NutriPATH
Integrative Pathology Services
P: 1300 688 522
E: info@nutripath.com.au
A: PO Box 442 Ashburton VIC 3142

TEST PATIENT
Sample Test Name
Sex : F
Date Collected : 00-00-0000
111 TEST ROAD TEST SUBURB
LAB ID: 00000000 UR#:0000000

TEST PHYSICIAN
DR JOHN DOE
111 CLINIC STREET
CLINIC SUBURB VIC 3000

INTEGRATIVE MEDICINE

URINE, SPOT

	Result	Range	Units

Organic Acids.

Organic Acids Introduction

Organic acids are metabolic intermediates that are produced in pathways of central energy production, detoxification and biotransformation, neurotransmitter breakdown, or intestinal microbial activity. Marked accumulation of specific organic acids detected in urine often signals a metabolic inhibition or blockage. The abnormality may be due to a nutrient deficiency, an inherited enzyme deficiency, toxic interference or drug effect.
Several analytes provide insight into gastrointestinal bacterial or fungal imbalance.
Elevations in any organic acids can contribute to metabolic acidosis
Symptoms from mild, chronic acidurias may not manifest until adulthood
The mitochondrial function markers include:
Fatty Acid Metabolism
Carbohydrate Metabolism
Citric Acid Cycle Intermediates

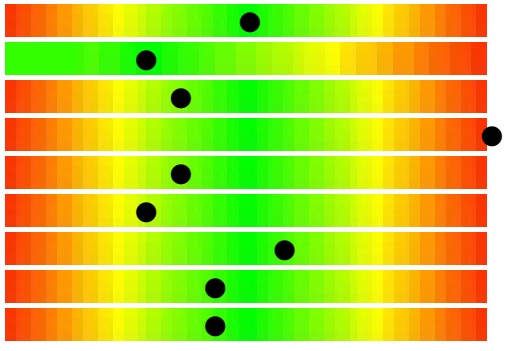
CITRIC ACID CYCLE Metabolites.

Citric Acid Cycle Metabolism

Citric Acid Cycle Metabolites serve both anabolic and catabolic functions. They are the final common pathway of energy release from catabolism of fats, proteins, and carbohydrates.
They are the source of basic structural moleules that are drawn away from the cycle to support organ maintenance and neurological function-anabolic processes
Crossroads of food conversion and utilization.
Spillage of Citric Acid Cycle intermediates into the urine may indicate mitochondrial inefficiencies in energy production. A block in any step may cause a build up of compounds that precede this step.
Amino acids supply carbon skeletons for maintaining mitochondrial concentrations.

Citrate, cis-Aconitate and Isocitrate ar the key organic acids in this biochemical pathway and are responsible for aerobic energy production

Pyruvic Acid.	3.40	0.60 - 6.61	ug/mgCR	
Lactic Acid.	0.19	0.00 - 1.58	ug/mgCR	
Citric Acid.	117.40	37.50 - 417.80	ug/mgCR	
cis-Aconitic Acid.	*59.60* *H	12.00 - 42.90	ug/mgCR	
Isocitric Acid.	16.60	7.80 - 45.90	ug/mgCR	
a-Ketoglutaric Acid.	35.88	10.40 - 168.30	ug/mgCR	
Succinic Acid	8.50	1.80 - 13.30	ug/gCR	
Fumaric Acid.	0.52	0.18 - 1.20	ug/mgCR	
Malic Acid.	0.68	0.30 - 1.45	ug/mgCR	



KETONE/FATTY ACID Metabolites

(*) Result outside normal reference range (H) Result is above upper limit of reference rang

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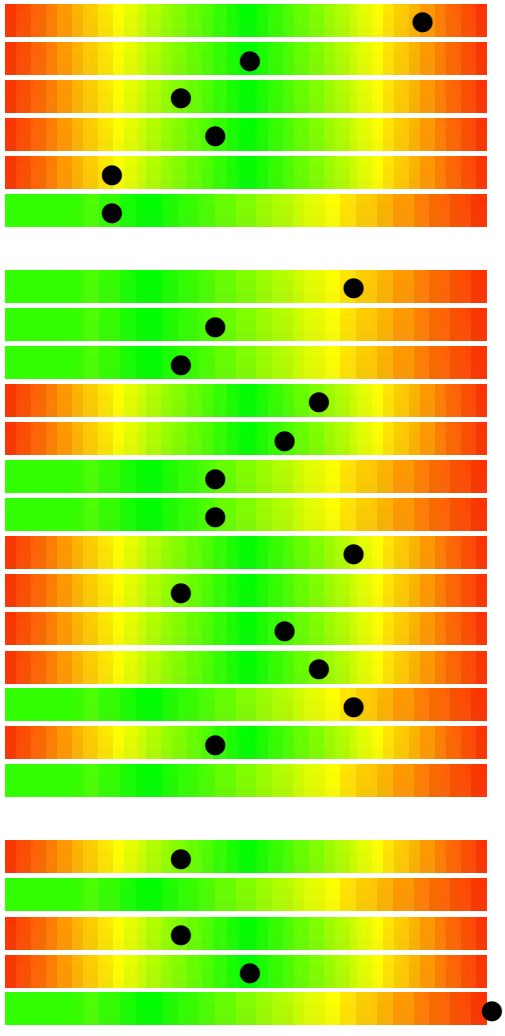
Ketone/Fatty Acid Metabolism

Fatty Acid Metabolism is needed for energy production.

Key Supplements:
Carnitine a metabolic cofactor synthesized from L-Lysine and L-methionine (as SAMe)
Conditionally essential nutrient
Fatty acid transport carrier from cytosol into mitochondria for beta -oxidation
Vitamin B2 (riboflavin)
Aids oxidative metabolism of fats within the mitochondria

Key Organic Acids:
Adipate (Adipic Acid) and Suberate (Suberic Acid)
Functional markers of carnitine insufficiency
Six and eight carbon dicarboxylic acids, respectively
Products of peroxisomal fatty acid oxidation
Indreased when carnitine insufficiency limits long chain fatty acid entry into mitochondria

Adipic Acid.	*3.34* *H	0.46 - 3.01	ug/mgCR
Suberic Acid.	1.43	0.34 - 2.34	ug/mgCR
Ethylmalonic Acid	1.78	1.09 - 4.22	ug/mgCR
Methyl-Succinic Acid	1.27	0.62 - 2.19	ug/mgCR
a-OH-Butyrate	*0.12* *L	0.16 - 2.76	ug/mgCR
b-OH-Butyrate	0.03	0.00 - 1.90	ug/mgCR
ORG. Acids for COFACTOR NEED.			
a-Ketoisovaleric Acid	0.18	0.00 - 0.22	ug/mgCR
a-Ketoisocaproic Acid	0.25	0.00 - 0.57	ug/mgCR
a-Keto-b-Methylvaleric Acid	0.19	0.00 - 0.69	ug/mgCR
beta-Hydroxyisovalerate	5.10	0.15 - 7.03	ug/mgCR
Methylmalonic Acid.	1.29	0.44 - 1.90	ug/mgCR
Formiminoglutamic Acid	3.8	0.0 - 9.0	ug/mgCR
Kynurenic Acid.	2.55	0.00 - 6.27	ug/mgCR
b-OH-b-Methylglutaric Acid	6.22	1.34 - 6.55	ug/mgCR
ParaHydroxyphenyllactate	0.51	0.31 - 1.21	ug/mgCR
Orotic Acid.	0.46	0.14 - 0.64	ug/mgCR
Pyroglutamic Acid.	20.57	6.66 - 23.87	ug/mgCR
Benzoate (OA)	0.35	0.00 - 0.41	ug/mgCR
Hippurate (OA)	278	12.9 - 663	ug/mgCR
Benzoic/Hippuric Acids Ratio	0.0	0.0 - 0.0	RATIO
BACTERIAL DYSBIOSIS MARKERS.			
ParaHydroxyBenzoate	1.1	0.5 - 2.7	ug/mgCR
Phenylacetic Acid.	0.0	0.0 - 0.0	ug/mgCR
2-OH-Phenylacetic Acid	0.55	0.35 - 1.04	ug/gCR
Indoleacetic Acid	2.23	0.18 - 3.98	ug/mgCR
Tricarballylate	*1.91* *H	0.00 - 0.79	ug/mgCR



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YEAST/FUNGAL DYSBIOSIS MARKERS.				
Citramalic Acid	*8.2* ***H**	0.0 - 7.0	ug/mgCR	
Arabinose.	**23.1**	0.0 - 42.3	ug/mgCR	
b-Ketoglutaric Acid.	**0.0**	0.0 - 0.0	ug/mgCR	
Tartaric Acid.	**6.4**	0.0 - 14.1	mmol/molCr	
Environmental Pollutants				
NEUROTRANSMITTER METABS.				
HVA	**2.7**	2.5 - 3.5	mmol/molC	
VMA	**2.9**	2.5 - 3.5	mmol/molC	
5HIAA	**3.1**	3.0 - 4.5	mmol/molC	
Quinolinate (OA)	**5.00**	0.00 - 6.10	ug/mgCR	

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Bacterial Dysbiosis Comment

PHENYLACETATE (PAA) COMMENT
- Intestinal bacterial action on polyphenols, tyrosine or phenylalanine results in PAA production
- Should only be present in background concentrations in healthy subjects
- Is trace product of endogenous phenylalanine, may accumulate in phenylketonuria (PKU)
- May accululate in schizophrenia
- Elimination may be redcud in depression
- May modulate estrogen-mediated cancers

IF PHENYLACETATE HIGH:
Causes: Intestinal bacterial overgrowth, Polypherol intake in the presence of PAA-producing bacteria, Malabsorption of phenylalanine due to low HCI in stomach, PKU

Symptoms/Conditions
Rule out PKU

TREATMENT:
Decrease sugars and amino acids

Indican (Indoleacetate) is a by-product from breakdown of dietary protein in the upper bowel. Bacteria are responsible for the production of indican. Indican is present only at low levels in a healthy person. An elevated level of urinary indican is an indication of upper bowel bacterial overgrowth or dysbiosis. Indican excretion is reduced when the intestines are populated with strains of lactobacillus.
TRICARBALLYLATE COMMENT:
- Tricarhballylate has extremely high affinity for magnesium, preengting its absorption
- Ruminant animal herds can develop severe magnesium deficiency from overgrowth of specific strains of ruminal bacteria that produce tricarballylate
- The disease is caused by overfeeding high-carbohydrate herbage

IF TRICARBALLYLATE HIGH:
Causes: Intestinal bacterial overgrowth, Associated with high dietary carbohydrate, Probably due to microaerophilic bacteria

Symptoms/Conditions
Elements tightly bound by tricarballylate causing decreased intestinal absorption (Magnesium, Calcium, Zinc)

TREATMENT:
- Magnesium, 400mg/day
- Calcium 800mg/day
- Zinc 40mg/d
- Restricted carbohydrate diet

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Cit Acid Cycle Metabs Comment

Pyruvate is the anaerobic breakdown product of glucose. Its further conversion to acetyl-CoA requires the pyruvate dehydrogenase enzyme complex. Pyruvate dehydrogenase requires cofactors derived from thiamin, riboflavin, niacin, lipoic acid, and pantothenic acid for optimal function.
Levels of pyruvate in the tissues are further controlled by the biotin-containing protein, pyruvate carboxylase, which controls the first step in the reformation of glucose from pyruvate. Multiple forms of pyruvate carboxylase deficiency, some of which are biotin responsive, have been reported.

cis-Aconitate Elevated:
An intermediate of the citric acid cycle, an elevated level of this organic acid may be an indication of poor supplies or metabolism of amino acids. A clinical sign is fatigue.
If elevated with orotate, isocitrate and citrate, suspect hyperammonia.

SUPPLEMENTATION RECOMMENDATIONS:
alpha Lipoic Acid, Vitamin B Complex, Cysteine, Iron, Magnesium, Manganese.

Malate Comment:
A high level of this organic acid may be indicative of a need for certain nutrients such as niacin (B3) and Coenzyme Q10.
A low level of this organic acid may be indicative of the need for aspartic acid.

Ketone/FA Metabolites Comment

ADIPIC ACID (ADIPATE) ELEVATED:
Elevated levels of this organic acid may be indicative of a disorder of fatty acid oxidation, carnitine deficiency or possible environmental toxin exposure.
Clinical symptoms may include general weakness/fatigue, mitochondrial dysfunction, nausea, hypoglycemia, recurrent infections, and sweaty feet odor.

SUPPLEMENTATION RECOMMENDATIONS:
B complex (B2, B5), CoQ10, L-Carnitine (may be contraindicatedin patients on thyroid medications), L-Lysine (precursor to L-Carnitine), Other nutrients involved in Carnitine synthesis (Mg, SAMe, Vit B6, ascorbic acid, iron, niacin)

OA Cofactor Need Comment
Hydroxymethylglutarate Comment:
This organic acid, when high, may be indicative of a low level of Coenzyme Q10 or poor synthesis.

Adrenergic Activity Comment
Vanilmandelate is a metabolite of both epinephrine and norepinephrine.

Serotonergic Activity Comment
[5HIAA] is within range. This is the major metabolite of Serotonin.

Xylene Exposure

3,4-Dimethylhippurate	**0.01**	0.00 - 0.11	ug/dg CR	
3-Methylhippurate	**0.06**	0.00 - 0.23	ug/dg CR	
2-Methylhippurate	**1.04**	0.00 - 7.20	ug/dg CR	

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Xylene Exposure Comment

Xylene:
Found in many solvents (paints, perfumes, etc.). Used in insecticide\pesticide application. Metabolized to Methylhippurate, which is measured in urine.
Xylene Metabolites (2-Methylhippurate or 3-Methylhippurate) Comment:
SOURCES OF EXPOSURE
Mainly by inhalation of vapors.
Natural component of petroleum and coal tar.
Motor and aviation fuel additive.
Automotive emissions, poor emission-control devices on older vehicles, poor maintenance practices, aviation fuel, waste and landfill sites, localized industrial discharges and spillage incidents, tobacco smoke.
Topical contact or inhalation of: varnish/polishers, paint, paint thinner, paint remover, shellac, rust preventatives, lacquers, inks, dyes, adhesives, cleaning fluids, degreasing agents, household cleaning products.
Used as a solvent for rubbers, synthetic resins, gums, inks, paint.
Fabric and leather treatments.
Used in the synthesis of plasticizers and in the manufacture of polyester fiber, film, insecticide formulations, and perfumes.
Occupational Exposure: paint and printing ink industries, automobile body and related repairers, photographic processing, rubber, leather, plastics and textile industries, flooring contractor.

EFFECTS
Depression of the central nervous system.
Neuropsychological and neurophysiological dysfunction.
Anemia, thrombocytopaenia, renal damage.
Irritation of mucous membranes, dermatitis, nausea, fatigue, headache, anxiety.
Dyspnea, cyanosis.

METABOLISM
Xylene is metabolized in the liver by cytochrome P-450-dependent multifunction oxidase enzymes, conjugated principally with glycine
and excreted in the urine as methylhippuric acids.
Conjugation with sulfate or glucuronic acid represents a minor pathway.
Urinary levels of 2, and 3-methylhippurate provide a valid complement to ambient monitoring. Although the 2-isomer exhibits a
longer half-life, the 3-isomer is the principle component making up 45-70% of co
Trimethylbenzene Metabolite (3,4-Dimethylhippurate) Comment:
SOURCES OF EXPOSURE
Mainly by inhalation of vapors.
Production occurs during petroleum refining.
Primary use is as a motor fuel additive.
Automotive emissions, poor emission-control devices on older vehicles, poor maintenance practices, diesel engine exhaust.
Solvent in coatings, paint thinners, wood preservatives, cleaners, dry cleaners, degreasers, aerosols, pesticides, printing and inks.
Component of white spirit, the most widely used solvent in the paint and coating industry.
Manufacture of pharmaceuticals, asphalt products, lacquers, varnishes, dyes, perfumes.
Occupational Exposure: scientific labs, janitors/cleaners, dry cleaning industry, automobile body and related repairers, construction laborers, house painters, screen cleaning processes, ski boots finishing, and telephone cable assembly.
People who do considerable home maintenance work or hobby work may be exposed via inhalation or dermal contact with the solvent.

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EFFECTS
Irritation of mucous membranes, dermatitis, dizziness, " d runkenness " , fatigue, headache, anxiety, nervousness.
Cyanosis, cognitive and motor impairment, apnea, bursts of perspiration, cardiac arrest.
Diarrhea, abdominal pains, nausea, blurred vision.
Low frustration tolerance, lack of initiative, apathy, depression, irritability (painter ' s syndrome) .
Neurotoxic.
Decreased erythrocyte, leukocyte and platelet counts.
Carcinogenic
Glomerulonephritis, renal dysfunction.

METABOLISM
Metabolized in the liver by cytochrome P-450 dependent multifunction oxidase enzymes, conjugated with glucuronic acid, glycine, or sulfates for urinary excretion.
Lipophilic and may accumulate in fat and fatty tissues.

Toluene Exposure

Hippurate	**278**	0.0 - 663	ug/mgCR	
Benzoate	**0.35**	0.00 - 0.41	ug/mgCR	

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Toluene Exposure Comment

Toluene:
Found in paints, glues, sanitizing agents, cigarette smoke.
Benzoate is metabolized to Hippurate. Elevations may cause elevated Hippurate independent of Toluene.
Toluene Metabolite (Hippurate) Comment:
SOURCES OF EXPOSURE
Mainly by inhalation of vapors. Produced from petroleum refining.
Automotive and aircraft emissions, poor emission-control devices on older vehicles, poor maintenance practices, high-density traffic locales, gasoline filling stations, refineries, tobacco smoke. The amount of toluene in a single cigarette may vary from 80 to 100 micrograms (?g) .
Blended into gasoline as a component to increase octane number.
Two thirds of its use as a solvent carrier in paints, inks, thinners, coatings, adhesives, degreasers, pharmaceutical products.
Household aerosols, spray paint cans, glues, varnishes, shellac, rust preventatives, solvent-based sanitizing agents and germicides, etc.
Additive in cosmetic products.
Occupational Exposure: paint, printing and leather finishing-industry, rubbercoating industry, shoemakers.
***Hippurate is also the end product of benzoate metabolism. Benzoate may be derived from foods containing sodium benzoate additive.**

EFFECTS
Depression or excitatory effect on the central nervous system -
euphoria followed by disorientation, tremulousness, mood lability, tinnitus, diplopia, hallucinations, dysarthria, ataxia, convulsions,
coma.
Irritation (eyes, nose, throat) , dizziness, taste and olfactory fatigue.
Drowsiness, headache, impaired cognitive and motor function, insomnia, anorexia.
Solvent abuse through " sniffing " toluene -containing products may lead to gross disorientation, neurological impairment and death.

METABOLISM
Toluene is metabolized in the liver by cytochrome P-450 dependent multifunction oxidase enzymes conjugated principally with glycine,
and excreted in the urine as hippuric acid.
Smaller amounts may be conjugated with glucuronic acid. Minor amou

BENZENE EXPOSURE.

t,t-Muconic Acid	**0.06**	0.00 - 0.11	ug/dg CR	

Trimethylbenzene Exposure

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Benzene Exposure Comment

Benzene Metabolite (Trans, trans-muconic acid) Comment:
Sources of Exposure
Natural component of crude and refined petroleum.
Automotive emissions, poor emission-control devices on older vehicles, poor maintenance practices, automotive-refueling operations and industrial emissions.
Emissions during the production of xylene, toluene, styrene and other compounds.
Discharge of industrial wastewater from chemical plants, chemical manufacturing sites, and petrochemical and petroleum industries.
Seepage from underground petroleum storage tanks, waste streams.
By-product of various combustion processes - wood burning, organic wastes, tobacco smoke.
First and second-hand smoke accounts for the largest source of benzene exposure for the general public. The amount of benzene in a single cigarette may vary from 5.9-90 micrograms.
Used in the manufacture of Styrofoam, resins, synthetic fibers and rubbers, gums, lubricants, dyes, glues, paints, and marking pens.
Used as a solvent in scientific labs, industrial paints, adhesives, paint removers/strippers, degreasing agents, carburetor cleaner, rubber cements, some arts and crafts supplies, manufacture of faux leather and rubber goods.
Off-gassing from building material, particleboard, carpet glue, textured carpet, liquid detergent, furniture wax, structural fires, high-density traffic locales, petrol stations.

Occupational Exposure: industries that produce or use benzene or benzenecontaining products - oil refineries, petroleum plants, tire manufacturers, paint and shoe manufacturing plant, petrol stations, active or passive cigarette-smoke inhalation, and areas of heavy vehicular traffic.
Interfering Factors: Sorbic acid and potassium sorbate, common food preservatives, are metabolized to muconic acid, wh

Styrene Exposure

Mandelate	**0.21**	0.00 - 0.31	ug/dg CR	
Phenylglyoxylate	***0.46* *H**	0.00 - 0.40	ug/dg CR	
Mandelate + Phenylglyoxylate	***0.68* *H**	0.00 - 0.64	ug/dg CR	

Styrene Exposure Comment

Styrene:
Used in the manufacturing of rubber, latex, and plastic products
Found in carpet backing, packaging materials, foam cups, etc. Central Nervous System depressant. Genotoxic. Metabolized to Phenylglyoxylate and Mandelate. Exposure best correlates to the sum of the two metabolites.

Phthalate Exposure

Monoethyl Phthalate	***0.27* *H**	0.00 - 0.09	ug/dg CR	
Phthalic Acid	**0.07**	0.00 - 0.50	ug/dg CR	
Quinolinate	**5.00**	0.00 - 6.10	ug/mgCR	

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Phthalate Exposure Comment

Phthalic Acid Ester Metabolite (Phthalate):
SOURCES OF EXPOSURE
Used in the manufacture of plastics to soften resins and impart flexibility. Most widely used plasticizers for the manufacture of polyvinyl chloride (P VC) plastics utilized in vinyl flooring and tile, wall covering, pool liners, tool handles, insulation of wires and cables, garden hoses, construction materials, weather-stripping, canvas tarps, upholstery, some food wrappersand containers, medical equipment containing flexible plastics such as blood bags and tubing, haemodialysis, children ' s toys, dishwasher baskets, notebook covers, flea collars, faux leather, shoe soles, traffic cones, latex adhesives, dyes, some pharmaceutical and pesticide formulations.
Detergents, lubricating oils, automobile parts, automobile undercoating, carpet backing, solvents, and personal-care products such as soaps, shampoo, hair spray, nail polish, and toothbrushes, baby-care products.
Diethyl Phthalate (parent compound of MEP) reported in over 70% of cosmetic products tested. Make fragrance in cosmetics and household products last longer.
Occupational Exposure: Plasticizer and PVC processing plants.
***Quinolinate - Phthalate esters perturb tryptophan metabolism resulting in the accumulation of quinolinic acid, an endogenous excitotoxin implicated in inflammatory neurological disorders.**
***Quinolinate is a metabolite of the essential amino acid tryptophan in the kynurenine pathway. This pathway is chiefly activated by IFN -gamma and IFN -alpha. Quinolinate is markedly elevated in the CNS following trauma or inflammation, and is implicated in neuronal injury through activation of the Nmethyl- D-aspartate (NMDA) receptor.**
Toxicity of phthalate esters, acting as metabolic disrupters, through accumulation o
PHTHALATE:
Used as 'plasticizers' to make plastics flexible. Found in 2\3 of cosmetic products. Also found in time-released drugs and pesticide formulations. Known endocrine disruptors. Linked to male fertility problems. May increase the production of Quinolinate, which plays a role in inflammatory disorders. Shown to have Genotoxic potential in lymphocytes.

HVA/5HIAA Ratio	**0.9**	0.0 - 1.2	RATIO	

Urban Pollution Index

Urban Pollution Comment

URBAN POLLUTION INDEX:
Research has showed that increased exposure to urban-type pollution increases levels of HVA and decreases 5-HIAA.
Looking at the ratio of HVA:5-HIAA may help in assessing one's overall exposure to pollution in general.

Creatinine, Urine Spot.	**9.3**	5.0 - 13.0	mmol/L	

Tests ordered: EPoll,OrgAcs2