample type | S/HP | | |
| | U24 | | |
| Expected value | 24U | Children (mg/kg/d) | Adult (mg/d) |
| | | <6 | 100-320 |
| | S/HP: | Age | Value (mg/dL) |
| | | 0-2 mth | 7.6-10 |
| | | 2-12 mth | 8.4-10.8 |
| | | 1-4 yr | 8.4-10.4 |
| | | 5-20 yr | 9.2-11 |
| | | 21-50 yr | 8.8-10.2 |
| | | >50 yr | 8.4-9.7 |
| Conversion factors | mmol/L x 4.01 = mg/dL | | |
| | mmol/L x 2 = mval/L | | |
| Sample stability in S/HP | 1 wk | at room temperature | |
| | 3 wk | at 2-8 °C | |
| | 8 mth | at -20 °C | |
| Sample stability in 24U | 2 d | at room temperature | |
| | 4 d | at 2-8 °C | |
| | 3 wk | at -20 °C | |

# Calprotectin

## Summary

As a non-invasive marker Calprotectin can be used as a sensitive screening test prior to perform endoscopy. The diagnosis of Calprotectin is used to recognize the acute inflammations and inflammation phases of the intestinal tract; suspicion of polyps, suspicion of colorectal carcinomas and as a prevention marker.

Negative values don't give any hints about an acute inflammatory disease of the intestinal tract, inflammatory episode nor about polyps of the intestinal tract < 10 mm.

Positive values point to inflammatoric disease of the intestinal tract, adenomas or tumorous processes. A coloscopic diagnostic examination should follow. The values over 2000 mg/kg can be detected according to the type, dimension and severity of the inflammatory disease of the intestinal tract. The values of a wide range with a median of 350 mg/kg can be found at adenomas or tumors.

| | | |
|---|---|---|
| Method | EIA | |
| TAT | 10-14 d | |
| Sample type | ST | |
| Expected value | < 50.00 mg/kg | |
| Sample stability | 1 d | at room temperature |
| | 2 d | at 2-8˚C |
| | Long term storage | at (-20˚C dry ice) |

# Cancer Antigen 125 II (CA 125 II)

## Summary

CA 125 is found in tumors and/or diseases of Ovary, bile ducts, gastrointestinal tract, mamma and pleural mesothelioma.

Markedly elevated levels have been found in benign liver diseases such as hepatitis and cirrhosis.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S /HP/EP/CP | |
| Expected value | Up To 35 IU/mL | |
| Sample stability | 4 d | at room temperature |
| | 5 d | at 2-8 °C |
| | 3 mth | at -20 °C |

# Cancer Antigen 15-3 (CA 15-3)

## Summary

CA 15-3 is found in tumor marker and/or diseases of Mamma, ovary, cervix and endometrium CA15-3 is used for treatment monitoring (progression or remission)

| | | |
|---|---|---|
| Remarks | Sample should not be taken from patients receiving therapy with high biotin doses<br>Until at least 8 hours following the last biotin administration. | |
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S /HP/EP | |
| Expected value | Up to 25 IU/mL | |
| Sample stability | 5 d | at 2-8 °C |
| | 3 mth | at -20 °C |

# Candida albicans Abs (IgA, IgG,IgM)

## Summary

Candida albicans is a disploid asexual fungus (a form of yeast) and a causal agent of opportunistic oral and vaginal infections in humans. Candidiasis usually develops in persons whose immunity is compromised (such as HIV-positive patients), most frequently in the presence of disturbed cellular immunity. Diabetes, pregnancy, progesterone therapy and intensive antibiotic treatment that eliminate the normal bacterial flora are among the predisposing factors. The mucosa is affected most often, less frequently the outer skin and inner organs (deep candidiasis). In oral cavity infections, a white, stubbornly adherent coating is seen on the cheek mucosa and tongue. Skin is mainly infected on the moist, warm parts of the body. Pathomorphologically similar to oral soor is vulvovaginitis.

Candida can spread to cause secondary infections of the lungs, kidneys and other organs. Chronic mucocutaneous candidiasis is observed as a sequel to damage of the cellular immune system.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative | |
| Sample stability | 2 d | at room temperature |
| | 2wk | at 2-8˚C |
| | 6mth | at - 20˚C |

# Cannabinoids (9-Carboxy-THC) (Marijuana)

## Summary

The acute effects of marijuana use, concomitant with the desired “high”, are memory impairment, time confusion, interference with learning, impaired motor skills and depersonalization. These effects are also manifested in chronic users in addition to cardiovascular, pulmonary, and reproductive effects. Marijuana is usually smoked, but maybe ingested, either incorporated into food or as a liquid extract (tea). It is rapidly absorbed from the lungs into the blood with rapid onset of effects; the onset is slower but prolonged when ingested.

| | | |
|---|---|---|
| Method | Turbidimetric /ICT | |
| TAT | Dy/A | |
| Sample type | US | |
| Expected value | Not detected /Negative | |
| Sample stability | 1 wk | at room temperature |
| | 1 mth | at 2-8˚C |
| | 3 yr | at -20˚C |

# Carbamazepine

## Summary

Carbamazepine is an antiepileptic drug for the treatment of epilepsy, trigeminal neuralgia, simple and complex, partial and generalized convulsive seizures in adults. It is effective as a sole agent in treating the above disorders, but can be used in combination with other antiepileptic drugs.

| | |
|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. |
| Method | FP |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | 4-10 μg/mL |
| Sample stability | 5d at room temperature |
| | 5 d at 2-8˚C |
| | 4 mth at -20˚C |

# Carbohydrate Antigen 19-9 (CA 19-9)

## Summary

CA 19-9 Assay values can assist in the differential diagnosis and monitoring of patients with pancreatic carcinoma. Patient with 10000 U/ml almost have distal metastasis. Elevated CA19-9 values are also found with a number of benign and inflammatory diseases of the gastrointestinal tract and the liver as well as cystic fibrosis. Undetectable CA 19-9 can be expected in patients and healthy persons with the rare blood group.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Up To 37 IU/mL | |
| Sample stability | 1 wk | at room temperature |
| | 1 mth | at 2-8 °C |
| | 3 mth | at -20 °C |

# Carcinoembryonic Antigen (CEA)

## Summary

High CEA concentrations are frequently found in cases of colorectal adenocarcinoma. Slight to moderate CEA elevations occur in 20-50% of benign diseases of the intestine, the pancreas, the liver and the lungs.

Smokers also have elevated CEA values.

CEA is indicated for therapy monitoring.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP/CP | |
| Expected value | Up To 4.6 ng/mL | |
| | Smokers: 3.5-10 ng/mL | |
| Conversion factors | ng/mL x 16.9 = mIU/mL | |
| | mIU/mL x 0.0592 =ng/mL | |
| Sample stability | 1wk | at room temperature |
| | 1 wk | at 2-8˚C |
| | 6 mth | at -20˚C |

# Catecholamines (Adrenaline/ Noradrenaline/L-Dopamine)

## Summary

The determination of Catecholamines ( Adrenaline/Noradrenaline and L-Dopamine) is important for the diagnosis of tumors of the sympathosuprarenal system,suspiction of neuroblastoma, pheochromocytoma or ganglioneuroma, arterial hypertension clarification.

| | |
|---|---|
| Remarks | 3 ml of the sample (Adrenaline/ Noradrenaline/ L-Dopamine) in equals amounts. |
| Method | ELISA |
| TAT | 2d |
| Sample type | EP |
| Expected value | Adrenaline: <100 pg/mL |
| | Noradrenaline: <600 pg/mL |
| | Dopamine: <100 pg/mL |
| Sample stability | 1d at Room temperature |
| | 2 d at 2-8 ˚C |
| | 1 mth at -20 ˚C |

# Cluster of Differentiation 4 (CD4)

## Summary

CD4 (T-helper cells) are a type of white blood cell that fights infection and considered as a primary targets of HIV.

The CD4 count is essential in assessing immune status and managing health care of HIV infected patients, the stage of HIV disease, guides treatment, and predicts how disease may progress.

| | |
|---|---|
| Method | FCM |
| TAT | 2 d |
| Sample type | EWB |
| Expected value | 500-1500 cells/mm3 |
| Sample stability | 2 d at 18-22˚C |

# Cluster of Differentiation 8 (CD8)

## Summary

The CD8 marker antigen expressed on the surface of cytotoxic T cells, T cell lymphoblastic lymphoma and hypo-pigmented mycosis fungoids, and also found on natural killer cells, cortical thymocytes, and dendritic cells. An elevated CD8 cell count is associated with an increased risk of HIV treatment failure for patients who initially achieved an undetectable viral load.

| | |
|---|---|
| Method | FCM |
| TAT | 2 d |
| Sample type | EWB |
| Expected value | 150-1000 cells/mm3 |
| Sample stability | 2 d at 18-22˚C |

# CD Markers

## Summary

### CD1a

CD1a expressed on dendritic cells and cortical thymocytes. CD1a antigen expression has been shown to be useful in differentiating Langerhans cells, powerful antigen presenting cells present in skin and epithelia, from interdigitating cells. CD1a associates with the beta2-microglobulin and is thought to play a role in antigen presentation.

### CD2

The CD2 antigen is an accessory molecule important in mediating the adhesion of activated T cells and thymocytes with antigen-presenting cells and target cells.

### CD3

The CD3 antigen is first detected in early thymocytes and its appearance probably represents one of the earliest signs of commitment to the T cell lineage.

### CD5

CD5 antigen is expressed in thymocytes, peripheral blood lymphocytes, T cell leukemias, lymphomas, activated T cells and on a subset of B cells located primarily in the mantle zones of normal lymph nodes. Antigen expression is also reported in T cell acute lymphocytic leukemias (T-ALL), some B cell chronic lymphocytic leukemias (B-CLL) as well as B and T cell lymphomas.

### CD7

The CD7 molecule is the earliest T cell specific antigen to be expressed in lymphocytes. CD7 antigen is also the only early marker to persist throughout differentiation.

CD7 antigen is reported to be found on the majority of peripheral blood T cells, most natural killer cells and thymocytes.

### CD11b

The CD11b (Mac-1) leukocyte associated protein is expressed on granulocytes, monocytes, macrophages and NK cells.

## CD11c

CD11c antigen marker is expressed by mononocytes and macrophages, neutrophils, myeloid dendritic cells and a small subset of lymphocytes. Monoclonal antibodies to CD11c have proven especially useful in the diagnosis of Hairy Cell Leukaemia, a B cell malignancy. Other haematological malignancies that express CD11c antigen include acute myeloid with a monocytoid component, systemic mastocytosis and lymphomas derived from histiocytes and dendritic cells.

## CD13

CD13 antigen (aminopeptidase N), is a member of the type II integral membrane metalloproteases.

CD13 antigen is a receptor for the coronaviruses which cause respiratory disease in humans and several animal species.

CD13 antigen is reported to be expressed on granulocytes, monocytes and their precursors, most acute myeloid leukemias and a smaller proportion of acute lymphoid leukemias.

Non-hematopoietic cells which express CD13 antigen include epithelial cells, renal proximal tubules, intestinal brush border, endothelial cells, fibroblasts, brain cells, bone marrow, osteoclasts and cells lining the bile canaliculi.

## CD14

CD14 antigen marker is expressed on cells of the myelomonocytic lineage including monocytes, macrophages and Langerhans cells.

Low expression is also reported on neutrophils and on B cells. CD14 antigen is a receptor for bacterial lipopolysaccharide (LPS, endotoxin) and the lipopolysaccharide binding protein (LBP).

## CD15

CD15 antigen marker ( X-hapten), is expressed on circulating human granulocytes, circulating monocytes and is absent from normal lymphocytes.

The CD15 antigen is also expressed on Reed Sternberg cells of Hodgkin's disease and some leukemias.

## CD16

CD16 antigen marker is expressed on natural killer (NK) cells, granulocytes, activated macrophages and a subset of T cells expres-sing alpha-beta or gamma-delta T cell antigen receptors. The CD16 antigen exists both as a glycosyl-phosphatidylinositol (GPI)-anchored protein in polymorphonuclear cells and as a transmembrane protein in NK cells

## CD19

CD19 antigen marker is present on the surface of B lymphocytes and follicular dendritic cells of the hematopoietic system.

CD19 is expressed from the earliest recognizable B cell lineage stage, through development to B cell differentiation but is lost on maturation to plasma cells.

## CD20

The CD20 antigen marker is expressed on normal and malignant human B cells normal B cells from peripheral blood, lymph node, spleen, tonsil, bone marrow, acute leukemias and chronic lymphocytic leukemias and is thought to act as a receptor during B cell activation and differentiation.

## CD22

The CD22 antigen marker (BL-CAM) is expressed early in B cell lymphocyte differentiation and on hairy cell leukemias.

## CD23

The CD23 antigen marker reported to be found on a sub-population of peripheral blood cells, B lymphocytes and on EBV-transformed B lymphoblastoid cell lines.

Expression of CD23 antigen has been reported on monocytes and dendritic cells.

## CD24

CD24 is a glycoprotein expressed at the surface of most B lymphocytes and differentiating neuroblasts.

## CD25

CD25 antigen marker is expressed in most B-cell neoplasms, some acute nonlymphocytic leukemias, neuroblastomas, and tumor infiltrating lymphocytes.

## CD29

CD29 antigen marker is reported to be expressed on most cells including all leukocytes, although only at low levels on granulocytes.

On T cells, CD29 antigen is expressed at higher levels on memory cells than on naive cells. The co-expression of CD4 and CD29 antigens is found in helper/inducer subpopulation of CD4 lymphocytes. CD29 antigen is one of several additional molecules reported to be found on the cell membrane of hepatocytes in cases of cirrhosis, alcoholic hepatitis and hepatitis C.

Reduced expression of CD29 antigen together with the β2 integrin, CD11b, has been reported on peripheral blood lymphocytes from Graves' disease patients.

## CD30

CD30 antigen marker is reported to be expressed on the surface of multinucleated Reed Sternberg cells, mononuclear Hodgkin's cells and in the majority of anaplastic large cell lymphomas.

The CD30 antigen is expressed in non-Hodgkin's lymphoma and virally transformed cells, eg EBV-transformed B cells.

Most T cell lymphomas are reported not to express CD30 antigen, with the exception of some pleomorphic T cell lymphomas.

## CD33

CD33 antigen marker is reported to appear on myelomonocytic precursor cells after antigen expression. It then continues to be expressed on both the myeloid and monocyte lineages.

It has been reported that expression of CD33 is restricted to monocytes, premyelocytes, myeloid blasts, some acute undifferentiated leukemias and acute lymphoblastic leukemias. The expression of CD33 antigen has been demonstrated to be an important marker for distinguishing myeloid from the lymphoid leukemias.

The CD34 antigen marker is expressed on human lymphoid, myeloid hemopoietic progenitor cells and vascular endothelium.

## CD35

The CD35 antigen marker ( CR1 or C3b/C4b R) mediates phagocytosis by neutrophils and monocytes of particles coated with C3b or C4b.

CD35 antigen is reported to be found on erythrocytes, B cells, a subset of T cells, monocytes, macrophages cultured in vitro, neutrophils, eosinophils, glomerular podocytes and follicular dendritic cells. Decreased levels of CD35 antigen has been reported on B cells in patients with HIV infection.

## CD38

The CD38 antigen marker is found on immature cells of the B and T cell lineages but not on most mature resting peripheral lymphocytes. It is also present on thymocytes, pre-B cells, germinal center B cells, mitogen-activated T cells, Ig-secreting plasma cells, monocytes, NK cells, erythroid and myeloid progenitors in the bone marrow and brain cells.

CD38 antigen has also been reported in neurofibrillary tangles, the pathological indicator of Alzheimer's disease that occurs in the neuronal perikarya and proximal dendrites.

## CD40

The CD40 antigen marker is a member of the tumor necrosis factor/nerve growth factor superfamily and shows a significant homology to the Hodgkin's disease-associated antigen, CD30.

The CD40 antigen is reported to be found on mature B cells except for plasma cells, most B cell leukemias and lymphomas, interdigitating reticulum cells, follicular dendritic cells and Reed Sternberg cells.

Outside the immune system, CD40 antigen is reported to be expressed on some epithelial cells of certain carcinomas and in malignant melanomas.

## CD43

The CD43 antigen is expressed on the membrane and in the cytoplasm of T cells and cells of myeloid lineage. Cells expressing the CD43 antigen are reported to include normal and neoplastic T cells. A small proportion of B cell chronic leukemias and centrocytic lymphomas are also reported to express CD43 antigen.

## CD44

The CD44 antigen marker (H-CAM) is reported to be expressed on T cells, B cells, monocytes, granulocytes, erythrocytes and weakly on platelets. Other CD44 antigen positive cell types are reported to include epithelial cells, glial cells, fibroblasts and myocytes.

Increased expression of CD44 antigen is found on some carcinomas and it has been reported that transition of tumor cell lines from non-metastatic to metastatic may be associated with changes in the expression of CD44 antigen variants.

## CD57

The CD57 antigen marker (HNK-1) is found on a subset of mononuclear cells with natural killer activity and on neuroectodermal cells expressing myelin-associated glycoprotein. Many cells which co-express CD57 and CD8 proteins are a subset of suppressor/cytotoxic T cells. These cells play a role in the rejection of grafts in acute graft versus host disease.

| | |
|---|---|
| Method | FCM |
| TAT | 2 d |
| Sample type | EWB |
| Expected value | Refer to Report |
| Sample stability | 1 d at 18-22˚C |

# Ceruloplasmin

## Summary

Lower ceruloplasmin synthesis, occurs as a consequence of missing Cu2+ incorporation into the molecule due to defective metallothionine this results in pathological deposits of copper in the liver (with accompanying development of cirrhosis), brain (with neurological symptoms), cornea (Kayser-Fleischer ring), and kidneys (hematuria, proteinuria, aminoaciduria).

The rare Menke's syndrome involves a genetically caused copper absorption disorder with concomitant lowering of the ceruloplasmin level. Protein loss syndromes and liver cell failures are the most important causes of acquired ceruloplasmin depressions. As ceruloplasmin is a sensitive reactant to the acute phase, increases occur during acute and chronic inflammatory processes. Great increases can lead to a green-blue coloration of the sera.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP | |
| Expected value | 20-60 mg/dL | |
| | 0-5 d: 5-40 mg/dL | |
| Conversion factors | mg/dL x 0.01 = g/L | |
| | mg/dL x 0.0746 = µmol/L | |
| | µmol/L x 13.40 = mg/dL | |
| Sample stability | 3 d | at 2-8 °C |
| | 1 mth | at -20°C |

# Chlamydia pneumonia ( IgA, IgG, IgM)

## Summary

Chlamydia belong to small bacteria, they grow obligatory intracellular, their caused diseases are categorized as Sexual Transmitted Diseases (STDs). Transmission also occurs through animals. A chronic process with slight symptomatology is typical for this disease; therefore diagnoses are provided too late or the infection is not detected at all. Sexually transmitted chlamydia are of importance as they represent the most frequent venereal disease. Infection leads to an antibody reaction after 6-8 weeks.

This type of chlamydia causes mostly mild respiratory infects such as bronchitidae, newborn pneumonia or atypical pneumonia. Associations with other diseases were described: asthma, chronic obstructive lung diseases, erythema nodosum, sarcoidosis, myocarditis, CHD, heart attack.

The detection of specific IgA antibodies indicates current infection. IgM is used as an indicator of acute/current infection while the detection of specific IgG antibodies indicates previous exposure.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:10 |
| Sample stability | 2 d at room temperature |
| | 2 wk at 2-8˚C |
| | 1 yr at -20˚C |

# Chlamydia trachomatis ( IgA, IgG, IgM)

## Summary

Chlamydia trachomatis has been called the “silent epidemic” of reproductive age women and is the most common sexually transmitted bacterial infection. The majority of chlamydial infections in women are asymptomatic and if untreated may cause Pelvic Inflammatory Disease (PID) and associated complications such as ectopic pregnancy, infertility, and chronic pelvic pain. C. trachomatis includes the agent of trachoma, inclusion conjunctivitis, lymphogranuloma venerum and uro-genital tract disease.

The detection of specific IgA antibodies indicates current infection. IgM is used as an indicator of acute/current infection while the detection of specific IgG antibodies indicates previous exposure.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:10 |
| Sample stability | 2 d at room temperature |
| | 2 wk at 2-8˚C |
| | 1 yr at -20˚C |

# Chlamydia trachomatis - DNA

## Summary

Chlamydia trachomatis has been called the “silent epidemic” of reproductive age women and is the most common sexually transmitted bacterial infection. The majority of chlamydial infections in women are asymptomatic and, untreated may cause Pelvic Inflammatory Disease (PID) and associated complications such as ectopic pregnancy, infertility, and chronic pelvic pain. C.trachomatis includes the agent of trachoma, inclusion conjunctivitis, lymphogranuloma venerum and uro-genital tract disease.

| | |
|---|---|
| Remarks | Not urinated for at least 2 hr. |
| Method | PCR |
| TAT | 2 d |
| Sample type | US/ENS |
| Expected value | Not detected |
| Sample stability | 2 d at room temperature |

# Cholesterol -High Density Lipoprotein (HDLC)

## Summary

Elevated HDL-cholesterol concentrations are protective against coronary heart disease, while reduced HDL-cholesterol concentrations, particularly in conjunction with elevated Triglycerides increase the cardiovascular risk. Strategies have emerged to increase the level of HDL-cholesterol to treat cardiovascular disease.

| | |
|---|---|
| Method | Colorimetric |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Male (♂): 35-55 mg/dL |
| | Female (♀): 45-65 mg/dL |
| | Good Progress: |
| | Male (♂) :>55 mg/dL |
| | Female (♀): >65 mg/dL |
| Conversion factors | mmol/L x 38.66 = mg/dL |
| | mg/dL x 0.0259 = mmol/L |
| Sample stability | 1 wk at room temperature |
| | 1 wk at 2-8˚C |
| | 3 mth at -20˚C |

# Cholesterol- Low Density Lipoprotein (C-LDL)

## Summary

Elevated LDL concentrations in blood and an increase in their residence time coupled with an increase in the biological modification rate results in the destruction of the endothelial function and a higher LDL-cholesterol uptake in the monocyte/macrophage system as well as by smooth muscle cells in vessel walls. The LDL-cholesterol value is the most powerful clinical predictor among all of the single parameters with respect to coronary atherosclerosis. Therefore, therapies focusing on lipid reduction primarily target the reduction of LDL-cholesterol which is then expressed in an improvement of the endothelial function, prevention of atherosclerosis and reducing its progression as well as preventing plaque rupture.

| | |
|---|---|
| Remarks | Fasting |
| Method | Colorimetric |
| TAT | Dy/AB |
| Sample type | S/EP |
| Expected value | Optimal: <100 mg/dL |
| | Borderline High: 130-159 mg/dL |
| | High: >160 mg/dL |
| Conversion factors | mmol/L x 38.66 = mg/dL |
| | mmol/L x 0.3866 = g/L |
| | mg/dL x 0.0259 = mmol/L |
| Sample stability | 1 wk at room temperature |
| | 1 wk at 2-8˚C |
| | 3mth at - 20˚C |

# Cholesterol,Total

## Summary

Cholesterol is an essential component of cell membranes and lipoproteins as well as a precursor for the synthesis of steroid hormones and bile acids.

LDL bound Cholesterol is transported to the peripheral tissues.

Elimination of the excessive cholesterol to the liver occurs through HDL.

Nutritional supplied cholesterol is absorbed to only 40%. Endogenous cholesterol synthesis is normally prevented by high concentrations of LDL cholesterol in plasma and increased alimentary cholesterol supply. However oral supply of long-chain polysaturated fatty acids (Triglycerides) or increased energy supply in general can lead to an increase of LDL cholesterol in plasma resulting in hypercholesterinemia and, as a consequence, to an elevated cardiovascular risk. Cholesterol elimination occurs mainly via bile. The inherited form of primary hypercholesterinemia leads to accumulation of LDL in plasma due to reduced transport of LDL cholesterol into the cell. Secondary hypercholesterolemia appears in cases of hypothyreoidism or kidney disorders as well as in pancreas or liver disorders.

Total cholesterol gives an overall level of cholesterol in the blood.

| | | |
|---|---|---|
| Remarks | Fasting | |
| Method | Colorimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP | |
| Expected value | Up To 200 mg/dL | |
| Conversion factors | mmol/L x 38.66 = mg/dL | |
| | mmol/L x 0.3866 = g/L | |
| | mg/dL x 0.0259 = mmol/L | |
| Sample stability | 1 wk | at room temperature |
| | 1 wk | at 2-8˚C |
| | 3mth | at -20˚C |

# Cholinesterase (CHE)

## Summary

The biological function of cholinesterase is unknown. Serum cholinesterase serves as an indicator of possible insecticide poisoning. It is measured as an index of liver function. In preoperative screening, cholinesterase is used to detect patients with atypical forms of the enzyme and hence avoid prolonged apnea caused by slow elimination of muscle relaxants. Depressed cholinesterase levels are found in cases of intoxication with organophosphorus compounds and in hepatitis, cirrhosis, myocardial infarction, acute infections and atypical phenotypes of the enzyme.

| | | |
|---|---|---|
| Remarks | Keep sample on ice. | |
| Method | Enzymatic | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Children, Male (♂), (Female (♀)>40 yr): | |
| | 5320-12920 U/L | |
| | Female (♀)Non pregnant & not taking contraceptives (16-39 yr): | |
| | 4260-11250 U/L | |
| | Female (♀) Pregnant or takes contraceptives (18-41 yr): | |
| | 3650-9120 U/L | |
| Conversion factors | U/L x 0.0167 = μkat/L | |
| | μkat/L x 60 = U/L | |
| Sample stability | 6 h | at room temperature |
| | 1 wk | at 2-8°C |
| | 1 yr | at -20°C |

# Chromium

## Summary

Chromium is an essential trace element in physiological concentrations which is necessary in carbohydrate and fat metabolism. Intoxication with chromium occurs through contact with or by intake of chrome trioxide (chromic acid), potassium chromate and potassium dichromate, which are found, for example, in chromium-containing colours.

Oral intake of chromate or dichromate solutions in toxic concentrations leads to mouth affection, green-colored mucous membranes and gastroenteritis. Shock, exsiccosis, anuria and uremia as well as toxic liver damages with severe jaundice can lead to death. Chrome dust inhalation could result in ulcers up to nose septum perforations. Injuries of the skin surface degenerate ulcerously with low curative tendency. Furthermore, it also provokes skin allergies and contact dermatitis.

| | | |
|---|---|---|
| Method | IPMS | |
| TAT | 10 – 14 d | |
| Sample type | S | |
| Expected value | < 1.0 µg/L | |
| Sample stability | 1 wk | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20 ˚C |

# Chromosomal Study

## Summary

Chromosomal analysis can be used to indicate Infertility, recurrent Abortion Deliveries of children with chromosomal abnormalities, abnormal findings in prenatal Chromosomal Analysis, Suspicion of Dysmorphic Syndrome, and Suspicion of gonosomal abnormalities.

Chromosome analysis of amniocytes karyotype is used to determine advanced Maternal age (<37 years or older), suspect prenatal screening, suspect ultrasound, suspicion of fetal malformation, parental chromosomal aberation, previous miscarriage or abortion, childbirth with chromosomal aberation, childbirth with malformations, mutagene exposure before or during pregnancy, suspicion of Neural Tube Defect (NTD), suspicion on embryonal virus infection. Additionally, fetal sex is determined during the course of the investigation, which may have implications when sex chromosome -associated disorders are suspected.

Chorionic Villus (CVS) sampling is a method for first trimester prenatal diagnosis of chromosome disorders used to determine advanced maternal age >34 years or older, previous abortion or miscarriage, parental chromosomal aberation, suspect prenatal screening, suspect ultrasound, childbirth with chromosomal aberations, childbirth with malformation, mutagene exposure before or during pregnancy.

| | | |
|---|---|---|
| Remarks | Use sterile heparin blood (without additives such as gel or plastic pearls). | |
| Method | Karyotyping | |
| TAT | 7 d – 14 d | |
| Sample type | HWB/ AF/ CVS/ D | |
| Expected value | Refer To Report | |
| Sample stability | 2 d | at room temperature |
| | 2 d | at 2-8 ˚C |

# Clostridium Difficile Endotoxin

## Summary

Clostridium difficile is a major cause of antibiotic-associated diarrhea, pseudomembranous colitis1 and an important cause of nosocomial infections in hospitals and nursing homers. Toxin A is enterotoxin which seems to interfere with the cytoskeleton of the intestinal epithelial cells rendering them non functional, while toxin B is a cytotoxin that induces strong cytopathic effects in tissue cultures cell lines.

| | |
|---|---|
| Method | ICT |
| TAT | Dy/A |
| Sample type | ST |
| Expected value | Negative |
| Sample stability | 3 d at 2-8˚C |

# Clozapine

## Summary

Clozpine is a Neuroleptic drug for the treatment of acute and chronic forms of schizophrenic psychoses, toxic concentrations may cause hypotension, cardiac abnormalities, respiratory depression, coma and death. A toxic range is not well established in children.

| | | |
|---|---|---|
| Method | LCMS | |
| TAT | 10 – 14 d | |
| Sample type | S | |
| Expected value | 50-700 μg/L | |
| Sample stability | 5 wk | at room temperature |
| | 2 mth | at 2-8˚C |
| | 2 mth | at -20˚C |

# Cocaine

## Summary

Cocaine (Benzoylmethylecgonine) is a central nervous system stimulant. Cocaine produces a short-lived, intense high which is extremely addictive. The signs and symptoms associated with the abuse of cocaine depend upon the amount used and the duration of use. With infrequent or low dose use a person may experience euphoria, lowered anxiety, talkativeness, decreased appetite, increased sexual arousal, increased alertness, and decreased fatigue. Physiologically there can be increased heart rate and blood pressure.

With increased dose or prolonged abuse (either binge or chronic) an individual may experience a set of secondary effects that can include increased anxiety, irritability, aggressiveness, paranoia and hypersexuality. Physiological effects can include dilated pupils, dry mouth, hippus, increased body temperature and tachycardia. In overdose situations, a person my experience hallucinations, coma or death. Crash symptoms typically follow binge abuse of cocaine. This phase is marked by extreme fatigue, depression, mental exhaustion and prolonged periods of sleep.

The apparent half life for cocaine is short, roughly 1 hour. That means that the time required for cocaine levels to decrease by half in the body is somewhere around 60 minutes. However, with chronic use, cocaine accumulates in the body and resulting a prolonged terminal elimination phase for cocaine and its metabolites. Very low concentrations of cocaine may be detected in urine during the initial few hours after use. In general, cocaine metabolites can be detected in urine 2-4 days after use for sporadic users and up to 12 days after use for chronic users or following a binge.

The Major uses of cocaine testing are to detect the presence of the drug, Suspicion of drug abuse of cocaine products, designer drugs, e.g. crack or similar products, and control during therapy.

| | | |
|---|---|---|
| Method | EIA | |
| TAT | Dy/A | |
| Sample type | US | |
| Expected value | Not detected | |
| Sample stability | 1 wk | at room temperature |
| | 1 mth | at 2-8˚C |
| | 3 yr | at -20˚C |

# Cold Agglutinins

## Summary

The cold agglutinins test is performed to detect the presence of antibodies in blood that are sensitive to temperature changes. The diseases most commonly diagnosed are mycoplasmal pneumonia, mononucleosis, mumps, measles, scarlet fever, some parasitic infections, cirrhosis of the liver, and some types of hemolytic anemia can also cause the formation of cold agglutinins. In addition to these illnesses, some people have a benign condition called chronic cold agglutinin disease, in which exposure to cold causes temporary clumping of red blood cells and consequent numbness in ears, fingers, and toes.

| | |
|---|---|
| Remarks | Serum clotted at 37°c for 30 min. |
| Method | AGGL |
| TAT | 2 d |
| Sample type | S |
| Expected value | Negative |
| Sample stability | 2 d at room temperature |
| | 2 wk at 2-8˚C |
| | 1 yr at -20˚C |

# Complement 3 (C3)

## Summary

A considerable decrease in C3 is found in patients with partial lipodystrophy or membranoproliferative glomerulonephritis when the C3-nephritis factor is present. As an acute phase protein, C3 is produced in an increased extent during inflammatory processes. It is elevated in systemic infections, non-infectious chronic inflammatory conditions (primarily chronic polyarthritis) and physiological states (pregnancy).

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | 90-180 mg/dL | |
| Conversion factors | g/L x 100 = mg/dL | |
| | mg/dL x 0.01 = g/L | |
| Sample stability | 4 d | at room temperature |
| | 8 d | at 2-8 °C |
| | 8 d | at -20 °C |

# Complement 4 (C4)

## Summary

The main use of C4 determinations is in assessing the course of hypocomplement conditions. As an acute phase protein, C4 is produced to an increased extent during inflammatory processes. It is elevated in systemic infections, noninfectious chronic inflammatory conditions (primarily chronic polyarthritis) and physiological states (pregnancy).

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | 10-40 mg/dL | |
| Conversion factors | mg/dL x 0.01 = g/L | |
| | mg/dL x 0.050 = µmol/L | |
| Sample stability | 2 d | at room temperature |
| | 2 d | at 2-8 °C |

# Complement Hemolytic 50 (CH50)

## Summary

Assessment of CH50 is useful in screening for genetic deficiencies and the functional activity in the complement system and in monitoring the progress of patients with immune complex disease.

Reduced activity levels indicate inherited complement deficiency, mixed cryoglobulineamia, immune complex disease, infectious and autoimmune processes, malignancy, trauma, burns, and liver disease. Elevated activity levels are seen in acute inflammatory conditions, leukemia, Hodgkin's disease, and sarcoma.

| | | |
|---|---|---|
| Method | LIA | |
| TAT | 10 – 14 d | |
| Sample type | S | |
| Expected value | >25 Units | |
| Sample stability | 2 h | at room temperature |
| | 2 wk | at 2-8°C |
| | long-term storage | at -70°C |

# Complete Blood Count (CBC)

## Summary

The complete blood count (CBC) is often used as a broad screening test to determine an individual's general health status. Screening for a wide range of conditions and diseases, help diagnose various conditions such as anemia, infection, inflammation, bleeding disorder or leukemia, monitoring the condition and/or effectiveness of treatment after a diagnosis and monitoring treatment that is known to affect blood cells, such as chemotherapy or radiation therapy.

| | | |
|---|---|---|
| Method | HPLC/ FCM | |
| TAT | Dy/AB | |
| Sample type | EWB | |
| Sample stability | 8 h | at room temperature |
| | 3 d | at 2-8 °C |

Expected value

| White Blood Count (WBC)(Leukocytes) | Age | Male ($X10^3$/uL) | Female ($X10^3$/uL) |
|---|---|---|---|
| Low levels associated with risk of infection; high levels indicates possible infection. | 0 d- 1 d | 9.4 - 34.0 | 9.4 - 34 |
| | 1d - 4 mth | 5.0- 19.0 | 5.0- 19.0 |
| | 4 mth - 4 yr | 6.0 - 17.0 | 6.0 - 17.0 |
| | 4 yr- 6 yr | 5.5 - 15.5 | 5.5 - 15.5 |
| | 6 yr - 8 yr | 5.0 - 14.5 | 5.0 - 14.5 |
| | 8 yr - 16 yr | 4.5 - 13.0 | 4.5 - 13.0 |
| | 16 yr+ | 4.0 - 11.0 | 4.0 - 11.0 |
| Hemoglobin (Hgb) | Age | Male (g/dL) | Female (g/dL) |
| Deficiency of iron and therefore of hemoglobin leads to anemia and decreased ability to carry oxygen to body tissues | 0 d- 5 mth | 9.0 - 12.8 | 9.0 - 12.8 |
| | 5 mth- 3 yr | 10.1 - 13.0 | 10.1 - 13.0 |
| | 3 yr- 10 yr | 10.7 - 15.0 | 10.7 - 15.0 |
| | 10 yr + | 13.0 - 17.5 | 11.5 - 16.0 |

| Hematocrit (Hct) | Age | Male (%) | Female (%) |
|---|---|---|---|
| Percent of whole blood that is comprised of red blood cells; measure of both the number and size of red blood cells | 0 yr + | 40 - 52 | 35 - 47 |
| **Mean Corpuscular Volume (MCV)** | **Age** | **Male (fL)** | **Female (fL)** |
| Measures the size of red blood cells. Larger or smaller than normal red blood cells may indicate anemia | 0 yr + | 80 - 96 | 80 - 96 |
| **Mean Corpuscular Hemoglobin (MCH)** | **Age** | **Male (pg)** | **Female (pg)** |
| Estimate of the amount of hemoglobin in the average red cell. Low levels indicate anemia | 0 yr + | 27 - 33 | 27 - 33 |
| **Mean Corpuscular Hemoglobin Concentration (MCHC)** | **Age** | **Male (g/dL)** | **Female (g/dL)** |
| Estimate of the concentration of hemoglobin in the average red cell. Low levels indicate anemia | 0 yr + | 30 - 36 | 30 - 36 |
| **Platelets (Thrombocytes)** | **Age** | **Male ($X10^3$/uL)** | **Female ($X10^3$/uL)** |
| Helps blood clotting in order to stop bleeding from injury. Decreased platelet count is called thrombocytopenia. | 0 yr + | 145 - 450 | 145 - 450 |
| **Red Blood Count (RBC)** | **Age** | **Male($X10^6$/uL)** | **Female($X10^6$/uL)** |
| (Erythrocytes) Low levels cause anemia and are associated fatigue | 0 yr + | 4.5 - 6 | 4.0 - 5.3 |
| **Neutrophils** | **Age** | **Male (%)** | **Female (%)** |
| These cells provide primary defense against bacterial infection | 0 yr + | 40 - 65 | 40 - 65 |
| **Lymphocytes** | **Age** | **Male (%)** | **Female (%)** |
| Many kinds of immune cells; protect against pathogens (bacteria, virus, fungi) and cancer | 0 yr + | 20 - 45 | 20 - 45 |

| Monocytes | Age | Male (%) | Female (%) |
|---|---|---|---|
| Germ eating cells. A low number can increase risk of getting sick from an infection, particularly of bacteria type. | 0 yr + | 2.0 - 10 | 2.0 - 10 |
| Eosinophils | Age | Male (%) | Female (%) |
| A type of phagocyte that produces the antiinflammatory protein histamine. A high number indicates allergies or parasitic | 0 yr + | 1.0 - 5.0 | 1.0 - 5.0 |
| Basophils | Age | Male (%) | Female (%) |
| Control inflammation and damage of tissues in the body. | 0 yr + | 0.2 - 1.0 | 0.2 - 1.0 |
| Neutrophils Counts | Age | Male ($X10^3$/uL) | Female ($X10^3$/uL) |
| | 0 yr + | 2.0 - 4.8 | 2.0 - 4.8 |
| Lymphocytes Counts | Age | Male ($X10^3$/uL) | Female ($X10^3$/uL) |
| | 0 yr + | 1.2 - 3 | 1.2 - 3 |
| Monocytes Counts | Age | Male ($X10^3$/uL) | Female ($X10^3$/uL) |
| | 0 yr + | 0.2 - 0.8 | 0.2 - 0.8 |
| Eosinophils Counts | Age | Male ($X10^3$/uL) | Female ($X10^3$/uL) |
| | 0 yr + | 0 - 0.4 | 0 - 0.4 |
| Basophils Counts | Age | Male ($X10^3$/uL) | Female ($X10^3$/uL) |
| | 0 yr + | 0 - 0.1 | 0 - 0.1 |
| Red Cell Distribution Width (RDW) | Age | Male (%) | Female (%) |
| | 0 yr + | 11.5 - 15.5 | 11.5 - 15.5 |
| Platelet Distrubution Width (PDW) | Age | Male (%) | Female (%) |
| | 0 yr + | 15.5 - 17.1 | 15.5 - 17.1 |
| Plateletcrit (PCT) | Age | Male (%) | Female (%) |
| | 0 yr + | 0.2 -0.4 | 0.2 -0.4 |

# Connecting peptide (C-Peptide)

## Summary

Measurements of C-peptide, insulin and glucose are used as an aid in the differential diagnosis of hypoglycemia (factitious hypoglycemia and hypoglycemia caused by hyperinsulinism). Measurements of C-peptide may, therefore, be an aid in the assessment of a residual β-cell function in the early stages of type-1 diabetes mellitus and for the differential diagnosis of Latent Autoimmune Diabetes of Adults (LADA) and type-2 diabetes.

A correlation was found between higher C-peptide levels and increasing hyperlipoproteinaemia and hypertension.

C-peptide concentrations are elevated in renal disease and may result from increased β-cell activity observed in hyperinsulinism, from renal insufficiency and obesity. Decreased levels are observed in: starvation, factitious hypoglycemia, hypoinsulinism (NIDDM, IDDM), Addison's disease and after radical pancreatectomy.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/ HP/ EP | |
| Expected value | 0.78 – 1.89 ng/mL | |
| Conversion factors | ng/mL (µg/L) x 0.33333 = nmol/L | |
| | ng/mL x 333.33 = pmol/L | |
| | nmol/L x 3.0 = ng/mL | |
| | pmol/L x 0.003 = ng/mL | |
| Sample stability | 4 h | at room temperature |
| | 1 d | at 2-8 °C |
| | 1 mth | at -20 °C |

# Coomb's direct

## Summary

The direct coomb's test (also known as the Direct Antiglobulin Test "DAT") is useful in the diagnosis of haemolytic disease of newborn, autoimmune haemolytic anaemia, and transfusion reaction.

| | |
|---|---|
| Method | AGGL |
| TAT | Dy/AB |
| Sample type | EWB |
| Expected value | Negative |
| Sample stability | 2 d at 2-8 ˚C |

# Coomb's indirect

## Summary

The indirect Coombs test (also known as the Indirect Antiglobulin Test "IAT") is used in the detection of incompatibility in cross matching tests, and in the diagnosis of hemolytic disease of the newborn. The IAT can also be used for compatibility testing, antibody identification, RBC phenotyping, and titration studies.

| | |
|---|---|
| Method | AGGL |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Negative |
| Sample stability | 2 d at 2-8 ˚C |

# Copper

## Summary

Copper effects are involved in the formation of connective tissue, in the free function of Central Nervous System (CNS) and in hematopoiesis. Other causes for elevated copper level are liver cirrhosis, tumors, infectious diseases, cholestasis, pregnancy, thyroid disturbances, nephritic syndrome, and rheumatic arthritis. Copper levels in urine reflect the free copper which is unlike to ceruloplasmin as a transporter protein. Increased copper level in urine is not sufficient for M. Wilson diagnosis.

| | | | |
|---|---|---|---|
| Method | Colorimetric | | |
| TAT | 24U: Dy/A | | |
| | S: Dy/AB | | |
| Sample type | S/24U | | |
| Expected value | 24: | 15-70 µg/d | |
| | S : Age | Male ( µg/dL) | Female (µg/dL) |
| | Newborn | 12-67 | 12-67 |
| | <10 yr | 30-150 | 30-150 |
| | + 10 yr | 70-140 | 80-155 |
| Conversion factors | µg/dL x 0.157 = µmol/L | | |
| | µmol/L x 6.354 =µg/dL | | |
| Sample stability in S | 2 wk | at room temperature | |
| | 2 wk | at 2-8˚C | |
| | > 1 yr | at -20˚C | |
| Sample stability in 24U | 3 d | at room temperature | |
| | 1 wk | at 2-8˚C | |
| | 1 yr | at -20˚C | |

# Coproporphyrins (Quantitative)

## Summary

Increased levels of coproporphyrins can indicate congenital erythropoietic porphyria or sideroblastic anaemia. Porphyria is a pathological state characterised by abnormalities of porphyrin metabolism and results in the excretion of large quantities of porphyrins in the urine and in extreme sensitivity to light. A large number of factors are capable of increasing porphyrin excretion, owing to different and multiple causes and etiologies: the main site of the chronic hepatic porphyria disease process concentrates on the liver, a functional and morphologic liver injury is almost regularly associated with this chronic porphyria, the toxic form due to occupational and environmental exposure takes mainly a subclinical course. Hepatic factors includes disturbance in coproporphyrinogen metabolism, which results from inhibition of coproporphyrinogen oxidase as well as from the rapid loss from, and diminished utilization of coproporphyrinogen in the hepatocytes, which may also explain why coproporphyrin, its autoxidation product, predominates physiologically in the urine; decreased biliary excretion of coproporphyrin leading to a compensatory urinary excretion, so that the coproporphyrin ring isomer ratio (1:3) becomes a sensitive index for impaired liver function and intrahepatic cholestasis; and disturbed activity of hepatic uroporphyrinogen decarboxylase. In itself, secondary coproporphyrinuria is not associated with porphyria symptoms of a hepatologic-gastroenterologic, neurologic, or dermatologic order, even though coproporphyrinuria can occur with such symptoms.

| | | |
|---|---|---|
| Remarks | Protect sample from light. | |
| Method | HPLC | |
| TAT | 2 d | |
| Sample type | 24U | |
| Expected value | Male (♂): 10-109 μg/d | |
| | Female (♀): 3-56 μg/d | |
| Sample stability | 4 d | at room temperature |
| | 1 wk | at 2-8˚C |
| | 1 mth | at -20˚C |

# Cordirone (Amidirone)

## Summary

Amidirone is an antiarrhythmic agent drug used for various types of cardiac dysrhythmias, both ventricular and atrial. Toxic concentrations may exacerbate arrhythmias, cause liver and lung toxicity, and thyroid dysfunction. The concentration of desmethylamiodarone, an active major metabolite, is also reported, but no therapeutic range is established. At steady-state, the metabolite concentration is similar to the amiodarone concentration.

| | |
|---|---|
| Remarks | 2 ml of the sample. |
| Method | HPLC |
| TAT | 4 d |
| Sample type | S |
| Expected value | Refer To Report |
| Sample stability | 6 wk at 2-8 ˚C |
| | 6 wk at -20˚C |

# Cortisol, Free

## Summary

Cortisol is a steroid hormone released from the adrenal cortex in response to an hormone called ACTH (produced by the pituitary gland), it is involved in the response to stress; it increases blood pressure, blood sugar levels, may cause infertility in women, and suppresses the immune system. Cortisol acts through specific intracellular receptors and has effects in numerous physiologic systems, including immune function, glucose-counter regulation, vascular tone, substrate utilization and bone metabolism.

Cortisol is excreted primarily in urine in an unbound (free) form. This test measures the amount of cortisol in urine. It is used to find and evaluate metabolic problems, such as Cushing's syndrome, may also be used to evaluate certain disorders that are related to how the hypothalamus, pituitary, and adrenal glands work together.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | 24U | |
| Expected value | 9-180 μg/d | |
| Sample stability | 1 wk | at 2-8 ˚C |
| | 1 wk | at – 20°C |

# Cortisol, Total

## Summary

The most important physiological effects of cortisol are the increase of blood glucose levels and its anti-inflammatory and immunosuppressive action. The cortisol status of a patient is used to diagnose the function or malfunction of the adrenal gland, the pituitary, and the hypothalamus. Thereby, cortisol serum concentrations are used for monitoring several diseases with an overproduction (e.g. Cushing's syndrome) or underproduction (e.g. Addison's disease) of cortisol and for the monitoring of several therapeutic approaches (e.g. dexamethasone suppression therapy in Cushing's syndrome and hormone replacement therapy in Addison's disease).

| | |
|---|---|
| Remarks | State sampling time. |
| Method | Blood Cortisol: ECLIA |
| | Urinary Cortisol: ELISA |
| TAT | Dy/AB |
| Sample type | S/HP/EP/CP |
| | |
| Expected value | 7-10 a.m.: 171-536 nmol/L |
| | 4-8 p.m.: 64-340 nmol/L |
| Conversion factors | nmol/L x 0.03625 = µg/dL |
| | nmol/L x 0.3625 = µg/L |
| | µg/dL x 27.586 = nmol/L |
| | µg/L x 2.7586 = nmol/L |
| Sample stability | 1 wk at room temperature |
| | 1 wk at 2-8 ˚C |
| | 3 mth at - 20 ˚C |

# Creatine Kinase (CK)

## Summary

Creatine kinase (CK), also known as creatine phosphokinase (CPK) or phospho-creatine kinase is one of the most useful enzymes for diagnostic purposes in acute myocardial infarction. CK is assayed in blood tests as a marker of myocardial infarction (heart attack), rhabdomyolysis (severe muscle breakdown), and muscular dystrophy and in acute renal failure. Drugs that can increase CK measurements: amphotericin B, ampicillin, anesthetics, anticoagulants, aspirin, clofibrate, dexamethasone, furosemide, morphine, alcohol, cocaine.

| | | |
|---|---|---|
| Remarks | Neither homlezyed nor lipemec sample. | |
| Method | Enzymatic | |
| TAT | Dy/AB | |
| Sample type | S/HP | |
| Normal value | Male (♂): 0.7-1.2 mg/dL | |
| | Female (♀): 0.5-0.9 mg/dL | |
| Conversion factors | U/L x 0.0167 = μkat/L | |
| Sample stability | 2d | at room temprature |
| | 1 wk | at 2-8°C |
| | 1 wk | at 20°C |

# Coxsackie A, B Abs (IgA,IgG,IgM)

## Summary

Coxsackie viruses are a highly contagious enterovirus separated into two groups, A and B.

Type A viruses cause herpangina (painful blisters in the mouth, throat, hands, feet, or in all these areas).Type A also cause conjunctivitis (inflammation of the eyelids and white area of the eye).

Type B viruses cause epidemic pleurodynia (fever, lung, and abdominal pain with headache that lasts about two to 12 days and resolves). Pleurodynia is also termed Bornholm disease.

The detection of IgG antibodies indicates past or recent exposure to the virus whereas the detection of IgM/IgA specific antibodies is suggestive of current infection with the virus.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:10 |
| Sample stability | 1 wk at 2-8°C |

## CK-MM/CK-MB/CK-BB/ Macro-CK Isoenzymes

### Summary

Elevated CKMM (muscle type) can usually be detected in intramuscular injection, myopathia, muscle dystrophia, poly-/dermatomyositis, infectious myositis, severe burn, rhabdomyolysis (Crush syndrome), constant physical stress.

The isoenzyme MB muscle-brain (hybrid type) of creatine kinase is relatively cardiac muscle specific. CK-MB levels rise 3-6 hours after a heart attack. If there is no further damage to the heart muscle, the peak level is found 12-24 hours and returns to normal 12-48 hours after cell death. CK-MB levels do usually not rise with chest pain caused by angina, pulmonary embolism (blood clot in the lung), or congestive heart failure.

The quantity of CK-BB in the tissue is usually small. The small quantity, coupled with its relatively short half-life (1-5 hours), results inCK-BB (brain type) activities that are generally low and transient and not usually measurable when tissue damage occurs. Highest concentrations are found in the central nervous system, the gastrointestinal tract, and the uterus during pregnancy.

Macro-CK are CK-variants with a high molecule-mass resulting in falsely-high CK-concentration. Macro-CK-type 1 results from a bond between CK-BB and specific antibodies, however, Macro-CK is without clinical significance. Macro-CK type 2 is a mitochondrial CK in oligomeric form which is often associated with severe disorders, e.g. tumors, liver-cirrhosis, Lyell´s-syndrome

| | | |
|---|---|---|
| Method | ELPH | |
| TAT | 10-14 d | |
| Sample type | S | |
| Expected value | Components | Reference Interval |
| | CK-MM | 96-100% |
| | CK-MB | < 3.0% |
| | CK-BB | 0% |
| | CK-Macro Type I | 0% |
| | CK-Macro Type II | 0% |
| Sample stability | 8 d at 2-8 ˚C | |
| | 1 mth at - 20 ˚C | |

# Creatine Kinase-Muscle Brain (CKMB)

## Summary

CK is one of the most useful enzymes for diagnostic purposes in acute myocardial infarction. It is found in skeletal muscles, heart, brain and lungs. Increased CKMB can usually be detected in heart attack patients about 3-4 hours after onset of chest pain. If the value of CKMB is elevated and the ratio of CKMB to total CK (relative index) is more than 2.5-3 (> 6%), it is likely that the heart was damaged and the relative index below this value suggests that skeletal muscles were damaged.

| | | |
|---|---|---|
| Method | Enzymatic | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | <25 U/L | |
| Conversion factors | U/L x 0.0167 = µkat/L | |
| Sample Stability in S | 8 h | at room temperature |
| | 8 d | at 2-8 °C |
| | 1 mth | at -20˚C |
| Sample Stability in HP | 8 h | at room temperature |
| | 5 d | at 2-8 °C |
| | 8 d | at -20˚C |

# Creatinine

## Summary

The assay of creatinine in serum or plasma is the most commonly used test to assess renal function. In addition to the diagnosis and treatment of renal disease, the monitoring of renal dialysis, creatinine measurements are used for the calculation of the fractional excretion of other urine analytes (e. g., albumin, and α-amylase). For this test a precisely timed urine collection (usually 24 hours) and a blood sample are needed.

| | | | | |
|---|---|---|---|---|
| Remarks | S/HP/EP: Not lipemic sample. | | | |
| Method | Jaffe rate blanked | | | |
| TAT | Dy/AB | | | |
| Sample type | S/HP/EP/U24/US | | | |
| Expected value | Gender | S/HP/EP (mg/dL) | 24U (mg/kg/d) | US (mg/dL) |
| | Male (♂) | 0.7-1.2 | 14-26 | 39-259 |
| | Female (♀) | 0.5-0.9 | 11-20 | 28-217 |
| Conversion factors | mmol/L × 11.3 = mg/dL | | | |
| Sample stability in S/HP/EP | 1 wk | at room temperature | | |
| | 1 wk | at 2-8 °C | | |
| | 3 mth | at -20 °C | | |
| Sample stability in 24U/US (without preservative) | 2 d | at room temperature | | |
| | 6 d | at 2-8 °C | | |
| | 6 mth | at -20 °C | | |
| Sample stability in 24U/US (with preservatives) | 3 d | at room temperature | | |
| | 8 d | at 2-8 °C | | |
| | 3 wk | at -20°C | | |

# CSF albumin

## Summary

CSF albumin is a measurement used to determine the levels of albumin in cerebrospinal fluid. It can be useful in distinguishing among causes of Meningitis. It is more likely to be elevated in bacterial meningitis than in viral meningitis.

| | |
|---|---|
| Method | Turbidmetric |
| TAT | Dy/A |
| Sample type | CSF |
| Expected value | <4 yrs: <45 mg/dL |
| | >4 yrs: 10-30 mg/dL |
| Sample stability | 8 h at room temperature |
| | 8 d at 2-8˚C |
| | 1 yr at -20˚C |

# CSF analysis

## Summary

Cerebrospinal Fluid (CSF) analysis may be used to help diagnose a wide variety of diseases and conditions affecting the central nervous system.

- Infectious diseases such as meningitis and encephalitis - analyzing is used to determine if infection is caused by bacteria, viruses or, less commonly, by tuberculosis, fungi or parasites, and to distinguish it from other conditions; may also be used to detect infections of or near the spinal cord or to investigate a fever of unknown origin.
- Bleeding (hemorrhaging) within the brain or skull.
- Diseases that cause inflammation or other immune responses such as the production of antibodies - these may include autoimmune disorders, such as Guillain-Barré syndrome or sarcoidosis, or diseases that cause the destruction of myelin, such as multiple sclerosis.
- Tumors located within the central nervous system (primary) or that spread to the central nervous system (metastatic cancer).

| | |
|---|---|
| Method | HPLC/ FCM/ Microscopy |
| TAT | Dy/A |
| Sample type | CSF |
| Expected value | Refer To Report |
| Sample stability | 8 h at room temperature |
| | 1 wk at 2-8˚C |
| | 1 yr at -20˚C |

# CSF IgG Index

## Summary

The determination of CSF IgG Index used as an aid in the diagnosis of multiple sclerosis. The elevation of IgG level in the Cerebrospinal Fluid (CSF) of patients with inflammatory diseases of the central nervous system (Multiple Sclerosis [MS], neurosyphilis, acute inflammatory polyradiculoneuropathy, subacute sclerosing panencephalitis) is due to local Central Nervous System (CNS) synthesis of IgG.

| | | |
|---|---|---|
| Method | Turbidmetric | |
| TAT | Dy/AB | |
| Sample type | CSF+S | |
| Expected value | 0.3-0.7 Index | |
| Sample stability | 8 h | at room temperature |
| | 8 d | at 2-8˚C |
| | 1 yr | at -20˚C |

# CSF Index (IgG/Albumin)

## Summary

The CSF index is the CSF IgG to CSF albumin ratio compared to the serum IgG to serum albumin ratio. The CSF index is, therefore, an indicator of the relative amount of CSF IgG compared to serum. Any increase in the index is a reflection of IgG production in the Central Nervous System (CNS). The IgG synthesis rate is a mathematical manipulation of the CSF index data and can also be used as a marker for CNS inflammatory diseases.

| | | |
|---|---|---|
| Method | Turbidmetric | |
| TAT | Dy/AB | |
| Sample type | CSF+S | |
| Expected value | <0.27 Index | |
| Sample stability | 8 h | at room temperature |
| | 8 d | at 2-8˚C |
| | 1 yr | at -20˚C |

# CSF Latex

## Summary

The Bacterial Antigen Test (BAT) screens Cerebrospinal Fluid (CSF) or other body fluids for antigens of classic bacterial meningitis pathogens (i.e., Streptococcus pneumoniae, Haemophilus influenzae type b [Hib], group B Streptococcus species, Neisseria meningitidis, and Escherichia coli K1). Meningitis has a wide variety of potential causes, either infectious or non infectious. If bacterial meningitis is not treated promptly and effectively, the disease is likely to be fetal. Early identification of the infecting agent can be considerable value in providing the patient with appropriate and adequate chemotherapy.

| | | |
|---|---|---|
| Remark | 2 mL of the sample. | |
| Method | Latex Agglutination | |
| TAT | Dy/A | |
| Sample type | CSF | |
| Expected value | Negative | |
| Sample stability | 1 d | at 2-8˚C |
| | Long term storage | at -20˚C |

# CSF Oligoclonal Banding

## Summary

The presence of oligoclonal bands in cerebrospinal fluid combined with their absence in blood serum often indicates that immunoglobulins are produced in central nervous system.

Oligoclonal bands are an important indicator in the diagnosis of multiple sclerosis. The presence of one band (a monoclonal band) may be considered serious, such as lymphoproliferative disease, or may simply be normal. More bands may reflect the presence of a disease. The bands tend to disappear from the cerebrospinal fluid as a person recovers from the neurological disease.

| | | |
|---|---|---|
| Method | ELPH | |
| TAT | 3 d | |
| Sample type | CSF+S | |
| Expected value | No Oligoclonal Band Detected | |
| Sample stability | 8 h | at room temperature |
| | 8 d | at 2-8˚C |
| | 1 yr | at -20˚C |

# Cyclic Adenosin-Monophosphate (CAMP)

## Summary

The elimination of cyclic AMP (generated from plasma and kidneys) is considered as total cyclic AMP. The quantity of renal AMP depends on the number of tubuli as well as on parathyroid hormone concentration.

Increased level of parathyroid hormone stimulates the adenylatcyclase in the renal cortex; which leads to an increased production of CAMP.

| | | |
|---|---|---|
| Remark | EP frozen sample. | |
| Method | RIA | |
| TAT | 10 – 14 d | |
| Sample type | EP/ 24U/ US | |
| Expected value | Cyclic AMP, EP | 17.0 - 36.0 nmol/L |
| | Cyclic AMP, Urine | 0.5 - 10.0 nmol/mL |
| Sample stability In EP | 1 h | at 2-8 °C |
| | 3 mth | at -20 °C |
| Sample stability In | 24u/us | 2h at room temperature |
| | 1 d | at 2-8 °C |
| | 3 mth | at -20 °C |

# CYFRA 21-1

## Summary

The main indication for CYFRA 21-1 is monitoring the course of non-Small Cell Lung Cancer (NSCLC) and course-monitoring in myoinvasive cancer of the bladder. Good specificity is shown by CYFRA 21-1 in relevance to benign lung diseases (pneumonia, sarcoidosis, tuberculosis, chronic bronchitis, bronchial asthma and emphysema). Slightly elevated values (up to 10 ng/mL) are rarely found in marked benign liver diseases and renal failure.

An unclear circular focus in the lung together with CYFRA 21-1 values > 30 ng/mL indicate with high probability the existence of primary bronchial carcinoma.

High CYFRA 21-1 serum levels indicate an advanced tumor stage and a poor prognosis. Successful therapy is documented by a rapid fall in the CYFRA 21-1 serum level into the normal range.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Up To: 3.3 ng/mL | |
| Sample stability | 1 mth | at 2-8 °C |
| | 6 mth | at -20 °C. |

# Cystic Fibrosis (CF)

## Summary

Cystic Fibrosis ( CF ) is an autosomal recessive genetic disorder that affects most critically the lungs, and also the pancreas, liver, and intestine. Difficulty breathing is the most serious symptom and results from frequent lung infections that are treated with antibiotics and other medications. Other symptoms, including sinus infections, poor growth, and infertility affect other parts of the body.

CF is caused by a mutation in the gene for the protein Cystic Fibrosis Transmembrane Conductance Regulator (CFTR).

| | | |
|---|---|---|
| Method | PCR | |
| TAT | 3 d | |
| Sample type | EWB/HWB | |
| Expected value | Normal Genotype | |
| Sample stability | 3 d | at room temperature |
| | 1 wk | at 2-8˚C |

# Cytomegalovirus IgG Abs (CMV- IgG)

## Summary

CMV infections are usually mild and asymptomatic. However, primary maternal CMV infection during pregnancy carries a high risk of intrauterine transmission which may result in severe fetal damage, including growth and mental retardation, jaundice and Central Nervous System (CNS) abnormalities. Transmission of infection requires intimate contact with infected excretions such as saliva, urine, cervical and vaginal excretions, semen, breast milk and blood. The determination of IgG antibodies will be found in old stages of manifested autoimmune disorders.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Negative: <1.0 | |
| Sample stability | 1 wk | at room temperature |
| | 1 mth | at 2-8 °C |
| | 6 mth | at -20 °C |

# Cytomegalovirus IgM Abs (CMV- IgM)

## Summary

CMV infections are usually mild and asymptomatic. However, primary maternal CMV infection during pregnancy carries a high risk of intrauterine transmission which may result in severe fetal damage, including growth and mental retardation, jaundice and Central Nervous System (CNS) abnormalities. Transmission of infection requires intimate contact with infected excretions such as saliva, urine, cervical and vaginal excretions, semen, breast milk and blood. The determination of IgM antibodies is a valuable indicator in the diagnosis of recent infection.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:10 |
| Sample stability | 1 wk at room temperature |
| | 1 mth at 2-8 °C |
| | 6 mth at -20 °C |

# D-Dimer

## Summary

D-Dimer is a very sensitive marker fibrin degradation product for the activation of coagulation. Monitoring the fibrin-specific degradation products can be used to:

• confirm or refute a tentative diagnosis.

• estimate the potential risk for patients with existing Disseminated Intravascular Coagulation (DIC).

• monitor an initiated therapy.

Elevated concentration of D-Dimer indicates increased coagulatory and fibrinolytic activity; in general a validated d-dimer test is useful in ruling out Deep Venous Thrombosis (DVT) and Pulmonary Embolism (PE). Apart from DVT, PE, and DIC, D-Dimer may reflect other causes associated with fibrin formation such as trauma, pregnancy complications, malignant disease or vascular abnormalities.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | HWB | |
| Expected value | < 0.5 µg FEU/mL | |
| Conversion factors | µg FEU/mL = mg FEU/L | |
| | µg FEU/mL x 1000 = ng FEU/mL | |
| Sample stability | 8 h | at room temprature |
| | 4 d | at 2-8 °C |
| | 6 mth | at -20 °C |

# Dengue Abs (IgG, IgM)

## Summary

Dengue fever (breakbone fever) is an infectious tropical disease caused by the dengue virus. Symptoms include fever, headache, muscle and joint pains, and a characteristic skin that is similar to measles. In a small proportion of cases the disease develops into the life-threatening dengue hemorrhagic fever, resulting in bleeding, low levels of blood platelets and blood plasma leakage, or into dengue shock syndrome, where dangerously low blood pressure occurs.

IgG antibody to dengue fever virus detected, which may indicate a current or past infection While IgM antibody to dengue fever virus indicate a current or recent infection. However, low levels of IgM antibodies may occasionally persist for more than 12 months post-infection.

| | | |
|---|---|---|
| Method | EIA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative | |
| Sample stability | 2 d | at room temperature |
| | 2wk | at 2-8 ˚C |
| | 6mth | at - 20 ˚C |

# Digoxin

## Summary

Digoxin is widely prescribed for the treatment of congestive heart failure and various disturbances of cardiac rhythm. Therapeutic use of digoxin improves the strength of myocardial contraction and results in the beneficial effects of increased cardiac output, decreased heart size, decreased venous pressure and decreased blood volume. Therapeutic administration inadvertently, yet frequently, results in toxicity. Most importantly, symptoms of digoxin toxicity often mimic cardiac arrhythmias for which the drug was originally prescribed.

| | | |
|---|---|---|
| Remark | Not hemolyzed nor lipemic sample. | |
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP | |
| Expected value | 0.9-2 ng/mL | |
| Conversion factors | nmol/L x 0.78 = ng/mL | |
| | ng/mL x 1.28 = nmol/L | |
| Sample stability | 2 wk | at room temperature |
| | 2 mth | at 2-8 ˚C |
| | 6 mth | at - 20 ˚C |

# Down Syndrome (DS)

## Summary

Down Syndrome (DS), also known as trisomy 21, is a chromosomal condition caused by the presence of all or part of a third copy of chromosome 21. It is typically associated with a delay in cognitive ability (mental retardation, or MR) and physical growth and a particular set of facial characteristics. A large proportion of individuals with Down syndrome have a severe degree of intellectual disability. Down syndrome is, therefore, a malformation and the frequency increases with increasing age of the mother (<30 yr).

### • Down syndrome Risk Calculation

1st Trimester (PAPP-a, free β-HCG)

The first trimester screen combines the results from three measurements (nuchal translucency, PAPP-a, free β-HCG) with maternal age risk factors. The concentration changes of Biochemical parameters (PAPP-a, free β-HCG) in the blood of the mother during the first trimester provides the increasing risk of having Down syndrome, 13 or 18 trisomy. Increased fluid is found in the Nuchal Translucency (NT) and abnormally high or low hCG , PAPP-A levels are also considered. NT measurement of up to 2 mm is normal about 11 weeks, and up to 2.8 mm by 13+6. As the NT increased, so does the risk of Down Syndrome and other abnormalities. Due to the entered data a calculation is possible only from week of gestation 11+0 and CRL of 45-84 mm.

Time period of the test: week gestation 8- 13.8

### • Down syndrome Triple Test

(AFP, free β-HCG, FE3)

The Triple test measures serum levels of AFP, estriol, and free β-hCG, with a 70% sensitivity and 5% false-positive rate. A high risk (more than 1:250-1/350) should be considered for amniocentesis normal results obtained with these tests point to normal constellation

regarding neural tube defects and trisomies 13,18 and 21.

*Neural tube defects are recognized by the AFP determination.

Time period of the test: week of gestation 14 to 22

• Down syndrome Quadruple Test

The quad marker screen measures serum levels of AFP, estriol, inhibin A and free β-hCG. High maternal serum levels of hCG and inhibin A with low levels of AFP and/or unconjugated estriol in pregnant women has been associated with an increased risk of carrying a Down syndrome fetus.

Time period of the test: week gestation 17 to 22.

• software also calculate the risk of having Edwards syndrome (trisomy 18), Neural Tube Defects like spina bifida (NTD).

| Remarks | |
|---|---|
| Provide DOB, Weight, LMP, Race, No. of embryos, Smoker, Diabetic. | |
| Method | Biochemical screening |
| TAT | 2 d |
| Sample type | S |
| Expected value | Refer To Report |
| Sample stability | 1 d at room temperature |
| | 1 wk at 2- 8˚C |
| | 1 yr at -20˚C |

# Duchenne Muscular Dystrophy (DMD)

## Summary

Duchenne Muscular Dystrophy (DMD) is a recessive X-linked form of muscular dystrophy, which results in muscle degeneration and eventual death. The disorder is caused by a mutation in the dystrophin gene, located on the human X chromosome. The main symptom of DMD, a progressive neuromuscular disorder, is muscle weakness associated with muscle wasting with the voluntary muscles being first affected, especially affecting the muscles of the hips, pelvic area, thighs, shoulders, and calf muscles. Muscle weakness also occurs in the arms, neck, and other areas, but not as early as in the lower half of the body. Calves are often enlarged. Symptoms usually appear before age 6 and may appear as early as infancy.

| | |
|---|---|
| Method | PCR |
| TAT | 4 d |
| Sample type | EWB |
| Expected value | Normal genotype |
| Sample stability | 1 d at room temperature |
| | 5 d at 2-8˚C |

# Entamoeba histolytica Ag

## Summary

Entameoba histolytica, one of the two Entamoeba species with similar morphology that infect humans, causes invasive intestinal and extraintestinal diseases, E.histolytica had clinical symptoms, dysentery-feces with mocus and blood, diarrhea, cramping abdominal pain, tenesmus rectal, flatulence, vomiting and headache. It is necessary to perform a confirmatory test e.g. by ELISA method on the specimens in order to determine whether the patient should be treated or to prevent patients from being given an unnecessary treatment.

| | |
|---|---|
| Method | ELISA |
| TAT | Dy/A |
| Sample type | ST |
| Expected value | Negative |
| Sample stability | 2 d at 2-8˚C |
| | 1 wk at -20˚C |

# Enteric Cytopathic Human Orphan Virus (ECHO Virus)

## Summary

ECHO is a virus found in the gastrointestinal tract and exposure to the virus causes other opportunistic infections and diseases. Echovirus is highly infectious, and its primary target is children. The echovirus is among the leading causes of acute febrile illness in infants and young children, and is the most common cause of aseptic. Infection of an infant with this virus following birth may cause severe systemic diseases, and is associated with high infant mortality rates. The echovirus can mimic symptoms caused by other common bacterial and viral infections. IgG antibody to ECHO virus detected, which may indicate a current or past infection While IgM antibody to the virus indicate a current or recent infection.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:10 |
| Sample stability | 2 d at room temperature |
| | 2wk at 2-8 ˚C |
| | 1 yr at - 20 ˚C |

# Epstein - Barr virus Abs (EBV-IgA,IgG,IgM)

## Summary

The Epstein–Barr Virus EBV (herpes family) is best known as the cause of infectious mononucleosis (glandular fever). It is also associated with particular forms of cancer, such as Hodgkin's lymphoma, Burkitt's lymphoma, nasopharyngeal carcinoma, and conditions associated with Human Immunodeficiency Virus (HIV) such as hairy leukoplakia and central nervous system lymphomas. There is evidence that infection with the virus is associated with a higher risk of certain autoimmune diseases, especially dermatomyositis, systemic lupus erythematosus, rheumatoid arthritis, Sjögren's syndrome, and multiple sclerosis.

Antibodies can be detected against several components of the Epstein-Barr Virus (EBV). These components are the EBV Early Antigen (EA), the Viral Capsid Antigen (VCA), and the Nuclear Antigen (EBNA). These several antigens are different proteins that are produced in the process (stages) of the virus' growth. Antibody to VCA is found both early and late in EBV infection while antibody to EBNA does not usually develop until recovery from first time infection of this virus.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:10 |
| Sample stability | 1 wk at 2-8˚C |
| | Long term storage at -20°C |

# Erythropoietin (EPO)

## Summary

The determination of Erythropoietin concentration serves as a diagnostic adjunct in determining the cause of anemia or erythrocytosis. Increased EPO levels are found in patients with tissue hypoxia including anemia, lung disease, cyanotic heart disease and chronic heart failure .EPO levels in patients with secondary anemia due to renal failure and other disorders such as Acquired Immune Deficiency Syndrome (AIDS) are generally inappropriately low for the degree of anemia. Low concentrations of EPO may give an early warning of kidney transplant rejection, anaemic patients with cancer, ulcerative colitis, sickle cell anemia and the anemia of prematurity. EPO also can be used to monitor AIDS patients undergoing Zidovudine (AZT) therapy. Some tumors produce EPO and, in these cases, EPO may be used as a tumor marker to monitor the effectiveness of treatment.

| | | |
|---|---|---|
| Remarks | Morning sample before 12 noon. | |
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | 3.22-31.9 mIU/mL | |
| Sample stability | 8 h | at room temperature |
| | 1 wk | at 2-8˚C |
| | 2 mth | at -20 ˚C |

# Estradiol II (E2)

## Summary

Estradiol is utilized clinically in the elucidation of fertility disorder in the hypothalamus-pituitary-gonad axis, gynecomastia, estrogen-producing ovarian and testicular tumors and in hyperplasia of the adrenal cortex. It is useful for monitoring the fertility therapy being undergone and determining the time of ovulation within the framework of the in vitro fertilization. Osteoporosis risk is increased from 43-50 years onwards if E2 values are below 30 pg/ml during pregnancy.

| | | |
|---|---|---|
| Remarks | Sample should not be taken from patients receiving therapy with high biotin doses until at least 8 hr following the last biotin administration. | |
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Male (♂) (0-10 yr) | <5.0-20 pg/mL |
| | Female (♀) (0-10 yr) | 6.0-27.0 pg/mL |
| | Male (♂) | 7.63-42.6 pg/mL |
| | Follicular | 12.5-166 pg/mL |
| | Ovulation | 85.8-498 pg/mL |
| | Luteal | 43.8-211 pg/mL |
| | 1st Trimester | 215->4300 pg/mL |
| | 2nd Trimester | 801-5763 pg/mL |
| | 3rd Trimester | 1810-13890 pg/mL |
| Conversion factors | pmol/L x 0.273 = pg/mL (ng/L) | |
| | pg/mL x 3.67 = pmol/L | |
| Sample stability | 1 d | at room temperature |
| | 3 d | at 2-8 ˚C |
| | 1 yr | at - 20 ˚C |

# Extractable Nuclear Antigens (ENA)

## Summary

Antibodies to Extractable Nuclear Antigens (ENA) have been associated with several autoimmune syndromes, and appear to be of diagnostic and/or prognostic significance in systemic sclerosis, mixed connective tissue disease, Sjögren's syndrome, polymyositis, dermatomyositis, systemic lupus erythematosus, and rheumatoid arthritis.

• Antibodies to the Sm (Smith) antigen are considered a specific serologic marker due to their high degree of specificity for Systemic Lupus Erythematosus (SLE). These antibodies are seen in up to 30% of SLE patients, and have been associated with active renal disease.

• Antibodies to RNP are not considered a specific serologic marker for rheumatic diseases including SLE, scleroderma, Sjögren's syndrome, sharp syndrome, poly-/dermatomyositis and rheumatoid arthritis.

• Antibodies to SSA (Ro) are originally described as nuclear RNA-protein antigens in patients with Sjögren's syndrome, SLE, Neonatal lupus erythematosus.

• Antibodies to SSB (LA) are found in patients with subacute cutaneous lupus, Sjögren's syndrome who develop vasculitis.

• Antibodies to Scl-70 antigens have been identified as a cellular enzyme; DNA topoisomerase Antibodies to Scl-70 have been reported in patients with progressive systemic sclerosis, particularly the subset of patients with diffuse scleroderma.

• Antibodies to Jo-1, which is the cellular enzyme histidyl tRNA synthetase, are found in patients with polymyositis or dermatomyositis, but not in other myopathies. Anti Jo-1 antibodies have also been shown to have a high association with interstitial lung disease seen in conjunction with myositis.

•Antibodies to Proliferating Cell Nuclear Antigen (PCNA), are found in patients with Systemic Lupus Erthematosus (SLE).

• Antibodies to dsDNA are found in patients with Systemic Lupus Erthematosus (SLE).

• Antibodies to Centromere Antigens (ACA) are found in patients with systemic sclerosis (limited and duffuse forms) and primary biliary cirrhosis.

• Antibodies to PML-Scl antigens are detected in patients with systemic sclerosis including overlap syndrome, Polymyositis/ systemic sclerosis ( Anti-PM-Scl75 positive and Anti- PM-Scl100 positive)

• Antibodies to Nucleosomrs have been found in patients with Systemic Lupus Erthematosus SLE.

• Antibodies to all five Histones types H1, H2A, H2B, H3, H4 they all constant find in drug-induced lupus erthematosus, Systemic Lupus Erthematosus (SLE), Rheumatoid Arthritis.

• Antibodies to Ribosomal P- protein (3 proteins of the 60S ribosomal subunit P0, P1, P2) are specific for Systemic Lupus Arthritis SLE.

• Antimitochondrial Antibodies (AMAs) are detectable in >90% of patients with Primary Biliary Cirrhosis (PBC).

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative | |
| Sample stability | 2d | at room temperature |
| | 2 wk | at 2-8 °C |
| | 1 yr | at -20˚C |

# Factor IX

## Summary

Factor IX is a glycoprotein Formed in the liver as a clotting physiologically-indifferent precursor (acarboxy-factor IX), it is converted to true factor IX by vitamin K-dependent carboxylase.

Acquired deficient states occur:

- During treatment with oral anticoagulants.
- In liver disease such as cirrhosis or hepatitis.
- In vitamin K absorption disorders, e.g. in hemorrhagic disease in neonates, icterus or treatment with antibiotics.

| | |
|---|---|
| Remarks | Centrifuge 10 min, Fresh sample. |
| Method | Clotting time |
| TAT | Dy/AB |
| Sample type | CP |
| Expected value | 60-150 % |
| Sample stability | 6 h at room temperature<br>2 wk at -20˚C |

# Factor V

## Summary

Factor V (also known as proaccelerin), congenital deficiency of factor V occurs in Owren's disease. Acquired deficiencies of factor V are associated with deficiencaies of the factors VII, X, and II, and are found in liver damage (cirrhosis, hepatitis), in fibrinolysis and in Disseminated Intravascular Coagulation (DIC).

| | |
|---|---|
| Remarks | Centrifuge 10 min, Fresh sample. |
| Method | Clotting time |
| TAT | Dy/AB |
| Sample type | CP |
| Expected value | 70-120 % |
| Sample stability | 4 h at room temperature |
| | 2 wk at -20˚C |

# Factor VII

## Summary

Factor VII is a vitamin K dependent glycoprotein. Acquired deficiencies are observed in cases of treatment with vitamin K antagonists, liver disease and vitamin K absorption disorders. Clinically relevant bleeding complications are observed at factor VII below 10% and serious bleeding complications below 1 %.

| | |
|---|---|
| Remarks | Centrifuge 10 min, Fresh sample. |
| Method | Clotting time |
| TAT | Dy/AB |
| Sample type | CP |
| Expected value | 50-150 % |
| Sample stability | 6 h at room temperature |
| | Unstable at 2-8 ˚C |

# Factor VIII

## Summary

The Factor VIII level is more or less decreased according to the type of von Willebrand disease. In certain conditions, factor VIII activity may increase severely to more than 250% of the normal activity. These conditions include thromboembolitic complications, coronary arteriosclerosis, renal failure and diabetes. High levels of factor VIII are a thrombosis risk factor, notably venous thrombosis. The factor VIII level is decreased in case of the presence of factor VIII inhibitor.

| | |
|---|---|
| Remarks | Centrifuge 10 min, Fresh sample. |
| Method | Clotting time |
| TAT | Dy/AB |
| Sample type | CP |
| Expected value | 60-150 % |
| Sample stability | 4 h at room temperature |
| | 2 wk at -20˚C |

# Factor XI

## Summary

Factor XI is a vitamin K dependent glycoprotein, factor XI deficiency is characterized by hemorrhagic syndrome of varying degrees of severity, generally following a dental extraction or surgical operation.

| | | |
|---|---|---|
| Remarks | Centrifuge 10 min, Fresh sample. | |
| Method | Clotting time | |
| TAT | Dy/AB | |
| Sample type | CP | |
| Expected value | 60-150 % | |
| Sample stability | 4 h | at room temperature |
| | 4 h | at 2-8˚C |
| | 2 wk | at -20˚C |

# Familial Mediterranean Fever (FMF)

## Summary

MEFV (Mediterranean Fever) is a human gene that provides instructions for making a protein called pyrin that plays a role in inflammation and in fighting infection. The MEFV gene is located on the short (p) arm of chromosome 16 at position 13.3. A few mutations delete small amounts of DNA from the MEFV gene, which can lead to an abnormally small protein. Among people with familial Mediterranean fever, this particular mutation is also associated with an increased risk of developing amyloidosis, a complication in which abnormal protein deposits can lead to kidney failure. The assay covers 12 mutations.

| | |
|---|---|
| Method | PCR |
| TAT | 2 d |
| Sample type | EWB/CWB |
| Expected value | Normal Genotype |
| Sample stability | 3 d at room temperature |
| | 1 wk at 2-8 ˚C |

# Ferritin

## Summary

The determination of Ferritin is a suitable method for ascertaining the iron metabolism, provides a representative measure of the body's iron

Testing Ferritin is important to indicate Iron deficiency anemia, monitoring blood donors and oral iron therapy, hemodialysis patients, and pregnant women.

The reason for the elevated values could be cell necrosis, blocked erthropoiesis or increased synthesis in tumor tissue.

| | | | |
|---|---|---|---|
| Method | ECLIA | | |
| TAT | Dy/AB | | |
| Sample type | S/HP/EP/CP | | |
| Expected value | Age | Male ( ng/mL) | Female ( ng/mL) |
| | 0 -4 mth | 80-500 | 80-500 |
| | 4 mth-10 yr | 15-150 | 15-150 |
| | + 10 yr | 3-0400 | 13-150 |
| Sample stability | 1 wk | at room temperature | |
| | 3 wk | at 2-8˚C | |
| | 1 yr | at -20˚C | |

# Fibrinogen

## Summary

Fibrinogen is important in the investigation of a possible bleeding disorder or thrombotic episode, follow-up on an abnormal Prothrombin Time (PT) or Partial Thromboplastin Time (PTT) and/or an episode of prolonged or unexplained bleeding. It also helps in monitoring the status of a progressive disease (such as liver disease) over time, or rarely, to monitor treatment of an acquired condition (such as Disseminated Intravascular Coagulation DIC). Sometimes, fibrinogen is important along with other cardiac risk markers such as C-Reactive Protein (CRP), to help determine a person's overall risk of developing cardiovascular disease.

| | | |
|---|---|---|
| Remarks | Fresh sample. | |
| Method | Latex Agglutination | |
| Sample type | CP | |
| Expected value | 200-400 mg/dL | |
| Sample stability | 1 wk | at room temperature |
| | 1 wk | at 2-8˚ C |
| | 1 mth | at -20˚C |

# Fibrinogen Degradation Product (FDP)

## Summary

An abnormal fibrinogenolytic activity shown by high levels of FDP in plasma can be found in clinical states such as eclampsia, carcinoma, post-operative complications, cardiac disorders, hepatic disorders, fibrinolysis, pulmonary embolism and Deep Vein Thrombosis (DVT).

| | | |
|---|---|---|
| Remarks | Fresh sample. | |
| Method | Latex Agglutination | |
| TAT | Dy/A | |
| Sample type | CP | |
| Expected value | Negative: | <5 µg/mL |
| Sample stability | 3h | at room temperature |
| | 1 d | at 2-8˚ C |
| | 1 mth | at -20˚C |

# Fluvoxamine

## Summary

Fluvoxamine (Luvox) is an antidepressant drug used for the treatment of Major Depressive Disorder (MDD), Obsessive Compulsive Disorder (OCD), and anxiety disorders such as Panic Disorder And Post-Traumatic Stress Disorder (PTSD). The determination of fluvaxamine level is important for monitoring therapy and indicates drug toxicity.

| | | |
|---|---|---|
| Method | GC | |
| TAT | 3 d | |
| Sample type | HP | |
| Expected value | 30-300 ng/mL | |
| Sample stability | 2 wk | at room temperature |
| | 2 wk | at 2-8˚C |
| | 6 mth | at -20˚C |

# Follicle-Stimulating Hormone (FSH)

## Summary

FSH and LH regulate and stimulate the growth and function of the glands (ovaries and tstes) synergistically.

Due to changes in ovarian function and reduced estrogen secretion, high FSH concentration occurs during menopause.

The determination of FSH with LH utilized for the following indications: congenital diseases chromosome aberrations, Polycystic Ovaries (PSO), amenorrhea (causes), suspicion of hypogonadism, differential diagnosis of hypogonadism (testicular or central), infertility, climax praecox, estrogen medication in the menopause, primary ovarian failure ( turner's syndrome).

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Children | 0.2-3.8 mIU/mL |
| | Male (♂) | 1.5-12.4 mIU/mL |
| | Follicular | 3.5-12.5 mIU/mL |
| | Ovulation | 4.7-21.5 mIU/mL |
| | Luteal | 1.7-7.7 mIU/mL |
| | Postmenopause | 25.8-134.8mIU/mL |
| Sample stability | 2 wk | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Food Intolerance Test (FIT)

## Summary

The FIT was developed to detect up to 181 different nutriments and determine the individual nutritional profile of each patient by detection of elevated IgG titers concentration in blood serum, in many cases food intolerance lead to chronic diseases which are typically resistant to conventional therapies.

### List of the 181 Food Tests

| | | | |
|---|---|---|---|
| Almonds | Blueberry | Chicken | Dates |
| Amarath | Broccoli | Chicorée | Dear Meat |
| Anchovy | Brussel Sprout | Chinese cabbage | Dill |
| Anis | Buttermilk | Chive | Dinkel |
| Apple | Cacao | Cinnamon | Duck |
| Apricot | Camembert | Citron | Egg white |
| Artichokes | Canderel | Clove | Egg yolk |
| Asparagus | Carob | Coconut | Eggplant |
| Aspergillus Niger | Carp | Codfish | Emmental |
| Avocado | Carrot | Coffee | Endive |
| Bakers yeast | Cashew | Coriander | Escarol |
| Baking powder | Cauliflower | Cottage Cheese | Fennel |
| Banana | Cayenne pepper | Cow's milk | Fig |
| Barley | Celeries | Cranberry | Garlic |
| asilic | Chamomile | Cress | Ginger |
| Beans | Cheese fond. | Cucumber | Glutamate |
| Beef | Cherries | Cumin | Goat's milk |
| Beet root | Chestnut | Curry | Goats Cheese |
| Blackberry | Chick Pea | Dandelion | Goose berry |

| | | | |
|---|---|---|---|
| Gouda | Maize | Pear | Sole |
| Grape green | Mallow | Peppercorn | Soya Bean |
| Grape red | Malt | Peppermint | Spinach |
| Grapefruit | Mandarin | Peppers | Sting. nettle |
| Green bean | Mango | Pineapple | Strawberry |
| Green cabbage | Mangold | Pistachio | Sugar Melon |
| Green pea | Majoram | Plaice | Sunflower Oil |
| Green pepper | Millet | Plum | Swed. turnip |
| Halibut | Mozzarella | Pork meat | Tea, black |
| Hazelnut | Mushroom | Potato | Thistle oil |
| Herring | Mussels | Pumpkin | Thyme |
| Honey | Mustard Corn | Quinoa | Tobacco |
| Honey Melon | Nectarine | Rabbit Meat | Tomatoes |
| Hop | Nutmeg | Radish | Trout |
| Horseradish | Oats | Raspberries | Tuna |
| Kiwi | Olive | Red bass | Turkey |
| Kohlrabi | Onion | Red currant | Vanilla |
| Lamb | Orange | Rice | Veal Meat |
| Laurel | Oregano | Roquefort | Walnut |
| Leek | Papaya | Rosemary | Water Melon |
| Lentils | Para nuts | Rye | Wh. cabbage |
| Lettuce | Para nuts | Sage | Wheat |
| Lime Flower | Parsley | Salmon | Yeast |
| Linseed | Parsley Root | Sardine | Yello Bean |
| Lobster | Peach | Sesame | Yoghurt |
| Mackerel | Peanut | Shrimp | Zucchini |

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | 10 – 14 d | |
| Sample type | S | |
| Expected value | Refer To Report | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8˚C |
| | Long term storage | at -20 ˚C |

# Fragile X Syndrome

## Summary

Fragile X Syndrome (FXS), Martin–Bell syndrome, or Escalante's syndrome is a genetic syndrome that is the most widespread single-gene cause of autism and inherited cause of mental retardation among boys. It results in a spectrum of intellectual disabilities ranging from mild to severe as well as physical characteristics such as an elongated face, large or protruding ears, and large testes (macroorchidism), and behavioral characteristics such as stereotypic movements (e.g. hand-flapping), and social anxiety.

Fragile X syndrome is associated with the expansion of the CGG trinucleotide repeat affecting the Fragile X Mental Retardation 1 (FMR1) gene on the X chromosome, resulting in a failure to express the Fragile X Mental Retardation Protein (FMRP), which is required for normal neural development. Depending on the length of the CGG repeat, an allele may be classified as normal (unaffected by the syndrome), a premutation (at risk of fragile X associated disorders), or full mutation (usually affected by the syndrome).

| | |
|---|---|
| Method | PCR |
| TAT | 5 d |
| Sample type | EWB |
| Expected value | Normal genotype |
| Sample stability | 3 d at room temperature |
| | 1 wk at 2-8˚C |

# Free Triiodothyronine (FT3)

## Summary

Determination of this hormone concentration is important for the diagnostic differentiation of euthyroid, hyperthyroid and hypothyroids states.

| | |
|---|---|
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S/HP/EP/CP |

| Expected value | Age | Value (pmol/L) |
|---|---|---|
| | <1 yr | 4.5-10.5 |
| | 1-6 yr | 3.8-8.2 |
| | 6-13 yr | 3.8-8.6 |
| | 13-17 yr | 2.8-7.1 |
| | + 17 yr | 3.00-7.00 |

| | |
|---|---|
| Conversion factors | pg/mL x1.536 =pmol/L |

| Sample stability | | |
|---|---|---|
| | 1 d | at room temperature |
| | 2 wk | at 2-8˚C |
| | 3 mth | at -20˚C |

# Free Thyroxine (FT4)

## Summary

The determination of free thyroxine is an important element in clinical routine diagnostics. Free T4 is measured together with TSH when thyroid function disorders are suspected. The determination of FT4 is also suitable for monitoring thyrosuppressive therapy.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP/CP | |
| Expected value | Age | Value (pmol/L) |
| | <1 yr | 13.9-26.1 |
| | 1-6 yr | 12.1-22.0 |
| | 6-13 yr | 13.9-22.1 |
| | 13-17 yr | 13.6-23.2 |
| | + 17 yr | 12.00-22.00 |
| Conversion factors | pmol/L x 0.077688 = ng/dL | |
| | ng/dL x 12.872 = pmol/L | |
| | pmol/L x 0.77688 = ng/L | |
| Sample stability | 2 d | at room temperature |
| | 8 d | at 2-8˚C |
| | 3 mth | at -20˚C |

# Fructose

## Summary

The measurement of Fructose in semen reflects the secretory function of seminal vesicles, and help in assessing the diagnosis and the management of male infertility.

| | | |
|---|---|---|
| Method | Colorimetric | |
| TAT | Dy/A | |
| Sample type | SF | |
| Expected value | 59-556 mg/dL | |
| Conversion factors | mg/dL x 0.0555= mmol/L | |
| | mmol/L x 18.02= mg/dL | |
| Sample stability | 1 d | at room temperature |
| | Several days | at - 20 ˚C |

# FV Leiden (Activated Protein C)

## Summary

FV Leiden (G1691A; R506Q): represents one of the most important genetic risk factors for inherited thrombophilia; leads to activated protein C resistance; occurs in 20-50% of patients with venous thromboembolism.

| | | |
|---|---|---|
| Method | PCR | |
| TAT | 2d | |
| Sample type | EWB/CWB | |
| Expected value | Normal Genotype | |
| Sample stability | 3 d | at room temperature |
| | 1 wk | at 2-8˚C |

# γ-Glutamyltransferase (GGT)

## Summary

γ-Glutamyltransferase is used in the diagnosis and monitoring of hepatobiliary diseases. Enzymatic activity of GGT is often the only parameter with increased values when testing for such diseases, and is one of the most sensitive indicators known. γ-glutamyltransferase is also a sensitive screening test for occult alcoholism. Elevated GGT activities are found in the serum of patients requiring long-term medication with phenobarbital and phenytoin.

| | |
|---|---|
| Method | Colorimetric |
| TAT | Dy/AB |
| Sample type | S/HP |
| Expected value | Male (♂) : 11-49 U/L |
| | Female (♀): 7-32 U/L |
| Conversion factors | U/L x 0.0167 = μkat/L |
| Sample stability | 1 wk at room temperature |
| | 1 wk at 2-8˚C |
| | 1 yr at -20˚C |

# Gentamicin

## Summary

At therapeutic serum concentrations, Gentamicin is capable of inhibiting the growth of many gram positive cocci, especially penicillinase-producing staphylococci and displays activity against most strains of Pseudomonas aeruginosa. At concentrations of 10 µg/mL (20.9 µmol/L), most strains of Ecoli, Proteus spp., Klebsiella, Aerobacter, Clostridium, Brucella spp., Salmonella, Serratia, and Shigella are inhibited. Because of these characteristics, gentamicin has been most successfully used in the treatment of serious infections, especially those caused by gram-negative bacilli.

| | |
|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. |
| Method | FP |
| TAT | Dy/A |
| Sample type | S/HP |
| Expected value | Peak Level: 6-10 µg/mL |
| | Trough Level: 0.5-2.0 µg/mL |
| Conversion factors | µg/ml x 2.09 = µmol/L |
| | µmol/L x 0.478 = µglmL |
| Sample stability | 4 h at room temperature |
| | 1 mth at 2-8˚C |
| | 1 mth at -20˚C |

# Glucose

## Summary

The most frequent cause of hyperglycemia is diabetes mellitus resulting from a deficiency in insulin secretion or action. A number of secondary factors also contribute to elevated blood glucose levels. These include pancreatitis, thyroid dysfunction, renal failure, and liver disease. A variety of conditions may cause low blood glucose levels such as insulinoma, hypopituitarism, or insulin induced hypoglycemia. Glucose measurements are used in the diagnosis and treatment of carbohydrate metabolism disorders including diabetes mellitus and idiopathic hypoglycemia. Glucose measurement in urine is used as a diabetes screening procedure and to aid in the evaluation of glucosuria, to detect renal tubular defects, and in the management of diabetes mellitus. Glucose measurement in cerebrospinal fluid is used for evaluation of meningitis, neoplastic involvement of meninges, and other neurological disorders.

| | |
|---|---|
| Remarks | Fasting |
| Method | Enzymatic (HK) |
| TAT | Dy/A |
| Sample type | S/D |

| | | |
|---|---|---|
| Expected value | S: | Adults: 74-106 mg/dL |
| | | 60-90 yr: 82-115 mg/dL |
| | | > 90 yr: 75-121 mg/dL |
| | | Children: 60-100 mg/dL |
| | | Neonates (1 d): 40-60 mg/dL |
| | | Neonates (> 1 d): 50-80 mg/dL |
| | 24U: | < 0.5 g/24 h |
| | US: | 1-15 mg/dL |
| | CSF: | Children: 60-80 mg/dL |
| | | Adults: 40-70 mg/dL |
| Conversion factors | mmol/L × 18.02 = mg/dL | |
| Sample stability | 1 wk | at room temperature |
| | 1 wk | at 2-8˚C |
| | 1 d | at -20˚C |

# Glucose-6-Phosphate Dehydrogenase (G6PD)

## Summary

G6PD deficiency is a hereditary, sex-linked condition carried on the X chromosome (genetic disorders) found mostly in males. Affected patients are at risk for hemolytic anemia, which can be induced by exposure to certain drugs and foods.

| | |
|---|---|
| Method | Colorimetric |
| TAT | Dy/AB |
| Sample type | EWB |
| Expected value | 4.6-13.5 U/g Hb |
| | Newborn: 4.6-20 U/g Hb |
| Sample stability | 8 h at room temperature |
| | 1wk at 2-8˚C |

# Haptoglobin (HAPT)

## Summary

A reduction in the level of free haptoglobin is indicative of intravascular hemolysis. Increased values are usually based on the acute phase function of haptoglobin and occur in inflammatory reaction.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S/EP | |
| Expected value | 30-200 mg/dL | |
| | <1 yr: 5-48 mg/dL | |
| Conversion factors | mmol/L × 18.02 = mg/dL | |
| Sample stability | 3 mth | at room temperature |
| | 8 d | at 2-8˚C |
| | 3 mth | at -20˚C |

# Hb Composition

## Summary

Over 400 mutant haemoglobins are now known, some of which may cause serious clinical effects, especially in the homozygous state or in combination with another abnormal haemoglobin. The abnormalities synthesized are divided into three groups:

1-Production of an abnormal protein molecule (e.g. sickle cell anemia).

2-Reduction in the amount of normal protein synthesis (e.g. thalassaemia).

3-Developmental anomalies (e.g. hereditary persistence of fetal haemoglobin).

The two mutant haemoglobins most commonly seen are HbS and HbC. Hb lepore, HbE, HbG- Philadelphia, HbD-Los Angeles and HbO-Arab are seen less frequently.

| | |
|---|---|
| Method | GEL/ HPLC |
| TAT | Dy/A |
| Sample type | EWB |
| Expected value | Hb A1: 96.5 - 98.5% |
| | HbA2: 1.5 - 3.5% |
| | HbF: < 2% |
| | HbF (up to 6 mth old) :7.2 – 39.2 % |
| Sample stability | 1 wk at 2-8 ˚C |

# Helicobacter pylorus Abs (H. pylori IgA, IgG)

## Summary

Helicobacter pylori (earlier Campylobacter pylori) is considered as an important and frequent pathogenic factor for the development of chronic gastritis of the antrum type (antral alcalization) with hypochlorhydria. An association with gastritis associated ulcer diseases is described. The bacterium is acid resistant and binds to substances, which are similar to blood-groups (increased occurrence in blood-group 0).

The infection is usually acquired in infancy, also depending on social environment. A first infection in adulthood is relatively rare. Sixty to 80% of the population is affected; however, not everybody presents symptoms. A connection with chronic urticaria is suspected.

The acute infection changes mostly into a persisting colonization of the anacidic gastral mucosa. This is supported by an additional enterovirus infection, or e.g. during undernourishment in childhood. The immune response is mainly production of IgG (chronic infection) and IgA (fresh infection).

| | |
|---|---|
| Method | ELISA/ECLIA |
| TAT | IgA : Dy/A |
| | IgG :Dy/AB |
| Sample type | S |
| Expected value | IgA: Negative: <1.0 |
| | IgG: Negative: <1.0 U/mL |
| Sample stability | 3 d at 2-8 ˚C |

# Helicobacter pylorus Antigen (H. pylori Ag)

## Summary

Helicobacter pylorus (H. pylori) is a bacterium that causes chronic inflammation of the inner lining of the stomach (gastritis) in humans. This bacterium also is considered as a common cause of ulcers worldwide. H pylori infection is most likely acquired by ingesting contaminated food and water, and through person to person contact. Infected individuals usually carry the infection indefinitely (for life) unless they are treated with medications to eradicate the bacterium. One out of every six patients with H. pylori infection may develop ulcers of the duodenum or stomach. H. pylorus also is associated with stomach cancer and a rare type of lymphocytic tumor of the stomach called MALT (mucosa-associated lymphoid tissue) lymphoma. In addition, several recent research papers have shown a link between diabetes, infections, elevated hemoglobin A1C levels, and H. pylori. This test is important to demonstrate H.pylori infection and particularly appropriate for children or those who prefer providing samples in the privacy of their home.

| | |
|---|---|
| Method | ICT |
| TAT | Dy/A |
| Sample type | ST |
| Expected value | Negative |
| Sample stability | 3 d at 2-8 ˚C |

# Hemoglobin A1c (HbA1c)

## Summary

In the erythrocytes, the relative amount of HbA converted to stable HbA1c increases with the average concentration of glucose in the blood. HbA1c reflects the average blood glucose level during the preceding 2 to 3 months. HbA1c is thus suitable to monitor long-term blood glucose control in individuals with diabetes mellitus. Glucose levels closer to the time of the assay have a greater influence on the HbA1c level.

| | | |
|---|---|---|
| Method | Turbidometric | |
| TAT | Dy/AB | |
| Sample type | EWB | |
| Expected value | 4.2-6.2 % | |
| Sample stability | 3d | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 6 mth | at -20 ˚C |

# Hemosiderin

## Summary

Hemosiderin is an iron-storage complex. It is always found within cells (as opposed to circulating in blood) and appears to be a complex of ferritin, denatured ferritin and other materials. Several disease processes result in deposition of larger amounts of hemosiderin, which can lead to organ damage. Hemosiderin may deposit in diseases associated with iron overload in chronic blood loss, which requires frequent blood transfusions, such as sickle cell anemia and thalassemia.

| | | |
|---|---|---|
| Method | Prussian blue stain | |
| TAT | Dy/A | |
| Sample type | US | |
| Expected value | Negative | |
| Sample stability | 1 h | at room temperature |
| | 1 d | at 2-8˚C |
| | 1 wk | at -20˚C |

# Hepatitis A Virus Abs (HAV-IgG,IgM), Total

## Summary

Total anti-HAV is positive at the onset of a hepatitis A infection (IgM). After natural infection, anti-HAV IgG antibodies can usually be detected lifelong and provide protection against the disease if the organism is reinfected. Upon vaccination against hepatitis A, anti-HAV IgG antibodies can be detected after 2 weeks. In the case of complete immunization, protection usually lasts for years. There is no limit value to define immune protection but anti-HAV values > 10-20 IU/L are generally considered to be protective against infection.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP/CP | |
| Expected value | Negative: <3 IU/mL | |
| Sample stability | 1 wk | at 2-8 °C |
| | 3 mth | at -20 °C |

# Hepatitis A Virus IgM Abs (HAV-IgM)

## Summary

An acute hepatitis A infection can be assumed if anti-HAV IgM antibodies are detected. Anti-HAV IgM antibodies can always be detected at the onset of the disease, and usually disappear 3 to 4 months later. Anti-HAV IgM can also be detected in some patients for a longer period of time, however, HAV IgM antibodies develop only very rarely after vaccination.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP/CP | |
| Expected value | Negative: <1.0 | |
| Sample stability | 1 wk | at 2-8 °C |
| | 6 mth | at -20 °C |

# Hepatitis B ( HBV-DNA )

## Summary

HBV DNA is detectable in the serum of persons with acute and chronic HBV infection. Monitoring HBV DNA levels is useful in determining the response of a person with chronic HBV infection to interferon treatment.

| | | |
|---|---|---|
| Remarks | Serum Separatation within max. 6 hrs. | |
| Method | PCR | |
| TAT | 2 d | |
| Sample type | S | |
| Expected value | Not detected < 6.00 IU/mL | |
| Sample stability | 3 d | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 6 wk | at - 20 ˚C to -80˚C |

# Hepatitis B core Abs (HBcAb)

## Summary

Hepatitis B virus belongs to the HEPADNA viruses,consisting of a core and a coat, which contains partial double-strand circular DNA and DNA polymerase; Incubation period is 60-180 days; the transmission is parenterally, sexually or perinatally. Prophylaxis: passive: immunoglobulin; prevention: vaccination (antibodies against surface ag), infectiosity: highly infectious if HBe antigen is present.

The test detects core IgG and IgM-antibodies combined in one test which indicates the suspicion of postacute hepatitis and the immunity status. HBc antibodies occur immediately after appearance of HBs-Ag.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP/CP | |
| Expected value | Negative: <1.0 | |
| Sample stability | 5 d | at 2-8 °C |
| | 3 mth | at -20 °C |

# Hepatitis B envelop Abs (HBeAb)

## Summary

Anti-HBs and anti-Hbe antibodies can - together with anti-HBc antibodies - persist the whole life after infection and are characteristic for immunity. However, in most cases anti-Hbe antibodies show lower persistence and are below the detection limit after several years. Disappearance of Hbe antigen and the occurrence of anti-HBe indicate the transition into the nonreplicative phase.

| | |
|---|---|
| Method | ELISA |
| TAT | Dy/AB |
| Sample type | S/HP/EP/CP |
| Expected value | Negative: >1.0 |
| Sample stability | 1 wk at 2-8 °C |
| | 3 mth at -20 °C |

# Hepatitis B envelop Antigen (HBeAg)

## Summary

HBeAg appears in serum during acute HBV infections and is detectable for a short period (days to weeks). The detection of HBeAg is generally associated with the presence of large quantities of the virus. In the recovery phase following acute hepatitis B, HBeAg is the first serological marker which becomes negative and is replaced by the corresponding antibody (anti-HBe). Demonstration of anti-HBe in these persons is an indication of the presence of precore stop codon mutants.

| | |
|---|---|
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S/HP/EP/CP |
| Expected value | Negative: <1.0 |
| Sample stability | 1 wk at 2-8 °C |
| | 3 mth at -20 °C |

# Hepatitis B surface Abs ( HBsAb)

## Summary

The presence of HBs antibodies in serum indicates contact with hepatitis B virus (or HBV surface Ag in vaccination). Simultaneously detectable HBc antibodies indicate acquired immunity by HBV; HBsAbs without HBc antibodies indicate vaccination.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/EP | |
| Expected value | Negative: <10 IU/L | |
| Sample stability | 5 d | at 2-8 °C |
| | 3 mth | at -20 °C |

# Hepatitis B surface Antigen ( HBsAg)

## Summary

The detection of HBsAg in human serum or plasma indicates an infection by the hepatitis B virus. HBsAg is the first immunological marker and is generally present some days or weeks before clinical symptoms begin to appear. HBsAg tests are also used to monitor the course of the disease in persons with acute or chronic HBV infections to prevent the transmission of the hepatitis B virus by blood and blood products and if necessary, to check the efficacy of an antiviral therapy. In addition, HBsAg tests are recommended as part of prenatal care, in order to be able to initiate suitable measures for preventing as far as possible the transmission of an HBV infection to the newborn child.

| | |
|---|---|
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S/HP/EP/CP |
| Expected value | Negative: <1.0 |
| Sample stability | 5 d at 2-8 °C |
| | 3 mth at -20 °C |

# Hepatitis C RNA (HCV-RNA)

## Summary

HCV-RNA test is intended for use, in conjunction with clinical presentation and other laboratory markers, as an aid in assessing viral response to antiviral treatment as measured by changes in serum or plasma HCV RNA levels. Recent data suggest that early changes in serum/plasma HCV RNA levels may predict a long-term response to interferon therapy.

| | | |
|---|---|---|
| Remarks | Serum Separatation within max. 6 hr. | |
| Method | PCR | |
| TAT | 2 d | |
| Sample type | S | |
| Expected value | Negative < 25 IU/mL (< 100 copy/mL) | |
| Sample stability | 3 d | at 2-8 ˚C |
| | 1 wk | at - 20 ˚C |

# Hepatitis C Virus Abs (anti-HCV)

## Summary

Infection with HCV frequently leads to chronic hepatitis and cirrhosis, and is associated with the development of hepatocellular carcinoma. Common extrahepatic manifestations comprise mixed cryoglobulinemia and other rheumatic diseases. Anti-HCV antibody tests are used alone or in combination with other tests (e.g. HCV-RNA) to detect an infection with hepatitis C virus and to identify blood and blood products of individuals infected with HCV.

| | | |
|---|---|---|
| Method | ECLIA/ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1.0 | |
| Sample stability | 3 d | at room temperature |
| | 3 wk | at 2-8 °C |
| | 3 mth | at-20 °C |

# Hepatitis Delta Virus (HDV Total)

## Summary

Hepatitis D is caused by Hepatitis D virus (Delta agent) which requires co-infection with Hepatitis B Virus (HBV) for its replication. Transmitted percutaneously or sexually through contact with infected blood or blood products, HDV is associated with the most severe forms of chronic and acute hepatitis in many HBsAg positive patients. The co-infection with HDV can lead to severe acute hepatitis disease with low risk of chronic stage development. The determination of HDV specific serological markers (HDV Ag, HDV IgM and HDV IgG) represents an important tool to the classification of the etiological agent, and follows up of infected patients and monitoring their treatment. A positive result for HDV total antibody may indicate either acute or chronic HDV infection. HDV antibodies appear transiently during acute infection, and typically disappear with resolution of the infection. HDV antibodies usually persist in chronic infection.

| | | |
|---|---|---|
| Method | EIA | |
| TAT | 7d | |
| Sample type | S | |
| Expected value | Negative: <1.0 | |
| Sample stability | 1 wk | at room temperature |
| | 2 mth | at 2-8˚C |

# Hepatitis Delta Virus IgM Abs (HDV-IgM)

## Summary

Hepatitis D is caused by Hepatitis D virus (Delta agent) which requires co-infection with Hepatitis B virus (HBV) for its replication. Transmitted percutaneously or sexually through contact with infected blood or blood products, HDV is associated with the most severe forms of chronic and acute hepatitis in many HBsAg positive patients. The co-infection with HDV can lead to severe acute hepatitis disease with low risk of chronic stage development. The serological detection of IgM class antibodies is an important marker for diagnosis and monitoring of patients during early infection period and post treatment recovery. Decreasing or low titers of IgM suggest early recovery during HDV co- and acute super-infection while constantly elevated level of IgM indicates possible progression to chronic carrier stage.

| | | |
|---|---|---|
| Method | EIA | |
| TAT | 3d | |
| Sample type | S | |
| Expected value | Negative :< 1.0 | |
| Sample stability | 1 wk | at room temperature |
| | 2 mth | at 2-8˚C |

# Hepatitis E Virus (HEV Total)

## Summary

HEV is a waterborne disease, and contaminated water or food supplies have been implicated in major outbreaks. Hepatitis E has a fecal-oral transmission route. Typical signs and symptoms of hepatitis include jaundice( yellow discoloration of the skin and sclera of the eyes, dark urine and pale stools), anorexia (loss of appetite), an enlarged, tender liver (hepatomegaly), abdominal pain and tenderness, nausea ,vomiting, and fever, although the disease may range in severity from subclinical to fulminant. There is no evidence for sexual transmission or for transmission by transfusion. Person to person transmission is uncommon.

The determination of HEV specific serological markers (HEV Ag, HEV IgM and HEV IgG) is important for monitoring treatment. A positive result for HEV total antibody may indicate either acute or chronic HEV infection.

| | |
|---|---|
| Method | EIA |
| TAT | 3 d |
| Sample type | S |
| Expected value | Negative: <1.0 |
| Sample stability | 1 wk at room temperature |
| | 2 mth at 2-8˚C |

# Hepatitis E Virus IgM Abs (HEV-IgM)

## Summary

HEV is a waterborne disease, and contaminated water or food supplies have been implicated in major outbreaks. Hepatitis E has a fecal-oral transmission route. Typical signs and symptoms of hepatitis include jaundice( yellow discoloration of the skin and sclera of the eyes, dark urine and pale stools), anorexia (loss of appetite), an enlarged, tender liver (hepatomegaly), abdominal pain and tenderness, nausea ,vomiting, and fever, although the disease may range in severity from subclinical to fulminant. There is no evidence for sexual transmission or for transmission by transfusion. Person to person transmission is uncommon.

The detection of IgM class antibodies is an important marker for diagnosis and monitoring early infection period and post treatment recovery. Decreasing or low titers of IgM suggest early recovery during HEV infection while constantly elevated level of IgM indicates possible progression to chronic carrier stage.

| | |
|---|---|
| Method | EIA |
| TAT | 10- 14 d |
| Sample type | S |
| Expected value | Negative: <1.0 |
| Sample stability | 1 wk at room temperature |
| | 2 mth at 2-8˚C |

# Herpes Simplex Virus I Abs (HSV-I IgG,IgM)

## Summary

Primary HSV-1 infections are typically acquired during childhood. Following oropharyngeal infection, the trigeminal ganglion becomes colonized and harbors latent virus. A major manifestation of HSV-1 infection in young children is gingivostomatitis, a serious infection of the gums, tongue, mouth, lip, facial area, and pharynx. In older people infected with HSV-1 upper respiratory tract infections and mononucleosis-like syndrome are very common. Genital HSV-1 results from self-inoculation or from oral sexual practices. The determination of IgG antibodies is a valuable indicator in the diagnosis of the beginning infection whereas IgM antibodies will be found in recent infection with Herpes virus.

| | | |
|---|---|---|
| Method | ELISA/IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | IgG: Negative: <1:10 | |
| | IgM: Negative: <1.0 | |
| Sample stability | 8 h | at room temperature |
| | 2 d | at 2-8 °C |
| | 3 mth | at -20 °C |

# Herpes Simplex Virus II Abs (HSV-II IgG,IgM)

## Summary

HSV-II viruses are sexually transmitted produce a wide spectrum of symptoms, e.g. mucous membrane and skin lesions and ocular, visceral, and central nervous system (CNS) disease, mucocutaneous infection, vulvovaginitis, herpes gentalis, herpetic proctitis, inoculation herpes, aczema herpeticum, meningoencephalitis, herpes neonatorum. The determination of IgG antibodies is a valuable indicator in the diagnosis of the beginning infection whereas IgM antibodies will be found in recent infection with Herpes virus.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative<1.0 | |
| Sample stability | 8 h | at room temperature |
| | 2 d | at 2-8 °C |
| | 3 mth | at -20 °C |

# HIV combi (HIV-Ag,HIV I & II Total Abs)

## Summary

The human immunodeficiency virus (HIV), the causative agent of Acquired Immunodeficiency Syndrome (AIDS), belongs to the family of retroviruses. HIV can be transmitted through contaminated blood and blood products, through sexual contact or from an HIV infected mother to her child before, during and after birth. Specific antigens are necessary to avoid failure in the detection of an HIV infection by immunoassay.

| | | |
|---|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. | |
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Negative: <1.0 | |
| Sample stability | 3 d | at room temperature |
| | 10 d | at 2-8 °C |
| | 3 mth | at -20 °C |

# Homocysteine (HCYS)

## Summary

Elevated total Homocysteine level is caused by four major factors, including: genetic deficiencies in enzymes involved in Homocystiene metabolism such as Cystathionine Beta-Synthase (CBS), Methionine Synthase (MS), and methylenetetrahydrofolate reductase (MTHFR), nutritional deficiency in B vitamins such as B6, B12 and folate, renal failure for effective amino acid clearance; and drug interactions, such as with nitric oxide, methotrexate and phenytoin that interfere with Homocystiene metabolism.

| | | |
|---|---|---|
| Method | Enzymatic | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | <60 yr: | 5-15 µmol/L |
| | >60 yr: | 5-20 µmol/L |
| Sample stability | 4 d | at room temperature |
| | 1 mth | at 2-8 °C |
| | 10 mth | at -20 °C |

# Human Chorionic Gonadotropin + β-subunit (HCG+β)

## Summary

Measurement of the hCG concentration permits the diagnosis of pregnancy just one week after conception. Elevated values here serve as an indication of chorionic carcinoma, hydatiform mole or multiple pregnancies. Depressed values indicate threatening or missed abortion, ectopic pregnancy, gestosis or intra-uterine death. Elevated hCG concentrations not associated with pregnancy are found in patients with other diseases such as tumors of the germ cells, ovaries, bladder, pancreas, stomach, lungs and liver.

| | |
|---|---|
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S/HP/EP/CP |

| Expected value | | |
|---|---|---|
| | Male (♂) | Up To 3 mIU/mL |
| | Non Pregnant | <4 mIU/mL |

| wk of gestation | HCG value | wk of gestation | HCG value |
|---|---|---|---|
| Wk 1 | 5-50 | Wk 2 | 20-500 |
| Wk 3 | 500-5000 | Wk 4 | 3000-19000 |
| Wk 8 | 14000-16900 | Wk 12 | 16000-160000 |
| Wk 14 | 15000-92100 | Wk 15 | 10600-64200 |
| Wk 16 | 9000-52800 | Wk 17 | 6700-47100 |
| Wk 18 | 6100-42100 | Wk 19 | 6800-42900 |

| Sample stability | | |
|---|---|---|
| | 1 d | at room temperature |
| | 3 d | at 2-8 ˚C |
| | 1 yr | at - 20 ˚C |

# Human Immunodeficiency Virus (HIV-I, II)

## Summary

This test is intended for use in conjunction with clinical presentation and other laboratory markers of disease progress for the clinical management of HIV infected patients. The test can be used to assess patient prognosis by measuring the baseline HIV RNA level or to monitor the effects of antiretroviral therapy by measuring changes in EDTA plasma HIV RNA levels during the course of antiretroviral treatment.

| | | |
|---|---|---|
| Method | PCR | |
| TAT | 2 d | |
| Sample type | EP | |
| Expected value | Not detected < 5.68 IU/mL | |
| Sample stability | 1 d | at room temperature |
| | 6 d | at 2-8˚C |
| | 1 mth | at -20˚C to -80˚C. |

# Human Leukocyte Antigen (HLA B27)

## Summary

HLA-B27 is a genetic risk factor for seronegative spondyloarthropathies, such as ankylosing spondylitis, reactive arthritis, juvenile rheumatoid arthritis and anterior uveitis .Ankylosing spondylitis (Morbus Bechterew) is the most prevalent spondyloarthropathy affecting 0.1-1% of the general population. Symptoms at the onset of the disease are usually nonspecific, posing difficulties for a firm diagnosis. Differentiating between HLA-B27 positive and negative patients is an important parameter for diagnosing spondyloarthropathies .Early and adequate treatment ameliorates the course of disease.

| | | |
|---|---|---|
| Method | PCR | |
| TAT | 2 d | |
| Sample type | EWB/CWB | |
| Expected value | Normal Genotype | |
| Sample stability | 3 d | at room temperature |
| | 1 wk | at 2-8˚C |

# Human Leukocyte Antigen A, B, C (HLA - A, B, C)

## Summary

HLA (A, B, C) Antigens play an important role in the body's immune response and have also been implicated in many disease processes, particularly those with an autoimmune component. HLA testing is done to reduce the likelihood of rejection after transplant and to avoid graft-versus-host disease. The success of a transplant depends on how closely the antigens match.

| | |
|---|---|
| Method | PCR |
| TAT | 2 d |
| Sample type | EWB/CWB |
| Expected value | Refer To Report |
| Sample stability | 3 d at room temperature |
| | 1 wk at 2-8˚C |

# Human Leukocyte Antigen DR (HLA-DR)

## Summary

HLA-DR are key molecules critical for numerous aspects of immune function, including T- cell selection, tolerance induction, antibody production, T-cell mediated immunity and inflammatory response. In transplant organ rejection, these molecules are often a common target of immune therapies to prevent the rejection of grafted tissues.

| | | |
|---|---|---|
| Method | PCR | |
| TAT | 2 d | |
| Sample type | EWB/CWB | |
| Expected value | Refer To Report | |
| Sample stability | 3 d | at room temperature |
| | 1 wk | at 2-8 ˚C |

# Human Leukocyte Antigen-B5 (HLA-B5)

## Summary

HLA-B is a human gene that provides instructions for making a protein that plays a critical role in the immune system. It has many different normal variations, allowing each person's immune system to react to a wide range of foreign invaders. HLA B5 plays an important role in the aetiology of Behçet's disease.

HLA-B is located on the short (p) arm of chromosome 6 at cytoband 21.3

| | |
|---|---|
| Method | PCR |
| TAT | 2 d |
| Sample type | EWB/CWB |
| Expected value | HLA –B 5 which associated with Behcet's diseases is not detected |
| Sample stability | 3 d at room temperature<br>1 wk at 2-8˚C |

# Human Papilloma Virus (HPV )

## Summary

Human papilloma virus is a Sexually Transmitted Infection (STI) produce epithelial tumors of the skin and mucous membranes. The majority of the known types of HPV cause no symptoms in most people, some types can cause warts (verrucae), while others can lead to cancers of the cervix, vulva, vagina, penis,oropharynx and anus, HPV has been linked with an increased risk of cardiovascular disease. In addition, HPV infections are strongly associated with an increased odds ratio of developing oropharyngeal (throat) cancer.

| | |
|---|---|
| Method | PCR |
| TAT | 2 d |
| Sample type | EnS/Tissue |
| Expected value | Not detected |
| Sample stability | 1 wk at 2-8 ˚C |

# Human Placental Growth Factor (PlGF)

## Summary

PlGF in cardiovascular diseases: PlGF can be detected in normal non-pregnant subjects at lower levels. Increased levels of PlGF can be found in patients with cardiovascular diseases as an indicator of micro - and macrovascular atherosclerosis and as a sign of pathological angiogenesis. In addition PlGF has been shown to be an independent predictor of cardiovascular morbidity and mortality in patients with type 1 und type 2 diabetes. In normal pregnancy, the pro-angiogenic factor PlGF increases during the first 2 trimesters and decreases as pregnancy progresses to term. In women who develop preeclampsia, PlGF levels have been found to be lower than in normal pregnancy.

| | |
|---|---|
| Remarks | State Gestational Age. |
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S |

| Expected value | wk of gestational | Value (pg/mL) |
|---|---|---|
| | 10-14wk | 29.4-183 |
| | 15-19wk | 65.7-203 |
| | 20-23wk | 125-541 |
| | 24-28wk | 130-1108 |
| | 29-33wk | 73.3-1108 |
| | 34-36wk | 62.7-972 |
| | 37-delivery | 52.3-659 |

| Sample stability | | |
|---|---|---|
| | 8 h | at 2-8 ˚C |
| | 4 mth | at -20 ˚C |

# Human T-cell Lymphotropic Virus (HTLV I, II)

## Summary

Human T-cell Lymphotropic Viruses (HTLVs) are pathogenic retroviruses that may cause severe haematological and neurological diseases in infected individuals. HTLV-I is known as the etiological agent of Adult T-Cell Leukemia / Lymphoma (ATL), HTLV-Associated Myelopathy / Tropical Spastic Paraparesis (HAM/TSP), and HTLV-associated uveitis. HTLV-II infection has also been associated with leukemia and neurological disease although it is less pathogenic than HTLV-I.

| | |
|---|---|
| Method | ELISA |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1.0 |
| Sample stability | 1 wk at 2-8 ˚C |

# Immunoglobulin A (IgA)

## Summary

Increased polyclonal IgA levels may occur in chronic liver diseases, chronic infections, autoimmune disorders (rheumatoid arthritis, systemic lupus erythematosis), sarcoidosis and Wiscott-Aldrich syndrome. Monoclonal IgA increases in IgA myeloma. Decreased synthesis of IgA is observed in acquired and congenital immunodeficiency diseases such as Bruton type agammaglobulinemia. Reduced levels of IgA can be caused by protein-losing gastroenteropathies and loss through skin from burns.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Age | Value (g/L) |
| | 0-1 yr | 0-0.83 |
| | 1-3 yr | 0.2-1 |
| | 4-6 yr | 0.27-1.95 |
| | 7-9 yr | 0.34-3.05 |
| | 10-11 yr | 0.53-2.04 |
| | 12-13 yr | 0.58-3.58 |
| | 14-15 yr | 0.47-2.49 |
| | 16-19 yr | 0.61-3.48 |
| | Adult | 0.7-4 |
| Conversion factors | mg/dL x 0.01 = g/L | |
| | g/L x 100 = mg/dL | |
| Sample stability | 8 mth | at room temperature |
| | 8 mth | at 2-8˚C |
| | 8 mth | at -20 ˚C |

# Immunoglobulin E (IgE )

## Summary

Elevated IgE concentration can be found in patients with allergic diseases such as hay fever, atopic bronchitis and dermatitis. The Ig E concentration in serum is normally very low. Elevated serum IgE concentration can also occur in non allergic diseases e.g. hyper-IgE syndrome and parasitic infection.

| | |
|---|---|
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S/HP/EP/CP |

**Expected value**

| Age | Value (IU/mL) |
|---|---|
| Neonates | Up To 1.5 |
| 1st yr | Up To 15 |
| 1-5 yr | Up To 60 |
| 6-9 yr | Up To 90 |
| 10-15 yr | Up To 200 |
| +15 yr | Up To 100 |

**Conversion factors**

mg/mL x 0.042 = IU/mL

IU/mL x 2.4 = mg/mL

**Sample stability**

| | |
|---|---|
| 1 wk | at room temperature |
| 1 wk | at 2-8 ˚C |
| 6 mth | at - 20 ˚C |

# Immunoglobulin G (IgG)

## Summary

Polyclonal IgG increases in serum/plasma may be present in systemic lupus erythematosis, chronic liver diseases (infectious hepatitis and Laennec's cirrhosis), infectious diseases and cystic fibrosis.

Monoclonal IgG increases in IgG-myeloma.

Decreased synthesis of IgG is found in congenital and acquired immunodeficiency diseases and selective IgG subclass deficiencies, such as Bruton type agammaglobulinemia. Decreased IgG concentrations in serum and plasma are seen in protein-losing enteropathies, nephrotic syndrome and through the skin due to burns. Increased IgG metabolism is found in Wiskott-Aldrich syndrome, myotonic dystrophy and with anti-immunoglobulin antibodies.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Age | value (g/L) |
| | 0-1 yr | 2.32-14.11 |
| | 1-3 yr | 4.53-9.16 |
| | 4-6 yr | 5.04-14.64 |
| | 7-9 yr | 5.72-14.74 |
| | 10-11 yr | 6.98-15.6 |
| | 12-13 yr | 7.59-15-49 |
| | 14-15 yr | 7.16 17.11 |
| | 16-19 yr | 5.49-15.84 |
| | + 20 yr | 7-16 |

| | |
|---|---|
| Conversion factors | mg/dL x 0.01 = g/L |
| | g/L x 6.67 = µmol/L |
| | µmol/L x 0.15 = g/L |
| | mg/L x 6.67 = nmol/L |
| | nmol/L x 0.15 = mg/L |

| | |
|---|---|
| Sample stability | 4 mth at room temprature |
| | 8 mth at 2-8 ˚C |
| | 8 mth at -20 ˚C |

# Immunoglobulin M (IgM)

## Summary

Increased polyclonal IgM levels are found in viral, bacterial, and parasitic infections, liver diseases, rheumatoid arthritis, scleroderma, cystic fibrosis and heroin addiction. Monoclonal IgM is increased in Waldenström's macroglobulinemia. Increased loss of IgM is found in protein-losing enteropathies and in burns. Decreased synthesis of IgM occurs in congenital and acquired immunodeficiency syndromes. Due to the slow onset of IgM synthesis, the IgM concentration in serum from infants is lower than in that from adults.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP | |
| Expected value | Age | Value (g/L) |
| | 0-1 yr | 0-1.45 |
| | 1-3 yr | 0.19-1.46 |
| | 4-6 yr | 0.24-2.1 |
| | 7-9 yr | 0.31-2.08 |
| | 10-11 yr | 0.31-1.79 |
| | 12-13 yr | 0.35-2.39 |
| | 14-15 yr | 0.15-1.88 |
| | 16-19 yr | 0.23-2.59 |
| | + 20 yr | 0.4-2.3 |

| | | |
|---|---|---|
| Conversion factors | mg/dL x 0.01 | = g/L |
| | g/L x 1.03 | = µmol/L |
| | g/L x 100 | = mg/dl |
| | µmol/L x 0.971 | = g/L |

| | | |
|---|---|---|
| Sample stability | 2 mth | at room temperature |
| | 4 mth | at 2-8 ˚C |
| | 6 mth | at -20 ˚C |

# Infectious Mononucleosis (IM)

## Summary

The symptoms of IM include fever, fatigue, sore throat, and nodes and can cause liver inflammation (hepatitis) and enlargement of the spleen in very rare cases, heart or central nervous system problems may occur. Diagnosis of IM made based on the presence of heterophile antibodies which belong to the IgM class.

| | |
|---|---|
| Method | Immunoassay |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Negative |
| Sample stability | 2 d at 2-8˚ C |

# Inhibin-A

## Summary

Inhibin-A is elevated in the serum of women carrying fetuses with Down syndrome. In the second trimester of pregnancy, measuring inhibin A in maternal serum in combination with measurements of alpha-fetoprotein and the β subunit of human chorionic gonadotropin, significantly improves the rate of detection of Down's syndrome.

In men, it is a hormone that inhibits FSH production. The inhibin alpha subunit joins either the beta A or beta B subunit to form a pituitary FSH secretion inhibitor.

Measuring Inhibin A has increased the detection of Down syndrome by as much as 8%.

| | | | |
|---|---|---|---|
| Method | ELISA | | |
| TAT | 2d | | |
| Sample type | S | | |
| Expected value | Female (♀): | Age | Value (pg/mL) |
| | | <11 yr | <4.7 |
| | | 11-17 yr: | <97.5 |
| | Male (♂): | <2.0 pg/mL | |
| | Premenopausal: | <97.5 pg/mL | |
| | Postmenopausal: | <2.1 pg/mL | |
| Sample stability | 1 d at room temperature | | |
| | 1 wk at 2-8˚C | | |
| | 1 yr at -20˚C | | |

# Inhibin-B

## Summary

Inhibin B it is correlates negatively with the serum concentration of FSH. Inhibin B is also a marker of ovarian function as well as exocrine testes function. It correlates positively with sperm density, the Johnsen Score, an indicator of the testes volume.

In patients with infertility, measuring inhibin B levels may provide useful information on spermatogenesis and possibly serve as a more direct marker of the spermatogenesis than FSH.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | 2d | |
| Sample type | S | |
| Expected value | 22-418 pg/mL | |
| Sample stability | 2 d | at 2-8˚C |
| | 1 mth | at -20˚C |

# Insulin

## Summary

A disorder in insulin metabolism leads to massive influencing of a number of metabolic processes. A too low concentration of free, biologically active insulin can lead to the development of diabetes mellitus. Possible causes of this include destruction of the β-cells (type I diabetes), reduced activity of the insulin or reduced pancreatic synthesis (type II), circulating antibodies to insulin, delayed release of insulin or the absence (or inadequacy) of insulin receptors. This condition is brought about by inhibition of gluconeogenesis, e.g. as a result of severe hepatic or renal failure, islet cell adenoma, or carcinoma. Reduced glucose tolerance during pregnancy always requires treatment. The clearly elevated risk of mortality for the fetus necessitates intensive monitoring.

| | | |
|---|---|---|
| Remark | Keep samples On Ice, Neither hemolyzed nor lipemic sample. | |
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP/CP | |
| Expected value | 2.6-24.9 μIU/ml | |
| Conversion factors: | μU/mL x 6.945 = pmol/L | |
| | pmol/L x 0.144 = μU/mL | |
| Sample stability | 4 h | at room temperature |
| | 1 d | at 2-8 ˚C |
| | 6 mth | at - 20 ˚C |

# Insulin like growth factor 1 (IGF 1)

## Summary

The measurement of serum IGF-1 is of recognize4d value in children with growth disorders and in the diagnosis and monitoring of acromegaly. IGF-1 concentrations change with :age, nutritional status, body composition and growth hormone secretion. In adults raised levels of IGF-1 are associated with a reduced risk of impaired glucose tolerance, favorable cardiovascular disease risk factors, and a lower incidence of coronary heart disease. Low levels of IGF-1 are associated with liver dysfunction. Pregnancy and late puberty are also associated with elevated serum IGF-1 levels.

| | | |
|---|---|---|
| Remarks | 500 µl, Freeze immediately, Fasting. | |
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Age | Value (ng/mL) |
| | <2 yr | 13.26 - 82.95 |
| | 3-6 yr | 12.29 - 240.57 |
| | 7-10 yr | 19.87 - 540.65 |
| | 11-17 yr | 16.61- 485.12 |
| | Adults | 91- 443 |
| Conversion factors | nmol/L x 7.63 = µg/L | |
| | µg/L x 0.131 = nmol/L | |
| Sample stability | Long term storage at - 20˚C | |

# Insulin Like Growth Factor Binding Protein-3(IGFBP-3)

## Summary

Human T-cell Lymphotropic Viruses (HTLVs) are pathogenic retroviruses that may cause severe haematological and neurological diseases in infected individuals. HTLV-I is known as the etiological agent of Adult T-Cell Leukemia / Lymphoma (ATL), HTLV-Associated Myelopathy / Tropical Spastic Paraparesis (HAM/TSP), and HTLV-associated uveitis. HTLV-II infection has also been associated with leukemia and neurological disease although it is less pathogenic than HTLV-I.

| | | |
|---|---|---|
| Remarks | Fasting. | |
| | Freeze sample immediately. | |
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Age | Value (ng/mL) |
| | <5 yr | 232-6595 |
| | 6-7 yr | 1463-7182 |
| | 8-12 yr | 1281-10000 |
| | 13-17 yr | 2034-5962 |
| | Adults | 1500-5580 |
| Sample stability | 1 d | at room temperature |
| | 1 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Interleukin 6 (IL-6)

## Summary

IL-6 detected in human body fluids in patients with acute bacterial infections.IL-6 production is rapidly induced in the course of acute inflammatory reactions associated with injury, trauma, stress, infection, brain death, neoplasia, and other situations. Sequential measurements of IL-6 in serum or plasma of patients admitted to the ICU (Intensive Care Unit) showed to be useful in evaluating the severity of SIRS (Systemic Inflammatory Response Syndrome), sepsis and septic shock and to predict the outcome of these patients. IL-6 is also useful as an early alarm marker for the detection of neonatal sepsis. IL-6 also plays a role in chronic inflammation e.g. Rheumatoid Arthritis (RA).

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Up To: 7 pg/mL | |
| Sample stability | 5 hr | at room temperature |
| | 1 d | at 2-8 ˚C |
| | 3 mth | at -20 ˚C |

# Iron

## Summary

Iron (non-heme) measurement is used in the diagnosis and treatment of diseases such as iron deficiency anemia, hemochromatosis (a disease associated with widespread deposit in the tissue of the two iron-containing pigments, hemosiderin and hemofuscin, and characterized by pigmentation of the skin), and chronic renal disease. Iron determinations are performed for the diagnosis and monitoring of microcytic anemia (e.g. due to iron metabolism disorders and hemoglobinopathy), macrocytic anemia (e.g. due to vitamin B12 deficiency, folic acid deficiency and drug-induced metabolic disorders of unknown origin) as well as normocytic anemias such as renal anemia (erythropoietin deficiency), hemolytic anemia, hemoglobinopathy, bonemarrow disease and toxic bone marrow damage.

| | |
|---|---|
| Method | Neither hemolyzed nor lipemic sample. |
| TAT | Dy/AB |
| Sample type | S/HP |

Expected value

| Age | Male (μg/dL) | Female (μg/dL) |
|---|---|---|
| <7 Mth | 36-156 | 36-156 |
| 7 Mth-12 yr | 43-184 | 43-184 |
| +12 yr | 59-158 | 37-145 |

| | | |
|---|---|---|
| Sample stability | 1 wk | at room temperature |
| | 3 wk | at 2-8˚c |
| | >1 yr | at -20˚C |

# Lactate (LACT)

## Summary

Anaerobic glycolysis markedly increases blood lactate and causes some increase in pyruvate levels, especially with prolonged exercise. The common cause for increased blood lactate and pyruvate is anoxia resulting from such conditions as shock, pneumonia and congestive heart failure. Lactic acidosismay also occur in renal failure and leukemia. Thiamine deficiency and diabetic ketoacidosis are associated with increased levels of lactate and pyruvate. Lactate levels in CSF are increased in bacterial meningitis, and also occur in hypocapnia, hydrocephalus, brain abscesses, cerebral ischemia and any clinical condition associated with reduced oxygenation of the brain and/or increased intracranial pressure.

| | | |
|---|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. | |
| Method | Colorimetric | |
| TAT | Dy/AB | |
| Sample type | FP/CSF | |
| Expected value | Venous: 8.1-15.3 mg/dL | |
| | Arterial: <11.3 mg/dL | |
| | CSF Neonates: 10-60 mg/dL | |
| | CSF 3-10 d: 10-40 mg/dL | |
| | CSF >10 d: 10-25 mg/dL | |
| | CSF Adult: 10-22 mg/dL | |
| Conversion factors | mmol/L x 9.009 = mg/dL | |
| | mg/dL x 0.111 = mmol/L | |
| Sample stability | 3 d | at room temperature |
| | 3 d | at 2-8 ˚C |
| | 3 d | at - 20 ˚C |

# Lactoferrin

## Summary

Lactoferrin in stool is considered as marker for activity of inflammatorious disorders such like Morbus Crohn, colitis ulcerosa, diverticulitis etc. Furthermore a differential diagnosis is possible between functional bowl disorders and colon irritabile, and for monitoring activity under steroidal therapy.

| | |
|---|---|
| Remarks | Sample dispatch refrigerated. |
| Method | EIA |
| TAT | 3 d |
| Sample type | ST |
| Expected value | < 7.2 µg/g St. |
| Sample stability | 1 h at room temperature |
| | 1 wk at 2-8˚C |
| | 1 wk at 20˚C |

# Lambda Light Chain

## Summary

Measurement of the various amounts of the different types of light chains aids in the diagnosis of multiple myeloma, lymphocytic neoplasms, Waldenström's macroglobulinemia, and connective tissue diseases such as rheumatoid arthritis or systemic lupus erythematosus. Pathological increases of a cell clone lead to elevated formation of monoclonal immunoglobulins or immunoglobulin fragments (free light chains), which bring about a change in the kappa:lambda ratio. A kappa:lambda ratio outside the normal range is indicative of monoclonal gammopathy.

This test encompasses both bound and free immunoglobulins of the light chain type.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | 0.93-2.42 g/L | |
| Conversion factors | mg/dL x 0.01 = g/L | |
| Sample stability | 4 d | at 2-8˚C |
| | 6 mth | at -20˚C |

# Lupus Erythematosus cell (LE cell)

## Summary

LE (Lupus Erythematosus) cells test measures the presence of a special cell found mostly in patients with Systemic Lupus Erythematosus, (SLE) a disease of protean clinical manifestations commonly with rash, arthralagia, fever, anemia, leukopenia, thrombocytopenia, and hypocomplementemia.) Some patients with rheumatoid arthritis, scleroderma, and drug sensitivities (drug-induced lupus erythematosus) also have a positive LE cells test. The antibody may be IgG, IgA, or IgM specificity but is most commonly of IgG class.

| | |
|---|---|
| Remarks | Fresh sample. |
| Method | AGGL |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Negative |
| Sample stability | 2 d at 2-8˚C |

# Lead

## Summary

Lead is a heavy metal found naturally in the environment. In adults, a low level of lead exposure isn't considered dangerous. However, in babies and young kids whose brains are still developing, even a small amount of lead can cause learning disabilities and behavioral problems. At higher levels, lead exposure can cause seizures, coma, and even death.

| | |
|---|---|
| Remarks | Fresh sample. |
| Method | Electrochemistry |
| TAT | Dy/A |
| Sample type | EWB/HWB |
| Expected value | Up To 10 µg/dL<br>Toxic Level: >25 µg/dL |
| Sample stability | 1 d at room temperature |

# Leishmania Abs (IgA ,IgG, IgM)

## Summary

Leishmania infantum is a causal agent of Kala-azar or visceral Leishmania and oriental Sore. Kala-zara is a serious disease characterized by fever, splenomegaly, anemia, weight loss and leukopenia. The incidence of L.infantum infection in AIDS patients is very high. The determination of IgG antibodies is a valuable indicator in the diagnosis of past infection, whereas IgM/IgA antibodies will be found in recent infection.

| | |
|---|---|
| Method | IF |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Negative: <1:40 |
| Sample stability | 1 wk at 2-8˚C |

# Leisteria Monocytogenes Abs (1/2a, 4b) (IgG, IgM)

## Summary

Listeria is typically a food-borne organism, transmitted through soil and water. A person can also ingest listeria by eating certain foods, such as deli meats and cold cuts, soft-ripened cheese, milk, undercooked chicken, uncooked hot dogs, shellfish, and coleslaw made from contaminated cabbage.

The most common clinical manifestation is diarrhea. A mild presentation of fever, nausea, vomiting, skin infections, septicemia and Influenza- like symptoms, difficulty breathing, and poor feeding, diarrhea may resemble a gastrointestinal illness, in case of infection during pregnancy it can lead to the death of the foetus inside the uterus.

The determination of IgG antibodies is a valuable indicator in the diagnosis of past infection, whereas IgM antibodies will be found in recent infection.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:100 |
| Sample stability | 2 wk at 2-8˚C |

# Lipase (LIP)

## Summary

The lipase activity determination has gained increasing international recognition because of its high specificity and rapid response. After acute pancreatitis the lipase activity increases within 4-8 hours, reaches a peak after 24 hours and decreases after 8 to 14 days. However, there is no correlation between the lipase activity determined in serum and the extent of damage to the pancreas.

| | |
|---|---|
| Method | Colorimetric |
| TAT | Dy/AB |
| Sample type | S/HP |
| Expected value | 13-60 U/L |
| Conversion factors | U/L x 0.0167 = μkat/L |
| Sample stability | 1 wk at room temperature |
| | 1 wk at 2-8˚c |
| | 1 yr at -20˚C |

# Lipoprotein (A)

## Summary

High lipoprotein (A) concentrations in serum correlates with premature manifestation of atherosclerosis and strokes. When lipoprotein (A) concentrations exceeds 0.30 g/L, the coronary risk is approximately doubled. In combination with elevated LDL-cholesterol concentrations, the risk increases approximately six-folds. An elevated lipoprotein (A) level is considered to be the most sensitive parameter for the development of coronary heart disease, irrespective of other plasma lipoproteins. Lipoprotein (A) should be determined together with total cholesterol, HDL-cholesterol and LDL-cholesterol as well as triglycerides when assessing the total arteriosclerotic risk. Lipoprotein (A) levels should be determined in patients suffering from dyslipoproteinemia, diabetes mellitus, renal failure, and cardiovascular or cerebrovascular disorders, as well as in premature onset of arteriosclerosis.

| | | |
|---|---|---|
| Remark | Fasting | |
| Method | Turbidimetric | |
| TAT | 3 d | |
| Sample type | S | |
| Expected value | Male (♂): (n = 154): 0.09 g/L (9 mg/dL) | |
| | Female (♀): (n = 187): 0.11 g/L (11 mg/dL) | |
| Conversion factors | g/L × 100 = mg/dL | |
| | g/L × 1000 = mg/L | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8 ˚C |
| | 3 mth | at - 20 ˚C |

# Lithium (Li)

## Summary

Serum Lithium concentration is measured , essentially, to ensure compliance and to avoid toxicity. Early symptoms of intoxication include apathy, sluggishness, drowsiness, lethargy, speech difficulties, irregular tremors, myoclonic twitchings, muscle weakness and ataxia. Levels higher than 1.5 mmol/L (12 hours after a dose) indicate a significant risk of intoxication

| | | |
|---|---|---|
| Method | Colorimetric | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Male (♂): 0.6-1.2 mmol/L | |
| | Female (♀): 0.4-1.0 mmol/L | |
| Conversion factors | mmol/L x 0.6941 = mg/dL | |
| | mg/dL x 1.441 = mmol/L | |
| Sample stability | 1 wk | at room temperature |
| | 1 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Liver / Kidney Microsomal Abs -1 (Lkm-1)

## Summary

The LKM antibody is quite rare but can be seen in patients with various types of hepatitis. The antibodies are usually found in autoimmune chronic active hepatitis and viral hepatitis.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:20 | |
| Sample stability | 4-7 d | at 2-8˚C |
| | 3-6 mth | at -20˚C |

# Luteinizing Hormone (LH)

## Summary

Determination of the LH concentration is used in the elucidation of dysfunctions within the hypothalamus-pituitary-gonads system. The determination of LH in conjunction with FSH is utilized for the following indications: congenital diseases with chromosome aberrations (e.g. Turner's syndrome), Polycystic Ovaries (PCO), clarifying the causes of amenorrhea, menopausal syndrome, and suspected Leydig cell insufficiency.

| | |
|---|---|
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S/HP/EP |
| Expected value | Children 0.2-1.4 mIU/mL |
| | Male (♂) 1.7-8.6 mIU/mL |
| | Follicular 2.4-12.6 mIU/mL |
| | Ovulation 14-95.6 mIU/mL |
| | Luteal 1-11.4 mIU/mL |
| | Postmenopause 7.7-58.5 mIU/mL |
| Sample stability | 1 d at room temperature |
| | 3 d at 2-8˚C |
| | 1 yr at -20˚C |

# Magnesium (Mg)

## Summary

Increased serum magnesium concentrations occur in renal failure, acute diabetic acidosis, dehydration, or Addison's disease. Hypermagnesemia has a depressing effect on the central nervous system, causing general anesthesia and respiratory failure. It alters the conduction mechanism of the heart, causing cardiac arrest. Hypomagnesemia may be observed in chronic alcoholism, malabsorption, severe diarrhea, acute pancreatitis, diuretic therapy, prolonged parenteral fluid therapy without magnesium supplementation, and kidney disorders such as glomerulonephritis and tubular reabsorption defects. Decreased serum magnesium concentrations may result in tetany, convulsions, and cardiac arrhythmias.

| | | |
|---|---|---|
| Method | Colorimetric | |
| TAT | Dy/AB | |
| Sample type | 24U/ S/HP | |
| Expected value | S/HP: 1.58-2.55 mg/dL | |
| Conversion factors | mmol/L x 2.43 = mg/dL | |
| | mg/dL x 0.411 = mmol/L | |
| | mval/L x 0.5 = mmol/L | |
| | mval/L x 1.22 = mg/dL | |
| | mval/L = mEq/L | |
| Sample stability in 24U | 3 d | at room temperature |
| | 3 d | at 2-8 ˚C |
| | 1 yr | at - 20 ˚C |
| Sample stability in S/HP | 1 wk | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 1 yr | at - 20 ˚C |

# Malaria Abs

## Summary

Malaria is caused by a parasite called Plasmodium, which is transmitted via the bites of infected mosquitoes. In the human body, the parasites multiply in the liver, and then infect red blood cells. Symptoms of malaria appear seven days or more (usually 10–15 days) after the infective mosquito bite. The first symptoms – fever, headache, chills and vomiting – may be mild and difficult to recognize as malaria. If not treated within 24 hours, P. falciparum malaria can progress to severe illness often leading to death. Children with severe malaria frequently develop one or more of the following symptoms: severe anaemia, respiratory distress in relation to metabolic acidosis, or cerebral malaria.

In adults, multi-organ involvement is also frequent. In malaria endemic areas, persons may develop partial immunity, allowing asymptomatic infections to occur. For both P. vivax and P. ovale, clinical relapses may occur weeks to months after the first infection, even if the patient has left the malarious area. These new episodes arise from “dormant” liver forms (absent in P. falciparum and P.malariae), and special treatment – targeted at these liver stages – is mandatory for a complete cure.

There are four parasite species that cause malaria in humans: Plasmodium falciparum, Plasmodium vivax, Plasmodium malariae, Plasmodium ovale. Plasmodium falciparum and Plasmodium vivax are the most common.Plasmodium falciparum is the most deadly. Transmission Malaria is transmitted exclusively through the bites of Anopheles mosquitoes. The intensity of transmission depends on factors related to the parasite, the vector, the human host, and the environment.

The presence of all antibodies isotypes (IgG/IgM/IgA) is specific to detect Plasmodium falciparum, Plasmodium vivax simultaneously in human serum, plasma or whole blood.

| | |
|---|---|
| Method | ICT |
| TAT | Dy/AB |
| Sample type | EWB |
| Expected value | Negative |
| Sample stability | 3 d at 2-8˚C |

# Measles Abs (IgA ,IgG,IgM)

## Summary

Measles (Rubeola ) is a highly contagious viral disease that can be fatal. The disease produces fever (temperature > 101 F [38.3 C]), a generalized rash that last greater than three days, cough, runny nose(coryza), and red eyes (conjunctivitis). The complications of measles that result in most deaths include pneumonia and inflammation of the brain (encephalitis). The determination of Measles IgM/IgA antibodies is intended for use as an aid in the diagnosis of recent infection with Measles Virus while IgG antibodies used for the diagnosis of primary infection and as a determination of immune status.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:10 | |
| Sample stability | 2d | at room temperature |
| | 1 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Mercury (Hg)

## Summary

Mercury steam can develop at room temperature. Both inhaling and oral intake is toxic for humans. Acute poisoning presents with headache, dizziness, trembling, sight and hearing disorders, mucous membrane inflammations, stomach and intestinal colic, metal taste in the mouth, bloody diar¬rhea, vomiting, kidney failure and blood pressure drop and collapse. A dose of approximately 150-300 mg of mercury is lethal.

Toxicity is based on enzyme blocking. In addition, the kidneys and neurons are mainly damaged. In chronic mercury intoxication (permanent exposure to small quantities) the symptoms manifest as tiredness, head and rheumatic pain, inflammations in the mucous membrane of the mouth, trembling and disturbances in the central nervous system up to death. Amalgam fillings have been controversially discussed.

| | |
|---|---|
| Method | AAS |
| TAT | 10- 14 d |
| Sample type | EWB |
| Expected value | < 5.00µg/L |
| | BAT < 50µg/L |
| Sample stability | 2 wk at 2- 8˚C |

# Methemoglobin (Met-Hb)

## Summary

Methemoglobin contains iron III (Hemiglobin) and can not transfer O2. The physiological met-Hb content in blood is less than 1.5% of the total hemoglobin concentration. Cyanosis is visible in increased Met-Hb concentrations. The cause of methemoglobinemia is often due to medicaments or toxic substances. Symptoms usually appear when the met-Hb reaches values of more than 40% of the complete Hb.

Methemoglobin levels increased by intake of methemoglobin generating drugs, such like phenacetin, sulfonamids, chinin, PAS, nitrites, nitrogen oxides, arsenic hydrogen, aromatic nitro- and amino compounds, chlorates, bromates.

| | |
|---|---|
| Method | PHO |
| TAT | 7-10 d |
| Sample type | EWB |
| Expected value | < 1.00 % |
| Sample stability | 8 h at 2-8 ˚C |

# Mucopolysaccharides

## Summary

Mucopolysaccharidoses are a group of metabolic disorders caused by the absence or malfunctioning of lysosomal enzymes needed to break down molecules called glycosaminoglycans - long chains of sugar carbohydrates in each cell that help build bone, cartilage, tendons, corneas, skin and connective tissue. Glycosaminoglycans (formerly called mucopolysaccharides) are also found in the fluid that lubricates our joints.

People with mucopolysaccharidosis, either do not produce enough of one of the 11 enzymes required to break down these sugar chains into simpler molecules, or they produce enzymes that do not work properly. Over time, these glycosaminoglycans collect in the cells, blood and connective tissues. The result is permanent, progressive cellular damage which affects appearance, physical abilities, organ and system functioning, and, in most cases, mental development.

Mucopolysaccharidoses test is used to monitor Glycosaminoglycans (GAGs) in patients previously diagnosed with Mucopolysaccharidosis (MPS).

| | |
|---|---|
| Method | SPHO |
| TAT | Dy/A |
| Sample type | US |
| Expected value | Negative |
| Sample stability | 1 mth at -20 ˚C |

# Mumps Abs (IgA , IgG,IgM)

## Summary

Mumps is an acute, self-limited, systemic viral illness characterized by the swelling of one or more of the salivary glands, typically the parotid glands, affected salivary glands show edema and lymphocytic infiltration. The determination of Mumps IgM/IgA antibodies is intended for use as an aid in the diagnosis of recent Mumps Virus while IgG antibodies used for the diagnosis of primary infection and as a determination of immune status.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:100 | |
| Sample stability | 1 wk | at 2-8˚C |
| | Long term storage | at -20˚C |

# Muscle-Specific Receptor Tyrosinkinase Abs (Anti MuSK)

## Summary

The measurement of autoantibodies to muscle- specific receptor tyrosine kinas (MuSK) may be useful in the assessment and management of patients with Acetylcholine Receptor Antibody (AChRAb)- negative Myasthenia Gravis (MG).

| | | |
|---|---|---|
| Method | RIA | |
| TAT | 10-14 d | |
| Sample type | S | |
| Expected value | Negative: < 0.5 nmoL/L | |
| Sample stability | 2 wk | at 2-8˚C |
| | 6 mth | at -20˚C |

# Mycobacterium tuberculosis (TB)

## Summary

TB infection usually occurs initially in the upper part (lobe) of the lungs. The body's immune system, however, can stop the bacteria from continuing to reproduce. Thus, the immune system can make the lung infection inactive (dormant). On the other hand, if the body's immune system cannot contain the TB bacteria, the bacteria will reproduce (become active or reactivate) in the lungs and spread elsewhere in the body.It may take many months from the time the infection initially gets into the lungs until symptoms develop. The usual symptoms that occur with an active TB infection are a generalized tiredness or weakness, weight loss, fever, and night sweats. If the infection in the lung worsens, then further symptoms can include coughing, chest pain, coughing up of sputum (material from the lungs) and/or blood, and shortness of breath. If the infection spreads beyond the lungs, the symptoms will depend upon the organs involved.

### TB Abs. (Screen ,IgM,IgA)

TB antibodies screening indicates for Persons at increased risk for latent Mycobacterium tuberculosis infection (e.g., recent immigrants, injection-drug users, and residents and employees of prisons and jails), persons at low risk for latent M. tuberculosis infection but whose future activity might place them at increased risk (e.g., health-care workers and military personnel), persons who are not considered to have an increased probability of M. tuberculosis infection but who require testing for other reasons.

| | | |
|---|---|---|
| Method | ELISA/ICT | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative | |
| Sample stability | 2d | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

## TB Culture

TB culture indicates for suspicion of TB, persisting teherapy resistant pneumonia, hemoptysis clarification pleural effusion.

| | |
|---|---|
| Remarks | Sample on 3 consecutive days. |
| Method | Culture |
| TAT | 40 d |
| Sample type | SP |
| Normal value | Negative |
| Sample stability | 3 d at 2-8˚C |
| | 2 wk at -20˚C |

## TB DNA

Highly recommended test for the best diagnosis of TB infection

| | |
|---|---|
| Method | PCR |
| TAT | 2 d |
| Sample type | SP/BW/US |
| Expected value | Not detected |
| Sample stability | 1 mth at room temperature |
| | 1 mth at 2-8˚C |

# Mycophenolic Acid (MPA)

## Summary

Mycophenolic acid is an immunosuppressant drug used in tissue transplants. It prevents graft rejection by the host's immune system. Monitoring its level is essential to optimize therapeutic effects, avoid toxicity, and assure compliance. Since mycophenolic acid can lower white blood cell counts and cause anemia, a Complete Blood Count (CBC) will frequently be ordered along with the mycophenolic acid test to evaluate the body's blood cell status.

| | | |
|---|---|---|
| Remarks | 2 mL of the sample. Just before dose. | |
| Method | HPLC | |
| TAT | 3 d | |
| Sample type | S | |
| Expected value | Refer To Report | |
| Sample stability | 6 wk | at room temperature |
| | 6 wk | at 2-8 ˚C |
| | 11 mth | at -20 ˚C |

# Mycoplasma pneumoniae Abs (IgA,IgG,IgM)

## Summary

Mycoplasma pneumoniae is a very small bacterium causes the disease mycoplasma pneumonia which is a contagious disease of children and young adult characterized by 9 to 12 day incubation period and followed by symptoms of an upper respiratory infection, dry cough, and fever.

If IgG antibody to Mycoplasma pneumoniae is detected, it indicates a past infection, whereas IgM/IgA indicates a current infection to the disease.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:64 | |
| Sample stability | 2 d | at room temperature |
| | 2wk | at 2-8 ˚C |
| | 1 yr | at - 20 ˚C |

# Myelin Associated Glycoprotein Abs (Anti-MAG)

## Summary

Elevated levels of Myelin-Associated Glycoprotein (MAG) has been implicated in inhibition of nerve regeneration in the Central Nervous System(CNS), including multiple sclerosis, subacute sclerosing panencephalitis, Guillain-Barré syndrome, chronic relapsing polyradiculoneuritis and carcinomatous polyneuropathy and also some patients with autoimmune diseases such as collagen diseases and myasthenia gravis.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Myoglobin (MYO)

## Summary

Myoglobin is liberated from damaged heart muscle cells such as occurs during acute myocardial infarction. An increase in myoglobin concentrations in blood can generally be detected 2 to 4 hours after the onset of pain, which is earlier than other cardiac markers such as CK, CK-MB or Troponin. Depending on the therapeutic reperfusion measures taken, the myoglobin concentration reaches its maximum value after 4 to 12 hours and then decreases relatively rapidly to normal levels due to renal elimination (biological half-life: approx. 15 minutes). A very rapid increase in the concentration of myoglobin occurs when therapeutic intervention is successful. The gradient of the concentration increase can be taken as an indication of the success of thrombolysis. The myoglobin determination is of particular value in exclusion diagnosis for myocardial infarction: if there is no increase in the myoglobin concentration 6 hours after the onset of pain and after a repeat determination within 4 hours, then acute myocardial damage can essentially be excluded. Increases in concentration of myoglobin not due to infarction may be a result of muscle trauma, crush syndrome, myopathy, muscle strain/stress, shock, rhabdomyolysis or decreased elimination due to renal failure.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | HWB | |
| Expected value | Male (♂): 16-76 ng/mL | |
| | Female (♀): 7-64 ng/mL | |
| Conversion factors | µg/L x 0.0571 = nmol/L | |
| | µg/L = ng/mL | |
| Sample stability | 2 d | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 3 mth | at -20 ˚C |

# Neisseria gonorrhoeae

## Summary

Neisseria gonorrhoeae is a species of Gram-negative cocci. it is pathogenic to humans who are its only natural host, and it is responsible for the disease Gonorrhea which is a sexually transmitted disease of worldwide importance. Neisseria gonorrhoeae can grow and multiply easily in warm- moist areas, including the cervix, uterus, and fallopian tubes in women, and in the urethra in women and men. The bacterium can also grow in the mouth, throat, eyes, and anus.

Neisseria gonorrhoeae typically infects the mucous membranes causing infections such as urethritis, cervicitis, salpingitis, pelvic inflammatory disease, proctitis, conjunctivitis and pharyngitis.

| | |
|---|---|
| Remarks | Urine first catch, not urinated for at least 2 h. |
| Method | PCR |
| TAT | 2 d |
| Sample type | US/EnS/UrS |
| Expected value | Not detected |
| Sample stability | 2 d at 2-8˚C |

# Neuron-Specific Enolase (NSE)

## Summary

(NSE) or γ-enolase, are primarily detectable in high concentrations in neurons and neuro-endocrine cells as well as in tumors originating from them. NSE is described as the marker of first choice in the monitoring of small cell bronchial carcinoma.

Neuroblastoma: NSE serum values above 30 ng/mL are found in 62 % of the affected children.

Apudoma: In 34 % of the cases elevated NSE values (> 12.5 ng/mL) are found in serum.

Seminoma: 68-73 % of the patients have a clinically significant NSE elevation.

Other tumors: Non-pulmonary malignant diseases show values above 25 ng/mL in 22 % of the cases (carcinomas in all stages). Brain tumors such as glioma, meningioma, neurofibroma, and neurinoma are only occasionally accompanied by elevated serum NSE values.

Benign disease: Elevated serum NSE concentrations (> 12 ng/mL) have been found in patients with benign pulmonary diseases and cerebral diseases.

Elevated values have been found in cerebrovascular meningitis, disseminated encephalitis, spinocerebellar degeneration, cerebral ischemia, cerebral infarction, intracerebral hematoma, subarachnoid hemorrhage, head injuries, inflammatory brain diseases, organic epilepsy, schizophrenia, and Jakob-Creutzfeld disease.

| | | |
|---|---|---|
| Remarks | Centrifuge in 1 hr max, keep sample On Ice. | |
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | <17 ng/mL | |
| Sample stability | 6 h | at room temperature |
| | 1 d | at 2-8 ˚C |
| | 3 mth | at -20 ˚C. |

# New Born Screening

## Summary

Newborn screening is intended as a public health program to identify infants with treatable conditions before they present clinically, or suffer irreversible damage.

### Remarks

- The ideal time for collection is between 36-48 hours of age.

- Newborn screenings are not valid after 14 days especially for screening beta-oxidation disorders (MCADD, LCHADD, and VLCADD). If the newborn is discharged before 36 hours of age, a repeated sample should be collected as soon as possible.

- Critically ill newborns should be screened before 36 hours of age. A pre-transfusion specimen is essential even before 36 h. The screening should be controlled 3 days after the last transfusion.

- If an initial screen is obtained prior to transfusion when the newborn is older than 36 hours of age, a repeat specimen is not necessary.

- Cord Blood is unacceptable for newborn screening.

| | |
|---|---|
| Method | LCMS/ PHO/ EIA |
| TAT | 2 d |
| Sample type | DB |

## The following examinations are recommended for newborn screening:

- 17-Hydroxyprogesterone (17-OH Prog.)

Hydroxyprogesterone is directly synthesized from progesterone or indirectly from 17α-OH pregnelonone. As an intermediate product of glucocorticoids and sexual hormone pathway, hydroxyprogesterone is accumulated in inherited enzyme deficiencies (hydroxylase deficiencies). Most common is the congenital Adrenogenital Syndrome (AGS) or Congenital Adrenal Hyperplasia (CAH) due to 21-hydroxylase deficiency.

Clinical symptoms: In the classic AGS, newborn girls show a virilized outer genital. The virilism consists out of slight clitoris enlargement as well as the formation of a pseudopenis. The inner genital, however, is female. Boys are born with normal genitalia. Without treatment a faster infantile growth can be observed in girls and boys. The children are too tall for their age. However, an accelerated closing of epiphysal fissures occurs. As a consequence, early growth interruption occurs resulting in dwarfism in adolescence.

In boys, premature pseudo puberty is observed at the age of 4 - 5 years. Boys show secondary pubic hair growth and increased penis size, however the testes are still normal for age. Girls show early pubic and general hair growth (hirsutism). Acne, less breast development, menstruation disturbances and infertility are further characteristics in girls.

The typical AGS with salt loss is characterized by additional problems. The infants (girls and boys) can present with “life-threatening salt-losing crisis” within the first weeks of life. They vomit, are apathetic and lose weight. Serious disturbances in electrolytes with acidosis might occur.

The non-typical “late onset AGS” in girls does not present with any physical abnormalities at birth. Otherwise the symptoms correspond to those of the simple AGS, however being less distinct. Early pubic hairs, increased hair-growth, acne, and menstruation disturbances as well as difficulties in conception are observed in girls or women.

Also boys present with early pubic hair growth and suffer from acne. In both women and men, insignificant reduced growth is observed. Biochemical changes are also found in the non-classic “cryptic AGS” but without presenting any clinical symptoms.

• Biotinidase

Biotinidase is essential for the recycling of the vitamin biotin; Clinical symptoms of biotinidase deficiency (“3 H-syndrome”) appear after weeks or months: developmental retardation, hypotonia, ataxia. Later on features of this disorder include neurological symptoms, such as developmental disabilities and seizures, and cutaneous symptoms, such as hair loss and skin rash, seborrhoic dermatitis, ceratoconjunctivitis, alopecia, candidiasis and occasional acidosis (metabolic acidosis; overlapped by respiratoric alcalosis), cramps, laryngeal stridor, deafness.

| Normal value | > 30 % |
|---|---|

• Glactose

Classical galactosemia (also known as galactosemia type I) is a disorder that affects how the body processes the simple sugar galactose. If infants with classical galactosemia are not treated promptly with a low-galactose diet, life-threatening complications appear within a few days after birth. Affected infants typically develop feeding difficulties, lethargy, failure to thrive, jaundice, liver damage, and abnormal bleeding. Other serious complications of this condition can include sepsis and shock. Classical galactosemia is caused by mutations in the GALT gene; it has an autosomal recessive pattern of inheritance.

| Normal value | < 15.0 mg/dl |
|---|---|

• Phenylalanine (PKU)

Babies with this disorder cannot process a substance called phenylalanine that is found in almost all food. Without treatment, phenylalanine builds up in the bloodstream and causes brain damage and mental retardation. When PKU is detected early, mental retardation can be prevented by feeding the child a special diet.

| Normal value | <129µmol/L |
|---|---|

• Glucose-6-Phosphat-Dehydrogenase( G6PD)

G6PD deficiency can result in neonatal jaundice and in life threatening reactions to several medications, foods and infections. Babies with G6PD deficiency appear normal at birth. They may experience neonatal jaundice and hemolysis that can be so serious as to cause neurologic damage or even death. Barring such severe complications in the newborn period, infants with G6PD deficiency generally experience normal growth and development.

Exposure to certain antimalarial drugs and sulfonamides, infection stress (such as upper respiratory or GI infections), environmental agents (e.g. moth balls), and eating certain foods (e.g. fava beans), each of which impact the patient's ability to handle oxidative reactions, can cause acute hemolytic anemia.

| Normal value | 4.6-20.0 U/g Hb |
|---|---|

• Thuyroid Stimulating Hormone (TSH)

The innate (primary) hypothyreoidism is a result of dysgenesis and affects 1 in 4000 newborns. It occurs 3 times more frequently in girls than in boys. Early symptoms: drinking weakness, muscular hypotonia, icterus prolongatus, open small fontanel. Symptoms usually appear later, from 8-12th week: myxoedema, blunt facial expression, macroglossia, wide open large fontanel, broad flat nose, scanty growth of hair, cool dry skin, constipation, muscular hypotonia, umbilical hernia, delayed psychomotoric development. Substitution therapy is required.

| Normal value | Up To 10 µIU/mL |
|---|---|

• Haemoglobinopathies

Any condition that results in the production of abnormal hemoglobin is included under the broad category of hemoglobinopathies, HbS-variants, newborns with HbC, D-Punjab or E-variants, less common beta-chain variants, variants of gamma/alpha-chains, alpha-thalassemia (alpha-chain deletions (HbH, Hb Bart, Hb Lepore), beta-thalassemia, The most well known condition in this group is sickle cell disease.

Early identification of individuals with sickle cell disease and other hemoglobinopathies allows treatment to be initiated in a timely fashion.

| Normal value | Refer To Report |
|---|---|

• Trypsin (IRT)

Immune Reactive Trypsin (IRT) is a pre-screening test for cystic fibrosis which measured from dried blood spot is 2-5 times higher in neonates with CF than normal controls. Levels decrease after 1-2 months. Positive results should be confirmed by DNA analysis of the CF gene (mutation Delta F508)

| Remarks | | |
|---|---|---|
| DB on filterpaper, store and dispatch at room temperature, not freezing, thermal effects can reduce enzyme activities. | | |
| Normal value | 1 - 20 d | < 60.0 ng/ml |
| | > 20 d | < 45.0 ng/ml |

• Amino Acid Disorders

Babies born with one of these disorders cannot breakdown certain waste products from their blood, such as amino acids or ammonia. This can lead to problems with the eyes, skin or general development, liver failure, coma or death if untreated. Treatment can range from special diets to liver transplantation and special medications. A baby with an amino acid disorder must have regular medical care by an experienced physician

| Normal value | Refer To Report |
|---|---|

The Newborn amino acids Screening Program will screen for:

- Argininosuccinic Acidemia (ASA, argininosuccinase)

People with ASA may appear normal at birth. After a few days of life, a newborn with ASA may develop poor feeding, lack of energy, vomiting, problems breathing, or seizures. If left untreated, brain damage, coma, and death will occur. Many symptoms of ASA can be prevented by immediate treatment and lifelong management.

- Citrullinemia (CIT)

People with CIT may appear normal at birth. After a few days of life, a newborn with CIT may develop poor feeding, lack of energy, vomiting, problems breathing, or seizures. If left untreated, brain damage, coma, and death will occur. Many symptoms of CIT can be prevented by immediate treatment and lifelong management.

- Homocystinuria (HCY)

People with HCY may appear normal at birth. If untreated, a person with HCY will develop progressive vision problems, tall stature, slender build, scoliosis, mental retardation or developmental delay, seizures, and an increased risk of stroke can occur. Many symptoms of HCY can be prevented by immediate treatment and lifelong management.

- Hypermethioninemia (MET)

People with MET usually have no symptoms. There have been reports of people with MET having foul breath, and having problems with the insulation surrounding the brain.

- Maple Syrup Urine Disease (MSUD)

Babies with MSUD are missing an enzyme needed to process three amino acids that are essential for the body's normal growth. When these are not processed properly, they can build up in the body, causing urine to smell like maple syrup or sweet, burnt sugar. These babies usually have little appetite and are extremely irritable. If not detected and treated early, MSUD can cause mental retardation, physical disability, and even death.

- Tyrosinemias (TYR-I, TYR-II)

People with TYR-I and TYR-II appear normal at birth. If left untreated, people with TYR-I and TYR-II will develop eye problems, blisters on the hands and feet, developmental delays, and behavioral problems.

- Phenylalanine (PKU)

Babies with this disorder cannot process a substance called phenylalanine that is found in almost all food. Without treatment, phenylalanine builds up in the bloodstream and causes brain damage and mental retardation. When PKU is detected early, mental retardation can be prevented by feeding the child a special diet.

Organic Acid Disorders

Organic acid disorders are a group of inherited metabolic conditions. Each organic acid disorder is associated with a specific enzyme deficiency that causes the accumulation of organic acids in blood and urine. The accumulated compounds or their metabolites are toxic, resulting in the clinical features of these disorders.

| Normal value | Refer To Report |
|---|---|

An elevated level of a particular acylcarnitine may indicate the possibility of one of several different organic acid disorders which include:

- 3-Hydroxy-3-Methylglutaryl-CoA Lyase Deficiency (HMG-CoA-Lyase-Deficiency)

HMG lyase deficiency occurs when an enzyme, called “HMG CoA lyase”, is either missing or not working properly. This enzyme has two jobs. The first is to help break down leucine. Leucine is found in all foods that contain protein. The second job is to help the body make ketone bodies from stored fat. When children with this condition eat food containing leucine, harmful substances build up in the blood. In addition, children with HMG lyase deficiency can’t make ketone bodies from stored fat like most people. So, when they don’t eat for a long period of time, they can develop low blood sugar (hypoglycemia) and serious health problems.

Therapy: Avoid going a long time without food, Low-leucine diet, including medical foods and formula, Medications (Lcarnitine)

- Glutaric AciduriaType I (GA-1)

GA-1 occurs when an enzyme called “glutaryl-CoA dehydrogenase” is either missing or not working properly. This enzyme’s job is to break down a substance called glutaryl-CoA. Glutaryl-CoA is made when the amino acids lysine, hydroxylysine and tryptophan are processed. It cannot be removed and it causes glutaric acid and other harmful substances build up in the blood and cause problems. Lysine and tryptophan are found in all foods that contain protein. Babies with GA-1 are usually healthy at birth, although many are born with a larger than average head size. Other symptoms usually start between two months and four years of age. GA-1 causes episodes of severe illness called metabolic crises. Some of the first symptoms are poor appetite, extreme sleepiness or lack of energy, irritability, jitteriness, nausea, vomiting, low muscle tone (floppy muscles and joints), muscle weakness. If untreated, other symptoms then follow: tics or spasms of the muscles, swelling of the brain or blood in the brain, coma, sometimes leading to death.

Therapy: Medication L-caritine sometimes glucose, insulin, avoid going a long time without food, Food plan, including medical foods and formula, Low-protein (lysine and tryptophan) diet, Medical foods and formula, Regular blood tests.

- Isobutyryl-CoA Dehydrogenase Deficiency (IBD)

Less than 30 people have been reported in the medical literature, and most people with IBD have no symptoms. IBD deficiency occurs when an enzyme, called “isobutyryl-CoA dehydrogenase”, is either missing or not working properly. This enzyme’s job is to help break down valine. When a child with IBD deficiency eats food containing valine, harmful substances build up in the blood and cause problems. Valine is found in all foods that contain protein. IBD deficiency is very rare and little is known about the effects. Symptoms included are enlarged, weakened heart, anemia, poor growth and low carnitine levels.

Therapy: Medications L-carnitine, Avoid going a long time without food, Low-valine food plan (including medical foods)

- Isovaleric Aciduria (IVA)

IVA occurs when an enzyme, called “isovaleryl-CoA dehydrogenase”, is either missing or not working properly. This enzyme’s job is to help break down a substance called “isovaleryl-CoA”. It is made in the body when the amino acid leucine is broken down. When a child with IVA eats food containing leucine, a substance called isovaleric acid builds up in the blood and causes problems. Leucine is found in all foods that contain protein. If not treated, many babies die during their first metabolic crisis. In those who survive, repeated episodes of metabolic crisis can cause brain damage. This can result in life-long learning problems or mental retardation.

Therapy: Low-leucine/ low-protein diet, medical foods and formula, Medications (Glycine, L-carnitine, bicarbonate, glucose)

- 2-Methylbutryl-CoA Dehydrogenase Deficiency (2MBCD)

2MBCD deficiency occurs when an enzyme, called “2-methylbutyryl-CoA dehydrogenase (2-MBCD)”, is either missing or not working properly. This enzyme’s job is to help break down isoleucine. When a child with 2MBCD deficiency eats food containing isoleucine, harmful substances may build up in the blood. Isoleucine is found in all foods that contain protein. If not treated, episodes of metabolic crisis can cause brain damage. This can lead to life-long learning problems or mental retardation.

Therapy: Avoid going a long time without food, Low-protein diet, including medical foods and formula, Medications (Lcarnitine)

- 3-Methylcrotonyl-CoA Carboxylase Deficiency (3MCC)

3MCC deficiency occurs when an enzyme, called “3-methylcrotonyl CoA carboxylase (3MCC)”, is either missing or not working properly. This enzyme’s job is to help break down leucine. When a child with 3MCC deficiency eats food containing leucine, harmful substances may build up in the blood and cause problems. Leucine is found in all foods with protein. If a metabolic crisis is not treated, it could result in death. In surviving babies and children, repeated episodes of metabolic crisis can cause brain damage. This can lead to life-long learning problems or mental retardation.

Therapy: Low-leucine diet, including medical foods and formula, Medications (L-carnitine).

- 3-Methylglutaconyl-CoA Hydratase Deficiency

3-Methyl Glutaconyl-CoA Hydratase is an enzyme involved in the metabolism of the amino acid Leucine. It is located in mitochondria along with other associated enzymes of leucine catabolism. Deficiency of the enzyme leads to impaired leucine breakdown and massive excretion of 3-methylglutaconic acid. The gene has been cloned and some mutations identified in affected patients. Few patients have been described with this disorder, but the disease seems to have a wide range of clinical severity. Some patients develop acute lifethreatening ca

diopulmonary symptoms soon after birth, whereas others have a more chronic picture with psychomotor retardation, hypotonia, failure to thrive, microcephaly, seizures, and spasticity. Some patients have acute episodes of vomiting, metabolic acidosis and lethargy progressing to coma. Carnitine levels are variably

low. Recurrent acidosis is occasionally seen with prolonged fasting and/or intercurrent illness. Speech retardation was the isolated neurological manifestation in one family.

- Methylmalonyl-CoA Mutase Deficiency (MMA)

Methylmalonic Acidemia can result from several different genetic disorders, including Methylmalonic-CoA mutase deficiency and defects of enzymes in cobalamin (vitamin B12) metabolism. Multiple DNA mutations for MMA have been identified. Because of the dependence of Methylmalonyl-CoA Mutase activity upon cobalamin metabolism and function, the different defects producing MMA have a similar clinical presentation. The clinical symptoms of methymalonic leucopen as recurrent vomiting, dehydration, respiratory distress, muscle hypotonia, and lethargy that can lead to coma and death is often seen in the first week of life. Metabolic acidosis is pronounced. Ketoacidosis, hyperglycinemia, hypoglycemia, and hyperammonemia are often found, along with leucopenia, thrombocytopenia, and anemia. This same scenario can present later in the first month of life, manifesting as failure-to-thrive and mental retardation. All patients are reportedly susceptible to infection. A long-term complication of MMA is renal failure.

- Some Adenosylcobalamin Synthesis Defects

Intracellular cobalamin is converted to adenosylcobalamin, coenzyme for Methylmalonyl-CoA mutase and to methylcobalamin, coenzyme for methionine synthase. Very rare disorders show genetic defects of these steps which are associated with combined homocystinuria and methylmalonic aciduriaders and are not able to produce adenosylcobalamin, a derivative of vitamin B12. Laboratory findings are high methylmalalonic acid level in urine and blood, long chain ketonuria, intermittent hyperglycinemia, hyperammonemia, pancytopenia. Clinical symptoms are retarded development, lethargy, failure to thrive, recurring vomiting, dehydration, respiratory distress, reduced muscle tone, enlarged liver, poor feeding, seizures and fine tremors.

- Maternal Vitamin B12 Deficiency

Vitamin B12 (cobalamin) is an essential cofactor in several metabolic pathways. Intracellular conversion of cobalamin to its two coenzymes, adenosylcobalamin in mitochondria and methylcobalamin in the cytoplasm, is necessary for the homeostasis of methylmalonic acid and homocysteine. One of these defects, the cblD defect, can cause isolated methylmalonic aciduria, isolated homocystinuria, or both. Affected persons present with multisystem clinical abnormalities, including developmental, hematologic, neurologic, and metabolic findings. The gene responsible for the cblD defect has not been identified.

- β-Ketothiolase Deficiency (BKD)

BKD occurs when an enzyme, called “Mitochondrial Acetoacetyl-CoA Thiolase” (MAT), is either missing or not working properly. This enzyme’s job is to help break down the amino acid isoleucine. When a child with BKD eats food containing isoleucine, harmful substances called organic acids build up in the blood and cause problems. Isoleucine is found in all foods that contain protein. BKD can cause episodes of illness called metabolic crises. Some of the first symptoms of a metabolic crisis are extreme sleepiness or lack of energy, vomiting, diarrhea, fever, poor appetite and ketones in the urine (substances created during the breakdown of fat). Other symptoms then follow: increased levels of acidic substances in the blood, called metabolic acidosis, low blood sugar, called hypoglycemia, coma, sometimes leading to death.

Therapy: Medication (L-carnitine, biocarbonate, glucose), avoid going a long time without food, low-protein diet, tracking ketone levels

- Propionic Aciduria (PA)

PA occurs when an enzyme called “Propionyl CoA Carboxylase” (PCC) is either missing or not working properly. This enzyme’s job is to change certain amino acids so the body can use them. When this enzyme is not working, substances called glycine and propionic acid, along with other harmful substances, build up in the blood and cause problems. Without treatment, brain damage can occur. This can result in mental retardation. If not treated, many babies with PA die within the first year of life.

Therapy: Low-protein diet, medical foods and medical formula, Avoid going a long time without food, Medication (Lcarnitine, biotin, bicarbonate, glucose), Regular blood and urine tests, Tracking of ketones

- Multiple-CoA Carboxylase Deficiency (GA-2)

GA-2 occurs when one of two different enzymes is either missing or not working properly. The enzymes responsible for GA-2 are called “Electron Transfer Flavoprotein” (ETF) and “ETF-ubiquinone oxidoreductase” (ETF:QO). The job of these enzymes is to help make energy for the body by breaking down certain fats and proteins from the food we eat.

They also break down fat and protein already stored in the body. Many babies with GA-2 have an odor that smells like “sweaty feet”. In addition, they often have serious heart and liver problems. Without treatment, most babies die within the first few weeks of life. Even with treatment, many babies with GA-2 die of severe heart problems within a few months.

Therapy: Avoid going a long time without food, low-fat, low-protein, high-carbohydrate diet, Riboflavin, L-carnitine and glycine supplements.

- Methylmalonic Aciduria (MMA)

MMA occurs when one of these special enzymes is either missing or not working properly. Without this enzyme, certain amino acids and fatty acids cannot be used correctly. This causes glycine, methylmalonic acid, and other harmful substances to build up in the blood and urine and cause health problems. There are a number of different forms of MMA. Some forms can be treated with vitamin B12 injections. Two types of MMA that often can be treated with vitamin B12 are Cobalamin A (CblA) deficiency and Cobalamin B (CblB) deficiency.

There are other forms of MMA which cannot be treated with vitamin B12. One of these is called ‘Mut 0’. It is caused by the absence of an enzyme called methylmalonyl-CoA mutase (MCM). Another type of MMA that does not respond to vitamin B12 treatment is called ‘Mut –‘. People with the ‘Mut–‘ type of MMA have too little of the MCM enzyme. Isoleucine, valine, methionine, and threonine are the four amino acids that cannot be

used correctly by people with MMA. These amino acids are found in all foods that contain protein. Large amounts are found in meat, eggs, milk, and other dairy products. Smaller amounts are found in flour, cereal, and some vegetables and fruits. Without treatment, brain and nerve damage can occur. This can cause mental retardation and problems with involuntary movements. Death is common in untreated babies and children.

Therapy: Medication (vitamin B12 injections in the form of hydroxocobalamin (OH-cbl) or cyanocobalamin (CN-cbl), Lcarnitine, biocarbonate, glucose), low-protein diet, medical foods and medical formula, avoid going a long time without food, regular blood and urine tests, tracking of ketones.

• Acylcarnitines

- Fatty Acid Oxidation Disorders

Fatty acid oxidation disorders are a group of inherited metabolic conditions that lead to an accumulation of fatty acids, and a decrease in cell energy metabolism. Each fatty acid oxidation disorder is associated with a specific enzyme defect in the fatty acid metabolic pathway and affects utilization of dietary and stored fat.

| Normal value | Refer To Report |
|---|---|

An elevated level of a particular acylcarnitine may indicate the possibility of one of several different fatty acid oxidation disorders which include:

- Carnitine-Acylcarnitine Translocase (CACT) Deficiency

CAT can cause serious brain damage and metabolic crises (muscle weakness, seizures, coma). Babies who are not treated usually die of heart problems, breathing problems, liver failure or high levels of ammonia in the blood.

Therapy: Avoid going a long time without food, low-fat, high carbohydrate diet, L-carnitine and MCT oil.

- Carnitine Palmitoyl Transferase I & II (CPT I & II) Deficiency

people with CPT-1A deficiency are usually healthy. However, repeated episodes may cause brain damage that can result in learning problems or mental retardation.

Therapy: Avoid going a long time without food, low-fat, high carbohydrate diet, Medium Chain Triglyceride oil (MCT). There are three clinical presentations of CPT II Deficiency. The classic form has adult onset of exercise-induced muscle weakness, often with rhabdomyolysis and myoglobinuria that can be associated with acute renal failure. CK levels are found to be elevated only during a symptomatic period. Carnitine levels are normal. A second phenotype is often fatal in the period from 3 to 18 months of age. Presentation can be onset of seizures with hepatomegaly, non-ketotic hypoglycemia, cardiomyopathy, hypotonia, and muscle weakness. Plasma free carnitine levels are low and acyl-carnitine high. A severe form presents in the newborn period with non-ketotic hypoglycemia, cardiomyopathy, muscle weakness, and renal dysgenesis in some patients. All of these patients have expired within days of birth. These different clinical presentations appear to be correlated with residual CPT II enzyme activity. Adult onset patients are found to have approximately 25% of normal activity while the other clinical groups have less than 15%.

Therapy: alter their lifestyle and refrain from rigorous exercise, avoid prolonged fasting, Medium-chain triglyceride oil, aggressive treatment of acutely ill infants with IV glucose and cardiac support is critical, L-Carnitine supplementation should be instituted.

-2,4 Dienoyl-CoA Reductase Deficiency

One patient has been reported with 2,4-Dienoyl-CoA Reductase Deficiency. This enzyme is necessary for the degradation of unsaturated fatty acids having even numbered double bonds. The patient was born with a small body habitus, a short trunk, arms and fingers, and microcephaly. She was readmitted to the hospital on day 2 of life with symptoms of sepsis, hypotonia, decreased feeding and intermittent vomiting. A low carnitine level was found in her plasma. She responded poorly to treatment in the hospital, and later developed respiratory acidosis and died at 4 months of age.

- Glutaric Aciduria Type I (GA-1)

Babies with GA-1 are usually healthy at birth, although many are born with a larger than average head size. Other symptoms usually start between two months and four years of age. GA-1 causes episodes of severe illness called metabolic crises. Some of the first symptoms are poor appetite, extreme sleepiness or lack of energy, irritability, jitteriness, nausea, vomiting, low muscle tone (floppy muscles and joints), muscle weakness. If untreated, other symptoms then follow: tics or spasms of the muscles, swelling of the brain or blood in the brain, coma, sometimes leading to death.

Therapy: Medication L-caritine sometimes glucose, insulin, avoid going a long time without food, Food plan, including medical foods and formula, Low-protein (lysine and tryptophan) diet, Medical foods and formula, Regular blood tests.

- Electron Transfer Flavoprotein (ETF) Dehydrogenase Deficiency ( Multiple Acyl-CoA Dehydrogenase Deficiency (MADD) )

Three clinical presentations are reported for MADD.Two newborn presentations are seen one with congenital anomalies, and one without. Those with congenital anomalies are often premature, and develop symptoms in the first 24-48 hours consisting of hypotonia, hepatomegaly, severe nonketotic hypoglycemia, metabolic acidosis and variable body odor of sweaty feet.

Dysmorphic facial features and dysplastic, cystic kidneys are present. Plasma carnitine levels are low. Those patients with no congenital anomalies have similar symptoms and metabolic abnormalities. With both neonatal presentations, most patients do not live past a few weeks, though some older survivors succumb at a few months of age from hypertrophic cardiomyopathy. Heart, liver and kidneys are infiltrated with fat. The third cohort of patients has a mild and/or later onset with variable symptoms including lipid storage myopathy.

Therapy: riboflavin (a precursor to FAD) and L-carnitine supplementation, dietary restriction of fats and protein has had variable results.

- Medium-Chain Acyl-Coenzyme A Dehydrogenase Deficiency (MCAD Deficiency)

people with MCADD are usually healthy. However, repeated episodes can cause permanent brain damage. This may result in learning problems, mental retardation or spasticity. Therapy: Avoid going a long time without food, low fat, high carbohydrate diet, L-carnitine.

- Short-Chain Acyl-Coenzyme A Dehydrogenase Deficiency (SCAD Deficiency)

SCADD was originally thought to be very rare. However newborn screening for this disorder revealed that SCADD is more common than previously believed. The actual incidence is unknown. Our bodies rely on fat when we don't eat for a stretch of time like when we miss a meal or when we sleep. SCADD is a highly variable and not well understood. Most babies found to have SCADD through newborn screening never have symptoms. In fact, so far, there have been only about 20 people with SCADD reported to have health effects. Things that cause stress, such as lack of sleep, going without food for too long, illness, or infection are thought to trigger episodes of illness called metabolic crisis in some children but not others.

Therapy: Avoid going a long time without food, low fat, high carbohydrate diet, L-Carnitine and Riboflavin (vitamin B2).

- Very Long-Chain Acyl-Coenzyme A Dehydrogenase deficiency (VLCAD deficiency)/ Long-Chain 3-Hydroxyacyl-Coenzyme A Dehydrogenase deficiency (LCHAD deficiency)

A child with VLCAD/LCHAD can develop breathing problems and seizures. Periods of hypoglycemia can happen with or without the other symptoms. Hypoglycemia can

cause a child to feel weak, shaky or dizzy with clammy, cold skin. If not treated, it can lead to coma, and possibly death.

Therapy: Avoid going a long time without food, low fat, high carbohydrate diet, MCT oil and L-carnitine, avoid prolonged exercise or exertion.

• 3-Hydroxyacyl-Coenzyme A Dehydrogenase Deficiency (M/SCHAD Deficiency)

M/SCHADD is highly variable and not well understood. Things that cause stress, such as lack of sleep, lack of food, illness or infection are thought to trigger episodes of illness called metabolic crises in some children with M/SCHADD but not in others.

Therapy: Avoid going a long time without food, L-carnitine supplements and other medications.

• Trifunctional Protein Deficiency

Prevalence: TFP deficiency very rare, actual incidence unknown. Infants with early TFP who remain untreated usually die of heart or breathing problems by three years of age. If muscle symptoms are not treated, kidney failure can occur.

Therapy: Avoid going a long time without food, low fat, high carbohydrate diet, MCT oil and L-carnitine, avoid exercise and extreme cold.

# Nicotine (Cotinine)

## Summary

Nicotine accumulates in the blood system by inhalative smoking which causes higher number of disabilities, pain, breathing problems, stroke and cancer.

| | | |
|---|---|---|
| Remarks | Preferable morning urine. | |
| Method | ICT | |
| TAT | Dy/AB | |
| Sample type | US | |
| Expected value | No Active Smoking | <10 ng/mL |
| | Heavy Smoking | >300 ng/mL |
| | Urine Active Smoking | >500 ng/mL |
| Sample stability | 2 d at 2-8˚C | |
| | 6 mth at -20˚C | |

# N-Terminal pro B-type Natriuretic Peptide (proBNP)

## Summary

NT-proBNP testing is useful for diagnosing acute decompensated heart failure. The high sensitivity of proBNP allows for the detection of mild forms of cardiac dysfunction in asymptomatic patients with structural heart disease. Clinical information and imaging procedures are used to diagnose left ventricular dysfunction, in subjects with left ventricular dysfunction serum and plasma concentrations of BNP increase as does the concentration of the putatively inactive amino-terminal fragment, NT-proBNP. Increased risk in proBNP also represents cardiac function and indicates patients scheduled for potentially cardiotoxic drugs or interventions causing fluid retention or volume overload (e.g. COX-2 inhibitors and nonsteroidal anti-inflammatory drugs).

| | | | |
|---|---|---|---|
| Method | ECLIA | | |
| TAT | Dy/AB | | |
| Sample type | HWB | | |
| Expected value | Age | Male (pg/mL) | Female (pg/mL) |
| | <50yr | 33.6-84 | 62-155 |
| | >50yr | 77.6-194 | 88.8-222 |
| Conversion factors | pmol/L x 8.457 = pg/mL | | |
| | pg/mL x 0.118 = pmol/L | | |
| Sample stability | 3 d at room temperature | | |
| | 6 d at 2-8˚C | | |
| | 1 yr at -20˚C | | |

# Occult Blood

## Summary

Most of diseases can cause hidden blood in the stool. Early stages, gastrointestinal problems such as colon cancer, ulcer, polyps, colitis, hemorrhoids, diverticuititis, and fissures are not shown any visible symptoms.

| | |
|---|---|
| Method | ICT |
| TAT | Dy/A |
| Sample type | ST |
| Expected value | Negative |
| Sample stability | 3 d at 2-8 ˚C |

# Opiates (OPI)

## Summary

The toxicity is caused by the content of alkaloids (morphine, codeine, thebaine, papeverine, narcotion and others). Effects in case of acute abuse: euphoretic, pain-reducing, relaxing, anticonvulsant, time and space changing impressions, dream stimulation. Opiates indicate drugscreening.

| | |
|---|---|
| Method | Kinetic/ICT |
| TAT | Dy/AB |
| Sample type | US |
| Expected value | Not detected/ Negative |
| Sample stability | 5 d at 2-8 ˚C |

# Osmolality

## Summary

The osmolality test is a snapshot of the number of solutes present in the blood (serum) or urine. It is ordered to help evaluate the body's water balance, its ability to produce and concentrate urine, to help investigate low sodium levels (hyponatremia), to detect the presence of toxins such as methanol and ethylene glycol, and to monitor osmotically active drug therapies such as mannitol. It is also ordered to help monitor the effectiveness of treatment for any conditions found.

**Serum osmolality** is primarily ordered to investigate hyponatremia. If a patient with significant hyponatraemia (serum sodium < 130 mmol/l) has a normal plasma osmolality, the patient may have pseudohyponatraemia due to excess lipids or proteins, or the sample may have been collected from a drip arm containing dextrose. If the patient has an increased osmolality it is likely the patient has reactive hyponatraemia due to an excess of solute pulling water out of cells. Examples of this include glucose in diabetes mellitus or hyperglycinaemia after trans-urethral resection of the prostate. The finding of a hypo-osmolar hyponatraemia ("true hyponatraemia") then leads to further investigation of the cause.

**Urine osmolality** is frequently ordered along with serum osmolality. It is used to help evaluate the body's water balance and to investigate increased and decreased urine output. Increased urine output may be due to increased fluid intake, lack of appropriate amounts of Antidiuretic Hormone (ADH), or due to diabetes, with increased glucose levels leading to increased urine output. Sometimes a urine osmotic gap is calculated and used to help evaluate the kidney's ability to excrete acid and reabsorb bicarbonate, to detect the presence of osmotically active molecules, and to compare with the serum osmotic gap.

| | |
|---|---|
| Method | Calculated |
| TAT | Dy/AB |
| Sample type | S/US/24U |
| Expected value | S : 275-298 mosm/kg |
| | 24U: Male (♂) :392-1092 mosm/kg |
| | Female (♀): 301-1093 mosm/kg |
| Sample stability | 3h at room temperature |
| | 1 wk at 2-8˚C |
| | 3 mth at -20 ˚C |

# Osteocalcin

## Summary

Osteocalcin (bone-gamma carboxyglutamic acid containing protein) is vitamin-K-dependent and can be stimulated by 1,25-dihydroxy vitamin D. Osteocalcin is often used as a biochemical marker for the bone formation process. It has been routinely observed that higher serum osteocalcin levels are relatively well correlated with bone diseases characterized by increased bone turnover, especially osteoporosis.

| | | |
|---|---|---|
| Remarks | Neither hemolyzed nor lipemic sample, keep on ice. | |
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | premenopause | 11-43 |
| | postmenopause | 15-46 |
| | Osteoporosis Patient | 13-48 |
| | Age | Male (ng/mL) |
| | 18-29 yr | 24-70 |
| | 30-50 yr | 14-42 |
| | 51-70 yr | 14-46 |
| Sample stability | 8h | at room temperature |
| | 3 d | at 2-8˚C |
| | 3 mth | at -20 ˚C |

# Ovulation Test

## Summary

Ovulation tests are used to determine the fertile days to maximize the efforts in trying to conceive. Ovulation is triggered by Luteinising Hormone (LH) which peaks two days before ovulation. Progesterone levels then begin to rise at the same time as the LH surge. After ovulation the ovocytes take 2-3 days to ripen making the most fertile period 5-6 days after the initial rise in progesterone. The most appropriate test for detecting ovulation is a serum progesterone concentration. This is performed approximately seven days before the predicted date of a menstrual period (day 1).

| | |
|---|---|
| Method | ICT |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Positive |
| Sample stability | 1 yr at 2-8 ˚C |

# Oxcarbazepine 10-OH Metabolite

## Summary

The kinetics of Oxcarbazepine and its active metabolite 10-Hydroxy-Carbazepine (10-OH-CZ) after a single oral dose was compared in healthy control subjects and in epileptic patients treated with phenobarbitone or sodium valproate. Oxcarbazine does not cause any enzyme induction. No dose adjustment is needed. The interaction with other medicaments is low. The tolerability is better than that of carbamazepine since kinetics is not affected by epoxide, which is described to be the main factor for intolerances.

| | | |
|---|---|---|
| Method | HPLC | |
| TAT | 10-14 d | |
| Sample type | S | |
| Expected value | < 3.0 mg/L | |
| Sample stability | 6 wk | at room temperature |
| | 6 wk | at 2-8˚C |
| | 3 mth | at -20˚C |

# Panel Reactive Antibody (PRA)

## Summary

Panel Reactive Antibody (also called Anti-HLA antibodies) is an immunological test routinely preformed on the blood of patients waiting for organ transplantation to give an estimate of the likelihood of finding a suitable donor.

Pregnancy, previous transplants, and blood transfusions are common causes for increased panel reactive antibody levels, patients receiving certain medications, such as erythropoietin, had a decrease in the amount of blood transfusions that were needed and a reduction in PRA levels. Preformed anti-HLA antibodies are associated with hyperacute rejection of kidney, heart and lung grafts, primary nonfunctional of liver grafts, and failure of platelet transfusions and accelerated and chronic loss of solid organ transplants. When present in blood or plasma donors, preformed anti-HLA antibodies may cause Transfusion-Related Acute Lung Injury (TEALI) in some recipients.

| | | |
|---|---|---|
| Method | MBA | |
| TAT | 2 d | |
| Sample type | Donor: EWB | |
| | Receiver: S | |
| | Or S | |
| Expected value | Negative: <1:10 | |
| Sample stability | 1 mth | at 2-8˚C |
| | 2 yr | at -20 ˚C |

# Pap smear

## Summary

A conventional pap smear is used as a screening test for evaluation of the lower female genital tract to detect the presence of inflammatory/infectious or benign proliferative conditions; detection of unsuspected or confirmation of suspected atypical, premalignant, or malignant changes; or follow-up of patients with known and/or treated premalignant or malignant lesions.

### unich Nomenclature

| Class | Criteria of classification | Recommendation |
|---|---|---|
| I | Normal image with enough representative cells; no damage due to technical, degenerative, or inflammatory factors | |
| II | Just enough cells, or only partly representative cells, and/or damage due to technical, degenerative, or inflammatory factors | |
| II W | Interpretation of cells is difficult because of their specific morphology or changes caused by technical, degenerative, or inflammatory factors; impossible to distinguish from mild or moderate dysplastic cells | Repeat smear after 1-6 months, possibly after treatment of vaginitis or after estrogen therapy |
| III | Interpretation of cells is difficult because of their specific morphology or changes caused by technical, degenerative, or inflammatory factors; impossible to distinguish from severe dysplastic cells or malignant tumor cells | Repeat smear after 1 month, possibly after treatment of vaginitis or after estrogen therapy; or histological examination (curettage, conization, exploratory excision), possibly colposcopy and HPV analysis |
| III E | Detection of endometrial cells or other atypical glandular cells after menopause without hormone replacement therapy, or in case of abnormal bleeding during hormone replacement therapy | Ultrasonography or histological examination (curettage, possibly conization) and/or laparoscopy |

| Class | Criteria of classification | Recommendation |
|---|---|---|
| III D | Detection of mild or moderate dysplastic cells, possibly with signs of HPV infection | Repeat smears at intervals of 3-6 months, with colposcopy and possibly also HPV analysis; after a course of more than a year, possibly histological examination (conization, curettage, exploratory excision) |
| IV A | Detection of severe dysplastic cells or squamous carcinoma in situ cells, possibly with signs of HPV infection | Histological examination (conization, curettage, exploratory excision); under special circumstances (pregnancy, post partum, severe inflammation) possibly repeat smears at control intervals of one month, with colposcopy and possibly HPV analysis |
| IV B | Detection of questionable squamosous carcinoma cells | Histological examination (conization, curettage, exploratory excision) after colposcopy and possibly HPV analysis |
| V | Detection of squamosous carcinoma cells, abnormal glandular cells, or other malignant tumor cells | Histological examination (conization, curettage, exploratory excision) after colposcopy and possibly HPV analysis; possibly laparoscopy |

**Remarks**

In premenopausal patients, obtain specimens during the second half of the menstrual period to avoid contamination by obscuring blood, Instruct the patient not to douche or engage in sexual intercourse within 24 hours of the procedure.

| | |
|---|---|
| Method | LBC |
| TAT | 2 d |
| Sample type | Cell prep+ Brush |
| Expected value | Refer To Report |
| Sample stability | 1 mth room temperature |
| | 6 mth 2-8 ˚C |

# Parainfluenza Virus I,II, III Abs (IgA,IgG,IgM)

## Summary

The majority of parainfluenza virus infections result in a non-specific upper respiratory tract infection, even in children. Parainfluenza virus infections in adults are relatively uncommon, and symptoms are usually less severe in adults than in children, the most common symptoms are milder influenza-like illness, fever, headache, cough, sore throat, bronchitis and the complications of the disease may induce bronchopneumonia, croup, bacterial superinfection. The Infections are spread via droplets, aerosols (upon contact)fomites (indirect contact transmission).

IF IgG antibodies to Parainfelunza is detected, it indicates a past parainfluenza virus infection, while IgM/IgA indicates a current infection to the disease.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:10 | |
| Sample stability | 2 d | at room temperature |
| | 2wk | at 2-8 ˚C |
| | 1 yr | at - 20 ˚C |

# Parvovirus B19

## Summary

Parvovirus occurs endemically, the infection is transmitted by droplets or more rarely parenteral. After an incubation period of 13-17 days, an erythema infectiosum, lymph node swellings and infuenzal symptoms occur. Arthralgies and arthritises also have been observed. Parvovirus B19 can lead to a temporary myelodysplasia with inhibition of the erythropoesis. In pregnant women parvovirus B19 is associated with hydrops fetalis due to severe fetal anemia, sometimes leading to miscarriage or stillbirth. The risk of fetal loss is about 10% if infection occurs before week 20 of pregnancy. The detection of Parvovirus B19-DNA indicates the infection clarification, virus persistency and infectivity, the suspicion of prenatal infection.

| | | |
|---|---|---|
| Method | PCR | |
| TAT | 5 d | |
| Sample type | EWB | |
| Expected value | Refer To Report | |
| Sample stability | 1 d | at room temperature |
| | 9 d | at 2-8˚C |

# Parietal Cells Abs (PCA)

## Summary

The detection of antibodies against parietal cells indicates forms of chronic atrophic gastritis, pernicious anemia, funicular myelosis, and various autoimmune endocrinopathies such as Basedow's and Addison's diseases.

| | | |
|---|---|---|
| Method | IF | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative: <1:20 | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8˚C |
| | 1 yr | at -20˚C |

# Phenobarbital (PHNO)

## Summary

Phenobarbital is one of the most commonly used drugs for the treatment of grand mal, psychomotor epilepsy, and other forms of focal epilepsy. Monitoring of the serum level of the drug is essential in order to achieve maximal seizure control while maintaining minimal blood levels to avoid negative side effects. As with other anti-convulsant drugs, it is imperative that each patient's dosage be individualized.

| | | |
|---|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. | |
| Method | FP | |
| TAT | Dy/AB | |
| Sample type | S/HP | |
| Expected value | 15-40 µg/mL | |
| Conversion factors | mg/L x 4.31 = µmol/L | |
| | µmol/L x 0.232 = mg/L | |
| Sample stability | 6 mth | at room temperature |
| | 6 mth | at 2-8 ˚C |
| | 6 mth | at -20 ˚C |

# Phenytoin (PHNY)

## Summary

Phenytoin has been used extensively for seizure control in patients having both grand mal epilepsy, cortical focal seizures, and temporal lobe epilepsy. Serum level monitoring of the drug is essential in order to achieve maximal seizure control while maintaining minimal blood levels to avoid negative side effect.

| | |
|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. |
| Method | FP |
| TAT | DY/AB |
| Sample type | S/HP |
| Expected value | Not detected |
| Conversion factors | mg /L x 3.96 = µmol/L |
| | µmol/L x 0.252= mg/L |
| Sample stability | 2 d at room temperature |
| | 5 mth at - 20 ˚C |
| | 1 mth at 2-8 ˚C |

# Philadelphia Chromosome

## Summary

Philadelphia chromosome or Philadelphia translocation is a specific chromosomal abnormality that is associated with Chronic Myelogenous Leukemia (CML). It is the result of a reciprocal translocation between chromosome 9 and 22, However, the presence of the Philadelphia (Ph) chromosome is not sufficiently specific to diagnose CML, since it is also found in acute lymphoblastic leukemia (ALL, 25–30% in adult and 2–10% in pediatric cases) and occasionally in Acute Mylogenous Leukemia (AML).

| | | |
|---|---|---|
| Method | PCR | |
| TAT | 3 d | |
| Sample type | EWB | |
| Expected value | Refer To Report | |
| Sample stability | 1 h | at room temperature |
| | 2 d | at 2-8˚C |

# Poliovirus I,II,III Abs

## Summary

Polio is a picornavirus (Enterovirus, Rhinovirus). Polio enters into cells orally by contaminated water, food or saliva and replicates in B- and T-cells in the small intestine. In the absence of a strong immune response, the virus enters into the blood stream and in about 1% the polio virus attacks motor neurons and the central nervous system causing a lifelong paralysis or death. The infectious polio virus is excreted in faeces from where it can spread to sewage and water supplies.

Symptoms usually start 7 to 14 days after exposure. Infected persons are most contagious a few days before and up to a few days after manifesting symptoms. However, persons with polio can spread the infection as long as the virus is present in throat or excreted in stool. The virus can be found in the throat for about 1 week after infection and in stool for >6 weeks.

The commonly used polio vaccine is Oral Polio Vaccine (OPV) and Inactivated Polio Vaccine (IPV).

The determination of polioviruses antibodies used to determine the immunity status.

| | | |
|---|---|---|
| Method | NT | |
| TAT | 10- 14 d | |
| Sample type | S | |
| Expected value | < 1:4 | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8 ˚C |
| | 1 yr | at -20˚C |

# Porphyrins (PORP), Total

## Summary

Urine porphyrins are useful for the evaluation of Acute Intermittent Porphyria (AIP), Hereditary Erythropoietic Porphyria (Morbus Guenther), ErythroHepatic Protoporphyria (EPP), Porphyria Variegata (PV). Porphyria Cutanea Tarda (PCT), hereditary coproporphyria, paraneoplastic porphyria (in prostate gland and liver tumors), acute and chronic lead poisoning induced porphyria (e.g. hexachlorbenzene, polychlorinated biphenyls, vinylchlorid, TCDD).

| | | |
|---|---|---|
| Remarks | Collect and dispatch light protected sample. | |
| Method | SPHO | |
| TAT | 10-14 d | |
| Sample type | 24U | |
| Expected value | <150 µg/d | |
| Sample stability | 4 d | at 2-8˚C |
| | 1 mth | at -20˚C |

# Procollagen Type 1 Amino-Terminal Propeptide (P1NP), Total

## Summary

The P1NP concentration is directly proportional to the amount of new collagen and has emerged as a reliable marker of bone turnover. It is routinely used to monitor bone formation and the growth response to growth hormone treatment. A useful combination is to use the test with a marker of bone resorption, such as crosslinks.

Concentrations are increased in patients with various bone diseases, including bone metastases and by therapies, which are characterized by increased osteoblastic activity. If the pre-treatment baseline level of bone formation is already high, a therapy to increase bone formation might not be necessary. If the relative levels of bone formation and bone resorption do not improve during therapy, another therapy must be considered.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Premenopause 15.13-58.59 ng/mL | |
| | Postmenopause (Receive hormone replacement therapy) | |
| | 14.28-58.92 ng/mL | |
| | (No hormone replacement therapy) | |
| | 20.25-76.31 ng/mL | |
| Sample stability | 1 d | at room temperature |
| | 5 d | at 2-8˚C |
| | 6 mth | at -20˚C |

# Prostate-Specific Antigen (fPSA), Free

## Summary

(fPSA) or the ratio (fPSA/tPSA) is an additional decision criterion for a prostate gland biopsy. The part of free PSA in relation to total PSA is considerably lower in patients with prostate gland carcinoma than in benign prostate hyperplasia.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | DY/AB | |
| Sample type | S/HP/EP/CP | |
| Expected value | Cut-off (Free/Total PSA Ratio): | |
| | Malignant: <0.19 | |
| | Benign: >0.19 | |
| Sample stability | 8 h | at room temperature |
| | 2 d | at 2-8 ˚C |
| | 3 mth | at -20 ˚C |

# Prostate-Specific Antigen (tPSA), Total

## Summary

The prostate specific antigen is a tissue specific secretion product of the prostate gland cells. It is used as organ specific tumor marker. In healthy men PSA supports the liquefaction of the seminalplasma.

Under cell stress (manipulation, inflammation, necrosis, tumor) PSA penetrates through the basal cell membrane and enters into the blood stream (increased values).

Prostate-Specific Antigen (PSA) is a valuable tool for the early detection and management of prostate carcinoma. It is, however, an imperfect tool because of poor specificity in the face of prostatic hypertrophy and other benign conditions. In routine diagnostics a PSA value of 10 ng/ml is considered as the approximate limiting value for the differentiation between Benign Prostata Hypertrophy (BHP) and prostate gland carcinoma. PSA is absent after prostatectomy.

Elevated concentration of PSA in serum is generally indicative of pathologic condition of the prostate (prostaitis, benign hyperplasia or carcinoma). The main areas in which PSA determinations are employed are the monitoring of progress and efficiency of therapy in patients with prostate or receiving hormonal therapy if it's not detectable anymore it provides information of the success of therapy .

| | | |
|---|---|---|
| Remarks | Give the exact patients age (NOT THE ESTIMATED). | |
| Method | ECLIA | |
| TAT | DY/AB | |
| Sample type | S/EP/CP. | |
| Expected value | Age | ng/mL |
| | <40 yr | Up To 1.4 |
| | 40-50 yr | Up To 2.0 |
| | 50-60 yr | Up To 3.1 |
| | 60-70 yr | Up To 4.1 |
| | >70 yr | Up To 4.4 |
| Sample stability | 8 h | at room temperature |
| | 2 d | at 2-8 ˚C |
| | 3 mth | at -20 ˚C |

# Protein C

## Summary

Protein C is a vitamin K-dependent protein synthesized primarily by hepatocytes. Protein C deficiency may lead to serious thrombotic events such as thrombophlebitis, deep vein thrombosis, or pulmonary embolism. Patients with a congenital heterozygous deficiency may present with venous thrombosis (purpura fulminans) during the neonatal period. Acquired Protein C deficiency may be seen in liver diseases, extensive thrombotic episodes, surgery, oral anticoagulation, antiphospholipid syndrome, etc. A decreased Protein C activity in plasma may be the result of low concentrations and function (type I) or only low function (type II).

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | CP | |
| Expected value | 72-160 % | |
| Sample stability | 1 wk | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 1mth | at -20 ˚C |

# Protein Electrophoresis

## Summary

Serum contains over 100 individual proteins each with specific set of functions which are subject to specific variation in concentration under different pathological conditions. Serum protein has been fractionated on the basis of their charge at a particular pH. These fractions have proven to be useful aid in the diagnosis and prognosis of certain diseases state.

| | | |
|---|---|---|
| Method | ELPH | |
| TAT | 2 d (S) | |
| | 3 d (24U) | |
| Sample type | S/24U | |
| Expected value | Normal Pattern | |
| Sample stability in S | 4 d | at room temperature |
| | 2 wk | at 2-8˚ C |
| | 6 mth | at -20˚ C |

# Protein S

## Summary

Protein S is a vitamin K-dependent protein synthesized in the liver. Protein S deficiency may lead to serious thrombotic events such as thrombophlebitis, deep vein thrombosis, or pulmonary embolism. Acquired Protein S deficiency may be seen during pregnancy, use of oral contraceptives, liver disease, oral anticoagulant therapy, diabetes mellitus, postoperative complications, septicemia and various inflammatory syndromes. In case of a Protein S deficiency total and free Protein S should be tested for pre-classification.

There are three types of hereditary protein S deficiency:

Type I – decreased protein S activity: decreased total protein S (=both bound and free protein S) levels and decreased free protein S levels (quantitative defect)

Type II – decreased protein S activity: normal free protein S levels and decreased total protein S levels (qualitative defect)

Type III – decreased protein S activity: decreased free protein S levels and normal total protein S levels (quantitative defect)

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | CP | |
| Expected value | Free protein S | 50-130 % |
| | Total protein S | 60-150 % |
| Sample stability | 4 h | at room temperature |
| | 4 h | at 2-8 ˚C |
| | 4 h | at -20 ˚C |

# Protein, Total

## Summary

Hypoproteinemia can be caused by diseases and disorders such as loss of blood, sprue, nephrotic syndrome, severe burns, salt retention syndrome and Kwashiorkor (acute protein deficiency). Hyperproteinemia can be observed in cases of severe dehydration andillnesses such as multiple myeloma.

The A/G ratio is commonly used as an index of the distributionof albumin and globulin fractions. Marked changes in this ratio can be observed in cirrhosis of the liver, glomerulonephritis, nephrotic syndrome, acute hepatitis, and lupus erythematosus as well as in certain acute and chronic inflammations. Total protein measurements are used in the diagnosis and treatment of a variety of diseases involving the liver, kidney, or bone marrow, as well as other metabolic or nutritional disorders.

| | | |
|---|---|---|
| Method | Colorimetric | |
| TAT | Dy/AB | |
| Sample type | S/HP/EP | |
| Expected value | Age | Value (g/dL) |
| | Newborn | 4.6-7 |
| | 1 wk | 4.4-7.6 |
| | 1-2 yr | 5.6-7.5 |
| | >3 yr | 6-8 |
| | Adult | 6.6-8.7 |
| Conversion factors | g/L x 0.1 = g/dL | |
| Sample stability | 1 mth | at 2-8 ˚C |
| | 6 mth | at -20 ˚C |

# Protein, Total

## Summary

Protein measurements in urine are used in the diagnosis and treatment of disease conditions such as renal or heart diseases, or thyroid disorders, which are characterized by proteinuria or albuminuria. Cerebrospinal Fluid (CSF) protein measurements are used in the diagnosis and treatment of conditions such as meningitis, brain tumors and infections of the central nervous system.

Elevated levels occur as a result of increased permeability of the blood-CSF barrier or with increased local synthesis of immunoglobulins.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | U24 | |
| | CSF | |
| Expected value | 24U: 28-141 mg/d | |
| | CSF: 15-45 mg/dL | |
| Conversion factors | mg/L x 0.1 = mg/dL | |
| | mg/L x 0.001 = g/L | |
| Sample stability in 24U | 1 d | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 1 mth | at -20 ˚C |
| Sample stability in CSF | 1 d | at room temperature |
| | 6 d | at 2-8 ˚C |
| | > 1 yr | at -20 ˚C |

# Pyruvate Kinase

## Summary

Pyruvate Kinase Deficiency (PKD) is the second most frequent enzyme deficiency of the erythrocyte, hereditary autosomal recessive. Poikilocytosis and increased hemolysis can be observed (chronic, non-sphaerocytic and Coombs-negative hemolytic anemia). Gall stones and splenomegaly are frequent. Patients who have recently received transfusions have normal donor cells that may mask PK deficient erythrocytes.

| | |
|---|---|
| Method | PHO |
| TAT | 10-14 |
| Sample type | EWB |
| Expected value | 5.3 – 17.3 U/g Hb |
| Sample stability | 3 wk at 2-8˚C |

# Reducing Substances

## Summary

Reducing substances urine is used for screening disorders with increased excretion of carbohydrate such as fructose, glucose, galactose, disaccharides, oligosaccharides, and succinylpurines.

Testing for reducing substances in stool is used in diagnosing the cause of diarrhea in children. Increased values in stool are consistent with primary or secondary disaccharidase deficiency and intestinal monosaccharide malabsorption. Similar intestinal absorption deficiencies are associated with short bowel syndrome and necrotizing enterocolitis. Stool reducing substances is also helpful in diagnosing between osmotic diarrhea caused by abnormal excretion of various sugars as opposed to diarrhea caused by viruses and parasites.

| | | |
|---|---|---|
| Method | Colorimetric | |
| TAT | Dy/AB | |
| Sample type | US/ST | |
| Expected value | Negative | |
| Sample stability | 3d | at 2-8 ˚C |
| | 1 wk | at -20 ˚C |

# Renin Active

## Summary

Renin Activity should be measured in the diagnosis of hypertension, hyperaldosteronism (primary hyperaldosteronism, secondary hyperaldosteronism with or without hypertension, pseudo-hyperaldosteronism, isolated deficit in mineral corticoids, hypokalemia (secondary hyperaldosteronism or primary hypermineralcorticism), insufficient response to antihypertensive treatment, detection of Renin producing tumors in the kidney, monitoring of glucocorticoid therapy. Elevated Renin levels are seen in secondary hyper-aldosteronism and Addison's disease decreased Renin levels are seen in patients with Cushing's disease, liver cirrhosisand severe cardiac failure.

| | | |
|---|---|---|
| Remarks | 1 ml of the sample, don't cool. | |
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | EP/S | |
| Expected value | Supine: 0.15-2.33 ng/mL/h | |
| | Errect: 1.31-3.95 ng/mL/h | |
| Sample stability | 4h | at room temperature |
| | Long term storge | at -20˚C |

# Respiratory Syncytial Virus Abs (RSV-IgA,IgG,IgM)

## Summary

Respiratory Syncytial Virus (RSV), which causes infection of the lungs and breathing passages, is a major cause of respiratory illness in young children. In adults, it may only produce symptoms of a common cold, such as a stuffy or runny nose, sore throat, mild headache, cough, fever, and a general feeling of being ill. But in premature babies and kids with diseases that affect the lungs, heart, or immune system, RSV infections can lead to other more serious illnesses.

If IgG antibodies to RSV are detected it indicates a past RSV infection, whereas IgM, IgA detection indicates a current infection to the disease.

| | |
|---|---|
| Method | IF |
| TAT | Dy/A |
| Sample type | S |
| Expected value | Negative: <1:10 |
| Sample stability | 2 d at room temperature |
| | 2wk at 2-8 ˚C |
| | 1 yr at - 20 ˚C |

# Rett Syndrome (RTT)

## Summary

Rett syndrome (RTT) is caused by mutations in the gene MECP2 located on the X chromosome, and can arise sporadically or from germline mutations. Classic Rett syndrome is a progressive neurodevelopmental disorder characterized by normal development until 6-18 months of age followed by rapid developmental regression, deceleration of head growth, loss of speech and acquired motor skills, and seizures; purposeful use of the hands is replaced by repetitive stereotyped hand movements. MECP2-Related disorders include Rett-like syndrome, severe congenital encephalopathy, or mild to severe mental retardation.

| | | |
|---|---|---|
| Method | Sequencing | |
| TAT | 7 d | |
| Sample type | EWB | |
| Expected value | Refer To Report | |
| Sample stability | 3 d | at room temperature |
| | 1 wk | at 2-8˚C |

# Rheumatoid Factor Abs (IgA, IgG, IgM)

## Summary

The presence of IgM Rheumatoid Factor (RF) in the serum is the sole serological indicator included in the ACR list of criteria for the diagnosis of RA. RFs are present in the serum of 75-80% of patients with RA at some time during the disease course. However, RFs are also found in the serum of patients with infectious and autoimmune diseases, hyperglobulinemia, B-cell lymphoproliferative disorders and in the aged population.

The determination of IgG antibodies is a valuable indicator in the diagnosis of past infection, whereas IgM/IgA antibodies will be found in recent infection.

| | |
|---|---|
| Method | ELISA |
| TAT | 3 d |
| Sample type | S |
| Expected value | Negative |
| Sample stability | 1 mth at 2-8 ˚C |

# Rheumatoid Factor II (RF-II)

## Summary

RF is impotent in the diagnosis of rheumatoid arthritis, but can also found in the other inflammatory rheumatic diseases and in various non-rehumatic diseases. it is also found in clinically healthy parsons over 60 years of age.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S/EP/HP | |
| Expected value | <14 IU/mL | |
| Sample stability | 2d | at room temprature |
| | 5 mth | at - 20 ˚C |
| | 1 mth | at 2-8 ˚C |

# Rotavirus

## Summary

Rotaviruses are the most frequent microorganisms in viral intestinal infections causing more than 70% of serious diarrhea in children worldwide. Infected children excrete viruses in high concentrations (highly infectious). The disorder manifests suddenly with severe stomach ache and diarrhea with blood and/or slime and fever.

| | |
|---|---|
| Method | ICT |
| TAT | Dy/AB |
| Sample type | ST |
| Expected value | Negative |
| Sample stability | 2 d at 2-8 ˚C |

# S100

## Summary

In patients suffering from malignant melanoma, especially stage II, III, and IV, elevated S100 serum levels may indicate disease progression. Serial measurements can be useful for follow-up and monitoring therapy success in these patients. S100 can be detected in patients with cerebral damage caused by several events,such as traumatic brain injuries or stroke. traumatic brain injuries or stroke

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Up To: 0.105 µg/L | |
| Conversion factors | µg/L x 1 = ng/mL | |
| | µg/L x 1000 = pg/mL | |
| | ng/mL x 1000 = pg/mL | |
| Sample stability | 8 h | at room temperature |
| | 2 d | at 2-8 °C |
| | 3 mth | at -20 °C |

# Salicylate (SALI )

## Summary

Salicylate is a common drug used in many formulations due to its analgesic and anti-inflammatory properties. Salicylate overdose can cause metabolic acidosis with a high anionic gap, gastrointestinal and central nervous system disturbances, as well as encephalopathy and renal failure.

| | |
|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. |
| Method | Enzaymtic |
| TAT | Dy/A |
| Sample type | S/HP |
| Expected value | 30-100 µg/mL |
| Conversion factors | µg/mL x 0.00724 = mmol/L |
| | µg/mL x 0.1 = mg/dL |
| Sample stability | 1 wk at room temperature |
| | 2 wk at 2-8˚C |
| | 6 mth at -20˚C |

# Salmonella typhi

## Summary

Salmonella typhi is a gram negative bacterium, transmitted by the ingestion of food or water contaminated with the feces or urine of Salmonella carrying animals or humans. Shellfish harvested from polluted regions or raw vegetables fertilized with human manure (night soil) may also harbor the bacterium. Diligent hand washing is important to prevent hand to mouth transmission. In addition, flies may passively carry the bacteria to food. Typhoid symptoms generally appear 1-2 weeks after infection. They include a high temperature (fever), that can reach 39–40C (103–104F), which usually increases throughout the day before falling the following morning, abdominal pain, constipation or diarrhea, vomiting, a dry cough, severe mental confusion, rashes, a swollen abdomen, a slow heartbeat, loss of appetite, weight loss, physical exhaustion and rapid breathing.

| | | |
|---|---|---|
| Method | AGGL | |
| TAT | 2 d | |
| Sample type | S | |
| Expected value | Negative: <1:80 | |
| Sample stability | 2d | at room temperature |
| | 2 wk | at 2-8 ˚C |
| | 1 yr | at - 20 ˚C |

# Schistosoma Abs (Bilharziasis)

## Summary

The main cause of schistomiasis is the dumping of human waste into water supplies. Hygienic disposal of waste would be sufficient to eliminate the disease. The immune system responds to eggs in liver causing hypersensitivity; an immune response is necessary to prevent damage to hepatocytes. The hosts' antibodies which bind to the tegument of the Schistosome don't bind for long since the tegument is shed every few hours. The schistosome can also take on host proteins.

Schistomiasis can be divided into three phases: (1) the migratory phase lasting from penetration to maturity,(2) the acute phase which occurs when the schistosomes begin producing eggs, and (3) the chronic phase which occurs mainly in endemic areas.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Negative | |
| Sample stability | 2 d | at room temperature |
| | 2 wk | at 2-8 ˚C |
| | 1 yr | at - 20 ˚C |

# Selenium

## Summary

Selenium is part of the essential trace elements and is a cofactor of some enzymes and proteins. Nutritive selenium supply is sufficient. The risk of an insufficient supply exists during pregnancy and lactation or in persons exposed to heavy metals and oxidation substances.
Selenium deficiency manifests primarily in the liver, heart, bone and joints. Selenium deficiency occurs after long-lasting parenteral nutrition, in patients with malabsorption syndrome, Keshan- (endemically appearing cardiomyopathy) and Kashin-Beck disease (endemic osteoarthropathy with severe joint deformation). Intoxications show clinical symptoms like discoloration of the fingernails, hair loss, coagulation malfunctions and breath that smells like garlic.
Foods containing selenium include egg yolk, fish, meat (particularly chicken and pork) as well as in nards.
The daily-required quantity is approx. 50-100 µg.

| | | |
|---|---|---|
| Method | AAS | |
| TAT | 10- 14 d | |
| Sample type | S | |
| Expected value | therp. range | 100 µg/L |
| | Age | Value (µg/L) |
| | 0-1 yr | 16 – 48 |
| | 2-6 yr | 23 – 114 |
| | 7- 14 yr | 36 – 112 |
| | 15- 18 yr | 44 – 98 |
| | + 18 yr | 50 - 120 |
| Sample stability | 2 wk | at room temperature |
| | 2 wk | at 2-8˚C |
| | 2 wk | at -20˚C |

# Serotonin

## Summary

Serotonin is a monoamine neurotransmitter. It is found extensively in the gastrointestinal tract of animals, and about 80 to 90 percent of the human body's total serotonin is found in the enterochromaffin cells of the gut, where it is used to regulate persistaltic movements. Serotonin is also synthesized in serotonergic neurons with different functions and effects, including control of appetite, mood and anger.

In blood, serotonin is stored in platelets and is active wherever platelets bind: as a vasoconstrictor to stop bleeding, but also as activator of fibrocytic mitoses to support healing. Because of these effects, overdoses of serotonin, or serotonin agonist drugs, may cause acute or chronic pulmonary hypertension from pulmonary vasoconstriction, or else syndromes of retroperitoneal fibrosis or endocardial fibrosis from overstimulation of serotonic growth receptors on fibrocytes. Extremely high levels of serotonin can have toxic and potentially fatal effects, causing a condition known as serotonin syndrome.

Diagnosis of carcinoid tumors with symptoms suggestive of carcinoid syndrome rests on measurements of circulating and urine serotonin, urine 5-HIAA (5-Hydroxyindoleacetic Acid), and serum chromogranin A, a peptide that is cosecreted alongside specific hormones by neuroectodermal cells. Urine serotonin measurements are not commonly employed in carcinoid tumor follow-up. The exceptions are patients with tumors that almost exclusively secrete 5-Hydroxytryptophan (HTP).

Urine serotonin is, in most circumstances, the least likely marker to be elevated. In these individuals, urine serotonin is the tumor marker of choice to monitor disease progression.

| | |
|---|---|
| Remarks | Blood sample must be frozen. |
| | Urine sample must be cooled. |
| Method | HPLC/ Calculation |
| TAT | 10-14 d |
| Sample type | EWB/ 24U |
| Expected value | EWB: 50-200 ng/mL |
| | 24U: 50 - 250μg/24h |
| Sample stability in EWB | 1 mth -20 ˚C |
| Sample stability in 24U | 1 mth 2-8˚C |
| | 2 mth -20 ˚C |

# Sickling Test

## Summary

The Sickling test detects disorders with Hb S, including sickle cell traits, combinations of the Hb S gene with α, β Thalassemias and β-chain structural haemoglobin variants disorders.

| | |
|---|---|
| Method | Sodium metabisulphite |
| TAT | Dy/AB |
| Sample type | EWB |
| Expected value | Negative |
| Sample stability | 1 d at room temperature |
| | 1 wk at 2-8 ˚C |

# Sirolimus (Rapamune)

## Summary

**Sirolimus**, is an immunosuppressant drug used to prevent rejection in organ transplantation; it is especially useful in kidney transplants. It is also used as a coronary stent coating.

The sirolimus level must be monitored over time because the drug has a narrow therapeutic Index or range of effective concentration. If the drug concentration is too low, organ rejection may occur; if it is too high, symptoms associated with toxicity may develop.

| | | |
|---|---|---|
| Method | LCMS | |
| TAT | 3 d | |
| Sample type | EWB | |
| Expected value | 3 -20 ng/mL | |
| Sample stability | 1 d | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 2 mth | at - 20 ˚C |

# Soluble Fms-Like Tyrosine Kinase-1 (sFlt-1 )

## Summary

Serum levels of PlGF (Placental Growth Factor) and sFlt-1 (soluble fms-like tyrosine kinase-1, also known as VEGF receptor-1) are altered in women with preeclampsia. Moreover, circulating levels of PlGF and sFlt-1 can discriminate normal pregnancy from preeclampsia even before clinical symptoms occur. In normal pregnancy, the pro-angiogenic factor PlGF increases during the first 2 trimesters and decreases as pregnancy progresses to term. In contrast, levels of the anti-angiogenic factor sFlt-1 remain stable during the early and middle stages of gestation and increase steadily until term. In women who develop preeclampsia, sFlt-1 levels have been found to be higher and PlGF levels have been found to be lower than in normal pregnancy. PlGF and sFlt-1 concentrations measured by immunoassay in maternal blood improve the diagnostic possibilities in preeclampsia which comprise clinical symptoms, proteinuria and uterine artery Doppler velocimetry.

| | | |
|---|---|---|
| Remarks | State Gestational Age. | |
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Gestational wk | Value (pg/mL) |
| | 10-14wk | 555-2361 |
| | 15-19wk | 470-2785 |
| | 20-23wk | 649-2944 |
| | 24-28wk | 630-3890 |
| | 29-33wk | 707-6688 |
| | 34-36wk | 978-9921 |
| | 37-delivery | 1671-11324 |
| Sample stability | 3 h | at room temperature |
| | 8 h | at 2-8 °C |
| | 1 mth | at -20 °C |

# Spinal Muscular Atrophy (SMA)

## Summary

Spinal Muscular Atrophy (SMA) is an autosomal recessive disease caused by a genetic defect in the SMN1 gene that codes SMN, a protein widely expressed in all eukaryotic cells. SMN1 is apparently selectively necessary for survival of motor neurons, as diminished abundance of the protein results in death of neuronal cells in the anterior horn of the spinal cord and subsequent system-wide muscle wasting (atrophy).

Spinal muscular atrophy is characterized by progressive muscle weakness caused by the degeneration of lower motor neurons that are responsible for controlling voluntary muscle movement including walking, crawling, swallowing, and head and neck control. The most common type of SMA is associated with respiratory failure and death before the age of two.

| | |
|---|---|
| Method | PCR |
| TAT | 7 d |
| Sample type | EWB |
| Expected value | Refer To Report |
| Sample stability | 5 d at room temperature |
| | 2 wk at 2-8˚C |

# Squamous Cell Carcinoma Antigen (SCCA)

## Summary

Squamous Cell Carcinoma (SCC) is a malignant cancer of epithelium that shows squamous cell differentiation. It can occur in several tissues, including uterine cervix, oral cavity, esophagus, lung, anal canal, and skin.

The SCCA, a tumour marker for squamous cell carcinoma, is used for monitoring the course and therapy of squamous cell carcinoma of the cervix, nasopharynx, ear, lungs and esophagus. Elevated levels of SCCA are diagnosed in patients with SCC of the cervix (85%) with carcinoma of the nasopharynx and the ear (60%). Sometimes raising SCCA exhibit renal insufficiency (10 ng / ml), in patients with hepatobiliary disease. Elevated levels of SCCA are also found in 31% of SCC of the lung, and in 17% of non small cell lung cancer.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | 2 d | |
| Sample type | S | |
| Expected value | Up To 1.5 ng/mL | |
| Sample stability | 2 d | at room temperature |
| | 4 mth | at 2-8 ˚C |
| | 4 mth | at - 20 ˚C |

# Stone Analysis

## Summary

The presence of kidney calculus is due to the conglomeration and the crystallization of matter in supersaturation. Nutritional, metabolical, genetical, anatomical, iatrogenical, neurological or infectious reasons can be the reasons of calculus formation. Passing a kidney stone through a ureter or the urethra may be painless or it may cause severe pain. A kidney stone may cause other symptoms, such as blood in the urine (hematuria), or a severe need to urinate. Chemical analysis of a kidney stone shows the type of stone which can guide treatment and give information that may prevent more stones from forming.

| | |
|---|---|
| Remarks | Keep dry |
| Method | Chemical analysis |
| TAT | Dy/A |
| Sample type | SN |
| Expected value | Refer To Report |
| Sample stability | Indefinitely from room temperature to -20 ˚C |

# T-Cell Receptor Rearrangement (TCR)

## Summary

Detection of rearranged T-cell receptor genes can be used to help establish a diagnosis of T-cell lymphoma, monitor for treatment response, and/or measure Minimal Residual Disease (MRD). T Cell Receptor (TCR) gene rearrangement is an important event in T cell ontogeny that enables T cells to recognize antigens specifically. Any dysregulation in this complex, yet highly regulated, process may result in disease.

The TCR genes are located on chromosomes 7 and 14.

| | | |
|---|---|---|
| Method | PCR | |
| TAT | 7 d | |
| Sample type | EWB | |
| Expected value | Refer To Report | |
| Sample stability | 1 d | at room temperature |
| | 5 d | at 2-8˚C |

# Testosterone, Free

## Summary

Measurement of the free or unbound fraction of serum testosterone has been proposed as a means of estimating the physiologically bioactive hormone. Free testosterone levels are elevated in women with hyperandrogenism associated with hirsutism in the presence or absence of polycystic ovarian disease. In addition, free testosterone measurements may be more useful than total testosterone in situations where SHBG is increased or decreased (e.g. hypothyroidism and obesity)

| | | |
|---|---|---|
| Remarks | Neither hemolyzed nor lipemic sample. | |
| Method | ELISA | |
| TAT | Dy/A | |
| Sample type | S | |
| Expected value | Male (♂) | 8-22 pg/mL |
| | Ovulatory | 0.5-2.3 pg/mL |
| | Oral Contraceptive | 0.5-1.7 pg/mL |
| | Postmenopause | 0.4-1.4 pg/mL |
| | Age | (Pg/mL) |
| | 0-10 yr | 0.13-0.7 |
| | 10-15 yr | 1.0-8.0 pg/mL |
| Sample stability | 2 d | at room temperature |
| | 1 wk | at 2-8˚C |
| | 6 mth | at -20˚C |

# Testosterone, Total

## Summary

Testosterone is the major androgenic hormone. It is responsible for the development of the male external genitalia and secondary sexual characteristics. In females, its main role is as an estrogen precursor. In both genders, it also exerts anabolic effects and influences behavior.

This test is used to measure the total amount of testosterone in blood. It is used to evaluate how well the sex organs (ovaries in females and testes in males) are working. The following are possible reasons why this test may be done:

- Absent or decreased secretion of sex hormones
- Adrenal gland cancer
- Congenital adrenal hyperplasia
- Hip fracture
- Polycystic ovaries

| | | | |
|---|---|---|---|
| Method | ECLIA | | |
| TAT | Dy/AB | | |
| Sample type | S/HP/EP | | |
| Expected value | Female (♀) | 0.06-0.82 ng/mL | |
| | Male (♂) | Age | (ng/mL) |
| | | <1 yr | 0.12-0.21 |
| | | 1-6 yr | 0.03-0.32 |
| | | 7-12 yr | 0.03-0.68 |
| | | 13-17 yr | 0.28-11.1 |
| | | +17 yr | 2.8-8.0 |
| Conversion factors | ng/mL x 3.47 = nmol/L | | |
| | ng/mL x 100 = ng/dL | | |
| | nmol/L x 0.288 = ng/mL | | |
| Sample stability | 1 wk | at 2-8˚C | |
| | 6 mth | at -20˚C | |

# Thalassemia

## Summary

Thalassemia are forms of inherited autosomal recessive blood disorders that originated in the Mediterranean region. caused by variant or missing genes that affect how the body makes hemoglobin. People with thalassemia make less hemoglobin and fewer circulating red blood cells than normal, which results in mild or severe anemia. Thalassemia can cause significant complications, including pneumonia, iron overload, bone deformities and cardiovascular illness. However this same inherited disease of red blood cells may confer a degree of protection against malaria, which is or was prevalent in the regions where the trait is common. In that respect, the various thalassemias resemble another genetic disorder affecting hemoglobin, e.g.sickle-cell disease.

Alpha-Thalassemia It is connected to the deletion in HBA1 and HBA2 genes of the 16p chromosome. Testing for it is important for the diagnosis of iron deficiency anemia, hypochromia and microcytosis.

| | |
|---|---|
| Method | PCR |
| TAT | 3 d |
| Sample type | EWB |
| Expected value | Refer to report |
| Sample stability | 3 d at room temperature |
| | 1 wk at 2-8˚C |

Beta-Thalassemia It is connected due to mutations in the HBB gene on chromosome 11. Testing for it is important for the diagnosis of hypochromic microcytic anemia, HBA2 and HBF- increase.

| | |
|---|---|
| Method | PCR |
| TAT | 3 d |
| Sample type | EWB/CWB |
| Expected value | Normal Genotype |
| Sample stability | 3 d at room temperature |
| | 1 wk at 2-8˚C |

# Thiopurine Methyltransferase (TPMT)

## Summary

TPMT is used as a screen to detect individuals with low (abnormal) TPMT activity that may be at risk for excessive myelosuppression when exposed to standard doses of thiopurines such as, azathioprine (Imuran) and 6-mercaptopurine (Purinethol). TPMT is the primary metabolic route for the inactivation of thiopurine drugs in the bone marrow. When TPMT activity is low, it is predicted that proportionately more 6-mercaptopurine is converted into the cytotoxic 6-thioguanine nucleotides, which will accumulate in the bone marrow and cause excessive toxicity. High TPMT activity means individuals are not predicted to be at low risk for bone marrow toxicity because of standard thiopurine dosing, but may be at risk for therapeutic failure due to excessive inactivation of thiopurine drugs.

| | |
|---|---|
| Method | HPLC |
| TAT | 4 d |
| Sample type | EWB |
| Expected value | 25-65 U/ML |
| Sample stability | 3 h at room temperature |
| | 6 d at 2-8˚C |

# Thyroid Stimulating Hormone (TSH)

## Summary

The determination of TSH serves as the initial test in thyroid diagnostics. Even very slight changes in the concentrations of the free thyroid hormones bring about much greater opposite changes in the TSH level. Accordingly, TSH is a very sensitive and specific parameter for assessing thyroid function and is particularly suitable for early detection or exclusion of disorders in the central regulating circuit between the hypothalamus, pituitary and thyroid.

| | |
|---|---|
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S/HP/EP/CP |

| Expected value | Age | Value (μIU/mL) |
|---|---|---|
| | <1 yr | 1.36-8.8 |
| | 1-6 yr | 0.85-6.5 |
| | 6-12 yr | 0.28-4.3 |
| | +12 yr | 0.27-4.2 |

| Sample stability | | |
|---|---|---|
| | 1 wk | at room temperature |
| | 1 mth | at 2-8˚C |

# Toxoplasma gondii Abs (Toxo-IgG,IgM)

## Summary

Toxoplasmosis is a common infection caused by the protozoan parasite Toxoplasma gondii. The infection is mainly acquired by ingestion of food or water that is contaminated by mature oocysts shed by cats or by undercooked meat containing tissue cysts.

Primary maternal Toxoplasma infection occurring during pregnancy may lead to severe damage of the fetus as the parasite can be transmitted across the placenta. The majority of infants with congenital infection does not present clinical symptoms at birth but may develop severe sequelae later in life like mental and psychomotor retardation, chorioretinitis and hearing loss. The determination of IgM antibodies is a valuable indicator in the diagnosis of recent infection while IgG antibodies will be found in old stages of manifested autoimmune disorders.

| | | |
|---|---|---|
| Method | ECLIA | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | IgM: Negative: <1.0 | |
| | IgG: Negative: <1 IU/mL | |
| | Grayzone: 1-3 IU/mL | |
| | Reactive: >3 IU/mL | |
| Sample stability | 3 d | at room temperature |
| | 3 wk | at 2-8°C |
| | 3 mth | at -20°C. |

# Transferrin

## Summary

Transferrin is used as a sensitive indicator of functional iron depletion in cases of iron deficiency, provides a better indication of the homozygous genotype than does Ferritin, in conjunction with Ferritin it gives conclusive prediction of the iron overloading in patients with chronic liver disease.

| | | |
|---|---|---|
| Method | Turbidimetric | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | 200-360 mg/dL | |
| Conversion factors | mg/dL x 0.01 = g/L | g/L x 12.6 = µmol/L |
| | g/L x 100 = mg/L | µmol x 0.0796 = g/L |
| Sample stability | 4 mth | at room temperature |
| | 8 mth | at 2-8˚C |
| | 6 mth | at -20˚C |

# Treponema Pallidum Hemagglutination Assay (TPHA)

## Summary

TPHA is a screening test for the recognition of total antibodies against Treponema pallidum which is a gram negative bacterium which causes the sexually transmitted disease syphilis. False positive results are observed in patients with immune disorders.

| | | |
|---|---|---|
| Method | IHA | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Negative | |
| Sample stability | 5 d | at 2-8 ˚C |
| | Long term storage | at -20˚C |

# Triglycerides

## Summary

The determination of triglycerides is utilized in the diagnosis and treatment of patients having diabetes mellitus, nephrosis, liver obstruction, lipid metabolism disorders and numerous other endocrine diseases.

| | | |
|---|---|---|
| Remarks | Fasting | |
| Method | Enzymatic colorimetric | |
| TAT | Dy/AB | |
| Sample type | S / EP / CP | |
| Expected value | < 2.26 mmol/L | |
| Sample stability | 2 d | at room temperature |
| | 1 wk | at 2-8˚C |
| | < 1yr | at -20˚C |

# Troponin I (Qualtitative,Quantitative)

## Summary

Troponin I is a highly sensitive marker for myocardial damage. Cardiac TnI allows differentiating between skeletal muscle lesions (e.g. rhabdomyolysis and polytraumatism) and myocardial injury. In cases of acute myocardial infarction (AMI), levels in serum rise about 3-6 hours after the onset of cardiac symptoms, peak at 12-16 hours, and can remain elevated for 4-9 days. Elevated levels have also been reported in cases of unstable angina pectoris (UAP) and congestive heart failure (CHF).Cardiac Troponin I is a well-established prognostic marker which can predict the near-, mid- and even long-term outcome of patients with acute coronary syndrome (ACS).

| | | |
|---|---|---|
| Method | ECLIA (Quantitative) | |
| | ICT (Qualitative) | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Negative: <0.01 ng/mL | |
| Sample stability | 2 h | at room temperature |
| | 1 yr | at -20 °C |

# Troponin T (Qualitative, Quantitative)

## Summary

Tropnin T Serves as an aid in the diagnosis of patients with suspected myocardial cell damage, as in the case of coronary syndromes (detection and ruling out of acute myocardial infarction and subacute myocardial infarction, determination of infarction size, risk stratification of patients with unstable angina pectoris by detecting minimal damages to the myocardium). It also helps in the diagnosis of inflammatory diseases of the myocardium (myocarditis) in cases of mechanical, chemical and electrical damage to the myocardial (contusion, PTCA, heart valve, heart operation, biopsy, cardiotoxic substances and catheter ablation).

| | |
|---|---|
| Remarks | Do not cool nor freeze the sample. |
| Method | ECLIA (Quantitative) |
| | ICT (Qualitative) |
| TAT | Dy/AB |
| Sample type | HWB/ S |
| Expected value | Negative: <0.01 ng/mL |
| Sample stability | 1 d at room temperature |
| | 1 yr at -20 ˚C |

# Unconjugated Estriol (FE3),Free

## Summary

Estriol (E3) is only produced in significant amounts during pregnancy as it is made by the placenta from 16-Hydroxydehydroepiandrosterone sulfate (16-OH DHEAS), an androgen steroid made in the fetal liver and adrenal glands. The placenta converts 16-OH DHEAS to estriol, and is the predominant site of estriol synthesis. Abnormally low levels of “unconjugated estriol” in a pregnant woman indicate chromosomal or congenital anomalies like Down syndrome or Edward’s syndrome. It is included as part of the Triple test and Quadruple test for screening fetal anomalies.

| | |
|---|---|
| Method | ECLIA |
| TAT | Dy/A |
| Sample type | S/HP/EP |

Expected value

| Gestational wk | Central 95% Range (ng/mL) |
|---|---|
| 27 | 2.9 – 12.7 |
| 28 | 3.3 – 14.3 |
| 29 | 3.7 – 16.0 |
| 30 | 4.1 – 17.9 |
| 31 | 4.6 – 19.9 |
| 32 | 5.1 - 22.1 |
| 33 | 5.7 – 24.4 |
| 34 | 6.3 - 27.0 |

| Expected value | Gestational wk | Central 95% Range (ng/mL) |
|---|---|---|
| | 35 | 7.0 – 29.7 |
| | 36 | 7.7 - >30 |
| | 37 | 8.5 - >30 |
| | 38 | 9.3 - >30 |
| | 39 | 10.2 - >30 |
| | 40 | 11.1 - >30 |

| | |
|---|---|
| Conversion factors | ng/mL x 3.467 = nmol/L |

| | | |
|---|---|---|
| Sample stability | 1 wk | at 2-8 ˚C |
| | 6 mth | at -20 ˚C |

# Urea

## Summary

Determination of blood urea nitrogen is the most widely used screening test for renal function. When used in conjunction with serum creatinine determinations it can aid in the differential diagnosis of the three types of azotemia: prerenal, renal and postrenal.

Elevations in blood urea nitrogen concentration are seen in inadequate renal perfusion, shock, diminished blood volume (prerenal causes), chronic nephritis, nephrosclerosis, tubular necrosis, glomerular nephritis (renal causes) and urinary tract obstruction (postrenal causes). Transient elevations may also be seen during periods of high protein intake. Unpredictable levels occur with liver diseases.

| | | | |
|---|---|---|---|
| Remarks | Morning spot urine | | |
| Method | Kinetic | | |
| TAT | Dy/AB | | |
| Sample type | S/HP/EP/FP | | |
| | U24/US | | |
| Expected value | S/HP/EP/FP | : | <65 yr: <50 mg/dL |
| | | | >65 yr: <71 mg/dL |
| | US | : | 847-2967 mg/dL |
| | 24-U | : | <35000 mg/d |

| | | |
|---|---|---|
| Conversion factors | Urea | Mg/dL x0.167=mmol/L |
| | | Mmol/L x 6.006 = mg/dL |
| | Urea/ U | g/24 h x 16.7 = mmol/d |
| | | mmol/d x0.06 =g/24 h |
| | | g/g crea x 1.883 = mol/mol crea |
| | | mol/mol crea x 0.531 =g/g crea |

| | | |
|---|---|---|
| Sample stability In U | 2 d | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 4 wk | at -20 ˚C |

| | | |
|---|---|---|
| Sample stability In S/EP/HP/FP | 1 wk | at room temperature |
| | 1 wk | at 2-8 ˚C |
| | 1 yr | at -20 ˚C |

# Uric Acid (UA)

## Summary

Uric acid measurements are used in the diagnosis and treatment of numerous renal and metabolic disorders, including renal failure, gout, leukemia, psoriasis, starvation or other wasting conditions, and of patients receiving cytotoxic drugs.

| | | | |
|---|---|---|---|
| Method | Enzymatic /Colorimetric | | |
| TAT | Dy/AB | | |
| Sample type | S/HP/EP/US/24U | | |
| Expected value | US: | 37-92 mg/dL | |
| | 24U: | 200-1000 mg/dL | |
| | S/HP/EP: | Male (♂)<65 yr | <7 mg/dL |
| | | Male (♂)>65 yr | <8.4 mg/dL |
| | | Female (♀) | <5.7 mg/dL |
| Conversion factors | mg/dL x 59.5 =μmol/L | | |
| | μmol/L x 0.0168 = mg/dL | | |
| Sample stability in U | 4 d | at room temperature | |
| | Unstable | at 2-8 ˚C | |
| | Unstable | at -20 ˚C | |
| Sample stability in S/EP/HP: | 3 d | at room temperature | |
| | 1 wk | at 2-8 ˚C | |
| | 6 mth | at -20 ˚C | |

# Vancomycin( VANC)

## Summary

Vancomycin is a complex glycopeptide antibiotic, which has been used to treat penicillinase-producing staphylococci. It is the drug of choice for the treatment of methicillin and related beta lactam antibiotic resistant Staphylococcus aureus as well as for the treatment of serious gram-positive infections where allergies to penicillin or cephalosporin play a role. Vancomycin is also used in the treatment of antibiotic-induced enterocolitis associated with Clostridium difficile and streptococcal or enterococcal endocarditis, the latter in conjunction with an aminoglycoside, when penicillin or ampicillin is not an option. Monitoring of peak and trough serum or plasma levels is necessary due to potentially serious side effects including ototoxicity, nephrotoxicity, phlebitis, and reversible neutropenia .

| | |
|---|---|
| Remarks | State through or peak level |
| Method | FP |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Peak Level: 25-40 μg/mL |
| | Trough Level: 5-10 μg/mL |
| Conversion factors | μg/mL x 0.690 = μmol/L |
| | μmol/L x 1.449 = μg/mL |
| Sample stability | 2 h at room temperature |
| | 2 d at 2-8 ˚C |
| | 1 mth at -20 ˚C |

# Varicella Zoster (Chicken Pox) Abs (IgA,IgM,IgG)

## Summary

Varicella-Zoster Virus (human herpes virus 3, HHV-3) is usually transmitted in respiratory a secretion which is acute and endemic disease follows primary contact with the virus, most commonly affected are children between 2 and 6 years of age. The course of disease is usually mild and complicated only in immunocompromised children. Rare fatal cases show multiple necrotic lesions in brain, lung (varicella pneumonia), kidneys (hemorrhagic nephritis), spleen, bone marrow, and occasionally in the intestinal tract.

IgG antibody to varicella-zoster detected, which indicate a past varicella-zoster infection, while IgM/IgA indicate a current infection to disease.

| | | |
|---|---|---|
| Method | ELISA | |
| TAT | Dy/AB | |
| Sample type | S | |
| Expected value | Negative: <1:10 | |
| Sample stability | 2 d | at room temperature |
| | 2wk | at 2-8 ˚C |
| | 1 yr | at - 20 ˚C |

# Vitamin A (Retinol)

## Summary

Vitamin A is a protecting substance for the complete ectoderm. It is important for the skin, eyes, mucous membranes of the respiratory system and gastrointestinal and urogenital tract. The determination of vitamin A levels is used to diagnose deficiency or over-dosage of the vitamin in the body.

Vitamin A deficiency leads to night blindness (deficieny of the rhodopsin synthesis), hemeralopia and reduced tear formation ("dry eye") as the first symptoms of a vitamin A defect. Deficiency also causes ceratoconjunctivitis sicca, Biot's spots and in extreme cases causes keratomalacia; Skin Hyperkeratosis with stratum corneum thickening, formation of little keratinin clots in the follicle ostiae and sweat gland ductuli (phrynoderma), in addition to squamous epithelium metaplasia at non cornifying mucous membranes, xerostomia, dysphagia and bluishly changed lips. Chronic vitamin A defects lead to a higher incidence of carcinoma (antineoplastic effect of the retinoids).

Hypervitaminosis leads to Nausea, headache, diffuse desquamation and a rise in transaminases and lipids.

Vitamin A is naturally found in carrots, peas, string beans, asparagus, bananas, milk and dairy products, butter, egg yolk, liver, kidneys and fish oil.

| | |
|---|---|
| Method | HPLC |
| TAT | 3 d |
| Sample type | S |
| Expected value | Male (♀): 425 – 831 µg/L<br>Female (♂): 402 – 697 µg/L |
| Sample stability | 1 mth at 2-8˚C<br>1 yr at -20˚C |

# Vitamin B1 (Thiamine)

## Summary

Thiamine is of importance for all organs using pyruvate and lactates as an energy source or show a high carbohydrate turnover or a high requirement for acetylic groups (nerve cells).

The determination of vitamin B1 levels is used to diagnose the deficiency or the over-dosage of the vitamin up take in the body. The deficiency of the vitamin shows neurological symptoms that include polyneuropathy, neuritis, areflexia, paresis vocal cord paralysis, neuromuscular degeneration (“dry form” of Beri-Beri) and cardiac symptoms including cardiac insufficiency, tachycardia, edema of lower extremities and effusions in serous caves (“wet form” of Beri-Beri).

Hypervitaminosis occurs very rarely and presents clinical symptoms that include > 100-fold of normal dosis headache, convulsions, paralysis and cardial rhythm disorders.

Thiamine is naturally found in grain or grain products, flour, meat, liver, yeast, egg, milk, vegetables and roots. Thiamine is destroyed easily by heating (cooking).

| | | |
|---|---|---|
| Method | HPLC | |
| TAT | 3 d | |
| Sample type | EWB | |
| Expected value | 20 – 100 ng/mL | |
| Sample stability | 4 h | at 2-8˚C |
| | 6 mth | at -20˚C |

# Vitamin B6 (Pyridoxalphosphat)

## Summary

The determination of vitamin B6 levels is used to diagnose the deficiency or the over-dosage of the vitamin up take in the body.

Vitamin B6 deficiency leads to skin eczema-like changes similar to seborrhoic dermatitis around the eyes, nose and mouth; cheilosis; glossitis. In pediatrics, B6 deficiency causes cerebral cramp attacks, Vit-B6 depending anemias, xanthurenaciduria, cysthathioninuria, homocystinuria, hyperornithinemia and oxalosis type I can appear in newborns (deficiency during pregnancy).

Hypervitaminosis occurs > 2 g/day and leads to neuropathy with ataxia, cerebral convulsions with changes in the EEGs, hypochromic anaemias and seborrhoic dermatitis.

The daily requirement is 2.0-2.6 mg and in pregnancy > 4 mg.

Vitamin B6 is naturally found in liver, kidney, brain, meat, fish, egg yolk, yeast, grains and rice.

| | | |
|---|---|---|
| Method | HPLC | |
| TAT | 10-14 d | |
| Sample type | EWB | |
| Expected value | 7.0 - 30.0 ng/mL | |
| Sample stability | 4 h | at 2-8˚C |
| | 2 mth | at -20˚C |

# Vitamin D (25-Hydroxyvitamin)

## Summary

Vitamin D is essential for bone health. In children, severe deficiency leads to bone-malformation, known as rickets. Milder degrees of insufficiency are believed to cause reduced efficiency in the utilization of dietary calcium. Vitamin D deficiency causes muscle weakness; reduced Vitamin D has also been associated with falls and fractures not explained by reduced bone density. Vitamin D deficiency is a common cause of secondary hyperparathyroidism. Elevations of PTH levels, especially in elderly vitamin D deficient adults, can result in osteomalacia, increased bone turnover, reduced bone mass and risk of bone fractures. Low vitamin D (25-OH) concentrations are also associated with lower bone mineral density. In conjunction with other clinical data, the results may be used as an aid in the assessment of bone metabolism. So far, vitamin D has been shown to affect the expression of over 200 different genes.

| | |
|---|---|
| Remark | Non freezed samples & Glass tube. |
| Method | ECLIA |
| TAT | Dy/AB |
| Sample type | S |
| Expected value | Desirable Healthy Concentration: >75 nmol/L |
| Conversion factors | nmol/L x 0.40 = ng/mL<br>ng/mL x 2.50 = nmol/L |
| Sample stability | 3 d at room temperature |

# Vitamin H (Biotin)

## Summary

Vitamin H belongs to the water-soluble, essential vitamins and is supplied by food as biotinlysins (biocytins).

Clinical symptoms: Skin: (seborrhoic) dermatitis predominately of the extremities, cheilosis, alopecia, signs of immune deficiency.

| | | |
|---|---|---|
| Remarks | Fasting, Glass tube | |
| Method | HPLC | |
| TAT | 2 d | |
| Sample type | S | |
| Expected value | 0.82-2.87 nmol/L | |
| Sample stability | 1 mth | at 2-8˚C |
| | 2 yr | at -20˚C |

# Vitamin K (Menaquinone)

## Summary

Menaquinone is considered as the effective K-vitamin (vitamin K2). It can be partly replaced by phylloquinone, which is supplied through vegetables (intact intestinal flora). In deficiencies, impaired coagulation and hemorrhagiae occur. In new born from vitamin K deficient mothers, intracranial bleedings can occur by hypoprothrombinemia. Chronic intestinal disease or malabsorption can lead to vitamin-K deficiency. Over-dosage symptoms are not described; however, the effect of oral anticoagulants such as coumarin can be reduced.

The daily requirement is approx. 0-1 mg/day.

| | | |
|---|---|---|
| Remarks | Dispatch light-protected. | |
| Method | LCMS | |
| TAT | 10-14 d | |
| Sample type | S | |
| Expected value | 50 - 900 ng/L (fas.) | |
| | <1800 ng/L (pp.) | |
| Sample stability | 1 mth | at 2-8˚C |
| | 6 mth | at -20˚C |

# Y Chromosome Deletion

## Summary

The Y chromosome is one of two sex-determining chromosomes (gonosomes) in mammals, including humans. DNA in the Y chromosome is passed from father to son.

Y chromosome infertility is usually caused by deletions of genetic material in regions of the Y chromosome called azoospermia factor (AZF) A, B, or C. Genes in these regions are believed to provide instructions for making proteins involved in sperm cell development.

| | |
|---|---|
| Method | PCR |
| TAT | 3 d |
| Sample type | EWB/CWB |
| Expected value | Refer To Report |
| Sample stability | 3 d at room temperature |
| | 1 wk at 2-8˚C |

# Zinc

## Summary

Zinc is necessary for the proper functioning of the immune system. This essential trace element is required for the activity of over 300 enzymes and is involved in most major metabolic pathways. The immune system depends on zinc in almost every aspect. Zinc bolsters the immune system and makes wounds heal faster. It is integral to the growth and maintenance of body tissues. It also plays a major role in the development of fetuses and the growth of children.

General signs of zinc deficiency are sleep disturbances, diarrhea, poor appetite, unhealthy skin, behavioral problems, inability to heal wounds, and chronic infections. Some individuals who have consistently low zinc values may have a genetic condition called pyroluria. This condition causes abnormally high kryptopyrroles in the urine which binds to zinc making it unavailable to the body. Ingesting too much zinc causes stomach cramps, nausea and vomiting. Very high doses over several months can cause anaemia through reducing the absorption of copper (another essential element for the body). It can also damage the pancreas and decrease the levels of high density lipoprotein (HDL i.e. the good form of cholesterol).

| | | |
|---|---|---|
| Remarks | 1% hemolysis results in a Zinc increase of 15%. | |
| Method | IF | |
| TAT | Dy/AB | |
| Sample type | S/HP/U24 | |
| Expected value | Newborn | 49.5-99.7 μg/dL |
| | Children | 63.8-110 μg/dL |
| | Male (♂) | 72.6-127 μg/dL |
| | Female (♀) | 70-114 μg/dL |
| | 24U | 300-800 μg/dL |

# Urine Analysis

A regular urinalysis often includes the following tests:

| | |
|---|---|
| **Color** | Many things affect urine color, including fluid balance, diet, medicines, and diseases. How dark or light the color is tells you how much water is in it. Vitamin B supplements can turn urine bright yellow. Some medicines, blackberries, beets, rhubarb, or blood in the urine can turn urine red-brown. |
| **Specific gravity** | This checks the amount of substances in the urine. It also shows how well the kidneys balance the amount of water in urine. The higher the specific gravity, the more solid material is in the urine. |
| **pH** | The pH is a measure of how acidic or alkaline (basic) the urine is. A urine pH of 4 is strongly acidic, 7 is neutral (neither acidic nor alkaline), and 9 is strongly alkaline. Sometimes the pH of urine is affected by certain treatments. |
| **Protein** | Protein normally isn›t found in the urine. Fever, hard exercise,pregnancy, and some diseases, especially kidney disease, may cause protein to be in the urine. |
| **Glucose** | Glucose is the type of sugar found in blood. Normally there is very little or no glucose in urine. When the blood sugar level is very high, as in uncontrolled diabetes, the sugar spills over into the urine. Glucose can also be found in urine when the kidneys are damaged or diseased. |
| **Nitrites** | Bacteria that cause a urinary tract infection (UTI) make an enzyme that changes urinary nitrates to nitrites. Nitrites in urine show a UTI is present. |
| **Leukocyte esterase (WBC esterase).** | Leukocyte esterase shows leukocytes (white blood cells [WBCs]) in the urine. WBCs in the urine may mean a UTI is present. |
| **Ketones** | Large amounts of ketones in the urine may mean a very serious condition, diabetic ketoacidosis, is present. A diet low in sugars and starches (carbohydrates), starvation, or severe vomiting may also cause ketones to be in the urine. |

| Urine Microscopic analysis | |
|---|---|
| **Red or white blood cells.** | Blood cells aren›t found in urine normally. Inflammation, disease, or injury to the kidneys, ureters, bladder, or urethra can cause blood in urine. Strenuous exercise, such as running a marathon, can also cause blood in the urine. White blood cells may be a sign of infection or kidney disease. |
| **Casts** | Some types of kidney disease can cause plugs of material (called casts) to form in tiny tubes in the kidneys. The casts then get flushed out in the urine. Casts can be made of red or white blood cells, waxy or fatty substances, or protein. The type of cast in the urine can help show what type of kidney disease may be present. |
| **Crystals** | Healthy people often have only a few crystals in their urine. A large number of crystals, or certain types of crystals, may mean kidney stones are present or there is a problem with how the body is using food (metabolism). |
| **Bacteria, yeast cells, or parasites.** | There are no bacteria, yeast cells, orparasites in urine normally. If these are present, it can mean you have an infection. |
| **Squamous cells** | The presence of squamous cells may mean that the sample is not as pure as it needs to be. These cells do not mean there is a medical problem, but your doctor may ask that you give another urine sample. |

# Stool Analysis

A stool analysis is a series of tests done on a stool (feces) sample to help diagnose certain conditions affecting the digestive tract. These conditions can include infection (such as from parasites, viruses, or bacteria), poor nutrient absorption, or cancer.

Stool analysis is done to:

- Help identify diseases of the digestive tract, liver, and pancreas. Certainenzymes (such as trypsin or elastase) may be evaluated in the stool to help determine how well the pancreas is functioning.
- Help find the cause of symptoms affecting the digestive tract, including prolonged diarrhea, bloody diarrhea, an increased amount of gas, nausea, vomiting, loss of appetite, bloating, abdominal pain and cramping, and fever.
- Screen for colon cancer by checking for hidden (occult) blood.
- Look for Ova, parasites, such as pinworms or *Giardia lamblia*.
- Look for the cause of an infection, such as bacteria, a fungus, or a virus.

| Stool analysis | |
|---|---|
| **Normal:** | The stool appears brown, soft, and well-formed in consistency.<br>The stool does not contain blood, mucus, pus, undigested meat fibers, harmful bacteria, viruses, fungi, or parasites.<br>The stool is shaped like a tube. |
| **Abnormal:** | The stool is black, red, white, yellow, or green.<br>The stool is liquid or very hard.<br>There is too much stool. |

# Culture

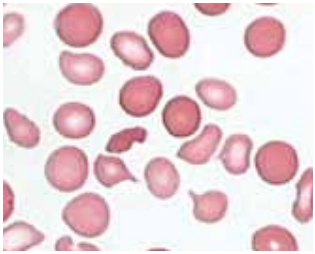
| Sample: Ascitic Fluid | | Sample: Blood | |
|---|---|---|---|
| Summary | Pathogenic Microorganisms | Summary | Pathogenic Microorganisms |
| A fluid culture detects bacteria in only 42%-65% of patients who have neutrocytic ascites and suspected spontaneous bacterial peritonitis. | Bacteroides species<br>Clostridium perfringens<br>Entrococci<br>Escherichia coli<br>Klepbsiella strains<br>Pseudomonas aeruginosa<br>Staphylococcus aureus<br>Streptococcus agalactiae<br>Streptococcus pneumoniea<br>Streptococcus pyogenes<br>Viridians Streptococci | Blood cultures should be obtained (prior to initiation of antimicrobial therapy) for any patient in whom there is suspicion of bacteremia or fungemia, including hospitalized patients and selected outpatients with fever and leukocytosis or leukopenia. However, a normal white blood cell count does not rule out bacteremia. Circumstances in which blood cultures are especially important include known or suspected sepsis, meningitis, osteomyelitis, arthritis, endocarditis, peritonitis, pneumonia, and fever of unknown origin. | Bacteroides species<br>Haemophilus influenzae<br>Haemophilus influenzae<br>Klepbsiella pneumoniea<br>Neisseria menengitidis<br>Pseudomonas aeruginosa<br>Salmonella species<br>Staphylococcus aureus<br>Streptococcus pneumoniea<br>Yerrsinia pestis |
| Sample: Ear swab | | Sample: Effusions (Hydrocele Fluid) | |
| Summary | Pathogenic Microorganisms | Summary | Pathogenic Microorganisms |
| This test detects and identifies bacteria from fluid or discharge found in the middle ear. It is used to help treat acute otitis media (inflammation of the middle ear) and chronic purulent otitis media. | Escherichia coli<br>Proteus species<br>Pseudomonas aeruginosa<br>Streptococcus agalactiae<br>Streptococcus pneumoniea<br>Streptococcus pyogenes<br>Vincent's organisms<br>Yerrsinia pestis | A hydrocele is a collection of fluid in the scrotum. Most develop for no apparent reason, are harmless, and can be left alone. If needed, a small operation can usually cure the problem. In a small number of cases, a hydrocele is due to an underlying problem with a testicle (testis). | Bancrofti microfilareiae<br>Brugia species<br>Occasionally Wuchereria |

| Sample: Effusion (Pleural and Pericardial Fluids) | | Sample:Effusion (Synovial Fluid) | |
|---|---|---|---|
| Summary | Pathogenic Microorganisms | Summary | Pathogenic Microorganisms |
| This test is used to invest inflammatory conditions (including possible bacterial, viral and fungal infections); and suspected primary or secondary neoplasm. | Mycoplasma pneumonia<br>Chlamydia trachomatis<br>Mycobacterium tuberculosis<br>Staphylococcus aureus<br>Streptococcus pneumonia<br>Enterobacteriacae and other gram negative Bacilli<br>Cryptococcus neoformans<br>Staphylococcus aureus<br>Streptococcus pneumonia<br>Haemophilus influenza<br>Enterobacteriacae<br>Pseudomonas spp.<br>Anaerobic bacteria<br>Mycobacterium tuberculosis<br>Actinomyces spp.<br>Coccidoides immitis<br>Aspergillus spp.<br>Candida spp.<br>Histoplasma capsulatum | Synovial fluid analysis may be diagnostic in patients with bacterial infections or crystal-induced synovitis. | Actinomycestes<br>Bacteroides species<br>Brucella species<br>Neisseria gonorrhoeae<br>Neisseria menengitidis<br>Salmonella species<br>Staphylococcus aureus<br>Streptococcus pneumoniea<br>Streptococcus pyogenes<br>Yerrsinia pestis |

| Sample:Feces (Stool) | | Sample :Nasopharangeal Aspirates and Nasal Swabs | |
|---|---|---|---|
| Summary | Pathogenic Microorganisms | Summary | Pathogenic Microorganisms |
| A stool analysis is a series of tests done on a stool (feces) sample to help diagnose certain conditions affecting the digestive tract, liver, and pancreas. Certainenzymes (such as trypsin or elastase) may be evaluated in the stool to help determine how well the pancreas is functioning.These conditions can include infection (such as from parasites, viruses, or bacteria), poor nutrient absorption, or cancer | Bacillus cereus<br>Campylobacter species<br>Clostridium difficile<br>Clostridium perfringens (A and C)<br>Escherichia coli<br>Other vibrio species<br>Salmonella species<br>Shigella species<br>Staphylococcus aureus<br>Vibrio cholera O1<br>Yerrsinia enterocolitica | A nasopharyngeal culture is a test used to diagnose upper respiratory infections. These are infections that cause symptoms such as a cough or runny nose. | Bordetella parapertussis<br>Bordetella pertussis<br>Corynebacterium diphtheria<br>Haemophilus influenza<br>Klebsiella species<br>Neisseria menengitidis<br>Streptococcus pneumoniea |
| Sample:Pus (Wounds, Absesses, Burns and Sinuses) | | Sample: Sputum | |
| Summary | Pathogenic Microorganisms | Summary | Pathogenic Microorganisms |
| This test is used To isolate and identify aerobic and anaerobic pathogenic organisms from pus specimen and sensitivity test. | Actinomyces israelii<br>Bacteroides species<br>Candida albicans<br>Clostridium perfringens<br>E.coli Enterococcus species Fusobactrium speciesKlebsiella species<br>Mycobacterium tuberculosis Nocardia species<br>Peptostreptococcus species Proteus species<br>Pseudomonas aeruginosa<br>Staphylococcus aureus<br>Streptococcus pyogenes | A sputum culture is a test to detect and identify bacteria or fungi (plural of fungus) that are infecting the lungs or breathing passages. Symptoms of a lung infection may include difficulty breathing, pain when breathing, or a cough that produces bloody or greenish brown sputum | Chlamydia pneumoniea<br>Haemophilus influenzae<br>Klepbsiella pneumoniea<br>Mycobacterium tuberculosis<br>Pseudomonas aeruginosa<br>Staphylococcus aureus<br>Streptococcus pneumoniea |

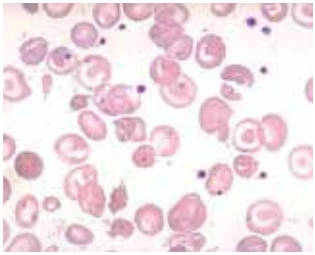
| Sample: Throat and mouth | | Sample: Urine | |
|---|---|---|---|
| Summary | Pathogenic Microorganisms | Summary | Pathogenic Microorganisms |
| A throat culture is a test to check for a bacterial or fungal infection in the throat. | Candida albicans,<br>Corynebacterium diphtheria<br>Corynebacterium ulcerans<br>Neisseria meningitides<br>Streptococcus pyogenes<br>Vincent's organisms (Borrelia Vincent, Leptotrichia buccalis, and Bbacteroides melaninogenicus) | A urine culture is a test to find and identify germs (usually bacteria) that may be causing a urinary tract infection (UTI). Urine in the bladder normally is sterile—it does not contain any bacteria or other organisms (such as fungi). But bacteria can enter the urethra and cause an infection. | Acinetobacter species<br>Citrobacter species<br>Enterobacter species<br>Enterococci<br>Epidedimis<br>Escherichia coli<br>Haemolytic streptococci<br>Haemolytic streptococci<br>Klebsiella species<br>Klebsiella strains<br>Neisseria gonorrhoeae<br>Proteus species<br>Pseudomonas aeruginosa<br>Pseudomonas species<br>Salmonella paratyphi<br>Salmonella typhi<br>Serratia species<br>Staphylococcus saprophyticus<br>Staphylococcus saprophyticus |

# Blood Films: Red blood cells



Anisochromia

Uneven coloration of erythrocytes can be found in: combined iron and folic acid deficiency, patients with sideropenia, especially at the beginning of the treatment (blood smear), and during blood transfusion.

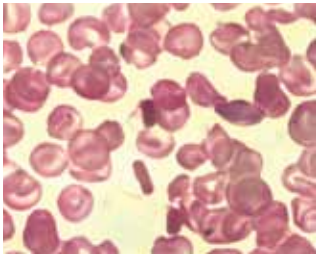


Beta-Thalassemia

A variety of genetic blood disorders are known as thalassemias, all of which are characterized by abnormal hemoglobin production.

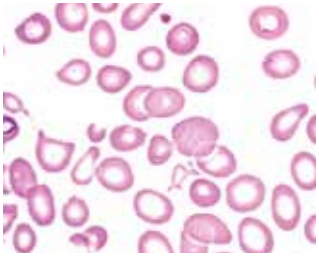
Elliptocytosis

Elliptocytosis is a hereditary disorder of the red blood cells (RBCs). In this condition, the RBCs assume an elliptical shape, rather than the typical round shape.



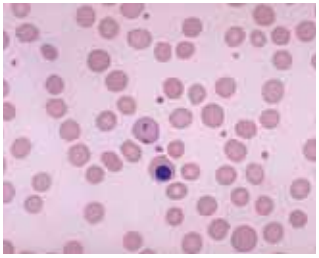
Hemoglobin C disease

- Rhomboid, tetragonal or rod shaped crystals of dense staining hemoglobin that often distort the cell.
- Crystals are found in patients with CC or SC disease, particularly after splenectomy.
- Typical hexagon crystals (shown here) are seen in CC disease only.



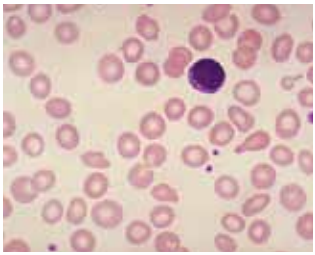
Hemoglobin H disease

An abnormal hemoglobin composed of four beta chains. It is caused by the reduced synthesis of the alpha chain. This abnormality results in Alpha Thalassemia.



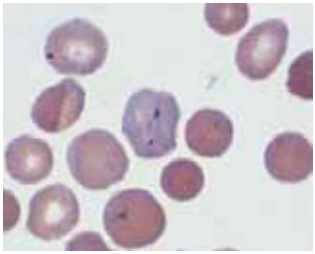
Howell-Jolly bodies

The RBC in the center of the field contains several Howell-Jolly bodies, or inclusions of nuclear chromatin remnants.



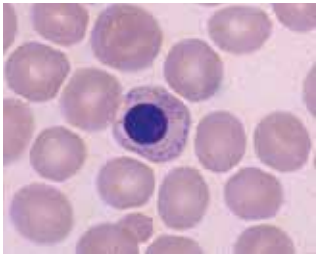
Iron Deficiency

The most common cause for a hypochromic microcytic anemia is iron deficiency. Persons most at risk are children and women in reproductive years (from menstrual blood loss and from pregnancy).



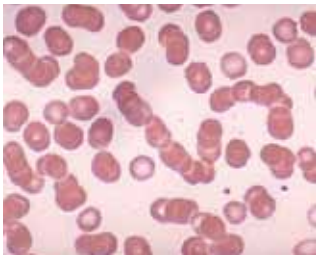
Malaria

Malaria parasites are visible within the red blood cells. They are stained a dark bluish color.



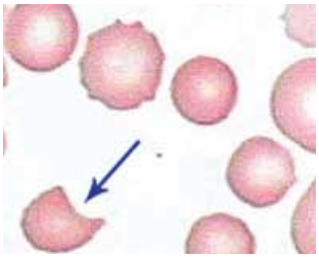
Nucleated RBC

The nucleated RBC in the center contains basophilic stippling of the cytoplasm. This suggests a toxic injury to the bone marrow, such as with lead poisoning. Such stippling may also appear with severe anemia.



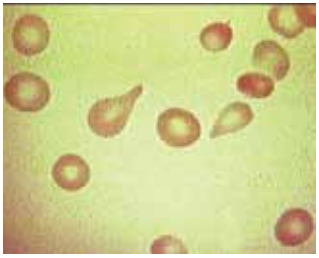
Rouleaux Formation

The RBC's here have stacked together in long chains. This is known as "Rouleaux Formation" and it happens with increased serum proteins, particularly fibrinogen and globulins.



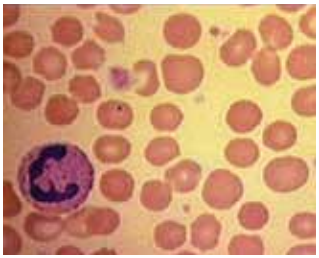
Schizocytes

Found in patients with congenital hemolyticanemia and megaloblastic anemias. Schizocytes are also formed in hemolytic anemias due to mechanical stress (micro-angiopathy, heart valve prosthesis, severe burns), cancer and myelofibrosis.



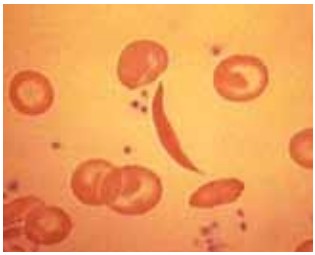
Sickle cell

Sickle cell anemia is an inherited blood disease in which the red blood cells produce abnormal pigment (hemoglobin). The abnormal hemoglobin causes deformity of the red blood cells into crescent or sickle-shapes.



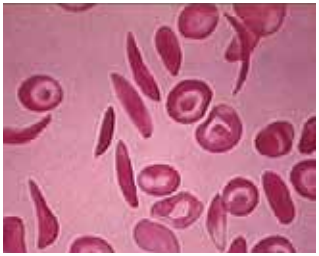
Spherocytosis

Spherocytosis is a hereditary disorder of the Red Blood Cells (RBCs), which may be associated with a mild anemia. Typically, the affected RBCs are small, spherically shaped, and lack the light centers seen in normal, round RBCs.



Target cells

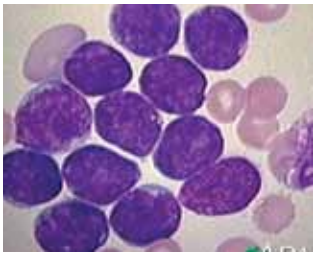
These abnormal Red Blood Cells (RBCs) resemble targets. These cells are seen in association with some forms of anemia, and following the removal of the spleen (splenectomy).



Tear-drop shape

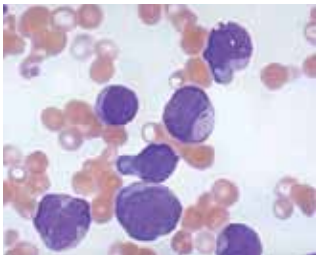
This photomicrograph shows one of the abnormal shapes that Red Blood Cells (RBCs) may assume, a tear-drop shape. Normally, RBCs are round.

# Blood Films: Leukemia



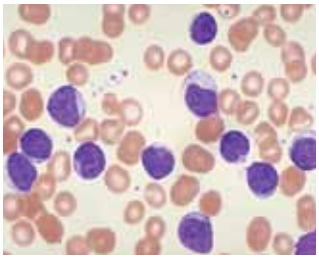
Acute Lymphocytic Leukemia

This picture shows the darkly-stained lymph cells (lymphoblasts) seen in Acute Lymphocytic Leukemia (ALL), the most common type of childhood leukemia.



Acute Myelogenous Leukemia

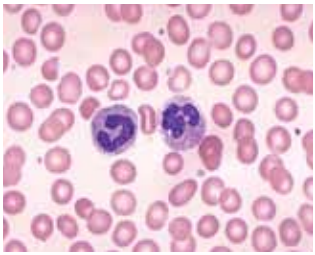
Here are very large, immature myeloblasts with many nucleoli. A distincitve feature of these blasts is a linear red “Auer rod” composed of crystallized granules. These findings are typical for Acute Myelogenous Leukemia (AML) that is most prevalent in young adults.



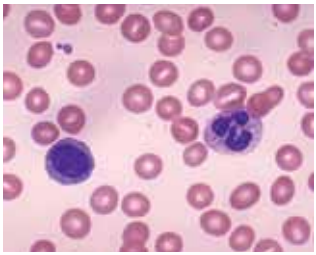
Chronic Lymphocytic Leukemia

These mature lymphocytes are increased markedly in number. They are indicative of Chronic Lymphocytic Leukemia (CML), a disease most often seen in older adults. This disease responds poorly to treatment, but it is indolent.

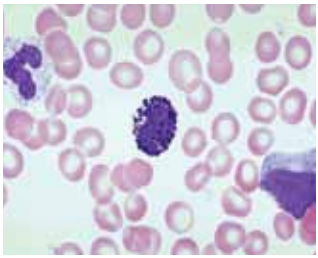
# Normal Blood Cells



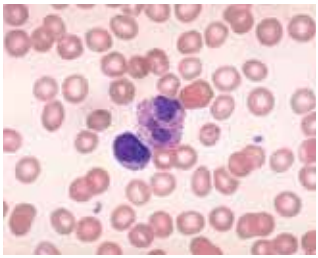
Normal Red Blood Cell



Normal White Blood Cells

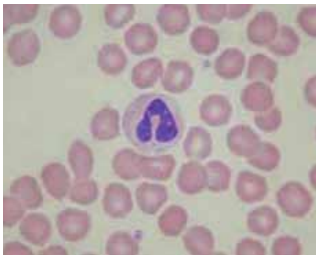


Normal Basophile



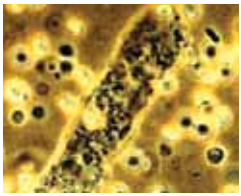
Normal Eosinophile

Normal Monocyte

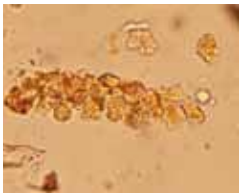


Normal Neutrophil

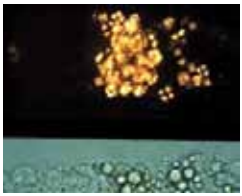
# Urine Casts



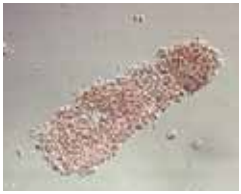
Bacterial Casts



Billirubin Casts



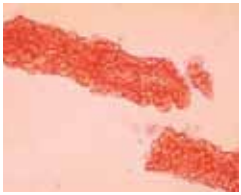
Fatty casts



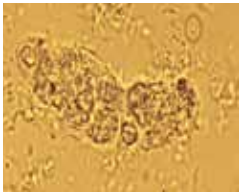
Granular Casts

Hyaline Casts

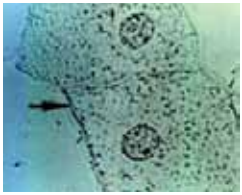
Leukocytes casts



Red blood casts

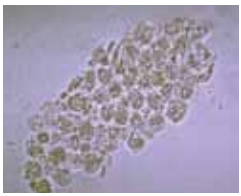


Renal tubular Casts

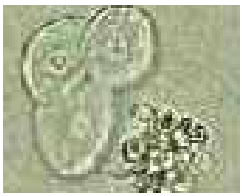


Squamous Cells Casts

Waxy Casts

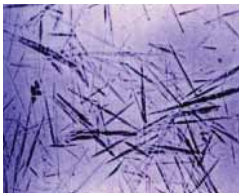


White blood cell casts

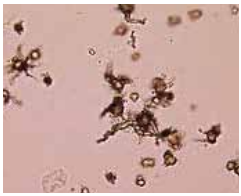


Yeast Casts

# Urine Crystals

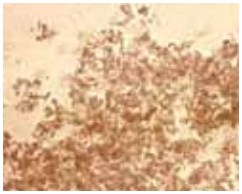


Acyclovir crystals

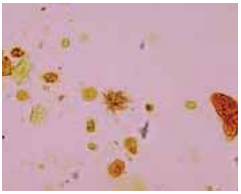


Ammonium biurate crystals

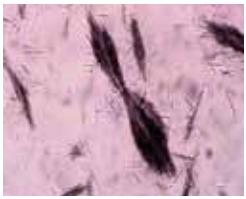
Amorphous phosphates Crystals



Amorphous urates Crystals



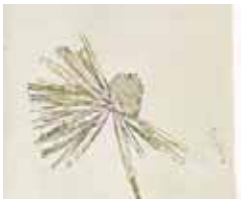
Bilirubin crystals



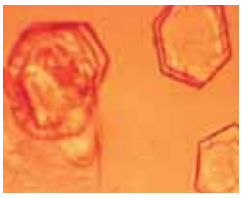
Amoxicillin crystals

Calcium carbonate crystals

Calcium Oxalate Crystals

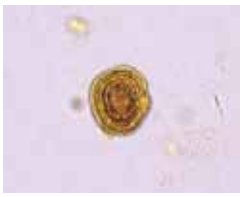


Calcium phosphate



Cystine crystals

Cholesterol crystals

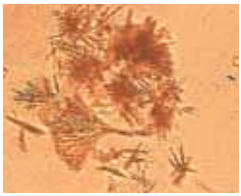


Leucine crystals

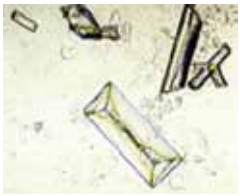
# Urine Crystals

struvite crystals

Sulfonamide crystals

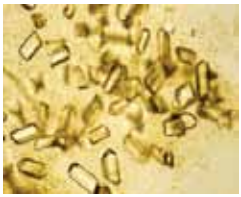


Sulfa crystals



Triple phosphate crystals

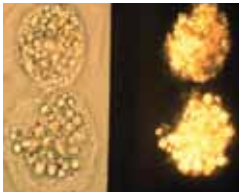
Tyrosine crystals



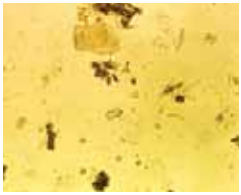
Uric acid Crystals

# Urine Artifacts and Contaminants

Cotton Fiber

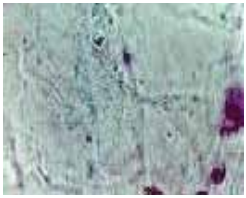


Fatty bodies

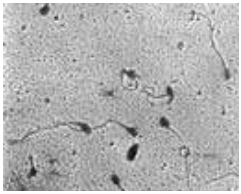


Feces

Hair

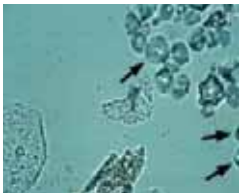


Mucous

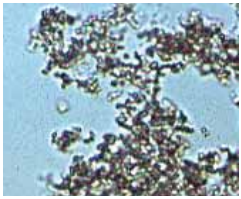


Sperm

Starch



Talcum Powder



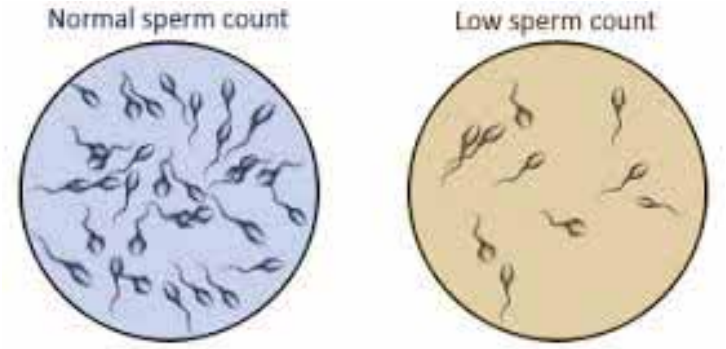
Urinary Artifacts

# Seminal Fluid: Sperm Count

Sperm count is a measure of the number of spermatozoa per ejaculation or per measured amount of semen, used as an indication of a man's fertility.

Sperm count is considered lower than normal if it is fewer than 15 million sperm per milliliter of semen. A low sperm count is also called oligospermia. A complete absence of sperm is called azoospermia.

The normal average of sperm count is between 20 and 40 million per milliliter.

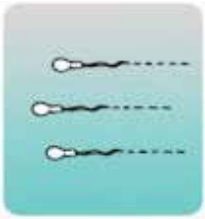



Sperm count

# Seminal Fluid: Sperm Motility

Sperm motility describes the ability of sperm to move properly through the female reproductive tract (internal fertilization) or through water (external fertilization) to reach the egg.

- **Grade a**: Sperm with progressive motility. These are the strongest and swim fast in a straight line. Sometimes it is also denoted motility IV.
- **Grade b**: (non-linear motility): These also move forward but tend to travel in a curved or crooked motion. Sometimes also denoted motility III.
- **Grade c**: These have non-progressive motility because they do not move forward despite the fact that they move their tails. Sometimes also denoted motility II.
- **Grade d**: These are immotile and fail to move at all. Sometimes also denoted motility I.



Grade (a)

Grade (b)

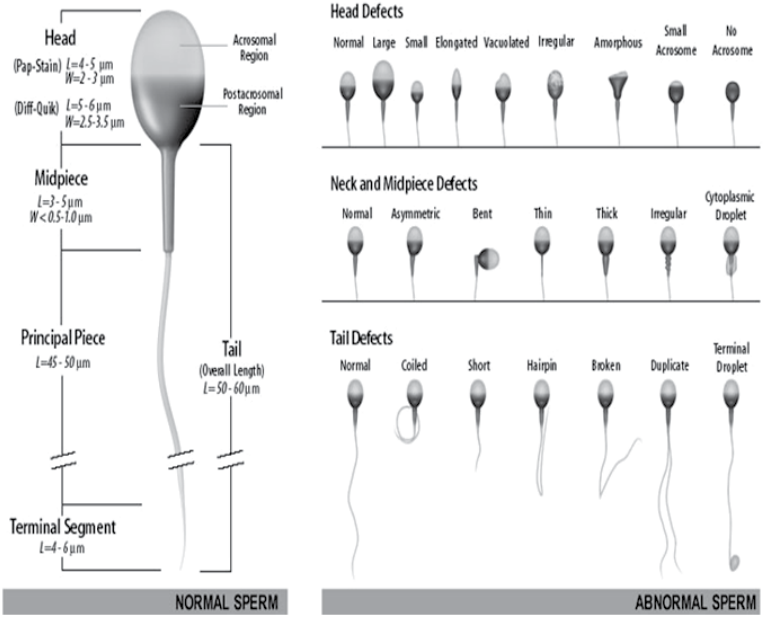
Grade (c )

Grade (d)

# Seminal Fluid: Sperm Morphology

Sperm morphology - the size and shape of sperm - is one of the things checked in a semen analysis for male infertility.

Sperm morphology results are reported as the percentage of sperm that appear normal when semen is viewed under a microscope. Normal sperm have an oval head with a long tail. Abnormal sperm have head or tail defects - such as a large or misshapen head or a crooked or double tail. These defects may affect the ability of the sperm to reach and penetrate an egg.



# Abbreviations

# Abbreviations

| | | | |
|---|---|---|---|
| 2d | Number of Days | FP | Fluorided Plasma |
| 24U | 24 Hour Urine Collection | FP | Fluorescence Polarisation |
| AAS | Atomabsorption | GC | Gaschromatography |
| AF | Amniotic Fluids | HP | Heparinized Plasma |
| AGGL | Agglutination | HPLC | High Performance Liquid Chromatography |
| BF | Body Fluids | HVS | Vaginal Swab |
| BM | Bone Marrow | HWB | Heparinized Whole Blood |
| BW | Bronchial Wash | ICT | Immuno Chromatography Test |
| CP | Citrated Plasma | IF | Immune Fluorescence |
| CSF | Cerebro Spinal Fluid | IHA | Indirect Haemagglutination Assay |
| CVS | Chorionic Villous Sample | IPMS | Inductive Coupled Plasma Mass Spectrom |
| CWB | Citrated Whole Blood | LCMS | Liquid Chromatography Mass Spectrometry |
| D | Different Samples | LIA | Luminescence Immuno Assay |
| DB | Dried Blood | LMP | Last Menstruation Period |
| DOB | Date Of Birth | MPA | Mulituplex Bead Array |
| Dy/A | Daily and done only once at morning. | NT | Neutralisation Test |
| Dy/AB | Daily and done randomly | PA | Particle Agglutination |
| ECLIA | Electrochemiluminescence Immuno Assay | PCR | Polymerase Chain Reaction |
| EIA | Enzyme Immuno Assay | PHO | Photometry |
| ELISA | Enzym Linked Sandwich Assay | RIA | Radio Immuno Assay |
| ELPH | Electrophoresis | S | Serum |
| EnS | Endo Servical Swab | SF | Seminal Fluid |
| EP | EDTA Plasma | SP | Sputum |
| ES | Ear Swab | SPHO | Spectral Photometry |
| EWB | EDTA Whole Blood | ST | Stool |
| FCM | Flowcytometry | UrS | Urethral Swab |
| FISH | Fluorescence in-situ Hybridisation | US | Spot Urine |

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Attaelmannan M, Levinson SS. Understanding and Identifying Monoclonal Gammopathy. Clin Chem 2000;46:1230-1238.

Ruibal A, Encabo G, Martinez-Miralles E, et al. CA 125 seric levels in nonmalignant pathologies. Bull Cancer (Paris) 1984;71(2):145-146.

Aardal E, Holm AC. Cortisol in Saliva - Reference Ranges and Relation to Cortisol in Serum. Eur J Clin Chem Clin Biochem 1995;33:927-932.

Abicht K, Samalouti V, Kroll W. Multicenter evaluation of new GGT and ALP reagents with new reference standardization and determination of 37 °C reference intervals. Clin Chem Lab Med 2001;39(Suppl.):S346[abstract].

Albers JJ, Marcovina SM, Kennedy H. International Federation of Clinical Chemistry Standardization Project for Measurements of Apolipoproteins A-I and B. II. Evaluation and Selection of Candidate Reference Materials. Clin Chem 1992;38:658-662.

Albertini A, Arosio P, Chiancone E, Drysdale J, eds. Ferritins and isoferritins as biochemical markers. Amsterdam/New York/Oxford: Elsevier, 1984.

Alexander E, Buyon J.P., Provost T.T., Guarnieri T. Anti-Ro/SS-A antibodies in the pathophysiology of congenital heart block in neonatal lupus syndrome, an experimantal model. Arthritis and Rheumatism;Vol. 35 No.2; 176-189; 1

Ameer B, Greenblatt DJ. Acetaminophen. Ann Intern Med 1977;87:202.

Anderson B, Sawyer DB. Predicting and preventing the cardiotoxicity of cancer therapy. Expert Rev Cardiovasc Ther 2008;6(7):1023-1033.

Arcieri GM, et al. Clinical research experience with gentamicin. Incidence of adverse reactions. Med J Aust 1970;1(Suppl.):30-34.

Arlt W. Androgen Therapy in Women. Eur J Endocrinol 2006;154(1):1-11.

Armbruster DA, Schwarzhoff RH, Hubster EC, et al. Enzyme immunoassay, kinetic microparticle immunoassay, radioimmunoassay, and fluorescence polarization immunoassay compared for drugs-of-abuse screening. Clin Chem 1993;39:2137-2146.

Arnett FC, Edworthy SM, Bloch DA et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum 1988;31:315-324.

Arnoux D, Boutiere B, Sanmarco M. [Antiphospholipid antibodies: clinical significance and biological diagnosis] Ann Biol Clin (Paris). 2000 Sep-Oct;58(5):557-74.

Arnqvist H, Olsson PO, von Schenck H. Free and Total Insulin as Determined after Precipitation with Polyethylene Glycol: Analytical Characteristics and Effects of Sample Handling and Storage. Clin Chem 1987;33(1):93-96.

# Bibliography


Bablok W et al. A General Regression Procedure for Method Transformation. J Clin Chem Clin Biochem 1988;26:783-790.

Bachoric P. Measurement of Low-Density-Lipoprotein. 245-263. In: Handbook of Lipoprotein Testing (eds. Rifai, Warnick and Dominiczak), 2nd edition, AACC press, 2000.

Barakat RM, Ekins P. Assay of vitamin B12 in blood: A simple method. Lancet 1961;2(7192):25-26.

Barhan D, Trinder P. An improved colour reagent for the determination of blood glucose by the oxidase system. Analyst 97;1972:142.

Barker JD, de Carle DJ, Annras S. Chronic excessive acetaminophen use and liver damage. Ann Intern Med 1977;87:299.

Barré-Sinoussi F, Chermann JC, Rey F, et al. Isolation of a T-lymphotropic Retrovirus from a Patient at Risk for Acquired Immune Deficiency Syndrome (AIDS). Science 1983;220:868-871.

Bartels H, Böhmer M. Micro-determination of creatinine. Clin Chim Acta 1971;32:81-85.

Bast RC, Klug TL, St. John E, et al. A radioimmunoassay using a monoclonal antibody to monitor the course of epithelial ovarian cancer. N Engl J Med 1983;309:883-887.

Bates M, Brandle J, Casaretto E, et al. An Abuscreen immunoassay for opiates in urine on the COBAS MIRA automated analyzer. Amer Acad Forensic Sci. Abstract 1991;37(6):1000.

Battino O, Croci D, Granata T, Bernardi G, Monza G. Changes in unbound and total valproic acid concentrations after replacement of carbamazepine with oxcarbazepine Ther Drug Monit 1992;14:376-379.

Bauer JD. Hemoglobin, porphyrin, and iron metabolism. In: Kaplan LA, Pesce AJ, eds. Clinical Chemistry, theory, analysis, and correlation. St. Louis: Mosby Company 1984:611-655.

Beastall GH, Ferguson KM, O'Reilly DSJ, et al. Assays for follicle stimulating hormone and luteinizing hormone: Guidelines for the provision of a clinical biochemistry service. Ann Clin Biochem 1987;24:246-262.

Beauregard C, Dickstein G, Lacroix A. Classic and recent etiologies of Cushing's syndrome: diagnosis and therapy. Treat Endocrinol 2002;1:79-94.

Becker DV, Bigos ST, Gaitan E, et al. Optimal use of blood tests for assessment of thyroid function (letter). JAMA 1993;269:273.

Becker W, Rapp W, Schenk HG, Störiko K. Methoden zur quantitativen Bestimmung von Plasmaproteinen durch Immunpräzipitation. Z Klin Chem Klin Biochem 1968;6:113-122.

Beniviste R, Davies J. Mechanisms of antibiotic resistance in bacteria. J Ann Rev Biochem 1973;42:471.

Bergenfelz A, Isaksson A, Lindblom P, et al. Measurement of parathyroid hormone in patients with

# Bibliography

primary hyperparathyroidism undergoing first and reoperative surgery. Br J Surg 1998;85:1129-1132.

Berggard I, Bearn AG. Isolation and Properties of a Low Molecular Weight β2-Globulin Occurring in Human Biological Fluids. J Biol Chem 1968;243:4095.

Bertolaccini ML, Atsumi T, Khamashta MA, et al. Autoantibodies to human prothrombin and clinical manifestations in 207 patients with systemic lupus erythematosus. J Rheumatol, Jun 1998, 25(6) p1104-8

BIO RAD Quantaphase B-12/Folate Radioassay Instruction Manual, März 1995.

Black HR, Quallich H, Gareleck CB. Racial differences in serum creatine kinase levels. Am J Med 1986;81:479-487.

Black J, et al. Pharmacology of gentamicin, a new broad-spectrum antibiotic. Ag Chemothe 1963:138.

Blankfield RP. Can natriuretic peptide levels predict the cardiovascular complications of COX-2 inhibitors and nonsteroidal anti-inflammatory drugs? J Am Board Fam Med 2006;19:178-182.

Blind E. Measurement of Intact Parathyroid Hormone by an Extracting Two-Site Immunometric Assay. In: Schmidt-Gayk H, Armbruster FP, Bouillon R, (eds). Calcium regulating hormones, vitamin D metabolites, an cyclic AMP. Heidelberg: Springer 1990:151.

Bobic B, Sibalic D, Djurkovic-Djakovic O. High levels of IgM Antibodies Specific for Toxoplasma gondii in Pregnancy 12 Years after Primary Toxoplasma Infection. Gynecol Obstet Invest 1991;31:182-184.

Boege F. Bence Jones-Proteine. J Lab Med 1999;23:477-482.

Bojunga J, Sarrazin C, Hess G, et al. Elevated plasma levels of N-terminal pro-brain natriuretic peptide in patients with chronic hepatitis C during interferon-based antiviral therapy. World J Gastroenterol 2006;12(36):5875-5877.

Bormer OP, Thrane-Steen K. Epitope group specificity of six immunoassays for carcino-embryonic antigen. Tumor Biol 1991;12:9-15. Guder WG, Narayanan S, Wisser H, et al. List of Analytes; Preanalytical Variables. Brochure in: Samples: From the Patient to the Laboratory. GIT-Verlag, Darmstadt 1996:10. ISBN 3-928865-22-6.

Borque L, Barozzi D, Ferrari L et al. The Determination of Rheumatoid Factors by an Immunoturbidimetric Assay on Boehringer Mannheim/Hitachi Analysis Systems. Klin Lab 1994;40:445-453.

Brandt, J.T.,D.A.Triplett, B. Alving, and I.M. Scharrer. Criteria for the diagnosis of lupus anticoagulants: an update [Review].Thromb. Haemost., 1995, 74:1185-1190.

Braun S, Röschenthaler F, Jarausch J, Klein G. Analyte Stability of CK-MB Activity and cTnT in ICU Patient Serum and Heparin Plasma. Poster presented at Medica 2004, Düsseldorf. (Roche Diagnostics GmbH No. 04587979990).

# Bibliography


Bretzel, R.G. Hypertonie, Mikroalbuminurie und Insulinresistenz bei Diabetes mellitus. Wiener Klin. Wochenschr. 1994; 106: 774 - 792.

Brewer HB, Gregg RE, Hoeg JM, et al. Apolipoproteins and Lipoproteins in Human Plasma: an overview. Clin Chem 1988;34:B4-B8.

Brewer JA, Tank ES. Yolk sac tumors and alpha-fetoprotein in first year of life. Urology 1993;42,1:79-80.

Buchthal F, Svensmark O. Serum concentration of diphenylhydantion (phenytoin) and phenobarbital and their relation to therapeutic and toxic effects. Psychiatr Neurol Neurochir 1971;(74):117-136.

Buffone GJ, Lewis SA, Josefsohn M et al. Chemical and immunochemical measurement of total iron-binding capacity compared. Clin Chem 1978;24:1788-1791.

Bunn HF, Gabbay KH, Gallop PM. The glycosylation of hemoglobin: relevance to diabetes mellitus. Science 1978;200:21-27.

Burrows B, Martinez FD, Halonen M, et al. Association of Asthma with Serum IgE Levels and Skin-Test Reactivity to allergens. N Engl J Med 1989;320:271-277.

Burtis CA, Ashwood ER, Bruns DE ed. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics, 4th ed. St. Louis, Missouri; Elsevier Saunders, 2006;1190.

Busch MP, Lee LLL, Satten GA, et al. Time course of detection of viral and serologic markers preceding human immunodeficiency virus type 1 seroconversion: implication for screening of blood and tissue donors. Transfusion 1995;35:91-97.

Canick JA, Saller DN Jr. Maternal serum screening for aneuploidy and open fetal defects. Obstet Gynecol Clin North Am 1993;20,3:443-454.

CDC. Prevention of Perinatal Transmission of Hepatitis B Virus: Prenatal Screening of all Pregnant Women for Hepatitis-B-Surface Antigen: Recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1988;37:341-355.

Chang J, Hoke C, Ettinger B, et al. Evaluation and interference study of hemoglobin A1c measured by turbidimetric inhibition immunoassay. Am J Clin Pathol 1998;109:274-278.

Chen YT, Luderer AA, Thiel RP, et al. Using proportions of free to total prostate-specific antigen, age, and total prostate-specific antigen to predict the probability of prostate cancer. Urology 1996;47:518-524.

Chevenne D, Letailleur A, Trivin F, et al. Effect of Hemolysis on the Concentration of Insulin in Serum Determined by RIA and IRMA. Clin Chem 1998;44(2):354-356.

Chiu SK, Collier CP, Clark AF, et al. Salivary cortisol on Roche Elecsys immunoassay system: pilot biological variations studies. Clin Biochem 2003;36:211-214.

# Bibliography


Clark PM. Assays for insulin, proinsulin(s) and C-peptide. Ann Clin Biochem 1999;36(5):541-564.

Clarke JT, Libke, RD, Regamey C, Kirby WM. Comparative pharmacokinetics of amikacin and kanamycin. Clin Pharm Ther 1974;15:610.

Clavel F, Guétard D, Brun-Vézinet F, et al. Isolation of a New Human Retrovirus from West Africa Patients with AIDS. Science 1986;233:343-346.

Coburn AF, Pauli RH. Limited observations on the antistreptolysin titer in relation to latitude. J Immunol 1935;29:515–521.

Cohen RJ, Haffejee Z, Steele GS, et al. Advanced Prostate Cancer With Normal Serum Prostate-Specific Antigen Values. Arch Pathol Lab Med 1994;118:1123-1126.

Cohn JS, McNamara JR, Schaefer EJ. Lipoprotein Cholesterol Concentrations in the Plasma of Human Subjects as Measured in the Fed and Fasted States. Clin Chem 1988;34:2456-2459.

Colcher D, Horan Hand P, Nuti M, et al. A Spectrum of monoclonal antibodies reactive with human mammary tumor cells. Proc Natl Acad Sci 1981;78(5):3199-3208.

Committee on patient preparation and specimen handling. Clinical Laboratory Handbook for Patient Preparation and Specimen Handling. Fascicle IV. Slokie, IL: College of American Pathologists; 1985.

Cook FV. Vancomycin revisited. Ann Intern Med 1978;88:813-818.

Cooper LZ, Alford CA. Rubella, in Infectious Diseases of the Fetus & Newborn Infant,: 5th Ed 2001, pp 347-88, ed Remington JS & Klein JO, Philadelphia: W.B. Saunders.

Costello-Boerrigter LC, Redfield MM, Rodeheffer RJ, et al. NT-proBNP is superior to BNP in the detection of Left Ventricular systolic dysfunction in the community. J Card Fail 2004;10(4) Suppl. 4.

Council Directive (2000/54/EC). Official Journal of the European Communities No. L262 from Oct. 17, 2000. rates for Down's Syndrome screening: How precisely can they be estimated and what factors influence their value? Statistics Medicine 1997;16:1481-1495.

Couroucé-Pauty AM, Plancon A, Soulier JP. Distribution of HBsAg Subtypes in the World. Vox Sang 1983;44:197-211.

Cowie MR, Jourdain P, Maisel A, et al. Clinical applications of B-type natriuretic peptide (BNP) testing. Eur Heart J 2003;24,1710-1718.

Cramer JA, Mattson RH, Bennett DM, Swick, CT. Variable free and total valproic acid concentrations in sole- and multi-drug therapy Ther Drug Monit 1986;8(4):411-415.

Crossley KB, Landesman B, Zaske D. An outbreak of infections caused by strains of Staphylococcus aureus resistant to methicillin and aminoglycosides. J Infect Dis 1979;139:273-279.

Cuckle HS. Improved parameters for risk estimation in Down's syndrome screening. Prenat Diagn 1995;15:1057-1065.

# Bibliography


Cuckle HS. Improved parameters for risk estimation in Down's syndrome screening. Prenat Diagn 1995;15:1057-1065.

Cunningham BA, Wang JL, Berggard I, et al. The complete amino acid sequence of β2-microglobulin. Biochemistry 1973;12:4811-4822.

Curtis GDW, Kraak WAG, Mitchell RG. Comparison of latex and haemolysin tests for determination of anti-streptolysin O (ASO) antibodies. J Clin Pathol 1988;41:1331–1333.

Da Fonseca-Wollheim F. Deamidation of glutamine by increased plasma γ-glutamyltransferase is a source of rapid ammonia formation in blood and plasma specimens. Clin Chem 1990;36:1479-1482.

Dandliker WB, Feigen GA. Quantification of the antigen-antibody reaction by the polarization of fluorescence. Biochem Biophys Res Comm 1961;5:299-304.

Daoud E, Bodor G, Weaver Ch, et al. (Washington University Case Conference) CA-125 Concentrations in Malignant and Nonmalignant Disease. Clin Chem 1991;37(11):1968-1974.

Daoud E, Bodor G, Weaver Ch, et al. (Washington University Case Conference) CA-125 Concentrations in Malignant and Nonmalignant Disease. Clin Chem 1991;37(11):1968-1974.

Darbar D, Davidson NC, Gillespie N, et al. Diagnostic value of B-Type Natriuretic Peptide Concentrations in Patients With Acute Myocardial Infarction. A J Cardiol 1996;78:284-287.

Dati F, Ringel KP. Reference values for serum IgE in healthy non-atopic children and adults. Clin Chem 1982;28(7):1556.

Davis HM, Zurawski VR Jr, Bast RC Jr, et al. Characterization of the CA 125 antigen associated with human epithelial ovarian carcinomas. Cancer Research 1986;46:6143-6148.

de Jong G, von Dijk IP, van Eijk HG. The biology of transferrin. Clin Chim Acta 1990;190:1-46.

Demers LM, Spencer CA. Laboratory support for the diagnosis and monitoring of thyroid disease. National Academy of Clinical Biochemistry, 2002;Section 3B.

Denis F, Leonard G, Sangare A, et al. Comparison of 10 Enzyme Immunoassays for Detection of Antibody to Human Immunodeficiency Virus Type 2 in West African Sera. J Clin Microbiol 1988;26:1000-1004.

Dericks-Tan JSE, Siedentopf HG, Taubert HD. Discordant Prolactin Values obtained with Different Immunoassays in an infertile Patient. J Lab Med 1997;21(9):465-470.

Detection and Quantification of Rubella IgG Antibody: Evaluation and Performance Criteria for Multiple Component Test Products, Specimen Handling, and Use of Test Products in the Clinical Laboratory; Approved Guideline. NCCLS Publication; 1997;Vol17,No17.

Dieterich,W. Et al. Serum antibodies in Celiac Disease. Clin. Lab., 2000, 46:361-364.

Domke, N. and Siegert, G. Phospholipidantikörper und ihre klinische Bedeutung. Zeitschr. Klin.Med. 1988; Heft 16: 1399 - 1401.

# Bibliography


Doumas BT, Kwok-Cheung PP, Perry BW et al. Candidate Reference Method for Determination of Total Bilirubin in Serum, Albumin standards and the measurement of serum albumin with bromcresol green: Development and Validation. Clin Chem 1985;31:1779-1789.

Dripps RD, Comroe JH Jr. Clinical studies on morphine I: The immediate effect of morphine administered intravenously and intramuscularly upon the respiration of normal man. Anesthesiology 1945;6:462.

Dubois T, Bisagni-Faure A,Coste J,Mavoungou E,Menkes CJ, Russo-Marie F, Rothhut B. High levels of antibodies to annexins V and VI in patients with rheumatoid arthritis. J Rheumatol. 1995 Jul;22(7):1230-4.

Duc J, Morel B, Peitrequin R, Frei PC. Identification of monoclonal gammopathies: a comparison of immunofixation, immunoelectrophoresis and measurements of kappa- and lambda-immunoglobulin levels. J Clin Lab Immunol 1988;26:141-146.

Dunn RT, Foster LB. Radioassay of serum Folate. Clin Chem 1973;19:1101-1105.

Dunstan FDJ, Gray JC, Nix ABJ, et al. Detection rates and false positive rates for Down's Syndrome screening: How precisely can they be Estimated and what factors influence their value Statistics Medicine 1997;16:1481-1495.

ECCLS. Determination of the catalytic activity concentration in serum of L-aspartate aminotransferase (EC 2.6.1.1, ASAT). Klin Chem Mitt 1989;20:198-204.

Eikelboom JW, Lonn E, Genest J Jr, et al. Homocystein(e)ine and cardiovascular disease: A critical review of the epidemiologic evidence. Ann Intern Med 1999; 131:363-375.

Ekins RP. Measurement of free hormones in blood. Endocr Rev 1990;11:5.

Elefath D, Cooney J, McDaniel R, et al. Effects of Frozen Storage of Serum on the Level of 22 Chemistry Analytes. Clin Chem 1991;37:931.

Enders G, Knotek F, Pacher U. Comparison of various serological methods and diagnostic kits for the detection of acute, recent and previous Rubella infection, vaccination and congenital infections. J Med Virol 1985;16:219-232.

Engelmann M, Landgraf R, Wotjak CT. The hypothalamic-neurohypophysial system regulates the hypothalamic-pituitary-adrenal axis under stress: an old concept revisited. Front Neuroendocrinol 2004;25:132-149.

Erali M, Bigelow RB, Meikle AW. ELISA for thyroglobulin in serum: recovery studies to evaluate autoantibody interference and reliability of thyroglobulin values. Clin Chem 1996;42(5):766-770.

European Patent 1577673 assigned to F. Hoffmann-LaRoche AG and Roche Diagnostics GmbH. The use of BNP-type peptides and ANP-type peptides for assessing the risk of suffering from a cardiovascular complication as a consequence of volume overload. Patent granted 30.07.2008.

# Bibliography


Fahie-Wilson M, Brunsden P, Surrey J, et al. Macroprolactin and the Roche Elecsys Prolactin Assay: Characteristics of the Reaction and Detection by Precipitation with Polyethylene Glycol. Clin Chem 2000;46:1993-1995.

Feldt-Rasmussen U. Analytical and clinical performance goals for testing autoantibodies to thyroperoxidase, thyroglobulin, and thyrotropin receptor. Clin Chem 1996;42(1):160-163.

Feldt-Rasmussen U. Analytical and clinical performance goals for testing autoantibodies to thyroperoxidase, thyroglobulin, and thyrotropin receptor. Clin Chem 1996;42(1):160-163.

Feltkamp T.E.W.. Antinuclear antibody determination in a routine laboratory. Ann.Rheum.Dis.;Vol.55; 723-727; 1996.

Ferguson JW, Richard JJ, O'Laughlin JW, Banks CV. Simultaneous spectrophotometric determination of calcium and magnesium with Chlorophosphonazo III. Anal Chem 1964;36: 796-799.

Fesus, L., And M. Piacentini. Transglutaminase 2: an enigmatic enzyme with diverse functions. Trends Biochem. Sci., 2002, 27:534-539.

Filella X, Fuster J, Molina R, et al. TAG-72, CA 19-9 and CEA as tumor markers in gastric cancer. Acta Oncologica 1994;33(7):747-751.

Franco RS. Ferritin. In: Pesce AJ, Kaplan LA, eds. Methods in clinical chemistry. St. Louis/ Washington/Toronto: CV Mosby Company, 1987: 1240-1242.

Frank EL, Moulton L, Little RR, et al. Effects of hemoglobin C and S traits on seven glycated hemoglobin methods. Clin Chem 2000;46 (6):864-867

Friedewald WF, Levy RI, Frederickson DS. Estimation of LDL-Cholesterol Concentration without Use of the Preparative Ultracentrifuge. Clin Chem 1972;18:499-502.

Fritsche HA Jr, Gelder FB. Serum tumor markers for pancreatic carcinoma. Immunol-Ser 1990;53:289-296.

Fritzler M.J.. Clinical relevance of autoantibodies in systemic rheumatic diseases. Mol.Biol. Rep.;Vol.23; 133-145; 1996.

Frösner G, Schomerus H, Wiedmann KH, et al. Diagnostic significance of quantitative HBsAg determination in acute and chronic hepatitis B infection. Eur J Clin Microbiol 1982;1:52-58.

Gallo RC, Salahuddin SZ, Popovic M, et al. Frequent Detection and Isolation of cytopathic Retroviruses (HTLV-III) from Patients with AIDS and at Risk for AIDS. Science 1984;224:500-503.

Gardner, et al. Serum thyroglobulin in normal subjects and patients with hyperthyroidism due to Graves` disease: effects of T3, iodine, 131J, and antithyroid drugs. Clin. Endocr. (Oxf.) 1979; 11: 585 - 594.

Gargouri Y, Julien R, Bois A et al. Studies on the detergent inhibition of pancreatic lipase activity. J

of Lipid Research 1983;24:1336-1342.

Geraci JE, Hermans PE. Vancomycin. Mayo Clin Proc 1983;58:88-91.

Gerbitz KD. Pankreatische B-Zellen Peptide: Kinetik und Konzentration von Proinsulin, Insulin und C-Peptid in Plasma und Urin, Probleme der Meßmethoden, klinische Aussage und Literaturübersicht. J Clin Chem Clin Biochem 1980;18(6):313-326.

Gerson B, ed. Essentials of Therapeutic Drug Monitoring New York: IGAKU-SHOIN: 1983:215-226.

Giannitsis E. Rationale for testing the cardiovascular risk for patients with COX-2 inhibitors on the basis of biomarker NT-proBNP. Clin Lab 2005;51(1-2):63-83.

Gibney J, Smith TP, McKenna TJ. Clinical relevance of macroprolactin. Clin Endocrinol 2005;62:633-643.

Gidal BE, Collins DM, Beinlich BR. Apparent valproic acid neurotoxicity in a hypoalbuminemic patient The Annals of Pharmacotherapy 1993;27(1):32-35.

Glick MR, Ryder KW, Jackson SA. Graphical Comparisons of Interferences in Clinical Chemistry Instrumentation. Clin Chem 1986; 32:470-475.

Glli M, Beretta G, Daldossi M, et al. Different anticoagulant and immunological properties of anti-prothrombin antibodies in patients with antiphospholipid antibodies. Thromb Haemost, Mar 1997, 77(3) p486-91

Gold P, Freedman SO. Demonstration of tumor-specific antigen in human colonic carcinomata. J Exp Med 1965;121:(3)439.

Goldfien A, Monroe SE. Ovaries. In: Greenspan FS, Baxter JD (eds), Basic & Clinical Endocrinology, 4th edition, Appleton & Lange, USA 1994; Chapter 10:419-470.

Goldstein DE, Little RR, Lorenz RA, et al. Tests of glycemia in diabetes. Diabetes Care 1995;18:896-909.

Gröschl M, Rauh M, Dörr HG. Circadian Rhythm of Salivary Cortisol, 17α-Hydroxyprogesterone, and Progesterone in Healthy Children. Clin Chem 2003;49(10):1688-1691.

Grützner, F.J.Diagnostik mit b2-Mikroglobulin. Inn.Med. 1982; 9: 45-56.

Guadagni F, Roselli M, Cosimelli M, et al. TAG-72 Expression and its Role in the Biological Evaluation of Human Colorectal Cancer. Anticancer Res 1996;16:2141-2148.

Gunter EW, Bowman BA, Caudill SP, et al. Results of an international round robin for serum and whole-blood folate. Clin Chem 1996;42(10):1689-1694.

Gutcho S, Mansbach L. Simultaneous radioassay of serum Folate and folic acid. Clin Chem 1977;23:1609-1614.

# Bibliography


Gutmann I, Wahlefeld A. Methods of Enzymatic Analysis. 2nd ed. Bergmeyer HU, ed. New York, NY: Academic Press Inc 1974:1464.

Hafner G, Endler T, Oppitz M et al. Effects of standardization with the new international reference preparation for proteins in human serum on method comparability and reference values. Clin Lab 1995;41:743-748.

Hammarström S, Shively JE, Paxton RJ, et al. Antigenic sites in carcinoembryonic antigen. Cancer Research 1989;49:4852-4858.

Haning RV. Using DHEAS to monitor androgen disorders. Contemp Ob/Gyn 1981;18(9):117-131.

Hasholzner U, Baumgartner L, Stieber P, et al. A. Significance of the tumor markers CA 125 II, CA 72-4, CASA and CYFRA 21-1 in ovarian carcinoma. Anticancer Res 1994;14(6B):2743-2746.

Haustein SV, Mack E, Starling JR, et al. The role of intra-operative parathyroid hormone testing in patients with tertiary hyperparathyroidism after renal transplatation. Surgery 2005;138(6):1066-1071.

Heringlake M, Heide C, Bahlmann L, et al. Effects of tilting and volume loading on plasma levels and urinary excretion of relaxin, NT-proANP, and NT-proBNP in male volunteers. J Appl Physiol 2004;97:173-179.

Hietarinta M., Lassila O..Clinical significance of antinuclear antibodies in systemic rheumatic disease. Ann.Med.;Vol.28; 283-291; 1996.

Hilkens J, Buijs F, Hilgers J, et al. Monoclonal antibodies against human milk-fat globule membranes detecting differentiation antigens of the mammary gland and its tumors. Int J Cancer 1984;34:197-206.

Hillmann G. Continuous Photometric Measurement of Prostate Phosphatase Activity. Zeitschrift fuer Klinische Chemie und Klinische Biochemie, 1971, vol. 9, no. 3, p. 273-274.

Hoermann R, Berger P, Spoettl G, et al. Immunological Recognition and Clinical Significance of Nicked Human Chorionic Gonadotropin in Testicular Cancer. Clin Chem 1994;40(12):2306-2312.

Hoffman BF. In: Fisch C, Surawicz B, eds. Effects of digitalis on electrical activity of cardiac fibers. Digitalis. New York, NY: Grune and Stratton 1969:93-109.

Hofmann W, Guder WG. A diagnostic program for quantitative analysis of proteinurea. J Clin Chem Clin Biochem 1989;27:589-600.

Hohnadel DC, Koller A. Urine protein total. In: Pesce AJ, Kaplan LA, editors. Methods in clinical chemistry, St. Louis, 1987, Mosby.

Hollinger FB. Hepatitis B virus. In Fields BN, Knipe DM (eds). Virology ed. 2 New York Raven Press 1990;2:2171-2236.

# Bibliography


Holloway WJ, et al. Gentamicin and kanamycin in the treatment of gram-negative sepsis: a comparative study. J Infect Dis 1971;124(Suppl):180-184.

Homburger HA. The Laboratory Evaluation of Allergic Diseases: Part I: Measurement Methods for IgE Protein. Lab med 1991;22:780-782.

Hoofnagle JH, Di Bisceglie AM. Serologic Diagnosis of Acute and Chronic Viral Hepatitis. Seminars in Liver Disease 1991;11(2):73-83.

http://en.wikipedia.org

http://humpath.com

http://labtestsonline.org

http://library.med.utah.edu

http://www.accu-chek.com

http://www.arulap.com

http://www.elsevier.com

http://www.euroclonegroup.it

http://www.euroimmun.com

http://www.fpnotebook.com

http://www.kidney.org

http://www.ncbi.nlm.nih.gov

http://www.nkdep.nih.gov

http://www.onelambda.com

http://www.orgentec.com

http://www.pacbio.com

http://www.provendiagnostics.com

http://www.remel.com

http://www.specialtylabs.com

http://www.synlab.com

http://www.viennalab.com

http://www.webmd.com

https://e-labdoc.roche.com

# Bibliography


Huffman DH, Crow JW, Pentikaimen P, Azarnoff DL. Clinical cardiac status, laboratory parameters and digoxin usage. Am Heart J 1976;91:28.

Hughes, G.R.V., Harris, E.N. and Gharavi, A.E. The Anticardiolipin Syndrome. J. Rheumatol. 1986; 13, 3: 486 - 489.

International Expert Committee report on the role of the A1c assay in the diagnosis of diabetes. Diabetes Care 2009;32(7):1327-34.

Jackson GG, et al. Pseudomonas bacteremia: pharmacologic and other bases for failure of treatment with gentamicin. J Infect Dis 1971;124(Suppl):130-185.

Jacobs DS, Kaster BL Jr., Demott WR, Wolfson WL. Laboratory Test Handbook. Stowe, OH: Lexi-Compl. Mosby 1990.

Jaspers JP, Van Oers RJM, Leerkes B. Nine Rheumatoid Factor Assays Compared. J Clin Chem Clin Biochem 1988;26:863-871.

Jeppsson JO, Kobold U, Finke A, et al. Approved IFCC reference method for the measurement of HbA1c in human blood. Clin Chem Lab Med 2002;40:78-89.

Johnson AM. A new international reference preparation for proteins in human serum. Arch Pathol Lab Med 1993;117: 29-31.

Johnson AM. A new international reference preparation for proteins in human serum. Arch Pathol Lab Med 1993;117:29-31.

Johnson VG, Schlom J, Paterson AJ, et al. Analysis of a human tumor-associated glycoprotein (TAG-72) identified by monoclonal antibody B72.3. Cancer Res 1986;46:850-857.

Jones RG, Aguzzi F, Bienvenu J et al. Use of Immunoglobulin Heavy-chain and Light-chain measurement in a multicenter trial to investigate Monoclonal components: I. Detection. Clin Chem 1991;37:1917-1921.

Josephson B, Gyllenswärd C. The Development of the Protein Fractions and of Cholesterol Concentration in the Serum of Normal Infants and Children. Scandinav J Clin Lab Investigation 1957; 9:29.

Kaburaki J, Kuwana M,Yamamoto M, Kawai S, Ikeda Y.Clinical significance of anti-annexin V antibodies in patients with systemic lupus erythematosus. Am J Hematol. 1997 Mar;54(3):209-13.

Kageyama N. A direct colorimetric determination of uric acid in serum and urine with uricase-catalase system. Clin Chim Acta 1971;31:421-426.

Kane J, Middle J, Cawood M. Measurement of serum testosterone in women; what should we do? Ann Clin Biochem 2007;44:5-15.

Kastrup, J. et al.An enzyme linked immunosorbent assay for measurement of human serum thyroglobulin. Evaluation of the influence of thyroglobulin autoantibodies. Scand. J. Lab. Invest. 1985; 45: 471 - 476.

# Bibliography


Kawamura, S. et al. Serum thyroglobulin changes in patients with Graves` disease treated with long term antithyroid drug therapy. J. Clin. Endocrinol.Metab. 1983; 56: 507 - 512.

Kenemans P, Bon GG, Kessler A, et al. Multicenter Technical and Clinical Evaluation of a Fully Automated Enzyme Immunoassay for CA 125. Clin Chem 1992;38/8:1466-1471.

Kjellman NIM, Johansson SGO, Roth A. Serum IgE levels in healthy children quantified by a sandwich technique (PRIST). Clin All 1976;6:51-59.

Klauke R, Schmidt E, Lorentz K. Recommendations for carrying out standard ECCLS procedures (1988) for the catalytic concentrations of creatine kinase, aspartate aminotransferase, alanine aminotransferase and γ-glutamyltransferase at 37 °C. Eur J Clin Chem Clin Biochem 1993; 31:907-909.

Klee GG. Clinical usage recommendations and analytic performance goals for total and free triiodothyronine measurements. Clin Chem 1996;42:155-159.

Kleeberg UR. Pathophysiologie und Diagnostik hämolytischer Anämien. Dtsch med Wschr 1975;100:1400.

Klein G, Berger A, Bertholf R et al. Abstract: Multicenter Evaluation of Liquid Reagents for CK, CK-MB and LDH with Determination of Reference Intervals on Hitachi Systems. Clin Chem 2001;47:Suppl. A30.

Kodama Y, Koike Y, Kimoto H, Yasunaga F, Takeyama M, Teraoka I, Fujii I. Binding parameters of valproic acid to serum protein in healthy adults at steady state Ther Drug Monit 1992;14:55-60.

Kojima S, Yokogava M, Tada T. Raised Levels of Serum IgE in Human Helminthiases. Am J Trop Med Hyg 1972;21(6):913-917.

Kolhouse JF, Kondo H, Allen NC, et al. Cobalamin analogues are present in human plasma and can mask cobalamin deficiency because current radioisotope dilution assays are not specific for true cobalamin. New Eng J Med 1978;299(15):785-792.

Koller A. Total serum protein. In: Kaplan LA, Pesce AJ, eds. Clinical Chemistry, Theory, Analysis, and Correlation. St. Louis: Mosby Company, 1984:1316-1319.

Koopman WJ, Schrohenloker RE. Arthritis Rheum 1980; 23:302-308.

Koumantakis G. Fluorescein Interference with Urinary Creatinine and Protein Measurements. Clin Chem 1991;37/10:1799.

Kraus M, Romisch J, Bastian B, Paques EP, Hartmann AA. Detection of human anti-annexin autoantibodies by enzyme immunoassays. J Immunoassay. 1992;13(3):411-39.

Kreutzer HJH. An immunological turbidimetric method for serum transferring determination. J Clin Chem Clin Biochem 1976;14: 401-406.

# Bibliography


Krieg M, Gunßer KJ. Vergleichende quantitative Analytik klinisch-chemischer Kenngrößen im 24-Stunden-Urin und Morgenurin. J Clin Chem Clin Biochem 1986;24:863-869.

Kuroki M, Haruno M, Arakawa F, et al. Reaction profiles of seven enzyme immunoassay kits for carcinoembryonic antigen (CEA) analyzed with purified preparations of CEA and related normal antigens. Clin Biochem 1992;25:29-35.

Lamson SH, Hook B. Comparison of Mathematical Models for the Maternal Age Dependence of Down's Syndrome Rates. Hum Genet Vol 1981;59:232-234.

Lang DA, Matthews DR, Peto J, et al. Cyclic oscillations of basal plasma glucose and insulin concentrations in human beings. N Engl J Med 1979;301:1023-1027.

Levy J, Klastersky J. Correlation of serum creatinine concentration and amikacin halflife. J Clin Pharmac 1975(Oct.):705-707.

Levy RH, Shen DD. Valproic acid absorption, distribution, and excretion. Antiepileptic Drugs. 4th ed New York: Raven Press, Ltd: 1995;605-619.

Leybold A, Junge W. Importance of colipase for the measurement of serum lipase activity. Adv clin Enzymol 1986;4:60-67.

Lievens M, Bienvenu J, Buitrago JMG et al. Evaluation of four new Tina-quant assays for determination of α-1-acid glycoprotein, α-1-antitrypsin, haptoglobin, prealbumin, kappa and lambda immunoglobulin light chains. Clin Lab 1996;42:515-520.

Lindsay JR, Nieman LK. Differential diagnosis and imaging in Cushing's syndrome. Endocrinol Metab Clin North Am 2005;34:403-421.

Linke R, Küppers R. Nicht-isotopische Immunoassays – Ein Überblick. In: Borsdorf R, Fresenius W, Günzler H et al., eds. Analytiker-Taschenbuch, Vol. 8. Berlin/Heidelberg/New York/Tokyo: Springer-Verlag, 1989: 127-177.

Litam, P. et al. Prognostic value of serum b2-microglobulin in low-grade lymphoma. Ann. Int.Med. 1990, 114: 855-860.

"Little RR, Wiedmeyer HM, England JD, et al. Interlaboratory standardization of measurements of glycohemoglobins. Clin Chem 1992;38:2472-2478.

Liu H, Montoya JL, Forman LJ, Eggers CM, Barham CF, Delgado M. Determination of free valproic acid: evaluation of the Centrifree System and comparison between high-performance liquid chromatography and enzyme immunoassay Ther Drug Monit 1992;14(5):13-521.

Ljunggren, H.G. et al. Role of b2-microglobulin in cancer. Cancer J. 1992, 5: 308-315.

Lobo RA, Paul WL, Goebelsmann U. Serum Levels of DHEAS in Gynecologic Endocrinopathy and Infertility. Obstet Gynecol 1981;57(5):607-612,57(1):69-73.

# Bibliography


Löhr B, El-Samalouti V, Junge W, et al. Reference Range Study for Various Parameters on Roche Clinical Chemistry Analyzers. Clin Lab 2008;55:465-471.

Loussert-Ajaka I, Brun-Vézinet F, Simon F, et al. HIV-1/HIV-2 Seronegativity in HIV-1 subtype O Infected Patients. Lancet 1994;343:1393-1394.

Luft BJ, Remington JS. Toxoplasmic encephalitis in AIDS. Clin Infect Dis 1992;15:211-222.

Maier H, Bossert-Reuther S, Junge W, et al. Calcium reference intervals re-established on Roche/ Hitachi and COBAS INTEGRA® systems. Clin Chem Lab Med 2006;9:A191 [abstract].

Malloy HT, Evelyn KA. The determination of bilirubin with the photoelectric colorimeter. J Biol Chem 1937;119:481-490.

Malloy HT, Evelyn KA. The determination of bilirubin with the photoelectric colorimeter. J Biol Chem 1937;119:481-490.

Malthiery Y, Lissitzky S. Primary structure of human thyroglobulin deduced from sequence of its 8448-base complementary DNA. Eur J Biochem 1987;165:491-498.

Mann K, Hörmann R. hCG (human chorionic gonadotropin). In: Thomas L (ed.). Clinical Laboratory Diagnosis, TH-Books, Frankfurt, 1st English edition 1998:971-976, deutsche Auflage 1998:992-997.

Mann K, Hörmann R. hCG (human chorionic gonadotropin). In: Thomas L (ed.). Clinical Laboratory Diagnosis, TH-Books, Frankfurt, 1st English edition 1998:971-976, deutsche Auflage 1998:992-997.

Mathieu M, Bretaudiere JP, Galteau MM, Giudollet J, Lalegerie P, Bailly M, et al. Recommendations for measuring the catalytic concentration of creatine kinase in human serum at 30 °C. Ann Biol Clin 1982;40:87-164.

Mazzachi BC, Peake MJ, Ehrhardt V. Reference Range and Method Comparison Studies for Enzymatic and Jaffé Creatinine Assays in Plasma and Serum and Early Morning Urine. Clin Lab 2000;46:53-55.

McCormick MH, Stark WM, Pittinger GE, Pittinger RC, McGuire JM. In: Antibiotics Annual. New York, NY: Medical Encyclopedia 1956:606-611.

McDonagh TA, Robb SD, Murdoch DR, et al. Biochemical detection of left-ventricular systolic dysfunction. Lancet 1998;351:9-13.

McIntosh RS, Asghar MS, Weetman AP. The antibody response in human autoimmune thyroid disease. Clin Sci 1997;(92)6:529-541.

McKenzie, J. M., Zakarija, M., Sato, A. Humoral immunity in Graves' disease. Clin. Endocrinol. Metab. 1978; 7: 31 - 45.

Meek B, van Gool T, Gilis H, et al. Dissecting the IgM antibody response during the acute and latent phase of toxoplasmosis. Diagn Microbiol Infect Dis 2001;41:131-137.

# Bibliography


Miedema K. Influence of hemoglobin variants on the determination of glycated hemoglobin. Klin Lab 1993;39:1029-1032

Moore TL, Dorner RN. Rheumatoid factors. Clin Biochem 1993; 26:75-84.

Morgensen CE. Microalbuminuria, renal disease, metabolic syndrome and risks in diabetes. Diabetes and Metabolic Syndrome: Clinical Research & Reviews 2007, 1: 127–133.

Morselli P, Bossi L. In: Woodbury DM, Penry JK, Pippenger CE, eds. Antiepileptic Drugs. New York: Raven Press 1982.

Moss DW, Henderson AR, Kachmar JF. Enzymes. In: Tietz NW, ed. Fundamentals of Clinical Chemistry, 3rd ed. Philadelphia, PA: WB Saunders Company, 1987:346-421.

Mueller T, Gegenhuber A, Poelz W, et al. Head-to-head comparison of the diagnostic utility of BNP and NT-proBNP in symptomatic and asymptomatic structural heart disease. Clin Chim Acta 2004;341:41-48.

Nagy B. Muscle disease. In: Kaplan LA, Pesce AJ, eds. Clinical Chemistry, theory, analysis, and correlation. St. Louis: Mosby 1984:514.

Naito HK. Reliability of Lipid, Lipoprotein and Apolipoprotein Measurements. Clin Chem 1988;34:B84-B94.

Natelson S. Microtechniques of Clinical Chemistry. Springfield, Il: Charles C Thomas 1975:147.

Nathan DM, Kuenen J, Borg R, et al. Translating the A1C assay into estimated average glucose values. Diabetes Care 2008;31:1473-1478.

National Cholesterol Education Program Recommendations for Measurement of High-Density Lipoprotein Cholesterol: Executive Summary. Clin Chem 1995;41:1427-1433.

NCCLS GP-16A2, Urineanalysis and Collection, Transportation and Preservation of Urine specimens, 2nd edition, 2001.

Occupational Safety and Health Standards: bloodborne pathogens. 29 CFR 1910.1030. Fed. Register July 1, 2001;17:260-273.

Owen WE, Roberts WL. Letter to the Editor: Cross-Reactivity of Three Recombinant Insulin Analogs with Five Commercial Insulin Immunoassays. Clin Chem 2004;50(1):257-259.

Pisani T, Gebski CP, Leary ET, et al. Accurate Direct Determination of Low-Density Lipopotein Cholesterol Using an Immunoseparation Reagent and Enzymatic Cholesterol Assay. Arch Pathol Lab Med 1995;119:1127.

Rasmussen K and Moller J. Total homocysteine measurement in clinical practice. Ann Clin Biochem 2000; 37:627-648.

Recommendations for Improving Cholesterol Measurement: A Report from the Laboratory

# Bibliography


Standardization Panel of the National Cholesterol Education Program. NIH Publication No. 90-2964, February 1990.

Refetoff S, Lever EG. The value of serum thyroglobulin measurement in clinical practice. JAMA 1983;250:2352-2357.

Refsum H, Smith AD, Ueland PM, et al. Facts and Recommendations about Total Homocysteine Determinations: An Expert Opinion. Clin Chem 2004; 50(1):3–32.

Reiber H, Thompson EJ, Grimsley G, Bernardi G et al. Quality Assurance for Cerebrospinal Fluid Protein Analysis: International Consensus by an Internet-based Group Discussion. Clin Chem Lab Med 2003;41:331-337.

Reiber H. Proteins in cerebrospinal fluid and blood: barriers, CSF flow rate and source-related dynamics. Res Neurol Neurosci 2003; 21: 79–96.

Reiners, C. Klinische Wertigkeit der Thyreoglobulinbestimmung im Serum. Akt. Endokr. Stoffw. 1984; 5: 76 - 82.

Reiter W, Stieber P, Reuter C, et al. Prognostic Value of Preoperative Serum Levels of CEA, CA 19-9 and 72-4 in Gastric Carcimoma. Anticancer Res 1997;17:2903-2907.

Remaley AT, Wilding P. Macroenzymes: Biochemical Characterization, Clinical Significance, and Laboratory Detection. Clin Chem 1989;35:2267–2270.

Remington JS, Mc Leod R & Desmonts G. Toxoplasmosis. Infectious Disease of the Fetus and Newborn Infant, 5th ed. W.B. Saunders: Philadelphia, 2001;205-346.

Renneberg J. Age related variations in anti-streptococcal antibody levels. Eur J Clin Microbiol Infect Dis 1989;8:792–795.

Report on the Symposium “Drug effects in clinical chemistry methods”, Breuer J, Eur J Clin Chem Clin Biochem 1996;34:385-386.

Reuning RH, Sams RA, Notari RE. Role of pharmacokinetics in drug dosage adjustment. I. Pharmacokinetic effect and apparent volume of distribution of digoxin. J Clin Pharmacol 1973;13:127-141.

Reynolds TM, Penney MD. The mathematical basis of multivariate risk screening: with special reference to screening for Down’s syndrome associated pregnancy. Ann Clin Biochem 1989;26:452-458.

Rice EW, Gorgan BS. Clin Chem 1962;8:181.

Richards AM, Nicholls GM, Yandle TG, et al. Plasma N-Terminal Pro-Brain Natriuretic Peptide and Adrenomedullin: New Neurohormonal Predictors of Left Ventricular Function and Prognosis After Myocardial Infarction. Circulation 1998;97:1921-1929.

# Bibliography


Richterich R, Colombo JP. Klinische Chemie. 4th ed. Basel: Karger S 1978:319-324.

Riesen WF. Lipoprotein (a) Bindeglied zwischen Fettstoffwechsel und Gerinnungssystem? Schweiz med Wschr 1991;121:1813-1818.

Rifai N, Warnick GR, McNamara JR, Belcher JD, Grinstead GF, Frantz Jr ID. Measurement of Low-Density-Lipoprotein Cholesterol in Serum: a Status Report. Clin Chem 1992;38:150-160.

Riff LJ, et al. Pharmacology of gentamicin in man.J Infect Dis 1971;124(Suppl):98-105.

Ringel KP, Dati F, Buchholz E. IgE-Normalwerte bei Kindern. Laboratoriumsblätter 1982;32:26-34.

Ritts RE Jr, Del Villano BC, Go VLV, et al. Initial clinical evaluation of an immunoradiometric assay for CA 19-9 using the NCI serum bank. Int J Cancer 1984;33:339-345.

Robertson DL, Anderson JP, Bradac JA, et al. HIV-1 nomenclature Proposal. Science 2000;288(5463):55-56.

Roberts-Thomson PJ, McEvoy R, et al. Ann Rheum Dis 1985;44:379-383.

Rock RC, Walker WG, Jennings CD. Nitrogen metabolites and renal function. In: Tietz NW, ed. Fundamentals of Clinical Chemistry. 3rd ed. Philadelphia: WB Saunders 1987:669–704.

Roddam AW, Rimmer J, Nickerson C, et al. Prostate-specific antigen: bias and molarity of commercial assays for PSA in use in England. Ann Clin Biochem 2006;43:35-48.

Rodriguez-Garcia MI, Fernandez JA, Rodriguez A, Fernandez MP, Gutierrez C, Torre-Alonso JC.Annexin V autoantibodies in rheumatoid arthritis.Ann Rheum Dis. 1996 Dec;55(12):895-900.

Roeschlau P et al. Z Klin Chem Klin Biochem 1974;12:226.

Rohlfing C, Connolly S, England J, et al. Effect of elevated fetal hemoglobin on HbA1c measurements: four common assay methods compared to the IFCC reference method. Clin Chem 2006;52 Suppl 6:A108.

Rosner W, Auchus RJ, Azzis R, et al. Position Statement: Utility, Limitations, and Pitfalls in Measuring Testosterone: An Endocrine Society Positions Statement. J Clin Endocrinol Metab 2007;92(2):404-413.

Rothenberg SP, DaCosta M, Rosenberg BS. A radioassay for serum folate: Use of a two-phase sequential incubation, ligand-binding system. New Eng J Med 1972;285(25):1335-1339.

Rothschild MA, Oratz M, Schreiber SS. Serum albumin. Hepatology 1988;8:385-401.

Rotschafer JC, Crossley K, Zaske DE, Mead K, Sawchuk RJ, Solem LD. Pharmacokinetics of vancomycin: observations in 28 patients and dosage recommendations. Antimicrob Agents Chemother 1982;22:391-394.

Ruf, J., Czarnocka, B., Ferrand, M. et al. Relationship between immunological structure and

# Bibliography

biochemical properties of human thyroid peroxidase. Endocrinology 1988; Vol. 125 (3): 1211 - 1218.

Ruibal A, Encabo G, Martinez-Miralles E, et al. CA 125 seric levels in nonmalignant pathologies. Bull Cancer (Paris) 1984;71(2):145-146.

Ruilope, L.M. et al. Randomly allocated study of the effects of standard therapy versus ACE inhibition on micro-albuminuria in essential hypertension. J. Hypert. Suppl. 1994;12: S59 - S63.

Rumack BH. Acetaminophen overdose. Arch Intern Med 1981;141:380.

Runnebaum B, Rabe T. Gynäkologische Endokrinologie und Fortpflanzungsmedizin Springer Verlag 1994; Band 1:36-38,70,116 Band 2:137,360,398-399,408-409,422-423. ISBN 3-540-57345-3, ISBN 3-540-57347-x.

Ruoslathi E, Engvall E, Kessler MJ. Chemical Properties of Alpha-Fetoprotein. In: Herberman RB, McIntire KR (eds). Immunodiagnosis of Cancer. New York: Marcel Dekker Inc 1979:101-117.

Rush D. Folate Supplements Prevent Recurrence of Neural Tube Defects, FDA Dietary Supplement Task Force. Nutrition Reviews 1992;50(1):22-28.

Rüter A, Smeds S, Lennquist S. Value of Serum Thyroglobulin Measurement in Patients Operated on for Well Differentiated Thyroid Carcinoma. Eur J Surg 1998;164:665-671.

Ryan MF. The role of magnesium in clinical biochemistry: an overview. Ann Clin Biochem 1991;28:19-26.

Saboori AM, Rose NR, Butscher WG, et al. Modification of a Nonincinerative Method for Determination of Iodine in Iodoproteins. Anal Biochem 1993;214:335-338.

Sacks BW, Bruns DE, Goldstein DE, et al. Guidelines and recommendations for laboratory analysis in the diagnosis and management of diabetes mellitus. Clin Chem 2002;48:436-472.

Sacks DB. Carbohydrates. In: Burtis CA, Ashwood ER, eds. Tietz Textbook of Clinical Chemistry. 3rd ed. Philadelphia: WB Saunders 1999:750-785.

Safi F, Roscher R, Beger HG. The clinical relevance of the tumor marker CA 19-9 in the diagnosing and monitoring of pancreatic carcinoma. Bull Cancer Paris 1990;77(1):83-91.

Sakai H, Fukuda G, Suzuki N, et al. Falsely Elevated Thyroid-Stimulating Hormone (TSH) Level Due to Macro-TSH. Endocr J 2009;56(3):435-440.

Salt WB II, Schenker S. Amylase-its clinical significance: a review of the literature [Review]. Medicine 1976; 55:269-281.

Sampson EJ, Baired MA, Burtis CA, Smith EM, Witte DL, Bayse DD. A coupled-enzyme equilibrium method for measuring urea in serum: Optimization and evaluation of the AACC study group on urea candidate reference method. Clin Chem 1980;26:816-826.

San Diego Declaration, Erythropoietin use and response in endstage renal disease. The American

# Bibliography


Society of Nephrology, Annual meeting, San Diego. J Am Soc Nephrol 1995; 3: 35.

Santiago JV. Lessons from the Diabetes Control and Complications Trial. Diabetes 1993;42:1549-1554.

Sapin R, Le Galudec V, Gasser F, et al. Elecsys Insulin Assay: Free Insulin Determination and the Absence of Cross-Reactivity with Insulin Lispro. Clin Chem 2001;47:602-605.

Sapin R. Review: Insulin Assays: Previously Known and New Analytical Features. Clin Lab 2003;49(3-4):113-121.

Sato Y, Nakata K, Kato Y, et al. Early recognition of hepatocellular carcinoma based on altered profiles of alpha-fetoprotein. N Engl J Med 1993;328(25):1802-1806.

Satoh A, Suzuki K, Takayama E, Kojima K, Hidaka T, Kawakami M, Matsumoto I, Ohsuzu F. Detection of anti-annexin IV and V antibodies in patients with antiphospholipid syndrome and systemic lupus erythematosus. J Rheumatol. 19

Schade, S.G., P.L. Feick, M.H. Imrie, and R.F. Schilling. In vitro studies on antibodies to intrinsic factor. Clin. Exp. Immunol. 1967, 2:399-413.

Schaefer EJ, Lamon-Fava S, Jenner JL et al. Lipoprotein(a) levels and risk of coronary heart disease in men - The Lipid Research Clinics Coronary Primary Prevention Trial. JAMA 1994;271:999-1003.

Schatz, H., Löbig, H. Diagnostische und prognostische Bedeutung von Antikörpern gegen Schilddrüsenmikrosomen und Thyreoglobulin. Akt. Endokr. Stoffw. 1989; 10: 146 - 153.

Scher HI, Kelly WK. Flutamide withdrawal syndrome: its impact on clinical trials in hormone-refractory prostate cancer. J Clin Oncol 1993;11:1566-1572.

Scherbaum,W. A., Berg, P. A.Autoimmune Schilddrüsenerkrankungen - Neuere Aspekte zur Pathogenese und Diagnostik. Dtsch.Med.Wschr. 1984; 109: 1574 - 1581.

Schlebusch H, Liappis N, Kalina E, et al. High sensitive CRP and creatinine: reference intervals from infancy to childhood. J Lab Med 2002;26:341-346.

Schmidt-Mathiesen H. Gynäkologie und Geburtshilfe. Schattauer Verlag 1992.

Schmitt, W. H. and Gross, W. L.. Antineutrophile zytoplasmatische Antikörper (ANCA). Internist 1995,No. 36, pp. 282 - 290.

Schneider M, Pott G, Gerlach U. Alpha-1-Antitrypsin Mangel. Klin Wschr 1986;64:197-205.

Schneider W, Marcovitz S, Al-Shammari S, et al. Reactivity of macroprolactin in common automated immunoassays. Clin Biochem 2001;34:469-473.

Schrader J, Köstering H, Scheler F. Klinische Relevanz der Bestimmung von Antithrombin III. Laboratoriumsblätter 1983;33:87-98.

# Bibliography


Schumann G et al. IFCC Primary Reference Procedures for the Measurement of Catalytic Activity Concentrations of Enzymes at 37 °C - Part 2. Reference Procedure for the Measurement of Catalytic Concentration of Creatine Kinase. Clin Chem Lab Med 2002; 40(6):635-642.

Schumann G, Dati F. Vorläufige Referenzbereiche für 14 Proteine im Serum (für Erwachsene) nach Standardisierung immunchemischer Methoden unter Bezug auf das internationale Referenzmaterial CRM 470. Lab Med 1995;19:401-403.

Schwartz GJ, Muñoz A, Schneider MF, et al. New Equations to Estimate GFR in Children with CKD. J Am Soc Nephrol 2009;20:629–637.

Schwarz S, Berger P, Wick G. The Antigenic Surface of Human Chorionic Gonadotropin as Mapped by Murine Monoclonal Antibodies. Endocrinology 1986;118(1):189-197.

Schwarzenbach G. The complexones and their analytical application. Analyst 1955;80:713-729.

Scott MG, Heusel JW, LeGrys VA, et al. Electrolytes and blood gases, in Tietz NW. Textbook of Clinical Chemistry. 3rd ed. Philadelphia, PA: WB Saunders Co 1999:1065-1066.

Seed M, Hoppichler F, Reaveley D et al. Relation of serum lipoprotein (a) concentration and apolipoprotein (a) phenotype to coronary heart disease in patients with familiar hypercholesterolemia. New Engl J Med 1990;322:1494-1499.

Seehofer D, Rayes N, Klupp J, et al. Predictive value of intact parathyroid hormone measurement during surgery for renal hyperparathyroidism. Langenbecks Arch Surg 2005;390(3):222-229.

Seetharam, B., S. Bose, and N. Li. Cellular Import of Cobalamin (Vitamin B-12). J.Nutr., 1999, 129:1761-1764.

Segal MA. A rapid electrotitrimetric method for determining CO2 combining power in plasma or serum. Am J Clin Pathol 1955;25:1212–1216.

Seino Y, Ogawa A, Yamashita T, et al. Application of NT-proBNP and BNP measurements in cardiac care: a more discerning marker for the detection and evaluation of heart failure. Eur J Heart Fail 2004;6(3):295-300.

Sell SS. Serological Cancer Markers. Humana Press 1992; ISBN 0-89603-209-4.

Semjonow A, Brandt B, Oberpenning F, et al. Discrepancies in assays impair the interpretation of prostate-specific antigen. Urology 1995;34:303-315.

Shaw LM. Keeping pace with a popular enzyme GGT. Diagnostic Medicine 1982; May/June:1-8.

Shen G.Q., Shoenfeld Y., Peter J.B..Anti-DNA, anti-histone and anti-nucleosome antibodies in systemic Lupus erythematosus and drug-induced Lupus. Clin. Reviews in Allergy and Immunology;Vol.16; 321-334; 1998.

Sherer Y. et al Anti-Prothrombin antibodies in thrombosis and atherosclerosis. Atherosclerosis and

# Bibliography


Autoimmunity 2001; 185-190

Sherwin AD, Robb JP, Lechter M. Improved control of epilepsy by monitoring plasma ethosuximide. Arch Neurol 1973;28:178-181.

Siedel J et al. AACC Meeting Abstract 34. Clin Chem 1993;39: 1127.

Siekmann L, Breuer H. Determination of Cortisol in Human Plasma by Isotope Dilution Mass Spectrometry. J Clin Chem Clin Biochem 1982;20:883-892.

Siekmeier R, März W, Scharnagl H et al. Bestimmung von Lipoprotein (a): Vergleich eines neuen latexverstärkten immunoturbidimetrischen Assay mit einem immunoradiometrischen Assay. J Lab Med 1996;20:294-298.

Sila A, Roselli M, Cosimelli M, et al. Clinical Utility of CA 72-4 Serum Marker in the Staging and Immediate Post-surgical Management of Gastric Cancer Patients. Anticancer Res 1996;16:2241-2248.

Silverman EK, Pierce JA, Province MA et al. Variability of pulmonary function in alpha-1-antitrypsin deficiency: Clinical correlation. Ann Intern Med 1989;111:982-991.

Silverman LM, Christenson RH. Amino acids and proteins. In: Burtis CA, Ashwood ER, eds. Tietz Fundamentals of Clinical Chemistry. 4th ed. Philadelphia: WB Saunders 1996:240-282.

Simon F, Mauclère P, Roques P, et al. Identification of a new human immunodeficiency virus type 1 distinct from group M and group O. Nat Med 1998;4(9):1032-1037.

Sing HP, et al. Clin Chem 1972;18:137.

Singer JM, Plotz CM. The latex fixation test. Am J Med 1956;21:888–892.

Sinico, R.A.,B.Bollini,E.Sabadini, L.Di Toma,and A.Radice.The use of laboratory tests in diagnosis and monitoring of systemic lupus erythematosus. J.Nephrol., 2002, 6:S20-27.

Skvaril F, Barandum S, Morell A, Fuffer F, Probst M. Imbalances of kappa/lambda immunoglobulin light chain ratios in normal individuals and in immunodeficient patients. In: Proteides of biological fluids, Peeters H, ed. 1975;23:415-420.

Slordal L, Spigset O. Heart failure induced by non-cardiac drugs. Drug safety 2006;29(7):567-586.

Smith CR, Norman MR. Prolactin and growth hormone: molecular heterogeneity and measurement in serum. Ann Clin Biochem 1990;27:542-550.

Smith TW, Haber E. Digoxin intoxication: the relationship of clinical presentation to serum digoxin concentration. J Clin Invest 1970;49:2377-2386.

Soldin SJ, Brugnara C, Wong EC, eds. Pediatric Reference Intervals, 5th edition. AACC Press 2005.

Sollid, L.M.Coeliac diasease:Dissecting a complex inflammatory disorder.Nature Rev., 2002,2:647-655.

# Bibliography


Sonntag O, Scholer A. Drug interferences in clinical chemistry: recommendation of drugs and their concentrations to be used in drug interference studies. Ann Clin Biochem 2001;38:376-385.

Sonntag O, Scholer A. Drug interferences in clinical chemistry: recommendation of drugs and their concentrations to be used in drug interference studies. Ann Clin Biochem 2001;38:376-385.

Sorice M, Pittoni V, Circella A, et al. Anti-prothrombin but not “pure” anti-cardiolipin antibodies are associated with the clinical features of the antiphospholipid antibody syndrome [letter]. Thromb Haemost, Oct 1998, 80(4)

Sorrell TC, Packham S, Shanker M, Foldes M, Munro R. Vancomycin therapy for methicillin-resistant Staphylococcus aureus. Ann Intern Med 1982;97:344-350.

Spencer C. International Thyroid Testing Guidelines. National Academy of Clinical Biochemistry, August 2001;Section 3E,11-14.

Stamey TA, Chen Z, Prestigiacomo AF. Reference Material for PSA: The IFCC Standardization Study. Clin Biochem 1998;31:475-481.

Stamey TA. Second Stanford conference on international standardization of prostate-specific antigen immunoassays: September 1 and 2, 1994. Urology 1995;45:173-184.

Standard Reference Materials from NERL, traceable to NIST (National Institute of Standards and Technology).

Staples A, LeBlond R, Watkins S, et al. Validation of the revised Schwartz estimating equation in a predominantly non-CKD population. Pediatr Nephrol 2010 Jul 22;25:2321–2326.

Stein EA, Myers GL. National Cholesterol Education Program Recommendations for Triglycerides Measurement: Executive Summary. Clin Chem 1995;41:1421-1426.

Stein W. Strategie der klinisch-chemischen Diagnostik des frischen Myokardinfarktes. Med Welt 1985;36:572-577.

Steinberg W. The clinical utility of the CA 19-9 tumor associated antigen. Am J Gastroenterol 1990;85(4):350-355.

Steinhoff J, Feddersen A, Wood WG, et al. β2-Microglobulinurie bei Cytomegalievirusinfektionen nach Nierentransplantation. Dtsch med Wschr 1991;116:1008-1012.

Steinmetz A (Universitätsklinik Marburg), personal communication.

Sternlieb I. Copper and the liver. Gastroenterology 1980;78:1615-1628.

Stieber P, Fateh-Moghadam A. Sensible Use of Tumormarkers. Dtsch: Tumormarker und ihr sinnvoller Einsatz. ISBN 3-926725-07-9 dtsch/engl. Juergen Hartmann Verlag Marloffstein-Rathsberg (1993).

Stieber P, Fateh-Moghadam A. Sensible Use of Tumormarkers. Dtsch: Tumormarker und ihr sinnvoller Einsatz. ISBN 3-926725-07-9 dtsch/engl. Juergen Hartmann Verlag Marloffstein-

# Bibliography


Rathsberg (1993).

Stiko Recommendations, Bundesgesundheitsbl. 1996;1/96:32-42.

Stollerman GH. Streptococcal antibodies in the diagnosis of rheumatic fever. In: Cohen AS, ed. Laboratory Diagnostic Procedures in the Rheumatic Diseases. Boston: Little, Brown 1967:168–215.

Stookey LL. FerroZine - a new spectrophotometric reagent for iron. Anal Chem 1970;42:779-781.

Strong JP,Naito HK, Scott MG, Roheim PS, Asztalos BF, Zilversmit DB, Srinivasan SR, Berenson GS, Wilson PWF, Scanu AM, Malikow MR. Atherogenesis: current topics on etiology and risk factors. Clin Chem 1995;41:132-133 No. 1.

Stuart KE, Anand AJ, Jenkins RL. Hepatocellular Carcinoma in the United States. Cancer 1996;77,11:2217-2222.

Study Group, European Atherosclerosis Society. Strategies for the prevention of coronary heart disease: A policy statement of the European Atherosclerosis Society. European Heart Journal 1987;8:77.

Sturgeon C. Practice Guidelines for Tumor Marker Use in the Clinic. Clin Chem 2002;48(8):1151-1159.

Subcommittee on Hyperbilirubinemia. Management of Hyperbilirubinemia in the Newborn Infant 35 or More Weeks of Gestation. Pediatrics 2004;114:297–316.

Suliman AM, Smith TP, Gibney J, et al. Frequent misdiagnosis and mismanagement of hyperprolactinemic patients before the introduction of macroprolactin screening: application of a new strict laboratory definition of macroprolactinemia. Clin Chem 2003;49(9):1504-1509.

Swan, F. et al. Beta 2 microglobulin cell surface expression as an indicator of resistance in lymphoma and its relation to the serum level. Blood 1988, 72: 258a.

Szasz G. Methods of Enzymatic Analysis. 2nd English ed. New York: Academic Press, Inc 1974:717.

Taketa K. Alpha-Fetoprotein in the 1990s. In: Sell SS. Serological cancer markers. Humana Press 1992;31-46, ISBN: 0-89603-209-4.

Talwar S, Squire IB, Davies JE, et al. Plasma N-terminal pro-brain natriuretic peptide and the ECG in the assessment of left-ventricular systolic dysfunction in a high risk population. Eur Heart J 1999;20:1736-1744.

Taussky HH, Schoor EA. A microcolorimetric method for the determination of inorganic phosphorus. J Biol Chem 1953;202:675.

The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. The Diabetes Control and Complications Trial Research Group.N Engl J Med 1993;329:977-986.

# Bibliography


The European Society of cardiology, Struthers AD. How to use natriuretic peptide levels for diagnosis and prognosis. Eur Heart J 1999;20:1374-1375.

Thefeld W, Hoffmeister H, Busch EW, et al. Referenzwerte für die Bestimmungen der Transaminasen GOT und GPT sowie der alkalischen Phosphatase im Serum mit optimierten Standardmethoden. Dtsch Med Wschr 1974;99:343-351.

Thiel RP, Oesterling JE, Wojno KJ, et al. A multicenter comparison of the diagnostic performance of free prostate-specific antigen. Urology 1996;48(6A):45-50.

Thienpont LM, De Brabandere VI, Stöckl D, et al. Candidate Reference Method for Determining Serum Cortisol,Use of cyclodextrins for prepurification of progesterone and testosterone from human serum prior to determination Based on Isotope Dilution-Gas Chromatography/Mass Spectrometry Using Heptafluoroburyrilation as Derivatization Method. Anal Biochem 1996;234:204-209.

Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). NIH Publication No. 01-3670; May 2001.

Thomas CMG, Reijnders FJL, Segers MFG, et al. Human Choriogonadotropin (HCG): Comparisons between Determinations of Intact HCG, Free HCG β-Subunit, and "Total" HCG + β in Serum during the First Half of High-Risk Pregnancy. Clinical Chemistry 1990;36(4):651-655.

Thomas L et al. Consensus of DGKL and VDGH for interim reference intervals on enzymes in serum. J Lab Med 2005; 29(5):301-308.

Thompson JA. Molecular cloning and expression of carcinoembryonic antigen gene family members. Tumor Biol 1995;16:10-16.

Thulliez P. Maternal and foetal infection: in Toxoplasmosis (eds D.H.M. Joynson, T.G. Wreghitt) Cambridge University Press, 2001:193-213 ISBN 0521 44328 8.

Tietz NW et al. Lipase in serum - the elusive enzyme: An overview. Clin Chem 1993;39:746-756.

Tiffany TO, Jansen JM, Burtis CA, Overton JB, Scott CD. Enzymatic kinetic rate and end-point analyses of substrate, by use of a GeMSAEC Fast Analyzer. Clin Chem 1972;18:829-840.

Town MH, Gehm S, Hammer B, Ziegenhorn J. J Clin Chem Clin Biochem 1985;23:591.

Trinder P. Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor. Ann Clin Biochem 1969;6:24.

Triplett DA. Lupus anticoagulants/antiphospholipid-protein antibodies: the great imposters. Lupus. 1996 Oct;5(5):431-5.

Troughton RW, Frampton CM, Yandle TG, et al. Plasma Amino-Terminal B-Type Natriuretic Peptide Measured by Elecsys 2010 Assay in a Trial of Hormone-guided Treatment for Heart Failure. Clin Chem 2003;49(7):1212-1215.

# Bibliography


Troupin A, et al. Carbamazepine, a double-blind comparison with phenytoin. Neurol 1977;27:511-519.

Truitt EG Jr, McKusick VA, Krantz C. Theophylline blood levels after oral, rectal, and intravenous administration and correlation with diuretic action. J Pharmcol Exp Ther 1958;10:310-315.

Tsang R, Marder H. Bone disease. In: Kaplan LA, Pesce AJ, eds. Clinical Chemistry, theory, analysis, and correlation. St. Louis: Mosby Company 1984:439-459.

Turner-Warwick M. Study of theophylline plasma levels after oral administration of theophylline compounds. Br Med J 1957:25.

Uller, R.P. and van Herle, A.J. Effect of therapy on serum thyroglobulin levels in patients with Graves` disease. J. Clin. Endocrinol.Metab. 1978; 46: 747 - 755.

Utiger RD. The pathogenesis of autoimmune thyroid disease. N Eng J Med 1991;325:278-279.

Vaarala O, Puurunen M, Manttari M, et al. Antibodies to prothrombin imply a risk of myocardial infarction in middle-aged men. Thromb Haemost, Mar 1996, 75(3) p456-9

Valdes R Jr, Jortani S, Gheorghiade M. Standards of laboratory practice: cardiac drug monitoring. Clin Chem 1998; 44:5:1096-1109.

Van Dalen A. Quality Control and Standardization of Tumor Marker Tests. Letter to the Editor: Tumor Biol 1993;14:131-135.

van der Heiden C, Bais R, Gerhardt W, Lorentz K, Rosalki S. Approved recommendation on IFCC methods for the measurement of catalytic concentration of enzymes. Part 8. IFCC method for lactate dehydrogenase. Eur J Clin Chem Clin Biochem1994; 32:639-655.

Van Duijnhoven HLP, Perqueriauz NCV, van Zon JPHM, et al. Large discrepancy between prostate specific antigen results from different assays during longitudinal follow-up of a prostate cancer patient. Clin Chem 1996;42:637-641.

Van Lente F, Marchand A, Galen RS. Evaluation of a nephelometric assay for haptoglobin and its clinical usefulness. Clin Chem 1979;25:2007.

VanAnken HC, Schiphorst ME. A kinetic determination of ammonia in plasma. Clin Chim Acta 1974;56:151-157.

Verhseghe PG,Thienpont LM, Van Brussel KA, De Leenheer AP. Efforts by industry toward standardization of serum estradiol-17β measurements. Clin Chem 1998;44(3):671-674.

Volpé R. Rational Use of Thyroid Function Tests. Crit Rev Clin Lab Sci 1997;34(5):405-438.

W. Heil, R. Koberstien, B. Zwata. References Ranges for Adults and children- Roche 2004;156-186.

Waaler E. On the occurrence of a factor in human serum activating the specific agglutination of sheep blood corpuscles. Acta Pathol Microbiol Scand 1940;17:172-178.

# Bibliography


Wada N, Chiba H, Shimizu C, et al. A Novel Missense Mutation in Codon 218 of the Albumin Gene in a Distinct Phenotype of Familial Dysalbuminemic Hyperthyroxinemia in a Japanese Kindred. Journal of Clinical Endocrinology and Metabolism 1997;82(10):3246-3250.

Wald NJ, Kennard A, Densem JW, et al. Antenatal maternal serum screening for Down's syndrome: results of a demonstration project. BMJ 1992;305:391-394.

Waldman TA, Polmar SH, Ballestra ST, et al. Immunoglobulin E in immunologic deficiency diseases. II. Serum IgE Concentration of Patients with Acquired Hypogammaglobulinemia, Thymoma and Hypogammaglobulinemia, Myotonic Dystrophy, Intestinal Lymphangiectasia and Wiskott-Aldrich Syndrome. J Immunol 1972;109(2):304-310.

Walker, J.D. et al.Glomerular structure in type-1 (insulin-dependent) diabetic patients with normo microalbuminuria. Kidn. Int. 1992; 41: 741 - 748.

Wang P, Izumi T, Fukuyama Y. Clinical significance of measurement of percent free valproic acid in epileptic children J Tokyo Wom Med Coll 1993;63:E216-E219.

Weber B, Fall EH, Berger A, et al. Reduction of Diagnostic Window by New Fourth-generation Human immunodeficiency Virus Screening Assays. J Clin Microbiol 1998;36(8):2235-2239.

Weichselbaum TE. Amer J Clin Path 1946;16:40.

Wians FH. The "Correct" PSA Concentration. Clin Chem 1996;42:1882-1885.

Wibell, L. et al. Serum b2-microglobulin in renal disease.Nephron 1973, 10: 320-331.

Wick M, Pinggera W, Lehmann P. Iron Metabolism, Anemias. Clinical Aspects and Laboratory, 5th ed. Vienna/New York: Springer-Verlag, 2003.

Wick M, Pinggera W, Lehmann P. Iron Metabolism. Diagnosis and Therapy of Anemias, 3rd ed. Vienna/New York: Springer-Verlag 1996.

Wiebe DA, Bernert JT. Clin Chem 1984;30:352.

Wieland H, Seidel D. Quantitative Lipoprotein Electrophoresis. In: Handbook of Electrophoresis, Vol III, ed. Lewis A., Boca Raton: CRC Press, 83-102, 1983.

Wiik, A.et al. The diversity of perinuclear antineutrophil cytoplasmic antibodies (pANCA) antigens. Proceedings of the 6th international ANCA workshop, France - July 1995, pp. 15 - 17.

Wild D. The Immunoassay Handbook. Stockton Press, 1994:338.

Wilder BJ, et al. Seizure Disorders. New York, NY: Raven Press 1981.

Wilding P, Zilva JF, Wilde CE. Transport of specimens for clinical chemistry analysis. Ann Clin Biochem 1977;14:301-306.

Williams WJ, Beutler E, Ersler AJ, Lichtman MA, eds. Hematology, 4th ed. New York: McGraw-Hill, 1990.

# Bibliography


Wu AHB, ed. Tietz Clinical Guide to Laboratory Tests, 4th ed. Philadelphia, PA: WB Saunders Company 2006:706-1100.

Wu AHB, ed. Tietz Clinical Guide to Laboratory Tests, 4th ed. St. Louis (MO): Saunders Elsevier; 2006:202,:306–307,1010pp.

Würzburg U, Hennrich N, Lang H. Bestimmung der Aktivität von Creatinkinase MB im Serum unter Verwendung inhibierender Antikörper. Klin Wschr 1976;54:357-360.

Wüst T, Beeser H, Lang HR. Diagnostik und Therapie der Verbrauchskoagulopathie. Intensivmed 1990;27:177-82.

Wuyts B, Bernard D, van den Noortgate N, et al. Reevaluation of Formulas for Predicting Creatinine Clearance in Adults and Children Using Compensated Creatinine Methods. Clinical Chemistry 2003;49:1011-4.

Yaneva M, Kirilov G, Zacharieva S. Midnight salivary cortisol, measured by highly sensitive electrochemiluminescence immunoassay, for the diagnosis of Cushing's syndrome. Cent Eur J Med 2009;4(1):59-64.

Yang GP, Levine S, Weigel RJ. A spike in parathyroid hormone during neck exploration may cause a false-negative intra-operative assay result. Arch Surg 2001;136:945-949.

Yeo KT, Wu AH, Apple FS, et al. Multicenter evaluation of the Roche NT-proBNP assay and comparison to the Biosite Triage BNP assay. Clin Chim Acta 2003,338:107-115.

Yilmaz FM, Yücel D. Effect of Addition of Hemolysate on Urine and Cerebrospinal Fluid Assays for Protein. Clin Chem 2006;52:152-153.

Yodfat,O., M. Blank, I. Krause and Y. Shoenfeld.The pathogenic role of antiphosphatidylserine antibodies: active immunization with antibodies leads to the induction of antiphospholipid syndrome. Clin. Immunol. Immunopathol.,V

Young DS. Effects of Preanalytical Variables on Clinical Laboratory Tests, 2nd ed. Washington, DC: AACC Press; 1997.

Zimmerman HJ, Henry JB. In: Henry JB, ed. Clinical Diagnosis and Management by Laboratory Methods. 17th ed. Philadelphia: WB Saunders, 1984:251-282.

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