OPEN ACCESS

Orapuh Journal

ORIGINAL ARTICLE

Journal of Oral & Public Health

Prevalence and associated factors of dyslipidemia among the population infected with *Helicobacter pylori* in Bunia town, Province of Ituri, Democratic Republic of the Congo

Bisingurege, K. F.[1], Tibamwenda, B. Y.[2], Mobali, W. C.[1], Ntanyungu, I. J.[1], Chelo, N. H.[3], Amuda, B. D.[4], Nyenke, B. G.[1], Kpane, C. F.[2], Magapa, C. M.[4], Mbabazi, R. J.[1], Manwa, B. B.[5], Batina, A. S.[6], & Tsongo, K. Z.[6]

[1]*Department of Internal Medicine, Shalom University of Bunia (USB), Democratic Republic of the Congo* [2]*Department of Internal Medicine, University of Bunia (UNIBU), Democratic Republic of the Congo* [3]*Department of Public Health, University of Goma (UNIGOM), Democratic Republic of the Congo* [4]*Higher Institute of Medical Techniques of Bunia (ISTM/Bunia), Democratic Republic of the Congo* [5]*Department of Pathological Anatomy, Official University of Bukavu (UOB), Democratic Republic of the Congo* [6]*Department of Internal Medicine, University of Kisangani (UNIKIS), Democratic Republic of the Congo*

ARTICLE INFO

Received: *27 December 2025*
Accepted: *14 February 2026*
Published: *30 March 2026*

Keywords:
Prevalence, associated factors, dyslipidemia, *Helicobacter pylori*, Bunia

Peer-Review: *Externally peer-reviewed*

© 2026 The Authors.

Re-use permitted under CC BY-NC 4.0
No commercial re-use or duplication.

Correspondence to:
Dr. Amuda Baba
amudadime@gmail.com

To cite:
Bisingurege, K. F., Tibamwenda, B. Y., Mobali, W. C., Ntanyungu, I. J., Chelo, N. H., Amuda, B. D., Nyenke, B. G., Kpane, C. F., Magapa, C. M., Mbabazi, R. J., Manwa, B. B., Batina, A. S., & Tsongo, K. Z. (2026). Prevalence and associated factors of dyslipidemia among the population infected with *Helicobacter pylori* in Bunia town, Province of Ituri, Democratic Republic of the Congo. *Orapuh Journal, 7*(2), e1420 https://dx.doi.org/10.4314/orapj.v7i2.20

ISSN: *2644-3740*

Published by Orapuh, Inc. (info@orapuh.org)

Editor-in-Chief: Prof. V. E. Adamu
Orapuh, F. Gaye R., Sukuta, Greater Banjul, The Gambia, editor@orapuh.org.

ABSTRACT

Introduction

Beyond gastric disease, *Helicobacter pylori* infection has been implicated in metabolic disturbances, including dyslipidemia; however, evidence from eastern Democratic Republic of the Congo remains limited.

Purpose

This study aimed to determine the prevalence and associated factors of dyslipidemia among individuals infected with *H. pylori* in Bunia, Ituri Province.

Methods

This cross-sectional study was conducted at the Evangelical Medical Centre Clinic (CME-Bunia) among 384 adult patients diagnosed with *H. pylori* infection from January to June 2025. For diagnosis, 0.5 g of stool was diluted in solvent and analyzed using a test strip. For lipid profiling, 5 mL of fasting venous blood was collected, and serum was analyzed for high-density lipoprotein (HDL-C), low-density lipoprotein (LDL-C), and triglycerides. A structured questionnaire was used to collect sociodemographic, medical, and lifestyle-related data. Data were analyzed using STATA version 15.2. Logistic regression was used for bivariate and multivariate analyses.

Results

The prevalence of dyslipidemia was 54.17%. The most frequent lipid abnormalities were combined elevated LDL-C and triglycerides (20.57%), followed by combined low HDL-C and elevated triglycerides (20.05%). Alcohol consumption of more than two glasses per day was associated with a tenfold increased risk of LDL-C disorder after adjustment for other variables (AOR = 9.96, 95% CI [4.56, 21.75], $p < .001$). Factors significantly associated with HDL-C disorder included age 31–40 years (AOR = 2.72, 95% CI [1.31, 5.64], $p = .007$), hypertension (AOR = 2.49, 95% CI [1.48, 4.19], $p = .001$), overweight (AOR = 1.80, 95% CI [1.11, 2.91], $p = .008$), and obesity (AOR = 2.30, 95% CI [1.24, 4.25], $p < .001$). Hypertension was associated with triglyceride disorder (AOR = 0.56, 95% CI [0.33, 0.93], $p = .026$). Smoking was significantly associated with total cholesterol disorder (AOR = 1.86, 95% CI [1.01, 3.42], $p = .046$).

Conclusion

Routine lipid profile testing should be introduced for patients diagnosed with *H. pylori* infection through systematic screening. Community health education should be strengthened to inform both the population and healthcare professionals about potential complications, given the high prevalence of *H. pylori* infection.

INTRODUCTION

Helicobacter pylori (*H. pylori*) is well known as a causative agent of peptic ulcer disease and gastric cancer. However, over the past decade, growing evidence has implicated this pathogen in a range of extragastric conditions, including cardiovascular and metabolic disorders (Chen et al., 2019; Pellicano et al., 2020; Tsay & Hsu, 2018). With an estimated global prevalence of approximately 48.5%, *H. pylori* infection affects individuals worldwide (Xie et al., 2024). The bacterium has been associated with lipid, metabolic, hematological, cardiovascular, and other systemic disorders (Pellicano et al., 2020; Shindler-Itskovitch et al., 2019). According to Fu et al. (2025), the relationship between triglyceride levels and *H. pylori* infection may be influenced by age, sex, blood glucose levels, body mass index (BMI), and chronic renal failure. Another study reported that among individuals with diabetes mellitus, *H. pylori* infection may increase the risk of dyslipidemia, suggesting that the bacterium contributes to metabolic disturbances beyond its established gastrointestinal effects (Yang et al., 2024).

Dyslipidemia refers to abnormalities in plasma lipid levels and may be quantitative, qualitative, or both. Quantitative dyslipidemia involves elevated plasma lipid levels, including increased total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), as well as reduced high-density lipoprotein cholesterol (HDL-C). These abnormalities may occur independently or in combination. Qualitative dyslipidemia refers to alterations in lipid composition, such as an increase in small, dense LDL-C particles, increased triglyceride content, or increased electronegativity of LDL-C (Wang et al., 2022; Oguejiofor et al., 2012).

According to the World Health Organization, dyslipidemia is defined as the presence of abnormal levels of one or more of the following: HDL-C, LDL-C, triglycerides, and total cholesterol. In this study, lipid abnormalities were defined according to the reference ranges of the machine used: triglycerides 40–150 mg/dL (1.7–4.5 mmol/L), total cholesterol > 200 mg/dL (> 5.2 mmol/L), LDL-C > 135 mg/dL (> 3.5 mmol/L), and HDL-C < 35 mg/dL (< 0.9 mmol/L) in men or < 40 mg/dL (< 1.0 mmol/L) in women (Oguejiofor et al., 2012; Yang et al., 2024).

Dyslipidemia is a major modifiable risk factor for cardiovascular disease, stroke, and type 2 diabetes mellitus. It is characterized by an abnormal lipid profile, including elevated cholesterol, triglycerides, or both, and/or reduced HDL-C. Data from the U.S. National Health and Nutrition Examination Survey (2003–2006) showed that lipid abnormalities were present in approximately 53% of American adults (Wang et al., 2022).

Studies have shown that *H. pylori* infection may interfere with multiple biological processes, contributing to the development of diseases outside the stomach. Kountouras et al. (2018) reported associations between *H. pylori* infection and vitamin B12 deficiency, insulin resistance, metabolic syndrome, diabetes mellitus, and nonalcoholic fatty liver disease. Furthermore, *H. pylori* infection may increase the risk of acute coronary syndrome, cerebrovascular disease, and neurodegenerative disorders (Quan et al., 2025). Although the exact mechanisms remain unclear, further research is needed to elucidate the pathogenesis of extragastroduodenal diseases associated with this infection (Tsay & Hsu, 2018). Kim et al. (2016) also reported that *H. pylori* infection is associated with dyslipidemia and other cardiovascular risk factors.

H. pylori is among the most widespread infectious agents worldwide. It induces chronic low-grade inflammation of the gastrointestinal tract and may lead to digestive diseases such as chronic gastritis, gastric ulcer, and gastric cancer. In addition, it has been implicated in extragastrointestinal diseases such as metabolic syndrome and nonalcoholic fatty liver disease (Wang et al., 2022).

A retrospective study evaluated lipid metabolism profiles among individuals with persistent *H. pylori* infection, those who had undergone eradication therapy, and those who had never tested positive for *H. pylori* using repeated ^13C-urea breath tests. The findings indicated that HDL-C and LDL-C levels continued to worsen in the persistently infected group, whereas improvement was more pronounced among individuals who underwent eradication therapy. These findings suggest that *H. pylori* eradication may reduce deterioration in lipid metabolism (Wang et al., 2022). The same study reported that eradication may be particularly beneficial among patients with high diastolic blood pressure, moderate-to-high mean

platelet volume, and low total protein levels (Wang et al., 2022).

Alterations in HDL-C and LDL-C levels in *H. pylori* infection may occur through changes in ghrelin and leptin secretion or impaired nutrient absorption. The infection may contribute to disturbances in lipid metabolism through acute-phase responses and chronic cytokine release, potentially leading to an atherogenic lipid profile and increased cardiovascular risk (Wang et al., 2022).

A study conducted in Tel Aviv reported that among 76,965 individuals, 1,397 (0.9%) experienced stroke, and 52.0% of those with stroke tested positive for *H. pylori*. The probability of stroke was significantly higher among individuals with *H. pylori* infection (adjusted odds ratio [aOR] = 1.16, 95% confidence interval [CI] [1.04, 1.29]), gastric ulcer (aOR = 1.50, 95% CI [1.18, 1.91]), and duodenal ulcer (aOR = 1.25, 95% CI [1.07, 1.46]). These findings support the hypothesis that stroke may be associated with a history of *H. pylori* infection (Shindler-Itskovitch et al., 2019).

Dyslipidemia appears to be common among individuals infected with *H. pylori*. Studies conducted in Iran (60%), China, and Ethiopia (71%) have reported high prevalence rates (Karim et al., 2018; Nigatie et al., 2022). A study in Uganda reported a prevalence of 41.58% (Kagoro et al., 2024). Dyslipidemia may manifest as increased total cholesterol, LDL-C, or triglycerides, or as decreased HDL-C (Gaonkar et al., 2025). However, prevalence rates vary depending on geographic and environmental factors.

A recent study found that the prevalence of *H. pylori* infection is higher in Africa (79.1%), South America (69.4%), and Asia (54.7%), while it is lower in North America (37.1%) and Oceania (24.4%). The same study reported that in Benin, the seroprevalence was 75.4% in an urban area (Cotonou) and 72.3% in a rural area (Pahou) (Saké et al., 2023). In Cameroon, a study conducted in Bafoussam also showed that *H. pylori* infection was associated with an increased risk of dyslipidemia. Therefore, eradication of *H. pylori* may be beneficial in reducing dyslipidemia and preventing cardiovascular disease (Fang et al., 2022; Park et al., 2021).

Despite previous studies estimating the prevalence of *H. pylori* infection in Bunia at approximately 67% (François et al., n.d.), updated evidence regarding dyslipidemia prevalence and associated factors among infected individuals remains limited. Therefore, this study aimed to determine the prevalence and associated factors of dyslipidemia among individuals infected with *H. pylori* in Bunia, Ituri Province, Democratic Republic of the Congo. Findings from this study may support the justification for routine lipid profile testing among all *H. pylori*-positive patients.

METHODS

Study Setting

This study was conducted at the Evangelical Medical Centre Clinic (CME) in Bunia from January to June 2025. CME is a tertiary referral hospital located in the Hoho health catchment area of Mbunya sub-county, serving the population of Bunia town in Ituri Province, northeastern Democratic Republic of the Congo. The facility includes an accredited provincial laboratory equipped with an automated clinical chemistry system and other diagnostic equipment.

The hospital employs specialist physicians, general practitioners, medical trainees, and clinical staff. The laboratory is adequately equipped and staffed to perform a variety of tests, including stool antigen testing for *H. pylori* and lipid profiling. The facility also has the capacity to perform biopsies.

Study Design

A cross-sectional study design was used.

Study Population

The study population consisted of all adult patients diagnosed with *H. pylori* infection who attended outpatient services at CME-Bunia between January and June 2025, totaling 401 individuals.

Inclusion and Exclusion Criteria

Inclusion criteria were: confirmed *H. pylori* infection based on stool antigen testing or biopsy following endoscopic sampling, provision of written informed consent, and fasting for at least 8 hours overnight before blood sampling for lipid profile testing.

Participants were excluded if they met any of the following criteria: pregnancy, HIV/AIDS, diabetes mellitus, malignancy, chronic liver failure, chronic kidney

failure, use of systemic corticosteroids or immunosuppressive drugs, or refusal to participate.

Sample Size

A total of 384 participants were included in the study. This represented all eligible patients among the 401 individuals who tested positive for *H. pylori* during the study period. Therefore, the study used an exhaustive sample to provide an accurate representation of dyslipidemia among *H. pylori*-infected individuals in Bunia.

Data Collection

Data collection was conducted in two phases.

Diagnosis of H. pylori Infection

A stool antigen test was performed for each participant. The test had a reported sensitivity of 96% and specificity of 95.7%. Approximately 0.5 g of stool was diluted in solvent and applied to a test strip, with an overall agreement of 95.4% (92.8%–97.3%). Diagnostic kits were manufactured by Safheal (Zhejiang Anji Saianfu Biotech Co., Ltd., China).

Lipid Profile Assessment

For lipid profiling, 5 mL of venous blood was collected from fasting participants. After centrifugation, serum was analyzed for HDL-C, LDL-C, triglycerides, and total cholesterol. Results were printed using the CYANSmart analyzer.

The analyzer reference ranges were: HDL-C 59–80 mg/dL, LDL-C 50–100 mg/dL, triglycerides 35–150 mg/dL, and total cholesterol 120–200 mg/dL.

Questionnaire Data

A structured questionnaire was administered to collect sociodemographic characteristics, medical history, and lifestyle-related information.

Data Analysis

Collected data were entered into Microsoft Excel 2021 and exported to STATA version 15.2 for analysis. The prevalence of dyslipidemia was presented using graphs with 95% confidence intervals. Lipid profile findings were summarized in tables. Factors associated with dyslipidemia were assessed using bivariate and multivariate logistic regression.

Variables with a p value ≤ .20 in bivariate analysis were included in the multivariate logistic regression model. Variables were considered statistically significant at $p \leq .05$. Measures of association were expressed as crude odds ratios (ORs) for bivariate analysis and adjusted odds ratios (AORs) for multivariate analysis, each reported with 95% confidence intervals and corresponding p values.

Variables

Independent variables included sociodemographic factors (age, sex, marital status, and residence), medical history variables (hypertension and body mass index), and lifestyle factors (smoking, alcohol consumption, and occupational stress). BMI classification followed World Health Organization criteria (World Health Organization, n.d.). Dependent variables included HDL-C, LDL-C, triglycerides, and total cholesterol.

Ethical Considerations

Ethical approval was obtained from the Ethics Committee of the University of Goma (approval letter reference: UNIGOM/CME/013/2023). Patients who tested positive for *H. pylori* were approached individually and provided with an information sheet. Written informed consent was obtained from all participants prior to enrollment.

RESULTS

Sociodemographic Characteristics and Lifestyle Habits

Table 1 presents the sociodemographic characteristics and lifestyle habits of participants infected with *H. pylori*. Participants aged ≥ 41 years were the most represented. Most participants were male (55.73%) and single (50.79%). The majority had primary-level education (78.13%), were Catholic (69.79%), and worked as farmers (71.35%). Most participants lived in urban areas (79.69%), reported no alcohol consumption (67.71%), and did not smoke (82.81%). Most participants did not have hypertension (78.39%), reported having stressful work (97.40%), and were overweight (49.48%).

Table 1:
Sociodemographic Characteristics and Lifestyle Habits of Participants With *H. pylori* Infection in Bunia (N = 384)

Characteristic	n	%
Age (years)		
21–30	52	13.54
31–40	101	26.30
41–50	130	33.85
≥ 51	101	26.30

Characteristic	n	%
Sex		
Male	214	55.73
Female	170	44.27
Marital status		
Married/cohabiting	95	25.00
Single	193	50.79
Widowed	70	18.42
Divorced	22	5.79
Education level		
Illiterate	24	6.25
Primary	300	78.13
Secondary	44	11.46
Higher	16	4.17
Religion		
Catholic	268	69.79
Muslim	45	11.72
Protestant	48	12.50
Traditional	23	5.99
Occupation		
Farmer	274	71.35
Civil servant	52	13.54
Business person	30	7.81
Other	28	7.29
Residence		
Rural	78	20.31
Urban	306	79.69
Alcohol consumption (per day)		
> 2 glasses	44	11.46
1–2 glasses	80	20.83
None	260	67.71
Smoking		
No	318	82.81
Yes	66	17.19
Hypertension		
No	301	78.39
Yes	83	21.61
Stressful job		
No	10	2.60
Yes	374	97.40
Body mass index (BMI)		
Underweight	2	0.52
Normal	120	31.25
Overweight	190	49.48
Obese	72	18.75

Note: Values are presented as frequency (n) and percentage (%).

Prevalence of Dyslipidemia

The overall prevalence of dyslipidemia was 54.17%. Triglyceride disorder was the most common abnormality, followed by HDL-C disorder and LDL-C disorder **(Table 2)**.

Table 2:
Prevalence of Dyslipidemia by Lipid Profile Marker Among *H. pylori*-Infected Participants in Bunia (N = 384)

Lipid marker	No, n (%)	Yes, n (%)
LDL-C disorder	226 (58.85)	158 (41.15)
HDL-C disorder	203 (52.86)	181 (47.14)
Triglyceride disorder	176 (45.83)	208 (54.17)
Total cholesterol disorder	308 (80.21)	76 (19.79)

Note: Dyslipidemia was defined according to the lipid reference values described in the Methods section.

Combined Lipid Abnormalities

The most frequent combined lipid abnormalities were LDL-C with triglycerides (20.57%) and HDL-C with triglycerides (20.05%) **(Table 3)**.

Table 3:
Prevalence of Combined Lipid Abnormalities Among *H. pylori*-Infected Participants in Bunia (N = 384)

Combined abnormality	No, n (%)	Yes, n (%)
LDL-C and HDL-C	305 (79.43)	79 (20.57)
LDL-C and triglycerides	305 (79.43)	79 (20.57)
LDL-C and total cholesterol	327 (85.16)	57 (14.84)
HDL-C and triglycerides	307 (79.95)	77 (20.05)
HDL-C and total cholesterol	341 (88.80)	43 (11.20)
Triglycerides and total cholesterol	343 (89.32)	41 (10.68)
LDL-C, HDL-C, and triglycerides	347 (90.36)	37 (9.64)
LDL-C, HDL-C, and total cholesterol	352 (91.67)	32 (8.33)
HDL-C, triglycerides, and total cholesterol	361 (94.01)	23 (5.99)
LDL-C, HDL-C, triglycerides, and total cholesterol	365 (95.05)	19 (4.95)

Note: Values are presented as frequency (n) and percentage (%).

Results of univariate analysis

Table 4 summarizes crude associations between lipid profile disorders (LDL-C, HDL-C, triglycerides, and total cholesterol) and participant characteristics. In univariate analysis, age group, marital status, education level, alcohol consumption, smoking, hypertension, and BMI were significantly associated with one or more lipid abnormalities. Variables with $p \leq .20$ were included in the multivariate regression models.

Table 4:

Univariate Associations Between Lipid Disorders and Participant Characteristics Among *H. pylori*-Infected Patients in Bunia (N = 384)

A. Sociodemographic Factors

Characteristic	n (%)	LDL-C OR (95% CI)	*p*	HDL-C OR (95% CI)	*p*	TG OR (95% CI)	*p*	TC OR (95% CI)	*P*
Age (years)									
21–30	52 (13.54)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
31–40	101 (26.30)	0.78 (0.39, 1.55)	.486	2.83 (1.38, 5.79)	.004	0.90 (0.45, 1.76)	.760	1.06 (0.42, 2.50)	.937
41–50	130 (33.85)	1.18 (0.61, 2.28)	.603	2.31 (1.16, 4.63)	.017	1.03 (0.54, 1.96)	.925	1.56 (0.68, 3.54)	.289
≥ 51	101 (26.30)	1.13 (0.57, 2.24)	.706	2.32 (1.13, 4.75)	.021	1.01 (0.51, 1.98)	.964	0.96 (0.39, 2.34)	.941
Sex									
Female	170 (44.27)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Male	214 (55.73)	0.73 (0.48, 1.11)	.147	1.45 (0.97, 2.18)	.068	0.84 (0.56, 1.26)	.419	1.42 (0.86, 2.35)	.169
Marital status									
Married/cohabiting	95 (25.00)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Single	193 (50.79)	1.56 (0.93, 2.60)	.086	2.57 (1.54, 4.29)	< .001	1.21 (0.74, 1.98)	.445	0.77 (0.42, 1.42)	.413
Widowed	70 (18.42)	1.18 (0.62, 2.25)	.611	1.66 (0.88, 3.15)	.116	1.34 (0.72, 2.49)	.352	1.14 (0.55, 2.37)	.715
Divorced	22 (5.79)	3.50 (1.33, 9.20)	.011	2.09 (0.81, 5.36)	.122	0.62 (0.23, 1.67)	.351	0.56 (0.15, 2.09)	.391
Education level									
Primary	300 (78.13)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Illiterate	24 (6.25)	1.14 (0.49, 2.67)	.747	1.20 (0.52, 2.77)	.659	1.33 (0.57, 3.06)	.500	0.87 (0.28, 2.64)	.808
Secondary	44 (11.46)	1.46 (0.77, 2.77)	.236	1.32 (0.70, 2.48)	.390	0.71 (0.37, 1.35)	.300	1.28 (0.59, 2.74)	.524
Higher	16 (4.17)	4.52 (1.52, 15.32)	.008	2.00 (0.71, 5.67)	.187	0.51 (0.17, 1.51)	.226	2.61 (0.91, 7.49)	.074
Religion									
Muslim	45 (11.72)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Catholic	268 (69.79)	1.99 (1.00, 3.97)	.049	0.69 (0.37, 1.31)	.270	2.02 (1.03, 3.97)	.041	2.40 (0.91, 6.36)	.076
Protestant	48 (12.50)	1.01 (0.41, 2.48)	.970	0.67 (0.29, 1.31)	.349	1.72 (0.73, 4.03)	.210	0.93 (0.25, 3.45)	.914
Traditional	23 (5.99)	2.25 (0.79, 6.39)	.120	0.51 (0.18, 1.43)	.203	2.87 (1.01, 8.12)	.046	1.68 (0.40, 6.99)	.473

B. Lifestyle and Clinical Factors

Characteristic	n (%)	LDL-C OR (95% CI)	*p*	HDL-C OR (95% CI)	*p*	TG OR (95% CI)	*p*	TC OR (95% CI)	*p*
Occupation									
Farmer	274 (71.35)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Civil servant	52 (13.54)	0.96 (0.52, 1.76)	.908	3.06 (1.62, 5.78)	.001	1.25 (0.69, 2.26)	.461	0.88 (0.41, 1.87)	.753
Business person	30 (7.81)	1.42 (0.66, 3.03)	.358	1.36 (0.64, 2.89)	.422	0.88 (0.41, 1.89)	.753	0.57 (0.19, 1.70)	.317
Other	28 (7.29)	0.67 (0.29, 1.54)	.352	1.36 (0.62, 2.96)	.437	0.54 (0.23, 1.09)	.155	0.62 (0.20, 1.85)	.394
Residence									
Rural	78 (20.31)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Urban	306 (79.69)	1.62 (0.96, 2.75)	.069	1.55 (0.93, 2.59)	.087	0.92 (0.56, 1.51)	.750	1.16 (0.61, 2.20)	.647
Alcohol consumption (per day)									
None	260 (67.71)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
1–2 glasses	80 (20.83)	4.26 (2.51, 7.23)	< .001	1.44 (0.87, 2.39)	.149	3.19 (1.88, 5.40)	< .001	1.13 (0.58, 2.20)	.713
> 2 glasses	44 (11.46)	9.96 (4.56, 21.75)	< .001	0.82 (0.42, 1.56)	.549	1.95 (1.02, 3.71)	.042	4.87 (2.47, 9.61)	< .001
Smoking									
No	318 (82.81)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Yes	66 (17.19)	1.14 (0.67, 1.96)	.612	1.93 (1.12, 3.31)	.017	1.05 (0.62, 1.79)	.839	1.85 (1.01, 3.38)	.046

https://dx.doi.org/10.4314/orapj.v7i2.20

Characteristic	n (%)	LDL-C OR (95% CI)	*p*	HDL-C OR (95% CI)	*p*	TG OR (95% CI)	*p*	TC OR (95% CI)	*p*
Hypertension									
No	301 (78.39)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Yes	83 (21.61)	1.05 (0.64, 1.72)	.831	2.38 (1.44, 3.94)	.001	0.56 (0.33, 0.93)	.025	1.27 (0.70, 2.28)	.424
Stressful job									
No	10 (2.60)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Yes	374 (97.40)	0.69 (0.19, 2.43)	.566	0.58 (0.16, 2.11)	.414	0.84 (0.23, 3.06)	.789		
Body mass index (BMI)									
Normal	120 (31.25)	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—	1.00 (ref)	—
Overweight	190 (49.48)	1.31 (0.81, 2.11)	.256	1.74 (1.09, 2.78)	.019	1.58 (0.99, 2.51)	.052	1.31 (0.71, 2.40)	.382
Obese	72 (18.75)	2.61 (1.43, 4.75)	.002	2.13 (1.18, 3.86)	.012	1.74 (0.96, 3.14)	.064	2.08 (1.02, 4.24)	.043

Note: OR = crude odds ratio; CI = confidence interval; TG = triglycerides; TC = total cholesterol; ref = reference category.

Results of multivariate analysis

Multivariate Analysis for LDL-C Disorder

Alcohol consumption remained independently associated with LDL-C disorder. Participants who consumed more than two alcoholic beverages per day had approximately 10 times higher odds of LDL-C disorder compared with non-drinkers (AOR = 9.96, 95% CI [4.56, 21.75], $p < .001$). Participants who consumed 1–2 glasses per day also had increased odds of LDL-C disorder (AOR = 4.26, 95% CI [2.51, 7.23], $p < .001$). The Hosmer–Lemeshow goodness-of-fit test suggested an adequate model fit ($p = .285$).

Table 5:
Multivariate Logistic Regression Analysis of Factors Associated With LDL-C Disorder Among *H. pylori*-Infected Participants (N = 384)

Characteristic	n (%)	AOR	95% CI	*p*
Alcohol consumption (per day)				
None	260 (67.71)	1.00	Reference	—
1–2 glasses	80 (20.83)	4.26	[2.51, 7.23]	< .001
> 2 glasses	44 (11.46)	9.96	[4.56, 21.75]	< .001

Note: AOR = adjusted odds ratio; CI = confidence interval. Hosmer–Lemeshow test: $p = .285$.

Multivariate Analysis for HDL-C Disorder

Age, hypertension, and BMI remained significantly associated with HDL-C disorder. Participants aged 31–40 years had nearly three times higher odds of HDL-C disorder compared with those aged 21–30 years (AOR = 2.72, 95% CI [1.31, 5.64], $p = .007$). Hypertension was also associated with increased odds of HDL-C disorder (AOR = 2.49, 95% CI [1.48, 4.19], $p = .001$). Overweight (AOR = 1.80, 95% CI [1.11, 2.91], $p = .008$) and obesity (AOR = 2.30, 95% CI [1.24, 4.25], $p < .001$) were significantly associated with HDL-C disorder. The Hosmer–Lemeshow test indicated adequate model fit ($p = .704$).

Table 6:
Multivariate Logistic Regression Analysis of Factors Associated With HDL-C Disorder Among *H. pylori*-Infected Participants (N = 384)

Characteristic	n (%)	AOR	95% CI	*p*
Age (years)				
21–30	52 (13.54)	1.00	Reference	—
31–40	101 (26.30)	2.72	[1.31, 5.64]	.007
41–50	130 (33.85)	2.32	[1.14, 4.70]	.019
≥ 51	101 (26.30)	2.08	[1.00, 4.31]	.049
Hypertension				
No	301 (78.39)	1.00	Reference	—
Yes	83 (21.61)	2.49	[1.48, 4.19]	.001
Body mass index (BMI)				
Normal	120 (31.25)	1.00	Reference	—
Overweight	190 (49.48)	1.80	[1.11, 2.91]	.008
Obese	72 (18.75)	2.30	[1.24, 4.25]	< .001

Note: Underweight was excluded from regression due to low frequency (n = 2). AOR = adjusted odds ratio; CI = confidence interval. Hosmer–Lemeshow test: $p = .704$.

Multivariate Analysis for Triglyceride Disorder

Hypertension remained significantly associated with triglyceride disorder (AOR = 0.56, 95% CI [0.33, 0.93], $p = .026$). The Hosmer–Lemeshow test indicated good model fit ($p = .690$).

Table 7:
Multivariate Logistic Regression Analysis of Factors Associated With Triglyceride Disorder Among *H. pylori*-Infected Participants (N = 384)

Characteristic	n (%)	AOR	95% CI	*p*
Age (years)				
21–30	52 (13.54)	1.00	Reference	—
31–40	101 (26.30)	0.92	[0.47, 1.82]	.828
41–50	130 (33.85)	1.04	[0.54, 2.00]	.888
≥ 51	101 (26.30)	1.05	[0.53, 2.08]	.867
Hypertension				
No	301 (78.39)	1.00	Reference	—
Yes	83 (21.61)	0.56	[0.33, 0.93]	.026

Note: AOR = adjusted odds ratio; CI = confidence interval. Hosmer–Lemeshow test: $p = .690$.

Multivariate Analysis for Total Cholesterol Disorder

Smoking was independently associated with total cholesterol disorder. Smokers had higher odds of total cholesterol abnormality compared with non-smokers (AOR = 1.86, 95% CI [1.01, 3.42], $p = .046$). The Hosmer–Lemeshow test suggested an adequate model fit ($p = .171$).

Table 8:
Multivariate Logistic Regression Analysis of Factors Associated With Total Cholesterol Disorder Among *H. pylori*-Infected Participants (N = 384)

Characteristic	n (%)	AOR	95% CI	*p*
Age (years)				
21–30	52 (13.54)	1.00	Reference	—
31–40	101 (26.30)	1.00	[0.41, 2.43]	.991
41–50	130 (33.85)	1.50	[0.65, 3.42]	.336
≥ 51	101 (26.30)	0.90	[0.37, 3.42]	.827
Smoking				
No	318 (82.81)	1.00	Reference	—
Yes	66 (17.19)	1.86	[1.01, 3.42]	.046

Note: AOR = adjusted odds ratio; CI = confidence interval. Hosmer–Lemeshow test: $p = .171$.

DISCUSSION

Prevalence of Dyslipidemia Among H. pylori-Infected Patients in Bunia

This study showed that among 384 participants, 208 had at least one lipid profile abnormality, corresponding to a dyslipidemia prevalence of 54.17%. The most common lipid disorder involved triglycerides, followed by HDL-C and LDL-C. Total cholesterol disorder was the least frequent.

This prevalence is lower than that reported in two Ethiopian studies. A comparative cross-sectional study conducted at the University of Gondar reported a dyslipidemia prevalence of 71.8% (95% CI [62.7, 79.7]), with LDL-C being the most common abnormality, followed by triglycerides (Nigatie et al., 2022). Another study conducted at Jimma University Medical Center reported a prevalence of 87.2% (Abdu et al., 2020). A study from Iran also reported a slightly higher prevalence (60.4%) than observed in the present study (Karim et al., 2018).

However, the prevalence observed in Bunia is higher than that reported in Fort Portal Regional Referral Hospital, Uganda (41.58%) (Kagoro et al., 2024), as well as the prevalence reported by Guzmán-Calderón (2016) (51.9%). These differences may be explained by variations in environment, dietary patterns, lifestyle, and other contextual factors.

Evidence suggests that *H. pylori* infection may contribute to dyslipidemia, and eradication therapy may reduce the risk of lipid metabolism deterioration (Park et al., 2021; Shimamoto et al., 2020; Watanabe et al., 2021).

Factors Associated With Dyslipidemia Among H. pylori-Infected Patients in Bunia

In this study, factors associated with dyslipidemia differed depending on lipid profile markers.

For LDL-C disorder, alcohol consumption was strongly associated with dyslipidemia (AOR = 9.96, 95% CI [4.56, 21.75], $p < .001$). For HDL-C disorder, age remained significantly associated ($p < .05$). In addition, hypertension was independently associated with HDL-C disorder (AOR = 2.49, 95% CI [1.48, 4.19], $p = .001$), as were overweight and obesity ($p < .05$). Regarding triglyceride disorder, hypertension remained significantly associated (AOR = 0.56, 95% CI [0.33, 0.93], $p = .026$). For total cholesterol disorder, smoking was significantly associated with abnormal total cholesterol levels (AOR = 1.86, 95% CI [1.01, 3.42], $p = .046$).

Kim et al. (2016) similarly reported that hypertension and BMI were associated with dyslipidemia among *H. pylori*-infected individuals. Their findings showed elevated LDL-

C (p < .001) and decreased HDL-C (p = .021) among infected individuals. Another study also reported that *H. pylori* infection was an independent risk factor for dyslipidemia based on multivariate logistic regression (OR = 1.13, 95% CI [1.03, 1.24], p = .006) and that infection correlated with BMI (OR = 1.13, 95% CI [1.11, 1.17], p = .001) (Suki et al., 2018).

A systematic review assessing the association between hypertension and *H. pylori* infection found that individuals infected with *H. pylori* had higher odds of hypertension (OR = 1.34, 95% CI [1.10, 1.63], p = .002, I^2 = 74%) (Fang et al., 2022).

Several studies have reported that eradication of *H. pylori* may mitigate lipid metabolism deterioration. For example, Wang et al. (2022) reported that LDL-C increased and HDL-C decreased during infection, whereas eradication was associated with an increase in HDL-C (p < .001) and a decrease in LDL-C. Shimamoto et al. (2020) also reported a negative effect of infection on HDL-C and triglyceride levels, consistent with other findings (Shimamoto et al., 2020; Yaslianifard et al., 2025).

A systematic review examining the association between *H. pylori* infection and dyslipidemia reported that infection increases LDL-C, triglycerides, and total cholesterol levels while decreasing HDL-C (Gaonkar et al., 2025). Similarly, Fu et al. (2025) reported an association between *H. pylori* infection and the triglyceride-glucose index in American adults.

Chen et al. (2019) also reported increased risk of metabolic diseases and diabetes mellitus among *H. pylori*-infected individuals. Likewise, Yaslianifard et al. (2025) highlighted the potential cardiovascular complications linked to *H. pylori* infection.

Overall, multiple studies suggest that chronic *H. pylori* infection may contribute to an atherogenic lipid profile, including increased LDL-C and triglycerides and decreased HDL-C (Basiak et al., 2022; Suki et al., 2018; Yang et al., 2024; Yaslianifard et al., 2025). This relationship may be explained by inflammation induced by *H. pylori* through cytokines such as interleukin-1 (IL-1), IL-6, and tumor necrosis factor alpha (TNF-α), which may disrupt lipid metabolism and promote atherosclerosis (Basiak et al., 2022; Yang et al., 2024; Yaslianifard et al., 2025; Yu et al., 2023).

Policy Implications

Routine lipid profile testing should be introduced for patients diagnosed with *H. pylori* infection.

Public Health Implications

Community-wide health education should be strengthened to inform both the population and healthcare professionals about the potential complications of *H. pylori* infection, given its high prevalence.

Study Strengths and Limitations

This study benefits from a relatively large sample of untreated patients with confirmed *H. pylori* infection, which reduces the likelihood of pharmacological treatment influencing lipid profile results. Additionally, the study relied on primary data, supporting data reliability. A validated diagnostic tool with high sensitivity and specificity was used, improving comparability with other studies.

However, limitations include recruitment from a single health facility, limited availability of comparable local studies within the province or country, and possible recall bias and social desirability bias related to participant self-reports.

CONCLUSION

The prevalence of dyslipidemia among *H. pylori*-infected participants in Bunia was 54.17%. Participants could present with one or multiple lipid profile abnormalities. Age, hypertension, overweight, obesity, alcohol consumption, and smoking were significantly associated with dyslipidemia in this population.

Routine lipid profile testing should be implemented for all patients diagnosed with *H. pylori* infection. In addition, community health education should be strengthened to increase awareness among the general population and healthcare professionals regarding the risk of complications, given the high prevalence of *H. pylori* infection.

These findings support the inclusion of *H. pylori* infection screening in dyslipidemia assessment strategies.

Abbreviations

HDL: High-density lipoprotein
LDL: Low-density lipoprotein
H. pylori: *Helicobacter pylori*
BMI: Body mass index

Acknowledgments: The authors sincerely thank the Cinquantenaire Hospital of Kisangani and the staff of the Department of Radiology and Medical Imaging for their support.

Ethical Approval: This study was approved by the Medical Ethics Committee of the University of Goma (UNIGOM/CEM/013/2023; October 26, 2023).

Conflicts of Interest: None declared.

ORCID iDs:

Bisingurege, K. F.[1]: https://orcid.org/0009-0003-8420-9767
Tibamwenda, B. F.[2]: https://orcid.org/0000-0001-5533-4479
Mobali, W. C.[1]: https://orcid.org/0009-0005-2422-3027
Ntanyungu, I. J.[1]: https://orcid.org/0009-0003-3086-2824
Chelo, N. H.[3]: https://orcid.org/0000-0003-4762-0218
Amuda, B. D.[4]: https://orcid.org/0000-0003-4672-0719
Nyenke, B. G.[1]: https://orcid.org/0000-0001-7058-3430
Kpane, C. F.[2]: https://orcid.org/0009-0000-6307-8049
Magapa, C. M.[4]: https://orcid.org/0009-0007-5925-7969
Mbabazi, R. J.[1]: https://orcid.org/0009-0005-5152-0406
Manwa, B. B.[5]: https://orcid.org/0009-0008-2785-7318
Batina, A. S.[6]: https://orcid.org/0000-0001-6422-7886
Tsongo, K. Z.[6]: https://orcid.org/0009-0005-1541-7524

Open Access: This original article is distributed under the Creative Commons Attribution Non-Commercial (CC BY-NC 4.0) license. This license permits people to distribute, remix, adapt, and build upon this work non-commercially and license their derivative works on different terms, provided the original work is properly cited, appropriate credit is given, any changes made are indicated, and the use is non-commercial. See: https://creativecommons.org/licenses/by-nc/4.0/.

REFERENCES

Abdu, A., Cheneke, W., Adem, M., Belete, R., & Getachew, A. (2020). Dyslipidemia and associated factors among patients suspected to have *Helicobacter pylori* infection at Jimma University Medical Center, Jimma, Ethiopia. *International Journal of General Medicine, 13*, 311–321. https://doi.org/10.2147/IJGM.S243848

Basiak, M., Kosowski, M., Hachula, M., & Okopień, B. (2022). Plasma concentrations of cytokines in patients with combined hyperlipidemia and atherosclerotic plaque before treatment initiation: A pilot study. *Medicina, 58*(5), Article 624. https://doi.org/10.3390/medicina58050624

Chen, Y.-Y., Fang, W.-H., Wang, C.-C., Kao, T.-W., Chang, Y.-W., Wu, C.-J., Zhou, Y.-C., Sun, Y.-S., & Chen, W.-L. (2019). *Helicobacter pylori* infection increases risk of incident metabolic syndrome and diabetes: A cohort study. *PLOS ONE, 14*(2), e0208913. https://doi.org/10.1371/journal.pone.0208913

Fang, Y., Xie, H., & Fan, C. (2022). Association of hypertension with *Helicobacter pylori*: A systematic review and meta-analysis. *PLOS ONE, 17*(5), e0268686. https://doi.org/10.1371/journal.pone.0268686

François, B. K., Zacharie, T. K., Salomon, B. A., Yves, B., Célestin, M. W., Herman, C. N., Floribert, K. C., & Jolie, R. (n.d.). *Infection à Helicobacter pylori: Aspect épidémio-clinique et facteurs associés dans la ville de Bunia et commune de Mahagi* [Unpublished manuscript].

Fu, W., Zhao, J., Chen, G., Lyu, L., Ding, Y., & Xu, L.-B. (2025). The association between *Helicobacter pylori* infection and triglyceride-glucose (TyG) index in U.S. adults: A retrospective cross-sectional study. *PLOS ONE, 20*(1), e0295888. https://doi.org/10.1371/journal.pone.0295888

Gaonkar, A., Zahiruddin, Q. S., Shabil, M., Menon, S. V., Kaur, M., Kumari, M., Sudan, P., Naidu, K. S., Thapliyal, S., Uikey, J., Kathuria, R., Chauhan, S. S., Verma, L., Sidhu, A., Bushi, G., Yusoff, R. B. M., Mehta, R., Satapathy, P., & Sah, S. (2025). Association of *Helicobacter pylori* infection and risk of dyslipidemia: A systematic review and meta-analysis. *JGH Open, 9*(3), e70128. https://doi.org/10.1002/jgh3.70128

Guzmán-Calderón, E. (2016). Relation between patients with gastric *Helicobacter pylori* infection and dyslipidemia. *Journal of Gastrointestinal Disorders and Liver Function, 1*(1), 1–4. https://doi.org/10.15436/2471-0601.15.004

Kagoro, F. B., Amandua, J., Abonga, C. L., Okurut, E., Nyenke, G. B., & Dana, W. (2024). Prevalence and factors associated with dyslipidemia among patients with *Helicobacter pylori* infection attending Fort Portal Regional Referral Hospital in Uganda: A cross-sectional study. *Research Square* (Preprint). https://doi.org/10.21203/rs.3.rs-4929147/v1

Karim, I., Zardari, A. K., Shaikh, M. K., Baloch, Z. A. Q., & Shah, S. Z. A. (2018). Dyslipidemia: *Helicobacter pylori*-infected patients. *The Professional Medical*

Journal, 21(5), 956–959. https://doi.org/10.29309/TPMJ/2014.21.05.2529

Kim, T. J., Lee, H., Kang, M., Kim, J. E., Choi, Y.-H., Min, Y. W., Min, B.-H., Lee, J. H., Son, H. J., Rhee, P.-L., Baek, S.-Y., Ahn, S. H., & Kim, J. J. (2016). *Helicobacter pylori* is associated with dyslipidemia but not with other risk factors of cardiovascular disease. *Scientific Reports, 6*(1), Article 38015. https://doi.org/10.1038/srep38015

Kountouras, J., Polyzos, S. A., Doulberis, M., Zeglinas, C., Artemaki, F., Vardaka, E., Deretzi, G., Giartza-Taxidou, E., Tzivras, D., Vlachaki, E., Kazakos, E., Katsinelos, P., & Mantzoros, C. S. (2018). Potential impact of *Helicobacter pylori*-related metabolic syndrome on upper and lower gastrointestinal tract oncogenesis. *Metabolism, 87*, 18–24. https://doi.org/10.1016/j.metabol.2018.06.008

Nigatie, M., Melak, T., Asmelash, D., & Worede, A. (2022). Dyslipidemia and its associated factors among *Helicobacter pylori*-infected patients attending University of Gondar Comprehensive Specialized Hospital, Gondar, northwest Ethiopia: A comparative cross-sectional study. *Journal of Multidisciplinary Healthcare, 15*, 1481–1491. https://doi.org/10.2147/JMDH.S368832

Oguejiofor, O. C., Onwukwe, C. H., & Odenigbo, C. U. (2012). Dyslipidemia in Nigeria: Prevalence and pattern. *Annals of African Medicine, 11*(4), 197–202. https://doi.org/10.4103/1596-3519.102846

Park, Y., Kim, T. J., Lee, H., Yoo, H., Sohn, I., Min, Y. W., Min, B.-H., Lee, J. H., Rhee, P.-L., & Kim, J. J. (2021). Eradication of *Helicobacter pylori* infection decreases risk for dyslipidemia: A cohort study. *Helicobacter, 26*(2), e12783. https://doi.org/10.1111/hel.12783

Pellicano, R., Ianiro, G., Fagoonee, S., Settanni, C. R., & Gasbarrini, A. (2020). Extragastric diseases and *Helicobacter pylori*. *Helicobacter, 25*(Suppl. 1), e12741. https://doi.org/10.1111/hel.12741

Quan, K., Huang, Z., Nie, S., & Li, X. (2025). The effect of *Helicobacter pylori* infection on dyslipidemia in Asia and outside Asia: A systematic review and meta-analysis. *Frontiers in Medicine, 12*, 1643218. https://doi.org/10.3389/fmed.2025.1643218

Saké, K., Ballè, M. C., Brun, L. V. C., Somitondji, N. M., Attinsounon, C. A., Adè, S., Alassani, C. A., Togbenon, L. D., Dovonou, C. A., & Akpo, M. T. A. (2023). *Helicobacter pylori* infection: Epidemiological, clinical and pathological aspects in a digestive endoscopy unit and the pathological anatomy service of Parakou in Benin Republic. *Open Journal of Gastroenterology, 13*(7), 225–236. https://doi.org/10.4236/ojgas.2023.137021

Shimamoto, T., Yamamichi, N., Gondo, K., Takahashi, Y., Takeuchi, C., Wada, R., Mitsushima, T., & Koike, K. (2020). The association of *Helicobacter pylori* infection with serum lipid profiles: An evaluation based on a combination of meta-analysis and a propensity score-based observational approach. *PLOS ONE, 15*(6), e0234433. https://doi.org/10.1371/journal.pone.0234433

Shindler-Itskovitch, T., Chodick, G., Shalev, V., & Muhsen, K. (2019). *Helicobacter pylori* infection and prevalence of stroke. *Helicobacter, 24*(1), e12553. https://doi.org/10.1111/hel.12553

Suki, M., Leibovici Weissman, Y., Boltin, D., Itskoviz, D., Tsadok Perets, T., Comaneshter, D., Cohen, A., Niv, Y., Dotan, I., Leibovitzh, H., & Levi, Z. (2018). *Helicobacter pylori* infection is positively associated with an increased BMI, irrespective of socioeconomic status and other confounders: A cohort study. *European Journal of Gastroenterology & Hepatology, 30*(2), 143–148. https://doi.org/10.1097/MEG.0000000000001014

Tsay, F. W., & Hsu, P. I. (2018). *Helicobacter pylori* infection and extra-gastroduodenal diseases. *Journal of Biomedical Science, 25*(1), Article 65. https://doi.org/10.1186/s12929-018-0469-6

Wang, Z., Wang, W., Gong, R., Yao, H., Fan, M., Zeng, J., Xu, S., & Lin, R. (2022). Eradication of *Helicobacter pylori* alleviates lipid metabolism deterioration: A large-cohort propensity score-matched analysis. *Lipids in Health and Disease, 21*(1), Article 52. https://doi.org/10.1186/s12944-022-01639-5

Watanabe, J., Hamasaki, M., & Kotani, K. (2021). The effect of *Helicobacter pylori* eradication on lipid levels: A meta-analysis. *Journal of Clinical Medicine, 10*(5), Article 904. https://doi.org/10.3390/jcm10050904

World Health Organization. (n.d.). *WHO discussion paper on obesity*.

Xie, L., Liu, G.-W., Liu, Y.-N., Li, P.-Y., Hu, X.-N., He, X.-Y., Huan, R.-B., Zhao, T.-L., & Guo, H.-J. (2024). Prevalence of *Helicobacter pylori* infection in China from 2014–2023: A systematic review and meta-analysis. *World Journal of Gastroenterology, 30*(43), 4636–4656. https://doi.org/10.3748/wjg.v30.i43.4636

Yang, C., You, N., Chen, Y., & Zhang, J. (2024). *Helicobacter pylori* infection increases the risk of dyslipidemia in a Chinese diabetic population: A retrospective cross-sectional study. *BMC Infectious Diseases, 24*(1), Article 730. https://doi.org/10.1186/s12879-024-09597-2

Yang, X.-T., Niu, P.-Q., Li, X.-F., Sun, M.-M., Wei, W., Chen, Y.-Q., & Zheng, J.-Y. (2024). Differential cytokine expression in gastric tissues highlights *Helicobacter pylori*'s role in gastritis. *Scientific Reports, 14*(1), Article 7683. https://doi.org/10.1038/s41598-024-58407-x

Yaslianifard, S., Sameni, F., Kazemi, K., Atefpour, Y., Hajikhani, B., Baradaran Bagheri, A., Yazdani, S., & Dadashi, M. (2025). Beyond the gut: A comprehensive meta-analysis on *Helicobacter pylori* infection and cardiovascular complications. *Annals of Clinical Microbiology and Antimicrobials, 24*(1), Article 18. https://doi.org/10.1186/s12941-025-00788-6

Yu, B., Xiang, L., Peppelenbosch, M. P., & Fuhler, G. M. (2023). Overlapping cytokines in *H. pylori* infection and gastric cancer: A tandem meta-analysis. *Frontiers in Immunology, 14*, 1125658. https://doi.org/10.3389/fimmu.2023.1125658