

TEST NUMBER: #####
 PATIENT NUMBER: #####
 GENDER: Female
 AGE: 60
 DATE OF BIRTH: dd-mm-yyyy

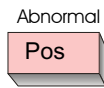
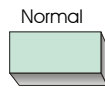
COLLECTED: dd/mm/yyyy
 RECEIVED: dd/mm/yyyy
 TESTED: dd/mm/yyyy

PRACTITIONER: **Nordic Laboratories**
 ADDRESS:

TEST NAME: H. Pylori add on For CSAPx2

Helicobacter Pylori Stool Antigen

H. pylori



Reference
 Neg

The HpSA enzyme immunoassay (EIA) is an in vitro qualitative procedure for the detection of H. Pylori antigens in the stool. Test results are intended to aid the diagnosis of H. Pylori infection, and to monitor response during and post therapy.

Date Collected: **01/01/2019**
 Date Received: **01/01/2019**
 Date Completed: **01/01/2019**

Comments:

PATIENT: Sample Report		TEST REF: TST-##-####
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INTRODUCTION

This analysis of the stool specimen provides fundamental information about the overall gastrointestinal health of the patient. When abnormal microflora or significant aberrations in intestinal health markers are detected, specific interpretive paragraphs are presented. If no significant abnormalities are found, interpretive paragraphs are not presented.

Helicobacter pylori

Helicobacter pylori (H. pylori) was detected by enzyme immunoassay in this stool specimen. H. pylori is a spiral-shaped, flagellated, micro-aerophilic organism, which resides primarily in the human gastric mucosa [1]. H. pylori is able to thrive in the high acidity of stomach by its ability to produce the enzyme urease [1]. Urease converts urea from saliva and gastric juices into bicarbonate and ammonia which are strong bases. This creates a neutralizing cloud of chemicals which surround H. pylori and allow it to survive in its environment.

H. pylori has worldwide distribution, most notably in developing countries where the rate of acquisition is up to 90% in children up to the age of five [2]. Overcrowding and poor sanitary conditions contribute to an increase of H. pylori infection [1]. In the United States, the projected incidence of H. pylori infection is 0.5 to 1%, with far fewer infections occurring in childhood [3]. About 50% of the rate of incidence of H. pylori infection in the U.S. occurs in adults over the age of 60 [3].

Research has indicated that a strong correlation exists between the presence of H. pylori and those suffering from chronic gastritis as well as gastric and duodenal ulcers [2]. H. pylori has been isolated in 90-95% of patients with duodenal ulcers and up to 80% of patients with gastric ulcers [4]. The rate of recurrence of duodenal ulcers is reduced dramatically following successful eradication of H. pylori (4% percent compared to 80% with ongoing infection) [5]. Recurrence of gastric ulceration with bleeding is virtually eliminated with successful H. pylori treatment vs. a 33% risk of rebleeding with untreated or unsuccessfully treated H. pylori infection [5]. One possible therapy often used includes "triple therapy" with bismuth, metronidazole and either amoxicillin or tetracycline [5]. Natural agents include garlic [6], mastic gum [7], tea catechins [8], and deglycyrrhized licorice [9].

H. pylori can colonize the stomach for many years or decades. It may remain inactive/asymptomatic or can cause on-going gastric inflammation via secretion of inflammatory substances such as CagA toxin and interleukin 8 [2]. It is not yet certain which host factors contribute to progression of inflammation and pathogenicity [2]. H. pylori is also recognized as a class I carcinogen for the development of adenocarcinoma [2]. Current research is also exploring the possible involvement of H. pylori in non-gastrointestinal conditions such as coronary heart disease, Reynaud's phenomenon, diabetes, and gallstone disease [10].

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7. Marone P, Bono L, Leone E, et al. Bactericidal activity of Pistacia lentiscus mastic gum against Helicobacter Pylori. J Chemother 2001;13(6):611-4.
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