Meta Analysis
Sample Work

A meta-analysis of the relationship between Helicobacter Pylori infection and ABO blood groups

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Abstract

This meta-analysis aimed to evaluate the association between ABO blood groups and the risk of Helicobacter pylori infection. Epidemiological studies were retrieved from MEDLINE/PubMed databases, and pooled effects estimates were obtained using fixed and random effects meta-analyses. Results showed that individuals with the O blood group were more likely to be infected with H. pylori (OR 1.163; 95% confidence interval (CI) 1.074-1.259; P < 0.001). Individuals with B and AB blood groups were less likely to be infected (OR 0.831; 95% CI 0.738-0.935; P = 0.002 and OR 0.709; 95% CI 0.605-0.832; P < 0.001, respectively).

The results suggest an estimated 16.3% increased odds of H. pylori infection among individuals with the O blood group. A better understanding of the underlying mechanisms could indicate potential prevention strategies for H. pylori infection if this association is causal.

Introduction

The human stomach mucosa is home to the Gram-negative spiral-shaped harmful bacteria known as Helicobacter pylori. Although the bacteria is prevalent in around 50% of the world's population (1, 2), only 10-15% of infected people develop symptoms, including peptic ulcer disease and stomach cancer (3, 4). The World Health Organization's (WHO) International Agency for Research on Cancer (IARC) classified H. pylori as a class I carcinogen in 1994 and identified it as the primary risk factor for stomach cancer (5). Knowing who is susceptible to infection and sickness is crucial, particularly in light of the rising prevalence of strains that are resistant to antibiotics and the absence of a reliable vaccination (6).

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Close human contact is necessary, yet the processes by which H. pylori is often acquired and its transmission mode are still unknown. Previous epidemiological studies demonstrated that poor living circumstances and home hygiene practices are significant risk factors for H. pylori infection (7, 8). Socioeconomic status was determined by occupation, family income level, and occupation. These elements are believed to partially account for the differences in H. pylori infection rates between different populations. A recent investigation showed that the prevalence of H. pylori infection was significantly greater in the sexual partners of H. pylori-infected people than in controls (9).

The literature on risk factors for H. pylori infection has mainly focused on environmental and lifestyle factors like smoking and diet (10). However, recent evidence suggests that genetic factors may also influence susceptibility. ABO blood group, a genetically determined trait with polymorphic expression, has been identified as a potential risk factor for H. pylori infection(11). Studies have shown a higher frequency of blood group O among patients with duodenal ulcers, suggesting that blood group O might also be a risk factor for acquiring H. pylori infection(12). ABO blood groups have also been investigated as risk factors for H. pylori-associated gastric cancer, but conflicting studies have been found due to multiple confounding effects(13). Since the discovery of the ABO blood group, there has been an ongoing interest in the potential role of blood groups in infectious diseases(14). Blood group antigens are receptors for toxins, parasites, and bacteria, facilitating colonization, invasion, or evading host clearance mechanisms(15-17). Previous studies have demonstrated that blood-group antigen-binding adhesion (BabA) mediates H. pylori adherence to human Lewisb blood-group antigens on gastric epithelial cells.

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Over the past few decades, animal model studies and epidemiological data have provided evidence for the possible risk factors for H. pylori infection that ABO groups may play (18–20). While some studies identified a favourable link between H. pylori infection and the O blood type (18, 21), others (19, 20, 22, 23) found no such relationship.

A well-known technique, meta-analysis, combines the data from smaller, inconclusive research to increase statistical power (24). The present systematic review and meta-analysis of the pertinent epidemiologic literature is being conducted to measure the relationship between the ABO blood group and H. pylori infection status.

Methods

Literature search and study selection

The MEDLINE/PubMed databases (up to October 2017) were searched for pertinent epidemiological studies using thorough inclusion and exclusion criteria. The studies have to meet the following criteria: ABO blood groups were identified using the slide method, tube test, microplate technology, column/gel centrifugation, surface imprinting of erythrocytes, use of synthetic and natural receptors for ABO blood group sensors, chromatography and filtration paper-based diagnostics, and any other molecular blood typing methods, (i) published in English; (ii) cross-sectional, cohort, case-control study, or randomized controlled trial; and (iii) any other mole (iv) H. pylori infection was diagnosed by the use of a stool antigen test, serum H. pylori-specific IgG utilizing ELISA, a 13C- or 14C-labelled urea breath test, an antral biopsy urease test, microbiological culture techniques, histological identification of organisms, hematoxylin and eosin staining, and hematoxylin and eosin stain or Giemsa staining. Reviews, letters, correspondences, editorials, and case reports were excluded from the analysis. Two researchers (ZC and BT) used three steps to independently assess the studies' suitability: title review, paper abstract review, and full-text review. To identify fresh competent works, references were also examined. The EndNote X7 program (Thomson Reuters Corporation, New York, NY, USA) was used to import pertinent documents. Documents were used to support the exclusion of papers.

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Study quality assessment

The Newcastle-Ottawa quality assessment scale (NOS) was developed and used to assess the quality of cross-sectional research. (25). Three elements were considered When generating a quality score: research group selection, group comparability, and exposure and result assessment. For cross-sectional and cohort studies, a score of 7 or above out of 10 was judged high quality.
Check out our study guide to learn more about Meta-Analysis.How to do a meta-analysis for a manuscript?

Statistical analysis

CMA (version 3) was used to examine reported odds ratios (ORs) and 95% confidence intervals (CIs) for the relationship between ABO blood types and H. pylori infection status. ORs and CIs for studies with published raw data had to be calculated. For the primary analysis, ABO blood group results were employed. In all trials, the relative risk was expressed as an OR and adjusted ORs were preferred over crude ORs. A random study effects model with heterogeneity was utilized to integrate individual impact estimates. To assess heterogeneity, Cochran Q and I2 statistics were utilized. Cochran Q was found statistically significant for heterogeneity with a P value of 0.10. A result for I2 of more than 50% exhibited considerable heterogeneity (26). To analyze publication/selection bias, funnel plots of research ORs vs the standard error logarithm of these ORs were utilized. The degree of asymmetry was determined using Egger's regression asymmetry test (27). Individual studies' impact and influence on total pooled results were assessed using sensitivity analysis (28, 29).

Following the primary analysis, subgroup analyses were conducted based on research quality (poor or high), study design, and study area (developed and developing). The current meta-analysis and review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (30) and Meta-analyses of Observational Studies in Epidemiology (MOOSE) (31).

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Results

Study characteristics

A meta-analysis was conducted on 2222 potentially relevant studies, identifying 30 eligible studies. Most of the studies were cross-sectional, with two cohorts and one randomized control trial. Most of the studies included adults aged 18-74, with only two involving children under 15. The majority of the studies used enzyme-linked immunosorbent assay (ELISA) in ten, histological analysis of hematoxylin and eosin in two, urea breath test in four, and a combination of ELISA, antral biopsy urease test, and Giemsa staining in two. The remaining 12 studies used various other techniques to define H.pylori. Most studies determined ABO type via serum hemagglutination methods, widely used in large-scale blood banking (Fig. 1).

Quality assessment

Of the 30 qualifying studies, 26 had better methodological quality (scoring 7), and three were deemed lower quality due to inadequate characterization of control groups, failure to publish response rates or a lack of correction for confounding factors. The median overall score was 7 out of 8, indicating that the studies were typically quality.

Meta-analysis of the association between O Blood groups and H. pylori infection

A meta-analysis of 30 studies found an increased odds of H. pylori infection in the O blood group compared to non-O blood groups (OR; 1.163; 95% CI; 1.074-1.259, $P < 0.001$). Sixteen of the 30 studies independently reported an OR greater than or equal to 1. However, substantial heterogeneity in the estimated effect size between the studies suggested that systematic effect size variability was unaccounted for (Figure 2). Study quality, design, and participant characteristics may have moderated the results. Subgroup analyses were conducted to determine how these variables moderated the overall results. The O blood group was also significantly associated with H. pylori infection when the data pool was restricted to seventeen studies conducted in a developing country. The pooled OR for twelve studies conducted in a developed country was weaker than statistically significant. Findings did not significantly change when the analysis was concentrated on the 26 high-quality studies.

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The pooled OR from the 27 cross-sectional studies was 1.185 (95% CI; 1.091 - 1.286; P 0.01) (Fig. S6). Because no significant departures from symmetry were found in the funnel plot (Fig. 3), publication bias did not appear to play a role in the connection between blood group O and H. pylori infection (Fig. 3). The Egger regression asymmetry test found no statistically significant publishing biases (b = 0.802, P = 0.486). Furthermore, the robustness of the observed outcome was explored by eliminating each study and re-analyzing the data sets sequentially, and no study was found to significantly change the pooled OR estimate (Online supplement Fig. S1).

A meta-analysis evaluating the relationship between the B blood group and H. pylori infection

A meta-analysis of 27 studies on the relationship between B blood type and H. pylori infection (Figure 4) revealed a somewhat lower risk of infection (OR=0.831 (95% CI; 0.738-0.935, p=0.002). Eighteen of the 27 studies independently reported an OR smaller than one, indicating significant heterogeneity (I2 = 62.31). A subgroup analysis of sixteen studies conducted in underdeveloped countries yielded a summary risk estimate of 0.741 (95% CI 0.633 - 0.868, P 0.001, Fig. S7). With 23 high-quality studies (OR = 0.850, 95% CI 0.752 - 0.959; P = 0.009; Fig. S8) and 24 cross-sectional studies (OR = 0.819, 95% CI 0.723 - 0.927; P = 0.002; Fig. S9), further stratified analysis by study quality and study type did not significantly affect the magnitude of the pooled estimate. Both visual inspections of the funnel plot (Fig. 5) and the results of Egger's test (Egger's test; b = -0.221, P = 0.792) revealed no indication of publication bias. Furthermore, a sensitivity analysis was performed by eliminating each research and re-analyzing the data sets, yielding almost comparable risk estimates (Online supplement Fig. S2).

Meta-analysis of the association between AB Blood group and H. pylori infection

There was considerable study-to-study heterogeneity (I2 = 49.2%) and a lowered risk of H. pylori infection, according to the meta-analysis of data from 27 research on the relationship between the AB blood type and H. pylori infection (Figure 6). The findings from the subgroup analyses based on different methodological criteria did not materially alter the size of pooled estimates (OR:0. 601 (95% CI 0.492 - 0.735, P < 0.001; 16 studies from developing country; Fig. S10, OR = 0.692, 95% CI 0.586 - 0.816; P < 0.01 in 23 high quality studies; Fig. S11, and OR=0.697; 95% CI; 0.591 - 0.822; P < 0.01 in 24 cross sectional studies Fig. S12). The funnel plot (Fig. 7) did not reveal any notable departures from symmetry that would indicate indications of publication bias.

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The Egger regression asymmetry test revealed no statistically significant publication bias (b = 0.775, P = 0.172). By systematically deleting each study and re-analyzing the data sets, the robustness of the observed outcome was examined, and no significant change was seen in the pooled OR estimate by any one study (Online supplement Fig. S3).

A meta-analysis evaluating the relationship between A blood types and Helicobacter pylori infection

Figure 8 displays the total pooled OR for the correlation between H. pylori infection and blood type A. There is no evidence to support a significant link between having an A blood type and an increased risk of H. pylori infection (OR=1.041; 95% CI; 0.958-1.132, p = 0.340). Additionally, there was strong evidence of heterogeneity (I2 = 74.68), although the funnel plot showed no signs of publication bias thanks to its symmetry (Fig. 8). Additionally, the Egger regression asymmetry test (Egger's test; b = 0.80198, P = 0.486) did not reveal any statistically significant publishing bias. The magnitude of the pooled estimates was unaffected significantly by subgroup analyses based on different methodological criteria (Data not shown). In the sensitivity analysis, no research significantly changed the pooled OR estimate (Data not shown).

Meta-analysis of the association between the Rh Blood group and H. pylori infection

Figure 9 depicts the total pooled OR for the Rh blood type and H. pylori infection. Overall, Rh positive was related to a non-significant lower risk of H. pylori infection (pooled OR 0.804 (95% CI 0.614-1.053). There was evidence of heterogeneity (P 0.0001), with between-study heterogeneity accounting for a considerable amount of the total range in the estimated impact (I2 =31.9%).

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Discussion

A meta-analysis of 30 studies found that the O blood group was associated with a 16.3% increase in the probability of H. pylori infection compared to the non-O blood group. Blood group B and AB were also associated with a 17% and 29% reduction in H. pylori infection compared to non-B and non-AB, respectively. The results did not vary significantly by study area, quality, or design. There was moderate to substantial heterogeneity in the estimated effect size between studies, suggesting that systematic effect size variability was unaccounted for. Study quality, design, and participant characteristics may have moderated the results. The subgroup analysis did not materially alter the size of pooled estimates, and no evidence was found for A and Rh blood group groups significantly associated with H. pylori infection risk. Further studies are needed to clarify the impact of blood groups on disease risk.

The meta-analysis's findings have limitations, including significant heterogeneity in the overall estimates, possibly due to methodological or population variations. An alternative approach is individual participant meta-analysis, which provides consistency across potentially mediating characteristics. The high statistical heterogeneity across the studies still limits the robustness of the results from subgroup analysis and sensitivity analysis. The results are prone to selection bias due to observational studies, and potential confounding from other risk factors cannot be ruled out. Additionally, the narrowing of the studies to only English may have missed suitable studies published in other languages. Although no evidence of publication bias was found, some unpublished studies may have been missed.

The thorough search method utilized in the PubMed database to discover relevant papers was a key strength of this analysis. Furthermore, reference lists of retrieved publications were verified to decrease the potential of missing eligible research. Furthermore, study eligibility was thoroughly checked by an independent reviewer and, where necessary, by an additional independent reviewer. After removing research of lesser quality, most of the relevant studies were determined to be of good methodological quality, and the findings were consistent. Specific inclusion criteria for the technique of ascertainment and other aspects were applied, thereby avoiding reporting bias that may arise from using a subjective self-reported approach. Our inclusion criteria demanded that the result of interest, H. pylori infection, be assessed objectively. The majority of research employed ELISA testing to identify anti-H. Pylori IgG antibodies to diagnose H. pylori infection. Most of the included studies employed conventional hemagglutination procedures for ABO phenotypic identification, which have been in clinical use for the main blood types for a century (32) and on which blood banking is based.

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Furthermore, the meta-analysis has strong statistical power due to using 30 studies and approximately 12,000 people.

Previous biochemical investigations in the ABO, Lewis, and Secretor histo-blood systems (33, 34) have postulated the underlying mechanism for the link between the ABO blood group and H. pylori infection. Individuals with the secretor phenotype convert the precursor oligosaccharide type 1 into H antigen type 1 but cannot synthesize A or B antigens due to a lack of GTA and GTB glycosyltransferases. As a result, they transform H type 1 antigen into Leb antigen in 90% of Lewis-positive patients. The high quantitative expression of this antigen in the gastric and duodenal mucosae of O blood group people with the secretor phenotype appears to promote susceptibility to H. pylori infection by giving a higher quantity of antigens(33). In this meta-analysis, the increased risk of H. pylori infection among persons in the O blood group but not in the non-O blood group (A, B, AB) matched the above-suggested molecular processes.

Furthermore, the higher risk of H. pylori infection in secretors compared to non-secretors in our study fits with the early hypothesis proposed by Borén et al.(33), in which bacteria choose to attach to the Lewis b antigen (Leb), which is rich in fucose and expressed on the surface of the epithelial cells of the gastric mucosa (33). The majority of the studies included in our analysis did not provide secretor frequencies in each A, B, and O individual; however, studies documented that the O and Le (a-b+) phenotypes express a greater quantity of these fucosylated antigens than other groups, which predisposes these carriers to H. pylori infection (33, 35), and influences the development of H. pylori-associated complications (33). Even though most epidemiological studies did not report data on specific strains of H. pylori about the ABO group, experimental evidence has shown a strain-dependent preference of BabA adhesin(s) in binding to MUC5AC glycoforms substituted with Leb, which may contribute to interindividual variability in host-microbe interactions(36).

In conclusion, our meta-analysis shows that the O blood type is associated with a higher risk of H. pylori infection than the non-O blood group. However, high-quality cohort studies are required to establish causation, which might considerably contribute to understanding the aetiology of H. pylori infection and identifying new prevention treatments. For example, if more controlled research establishes the link between O blood type and H. pylori infection, O blood group status may be classified as one of H. pylori risk factors, and ABO blood typing may thus form part of a multidimensional strategy for H. pylori risk assessment.

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Ten simple rules for carrying out and writing meta-analyses

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Figure Legends

Fig. 1. Flow diagram of the study selection procedure

Fig. 2. Forest plot of the association between O blood group and H. pylori infection. For each study, the box represents the fixed and random effects odds ratio and the line the 95% confidence intervals. The size of each box indicates the relative weight of each study in the meta-analysis. Test for overall fixed and random effects: z = 3.731, P < 0.001, I2 = 85.52, P < 0.001; z = 1.818, P = 0.069, respectively.

Fig. 3. Funnel plot of standard error by log odds ration for the association between O blood group and H. pylori infection. Egger's test; b = 0.80198, P = 0.48672.

Fig. 4. Forest plot of the association between B blood group and H. pylori infection. For each study, the box represents the fixed and random effects odds ratio and the line the 95% confidence intervals. The size of each box indicates the relative weight of each study in the meta-analysis. Test for overall fixed and random effects: z = -3.073, P = 0.002, I2 = 62.31, P < 0.001; z = -1.858, P = 0.063, respectively.

Fig. 5. Funnel plot of standard error by log odds ration for the association between B blood group and H. pylori infection. Egger's test; b = -0.221, P = 0.79117.

Fig. 6. Forest plot of the association between AB blood group and H. pylori infection. For each study, the box represents the fixed and random effects odds ratio and the line the 95% confidence intervals. The size of each box indicates the relative weight of each study in the meta-analysis. Test for overall fixed and random effects: z = -4.235, P < 0.001, I2 = 49.27, P = 0.002; z = -2.027, P = 0.043, respectively.

Fig. 7. Funnel plot of standard error by log odds ration for the association between AB blood group and H. pylori infection. Egger's test; b = 0.77514, P = 0.17212

Fig. 8. Forest plot of the association between A blood group and H. pylori infection. For each study, the box represents the fixed and random effects odds ratio and the line the 95% confidence intervals. The size of each box indicates the relative weight of each study in the meta-analysis. Test for overall fixed and random effects: z = 0.953, P = 0.340, I2 =74.68, P < 0.001; z = -0.213, P = 0.831, respectively.

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Fig. 9. Forest plot of the association between Rh blood group and H. pylori infection. For each study, the box represents the fixed and random effects odds ratio and the line the 95% confidence intervals. The size of each box indicates the relative weight of each study in the meta-analysis. Test for overall fixed effects and random effects: z = -1.583, P = 0.113, I2 = 31.999, P = 0.184; z= -1.306, P=0.192, respectively.

Fig. 10. Forest plot of the association between secretor status and H. pylori infection. Test for overall fixed and random effects: z = 1.067, P = 0.286, z= 1.001, P=0.317, respectively. I2 = 18.157, P = 0.184.

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