Kirkuk Journal of Medical Sciences

ORIGINAL ARTICLE

Association between *Helicobacter pylori* Stool Antigen Positivity and Type 2 Diabetes Mellitus among Patients in Kirkuk City

Burhan Ahmed Mohammed [1], Summer Saad Abdulhussain [2,*], Osama Hasan Othman [3], Saman Serwan Kaka Abdullah [4]

[1]*Department of Medical Laboratory Techniques, College of Medical Technology, Al-Kitab University, Kirkuk, Iraq*
[2]*Department of Medicine, College of Medicine, University of Kirkuk, Kirkuk, Iraq*
[3]*Department of Medical Laboratory Techniques, College of Medical Technology, Al-Kitab University, Kirkuk, Iraq*
[4]*Department of Medicine, Northampton General Hospital, Northampton, United Kingdom*

[*]*Corresponding author email: summers.a@uokirkuk.edu.iq*

Received: 20 January 2026
Accepted: 28 May 2026
Published online: 12 June 2026

How to cite this article:

Mohammed BA, Abdulhussain SS, Othman OH, Abdullah SSK. Association between *Helicobacter pylori* stool antigen positivity and type 2 diabetes mellitus among patients in Kirkuk City. *Kirkuk Journal of Medical Sciences*. 2026;14(1):80–85.
DOI: 10.32894/kjms.2026.168820.1284

ABSTRACT

Background: *Helicobacter pylori* infection is one of the most prevalent chronic bacterial infections. Several studies suggest a possible association between *H. pylori* infection and type 2 diabetes mellitus (T2DM), potentially mediated by chronic inflammation and metabolic dysregulation. This study aimed to evaluate the association between T2DM and active *H. pylori* infection among patients in Kirkuk City, Iraq.

Methods: This cross-sectional study was conducted at the Consulting Clinic of Azadi Teaching Hospital, Kirkuk, Iraq, between April 2022 and January 2023. A total of 185 participants aged 18–77 years were enrolled, including 112 patients with T2DM and 73 non-diabetic individuals. *H. pylori* infection was detected using a stool antigen immunochromatographic test. Data were analyzed using IBM SPSS Statistics version 26. Categorical variables were compared using Fisher's exact test, and crude odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. Stratified analyses were performed according to gender, age group, and smoking status.

Results: The mean age of participants was 48.6 ± 13.2 years. *H. pylori* positivity was observed in 82/112 (73.2%) patients with T2DM and 38/73 (52.1%) non-diabetic individuals. The crude OR for *H. pylori* infection among T2DM patients compared with non-diabetic participants was 2.52 (95% CI: 1.35–4.69; P = 0.004). Stratified analyses showed a generally consistent direction of association across subgroups, although statistical significance was not observed in all strata. Only crude ORs were calculated, and no adjusted analysis was performed. Therefore, the results may be affected by confounding factors and cannot establish an independent association.

Conclusion: Stool antigen-confirmed *H. pylori* infection was more prevalent among patients with T2DM than among non-diabetic participants in this cross-sectional study.

Key words: *Helicobacter pylori*; Type 2 diabetes mellitus; Stool antigen test; Iraq

ISSN: 2790-0207 (Print), 2790-0215 (Online).

INTRODUCTION

Helicobacter pylori (*H. pylori*) is a short, spiral-shaped, Gram-negative, microaerophilic bacterium that primarily colonizes the gastric antral mucosa. It can cause gastric mucosal changes ranging from mild inflammation to malignancy and is one of the most prevalent pathogens worldwide [1]. Chronic *H. pylori* infection is recognized as a major risk factor for gastritis, gastric ulcer, duodenal ulcer, gastric adenocarcinoma, and gastric mucosa-associated lymphoid tissue lymphoma [2]. Several extra-gastric conditions, including iron deficiency anemia, immune thrombocytopenic purpura, megaloblastic anemia, diabetes mellitus, cardiovascular diseases, and certain neurological disorders, have also been linked to *H. pylori* infection [3].

Several studies have reported a higher prevalence of *H. pylori* infection among people with T2DM than among non-diabetic individuals [1]. T2DM is a chronic metabolic disorder characterized by elevated blood glucose levels due to insulin resistance and relative insulin deficiency. In contrast, a non-diabetic state is defined by normal fasting blood glucose and HbA1c levels, indicating adequate insulin function and maintained glucose homeostasis [4]. Approximately half of the global population is estimated to be infected with *H. pylori*; however, prevalence differs between countries and is generally higher in developing regions [5]. *H. pylori* infection and diabetes mellitus are two common health conditions, and *H. pylori* infection has been described as one of the common infectious problems associated with diabetes, particularly in patients with gastric symptoms [6].

Epidemiological studies have suggested an association between *H. pylori* infection, insulin resistance, and metabolic syndrome [7, 8]. *H. pylori* may disrupt glucose and lipid metabolism, which are often already impaired in individuals with diabetes mellitus, particularly those with poor metabolic control [9]. The possible relationship between *H. pylori* prevalence and diabetes has therefore attracted increasing research interest [10]. However, findings regarding the prevalence of *H. pylori* among diabetic patients remain conflicting and inconsistent [6]. Some studies have not found a correlation between *H. pylori* infection and insulin resistance or metabolic syndrome [11].

Stool antigen testing can identify active *H. pylori* infection, unlike serological tests, which cannot reliably distinguish current from past infection [12]. Evidence regarding the association between active *H. pylori* infection and T2DM remains inconsistent because of differences in study design, population characteristics, and diagnostic methods. In addition, local data from Kirkuk are limited. Therefore, this study aimed to assess the association between stool antigen-confirmed *H. pylori* infection and T2DM among patients attending Azadi Teaching Hospital in Kirkuk City.

MATERIALS AND METHODS

Study design and setting

This cross-sectional analytical study was conducted at the Consulting Clinic of Azadi Teaching Hospital, Kirkuk, Iraq, between April 2022 and January 2023.

Participants

A total of 185 participants aged 18–77 years were included in the study, comprising 112 patients diagnosed with T2DM and 73 non-diabetic individuals. The diagnosis of T2DM was based on World Health Organization criteria, including fasting plasma glucose $\geq$ 126 mg/dL, 2-hour postprandial glucose $\geq$ 200 mg/dL, or glycated hemoglobin (HbA1c) $\geq$ 6.5%.

Inclusion and exclusion criteria

The inclusion criteria were age between 18 and 77 years, confirmed diagnosis of T2DM for the case group, and normal fasting blood glucose, postprandial glucose, and HbA1c levels for the non-diabetic group. Participants were excluded if they had antibiotic use within 4 weeks, proton pump inhibitor use within the previous 2 weeks, bismuth compound use within 4 weeks, previous *H. pylori* eradication therapy, pregnancy, immunosuppressive conditions, major gastrointestinal malignancy, or severe systemic illness affecting immune status.

Stool specimen collection and *H. pylori* testing

H. pylori infection was assessed using a rapid stool antigen immunochromatographic assay manufactured by Spectrum Diagnostics, Egyptian Company for Biotechnology, Obour City Industrial Area, Cairo, Egypt, and performed according to the manufacturer's instructions. Approximately 1–2 g of stool was collected from each participant in a clean, dry, leak-proof container. Whenever possible, specimens were tested within 6 hours of collection. If testing was delayed, specimens were stored at 2–8 °C for up to 3 days; for longer storage, specimens were kept below −20 °C. The assay was used to detect active *H. pylori* infection. Participants were recruited using convenience sampling during the study period. Because individual-level data on several potential confounders were unavailable, adjustment for BMI, socioeconomic status, residence, gastrointestinal symptoms, diabetes duration, glycemic control, HbA1c level, and medication use could not be performed; therefore, residual confounding cannot be excluded.

Statistical analysis

Data were entered and analyzed using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA). Categorical variables were summarized as frequencies and percentages, whereas continuous variables were expressed as mean ± standard deviation (SD). The distribution of age was assessed using the Shapiro–Wilk test and was approximately normal. The association between T2DM and *H. pylori* infection was evaluated using Fisher's exact test. Crude odds ratios (ORs) with corresponding 95% confidence intervals (CIs) were calculated to estimate the strength of association.

Stratified analyses were conducted according to gender, age group, and smoking status to assess the consistency of the association across subgroups. These analyses were considered exploratory, and no adjustment for multiple comparisons was performed; therefore, subgroup-specific *P*-values should be interpreted cautiously. A multivariable logistic regression model was initially planned to adjust for potential confounders, including age, sex, and smoking status. However, this analysis could not be performed because aggregated data rather than individual-level observations were used. Accordingly, only crude associations are reported. Given the relatively high prevalence of *H. pylori* infection in the study population, the reported ORs may overestimate the magnitude of association and are interpreted with caution. As this was a cross-sectional study, outcomes are reported as prevalence or positivity rates rather than incidence. A *P*-value < 0.05 was considered statistically significant.

RESULTS

Overall association between T2DM and *Helicobacter pylori*

A total of 185 participants were included in the study, comprising 112 patients with T2DM and 73 non-diabetic individuals. The mean age of participants was 48.6 ± 13.2 years (range: 18–77 years). The mean age was higher among T2DM patients (52.1 ± 11.4 years) than among non-diabetic participants (43.3 ± 13.8 years).

Overall analysis showed that the prevalence of *H. pylori* infection was higher among T2DM patients than among non-diabetic individuals. Specifically, 82 of 112 (73.2%) T2DM patients tested positive for *H. pylori*, compared with 38 of 73 (52.1%) non-diabetic participants. The crude OR for *H. pylori* infection among T2DM patients compared with non-diabetic participants was 2.52 (95% CI: 1.35–4.69; $P = 0.004$), indicating a positive crude association between T2DM and *H. pylori* infection.

Stratified analysis by gender

The association between T2DM and *H. pylori* infection stratified by gender is presented in Table 1. Among males, 42 of 60 (70.0%) T2DM patients were positive for *H. pylori*, compared with 15 of 37 (40.5%) non-diabetic males, yielding an OR of 3.42 (95% CI: 1.49–7.60; $P = 0.005$). Among females, 40 of 52 (76.9%) T2DM patients were positive, compared with 23 of 36 (63.9%) non-diabetic females, yielding an OR of 1.88 (95% CI: 0.75–4.87; $P = 0.231$). These subgroup-specific estimates represent the association between T2DM and *H. pylori* infection within each gender stratum and do not indicate an independent effect of gender.

Stratified analysis by age group

Age-stratified results are presented in Table 2. The prevalence of *H. pylori* infection among T2DM patients compared with non-diabetic individuals was 29/41 (70.7%) versus 18/33 (54.5%) in the 18–37-year age group (OR = 2.01, 95% CI: 0.78–5.53; $P = 0.224$), 36/46 (78.3%) versus 16/28 (57.1%) in the 38–57-year age group (OR = 2.70, 95% CI: 0.91–7.37; $P = 0.069$), and 17/25 (68.0%) versus 4/12 (33.3%) in the 58–77-year age group (OR = 4.25, 95% CI: 0.91–15.11; $P = 0.076$). Although higher odds were observed in older age strata, the associations did not reach statistical significance within individual strata. These age categories were used for stratified descriptive analysis only and do not represent independent or adjusted effects.

Stratified analysis by smoking status

The association between T2DM and *H. pylori* infection stratified by smoking status is presented in Table 3. Among smokers, 31 of 43 (72.1%) T2DM patients were positive for *H. pylori*, compared with 15 of 25 (60.0%) non-diabetic smokers, yielding an OR of 1.72 (95% CI: 0.62–4.67; $P = 0.420$). Among non-smokers, 51 of 69 (73.9%) T2DM patients were positive, compared with 23 of 48 (47.9%) non-diabetic individuals, yielding an OR of 3.08 (95% CI: 1.42–6.46; $P = 0.006$). The observed association within subgroups reflects the relationship between T2DM and *H. pylori* infection within each smoking category and does not indicate an independent effect of smoking.

Table 1. Association between T2DM and *H. pylori* infection stratified by gender.

Gender	Group	*H. pylori* positive, n (%)	*H. pylori* negative, n (%)	OR (95% CI)	*P*-value
Male	T2DM (n = 60)	42 (70.0)	18 (30.0)	3.42 (1.49–7.60)	0.005
	Non-DM (n = 37)	15 (40.5)	22 (59.5)	Reference	—
Female	T2DM (n = 52)	40 (76.9)	12 (23.1)	1.88 (0.75–4.87)	0.231
	Non-DM (n = 36)	23 (63.9)	13 (36.1)	Reference	—

Values are presented as row percentages within each gender stratum. Odds ratios compare T2DM with non-diabetic individuals within each gender stratum using Fisher's exact test. CI, confidence interval; OR, odds ratio; T2DM, type 2 diabetes mellitus.

Table 2. Association between T2DM and *H. pylori* infection stratified by age group.

Age group (years)	Group	*H. pylori* positive, n (%)	*H. pylori* negative, n (%)	OR (95% CI)	*P*-value
18–37	T2DM (n = 41)	29 (70.7)	12 (29.3)	2.01 (0.78–5.53)	0.224
	Non-DM (n = 33)	18 (54.5)	15 (45.5)	Reference	—
38–57	T2DM (n = 46)	36 (78.3)	10 (21.7)	2.70 (0.91–7.37)	0.069
	Non-DM (n = 28)	16 (57.1)	12 (42.9)	Reference	—
58–77	T2DM (n = 25)	17 (68.0)	8 (32.0)	4.25 (0.91–15.11)	0.076
	Non-DM (n = 12)	4 (33.3)	8 (66.7)	Reference	—

Values are presented as row percentages within each age group. Odds ratios compare T2DM with non-diabetic individuals within each age stratum using Fisher's exact test. CI, confidence interval; OR, odds ratio.

Table 3. Association between T2DM and *H. pylori* infection stratified by smoking status.

Smoking status	Group	*H. pylori* positive, n (%)	*H. pylori* negative, n (%)	OR (95% CI)	*P*-value
Smoker	T2DM (n = 43)	31 (72.1)	12 (27.9)	1.72 (0.62–4.67)	0.420
	Non-DM (n = 25)	15 (60.0)	10 (40.0)	Reference	—
Non-smoker	T2DM (n = 69)	51 (73.9)	18 (26.1)	3.08 (1.42–6.46)	0.006
	Non-DM (n = 48)	23 (47.9)	25 (52.1)	Reference	—

Values are presented as row percentages within each smoking category. Odds ratios compare T2DM with non-diabetic individuals within each smoking stratum using Fisher's exact test. CI, confidence interval; OR, odds ratio.

DISCUSSION

The present study found a statistically significant crude association between T2DM and *H. pylori* infection among patients in Kirkuk City. The prevalence of *H. pylori* infection was higher in individuals with T2DM (73.2%) than in non-diabetic participants (52.1%), with a crude OR of 2.52 (95% CI: 1.35–4.69). These findings indicate a positive association between T2DM and *H. pylori* infection. This observation is consistent with previous studies that reported a higher prevalence of *H. pylori* infection among patients with T2DM [13].

Several biological mechanisms may explain this relationship. Impaired immune function in individuals with diabetes, including reduced neutrophil activity and altered cytokine responses, may increase susceptibility to persistent infection [14]. In addition, delayed gastric emptying and autonomic neuropathy associated with T2DM may facilitate *H. pylori* colonization. Chronic low-grade inflammation in diabetes may further contribute to a favorable environment for bacterial persistence [15].

Stratified analyses by gender, age group, and smoking status were performed to explore the consistency of the association across subgroups. The direction of association between T2DM and *H. pylori* infection was generally consistent across these strata. The findings of the present study align with those reported in a study conducted in Kirkuk, Iraq [16]. In the present study, *H. pylori* positivity among T2DM patients was 76.9% in females and 70.0% in males. This finding is broadly comparable with research conducted in Erbil, Iraq [17]. In addition, a recent updated meta-analysis of 45 case-control studies reported a significant association between *H. pylori* infection and diabetes mellitus [18].

The age-stratified analysis showed higher crude odds of *H. pylori* positivity among T2DM patients than among non-diabetic individuals in all age groups, although the subgroup associations did not reach statistical significance. These findings should be interpreted cautiously because the subgroup analyses were exploratory and limited by small sample sizes. The observed variation in statistical significance across subgroups may reflect differences in sample size and statistical power rather than true effect modification.

Smoking status was evaluated only descriptively in this study, and the stratified findings should not be interpreted as evidence of an independent smoking effect because no adjusted model was performed. The significant finding within the non-smoker subgroup reflects the association between T2DM and *H. pylori* infection within that subgroup, rather than a direct ef-

fect of smoking. These findings are consistent with research from Saudi Arabia that reported no significant association between *H. pylori* prevalence and smoking status [19]. In a single-center Romanian endoscopic study, *H. pylori* infection was more frequently observed among patients with T2DM than among non-diabetic controls, although the difference did not reach statistical significance. The same study also reported that smoking was significantly associated with a higher frequency of *H. pylori* infection [20].

Given that *H. pylori* infection was relatively common in this study population, ORs may overestimate the strength of association compared with prevalence ratios. Therefore, the reported crude OR of 2.52 should be interpreted as indicating a positive association rather than a direct estimate of relative risk. Several limitations should be acknowledged. First, the cross-sectional design precludes establishing temporal or causal relationships between T2DM and *H. pylori* infection. Second, convenience sampling may limit the generalizability of the findings and introduce selection bias. Third, the analysis was based on aggregated data, which prevented multivariable regression and adjustment for potential confounding variables such as age, sex, and smoking status. As a result, the findings represent crude associations and may be influenced by residual confounding. Finally, although stool antigen testing is a useful method for detecting active infection, some degree of misclassification cannot be completely excluded.

Despite these limitations, the study provides local evidence from Kirkuk City, where data on the association between T2DM and *H. pylori* infection are limited. The use of a stool antigen test for active infection and the inclusion of both diabetic and non-diabetic participants strengthen the relevance of the findings.

CONCLUSION

Stool antigen-confirmed *H. pylori* infection was significantly more prevalent among patients with T2DM than among non-diabetic participants in Kirkuk City. These findings suggest a possible association between T2DM and active *H. pylori* infection; however, due to the cross-sectional design and the absence of adjusted analysis, a causal or independent relationship cannot be established. Further large-scale prospective studies, with appropriate adjustment for socioeconomic, metabolic, lifestyle, and gastrointestinal factors, are recommended to confirm this association and clarify its potential causal mechanisms.

ETHICAL DECLARATIONS

Ethics Approval and Consent to Participate

The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Ethics Committee of the College of Medicine, University of Kirkuk, Iraq (Approval Issue No. 16 B; date: 6 April 2022). Written informed consent was obtained from all participants.

Consent for Publication

Not applicable.

Availability of Data and Material

The datasets are available from the corresponding author upon reasonable request.

Competing Interests

The authors declare that they have no competing interests, financial or otherwise, that could be perceived as influencing the work reported in this manuscript.

Funding

Self-funded.

Use of Generative Artificial Intelligence

The authors declare that ChatGPT, developed by OpenAI, was used only during the final stage for language editing and grammar improvement. It was not used for data creation, data analysis, or interpretation of the results.

Authors' Contributions

All authors contributed to the literature review, study design, data collection, statistical analysis, and manuscript preparation. All authors read and approved the final version of the manuscript.

REFERENCES

[1] Buzás GM, Birinyi P, Newer, older, and alternative agents for the eradication of Helicobacter pylori infection: a narrative review. *Antibiotics* 2023;12(6):946. https://doi.org/10.3390/antibiotics12060946

[2] Liu D, Ren L, Zhong D, Zhang W, Li WW, Liu J, et al., Association of serum vitamin D levels on Helicobacter pylori infection: a retrospective study with real-world

data. *BMC gastroenterology* 2023;23:391. https://doi.org/10.1186/s12876-023-03037-2

[3] Elbehiry A, Marzouk E, Aldubaib M, Abalkhail A, Anagreyyah S, Anajirih N, et al., Helicobacter pylori infection: current status and future prospects on diagnostic, therapeutic and control challenges. *Antibiotics* 2023;12(2):191. https://doi.org/10.3390/antibiotics12020191

[4] Młynarska E, Czarnik W, Dzieża N, Jędraszak W, Majchrowicz G, Prusinowski F, et al., Type 2 diabetes mellitus: new pathogenetic mechanisms, treatment and the most important complications. *International journal of molecular sciences* 2025;26(3):1094. https://doi.org/10.3390/ijms26031094

[5] Mladenova I, Clinical relevance of Helicobacter pylori infection. *Journal of Clinical Medicine* 2021;10(16):3473. https://doi.org/10.3390/jcm10163473

[6] Sahoo OS, Mitra R, Bhattacharjee A, Kar S, Mukherjee O, Is diabetes Mellitus a Predisposing factor for Helicobacter pylori infections? *Current diabetes reports* 2023;23:195–205. https://doi.org/10.1007/s11892-023-01511-5

[7] Yadav S, Singh P, Singh N, Verma S, Singh S, Helicobacter pylori infection: the extragastric manifestations - a review. *International Journal of Health Sciences and Research* 2024;14(7):145–153. https://doi.org/10.52403/ijhsr.20240718

[8] Moalim NB, Ali AH, Elsheikh EG, Ali DB, Adam AA, Association between Helicobacter pylori and diabetes mellitus. *Journal of Drug Delivery and Therapeutics* 2023;13(11):102–106. http://dx.doi.org/10.22270/jddt.v13i11.6008

[9] Baryshnikova NV, Ermolenko EI, Leontieva GF, Uspenskiy YP, Suvorov AN, Helicobacter pylori infection and metabolic syndrome. *Exploration of Digestive Diseases* 2024;3:414–427. https://doi.org/10.37349/edd.2024.00058

[10] Banavre F, Karimian P, Motamed B, Ebrahim-Saraie HS, Impact of Helicobacter pylori infection on diabetes: A cross-sectional investigation in Northern Iran. *Journal of Current Biomedical Reports* 2025;6(2):36–40. https://doi.org/10.61882/jcbior.6.2.318

[11] Ye J, Feng T, Su L, Li J, Gong Y, Ma X, Interactions between Helicobacter pylori infection and host metabolic homeostasis: A comprehensive review. *Helicobacter* 2023;28(6):e13030. https://doi.org/10.1111/hel.13030

[12] Kang X, Gou L, Chen X, Wang Y, Liu Y, Zhang D, Application value and performance of stool antigen detection in the diagnosis of Helicobacter pylori infection. *European Journal of Clinical Microbiology & Infectious Diseases* 2026;45(1):69–77. https://doi.org/10.1007/s10096-025-05281-8

[13] Hafiz Q, Ikram O, Zia M, Theba F, Ikram N, Tariq A, Helicobacter pylori infection among type 2 diabetics: A case control study. *International Journal of Research in Medical Sciences* 2020;8(3):1047–1050. http://dx.doi.org/10.18203/2320-6012.ijrms20200779

[14] Dilworth L, Facey A, Omoruyi F, Diabetes mellitus and its metabolic complications: the role of adipose tissues. *International journal of molecular sciences* 2021;22(14):7644. https://doi.org/10.3390/ijms22147644

[15] Gharavi M, Zarei J, Roshani Asl P, Yazdanyar Z, Sharif M, Rashidi N, Determination of Helicobacter pylori infection prevalence by non-invasive methods. *Avicenna Journal of Clinical Microbiology and Infection* 2022;9(2):55–62. https://doi.org/10.34172/ajcmi.2022.09

[16] Sulyman MA, Kadir MAA, Tahir SS, Correlation between Helicobacter pylori and type 2 diabetes in Kirkuk-Iraq. *World Bulletin of Public Health* 2022;9:24–29. https://www.scholarexpress.net/index.php/wbph/article/view/772

[17] Taha AB, Helicobacter pylori infection associated with type 2 diabetes: a case–control study. *ARO-The scientific Journal of Koya University* 2024;12(2):229–233. https://doi.org/10.14500/aro.11824

[18] Hadianfard H, Darehbagh RR, Ahmadpooryan K, Mohamadi P, Moradi Y, The association between Helicobacter pylori infection and diabetes mellitus: an updated meta-analysis of 45 case-control studies. *Journal of Health, Population and Nutrition* 2025;44(1):330. https://doi.org/10.1186/s41043-025-01088-7

[19] Darraj MA, Eradication rate and factors influencing Helicobacter pylori infection clearance using standard triple therapy at a single centre in Jazan Region, Saudi Arabia: a retrospective study. *International Journal of General Medicine* 2024;17:2627–2634. https://doi.org/10.2147/IJGM.S456865

[20] Munteanu SN, Huțanu D, Filip AM, Cozac-Szőke AR, Mocan S, Negovan A, Type 2 Diabetes Mellitus and Helicobacter pylori Gastritis in Patients Referred for Endoscopy—A Single-Center Romanian Study. *Life* 2024;14(9):1160. https://doi.org/10.3390/life14091160