Gastrointestinal Diseases

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   - Hemoglobin-Haptoglobin-Complex
   - Helicobacter pylori
   - Histamin
   - Lysozyme
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   - MutaGEL® Laktase (AS)
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   - CRP (C-reactive Protein)
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   - Diamine Oxidase (DAO)
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   - S100A12 (Calgranulin C, EN-RAGE)
   - Salmonella sp.
   - Secretory IgA (slgA)
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   - Zonulin

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   - β-Defensin 2
   - Diamine Oxidase (DAO)
   - EDN (EPX)
   - Enterovirus
   - Ferritin
   - Gladin
   - Gliadorphin (Gliadomorphin)
   - Hemoglobin
   - Hemoglobin-Haptoglobin-Complex
   - Helicobacter pylori
   - Histamin
   - Lysozyme
   - MutaGEL® Aldolase B
   - MutaGEL® HLA-DQ 2+8
   - MutaGEL® Laktase (AS)
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   - S100A8/A9 (MRP 8/14, Calprotectin)
   - S100A12 (Calgranulin C, EN-RAGE)
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   - β-Defensin 2
   - Diamine Oxidase (DAO)
   - EDN (EPX)
   - Enterovirus
   - Ferritin
   - Gladin
   - Gliadorphin (Gliadomorphin)
   - Hemoglobin
   - Hemoglobin-Haptoglobin-Complex
   - Helicobacter pylori
   - Histamin
   - Lysozyme
   - MutaGEL® Aldolase B
   - MutaGEL® HLA-DQ 2+8
   - MutaGEL® Laktase (AS)
   - Norovirus
   - Myeloperoxidase (MPO)
   - Pancreatic Amylase
   - Pancreatic Lipase
   - PMN-Elastase
   - S100A8/A9 (MRP 8/14, Calprotectin)
   - S100A12 (Calgranulin C, EN-RAGE)
   - Salmonella sp.
   - Secretory IgA (slgA)
   - TNF-α
   - Zonulin

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   - D-Arabinitol
   - Calprotectin (PhiCal® Calprotectin)
   - CRP (C-reactive Protein)
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   - β-Defensin 2
   - Diamine Oxidase (DAO)
   - EDN (EPX)
   - Enterovirus
   - Ferritin
   - Gladin
   - Gliadorphin (Gliadomorphin)
   - Hemoglobin
   - Hemoglobin-Haptoglobin-Complex
   - Helicobacter pylori
   - Histamin
   - Lysozyme
   - MutaGEL® Aldolase B
   - MutaGEL® HLA-DQ 2+8
   - MutaGEL® Laktase (AS)
   - Norovirus
   - Myeloperoxidase (MPO)
   - Pancreatic Amylase
   - Pancreatic Lipase
   - PMN-Elastase
   - S100A8/A9 (MRP 8/14, Calprotectin)
   - S100A12 (Calgranulin C, EN-RAGE)
   - Salmonella sp.
   - Secretory IgA (slgA)
   - TNF-α
   - Zonulin
1. Colorectal cancer: prevention and early detection

Colorectal cancer includes cancerous growths in the colon, rectum and appendix. With more than 900,000 deaths worldwide per year, colorectal cancer is the fourth most common form of cancer in the United States and the third leading cause of cancer-related death in the Western world.

Most colon cancers arise sporadically but about 10% of afflicted patients have a familiar genetic disposition. During pathogenesis, adenomatous polyps in the colon arise and are usually benign at the beginning. Some polyps however develop into cancer over years.

This time window offers the chance for prevention because colon cancer has a good healing prognosis - if diagnosed early. The risk for polyps increases significantly beyond age 50. Hence, most public health care services offer preventive programmes for people above 50 that include colonoscopy and screening tests for faecal occult blood. Immunological assays specifically detect human hemoglobin in stool and have demonstrated superior performance in the past years in comparison to guaiac-based chemical occult blood tests.

Fig.: International colorectal cancer incidence rates by gender. Worldwide, there are 945,000 cases of colorectal cancer per year and 492,000 deaths (Parkin et al., 2005).
Hemoglobin
Hemoglobin-Haptoglobin-Complex

In contrast to commercially available rapid tests based on guaiac dye for the
determination of hemoglobin, this hemoglobin ELISA does not require previous
adherance to a diet (no raw meat etc.) and recognises human hemoglobin in 100-fold
lower concentrations. This avoids false-negative results. Due to the choice of antibo-
dies, false-positive results are practically absent.
Recent data show that when determining the hemoglobin-haptoglobin-
complexes (Hb-Hp complexes), the clinical specificity and sensitivity can be increased
in cases of polyps and carcinoma.

Indications

- Occult blood in stool
- Crohn’s disease, ulcerative colitis
- Suspicion of colon carcinoma
- Polyps in the colorectum

Reference Values Hemoglobin
Stool: < 2 µg/ml

Reference Values Hemoglobin-Haptoglobin-Complex
Stool: < 2 µg/ml

Hemoglobin (ELISA)
Sample volume 100 µl
Matrix Stool
Detection limit 0.447 µg/ml
Calibrators 0.21 - 50 µg/g
Incubation time 1 h; 1 h; 15 min
Tests 96
Cat. No. K 7816D

Hemoglobin-Haptoglobin-Complex (ELISA)
Sample volume 100 µl
Matrix Stool
Detection limit 0.081 µg/ml
Calibrators 0.21 - 50 µg/g
Incubation time 1 h; 1 h; 15 min
Tests 96
Cat. No. K 7817D

1-Point-Calibration is possible for both assays
(Cat. No. K 7836D, K 7837D)

References
Schmidt-Gayk H et al. (1994) Clin Lab 40:77-81
2. **Inflammatory bowel diseases**

Chronic diarrhea combined with gastrointestinal symptoms without infectious causes is indicative for a chronic, functional bowel disease.

*In irritable bowel syndrome (IBS)*, digestion is impaired but the gut is not pathologically altered. The treatment of IBS differs individually and includes motility-modulating agents or even anti-depression therapy.

*In contrast to IBS, inflammatory bowel diseases (IBD) like Morbus Crohn or Ulcerative Colitis are caused by inflammatory pathologies of the gut mucosa and pose a severe health threat. IBD therapy therefore relies on inhibition of inflammation.*

Since the symptoms for IBD and IBS overlap to a large degree, Immundiagnostik offers a broad portfolio of user-friendly ELISAs for the differential diagnosis of the diseases. Furthermore, we provide exclusive assays for monitoring and individual modulation of IBD-therapy which enable a comprehensive patient management.

The gut mucosa is an important immunocompetent system which performs local and systemic defense and control functions. To protect the organism, the mucosa establishes a powerful mechanic and immunological barrier - a complex system of immune and epithelial cells as well as a bacterial shield, the physiological intestinal flora.

**Disruptions of the intestinal barrier**, e.g. by mucosa irritations can increase the intestinal permeability and can lead to a *"leaky gut syndrome"*, an overactivation of the immune system with elevated release of pro-inflammatory cytokines.

As a result, food intolerances, susceptibility to infections or nutrient deficiencies can occur. A pro-inflammatory alteration of the intestinal mucosa has also been observed in IBD patients. The status of the intestinal barrier and of the intestinal immune system can be interpreted by an evaluation of marker proteins with the Immundiagnostik ELISA portfolio.

**Indications**
- Exclusion of intestinal infections
- Differentiation of IBS and IBD
- Diagnosis of Morbus Crohn, Colitis ulcerosa
- Therapy monitoring of IBD patients
- Integrity of the intestinal barrier
- Analysis of the intestinal immune system
Albumin

Changes in the albumin concentration in plasma, urine and stool are mainly caused by distribution defects, rather than metabolic defects. In the case of nutrition withdrawal, the concentration of albumin falls below the lower reference range after one week at the earliest. In the case of protein deficient nutrition, the extent of edemas slightly correlates with the albumin concentration. Higher losses of albumin, e.g. nephrotic syndrome, leads to an increased synthesis.

Increased albumin as well as increased haemoglobin concentrations in the stool are not only found in cases of colorectal carcinoma, but also in patients with polyps and chronic inflammatory bowel diseases (Crohn's disease, ulcerative colitis).

**Indications**

- Detection of bleeding in the lower gastrointestinal tract
- Identification of colorectal carcinomas
- Crohn's disease, ulcerative colitis
- Examination of risk groups

<table>
<thead>
<tr>
<th>Reference Values Albumin</th>
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<tbody>
<tr>
<td>Plasma, Serum</td>
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<tr>
<td>Urine</td>
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<td>Stool</td>
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<table>
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<tr>
<th>Albumin (ELISA)</th>
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<tr>
<td>Sample volume</td>
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<td>Matrix</td>
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<td></td>
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<tr>
<td>Detection limit</td>
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<tr>
<td>Calibrators</td>
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<tr>
<td>Incubation time</td>
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<tr>
<td>Tests</td>
</tr>
<tr>
<td>Cat. No.</td>
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</tbody>
</table>

References

John et al. (1994) Clin Lab 40:77-81
\(\alpha_1\)-Antitrypsin

(Alpha-1-Proteinase-Inhibitor)

The \(\alpha_1\)-antitrypsin acts as a primary inhibitor of elastase from polymorphonuclear neutrophilic granulocytes (PMN) and is released during inflammatory processes in order to reduce the proteolytic activity of the PMN elastase in the inflammation region. In addition, it inhibits, via complex formation, a series of serin proteinase such as blood-clotting proteinases, trypsin, chymotrypsin, etc. Thus, the \(\alpha_1\)-antitrypsin plays an important regulatory as well as antiinflammatory role.

The \(\alpha_1\)-antitrypsin is a linear glycoprotein with a molecular weight of ca. 52 kDa (394 amino acid residues), a free cysteine residue and three carbohydrate side chains. It is predominantly synthesized in the liver but also by intestinal macrophages, monocytes and epithelial cells. Although \(\alpha_1\)-antitrypsin is the main serine proteinase inhibitor in human plasma, the proof of fecal \(\alpha_1\)-antitrypsin has become an important marker for intestinal protein loss and permeability as it is able to resist degradation in the gut due to its anti-proteolytic activity. It will, therefore, stay intact and it is possible to detect it in the feces using an immunoassay.

Moreover, the measurement of fecal \(\alpha_1\)-antitrypsin concentration is used to evaluate and monitor chronic inflammatory intestinal diseases. In the clinical routine, the \(\alpha_1\)-antitrypsin-clearance (ratio of the \(\alpha_1\)-antitrypsin-ELISA-values of stool and serum samples) has been established along with the sole determination of the 24h-\(\alpha_1\)-antitrypsin-secretion in stool. Thus, the group of J. S. Fordtran (Strygler et al. 1990) reports that the sole determination of the \(\alpha_1\)-antitrypsin-concentration in stool yielded false positive or false negative results in 21% of the patients compared to the \(\alpha_1\)-antitrypsin clearance measurements.

In a comparative study with the radial immune diffusion (RID), routinely used in the clinical diagnostics, the \(\alpha_1\)-antitrypsin-ELISA developed by Immundiagnostik demonstrated significant advantages in the analysis of serum, stool and Caco-2-cell culture supernatants (Faust et al. 2001):

- The \(\alpha_1\)-antitrypsin concentrations obtained by the ELISA were on average about 30 % higher than the corresponding values from the radial immunodiffusion measurements.
- Only the ELISA-system detected \(\alpha_1\)-antitrypsin in the cell culture supernatants.

The results clearly demonstrate that our \(\alpha_1\)-antitrypsin-ELISA-test is more sensitive than other routinely used methods and that it recognizes the hepatic as well as the enteral \(\alpha_1\)-antitrypsin form. This newly developed test represents a promising alternative to the use of current clinical routine methods. It is superior to the radial immune diffusion especially in cases with extremely high protein loss through the intestine. The combination of two specific antibodies eliminates, to a large extent, the possibility of false negative results guaranteeing reliable diagnoses. This newly developed test is a non-invasive, simple test for the detection of protein loss through the intestine.

**Indications**

- Enteric protein loss and intestinal permeability
- Morbus Crohn
- Necrotic enterocolitis
- Viral, bacterial or allergic inflammation
2. Inflammatory bowel diseases

<table>
<thead>
<tr>
<th>Reference values $\alpha_1$-Antitrypsin</th>
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<tbody>
<tr>
<td>Stool (100mg)</td>
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<td>Serum</td>
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<tr>
<th>$\alpha_1$-Antitrypsin (ELISA)</th>
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<tbody>
<tr>
<td>Sample volume</td>
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<tr>
<td>Matrix</td>
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<tr>
<td>Detection limit</td>
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<tr>
<td>Calibrators</td>
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<tr>
<td>Incubation time</td>
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<table>
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<th>$\alpha_1$-Antitrypsin Clearance (ELISA)</th>
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<tr>
<td>Calibrators</td>
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<tr>
<td>Tests</td>
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<td>Cat. No.</td>
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</tbody>
</table>

![Figure: Comparison ELISA versus RID for stool and serum samples of alpha$_1$-proteinase inhibitor (Faust D et al. 2001, Z Gastroenterol 39: 769-74)](image)

References
Faust et al. (2001) Z Gastroenterol 39:769-774
Karbach et al. (1989) Gastroenterol 27:362
**PhiCal® Calprotectin**

Calprotectin (MRP 8/14) is a heterodimer of two calcium-binding proteins present in the cytoplasm of neutrophils and expressed by the membranes of monocytes. It constitutes nearly 60% of the soluble cytosol proteins in neutrophils and plays a central role in neutrophil defense. Upon neutrophil activation or endothelial adhesion of monocytes, calprotectin is released and may be detected in serum, body fluids or stool as a potentially useful clinical inflammatory marker.

The acute phase protein shows a high stability in faeces (stable for one week at room temperature) and has been established as a faecal marker of inflammatory bowel diseases (IBD). It allows a reliable differentiation between organic intestinal diseases (e.g. chronic inflammatory diseases, infectious diseases, polyps, colon cancer) and functional intestinal diseases (e.g. irritable bowel syndrome).

Calprotectin is ideal for monitoring disease activity (e.g. of M. Crohn or after polyp resection) and early detection of relapse. It shows high sensitivity in the detection of colorectal carcinoma (CC) and polyps (CRC: sensitivity 100%, polyps: sensitivity 88%). Due to the highly specific monoclonal antibodies that the immunoassay is based on, the test detects only human calprotectin. The linear part of the calibration curve was optimised for easier differentiation between negative values, poorly increased values and high calprotectin values. This differentiation is important for excluding functional intestinal diseases (e.g. irritable bowel syndrome) and for the diagnosis and monitoring of organic intestinal diseases.

The better performance of the monoclonal test system in comparison to the polyclonal system has been shown in a study about prospective validation of faecal leucocyte markers in the differential diagnosis of chronic diarrhoea. There our monoclonal test system has proven to have a better performance than the polyclonal system which was used. Naumann et al. (2004) also found that of the investigated stool parameters (Calprotectin, MPO, Lactoferrin) only calprotectin was qualified for discriminating between an organic diarrhoea and a functional diarrhoea.

**Determination of faecal Calprotectin**

- Chronic diarrhoea
- Morbus Crohn, Colitis ulcerosa
- Polyps

![PhiCal® is a German trademark of Immundiagnostik AG, Bensheim](image)

**Reference Values Calprotectin**

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<tr>
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<tbody>
<tr>
<td>Stool:</td>
<td>&lt; 15 µg/g</td>
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<tr>
<td>Grey area:</td>
<td>15 - 50 µg/g</td>
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<td>Cut off:</td>
<td>50 µg/g</td>
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**Calprotectin in faeces:**

**High stability in faeces**

*Ideal for monitoring disease activity (e.g. of M. Crohn or after polyp resection) and early detection of relapse*

*For the discrimination between an organic and a functional diarrhoea*

**High sensitivity in detection of colorectal carcinoma (CC) and polyps**

![NEW!](image)

**PhiCal® Calprotectin (Stool) (not sold in the USA)**

<p>| | |</p>
<table>
<thead>
<tr>
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<tr>
<td>Sample volume</td>
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<td>Matrix</td>
<td>Stool</td>
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<tr>
<td>Standards</td>
<td>13 - 840 ng/ml</td>
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<td>Incubation time</td>
<td>1 h; 1 h; 10-20 min</td>
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<td>Test principle</td>
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<tr>
<td>Tests</td>
<td>96 Determinations</td>
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<td>Cat. No.</td>
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![NEW!](image)

**PhiCal® Calprotectin (Stool) (not sold in the USA)**

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</table>
S100A8/A9 in biological liquids

The heterocomplex Calprotectin consists of the two proteins, S100A8 (calgranulin A) and S100A9 (calgranulin B), also designated as MRP8 and MRP14, respectively. Expression of S100A8 and S100A9 in epithelial tissues was first described in context with squamous epithelia and with murine and human wound repair. More recently, an association of S100 protein expression with adenocarcinomas in humans has emerged.

Various conditions have shown significant correlation of S100A8/A9 (or S100A8, S100A9) levels with disease activity:

- Concentrations of S100A8/A9 in serum, and particularly in synovial fluid, correlate strongly with disease activity in rheumatoid arthritis.
- Plasma S100A8/A9 levels are very early, specific and sensitive prediction markers for acute rejection in kidney allograft transplantation.
- Serum S100A8/A9 concentration is a prognostic marker of recurrent infection and of poor survival in alcoholic liver cirrhosis.
- S100A8/A9 is useful for evaluating the extent of periodontal inflammation.
- In cerebral malaria, S100A8/A9 expression correlates with microglial activation in brain.
- S100A8/A9 is present in urinary stones and in dental calculus.
- S100A9 in serum may serve as a useful marker for discrimination between prostate cancer and benign prostatic hyperplasia (BPH).

For Research only; not suitable for human samples

S100A8/A9 (MRP 8/14, Calprotectin, Mouse/Rat)

<table>
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<tr>
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<tbody>
<tr>
<td>Matrix</td>
<td>Serum, plasma, urine, cell culture supernatant, tissue extract Stool (100 mg)</td>
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<tr>
<td>Standards</td>
<td>0 - 15.6 ng/ml</td>
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<td>Incubation time</td>
<td>4 x 1 h, 20 min</td>
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<td>Test principle</td>
<td>ELISA</td>
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<tr>
<td>Tests</td>
<td>96</td>
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</table>

Only for research purposes: S100A8/A9 in biological liquids

Suitable for mouse/rat and other animal samples

Not suitable for human samples

*Not sold in the USA*
2. Inflammatory bowel diseases

<table>
<thead>
<tr>
<th>S100A12 (Calgranulin C, EN-RAGE)</th>
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<tbody>
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<td><strong>Sample volume</strong></td>
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<td><strong>Matrix</strong></td>
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<td><strong>Standards</strong></td>
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<td><strong>Incubation time</strong></td>
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<td><strong>Test principle</strong></td>
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References
Loitsch SM et al. (2010). Gastroenterol 138:5, Suppl 1, S-S28
Pezzilli R et al. (2007) J Gastroenterol 42(9):754-60
Schroeder O et al. (2007) Aliment Pharmacol Ther
Shastri YM et al. (2006) Poster presented at Conference of Indian Society of Gastroenterology, November
Langhorst J et al. (2005) Inflamm Bowel Dis 11:1085-1091
Tibble JA et al. (1999) Gut 44:35

Sample volume 100 mg
Matrix Stool
Standards 0.22-54 ng/ml
Incubation time 2 x 1 h
Test principle ELISA
Tests 96
Cat. No. K 6938
C-reactive protein (CRP) is an acute phase protein which is synthesized in the liver and released into the circulation. The CRP concentration in blood rises during inflammatory (infectious and non-infectious) diseases. CRP is considered a non-specific parameter which is useful for the evaluation of the severity of an inflammation. Elevated CRP levels always have to be clarified, even without clinical symptoms.

Generally, the CRP concentration in plasma mirrors a disease activity with a lag of 12-24 hours. CRP values close to the normal range (highly sensitive CRP or hsCRP) are an important risk parameter for cardiovascular diseases (Ridker et al. 1997).

In addition, CRP is utilized as a marker for intestinal inflammation. The determination in serum is an additional tool in primary diagnosis to border irritable bowel syndrome. Furthermore, CRP levels are analysed as part of IBD therapy monitoring (Langhorst et al., 2008; Vermeire et al., 2006). During remission, persistent elevated CRP concentrations indicate a higher relapse risk in IBD patients. The combination of CRP with other laboratory parameters such as calprotectin or PMN-elastase strengthen the diagnostic value in the evaluation of an intestinal inflammation.

**Indications**

- Intestinal inflammations
- Differential diagnosis of chronic inflammatory bowel diseases
- Therapy monitoring of chronic inflammatory bowel diseases

**References**

**β-Defensin 2**

The β-defensins are an integral part of the congenital immune system and contribute with their antimicrobial effect to the barrier function of the intestinal epithelial cells. Defensins exert a variable degree of antimicrobial activity against bacteria, fungi, and some enveloped viruses. Vertebrate defensins are classified as α- or β-defensins, based on their pattern of disulfide bridges. Nine human defensins of epithelial origin have been found, three of them being β-defensins (HBD-1, -2 and -3).

The expression of β-Defensins is induced by the pro-inflammatory cytokines and also through microorganisms (e.g. *E. coli*, *H. pylori* or *P. aeruginosa*).

A reduced β-defensin 2 expression can, for example, be observed in the intestinal mucous of patients with Crohn’s disease. The defense system of the mucous membrane is therefore restricted and allows an increased invasion of bacteria, which could possibly lead to a typical infection in Crohn’s disease patients.

Whether the reduced β-defensin-2 expression could even play a role in the development of Crohn’s disease is currently being researched, as is the possibility that it is the probiotic bacterium which produces β-defensin.

**Indications**

- Inflammatory bowel diseases (IBD)
- Research of intestinal barrier function

<table>
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<tr>
<th>β-Defensin 2 (ELISA)</th>
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<tbody>
<tr>
<td>Sample volume</td>
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<td>Matrix</td>
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<td>Standards</td>
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<tr>
<td>Tests</td>
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<td>Cat. No.</td>
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**Reduced β-defensin expression with Crohn’s disease (HBD-2)**

**Increased β-defensin expression with Colitis ulcerosa (HBD-2)**

**Also available for your research:**

→ β-Defensin 5 ELISA and
→ β-Defensin 6 ELISA

References
Ferritin

Iron deficit and the resulting anaemia are common disorders which can be caused e.g. by malnutrition, malabsorption or bleedings. The determination of iron and the binding capacity of total iron is therefore clinically relevant. Molecules like hemoglobin, hemosiderin, myoglobin and cytochromes act as iron reservoirs in the body. In most tissues however, ferritin is the prevalent iron storage. Human ferritin has a molecular weight of 450 kDa and consists of a protein shell covering an iron core - each ferritin molecule can carry up to 4000 Fe$^{2+}$ atoms. Almost 20% of total iron in healthy individuals is stored in this way.

The function of ferritin is the oxidation of Fe$^{2+}$, the transport of Fe$^{3+}$ into the core and its mobilisation. High ferritin concentrations can be detected mainly in liver, spleen and bone marrow.

The direct determination of iron in blood is not applicable for the detection of an iron deficit since these values are too inconsistent. Although the majority of ferritin molecules is intracellular, the ferritin concentration in serum is a meaningful parameter for the assessment of the total iron storage in the organism.

The analysis of the ferritin level is meanwhile routine in laboratory diagnostics and serves as a significant parameter in the diagnosis of anaemia and hemochromatosis. High ferritin concentrations indicate an iron overload which might be caused by hemochromatosis. Furthermore, ferritin status is used to monitor the iron supply in pregnant women, blood donors and dialysis patients. Ferritin serum concentrations also serve as an additional diagnostic parameter in inflammations, chronic liver diseases and tumors.

**Indications**

- Diagnosis of iron deficit / anaemia
- Diagnosis and therapy monitoring of hemochromatosis
- Iron status in pregnant women, blood donors and dialysis patients

**For Research Use only**

<table>
<thead>
<tr>
<th>Ferritin (ELISA)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sample volume</strong></td>
<td>20 µl</td>
</tr>
<tr>
<td><strong>Matrix</strong></td>
<td>Serum</td>
</tr>
<tr>
<td><strong>Calibrators</strong></td>
<td>15 – 1000 ng/ml</td>
</tr>
<tr>
<td><strong>Incubation time</strong></td>
<td>45 min; 20 min</td>
</tr>
<tr>
<td><strong>Tests</strong></td>
<td>96 determinations</td>
</tr>
<tr>
<td><strong>Cat.No.</strong></td>
<td>KD1872</td>
</tr>
</tbody>
</table>

**References**

Valberg L (1980) CMAJ. 122: 1240
Hazard JT et al. (1977) J. Blood. 49: 139
Smimes MA (1974) Blood. 43:581
Lysozyme

Lysozyme (muramidase) is a protein with a molecular weight of approx. 15 kDa and belongs to the group of alkaline glycosidases. Lysozyme is synthesised by granulocytes, monocytes and macrophages. The main source of faecal lysozyme is the intestinal granulocytes.

Lysozyme can be detected in all cells of the inflammatory infiltrate during an acute flare of Crohn’s disease. To some extent, lysozyme is also secreted actively by mononuclear cells into the bowel lumen.

**Indications**

- Diagnosis and monitoring of Crohn's disease, Boech's disease (in serum)
- Bacterial, viral, allergenic or autoimmune related bowel inflammations of allergenic or auto immune origin

### Reference values Lysozyme

<table>
<thead>
<tr>
<th>Sample</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stool</td>
<td>&lt; 600 ng/ml</td>
</tr>
</tbody>
</table>

### Lysozyme (ELISA)

<table>
<thead>
<tr>
<th>Sample volume</th>
<th>100 µl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matrix</td>
<td>Stool (100 mg), Serum</td>
</tr>
<tr>
<td>Detection limit</td>
<td>0.5 ng/ml</td>
</tr>
<tr>
<td>Calibrators</td>
<td>1.1 – 30 ng/ml</td>
</tr>
<tr>
<td>Incubation time</td>
<td>1 h; 1 h; 15 min</td>
</tr>
<tr>
<td>Tests</td>
<td>96</td>
</tr>
<tr>
<td>Cat. No.</td>
<td>K 6900</td>
</tr>
</tbody>
</table>

Also available as 1-point-calibration test (Cat. No. K 6901)

References

Langhorst J et al. (2005) Inflamm Bowel Dis 11(12):1085-91
Myeloperoxidase (MPO)

The granules of neutrophils (approx. 70 % of the white blood cells) contain a large number of different enzymes. Myeloperoxidase (MPO) catalyses the oxidation of substances via $\text{H}_2\text{O}_2$. The MPO $\text{H}_2\text{O}_2$ system has a toxic effect on many microorganisms such as bacteria, fungi, viruses and mycoplasma.

The efficiency of the bactericidal myeloperoxidase $\text{H}_2\text{O}_2$ system is increased by PMN-Elastase. The MPO determination in the stool reflects the inflammatory activity of Crohn’s disease or ulcerative colitis.

**Indications**

- Marker for inflammatory activities in the gastrointestinal tract
- Renal transplant rejection

### Reference Values Myeloperoxidase (MPO)

<table>
<thead>
<tr>
<th>Matrix</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stool</td>
<td>$&lt; 2000$ ng/ml</td>
</tr>
<tr>
<td>Serum</td>
<td>mean value $340$ ng/ml (SD 176.7)</td>
</tr>
<tr>
<td>EDTA-Plasma</td>
<td>mean value $98.31$ ng/ml (SD 62.9)</td>
</tr>
</tbody>
</table>

### MPO (ELISA)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
<td>100 µl</td>
</tr>
<tr>
<td>Matrix</td>
<td>Stool (100mg), Urine</td>
</tr>
<tr>
<td>Calibrators</td>
<td>3.6 – 100 ng/ml</td>
</tr>
<tr>
<td>Incubation time</td>
<td>1 h; 1 h; 1 h; 10-20 min</td>
</tr>
<tr>
<td>Tests</td>
<td>96</td>
</tr>
<tr>
<td>Cat. No.</td>
<td>K 6630</td>
</tr>
</tbody>
</table>

References:

- Kazunori et al. (1996) Am J Gastro, Vol 91(S)
PMN-Elastase

PMN-Elastase from human polymorphonuclear granulocytes is a glycoprotein of 30 kDa and belongs to the group of serine proteases. Active PMN-Elastase is released from azurophil granula of neutrophil granulocytes after irritation or disintegration. The determination of the PMN-Elastase in stool is used to record inflammatory reactions where neutrophil granulocytes are involved. Especially in Crohn's disease the inflammatory processes go hand in hand with an increased phagocytic activity and the biological decay of these cells and thus leads to an increased release of PMN-Elastase and other lysosomal enzymes.

**Indications**

- Diagnosis of Crohn's disease
- Chronic arthropathy
- Pancreatitis
- Bacterial infection, sepsis

**Reference Values PMN-Elastase**

<table>
<thead>
<tr>
<th>Matrix</th>
<th>Stool</th>
<th>62 ng/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Serum</td>
<td>19 - 78 ng/ml</td>
</tr>
</tbody>
</table>

**PMN-Elastase (ELISA)**

<table>
<thead>
<tr>
<th>Sample volume</th>
<th>100 µl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matrix</td>
<td>Stool, Serum, Plasma, Seminal plasma, Synovia</td>
</tr>
<tr>
<td>Detection limit</td>
<td>0.12 ng/ml</td>
</tr>
<tr>
<td>Calibrators</td>
<td>0.12 - 3.3 ng/ml</td>
</tr>
<tr>
<td>Incubation time</td>
<td>3x1 h; 10-20 min</td>
</tr>
<tr>
<td>Tests</td>
<td>96</td>
</tr>
<tr>
<td>Cat. No.</td>
<td>K 6840</td>
</tr>
</tbody>
</table>

**Short incubation time (3 h)**

**Designed to be suitable for processing small amounts of specimen also**

**References**

Langhorst J et al. (2007) Z Gastroenterol 45: P261
Langhorst J et al. (2005) Inflamm Bowel Dis 11(12):1085-91
Heinichen et al. (1995) Clin Lab 41:539-545
Oremek et al. (1995) MTA 10:273-278
### Secretory IgA (sIgA)

Secretory IgA consists of two IgA monomers, which are connected to each other by a J-chain and contain a secretory component. They are produced in plasma cells located in the lamina propria of the mucous membranes and are found in body secretions, such as saliva, tears, nasal mucus, tracheobronchial mucus, gastrointestinal secretions, breast milk and colostrum.

The formation of secretory IgA occurs independently of the serum IgA synthesis. Therefore, a lack of serum IgA does not necessarily mean a lack of secretory IgA. Neonates and infants are supplied with sIgA through breast milk and are therefore passively immunized against gastrointestinal infections.

Conclusions concerning the endogenic defence of the intestinal mucosa can be drawn from the concentration of the sIgA in stool. A deficiency of sIgA points to a diminished activity of the mucosa immune system, whereas increased sIgA values indicate increased activity and a local inflammation of the intestinal mucosa.

### Indications

- Proof of an imbalanced immunological barrier of the intestinal mucosa
- Autoimmune diseases

### Reference Values sIgA (Age > 16 years)

| Matrix   | Stool 510 - 2040 µg/ml | Saliva 102 - 471 µg/ml |

### Also available as 1-point calibration test

**sIgA (ELISA)**

- **Sample volume**: 100 µl
- **Matrix**: Stool (100 mg), body liquids
- **Detection limit**: 13.4 ng/ml
- **Calibrators**: 22.2 – 600 ng/ml
- **Incubation time**: 1 h; 1 h; 10-20 min
- **Test principle**: ELISA
- **Tests**: 96
- **Cat. No.**: K 8870

### For Research only:

**sIgA-1 (ELISA)**

- **Sample volume**: 100 mg
- **Matrix**: Stool
- **Calibrators**: 3,3-100 U
- **Incubation time**: 1 h; 1 h; 15 min
- **Tests**: 96
- **Cat. No.**: K 6863

**sIgA-2 (ELISA)**

- **Sample volume**: 100 mg
- **Matrix**: Stool
- **Calibrators**: 3,3-100 U
- **Incubation time**: 1 h; 1 h; 15 min
- **Tests**: 96
- **Cat. No.**: K 6864

### References

TNFα

Tumour Necrosis Factor alpha (TNFα) belongs to the pro-inflammatory cytokines that encourage and uphold infection reactions. Cytokines, produced by macrophages and t-cells, play a central role in both acute and chronic infections.

The TNFα concentration is raised in the affected joints in many rheumatic diseases (rheumatoid arthritis, chronic poly-arthritis, ankylosing spondylitis i.e. M. Bechterew disease) and plays a significant role in the joint destruction and in the courses of other diseases. Even in Crohn's disease, an overproduction of TNFα can be observed that obviously affects the course of the disease.

In the 1990’s, pharmaceutical companies developed bio-technologically produced medications that aimed at hindering TNFα (“TNFα blockers”) and therewith produced a positive effect on the various disease symptoms (Feldmann et al. 2001).

<table>
<thead>
<tr>
<th>TNFα (ELISA)</th>
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</thead>
<tbody>
<tr>
<td>Sample volume</td>
</tr>
<tr>
<td>Matrix</td>
</tr>
<tr>
<td>Calibrators</td>
</tr>
<tr>
<td>Incubation</td>
</tr>
<tr>
<td>Tests</td>
</tr>
<tr>
<td>Cat. No.</td>
</tr>
</tbody>
</table>

References
**TNFα-Blocker Therapy Monitoring**

1. **Drug-level determination**

Tumor Necrosis Factor alpha (TNFα) belongs to the pro-inflammatory cytokines that establish and sustain inflammation reactions. Cytokines, produced by macrophages and T-cells, play a central role in both acute and chronic inflammations.

The TNFα concentration is elevated in the affected joints in many rheumatic diseases (rheumatoid arthritis, chronic poly-arthritis, ankylosing spondylitis i.e. M. Bechterew disease, psoriasis) and plays a significant role in joint destruction as well as in other manifestations of the diseases. Even in Crohn's disease, an overproduction of TNFα has been observed, obviously affecting the activity of the disease.

In the 1990's, pharmaceutical companies developed biotechnologically produced drugs that are aimed to neutralize TNFα ("TNFα blockers") and expected to have a positive effect on the various symptoms of the diseases.

In 1998, the first TNFα blockers were approved for use in the therapy of rheumatoid arthritis. Since then other TNFα blockers have been marketed:

- Infliximab (Remicade®),
- Adalimumab (Humira®).

The two TNFα blockers are approved for treatment of rheumatoid arthritis as well as ankylosing spondylitis and psoriasis. Although the TNFα blockers differ in respect to their chemical structures and mechanisms of action, the pharmacological effects are the same for both substances. They are comparable in their effectiveness in facilitating improvement in clinical symptoms of rheumatoid arthritis, but in Crohn's disease, Infliximab has proved to be the most effective one.

The therapeutic effect of the anti TNFα antibodies on chronic inflammations, i.e. Crohn's disease or rheumatoid arthritis, depends on the serum concentration of the corresponding pharmaceutical. In many patients, the treatment is of limited value, because of a fast degradation of the pharmaceutical or generation of antibodies against it. For a better control of the therapy, drug level monitoring is necessary.

Our ELISA test can be used for monitoring anti-TNFα-antibodies (e.g. Remicade®, Humira®) and provides a basis for possible preventive strategies.

**Indication:**

- Continuous TNFα Blocker therapy monitoring

---

**With our ELISA test, anti-TNFα-therapeutic antibodies can be detected**

**As an orientation:**

**To ensure clinical effectiveness of the TNFα inhibitor, the serum level should be at/above 12 mg/ml in week 4 and at/above 5 mg/ml in week 8.**

<table>
<thead>
<tr>
<th>TNFα-Blocker-Monitoring (drug-level, e.g. Remicade®)</th>
<th>TNFα-Blocker-Monitoring (drug-level, e.g. Humira®)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
<td>Sample volume</td>
</tr>
<tr>
<td>100 µl</td>
<td>100 µl</td>
</tr>
<tr>
<td>Matrix</td>
<td>Matrix</td>
</tr>
<tr>
<td>Serum</td>
<td>Serum</td>
</tr>
<tr>
<td>Incubation</td>
<td>Incubation</td>
</tr>
<tr>
<td>1h, 1h, 10-20min</td>
<td>4h, 1h, 10-20min</td>
</tr>
<tr>
<td>Test principle</td>
<td>Test principle</td>
</tr>
<tr>
<td>ELISA</td>
<td>ELISA</td>
</tr>
<tr>
<td>Tests</td>
<td>Tests</td>
</tr>
<tr>
<td>96</td>
<td>96</td>
</tr>
<tr>
<td>Cat. No.</td>
<td>Cat. No.</td>
</tr>
<tr>
<td>K 9655</td>
<td>K 9657</td>
</tr>
</tbody>
</table>
2. ADA (anti-drug-antibodies) determination

Problems in the therapy with TNFα antibodies occur when the human organism develops antibodies against the therapeutical antibody i.e. against the TNFα-blocker (among others). In a cohort study 61% of M. Crohn patients treated with Infliximab reacted with the development of antibodies against Infliximab (Baert et al. 2003). The presence of antibodies in 7-19% of patients could be associated with infusion reactions. Allergic reactions, and also therapy failure, could be attributed to the development of such antibodies. Through the parallel administration of immunosuppressants (mostly Methotrexat) a reduction of antibody development could, at least, be achieved (Colombel et al. 2004).

Our ELISAs enable the detection of human antibodies against:
- Adalimumab (fully humanistic antibody to TNFα, e.g. Humira®)
- Infliximab (chimeric, monoclonal antibody to TNFα, e.g. Remicade®)
- Etanercept (genetically produced variant of TNF receptors, e.g. Enbrel®)

Indications:
- Therapy monitoring for
  - therapy with antibodies against TNFα or
  - therapy with TNFα-receptors

### TNFα-Blocker-ADA
(anti-drug-antibodies, e.g. Remicade®)

- **Sample volume**: 5 μl
- **Matrix**: Serum
- **Incubation**: o.N., 1h, 5-10min
- **Test principle**: ELISA
- **Tests**: 96
- **Cat. No.**: K 9650

### TNFα-Blocker-ADA
(anti-drug-antibodies, e.g. Humira®)

- **Sample volume**: 5 μl
- **Matrix**: Serum
- **Incubation**: o.N., 1h, 5-10min
- **Test principle**: ELISA
- **Tests**: 96
- **Cat. No.**: K 9652

### TNFα-Blocker-ADA
(anti-drug-antibodies, e.g. Enbrel®)

- **Sample volume**: 5 μl
- **Matrix**: Serum
- **Incubation**: o.N., 1h, 5-10min
- **Test principle**: ELISA
- **Tests**: 96
- **Cat. No.**: K 9653

References
Seow et al. (2009) Gut, publ. online, doi:10.1136/gut.2009.183095
Bendtz et al. (2009) Scand J Gastroenterol 44:774-781
Ainsworth et al. (2008) Am J Gastroenterol 103:944-948
Bender N et al. (2006) Rheumatol Int 2006 Sep 28 [Epub ahead of print]
Zonulin

Zonulin is a novel human protein analogue to the Vibrio cholerae derived Zonula occludens toxin, which participates in tight junctions between cells of the wall of the digestive tract. Zonulin binds to a specific receptor on the surface of intestinal epithelia and triggers a cascade of biochemical events which induces tight junction disassembly and a subsequent permeability increase of the intestinal epithelia, allowing some substances to pass through and activate immune reactions.

A. Fasano and his co-workers found out that the zonulin-zonulin-receptor-system is more activated in celiac disease and type 1 diabetes mellitus patients. Patients with active celiac disease showed higher levels of zonulin and anti-zonulin antibodies compared to non-celiac patients and patients in remission, who were eating a gluten-free diet.

In addition, it was reported that many people who suffer from celiac disease also suffer from other autoimmune disorders. It is suggested that increased levels of zonulin are a contributing factor to the development of celiac disease and other autoimmune disorders such as insulin dependent diabetes, multiple sclerosis, and rheumatoid arthritis.

References
De Magistris MT (2006) 24 Suppl 2: S2-60-1
Hilbig H et al. (2008) Poster presented at the CIMT Meeting, 15.-16.05.2008, Mainz, Germany
3. **Exocrine pancreatic function**

*The pancreas is an important exocrine gland of the digestive system. It secretes enzymes and their precursors for the cleavage of proteins, sugars, nucleic and fatty acids. This enzymatic "cocktail" enables the decomposition of food in the small intestine.*

*Next to this basic exocrine function, the pancreas acts as an endocrine gland by releasing hormones that regulate the blood sugar level into the circulation, e.g. insulin and glucagon.*

*Immundiagnostik offers assays for the analysis of the exocrine pancreatic functions which can be used for diagnosis and therapy monitoring of diseases, such as exocrine pancreas insufficiency or chronic pancreatitis.*
Chymotrypsin

Chymotrypsin is a serine protease, which is secreted as an excretory enzyme from the pancreas after food intake into the duodenum. Here food proteins are hydrolytically cleaved, preferentially next to aromatic residues. A small part of the active form of the enzyme is excreted in the stool.

In pancreas insufficiency secondary to a chronic pancreatitis, the secretion of the enzyme is reduced markedly. In the past an exocrine pancreatic insufficiency within the scope of a chronic pancreatitis was diagnosed e.g. by determining faecal elastase or chymotrypsin activity in stool. The colorimetric determination of chymotrypsin activity, however, is less reliable because the enzyme’s activity is influenced by enzyme inhibitors in stool. Our ELISA allows the determination of the chymotrypsin concentration in stool.

Besides the determination of the chymotrypsin concentration, we offer a photometric test for the determination of chymotrypsin activity in stool.

**Indications**

- Chronic pancreatitis
- Exocrine pancreas insufficiency

**References**

Goldberg et al. (1971) Comp Biochem Physiol 83B: 697
Pancreatic Amylase

Like lipase and elastase, pancreatic amylase is synthesised in the pancreas. In the case of chronic pancreatitis, pancreatic amylase concentration is decreased. Some forms of disease (alcoholism, accidental trauma, fibrosis), are associated with a pancreatic insufficiency. In the routine laboratory pancreatic amylase has proved itself to be a genuine alternative to elastase-I in stool diagnosis with apparently higher clinical specificity and sensitivity.

Indications

- Chronic pancreatitis

<table>
<thead>
<tr>
<th>Reference Values Pancreatic Amylase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum</td>
</tr>
<tr>
<td>Stool</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pancreatic Amylase (ELISA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
</tr>
<tr>
<td>Matrix</td>
</tr>
<tr>
<td>Calibrators</td>
</tr>
<tr>
<td>Incubation time</td>
</tr>
<tr>
<td>Tests</td>
</tr>
<tr>
<td>Cat. No.</td>
</tr>
</tbody>
</table>

References
Pancreatic Lipase

The pancreatic lipase (triacylglycerol acylhydrolase) is a specific pancreatic enzyme which hydrolyzes primarily glycerol ester of long chain fatty acid. The activity of this enzyme is elevated in patients with acute pancreatitis. The determination of pancreatic lipase is therefore an important parameter for the detection and differentiation of pancreatic diseases, esp. in combination with the determination of the pancreatic amylase.

Conventional turbidimetric methods to assess lipase activity are complex, non-specific and exhibit a low sensitivity. In contrast, our ELISA detects human pancreatic lipase in serum quantitatively and with high specificity.

<table>
<thead>
<tr>
<th>Pancreatic Lipase (ELISA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
</tr>
<tr>
<td>Matrix</td>
</tr>
<tr>
<td>Calibrators</td>
</tr>
<tr>
<td>Incubation time</td>
</tr>
<tr>
<td>Tests</td>
</tr>
<tr>
<td>Cat. No.</td>
</tr>
</tbody>
</table>

References
Lott JA et al. (1986) Clin Chem 32:1290
Lott JA et al. (1986) Clin Enzymol: 245
4. **Food intolerances**

Food intolerances include enzyme defects (e.g. lactose or histamine intolerance) as well as immunological intolerances (e.g. gluten intolerance).

Often, these intolerances are caused by gene defects and are therefore congenital. In addition, they can be caused by diseases or diet.

Depending on the kind of food intolerance, different methods are used for diagnosis. Next to provocation tests and bowel biopsies, a number of laboratory parameters in urine, serum/plasma or stool along with DNA-analysis tools enable a reliable detection of food intolerances.

Immundiagnostik offers specific assays for the detection and differentiation of various intolerances.

A special focus of Immundiagnostik is the basic understanding of gluten intolerance and the supply of comprehensive laboratory analysis tools for the detection of even atypical and silent forms.
EDN (Eosinophil-derived Neurotoxin)

EDN (eosinophil derived neurotoxin, eosinophil protein x, EPX) measuring in stool is recommended for diagnosing a food allergy with an immediate reaction or to test the clinical efficiency of an elimination diet. EDN measurements also support an examination of the integrity of the intestinal mucous, when looking into an inflammmable intestinal disease, investigating Colon Carcinoma or for the diagnosis of an intestinal parasite.

The classic ways of diagnosing an allergy (determine allergy specific IgE antibodies and the prick-test) are only limitedly reliable when used to determine a food allergy. For example a normal IgE level and a negative result from the prick-test doesn't rule out an intestinal food allergy. In this case, an EDN measurement in stool is recommended.

EDN, a cationic glycoprotein, which is released by activated eosinophiles, has strong cytotoxic characteristics and plays a large part in virus prevention. It is released by the eosinophile granules in places where eosinophiles are mainly to be found, in the skin, lungs, urogenital and gastrointestinal tracts, that is, in the organs which act as an entry point for pathogen. The accumulation of EDN in the intestine is associated with tissue damage.

Measuring EDN in stool can serve as an objective parameter for a current clinical or sub-clinical chronic inflammation which is noticeable in the gastrointestinal area. With Colitis ulcerosa and Crohn's disease, the EDN measurement helps the evaluation of a disease's activity and the prediction of it possible.

**Indications**

- Proof of a food allergy with immediate reaction
- Assessment of an elimination diet
- Proof of damaged integrity of the intestinal mucous membrane caused by an invasive disease (e.g. CED, CC etc.)
- Proof of intestinal parasites

<table>
<thead>
<tr>
<th>EDN (ELISA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
</tr>
<tr>
<td>Matrix</td>
</tr>
<tr>
<td>Detection limit</td>
</tr>
<tr>
<td>Standards</td>
</tr>
<tr>
<td>Incubation time</td>
</tr>
<tr>
<td>Tests</td>
</tr>
<tr>
<td>Cat. No.</td>
</tr>
</tbody>
</table>

References
Bischoff SC et al. (1997) Dig Dis Sci 42(2) 394-403
Petersen CGB et al. (2002) Am J Gastroenterol 97(7) 1755-1762
Gluten sensitivity

Gliadin / Transglutaminase Antibodies

Celiac disease/ gluten intolerance is a chronic gastrointestinal disease with a prevalence of about 1:250 in Europe.

The definition of gluten intolerance has to be revised due to new data regarding its prevalence. The classic diagnosis of fully developed celiac disease by biopsy-proven villous atrophy only represents the tip of the iceberg.

The iceberg model of Logan clearly indicates that the majority of patients with gluten intolerance do not present with fully developed celiac disease. The early diagnosis and the subsequent initiation of a gluten-free diet is instrumental in preventing a total atrophy of the intestinal mucous layer.

In patients presenting with long-term, unspecific abdominal complaints secondary to gluten intolerance, the quality of life can be improved considerably after an early diagnosis and a timely initiation of a gluten-free diet. Gluten intolerance is, next to its classic symptoms, associated with other autoimmune diseases (rheumatoid arthritis, dermatitis herpetiformis Duhring or diabetes mellitus) or with a risk of miscarriage.

We offer a unique product line for the purpose of evaluating unspecific abdominal complaints, especially when gluten intolerance is suspected. Beside the assays for the determination of anti-transglutaminase IgA antibodies from serum, we offer tests for the determination of anti-transglutaminase sigA/IgA from faeces. The tissue transglutaminase and also the epidermal transglutaminase have been proven to be associated with gluten intolerance. The epidermal transglutaminase especially is associated with extraintestinal manifestations like Dermatitis herpetiformis Duhring.

Gluten intolerance is caused by gliadins or analogous proteins in cereals. It is well known that this (auto-)immunological disease strikes individuals with a special genetic profile: there is a strong association with specific risk alleles encoding the heterodimeric HLA-DQ2 molecule (>90 % of the patients) or respectively the HLA-DQ8 molecule.

The MutaGEL HLA-DQ 2+8 test allows the individual HLA-DQ genotyping by amplification of the encoding alleles (for 24 determinations). For this purpose, only a small sample of EDTA-blood is necessary for the preliminary extraction of DNA (reagents not included). The subsequent processing with molecular biological methods leads to specific amplification products detectable by gel-electrophoresis.

Indications

- Poor appetite and failure to gain weight
- Iron-deficiency anaemia
- Neurological disturbances (e.g., depression, lethargy)
- Infertility
- Recurrent abortions (Untreated pregnant women are at risk of miscarriage and at risk of having a baby with a congenital malformation)
- Dermatitis herpetiformis Duhring
- Rheumatoid arthritis symptoms

<table>
<thead>
<tr>
<th>Reference Values Anti-Transglutaminase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum</td>
</tr>
<tr>
<td>≥ 7 AU positive</td>
</tr>
<tr>
<td>Stool</td>
</tr>
<tr>
<td>&gt; 100 U/I positives</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reference Values Anti-Gliadin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum</td>
</tr>
<tr>
<td>≥ 18 AU positive</td>
</tr>
<tr>
<td>Stool</td>
</tr>
<tr>
<td>&gt; 100 U/I positives</td>
</tr>
</tbody>
</table>
4. Food intolerances (Gluten sensitivity)

*Anti-gliadin-assay (particularly the IgG subclass) can yield false-positive results in gastrointestinal conditions other than CD, including cow’s milk protein intolerance and parasite infections (Fasano and Catassi, 2001)

<table>
<thead>
<tr>
<th>Affected organ</th>
<th>Serum diagnostics</th>
<th>Stool diagnostics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal tract</td>
<td>Anti-tTG-IgA</td>
<td>Anti-Gliadin-slgA* Anti-tTG-slgA</td>
</tr>
<tr>
<td>Joints</td>
<td>Anti-tTG-IgA</td>
<td>Anti-tTG-slgA</td>
</tr>
<tr>
<td>Skin</td>
<td>Anti-TG_e-IgA</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Product</th>
<th>Sample volume</th>
<th>Matrix</th>
<th>Calibrators</th>
<th>Incubation time</th>
<th>Test principle</th>
<th>Tests</th>
<th>Cat.No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-human epidermal Transglutaminase IgA [anti-TG_e-IgA]</td>
<td>100 µl</td>
<td>Serum, Plasma</td>
<td>Cut off</td>
<td>1h; 1h; 30min</td>
<td>ELISA</td>
<td>96</td>
<td>K 9396</td>
</tr>
<tr>
<td>Anti-human tissue Transglutaminase IgA [anti-tTG-IgA]</td>
<td>100 µl</td>
<td>Serum, Plasma</td>
<td>Cut off</td>
<td>1h; 1h; 30min</td>
<td>ELISA</td>
<td>96</td>
<td>K 9399</td>
</tr>
<tr>
<td>Anti-human tissue Transglutaminase IgG [anti-tTG-IgG]</td>
<td>50 µl</td>
<td>Serum, Plasma</td>
<td>Cut off</td>
<td>2h</td>
<td>ELISA</td>
<td>96</td>
<td>K 9398</td>
</tr>
<tr>
<td>Anti-human tissue Transglutaminase slgA [anti-htTG-slgA]</td>
<td>100 mg</td>
<td>Stool</td>
<td>Cut off</td>
<td>2h; 1h; 15-25min</td>
<td>ELISA</td>
<td>96</td>
<td>K 9393</td>
</tr>
<tr>
<td>anti-Gliadin IgA</td>
<td>100 µl</td>
<td>Serum</td>
<td>Cut off</td>
<td>1h; 1h; 15min</td>
<td>ELISA</td>
<td>96</td>
<td>K 9310</td>
</tr>
<tr>
<td>anti-Gliadin IgG</td>
<td>50 µl</td>
<td>Serum, Plasma</td>
<td>Cut off</td>
<td>2h</td>
<td>ELISA</td>
<td>96</td>
<td>K 9300</td>
</tr>
<tr>
<td>anti-Gliadin slgA</td>
<td>100 mg</td>
<td>Stool</td>
<td>Cut off</td>
<td>2h; 1h; 15-25min</td>
<td>ELISA</td>
<td>96</td>
<td>K 9311</td>
</tr>
<tr>
<td>MutaGEL® HLA-DQ 2+8</td>
<td>200 µl</td>
<td>DNA (e.g. whole blood, cheek swab)</td>
<td>Cut off</td>
<td>2h</td>
<td>PCR (alle specific)</td>
<td>24</td>
<td>KE09020</td>
</tr>
</tbody>
</table>

References
Schütz et al. (1998) Glut enunverträglichkeit EHK 11:807-810
Stern et al. (1998) Der Kinderarzt 2:159164
Gliadorphin (Gliadomorphin)

Gliadorphin is a 7 amino acids peptide which is formed during digestion of the gliadin component of the gluten protein. Gluten-derived peptides bind to opioid receptors in the brain and exhibit morphine-like effects, for example like heroin. These compounds have been shown to react with areas of the brain which are involved in speech and auditory integration. Urine samples from people with autism, schizophrenia, and celiac disease contain high amounts of gliadorphin. It is suspected that this peptide may also be elevated in other disorders such as chronic fatigue, fibromyalgia, and depression. Symptom remission has been observed after exclusion of wheat and dairy products from the diet.

Indications

- Autism
- Schizophrenia
- Celiac disease

<table>
<thead>
<tr>
<th>Gliadorphin (Gliadomorphin) (ELISA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
</tr>
<tr>
<td>Matrix</td>
</tr>
<tr>
<td>Standards</td>
</tr>
<tr>
<td>Incubation time</td>
</tr>
<tr>
<td>Tests</td>
</tr>
<tr>
<td>Cat.No.</td>
</tr>
</tbody>
</table>
Lactose intolerance

MutaGEL® Laktase

Patients with lactose intolerance are not able to digest milk sugar (lactose) taken in with food. Due to this fact, these persons subsequently suffer under malabsorption problems like nausea, flatulence, diarrhoea or stomach pain.

The most important reason for lactose intolerance is founded in a genetical lack of the enzyme lactase which is responsible for the degradation of milk sugar in the organism. This common gene defect is very easy to detect by analysing the T/C base replacement at position -13910 from the regulatory region of the lactase gene. If this point mutation is homozygous, a lactase deficiency and subsequent lactose intolerance is predetermined. The manifestation of the disease occurs with about 20 years of age and the prevalence of the homozygous mutation in Germany is more than 15%.

The kit “MutaGEL® Laktase” allows the detection of the common T13910C polymorphism in the lactase gene LCT.

Indications

- Nausea, cramps, bloating, gas, and diarrhea, which begin about 30 minutes to 2 hours after drinking or eating fluids, foods containing lactose

<table>
<thead>
<tr>
<th>MutaGEL® Laktase (AS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
</tr>
<tr>
<td>Matrix</td>
</tr>
<tr>
<td>Test principle</td>
</tr>
<tr>
<td>Tests</td>
</tr>
<tr>
<td>Cat. No.</td>
</tr>
</tbody>
</table>

References
**Fructose intolerance**

**MutaGEL® Aldolase B**

The liver isoenzyme Aldolase B is critical for sugar metabolism, and a catalytic deficiency due to mutations in its gene may result in hereditary fructose intolerance (HFI) syndrome, with hypoglycaemia and severe abdominal symptoms.

The autosomal recessive disorder HFI is a potentially lethal inborn error in metabolism and the disease poses diagnostic problems because of in part very unspecific clinical manifestations. The present Aldolase B PCR test is useful for the detection of the three most common mutations critical for gluconeogenesis and fructose metabolism: A149P (60%), A174D (11%) and N334K (8%). These mutations may account together for more than 80% of all known mutations causing HFI and their screening will be helpful for suited therapy of afflicted patients.

<table>
<thead>
<tr>
<th>MutaGEL® Aldolase B</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
<td>200 µl</td>
</tr>
<tr>
<td>Matrix</td>
<td>DNA (e.g. whole blood, cheek swab)</td>
</tr>
<tr>
<td>Test principle</td>
<td>PCR (allel specific)</td>
</tr>
<tr>
<td>Tests</td>
<td>24</td>
</tr>
<tr>
<td>Cat. No.</td>
<td>KE 09013</td>
</tr>
</tbody>
</table>
Histamin intolerance

Diamine Oxidase (DAO)

Diamine oxidase (DAO) is a histamine-metabolizing enzyme. Although DAO is found practically in the whole body, the most important site of its action is the intestine. The enzymatic activity of DAO determines the histamine degradation speed. In the case of DAO deficiency or inhibition, incorporated or endogenous histamine cannot be degraded quickly enough, and the symptoms of histamine intolerance are presented. Millions of people suffer from gastrointestinal problems, migraine, irritations of nasal mucosa and other allergy-like symptoms after consumption of certain nutrients. Too much histamine in the body can be the reason for this wide range of symptoms. Another possibility for reduced DAO function could be the intake of activity-inhibiting substances, such as alcohol or medication.

Histamine induced food intolerance is not IgE-mediated. Determination of the DAO activity in serum or plasma is a suitable marker for diagnosis of histamine intolerance and the associated symptoms.

With our easy-to-use, reliable and standardised test kit it is possible to quantify the biological activity of DAO in the circulation. Only 50 µl of serum are needed for the test, results are available within 3 hours.

Indications:
- Detection of histamine intolerance
- Monitoring of a histamine-free diet

DAO \(^4\)H (REA)

<table>
<thead>
<tr>
<th>Sample volume</th>
<th>100 µl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matrix</td>
<td>Serum</td>
</tr>
<tr>
<td>Calibrators</td>
<td>2.1 - 80 U/ml</td>
</tr>
<tr>
<td>Incubation time</td>
<td>2,5 h</td>
</tr>
<tr>
<td>Tests</td>
<td>96</td>
</tr>
<tr>
<td>Cat. No.</td>
<td>K 8220</td>
</tr>
</tbody>
</table>

DAO (ELISA)

<table>
<thead>
<tr>
<th>Sample volume</th>
<th>25 µl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matrix</td>
<td>Serum, Plasma</td>
</tr>
<tr>
<td>Incubation time</td>
<td>2 h, 1 h, 1 h, 10-20 min</td>
</tr>
<tr>
<td>Tests</td>
<td>96</td>
</tr>
<tr>
<td>Cat. No.</td>
<td>K 8500</td>
</tr>
</tbody>
</table>

References
## Histamin

### Histamin* (ELISA)

<table>
<thead>
<tr>
<th>Sample volume</th>
<th>100 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Matrix</td>
<td>Stool</td>
</tr>
<tr>
<td>Calibrators</td>
<td>10–2560 ng/ml</td>
</tr>
<tr>
<td>Incubation time</td>
<td>15 min; 30 min; 10 min; 15 min</td>
</tr>
<tr>
<td>Tests</td>
<td>96</td>
</tr>
<tr>
<td>Cat. No.</td>
<td>K 6861</td>
</tr>
</tbody>
</table>

* For Research Use only

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**New for your research:**

Our ELISA for the quantitative determination of Histamin in Stool
5. **Infectious diseases**

*Infections of the intestinal tract with bacteria, viruses or parasites can cause life threatening acute diarrhea. In addition, frequent or chronic infections play a role in the pathogenesis of a number of intestinal diseases. For routine screening, Immundiagnostik offers a comprehensive, automatable assay portfolio (ELISA, PCR) for pathogen detection.*

*Microscopic picture of the yeast Candida albicans*
Helicobacter pylori Antigen

Helicobacter pylori (H. pylori) is a spiral-shaped bacterium that can be found in the human stomach and duodenum. In order to be able to survive in the extremely acid environment of the stomach, H. pylori bacteria produce urease which in turn metabolises urea into bicarbonate and ammonia. Particularly, the highly corrosive ammonia affects the gastric mucosa adversely and might cause severe damage. Besides a possible gastritis, an H. pylori infection could eventually lead to a duodenal ulcer or a gastric tumour, resulting from the persisting immune response to the infection.

Traditional diagnosis of an H. pylori infection requires invasive measures such as gastroscopy and biopsy, which for most patients are rather burdensome. Alternatively, measuring H. pylori antibodies in serum or the 13C-urease breath test provides information on a possible H. pylori infection.

Our Helicobacter pylori Antigen ELISA offers a tool for the detection of the H. pylori antigen in faeces. The advantages of a faecal antigen test are obvious:

- non-invasive sample collection
- cost efficient alternative to the golden standard of gastroscopy
- equivalent sensitivity and specificity to the 13C-urease breath test
- for follow-up of treatment more useful than serological test since a comparison of pro- and post-treatment is necessary

Indications

- Initial diagnosis of a H. pylori infection
- Follow-up of treatment

<table>
<thead>
<tr>
<th>Helicobacter pylori antigen (ELISA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
</tr>
<tr>
<td>Matrix</td>
</tr>
<tr>
<td>Calibrators</td>
</tr>
<tr>
<td>Incubation time</td>
</tr>
<tr>
<td>Tests</td>
</tr>
<tr>
<td>Cat. No.</td>
</tr>
</tbody>
</table>

References


Also available at Immundiagnostik for serum und plasma samples:

- Helicobacter pylori IgA ELISA (KBE-HLAK09)
- Helicobacter pylori IgA CLIA (KBC-HLAK09)
- Helicobacter pylori IgG ELISA (KBE-HLGG08)
- Helicobacter pylori IgG CLIA (KBC-HLGG08)
Norovirus

Gastrointestinal infections can cause life-threatening diseases ultimately leading to death. It was recently shown, that the genetic heterogeneous group of Noroviruses (formally known as Norwalk-like viruses) are the major cause of non-bacterial gastroenteritis worldwide. Human Noroviruses are small, non-enveloped viruses with a ssRNA (single stranded) genome.

Noroviruses belong to the family of Caliciviridae and are divided into genotype I and II. These viruses are resistant against higher temperatures (60°C), acid (pH 3) and chlorit (10mg/L). The viruses are transmitted via contaminated food and water but also from person-to-person and are highly contagious.

<table>
<thead>
<tr>
<th>Test Type</th>
<th>Sample Volume</th>
<th>Matrix</th>
<th>Test Principle</th>
<th>Tests</th>
<th>Cat. No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>MutaPLATE® Norovirus</td>
<td>200 µl</td>
<td>RNA (Stool)</td>
<td>real time RT-PCR (for open system)</td>
<td>96</td>
<td>KG1934196</td>
</tr>
<tr>
<td>MutaPLEX® Norovirus</td>
<td>200 µl</td>
<td>RNA (Stool)</td>
<td>real time RT-PCR (for open system) (+ extraction control)</td>
<td>32</td>
<td>KG190132</td>
</tr>
<tr>
<td>MutaREAL® Norovirus</td>
<td>200 µl</td>
<td>RNA (Stool)</td>
<td>real time RT-PCR</td>
<td>96</td>
<td>KG2934196</td>
</tr>
<tr>
<td>MutaREX® Norovirus</td>
<td>200 µl</td>
<td>RNA (Stool)</td>
<td>real time RT-PCR (+ extraction control)</td>
<td>96</td>
<td>KG290196</td>
</tr>
</tbody>
</table>

Literature
Enterovirus

Human Enteroviruses are small RNA viruses which belong to the family of picornaviridae. The ubiquitous pathogen is responsible for about 500 million infections per year worldwide. The 64 serotypes are divided in the following groups:

* Polioviruses Typ 1-3
* Coxsackieviruses with subgroups A and B
* Echoviruses
* Enteroviruses 68-71

In moderate climates there is a seasonal accumulation of enterovirus infections in late summer and fall. Transmission of enteroviruses occur via the faecal-oral route. Airborne infections are also common. The viruses are transmitted between humans through stool or through saliva-contaminated objects and are excreted during the acute disease phase. In addition, virus particles can be detected up to several weeks after the symptoms have subsided. The median incubation time for most enterovirus infections is 3-5 days.

Diseases caused by enteroviruses are very complex and can be life threatening, especially for children. Among them are febrile illness, conjunctivitis, herpangina, hand foot and mouth disease, gastroenteritis, generalized skin rashes, meningitis, pneumonia, myocarditis, pericarditis, hepatitis, encephalitis, paralyses, fetal damage up to severe neonatal diseases with pneumonia, myocarditis and meningoencephalitis.

Enteroviruses are excreted mainly in stool and, depending on the virus type, through the pharyngeal route. In acute disease, the diagnosis should preferably be based on PCR pathogen detection.

Our PCR kits are screening tests for the qualitative determination of human enterovirus RNA (Polio-, Coxsackie A-, Coxsackie B- and echoviruses) in clinical samples (stool, whole blood, plasma, respirational tract samples, cerebrospinal fluid).

Highest quality standard of our PCR kits: 2010 evaluated by the international panel "Quality Control for Molecular Diagnostics" with best result (100%)

The CE-marked kits are optimized for enterovirus detection in stool

### MutaREAL® Enterovirus
- Sample volume: 200 µl
- Matrix: RNA (biol. liquids)
- Test principle: real time RT-PCR
- Tests: 96
- Cat. No.: KG2900296

### MutaPLEX® Enterovirus
- Sample volume: 200 µl
- Matrix: RNA (biol. liquids)
- Test principle: real time RT-PCR for open systems + extraction control
- Tests: 96
- Cat. No.: KG190296

### MutaPLATE® Enterovirus
- Sample volume: 200 µl
- Matrix: RNA (biol. liquids)
- Test principle: real time RT-PCR for open systems
- Tests: 96
- Cat. No.: KG1900296

### MutaREX® Enterovirus
- Sample volume: 200 µl
- Matrix: RNA (biol. liquids)
- Test principle: real time RT-PCR + extraction control
- Tests: 96
- Cat. No.: KG290296
Salmonella sp.

Salmonella are the main cause for food intoxications: In more than 65% of all cases, egg products, meat and poultry are the common foods which transmit salmonella. Therefore, the European regulations specify that none of the roughly 2000 known salmonella subtypes must be detected in a 25 g food sample. Until now, the necessary analysis has been performed with bacteriological cultures which take up to 5 days.

The PCR offers a far more sensitive and time saving alternative (one day) to the conventional microbiological analysis of Salmonella sp. in various food samples. In addition, the PCR allows the pathogen detection in stool samples and is therefore ideal for a clinical setting.

<table>
<thead>
<tr>
<th>MutaREAL® Salmonella</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample volume</td>
</tr>
<tr>
<td>Matrix</td>
</tr>
<tr>
<td>Test principle</td>
</tr>
<tr>
<td>Tests</td>
</tr>
<tr>
<td>Cat. No.</td>
</tr>
</tbody>
</table>
D-Arabinitol

The yeast *Candida albicans* is present in most human mucous membranes of the mouth and pharynx, the genital area and the intestinal tract. If the immune system is weakened or the physiological bacterial intestinal flora disrupted, the yeast multiplies and leads to a generalized candidiasis. Typical symptoms of a candidiasis include general weakness, recurring vaginal infections, fungal skin infections, fatigue, memory impairments, irritability, headaches, poor concentration, joint aches and flatulence.

A systemic candidiasis is often diagnosed late. As a consequence of the multiplication of the yeast in the circulation, multiple organs can be affected. Complications include endocarditis, endophthalmitis or osteomyelitis. During a systemic candidiasis, pathogen detection occurs in blood, liquor, joint fluid and endotracheal samples and bronchial lavages.

An additional possibility to diagnose invasive yeast infections is the determination of D-Arabinitol in serum or urine. D-Arabinitol is a characteristic yeast metabolite. While *Candida ssp.* produce exclusively D-Arabinitol, the human metabolism generates L-Arabinitol. D-Arabinitol serum levels rise during an invasive candidiasis, when the yeast multiplies in the organism.

Apart from the gas chromatographic determination easy-to-use tests have been missing so far. Our Arabinitol test is a straightforward colorimetric enzyme assay for the manual and automatic analysis of D-Arabinitol in serum or urine.

**Indications**

- Detection of a Candida infection
- Determination of the severity of a Candida infection
- Therapy monitoring
- Choice of the appropriate medication

<table>
<thead>
<tr>
<th>D-Arabinitol</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sample volume</strong></td>
</tr>
<tr>
<td><strong>Matrix</strong></td>
</tr>
<tr>
<td><strong>Test principle</strong></td>
</tr>
<tr>
<td><strong>Calibrators</strong></td>
</tr>
<tr>
<td><strong>Tests</strong></td>
</tr>
<tr>
<td><strong>Cat. No.</strong></td>
</tr>
</tbody>
</table>

**Colorimetric test:**

- Simple handling
- Automatable
More tests for the detection of viral and bacterial infections (determination in stool) on request:

<table>
<thead>
<tr>
<th>Test</th>
<th>Code</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Serazym Adenovirus</strong></td>
<td>HW/E-017</td>
<td>96 determinations</td>
</tr>
<tr>
<td>Kit for automate</td>
<td>HW/E-017-A2</td>
<td>2 x 96 determinations</td>
</tr>
<tr>
<td><strong>Serazym Astrovirus</strong></td>
<td>HW/E-045</td>
<td>96 determinations</td>
</tr>
<tr>
<td>Kit for automate</td>
<td>HW/E-045-A2</td>
<td>2 x 96 determinations</td>
</tr>
<tr>
<td><strong>Serazym Rotavirus</strong></td>
<td>HW/E-020</td>
<td>96 determinations</td>
</tr>
<tr>
<td>Kit for automate</td>
<td>HW/E-020-A2</td>
<td>2 x 96 determinations</td>
</tr>
<tr>
<td><strong>Serazym Giardia lamblia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kit for automate</td>
<td>HW/E-038</td>
<td>96 determinations</td>
</tr>
<tr>
<td><strong>Serazym Entamoeba histolytica</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kit for automate</td>
<td>HW/E-018</td>
<td>96 determinations</td>
</tr>
<tr>
<td><strong>Serazym Clostridium difficile</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Toxin A/B</strong></td>
<td>HW/E-040</td>
<td>96 determinations</td>
</tr>
<tr>
<td>Kit for automate</td>
<td>HW/E-040-A2</td>
<td>2 x 96 determinations</td>
</tr>
<tr>
<td><strong>Serazym Verotoxin 1+2</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kit for automate</td>
<td>HW/E-030</td>
<td>96 determinations</td>
</tr>
<tr>
<td>HW/E-030-A2</td>
<td>2 x 96 determinations</td>
<td></td>
</tr>
<tr>
<td><strong>Serazym Campylobacter</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kit for automate</td>
<td>HW/E-093</td>
<td>96 determinations</td>
</tr>
<tr>
<td>HW/E-093-A2</td>
<td>2 x 96 determinations</td>
<td></td>
</tr>
</tbody>
</table>
Appendix:
Stool sample preparation systems

We can spare you the cumbersome and unpleasant stool weighing procedure: Our special, cost-effective sample tube enables the preparation of a defined sample solution with minimal stool contact. For sample collection, the dip stick with screw cap is used to collect the sample (15 mg), excessive stool is stripped off at the cone-shaped insert (s. Fig.). The stool sample is now ready for storage or dilution with buffer. The resulting sample suspension can be used with our ELISAs for the determination of a respective parameter in stool.

The sample tubes can be ordered empty or filled with buffer for the preparation of a defined stool sample suspension.

*Fig.: Practical preparation of stool samples*

- time saving
- hygienic
- minimal stool contact
- suited for direct use on automates

<table>
<thead>
<tr>
<th>Stool sample preparation systems</th>
<th>Cat. No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>unfilled</td>
<td>K 6998SAS</td>
</tr>
<tr>
<td>with buffer (dilution 1:50)</td>
<td>...SASP1</td>
</tr>
<tr>
<td>with buffer (dilution 1:100)</td>
<td>...SASP2</td>
</tr>
</tbody>
</table>

... = Cat. Nr. of the respective ELISA test for the determination of a parameter in stool.
Please send me the following informative material:

- Product List
- Catalogue "Immunochemicals, Antibodies, Antigens"
- Manuals of these products:
  - Skeletal System
  - Oxidative Stress
  - Cardiovascular and Renal Systems
  - Orthomolecular Medicine
  - Molecular Biology
  - HPLC-Applications / LC-MS/MS-Applications

Name/Address:

Phone: ____________________________
Fax: ____________________________
Date: ____________________________
E-Mail: ____________________________