Chologenic Diarrhea
Determination of bile acids in stool
_IDK® Bile Acids_ (page 2)

_IDK® Bile Acids_

Vitamin D-binding protein and vitamin D
Are you able to measure all isoforms of vitamin D-binding protein? (page 3)

Hepcidin 25
Immundiagnostik AG receives award at the Falk Symposium in Frankfurt (page 4)

Reviewed for you...
- Inflammatory Bowel Disease
  → Therapy monitoring (page 5)
- Celiac Disease (CD)
  → Screening for patients at risk (page 5)
  → Monitoring of gluten free diet (page 6)
  → genetic CD analysis (page 6)
- Ankylosing Spondylitis
  → Calprotectin in stool (page 7)
- Bladder Cancer
  EGF receptor (page 7)
- Psoriasis
  β-defensin 2 (page 7)
- Coronary Heart Disease
  L-kynurenine: a novel marker for inflammation (page 8)
- Differential diagnosis between bacterial and aseptic meningitis
  Lactoferrin (page 8)
- Diabetes mellitus Type 2
  Glutathione (page 9)

News from the product line
- _IDK®_: The new company brand (page 9)
- _IDK Extract®_
  New buffer for the comprehensive stool analysis in a single extraction step for sample preparation (page 9)
- _IDK® Bile Acids_
  Assay for the _in-vitro_ determination of bile acids in stool (page 2 und page 10)

LABORATORY TIP
Stability of vitamins in blood samples (page 11)

_IDK® Product Tuning_
Improvements for _IDK®_ products (page 10)

Scientific conferences & Events
(page 10)
Several studies have documented bile acid malabsorption in up to 50% of patients with chronic diarrhea (1; 2).

Cholegenic diarrhea appears in clinical pictures which are caused by morphological or structural changes in the organism. These include disease and resection of the ileus, such as Crohn’s disease or ileitis following radiotherapy and small bowel overgrowth syndrome, a situation where colon bacteria reach the ileum and metabolize the bile acids which hinders their re-absorption.

Besides, cholegenic diarrhea may occur without morphologic changes. This is caused by functional anomalies of the re-absorption in the enterohepatic circulation. The reasons are diverse and not fully understood. In literature, the following causes are discussed (1):

• genetic mutation in the Na⁺-dependent transport mechanism of the re-absorption
• accelerated transit in ileum and thus reduced re-absorption
• changes of enzymes, receptors and regulators for the re-absorption of bile acids
• anomalies in bile acid recycling

Cholegenic diarrhea can arise as an adverse reaction of therapies, e.g. biguanid (metformin) for the treatment of diabetes mellitus type 2 or polycystic ovarian syndrome.

Even patients suffering from AIDS, a cholegenic diarrhea may occur as a sign of infection or of a side effect of a drug.

**IDK® Bile Acids** – a non-invasive assay for routine use in differential diagnosis of chronic diarrhea.

**Expert’s statements from literature:**

„Total direct costs for one irritable bowel syndrome patient per year amounted to 791.48 €, comprising roughly 25% for physician visits and tests, 50% for drugs and 25% for hospitalization. Including indirect costs for sick leave, total costs were 994.97 € per patient per year.“ (3)

„Therefore, from an epidemiological perspective and, because it can be specifically treated, patients presenting with irritable bowel syndrome with diarrhea (IBS-D) or chronic diarrhea should be screened for bile acid malabsorption. … measurement of fecal bile acid excretion should be done.“ (1)
25-hydroxy-vitamin D (25(OH)D) and vitamin D-binding protein (VDBP) form a complex which can be absorbed into proximal epithelial tubule cells by receptor-mediated endocytosis. The receptor is megalin. Therefore, 25(OH)D bound to VDBP is the bioavailable form. 25(OH)D represents the storage form of vitamin D in the organism. In the kidney, it is metabolized into the biological active form 1,25-dihydroxyvitamin D in a parathyroid hormone dependent manner.

As a general rule, more than 90 % of black people are homozygous for the Gc1F genotype of VDBP, while more than 90 % of white people are homozygous for the genotype Gc1S or GC2. Moreover, a variety of genetic variants giving rise to VDBP isoforms with differing affinity to vitamin D has been described.

A study of Powe et al. reported differences in the plasma concentration of VDBP by homozygous carriers of the phenotypes.

This is caused by the choice of the assay: While using immunoaassays with monoclonal antibodies, as used in Powe's study, different VDBP isoforms will be detected more or less accurately, depending on their structure. Assays based on monoclonal antibodies differentiate between Gc1F and Gc1S. This leads to presumed lower VDBP results for Gc1F carriers. Subsequently, VDBP will be underrepresented and the bioavailable 25(OH)D will be overestimated.

This observation is confirmed by other groups: using an assay for the immunological determination of VDBP based on monoclonal antibodies results in a reduction of 50 % for black people compared to white people. While using tests on the basis of polyclonal antibody material results in similar concentration of VDBP for both populations.

There even exist considerable differences concerning the recovery rate while comparing assays based on monoclonal vs. polyclonal antibody material.

Standard VDBP assays should therefore be based on polyclonal antibody material raised against VDBP to detect all isoforms in the same manner.

The vitamin D-binding protein ELISA of Immundiagnostik AG (cat. no. K 2314) is based on polyclonal antibody material and shows a recovery rate of approximately 100 %.
HEPCIDIN 25

Immundiagnostik AG receives award at Falk Symposium in Frankfurt

As a negative regulator of the iron absorption from diet and the iron distribution in the tissue, hepcidin plays a central role in iron metabolism and is deemed to be an early predictive marker for functional iron deficiency. In the case of absolute iron deficiency, the hepcidin synthesis is reduced to enhance iron absorption. During inflammation on the other hand, hepcidin synthesis is stimulated by the messenger interleukin-6, which in consequence empties the iron reservoir. Because of different regulatory mechanisms, hepcidin is a useful marker for the differential diagnosis of absolute iron deficiency anemia (low serum level) and anemia with chronic inflammatory diseases (high serum level).

At the annual Falk Symposium (March 6th – 7th 2015 in Frankfurt, „Critical Evaluation of Current Concepts and Moving to New Horizons in the Management of IBD“), the poster titled „Serum hepcidin levels predict intestinal iron absorption with inflammatory bowel disease“ was honored by the jury with a 3rd price.

The poster emphasizes the relevance of hepcidin as a key hormone for the regulation of the iron metabolism. For the first time, a link between hepcidin and iron absorption in patients with irritable bowel syndrome (IBS) was established: Determination of hepcidin can identify non-responders of an iron substitution therapy.

Basal hepcidin levels in patients with IBS, but even patients with iron deficiency anemia, show a higher predictive value for a successful substitution therapy than TSAT or with iron deficiency anemia, show a higher predictive value for a successful substitution therapy than TSAT or transferrin saturation (TSAT). However, hepcidin levels underlay a circadian characteristic, the hepcidin concentration is reduced to enhance iron absorption. During inflammation on the other hand, hepcidin synthesis is stimulated by the messenger interleukin-6, which in consequence empties the iron reservoir. Because of different regulatory mechanisms, hepcidin is a useful marker for the differential diagnosis of absolute iron deficiency anemia (low serum level) and anemia with chronic inflammatory diseases (high serum level).

Hepcidin, a 25 amino acid cysteine-rich peptide which has recently emerged as a master regulator of iron homeostasis, is believed to affect iron metabolism by regulation of iron absorption in the gut and iron recycling from macrophages. Hepcidin functions as a hormone that is regulated by a variety of factors (iron status, inflammation and infection). Although hepcidin expression has been shown to be strongly associated with iron absorption as expression of iron transport proteins in healthy women (1) and in patients with iron deficiency (2), a significant relationship between hepcidin expression and iron absorption in patients with inflammatory bowel disease (IBD) has not been established to date.

The objective of this study was to assess the relationship between serum concentrations of hepcidin and iron absorption in IBD patients using an oral iron absorption test. A second objective was to compare the strength of this relationship with transferrin saturation (TSAT), serum ferritin and hepcidin in isolation significantly predicted iron absorption test. A second objective was to compare the strength of this relationship with transferrin saturation (TSAT), serum ferritin and hepcidin in isolation significantly predicted iron absorption.

Serum hepcidin levels predict intestinal iron absorption in patients with inflammatory bowel disease

M. Wiesenthal1, F. Hartmann1, T. Iqbal1, A. Dignass1, J. Stein1,4

1 Cohn Callia Center, Frankfurt, Germany; 2-Bruninghuus University Hospital, Bornsberg, United Kingdom; 3-Immundiagnostik Marburg Kronsteinwirta, Frankfurt, Germany; 4-Kronsteinwirta, Schwalbach, Frankfurt, Germany.

In conclusion, we demonstrate that hepcidin levels in IBD patients with iron deficiency anemia can be predicted from patients’ baseline hepcidin levels, which have been correlated with iron absorption.

Serum hepcidin levels predict intestinal iron absorption in patients with inflammatory bowel disease.
Poster 139 presented on Falk Symposium 196, March 6 – 7, 2015 , Frankfurt, Germany
GASTROENTEROLOGY/ NUTRITION

Therapy monitoring of inflammatory bowel disease (IBD)

TNFα blockers are used for the treatment of IBD for several years. Due to inflammatory pathological changes of the intestinal mucosa, they often disappear from circulation. The generation of anti-drug antibodies (ADA) reduces their efficiency further. Determination of anti-TNFα trough level and the corresponding anti-drug-antibodies makes monitoring possible. By this strategy the treating physician is able to individually adapt the therapy (see table).

Celiac disease: Screening for patients at risk

Unrecorded cases for celiac disease (CD) are high because of the lack of typical clinical signs. Based on gastroenterological symptoms the prevalence for CD is 1:2500, but based on screening data the prevalence is 1:500.

Patients with typical gastrointestinal symptoms and people at elevated risk (first grade relatives of CD patients, patients with diabetes mellitus or other autoimmune diseases) should be included into a screening program. In the screening anti-transglutaminase IgA and total IgA should be determined. If the IgA level is under the age-adjusted normal range, anti-transglutaminase IgG or anti-gliadin IgG should be measured. All tests have to be performed on a gluten-containing diet. A prompt gastroscopy with duodenal biopsy to determine the Marsh criteria is recommended.

Genetic tests for haplotype HLA-DQ 2 and 8 have to be used in diagnosis of exclusion.
Celiac disease: Monitoring of gluten-free diet

A study on monitoring of gluten-free diet (GFD) was intended to prove that IgA and IgG antibodies against the disease inducing deamidated gliadin peptide (DGP) are more effective for the monitoring of patients with celiac disease (CD) on gluten-free diet (GFD) than anti-transglutaminase type 2 (TG2) IgA. Therefore, children were tested promptly after diagnosis of CD and on GFD. In total, 411 sera of 91 IgA positive children with CD proven by biopsy were included. 98 children with normal duodenal histology were defined as a control group.

Tests for the determination of TG2-IgA, DGP-IgG and DGP-IgA were used according to manufacturer’s information (TheBindingSite, Euroimmun, Phadia and INOVA).

<table>
<thead>
<tr>
<th>CD Diagnosis</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>TG2-IgA</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>DGP-IgG</td>
<td>90 – 100%</td>
<td>97 – 100%</td>
</tr>
<tr>
<td>DGP-IgA</td>
<td>67 – 86%</td>
<td></td>
</tr>
</tbody>
</table>

The test for the determination of TG2-IgA was the most suitable for the diagnosis of CD.

In 47 – 90% of the cases the TG2-IgA titers were above the 10-fold of the upper limit of the normal value. Here the authors demand a harmonization of the tests.

During the GFD, DGP-IgA normalized earlier than DGP-IgG and TG2-IgA. Non-adherence to GFD was proven best with positive TG2-IgA, independent from the assay used.

Combined determination of TG2-IgA and DGP-IgG did not improve the diagnostic value for CD compared to TG2-IgA alone.

Conclusion: Antibodies against tissue transglutaminase are most suitable for the diagnosis as well as for the monitoring of gluten-free diet.

Publication:

Genetic CD analysis: PCR tests for the determination of the specific HLA-DQ2/8 allele for predisposition

Celiac disease (CD) is strongly genetically linked to the human leukocyte antigen haplotypes (HLA)-DQ2 and -DQ8. Therefore, in cases where the diagnosis is not clear or doubtful, a genotyping for differential diagnosis or for diagnosis of exclusion should be done. These cases include asymptomatic individuals with CD-associated accompanying symptoms. The patients have only low antibody concentrations or are negative for CD-specific antibodies but show mild infiltrating changes in biopsy material of the proximal small intestine.

Here – and especially in young children under 2 years of age – the newly prepared ESPGHAN guideline waives another biopsy of the small intestine and highly recommends HLA-DQ genotyping as a “first-line” diagnosis! This HLA-DQ2/8 genotyping can be done quickly and safely with the classical agarose gel PCR tests, as well as with innovative real time PCR tests from Immundiagnostik AG.

Here – and especially in young children under 2 years of age – the newly prepared ESPGHAN guideline waives another biopsy of the small intestine and highly recommends HLA-DQ genotyping as a “first-line” diagnosis! This HLA-DQ2/8 genotyping can be done quickly and safely with the classical agarose gel PCR tests, as well as with innovative real time PCR tests from Immundiagnostik AG.

- anti-human-Tissue-Transglutaminase IgA (anti-hTGF/TGc/TG2 IgA) ELISA K 9399
- anti-Gliadin sIgA ELISA K 9311
- anti-human-Tissue-Transglutaminase sIgA (anti-hTGsIgA) ELISA K 9393
- anti-human-epidermal Transglutaminase IgA (anti-heTG/TGe/TG3 IgA) ELISA K 9396
- MutaGEL® HLA DQ 2+8 PCR KE09020
- MutaPLATE® HLA DQ 2+8 (TM) real time PCR KF190596
- IDK® Caso/Glia-Peptide LC-MS/MS KM8000

Publication:
**SKELETAL SYSTEM**

**Ankylosing spondylitis: Calprotectin is elevated in patient’s stool but not in serum**

While examining inflammatory processes in the gut of patients with ankylosing spondylitis (AS), calprotectin in stool and serum was analyzed. The results of the determinations were set in relation to gastrointestinal symptoms, therapy forms and the course of disease. All patients who met the modified New York criteria for AS were invited to take part in the study. They were asked by questionnaires for their medication, symptoms and course of disease. Mobility testing of the back was performed and stool and serum were analyzed for calprotectin.

Two thirds of the AS patients showed elevated values for fecal calprotectin without an association with gastrointestinal symptoms. Calprotectin in serum was predominantly normal in patients with diagnosed AS which is in contrast to other rheumatoid diseases.

Calprotectin in stool might be an early marker for subclinical gastrointestinal inflammation. Further studies are required.

**Publication:**
Calprotectin in ankylosing spondylitis – frequently elevated in feces, but normal in serum.
Scand J Gastroenterol 47(4): 435-44

---

**NEPHROLOGY**

**Bladder cancer: EGF receptor discovered as a possible predictive biomarker for survival**

The article from Bryan et al. examined 8 different bladder cancer cell lines on their secreted proteins. These data were compared with data from the Human Protein Atlas, and EGFR was selected as a possible risk factor.

In a clinical study, EGFR was determined in urine of 60 controls and 436 patients with bladder cancer in a long term follow-up. In this survey, EGFR was identified as an independent indicator for a poor survival prognosis with a Hazard ratio of 2.89 (95 % CI 1.81–4.62, P<0.001).

Multivariate models including EGFR as well as EpCAM showed that both markers in combination (elevated value for EGFR and decreased value for EpCAM) are specific for the prediction of the survival rate.

EGFR and EpCAM are simple and useful parameters for the examination and treatment of very aggressive bladder cancer.

**Publication:**
Protein shedding in urothelial bladder cancer: prognostic implications of soluble urinary EGFR and EpCAM
Br J Cancer 112(6): 1052-8

---

**DERMATOLOGY**

**Psoriasis: β-defensin 2, a serum marker for disease activity in psoriasis**

Studies show that human β-defensin 2 (hBD-2) is expressed in psoriatic epidermal lesions, but the in-vivo relevance is not clear yet. Jansen et al. detected high concentrations of hBD-2 in the serum of psoriatic patients but not in patients with atopic dermatitis. These concentrations strongly correlated with the activity of the disease according to the PASI score.

Serum hBD-2 seems to be a useful surrogate marker for the disease activity of psoriasis. The differing concentration of hBD-2 in psoriasis and atopic dermatitis might explain the differences in the infection rate of both diseases.

**Publication:**
Jansen PAM et al. (2009)
Beta-defensin-2 protein is a serum biomarker for disease activity in psoriasis and reaches biologically relevant concentrations in lesional skin.

---

**β-defensin 2**
ELISA K 6500
CARDIOVASCULAR AND RENAL SYSTEMS

Coronary heart disease: L-kynurenine as a novel inflammation marker

A huge Norwegian study, with more than 3224 patients included, identified the urinary L-kynurenine-tryptophan ratio (KTR) as a predictor for severe adverse events during angiography (death – odds ratio per pmol/l 2.93; p<0.001). Also, patients with high L-kynurenine levels after the survival of a cardiac arrest are at risk to suffer from a severe adverse event (death at ICU – odds ratio per pmol/l 1.43; p<0.002; see table 2)

Urinary kynurenine-tryptophan ratio is a novel biomarker with negative predictive value for patients with stable coronary heart disease.

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>Hazard ratio (95% confidence interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major coronary events</td>
<td>1.43 (1.29–1.59)</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>1.44 (1.29–1.59)</td>
</tr>
<tr>
<td>Ischaemic stroke</td>
<td>1.12 (0.91–1.37)</td>
</tr>
<tr>
<td>All-cause mortality</td>
<td>1.38 (1.23–1.54)</td>
</tr>
<tr>
<td>CVD mortality</td>
<td>1.53 (1.33–1.75)</td>
</tr>
<tr>
<td>Non-CVD mortality</td>
<td>1.20 (1.00–1.44)</td>
</tr>
</tbody>
</table>


Differential diagnosis between bacterial and aseptic meningitis: Lactoferrin as a biomarker

Dastych et al. examined the diagnostic significance of calprotectin and lactoferrin in cerebrospinal fluid (CSF) to differentiate between bacterial and aseptic meningitis. Therefore, 23 patients with bacterial and 50 patients with aseptic meningitis were tested for calprotectin and lactoferrin as well as the established biomarkers (glucose, total protein, lactate, polymuclear cells) in CSF.

Compared with other biomarkers (calprotectin, polymuclear cells, total protein and lactate), lactoferrin could be identified as an efficient marker for the differentiation between bacterial and aseptic meningitis besides the number of polymuclear cells and lactate.

ENDOCRINOLOGY

Glutathione, a prognostic marker for diabetes mellitus type 2

In his review article, M. Landenberger describes the changes of amino acid concentrations in blood of patients under permanent stress. Reduced glutathione levels are lower under these circumstances. The consequence is a reduced detoxification of the organism and therefore an increased risk for cancer and diabetes.

Publication:
Landenberger M (2015)
Gibt es Frühmarker für den Diabetes mellitus Typ 2?
ZAEN magazin Vol 7(1): 20-22

NEWS FROM THE PRODUCT LINE

PhiCal® Calprotectin ELISAs are now named IDK® Calprotectin ELISAs

With our calprotectin ELISAs, we start a renaming of all ELISA products of Immundiagnostik AG to bundle our products under one company brand: thereby all tests receive a quality mark of Immundiagnostik:

The proven characteristics of the ELISAs are not changed!

One single extraction step using only one tube – this saves time and costs in the laboratory.

IDK Extract®: New buffer for the comprehensive stool analysis in a single extraction step for sample preparation

The following parameters can be measured from the same stool sample diluted with IDK Extract®:

- Calprotectin
- Pancreatic Elastase
- Lactoferrin
- Hemoglobin
- Hemoglobin-Haptoglobin complex
- anti-Transglutaminase antibodies
- anti-Gliadin antibodies
- sIgA
- α1-Antitrypsin
- Albumin
- EDN
- Lysozyme
- β-defensin 2
- bile acids

At the same time, we would like to draw your attention to a new system to order single components of the kits: To reorder single components, please use the catalogue number (see manual, chapter material supplied) followed by the label as product number.

Important: In your reorder, please specify the lot number of the kit in use.

### IDK® Calprotectin ELISAs

- IDK® Calprotectin (MRP 8/14) (Stool, 1h) ELISA K 6927
- IDK® Calprotectin (MRP 8/14) (Stool, 1h, 1-Pt.-Cal.) ELISA K 6967
- IDK® Calprotectin (MRP 8/14) (Urine) ELISA K 6928
- IDK® Calprotectin (MRP 8/14) (Serum, plasma) ELISA K 6935

### IDK® Extract®

- BCAA colorimetric K 7016
- ImAnOx® (TAS/TAC) colorimetric KC5200
- Glutathione (GSH/GSSG) HPLC KC1800

Stool Sample Preparation
...completely relaxed

Stool sample preparation system filled with extraction buffer IDK Extract® K 6999
**NEWS FROM THE PRODUCT LINE**

**IDK® Bile Acids: Assay for the in-vitro determination of bile acids in stool**

*IDK® Bile Acids* is a non-invasive assay for routine use in differential diagnosis of chronic diarrhea (CE marked) (see page 2).

**Product announcement:** An assay for the determination of bile acids in serum and plasma will be available shortly.

*IDK® Bile Acids* colorimetric K 7878

**IDK® PRODUCT TUNING**

**Gastroenterology**

*IDK® Bile Acids* (K 7878), the research product for the *in-vitro* determination of bile acids in stool, received **CE mark** and is now ready for **routine use**.

**Cardiovascular and Renal Systems**

*anti-oxLDL ELISA* (K 7809), assay for the quantitative determination of anti-ox-LDL antibodies in EDTA plasma and serum, received **CE mark**.

**Skeletal System**

*25-OH vitamin D₃/D₂ LC-MS/MS* (KM5000), test kit for the determination of 25-hydroxyvitamin D₃ and 25-hydroxyvitamin D₂ in serum and plasma, **contains 5 calibrators**.

*total sRANKL ELISA* (K 1016), test kit for the determination of free and OPG-bound sRANKL in serum, has been cited in **new literature**.

**Complementary Medicine**

*Glutamate, colorimetric* (K 7731), is now validated for the determination in **serum and plasma**.

**SCIENTIFIC CONFERENCES & EVENTS**

Here you are welcome to meet us in person.

**September 2015**

- **ESPEN Congress on Clinical Nutrition and Metabolism**
  September 5th–8th, Lisboa, Portugal
  Booth no. 8

- **Immundiagnostik Gastro-Symposium**
  September 12th, Mainz, Germany

- **Annual Meeting of the German Society of Nephrology**
  September 12th–15th, Berlin, Germany
  Booth no. G 04

- **3rd Laboratory Forum – Diagnosticum**
  September 16th, Dresden, Germany

- **Visceral Medicine**
  September 16th–17th, Leipzig, Germany
  Booth no. B.12

- **Annual Meeting of the Working Group of Accredited Laboratories**
  September 18th–19th, Essen, Germany

- **Austrian Society of Pediatric Medicine (ÖGKJ)**
  September 24th–26th, Eisenstadt, Austria

**October 2015**

- **ASBMR Annual Meeting**
  October 9th–12th, Seattle, USA
  Booth no. 419

- **German United Society of Clinical Chemistry and Laboratory Medicine (DGKL)**
  October 14th–17th, Leipzig, Germany

- **UEGW (United European Gastroenterology Week)**
  October 24th–28th, Barcelona, Spain
  Booth no. 129

**November 2015**

- **ASN Kidney Week**
  November 3rd–8th, San Diego, USA
  Booth no. 1638

- **Congress Bone and Muscles**
  November 6th–7th, Würzburg, Germany

- **Annual Conference of Environmental Medicine**
  November 13th–14th, Berlin, Germany

- **MEDICA**
  November 16th–19th, Düsseldorf, Germany; Hall 3 Booth no. E59

*We are looking forward to your visit at our booth!*
Stability of vitamins in blood samples

Vitamins are sensitive against different chemical and physical factors like oxidation, high temperature, light, pH and ion strength. Therefore, Cuerq et al. studied the preanalytical stability of the vitamins A, E, K, B1, B2, B6, B9, B12, C and the carotenoids with various blood collection systems (EDTA, heparin, clot activator) in blood samples of healthy subjects. The authors concluded as follows:

- Only vitamin B6, vitamin C and serum folate showed clinical and statistical changes by delayed centrifugation
- Vitamin B9 gradually increased with time
- Serum folate significantly decreased with time
- As expected, vitamin C concentrations decreased after exposure to light at room temperature with or without acidifying the sample. Clinical significant changes were observed after storage at room temperature and under light exposure for 6 to 24 hours. The accepted storage time was 24 hours for storage at +4 °C.
- The optimal storage temperature was determined to be -20 °C, here only minimal changes for storage times of up to 48 hours were observed. There was no difference for acidified samples for a 28 day storage at -20 or -80 °C.
- Vitamins A, E, K, B1, B2, B12, folate and carotenoids showed a good stability for up to 48 hours at room temperature and under light exposure.

Publication:
Overview of the in vitro stability of commonly measured vitamins and carotenoids in whole blood

Please find more information on our website www.immundiagnostik.com

EDITING: Dr. Sonja Bastian, Dr. Corinna Berger, Dr. Susanne Duncker, Dr. Anastasia Stemke, Dr. Karl Florian Wintgens

IMPRESSUM
Immundiagnostik AG
Stubenwald-Allee 8a
64625 Bensheim, Germany
Phone: +49 6251 701900
Fax: +49 6251 849430
info@immundiagnostik.com
www.immundiagnostik.com

Pictures (if not stated otherwise): Shutterstock