

## D-Dimers

**Synonyms:** Fibrin Breakdown Product-D-Dimer

**Background:** Plasmin degrades fibrin clots to D-dimers and other fibrin degradation products (FDP). D-dimers are formed by plasmin degradation of fibrin, it is not formed from intact fibrinogen, indicating preceding fibrin forming. D-dimers and FDP are positive in disseminated intravascular coagulation (DIC), thrombosis, liver diseases (decreased hepatic clearance). Increased values occur during pregnancy, post operatively, during bleeding, hemodialysis, eclampsia, sickle cell crisis, and in cancer patients.

Clinically useful in the diagnosis of DIC, of deep venous thrombosis and pulmonary embolism. Used in monitoring thrombolytic therapy, which increases D-dimers.

**Sampling:** 3 mL of citrate plasma. Stable at room temperature for 8h, on ice 1 day.

**Reference Interval:** <0.5 µg/mL

## Dehydroepiandrosterone Sulphate (DHEA-S), Serum

**Related Information:** Adenocorticotrophic Hormone (ACTH), Plasma  
Androstenedione, Serum  
Cortisol, Free, Urine  
Cortisol, Serum or Plasma  
Estradiol, Serum  
17-alpha-Hydroxyprogesterone (17-OHP)  
Testosterone, Serum

**Background:** DHEA-S and dehydroepiandrosterone are synthesized by the adrenal cortex controlled by adrenocorticotropin (ACTH). In men, in addition to the adrenal cortex, approx. 5% of DHEA-S and 10%-25% of DHEA are produced by the testes. DHAE and DHAE-S are weak androgens but are converted by peripheral tissue into androstenedione and testosterone and into estrogens. Half life time of DAEH-S is 10-20h of DHEA is 1-3h, resulting in an up to 500 times higher serum concentration of DAEH-S. DHEA in opposite to DHEA-S, has a diurnal variation similar to cortisol.

In men DHEA-S levels are linked to greater fitness, higher testosterone levels. DHEA was added in 1996 to the list of prohibited substances by the International Olympic Commission.

Used in the assessment of

-adrenal hyperfunction: DHEA is elevated in congenital adrenal hyperplasias (11 beta hydroxylase and 21 beta hydroxylase forms) as well as in adrenal neoplasms.

-adrenal insufficiency: resulting in low basal DHEA-S levels.

**Sampling:** 1 mL serum

**Reference Interval:** (µg/dL)

Age	Male	Female
Children		
1-5 days	12-254	10-248
1 month to 5 years	1-41	5-55
6-9 years	2-145	2-140
10-11 years	15-115	15-260
12-17 years	20-555	20-535
Adults		
18-30 years	125-619	45-380
31-50 years	59-452	12-379
51-60 years	20-413	post menopausal 30-260
61-83 years	12-285	

**Dengue Fever, Serology**

**Background:** The Dengue virus is an arthropod borne virus (Arbovirus). Transmitted by the *Aedes aegypti* mosquito (also a vector for yellow fever) dengue virus infects 20 million people/year worldwide in tropical areas, especially in the Caribbean. Humans and monkeys are reservoirs. Two clinical courses are known: Classic dengue (breakbone fever) with a sudden influenzalike onset and severe muscle and joint pain. Leukopenia and maculopapular rash is common. Spontaneously resolving after 1-2 weeks, rarely fatal.

Dengue hemorrhagic fever initially resembles classic dengue fever but shock and hemorrhage in the gastrointestinal tract and skin develop. Fatality rate 10%. The severe form occurs particularly in southern Asia. There is no antiviral drug or vaccine available.

**Sampling:** 2 mL serum, each at the beginning and convalescent sample

**Reference Interval:** Antibody titer IgM, IgG negative: < 1:10

**11-Deoxycorticosteron (DOC), Plasma**

**Related Information:** Cortisol, Serum or Plasma  
11-Deoxycortisol, Plasma  
21-Deoxycortisol, Plasma

**Background:** DOC and 11-hydroxycortisol are precursors of aldosterone. Both have also mineralocorticoid activity, and may be the cause of hypertension.

The most prevalent (95%) congenital enzyme defect of the adrenal cortex is due to 21 hydroxylase deficiency, less prevalent are 11-hydroxylase, 17-hydroxylase, 3-beta hydroxysteroid dehydrogenase; 20-22-desmolase and the 18-hydroxylase and 18-hydroxysteroid dehydrogenase defects.

DOC is increased in serum (besides other metabolites of corticosterone) in 18-hydroxylase defects and present clinically as adrenocortical insufficiency (salt loss) without virilization, without hypertension and normal sexual development.

**Sampling:** 2 mL heparinized plasma

<b>Reference Interval:</b>	Premature:	<105 ng/dL
	Newborn: (first week)	<105 ng/dL
	Children:	
	1 month to 1 year	7 – 49 ng/dL
	2 to 10 years	2 – 34 ng/dL
Adult:	2 – 15 ng/dL	
	after ACTH stimulation:	< 90 ng/dL

## Diazepam, Serum

**Related Information:** Ethanol, Blood, Serum or Urine

**Synonyms:** Aliseum®; Alupram®; Atensine®; Diastat®; Diazemuls®; Di-Tran®; Lamra®; Solis®; Stesolid®; Tensium®; T-Quil®; Valium®; Valrelease®; Vatran®; Vazepam®; Zetran®.

**Background:** As a tranquilizer, diazepam is indicated in anxiety, panic attacks, muscle spasm, control of seizures in acute situations, and treatment of ethanol withdraw syndrome. It is metabolized to the active compound nordiazepam (N-desmethyldiazepam) with a half life of 2-4 days. Bioavailability 100%; urinary excretion 1%; plasma binding 98% lower in renal disease, cirrhosis, nephritic syndrome, pregnancy, neonates, burn patients elderly; volume of distribution 1 L/kg increased in cirrhosis, hypoalbuminemia, elderly; half life 30-56h increased in elderly, cirrhosis; bioactive CNS half life 1h; peak time 1-2h; peak concentration I.V.: 400-500 ng/mL after 5-10 mg IV dose, oral: 300-350 ng/mL after 10 mg oral dose.

**Sampling:** 2 mL serum, do not freeze. Peak 1h after oral dose, 15 min after IV.

**Reference Interval:**

Diazepam	Therapeutic values	200 - 500 ng/mL
		300 - 400 ng/mL provides anxiolytic effect
		> 600 ng/mL provides control of seizures
	Toxic values	> 1000 ng/mL

N-desmethyldiazepam	Therapeutic values	600-1500 ng/mL
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## Digitoxin, Serum

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**Related Information:** Digoxin, Serum

**Background:** Digitoxin differs from digoxin by the absence of a hydroxyl group at C12 resulting in a less hydrophilic compound. The compound is not available in the US.

Bioavailability 90%; urinary excretion 32%; plasma binding 90%-97%; volume of distribution 0.7L/kg; half life time 150-250h.

Limitations: Patients on digoxin cannot be monitored for digitoxin!

**Sampling:** 2 mL serum taken 6-12h after dosing

**Reference Interval:**

Therapeutic values: 15.0 – 30.0 ng/mL

Toxic values: Levels of > 35 ng/mL are in 80% of the patients associated with clinical toxicity

## Digoxin, Serum

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**Related Information:** Amiodarone, Serum  
Digitoxin, Serum  
Flecainide, Serum or Plasma  
Magnesium (Mg), Serum  
Magnesium (Mg), Urine  
Potassium, Serum or Plasma  
Verapamil, Serum or Plasma

**Synonyms:** Allocar®; Cardioreg®; Digacin; Lanocor®; Lanoxicaps®; Lanoxin®; Lenoxin®; Purgoxin®

**Background:** Digoxin is used in atrial fibrillation and in the treatment of heart failure.

Bioavailability 60%-80%; urinary excretion 50%-70%; plasma binding 20%-25% decreased in renal disease; volume of distribution 7 L/kg; plasma half life 26%-52% decreased in hyperthyroid patients and increased in renal disease, congestive heart failure, elderly, hypothyroid patients, peak time 1-3h.

Steady state reached after 5 days. Levels are increased by quinidine, verapamil, amiodarone, cyclosporine, spironolactone, propafenone through decreased clearance.

Limitations: Low frequent cross reaction with digitoxin, results therefore are not valid in patients on digitoxin. Other digitalis derivatives cross react too.

**Sampling:** 2 mL serum, at least 6 h after dose, best immediately before next dose.

**Reference Interval:**

Therapeutic 0.7-2 ng/mL, (revised to 0.5-0.8 ng/mL)

Toxic starting to be possibly harmful >1.2 ng/mL, particularly in hypokalemia or hypomagnesemia patients.

Highly toxic >2.4 ng/mL. Concentrations of 1.7, 2.5 and 3.3 ng/mL are found to be associated with 10%, 50% and 90% probability of digoxin induced arrhythmias, respectively.

## Dihydrotestosterone, Serum

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**Related Information:** Testosterone, Serum

**Synonyms:** DHT

**Background:** 5-alpha reductase, located in the skin, prostate, internal genitalia, metabolize testosterone to DHT. Since testosterone, which has less androgenic potency than DHT, can be converted to estradiol or to DHT, deficiency in 5-alpha reductase in a rare autosomal recessive disorder in 46XY phenotypic males, patients may develop hypospadias, urogenital sinus opening to the perineum, a blind vaginal pouch and normal testes. Testosterone levels are normal at puberty but DHT is low.

DHT is the parameter which correlates well with male sexual function.

**Sampling:** 2 mL serum

**Reference interval:**

Males	< 20 years	150 – 1240 pg/mL
	20- 39 years	155-553 pg/mL
	> 40 years	150-980 pg/mL
Females	20-39 years	50-250 pg/mL
	> 40 years	50-137 pg/mL

**Diphtheria see** *Corynebacterium diphtheriae*

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**Dopamine, Plasma see** Catecholamines, Plasma

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**Dopamine, Plasma see** Catecholamines, Urine

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## Drug of Abuse Screen, Urine

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**Related Information:** Amphetamine, Urine  
 Cannabinoids (Marijuana Metabolites) Immunological Drug Screen, Urine  
 Cocaine, Urine  
 Diazepam, Serum  
 Ethanol, Blood, Serum or Urine  
 Flunitrazepam, Urine  
 Methadone, Urine  
 Opiates, Quantitative, Urine

**Test includes:** Amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine, methadone, opiates (codeine, heroin, morphine), pH of the urine

**Sampling:** 10 mL random urine, keep refrigerated. If forensic, take precautions to make sure the sample is not substituted, diluted or chemicals added for drug destroy or test disturbance. Ph of the urine is included in the test, further precautions are specific gravity, creatinine to rule out adulteration.

**D-Xylose Absorption Test, Serum** see Xylose Absorption Test, Serum

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## Echinococcosis, Serology

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**Background:** Larval stages of the cestodes (tapeworms) *E. granulosus*, *E. multilocularis* and *E. vogeli* causes diseases in humans.

*E. granulosus* is one of the smallest tapeworms composed of a scolex and 3 proglottides. Definitive hosts are dogs or other canids, the intermediate host are sheep, caribou, deer, moose, pigs or men. The life cycle involves the canid's intestine where eggs are liberated and may be ingested by the intermediate host. The oncosphere embryos emerge in the small intestine and migrate primarily to the liver but also to the brain, the lung or into bones where they develop in a unilocular fluid filled hydatid cyst. The inner layer produces protoscoleces which may infect dogs by contaminated food.

*E. multilocularis*: Main definitive hosts are foxes; the intermediate hosts are various rodents. Human infection is due to accidental ingestion of food contaminated with fox feces, affecting primarily hunters and trappers. Endemic areas are in northern Europe, Siberia, Western Canada, and Alaska. In the human liver, the larvae form multiloculated cysts with few protoscoleces. Since an outer fibrous capsule is not build up, cysts can proliferate and honeycomb like tissue may form (alveolar form).

Polycystic hydatid disease of *E. vogeli* very rarely occurs in humans.

Limitations: Serologic sensitivity for the alveolar form is higher, also higher for the liver than for pulmonary infection. Non specific cross reactivity with other helminths is up to 50%. False positive results are rarely seen in patients with cirrhosis and lupus. False negative sometimes in cases of large cysts or dead cysts.

**Sampling:** 1 mL serum

**Reference Interval:** Report of diagnostic finding of the immunoblot antibody assays for *E. granulosus* and *E. multilocularis*

## Echo Virus, Serology

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**Background:** Echo is an acronym for enteric cytopathic human orphan. Echoviruses have a similar structure as other enteroviruses which are members of the single stranded RNA picornavirus family. The transmission modus of the more than 40 serotypes is the fecal oral route. The viruses in the group are the cause of aseptic meningitis, upper respiratory tract infection, febrile