



Diagnostics

GastroPanel

Biochemical Investigation

RISK ASSESSMENT

Pepsinogen I EIA

Product Information

Pepsinogen I ELISA kit 96 wells Cat. No. 601010.01

Background

This test is intended to identify patients who have an advanced atrophic gastritis in the gastric corpus and who, correspondingly, are at increased risk for gastric cancer (1, 2). The serum or plasma PGI (S-PGI, P-PGI) assay is a reliable tool for detecting patients with advanced atrophic corpus gastritis (3-6); the sensitivity and specificity of the test are 92 % and 90 %, respectively.

Pepsinogen I (PGI) is a precursor enzyme of pepsin and is synthesized by the chief cells and neck cells of the gastric corpus (from so-called oxyntic glands of the gastric mucosa). The major part of PGI is secreted into the gastric lumen but a small amount can be found in the blood. The S/P-PGI level reliably correlates with the number of chief cells in the gastric corpus mucosa. Correspondingly, the loss of chief cells results in a linear decrease in S/P-PGI. The loss of chief cells is, on the other hand, a result of atrophic gastritis.

For unknown reasons, atrophic gastritis increases the risk of gastric cancer, the risk being even 5-fold in patients with advanced atrophic gastritis in the corpus and even 90-fold in advanced atrophic pangastritis (both antrum and corpus affected) compared to the cancer risk in persons with normal gastric mucosa (2).

The screening of middle-aged (50-69 years) , smoking men in Finland with the S-PGI test has revealed that a low S-PGI level (<25 µg/l) is detected in 9.8 % of men of whom 4.7 % revealed either a gastric cancer or precancerous lesion by endoscopy (7). Corresponding results have also been published in earlier studies (8-17).

Principle of the Assay

This PGI ELISA is based on a sandwich enzyme immunoassay technique with a PGI specific capture antibody adsorbed on a microplate and a detection antibody labeled with horseradish peroxidase (HRP).

Assay Highlights

- Blank solution, three calibrators, control and red colored dilution buffer
- Specific: no cross-reactivity with Pepsinogen II
- Sensitive: 1.2 m g/l detection limit
- Excellent intra and inter assay precision
- 2 hour total incubation time: fast turnaround of patient results
- Protocol is easily adapted for automated systems

Pepsinogen II ELISA

Product Information



Pepsinogen II EIA Kit 96 wells Cat. No. 601020.01

Background

Pepsinogen II is produced by chief cells and mucous neck cells of the gastric mucosa, in pyloric glands in the gastric antrum and Brunner's glands in the proximal duodenum. The ratio of concentration of Pepsinogen I (PGI) to PGII in serum or plasma of normal subjects is about 4:1 (1). The PGI/PGII ratio decreases linearly with increasing grade of atrophic gastritis in the corpus (2, 3). The ratio is < 2.5 when the atrophic gastritis is advanced (moderate or severe) in the gastric corpus (3). It has been shown that the risk of gastric cancer is increased (5-fold) when the PGI/PGII ratio is low (5-14). This test is intended to be used as an additional tool in the diagnosis of atrophic corpus gastritis, which is also a risk state for gastric cancer (2, 4). The Pepsinogen II assay is used together with the Pepsinogen I assay by which the PGI/PGII ratio is determined.

Principle of the Assay

This PGII ELISA is based on a sandwich enzyme immunoassay technique with a PGII specific capture antibody adsorbed on a microwell plate and detection antibody labeled with horseradish peroxidase (HRP).

Assay Highlights

- Blank solution, three calibrators, control and red colored dilution buffer
- Sensitive: < 1.0 µ g/l detection limit
- Excellent within- and between-assay precision
- Total incubation time 2.5 hours , enables fast turnaround of results
- Protocol is easily adapted for automated platforms

References

Gastrin-17 EIA

Product Information

Gastrin-17 ELISA Kit 96 wells Cat. No. 601030.01

Background

This test is intended to identify *Helicobacter pylori* infected patients who have an advanced atrophic gastritis in the gastric antrum – these patients have an abnormally low S/P-G-17 - and who, correspondingly, are at an increased risk for gastric cancer and for peptic ulcer disease [(1-5), for reviews, see Ref. (6,7)]. For unknown reasons, atrophic antrum gastritis increases the risk of gastric cancer, the risk being even 18-fold in patients with advanced atrophic antrum gastritis compared to the cancer risk in persons with a normal antrum mucosa (2). On the other hand, abnormally high S/P-G-17 concentrations can be used as a biomarker of hypo- or achlorhydria and can be seen as a sign of atrophic gastritis that is limited to the gastric corpus. Moreover, S/P-G-17 levels can be used in differentiation of hypergastrinemias of neoplastic origin from those of non-neoplastic origin - G-17 does not rise, in contrast to high molecular mass forms of gastrins, in patients with gastrinoma-tumors. The measurement of serum/plasma G-17 may also be used to monitor patients who have undergone successful gastric surgery - secretion of G-17 into circulation is practically zero after successful antrectomy.

The antral hormone gastrin (gastrin-17) regulates gastric acid secretion and growth of the gastric mucosa (8). As a result of the cellular post-translational maturation process of progastrin, the G cells in the antrum release a mixture of different acid stimulatory gastrins and other precursor fragments into the circulation (9). This mixture comprises gastrin-71, -52, -34, -17, -14, and -6, all of which are carboxyamidated and circulate in an O-sulfated and nonsulfated form. The dominant gastrin forms in serum/plasma from healthy humans are amidated gastrin-34 and -17 [for a review, see Ref. (10)], of



which the latter, G-17 is the predominant and potent tissue form in healthy antral mucosa and almost exclusively produced by the antrum G-cells.

The secretion of G-17 from the G-cells in the antrum mucosa is a result of a stimulation by various factors e.g. of dietary protein stimulus. The high acidity in the stomach starts to inhibit the secretion of G-17 (11). In a normal stomach, protein stimulation or the lack of acid will result in an increase in the S/P-G-17 level. In advanced or severe atrophic gastritis of the antrum the fasting (basal) level of G-17 in serum/plasma is low and no increase will occur in connection with stimulation. The magnitude of the decline of the G-17 concentration and its response to stimulus depends on the degree of atrophy: the more severe the atrophy, the lower the concentration and the weaker the increase in the G-17 levels. The G-17 ELISA method is specific for amidated gastrin-17 measurements in serum/plasma (12).

Principle of the Assay

This G-17 ELISA is based on a sandwich enzyme immunoassay technique with a G-17 specific first antibody adsorbed to a microplate, a G-17 specific second antibody binding to the G-17 in the sample, followed by biotinylated IgG, which binds to the second antibody. Avidin, labeled with horseradish peroxidase (HRP), is used for signal production and enhancement.

Assay Highlights

- Blank solution, three calibrators, control and red colored dilution buffer
- Specific: no cross-reactivity with gastrin-34, gastrin-14 or CCK
- Sensitive: 2.0 pmol/l detection limit enabling baseline measurements to be taken
- Excellent intra- and interassay precision
- 4.5 hours total incubation time: Allows for same day reporting
- Protocol is easily adapted for automated platforms

Helicobacter pylori IgG Antibodies ELISA

Product Information

Helicobacter pylori IgG ELISA Kit 96 wells Cat.No. 601040.01

Background

Helicobacter pylori infection is the most important cause of chronic gastritis. Another mechanism for gastritis and severe atrophic gastritis is the autoimmune mechanism, which can also be activated by an *H. pylori* infection (1, 2). This kit is intended to aid in the diagnosis of *H. pylori* infection.

Helicobacter pylori is a spiral shaped, gram-negative bacterium that colonizes in the human stomach. The organism is found in the mucous layer of the stomach overlying the gastric epithelium and it does not appear to invade tissue. However, the mucosa underneath the area of the *H. pylori* colonization is invariably inflamed; this condition is referred to as chronic superficial or non-atrophic gastritis, which, if untreated persists for life (1). Nevertheless, the chronic inflammatory process can lead to atrophic gastritis, which has been linked with peptic ulceration and gastric cancer, two of the most important diseases of the upper gastrointestinal tract (3-6). The presence of antibodies to GagA *H. pylori* strains have been linked with the development of atrophic gastritis in corpus (7).

The epidemiological evidence of a link between gastric adenocarcinoma and mucosa associated lymphoid tissue (MALT) lymphoma (8,9) and *H. pylori* infection has resulted in the classification of the organism as a group I carcinogen (10).

Principle of the Assay

The *H. pylori* IgG antibody test is a qualitative test based on an enzyme immunoassay (EIA) utilizing a horseradish peroxidase conjugated detection antibody.



Assay Highlights

- Red colored diluent buffer
- Excellent intra- and interassay precision
- Results in less than 2 hours affording a fast patient turnaround
- Protocol is easily adapted for automated platforms