

Association Between *Helicobacter Pylori* Infection and Pulmonary Function Test Parameters in Asymptomatic Adults

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Abstract

Background: The gastrointestinal tract is the main organ affected by *Helicobacter pylori*, a common chronic bacterial illness. There is growing evidence that *H. pylori*-induced chronic systemic inflammation may cause extra-gastric symptoms, such as possible respiratory system impacts. Investigating the relationship between *H. pylori* infection and pulmonary function test (PFT) characteristics was the goal of this investigation. **Patients and Methods:** Fifty of the 105 patients in this study had an *H. pylori* infection, was conducted while a control group of 55 seemingly healthy, nonsmoking volunteers with negative *H. pylori* infection was matched to the patients (cases) in terms of age, sex, height, weight, and body mass index (BMI). Pulmonary function tests were done for both groups. Data analysis was done by using the variance analysis method for the independent T-test and Chi-Square Test. **Result:** The positive group for *H. pylori* is significantly linked to lower forced expiratory volume in one second (FEV₁) and the FEV₁/FVC ratio when compared to negative controls. The means of forced vital capacity (FVC), although it has no significant relation, is lower than that of the control group. **Conclusion:** Our research shows a strong correlation between lower pulmonary function parameters and *H. pylori* seropositivity.

Keywords: Pulmonary Function tests, Respiratory disorders, *Helicobacter pylori*.

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Introduction

The World Health Organization (WHO) states that prevention, control, and treatment of respiratory problems must be given high priority in international health decision-making because they are one of the leading causes of sickness and death globally.[1]. Infections are currently the most common cause of respiratory illnesses in both adults and children, with varying results based on the host, environmental conditions, and the causative agent. Examining whether non-traditional agents play a causal role in the pathogenesis of respiratory disorders is crucial, as established etiologic agents and risk factors account for only a portion of cases [2].

About half of all people worldwide are infected with the spiral-shaped Gram-negative bacterium *H. pylori*,

which colonizes the human gastric mucosa.[3]. It is frequently linked to low socioeconomic position and is typically acquired during childhood. Gastroduodenal disorders such peptic ulcer disease, gastric cancer, and mucous-associated lymphoid tissue lymphoma are brought on by *H. pylori* infection [4]. There is also evidence about the association of *H. pylori* with non-gastrointestinal diseases, like diabetes, respiratory, coronary artery disease, and Parkinson's disease [5]. Both oropharyngeal dysphagia and gastroesophageal reflux diseases may raise the likelihood of *H. pylori* colonization in the airways, which could exacerbate symptoms and cause lung dysfunction.[5]

H. pylori infection has also been linked to vitamin B12 deficiency, idiopathic thrombocytopenic purpura, and unexplained iron deficiency anemia; nevertheless, there is

ongoing discussion on various extra-gastric disorders.[6]. A number of studies were conducted and published in English between 1965 and 2021. Patients with chronic obstructive pulmonary disease, bronchial asthma, bronchiectasis, TB, and cystic fibrosis have all shown controversial results. Larger studies with a suitable epidemiological design are needed because there is currently no conclusive evidence linking *H. pylori* infection to respiratory disorders.[7]. However, its potential impact on pulmonary function remains underexplored. Chronic low-grade inflammation caused by *H. pylori* may have subclinical effects on the respiratory system, possibly contributing to subtle changes in lung function even in individuals without known respiratory disease [8].

There are two types of procedures for diagnosing *H. pylori* infection: invasive and non-invasive. The former relies on biopsy specimens taken during endoscopy. The clinical setting influences the test selection. The sensitivity and specificity of these techniques differ, which could result in biases in misclassification.[9]. While serologic testing for *H. pylori* IgG has a specificity of less than 80% for active *H. pylori* infection, urea breath tests and stool antigen tests have excellent sensitivity and specificity (above 90%) among non-invasive procedures, which is why we chose them for this study.[11,10].

The most popular and inexpensive test for respiratory function is spirometry [19]. Forced vital capacity (FVC), forced expiratory volume in the first second (FEV1), and the FEV1/FVC ratio are the most often employed metrics in this test [20]. The two indices of FVC and FEV1 are related to the speed of exhaled air flow inside the trachea, and some factors, such as increased BMI, will have a negative effect on it [18].

Aim of the study

This study aims to examine whether asymptomatic *H. pylori*-positive adults show variations in pulmonary function test relative to *H. pylori*-negative controls using standard spirometry.

Patients and Methods

Fifty of the 105 patients in this study had an *H. pylori* infection was conducted in Basrah Teaching Hospital, Iraq,

from 30 June 2025 to 31 October 2025, which determined by the stool antigen test. Additionally, a control group of 55 seemingly healthy, nonsmoking volunteers with negative *H. pylori* infection was matched to the patients (cases) in terms of age, sex, height, weight, and body mass index (BMI). The internal medicine consulting professor at the Private Medical Clinic was the source of the patients. The University of Basrah's College of Dentistry's local ethics committee gave its approval to the study. Each participant provided written, fully informed permission.

Inclusion criteria (cases)

Inclusion criteria for patients include age from 18 years and above with a history of *H. pylori* infection, and having some kinds of pulmonary problems at the time of visit, non-smoking or ex - smoked for less than 6 months, not taking *H. pylori* eradication treatment, or taking any acid-suppressive treatment, and no morbid obesity.

The exclusion criteria (control)

The exclusion criteria for the control group include non-smokers, no known gastrointestinal pathological conditions, including *H. pylori* infection or taking any acid-suppressive treatment, and no morbid obesity.

All participants underwent the following assessments: a complete medical history, including personal, family, and pharmacological therapy, as well as alcohol consumption, smoking, and environmental risk factors; a review of the patient's past medical history; and a thorough clinical examination, which included assessment of COPD symptoms and signs. All participants were subjected to a measurement of the *H. pylori* stool antigen test and a pulmonary function test using the MIR Spirolab datasheet code. 911081x (spiro + oximetry).

Statistical analysis

The data was coded and entered using SPSS (Statistical Package for the Social Sciences) version 25. For categorical data, use frequency (count) and relative frequency (%); for quantitative data, use standard deviation, mean, minimum, and maximum. For quantitative data, the independent T-test is used to compare the mean of the cases and control group; for qualitative data, the Chi-Square test is used; a P value of less than 0.05 is deemed statistically significant.

The results

A total of 105 participants were included in the analysis (55 in the control group with an *H. pylori* negative test and 50 cases with an *H. pylorus* positive test). The two groups were comparable in terms of age and gender distribution ($p > 0.05$), as shown in tables no. 1 and 2, and comparable for, weight, and body mass index distribution, and height ($p > 0.05$), as shown in table no. 3. Participants with *H. pylori* infection (cases) had a statistically significant lower mean FEV1 (2.286 L vs. 2.537 L, $p = 0.031$) and a lower FEV1/FVC ratio (72.55% vs. 78.292%, $p = 0.002$) compared to the control group. There was no significant difference in FVC between the groups ($p = 0.177$), as shown in table no 4.

Table (1): the distribution of the cases and the control group according to age

	Groups	N	Mean	Std. Deviation	P value
Age	control group (<i>H pylori</i> -)	55	39.84	13.639	0.894*
	cases group (<i>H pylori</i> +)	50	39.48	13.780	

*p-value from Independent Samples T-Test

Table (3): the distribution of cases and the control group according to the anthropometric measurements

	control group	N	Mean	Std. Deviation	P value
Height	control group (<i>H pylori</i> -)	55	161.91	9.847	0.884*
	cases group (<i>H pylori</i> +)	50	162.20	10.492	
Weight	control group (<i>H pylori</i> -)	55	78.47	16.952	0.857*
	cases group (<i>H pylori</i> +)	50	77.82	20.029	
BMI	control group (<i>H pylori</i> -)	55	29.99	6.63101	0.776*
	cases group (<i>H pylori</i> +)	50	29.58	7.33983	

* p-value from Independent Samples T-Test

Table (2): the distribution of the cases and the control group according to gender

	Gender		Total	P value
	Male	Female		
control group (<i>H pylori</i> -)	20	35	55	0.840**
cases group (<i>H pylori</i> +)	17	33	50	
Total	37	68	105	

**p-value from Chi-Square Test

Table (4): The distribution of the cases and the control group according to the pulmonary function test

	Groups	N
FEV1	control group (<i>H pylori</i> ⁻)	55
	cases group (<i>H pylori</i> ⁺)	50
FVC	control group (<i>H pylori</i> ⁻)	55
	cases group (<i>H pylori</i> ⁺)	50
FEV1/FVC	control group (<i>H pylori</i> ⁻)	55
	cases group (<i>H pylori</i> ⁺)	50

p-value from Independent Samples T-Test

Discussion

This case-control study investigated the potential relationship between *H. pylori* infection and impaired lung function measures. Our main results show that, after controlling for important confounders like age, gender, smoking status, weight, height, and BMI, the positivity group for *H. pylori* is significantly linked to lower forced

expiratory volume in one second (FEV₁) and the FEV₁/FVC ratio when compared to seronegative controls. The means of forced vital capacity (FVC), although they have no significant relation, are lower than those of the control group. These findings contribute to the increasing evidence connecting chronic extra-gastric *H. pylori* infection to systemic inflammatory illnesses by suggesting that *H. pylori* infection may be an independent risk factor for restrictive and, to a lesser extent, obstructive patterns of lung diseases.

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The idea of a pulmonary deficit is supported by the reported inverse connection between *H. pylori* positivity and FVC and FEV₁. This result agrees with several earlier epidemiological investigations. For example, studies by Joan et al. (2008) (16) and Sabah Ali et al. (2015) (17) found that *H. pylori*-positive people had an inverse relationship between *H. pylori* infection and asthma. Research done by [2] concluded that patients with a positive *H. pylori* infection, although they had a significantly lower mean of FEV₁, a nonsignificant lower FEV₁/FVC ratio, which is also consistent with our research. Another study indicated that there is a negative relation between *H. pylori* infection and FEV₁ but not with FVC nor with FEV₁/FVC ratio (22, 23). Research conducted in by (24) concluded that there is a significant reduction in the mean of FEV₁ among positive *H. pylori* participants. A coexisting obstructive component is also suggested by the small but significant decrease in the FEV₁/FVC ratio in our group, which has been noted in some research on chronic obstructive pulmonary disease (COPD).

Although many researchers agreed with our finding, others may have an opposite idea, like research by (25,21,2,22,23,24) which showed no significant differences between pulmonary function parameters and *H. pylori* infection, and this perhaps due to different study designs

and sample sizes, or may be due to different measurement methods of parameters used.

The mechanisms underlying this potential connection between *H. pylori* infection and lung conditions have not yet been discovered by research. One theory is that extra-gastric disorders arise because of *H. pylori* infection's long-term production of inflammatory mediators. These pathogenic strains are known to cause the release of several proinflammatory cytokines, such as TNF- α , IL-1, and IL-8. It is also believed that these cytokines play a role in pathophysiology. Another possible explanation is that human lung epithelial cells and *H. pylori* proteins, such as heat shock protein 60, share antigenic similarities that may cause an autoimmune cross-reactive reaction and long-term tissue damage.

Our research has several advantages. The accuracy of our pulmonary function data is guaranteed by using standardized spirometry in accordance with ATS/ERS recommendations. The conclusion of an independent connection is strengthened since we carefully adjusted for

significant possible confounders, such as age. Gender, smoking, socioeconomic level, and BMI. This hypothesis might be effectively investigated thanks to the case-control methodology.

These results must be implemented in the context of significant restrictions, though. First off, our study's cross-sectional design makes it impossible to establish causation. We are unable to draw firm conclusions on whether an underlying susceptibility increases the probability of both chronic infection and pulmonary impairment, or whether *H. pylori* infection precedes and contributes to lung function loss. Second, to ascertain *H. pylori* status, future research using stomach biopsies or urea breath testing would yield a more precise classification. Thirdly, even though we control several confounders, it is impossible to completely rule out residual confounding caused by unmeasured factors such as exposure to ambient air pollution, early-life respiratory illnesses. Lastly, our sample size might not have been sufficient for more detailed subgroup analyses.

Our results have potentially important clinical consequences. If there is a causative association, *H. pylori* eradication therapy may be considered as a novel therapeutic approach, especially for people who have a high risk of pulmonary disease or early indicators of lung function loss. It implies that screening for chronic infections such as *H. pylori* may be beneficial for people with inexplicable restrictive lung disease.

Conclusion

In summary, our research shows a strong correlation between lower pulmonary function parameters and *H. pylori* positivity. These findings add to the growing understanding of *H. pylori* as a systemic health modulator that extends beyond the gastrointestinal tract. To determine temporality and dose-response correlations, future studies should use longitudinal cohort designs with direct assessment of active infection (like a breath test). To determine its clinical significance and investigate a possible causal relationship, intervention studies evaluating the effect of *H. pylori* eradication on the course of pulmonary function loss are also necessary.

References

1. Levine SM, Marciniuk DD. Global impact of respiratory disease: what can we do, together, to make a difference?. *Chest*. 2022 May 1;161(5):1153-4. DOI: <https://doi.org/10.1016/j.chest.2022.01.014>
2. Durazzo M, Adriani A, Fagoonee S, Saracco GM, Pellicano R. *Helicobacter pylori* and respiratory diseases: 2021 update. *Microorganisms*. 2021 Sep 26;9(10):2033. <https://doi.org/10.3390/microorganisms9102033>
3. Wang L, Guan Y, Li Y, Liu X, Zhang Y, Wang F, Kong L, Guo Q. Asociación entre enfermedades respiratorias crónicas y *Helicobacter pylori*: un metaanálisis. *Archivos de Bronconeumología*. 2015 Jun 1;51(6):273-8. <http://dx.doi.org/10.1016/j.arbres.2014.03.019>
4. Behrendt CE. Mild and moderate-to-severe COPD in nonsmokers: distinct demographic profiles. *Chest*. 2005 Sep 1;128(3):1239-44. <https://doi.org/10.1378/chest.128.3.1239>
5. Gonzalez-Saitz A, Diez-Manglano J. *Helicobacter pylori* infection in patients with chronic obstructive pulmonary disease. A systematic review and meta-analysis. *Revista Clínica Española (English Edition)*. 2025 Feb 7. <https://doi.org/10.1016/j.rceng.2024.12.004>
6. Malfertheiner P, Mégraud F, O'Morain CA, Gisbert JP, Kuipers EJ, Axon AT, Bazzoli F, Gasbarrini A, Atherton J, Graham DY, Hunt R. Management of *Helicobacter pylori* infection—the Maastricht V/Florence consensus report. *Gut*. 2017 Jan 1;66(1):6-30. <https://doi.org/10.1136/gutjnl-2016-312288>
7. <https://medlineplus.gov/>
8. Doulberis M, Papaefthymiou A, Polyzos SA, Boziki M, Kazakos E, Tziritidou-Chatzopoulou M, Vardaka E, Hammrich C, Kulaksiz H, Riva D, Kiosses C. Impact of *Helicobacter pylori* and metabolic syndrome-related mast cell activation on cardiovascular diseases. *Frontiers in Gastroenterology*. 2024 Mar 4;3:1331330. <https://doi.org/10.3389/fgstr.2024.1331330>
9. Isaeva G, Isaeva R. Molecular methods for the detection of *Helicobacter pylori*. *Minerva Biotecnologica*.

2020 Dec 1;32(4):182-7. <https://doi.org/10.23736/S1120-4826.20.02665-8>

10. Chey WD. Practice Parameters Committee of the American College of Gastroenterology. American College of Gastroenterology guideline on the management of *Helicobacter pylori* infection. *Am J Gastroenterol.* 2007;102:1808-25. doi: 10.1111/j.1572-0241.2007.01393.x

11. Pellicano R, Ribaldone DG, Fagoonee S, Astegiano M, Saracco GM, Megraud F. A 2016 panorama of *Helicobacter pylori* infection: key messages for clinicians. *Panminerva medica.* 2016 Dec 1;58(4):304-17.

12. Chatrangsun B, Vilaichone RK. Endoscopic diagnosis for *H. pylori* infection: white light imaging (WLI) vs. image-enhanced endoscopy (IEE). *Asian Pacific Journal of Cancer Prevention: APJCP.* 2021 Sep;22(9):3031. <http://10.31557/apjcp.2021.22.9.3031>.

14. Shichijo S, Endo Y, Aoyama K, Takeuchi Y, Ozawa T, Takiyama H, Matsuo K, Fujishiro M, Ishihara S, Ishihara R, Tada T. Application of convolutional neural networks for evaluating *Helicobacter pylori* infection status on the basis of endoscopic images. *Scandinavian journal of gastroenterology.* 2019 Feb 1;54(2):158-63. <https://doi.org/10.1080/00365521.2019.1577486>

15. C.H. Lin, P.I. Hsu, C.D. Tseng, P.J. Chao, I.T. Wu, S. Ghose, C.A. Shih, S.H. Lee, J. H. Ren, C.B. Shie, T.F. Lee, Application of artificial intelligence in endoscopic image analysis for the diagnosis of a gastric cancer pathogen-*Helicobacter pylori* infection, *Sci. Rep.* 13 (2023) 13380. <http://10.1038/s41598-023-40179-5>

16. Reibman J, Marmor M, Filner J, Fernandez-Beros ME, Rogers L, Perez-Perez GI, Blaser MJ. Asthma is inversely associated with *Helicobacter pylori* status in an urban population. *PloS one.* 2008 Dec 29;3(12): e4060. doi:10.1371/journal. Pone .0004060

17. Alhelu SA, Alkaaby AR, Majeed AJ, Almuhanah SJ. Correlation Between *Helicobacter Pylori* Infection and Severity of Asthma وبصرًا وبرلا قدشو تباوبلا تاوبلما تباصلًا. KUFA JOURNAL FOR NURSING SCIENCES. 2015;5(2).

18. Enayatjazi M, Enayatjazi S, Ghadiri N, Fathi A. Effect of 8 weeks of Endurance Training on Volume and Capacity of Lungs (FEV1/FVC, FEV1, FVC) and Its Relationship with BMI in Male Smoking students. *New*

Approaches in Exercise Physiology. 2023 Jun 1;5(9):32-43. <https://doi.org/10.22054/nass.2023.74506.1139>

19. Páez-Mora CD, Zona DC, Angarita-Sierra T, Rojas-Paredes ME, Cano-Trejos D. Changes in lung function and dyspnea perception in Colombian Covid-19 patients after a 12-week pulmonary rehabilitation program. *Plos one.* 2024 Nov 15;19(11):e0300826. <https://doi.org/10.1371/journal.pone.0300826>

20. Kattainen S, Lindahl A, Vasankari T, Ollila H, Volmonen K, Piirila P, et al. Lung function and exercise capacity 6 months after hospital discharge for critical COVID-19. *BMC Pulm Med [Internet].* 2022 Dec 1 [cited 2022 Sep 14];22(1). Available from: <https://pubmed.ncbi.nlm.nih.gov/35733179/>

21. Tsang KW, Lam WK, Chan KN, Hu W, Wu A, Kwok E, Zheng L, Wong BC, Lam SK. *Helicobacter pylori* seroprevalence in asthma. *Respiratory medicine.* 2000 Aug 1;94(8):756-9. <https://doi.org/10.1053/rmed.2000.0817>

22. Roussos A, Philippou N, Gourgoulis KI. *Helicobacter pylori* infection and respiratory diseases: a review. *World journal of gastroenterology.* 2003 Jan 15;9(1):5. <http://www.wjgnet.com/1007-9327/9/5.htm>

23. Kanbay M, Kanbay A, Boyacioglu S. *Helicobacter pylori* infection as a possible risk factor for respiratory system disease: a review of the literature. *Respiratory medicine.* 2007 Feb 1;101(2):203-9. doi: 10.1016/j.rmed.2006.04.022

24. Center WC, Center GC. *Helicobacter Pylori* infection in never-smoking male patients with chronic obstructive pulmonary disease and its relation to lung function. *American Journal of Immunology.* 2012;8(4):146-53. doi:10.3844/ajisp.2012.146.153

25. Lee HY, Kang HR, Lee JK, Heo EY, Choi SH, Kim DK. The effect of *Helicobacter pylori* infection on the decline of lung function in a health screening population. *Annals of Palliative Medicine.* 2020 Sep;9(5):3115122-3122.

<http://dx.doi.org/10.21037/apm-20-850>