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• ORIGINAL RESEARCH •

Association of *H. pylori* infection with gastric carcinoma: a Meta analysis

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Abstract

AIM: **To follow the principles of evidence based medicine to reach the integrated results of these studies.**

METHODS: **Twenty-one papers of case-control studies were selected, including 11 on gastric cancer, 7 on precancerous lesion of stomach and 3 on lymphoma of stomach. Meta analysis was used to sum up the odds ratios (OR) of these studies.**

RESULTS: ***H. pylori* vsgastric cancer (intestinal and diffuse type): the odds ratio from the fixed effect model is 3.0 016 (95% CI: 2.4197-3.7234, *P*<0.001). *H. pylori vs* precancerous lesion of stomach: a random effect model was used to calculate the summary odds ratio and its value is 2.5635 (95% CI: 1.8477-3.5566, *P*< 0.01). *H. pylori vs* lymphoma of stomach: though the quantity of literature is too small to make Meta analysis, the data of these 3 studies show that lymphoma of stomach is highly associated with *H. pylori* infections.**

CONCLUSION: **Since it had been revealed that *H. pylori* infection pre-exists in gastric carcinoma and precancerous lesions, the results of Meta analysis present a strong evidence to support the conclusion that *H. pylori* infection is a risk factor for gastric carcinoma.**

Subject headings *Helicobacter pylori*; Helicobacter infections; stomach neoplasms/microbiology; evidence-based medicine; meta-analysis

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INTRODUCTION

Since *H. pylori* was founded in 1983, the association of *H. pylori* with *H. pylori* related diseases has become the hot spot of gastroenterological studies. Gastric carcinoma is the most important disease among *H. pylori* related diseases. It is believed that *H. pylori* is one of the important causes of gastric carcinoma. But there is still lack of the final conclusion and the definite mechanisms of their association.

In China, malignant tumor is the second death cause of men and the third death cause of women according to the investigation of death cause in 29 provinces, municipalities and autonomous regions conducted from 1 973 to 1975. The annual average mortality rate of gastric carcinoma is as high as 16 per 100 thousand, which is the leading death cause among malignant tumors, and the rates of *H. pylori* infection are higher than 50 percent in the mainland of China[1]. In t he past two decades, Chinese medical researchers and clinical workers have done much a great amount of studies to reveal the relationship and the mechanisms of the association of *H. pylori* with gastric carcinoma. Most of them agree to the opinion that *H. pylori* is a risk factor for gastric carcinoma, but a certain number of them hold different points of view[2-19].

Eslick *et al*[20] have workedout the summary odds ratio of 2.04 of those same studies made in Western countries. In this paper, we reviewed all the literatures of studies in China on the relationship between *H. pylori* and gastric carcinoma as well as precancerous lesions of stomach published from 1995 in order to reach a summary conclusion using statistical methods.

MATERIALS AND METHODS

Literature

A CBM (Chinese Biomedical Database) search for articles published from 1995 was performed with the MeSH headings "*Helicobacter pylori*," "gastric carcinoma (cancer),""precancerous lesion of stomach" and "lymphoma of stomach." More than 100 papers were retrieved. Since most of them had no appropriate controls or their data did not meet the requirements of Meta analysis, only 21 papers of case-control studies were selected, including 11 about gastric cancer (Table 1), 7 on precancerous lesions of stomach (Table 2) and 3 on lymphoma of stomach (Table 3).

Data

Eleven case-control studies on the relationship between *H. pylori* infection and gastric cancer (intestinal and diffuse type) included totally 820 patients and 11-647 controls. Among them, 7 attained significant results, and 4 did not (Table 1).

Seven case-control studies on the relationship between *H. pylori* infection and precancerous lesions of stomach included totally 1978 patients and 6076 controls. All of them had significant results (Table 2).

Three case-control studies on the relationship between *H. pylori* infection and lymphoma of the stomach included totally 83 patients and 143 controls. All of them had significant results (Table 3).

Methods

In the statistical analysis, Meta analysis method with fixed effect model and random effect model was used to reach the integrated conclusion[42].

Table 1 Eleven case-control studies of *H. pylori vs.* gastric cancer[21-31]

No.	Cases		Controls		OR	95%CI	X^2	*P* value
	Hp (+)	*Hp* (-)	*Hp* (+)	*Hp* (-)				
1	61	13	49	25	2.394	1.110-5.163	4.284	0.038
2	13	3	78	68	3.778	1.033-13.818	3.475	0.062
3	71	21	46	46	3.381	1.791-6.384	13.52	0.000
4	99	23	6	8	5.793	1.841-18.155	8.400	0.004
5	35	16	34	87	5.597	2.746-11.408	22.873	0.000
6	101	35	6236	4628	2.142	1.455-3.151	14.959	0.000
7	16	9	32	43	2.389	0.937-6.092	2.618	0.106
8	21	19	3	12	4.421	1.080-18.093	3.457	0.063
9	55	21	12	10	2.183	0.821-5.805	1.75	0.186
10	90	13	89	50	3.889	1.977-7.653	15.500	0.000
11	60	25	35	50	3.429	1.815-6.475	13.743	0.000
Summary	622	198	6620	5027	3.0016	2.4197-3.7234	99.9483	0.000

*the literatures that did not attain significant results.

Table 2 Seven case-control studies of *H. pylori vs.* precancerous lesion of stomach[32-38]

No.	Cases		Controls		OR	95%CI	X^2	*P* value
	Hp (+)	*Hp* (-)	*Hp* (+)	*Hp* (-)				
1	344	194	1467	1311	1.585	1.309-1.918	22.089	0.000
2	67	29	175	180	2.376	1.466-3.851	11.955	0.000
3	103	38	26	53	5.525	3.036-10.056	31.988	0.000
4	427	252	992	892	1.524	1.273-1.824	20.736	0.000
5	99	41	12	28	5.634	2.614-12.144	20.128	0.000
6	134	35	272	201	2.829	1.870-4.282	24.490	0.000
7	188	27	332	135	2.831	1.805-4.442	20.836	0.000
Summary	1362	616	3276	2800	2.5635	1.8477-3.5566	31.7540	0.000

Table 3 Three case-control studies of *H. pylori vs.* lymphoma of stomach[39-41]

No.	Cases		Controls		OR	95%CI	X^2	*P* value
	Hp (+)	*Hp* (-)	*Hp* (+)	*Hp* (-)				
1	24	5	13	16	5.908	1.762-19.810	7.465	0.006
2	15	0	36	24	10.811	1.347-86.798	5.692	0.017
3	34	5	31	23	5.045	1.709-14.896	8.176	0.004

RESULTS

H. pylori infection vs gastric cancer

All 11 odds ratios were statistically homogenous ($P>0.05$). Summary odds ratio for gastric cancer related to *H. pylori* infection was 3.0016 using fixed effect model (95% CI 2.41 97-3.7234, Table 1). Figure 1 shows the summary odds ratio and odds ratios and their 95%CI of 11 case-control studies on the relationship between *H. pylori* infection and gastric cancer.

H. pylori infection vs precancerous lesion of stomach

Since 7 odds ratios were not statistically homogenous ($P<0.05$), random effect model was used to calculate the summary odds ratio. The summary odds ratio for precancerous lesions of stomach related to *H. pylori* infection was 2.5635 (95% CI 1.8477-3.5566, Table 2). Figure 2 shows the summary odds ratio and odds ratios and their 95%CI of 7 case-control studies on the relationship between *H. pylori* and precancerous lesions of the stomach.

H. pylori vs lymphoma of stomach

Though the quantity of literature is too small to make Meta analysis, the data of these 3 studies show that lymphoma of the stomach was highly associated with *H. pylori* infections. Figure 3 shows the odds ratios and their 95%CI of 3 case-control studies on the relationship between *H. pylori* infection and lymphoma of the stomach.

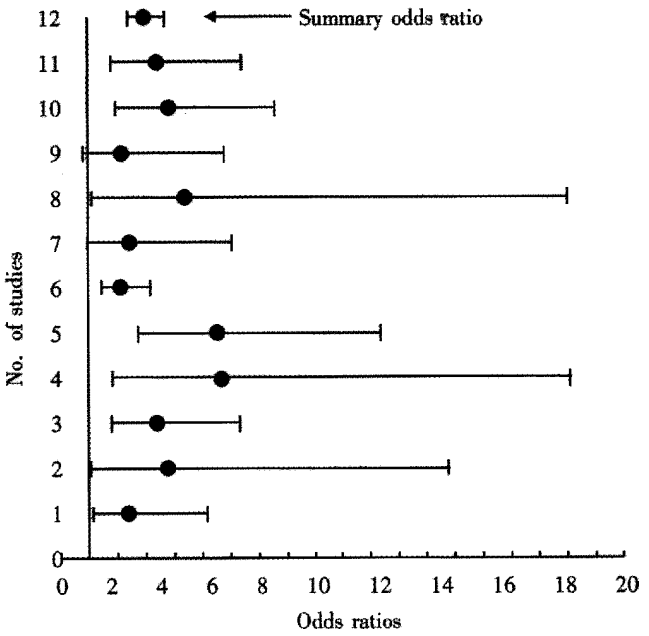

Figure 1 Odds ratios and summary odds ratio with 95%CI of 11 case-control studies.

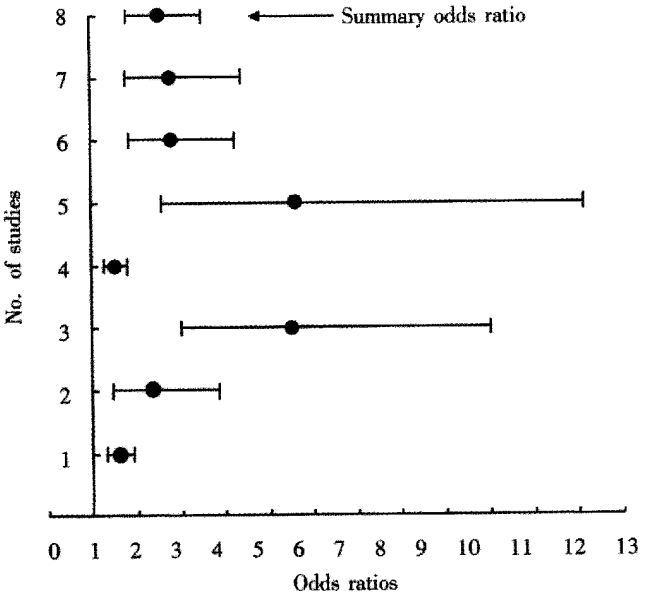

Figure 2 Odds ratios and summary odds ratio with 95%CI of 7 case-control studies.

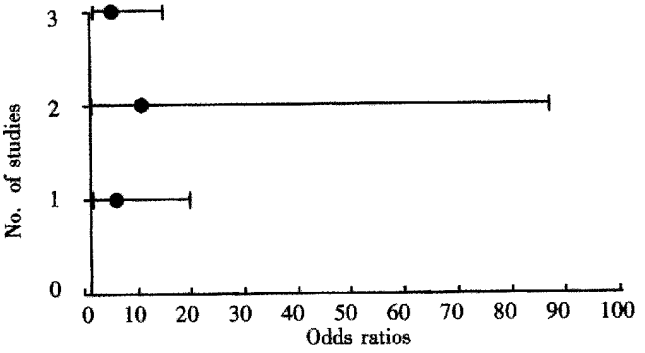

Figure 3 Odds ratios with 95%CI of 3 case-control studies.

DISCUSSION

Since it had been revealed that *H. pylori* infection pre-exists in gastric carcinoma and precancerous lesions, the result of the Meta analysis present a strong evidence to support the conclusion that *H. pylori* infection is a risk factor for gastric carcinoma.

The following statements might explain the association of *H. pylori* infection with gastric carcinoma: ① gastric carcinoma is caused by *H. pylori* infection; ② *H. pylori* infection is caused by gastric carcinoma; ③ there are some certain factors for *H. pylori* infection and gastric carcinoma[9]. As we all know that *H. pylori* infections also exist in the gastric mucosa of nearly normal persons, it is impossible that *H. pylori* infection is caused by gastric carcinoma. Numerous studies support the point that *H. pylori* infection exist before the occurrence of gastric carcinoma[43-48], indicating that *H. pylori* infection is not a secondary infection but a primary infection. It was reported that. Mongolia gerbils have been infected with *H. pylori* through mouth route, which is implanted in their stomach for a long time. Twenty-six weeks after the infection, severe chronic active gastritis, ulcer and intestinal metaplasia occurred in the stomachs of the gerbils. And 37% of the gerbils involved in the study were attacked with gastric adenocarcinoma 62 weeks after the infection[10].

The development and occurrence of gastric carcinoma is a long-lasting process and the effect of multiple factors. It is accepted by numerous scholars that *H. pylori* is an important risk factor for gastric carcinoma. In addition to the studies that support the association of *H. pylori* with gastric carcinoma, some researchers investigated the relationship between *H. pylori* and other kinds of carcinoma such as cancer of the colon, rectum, esophagus etc. and they made a negative conclusion, which proved the effect of *H. pylori* in gastric carcinoma in the other directions. However, *H. pylori* is not the unique etiological factor for gastric carcinoma. The association of the occurrence of gastric carcinoma with *H. pylori* infection should be considered from the angle of the multi-agent compound etiological theory[49-62].

According to the studies that support the opinion of association of *H. pylori* with gastric carcinoma, It can be assumed that t he canceration of gastric mucosa took place under the action of *H. pylori* in the following process: *H. pylori* related gastritis of antrum→ atrophic gastritis→intestinal metaplasia→gastric carcinoma.

There are many problems we are going to face. The infection rate of *H. pylori* is very high in the pop ulation surveys. But why most of those with *H. pylori* infection were not atta cked wit h gastric carcinoma. What are the other factors acting together with *H. pylori* to cause gastric carcinoma? How do they act? We must do more prospective interventional trial to answer these questions. It will be more helpful to do random interventional trial of eradication of *H. pylori* infection among the high-risk groups of gastric carcinoma and precancerous lesions to observe if their incidence rate of gastric carcinoma will decrease. In China, though very few of this kind of prospective studies have been made, an interventional trial of eradication of *H. pylori* is being made in Shandong and Fujian provinces. It will be of great help in revealing the exact mechanism of *H. pylori* in the process of gastric mucosa canceration.

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