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ORIGINAL RESEARCH

Mechanism of Chronic Atrophic Gastritis: A 20-Year Bibliometric Analyses

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Background and Purpose: Chronic atrophic gastritis (CAG), affecting approximately 20–30% in high-risk populations, contributes to significant morbidity and mortality due to its progression to gastric cancer. Despite two decades of research into its pathogenesis, the vast body of literature has not yet been systematically mapped. A comprehensive bibliometric analysis mapping the field's evolution, collaborative networks, and knowledge gaps remains lacking. Therefore, we conduct a 20-year bibliometric analysis (2005–2024) of research on the mechanism of CAG to identify seminal works, emerging themes, evaluate global collaboration networks, and highlight translational challenges and opportunities.

Patients and Methods: Data were retrieved from the Web of Science Core Collection (WoSCC) spanning from January 1, 2005, to December 31, 2024. Bibliometric analysis was performed using CiteSpace and VOSviewer to analyze publication trends, influential authors and institutions, keyword clusters, and citation bursts.

Results: A total of 954 papers were identified, with China leading in publication output (41.51%), followed by the USA (15.20%). The USA demonstrated high centrality in international collaboration. Key journals included WORLD J GASTROENTERO and GASTROENTEROLOGY. Prolific authors such as Liu Yuetao and co-cited authors like CORREA P were identified. Keyword analysis revealed "Helicobacter pylori" as the most prominent term, with clusters focusing on traditional Chinese medicine, macrophage biology, and gastric intestinal metaplasia.

Conclusion: The study highlights the significant research output and collaboration in CAG, emphasizing the importance of interdisciplinary approaches and international partnerships. Future research should focus on integrating traditional knowledge with modern mechanistic studies and addressing emerging themes such as microbiome dysbiosis and precision medicine.

Keywords: chronic atrophic gastritis, bibliometrics, Helicobacter pylori, mechanistic studies

Introduction

Chronic atrophic gastritis (CAG), characterized by the loss of gastric glandular structures and replacement by intestinal-type epithelium or fibrous tissue, represents a critical precancerous stage in the Correa cascade of gastric carcinogenesis.[1] With a global prevalence of approximately 20–30% in high-risk populations, CAG is closely associated with Helicobacter pylori infection, autoimmune mechanisms, and environmental factors, contributing to significant morbidity and mortality due to its progression to gastric cancer.[2,3] Over the past two decades, advances in molecular biology, genomics, and clinical diagnostics have deepened our understanding of CAG's pathogenesis, including dysregulated immune responses, oxidative stress, and epigenetic alterations.[4,5] However, despite extensive research, the heterogeneity of CAG mechanisms, challenges in early diagnosis, and gaps in targeted therapies remain unresolved.[6]

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Bibliometric analysis, a quantitative tool for evaluating scientific literature, has emerged as a powerful method to map research trends, identify knowledge gaps, and forecast future directions in medical fields.[7] Previous bibliometric studies have illuminated evolving paradigms in hepatology, such as treatment of liver fibrosis and mechanism of liver regeneration research,[8,9] yet a comprehensive analysis focusing on the mechanism of CAG is lacking. This gap hinders a systematic synthesis of interdisciplinary contributions, geographic collaboration patterns, and translational research priorities in CAG.

Despite two decades of research into its pathogenesis, the vast body of literature has not yet been systematically mapped. A comprehensive bibliometric analysis mapping the field's evolution, collaborative networks, and knowledge gaps remains lacking. Therefore, a 20-year bibliometric analysis (2005–2024) to delineate the intellectual landscape of research on the mechanism of CAG research is about to be conducted. By analyzing publication trends, influential authors, institutions, and keyword clusters, we aim to (1) identify seminal works and emerging themes, (2) evaluate global collaboration networks, and (3) highlight translational challenges and opportunities. Our findings will provide researchers, clinicians, and policymakers with actionable insights to optimize resource allocation and foster innovation in CAG management.

Materials and Methods

Data Selection

Relevant data were retrieved on January 14th, 2025 from the most influential database, the Web of Science Core Collection (WoSCC). The retrieval time span was from January 1st, 2005 to December 31st, 2024. The language was limited to English, and the article type was restricted to Articles or Reviews. Using Boolean logic operators, search terms such as "the mechanism of chronic atrophic gastritis" OR "the mechanism of chronic gastritis" OR "the mechanism of atrophic gastritis" were employed. The results were selected in the form of "Full Record and Cited References" and downloaded in the "Plain Text" document format. Subsequently, as the file format "download_*.txt" was only recognized by CiteSpace, the downloaded files were renamed accordingly. Additionally, ethical approval was not required for this study since the data were directly retrieved and exported from the WoSCC database.

Data Analysis and Visualization

Nowadays, widely used bibliometric software includes CiteSpace, VOSviewer, UCINET, SciMAT, Pajek, and Bicomb.[10] However, there is no consensus on which software is superior. Taking into account their unique features and aligning with our practical requirements, CiteSpace [version 6.3.R1 (64-bit)] and VOSviewer (version 1.6.20) were selected for bibliometric analysis in this study.[11,12]

CiteSpace, a citation visualization software based on Java and developed by Professor Chaomei Chen, also serves as a bibliometric analysis tool. It can identify potential research hotspots and trends within a specific field by generating knowledge-maps.[13] In this study, the annual growth trend of publication outputs, countries/regions and institutions, journals and co-cited journals, authors and co-cited authors, the occurrence of keywords, co-cited references, and reference bursts were analyzed and visualized to understand the mechanism of CAG.

VOSviewer, a bibliometric mapping software developed by Leiden University and based on Java, excels in visualizing scientific knowledge and managing large bibliometric maps derived from network data.[12] Utilizing bibliographic and text data, the software analyzes and visualizes productive journals and authors, relevant knowledge maps, keyword research, and cluster maps, serving as a complement to the capabilities of CiteSpace software.

Methods

We employed CiteSpace (v6.3.R1) and VOSviewer (v1.6.20) for bibliometric analysis and visualization.

CiteSpace was used to analyze publication trends, collaboration networks, keyword bursts, and reference co-citation patterns. Parameters included: time slicing (2005–2024), node types (eg, country, institution, keyword), and pruning (pathfinder algorithm).

VOSviewer complemented this by mapping co-authorship, journal co-citation, and keyword co-occurrence networks. The minimum occurrence threshold for keywords was set to 5.

Microsoft Office Excel 2010 was used for data management and preliminary trend analysis.

All visualizations (eg, network maps, timeline views) were generated to highlight evolutionary trends and research hotspots in CAG mechanisms.

Results

Figure 1 depicts the flow chart of bibliographic retrieval and research steps undertaken in this study.

Annual Growth Trend of Publications

According to the criteria for data selection, a total of 954 papers on the mechanism of CAG were retrieved from WoSCC between 2005 and 2024 (Supplementary Material). These comprised 717 (75.16%) original articles and 237 (24.84%) reviews. As illustrated in Figure 2, the quantity of publications regarding the mechanism of CAG has been increasing steadily and progressively over the years.

Productive Countries/Regions and Institutions

A total of 3259 institutions, originating from 177 countries/regions, collaboratively authored 954 research papers. Table 1 illustrated that CHINA led with the highest count of published outputs, totaling 396, which constituted 41.51% of the entire publications. The USA followed with 145 publications, representing 15.20%, while JAPAN had 86 (9.01%), ITALY 64 (6.71%), and SOUTH KOREA 62 (6.50%). From Table 1, it was evident that CHINA and the USA significantly outpaced other countries/regions in terms of publication numbers. Within the top 10 ones, solely CHINA and BRAZIL were categorized as developing countries. Moreover, the USA demonstrated a greater centrality with a score of 0.43, signifying its crucial role as a connector in international cooperation. Regarding the institutions, Chengdu University of Traditional Chinese Medicine and Vanderbilt University (n = 23, 2.41%) topped the list with the highest number of publications, succeeded by Chinese People's Liberation Army General Hospital (n = 22, 2.31%), and Veterans

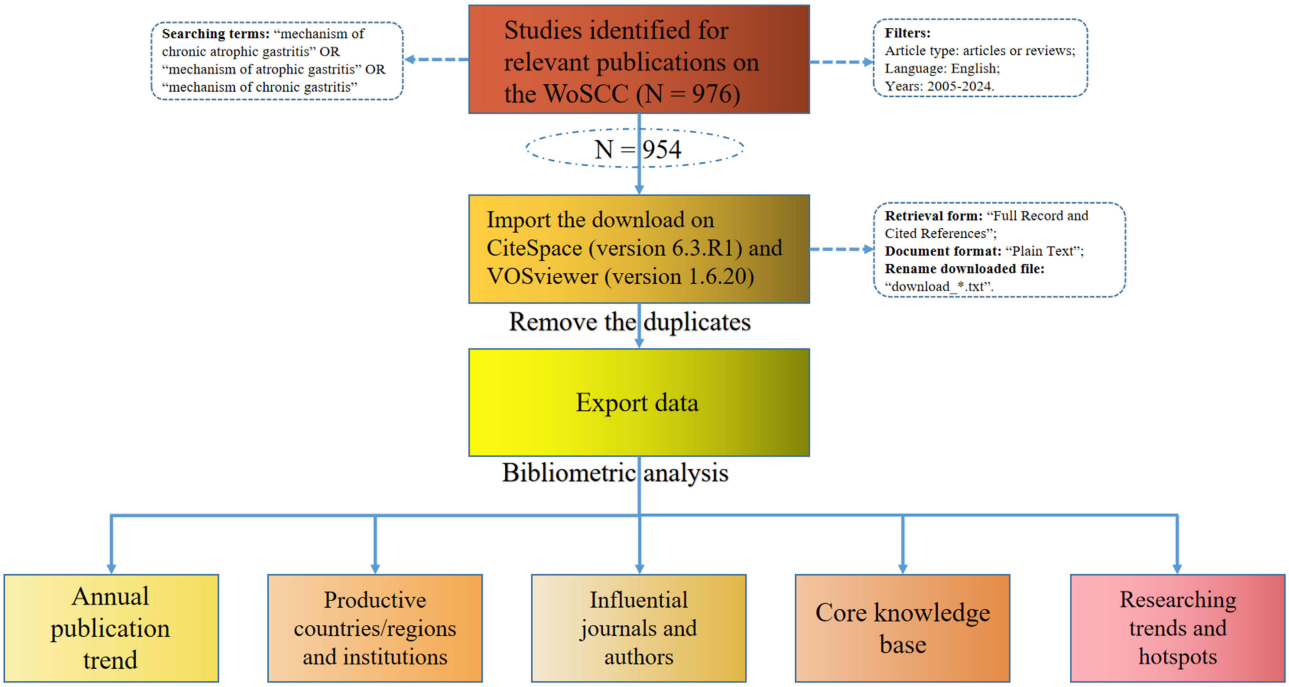

Figure 1 Flowchart of this study.

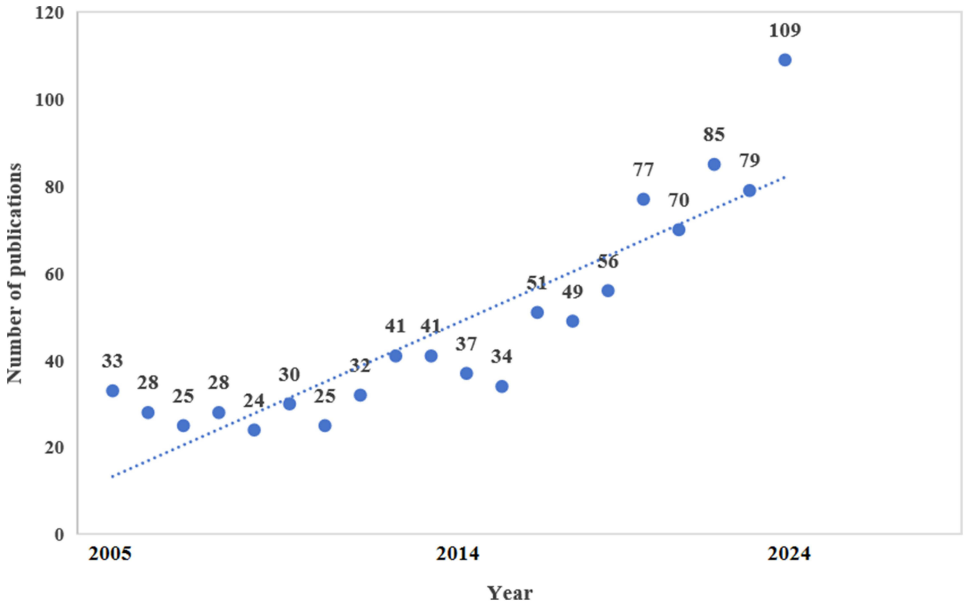

Figure 2 Chronological trend of publications about the mechanism of chronic atrophic gastritis.

Health Administration along with the US Department of Veterans Affairs (n = 20, 2.10%). Nevertheless, it was worth mentioning that among the top 10 institutions, all were from either CHINA or USA.

As shown in Figure 3A (countries/regions co-occurrence map), it illustrated a dense network of connections between nations, particularly highlighting numerous collaborations between CHINA and the USA. In Figure 3B (institutions co-occurrence map), the widespread distribution of yellow and light green years suggested that the period starting from 2018 had seen the most intense inter-institutional cooperation, with fewer such collaborations occurring prior to this time.

Productive Journals and Co-Cited Journals

To examine the journals with the highest quantity of published articles and co-citations within the CAG mechanism, VOSviewer (version 1.6.20) and CiteSpace [version 6.3.R1 (64-bit)] were utilized for conducting analyses on co-citation and journals being co-cited. The statistical findings indicated that 954 documents were published across 430 academic

Table 1 The Top 10 Countries/Regions and Institutions Involved Studies on the Mechanism of Chronic Atrophic Gastritis

Rank	Country/ Region	Article Count (%)	Centrality	Year	Institution (Country/Region)	Article Count (%)	Centrality	Year
1	Peoples R China	396 (41.51%)	0.08	2005	Chengdu University of Traditional Chinese Medicine (CHINA)	23 (2.41%)	0.03	2020
2	USA	145 (15.20%)	0.43	2005	Vanderbilt University (USA)	23 (2.41%)	0.23	2005
3	Japan	86 (9.01%)	0.08	2005	Chinese People's Liberation Army General Hospital (CHINA)	22 (2.31%)	0.03	2020
4	Italy	64 (6.71%)	0.1	2005	Veterans Health Administration (USA)	20 (2.10%)	0.09	2005
5	South Korea	62 (6.50%)	0.07	2005	US Department of Veterans Affairs (USA)	20 (2.10%)	0.09	2005
6	Germany	46 (4.82%)	0.15	2007	Shanxi University (CHINA)	19 (1.99%)	0	2017
7	England	26 (2.73%)	0.23	2006	Shanghai University of Traditional Chinese Medicine (CHINA)	16 (1.68%)	0.01	2017
8	Australia	23 (2.41%)	0.05	2005	VA Tennessee Valley Healthcare System (USA)	16 (1.68%)	0.08	2005
9	France	18 (1.89%)	0.04	2006	Nanjing University of Chinese Medicine (CHINA)	16 (1.68%)	0.06	2017
10	Brazil	18 (1.89%)	0.05	2009	China Medical University (CHINA)	15 (1.57%)	0.05	2010

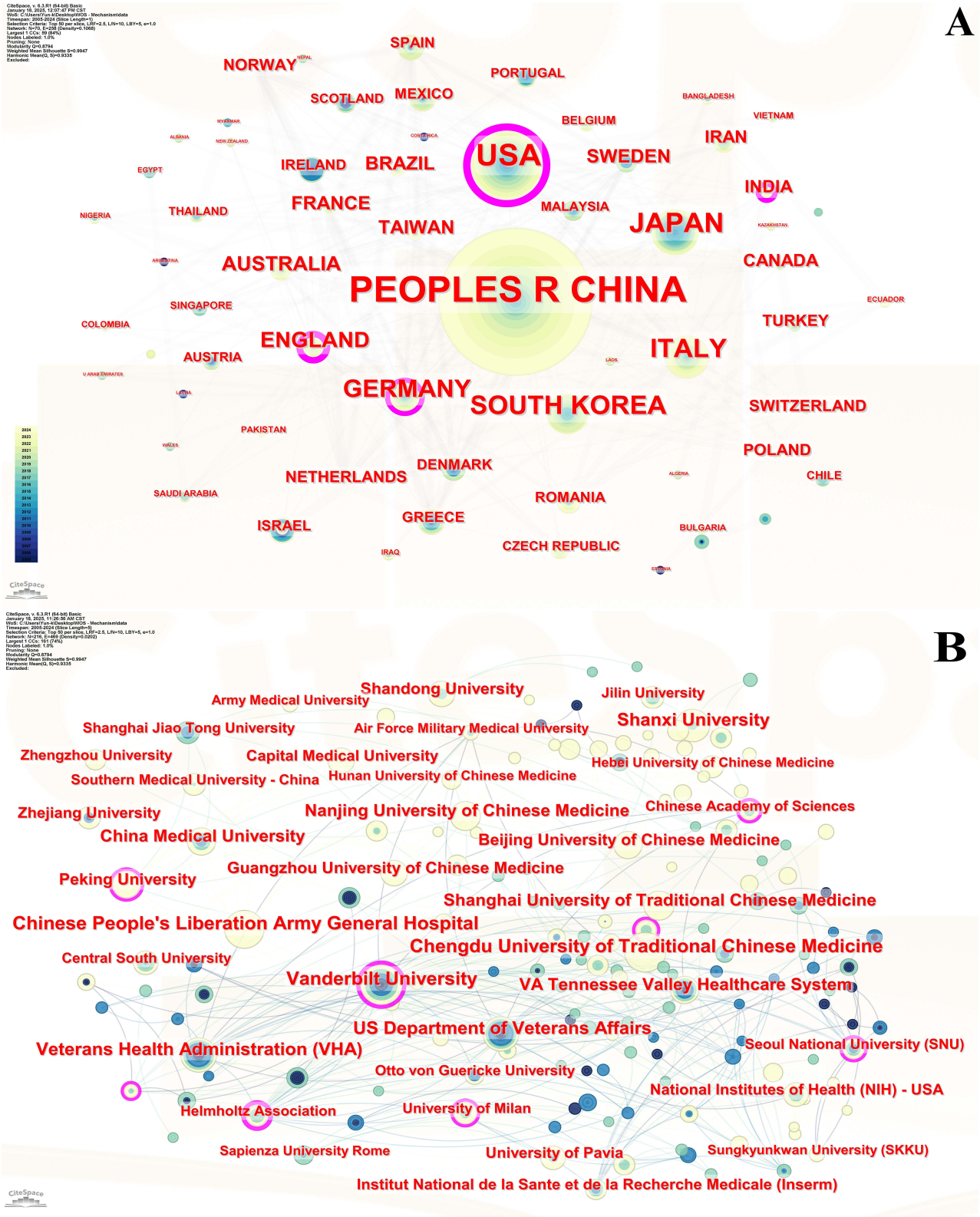

Figure 3 Co-occurrence maps (**A**) Countries; (**B**) Institutions). Notes: The size of node represents the co-occurrence frequencies while the links reflect the co-occurrence relationships. The color of node and line indicates different years.

Table 2 The Top 10 Journals and Co-Cited Journals Associated with the Mechanism of Chronic Atrophic Gastritis

Journals	Citation	IF (2024)	JCR (2024)	Cited-Journals	Count	IF (2024)	JCR (2024)
World J Gastroentero	1395	4.3	Q1	Gastroenterology	629	25.7	Q1
Gastroenterology	1225	25.7	Q1	Gut	547	23.0	Q1
J Ethnopharmacol	735	4.8	Q1	World J Gastroentero	459	4.3	Q1
Cancer Lett	575	9.1	Q1	Helicobacter	403	4.3	Q1
Helicobacter	467	4.3	Q1	P Natl Acad Sci USA	362		
PLOS One	461	2.9	Q1	PLOS One	351	2.9	Q1
Gut	439	23.0	Q1	Nature	340	50.5	Q1
Cell Commun Signal	400	8.2	Q1	Cancer Res	330	12.5	Q1
Int J Cancer	366	5.7	Q1	Am J Gastroenterol	325	8.0	Q1
Front Microbiol	338	4	Q2	New Engl J Med	257	96.2	Q1

journals. Table 2 listed the top 10 journals and co-cited journals related to the mechanism of CAG. Within these journals, WORLD J GASTROENTERO, with 1395 citing articles, was the most published, succeeded by GASTROENTEROLOGY (n = 1225), J ETHNOPHARMACOL (n = 735), CANCER LETT (n = 575), and HELICOBACTER (n = 467). Additionally, within the Q1 JCR (Journal Citation Reports) category, there were 9 journals, among which GASTROENTEROLOGY (IF = 25.7) boasted the highest impact factor (IF). From the entire list of journals, the top 300 with the strongest total link strength were selected to construct the overlay map (Figure 4A), which could effectively illustrate the productive journals based on the color timeline.

Regarding the journals most frequently cited in Table 2, GASTROENTEROLOGY (n = 629) topped the list, closely followed by GUT (n = 547), WORLD J GASTROENTERO (n = 459), HELICOBACTER (n = 403), and P NATL ACAD SCI USA (n = 362). Notably, all these journals occupied the Q1 region in the JCR, with NEW ENGL J MED boasting the highest IF of 96.2. The network diagram in Figure 4B adeptly showcased the top 200 co-cited journals, selected based on their strongest total link strength.

Productive Authors and Co-Cited Authors

The results of the bibliometric analysis conducted using VOSviewer (version 1.6.20) indicated that 5612 authors were retrieved. As presented in Table 3, Liu Yuetao ranked first with 16 published papers, followed by Zhao Yanling and Qin Xuemei (each with 14 papers), Wang Ruilin (11 papers), and Wei Shizhang (9 papers). Additionally, with the chosen threshold of a minimum of 2 documents per author, a total of 578 authors were ultimately selected to construct the network map. As illustrated in Figure 5A, various colors denoted different clusters, signifying close collaboration between clusters, such as those between Liu Yuetao and Zhao Yanling, Liu Yuetao and Hao Xinyu, Liu Yuetao and Cheng Chun, and so forth. Moreover, active collaborations related to the mechanism of CAG were readily observed, particularly among authors within the same cluster, including collaborations between Zhao Yanling and Wang Ruilin, Zhao Yanling and Li Haotian, Zhao Yanling and Wei Shizhang, among others.

Co-cited authors are those who are cited together in a range of papers. A total of 28,500 co-authors were identified. As shown in Table 3, the top 10 co-cited authors were all cited more than 90 times. The most frequently cited was CORREA P (n = 259), followed by MALFERTHEINER P (n = 159), YAMAOKA Y (n = 137), PEEK RM (n = 125), and BACKERT S (n = 121). Furthermore, authors with at least 20 co-citations (n = 236) were selected to create a density map, which effectively displayed the high-frequency co-cited authors through a gradient of yellow. From Figure 5B, it is evident that the yellow region corresponding to CORREA P was the darkest, indicating that this author was the most co-cited in the field.

Keyword Co-Occurrence, Clusters, and Evolution

VOSviewer (version 1.6.20) offered keyword co-occurrence and network cluster analysis. Utilizing this software, we extracted a total of 4609 keywords. Given the minimal impact of infrequently occurring keywords, we established a threshold of "minimum number of occurrences of a keyword ≧ 5" for selection. Consequently, 365 keywords met this

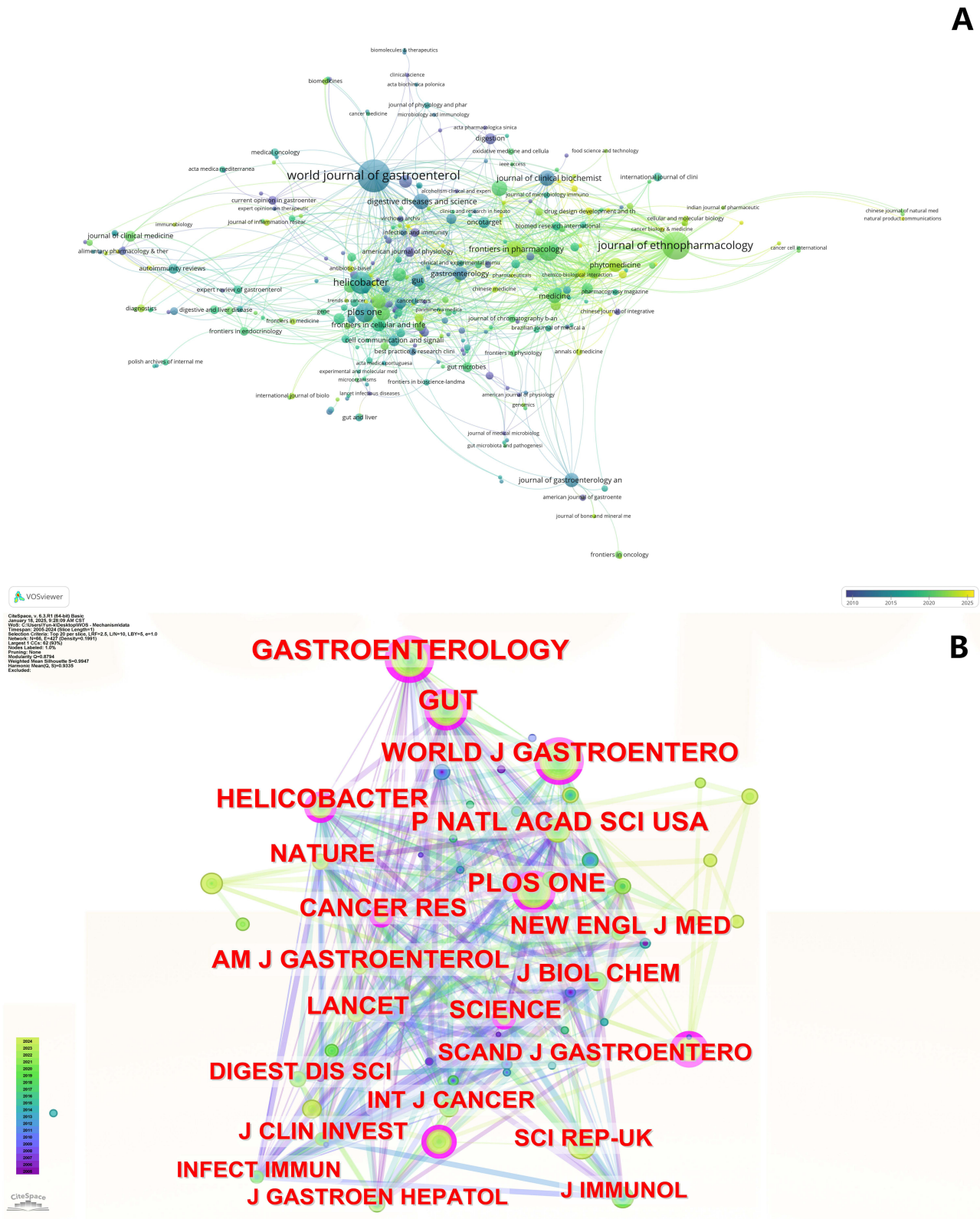

Figure 4 The density maps (**A**) Journals; (**B**) Co-cited journals). Notes: The size of word and round, and the opacity of yellow are positively associated with the co-cited frequency.

Table 3 The Top 10 Authors and Co-Cited Authors Involved Studies on the Mechanism of Chronic Atrophic Gastritis

Rank	Author	Document	Co-Cited Author	Citation
1	Liu, Yuetao	16	Correa P	259
2	Zhao, Yanling	14	Malfertheiner P	159
3	Qin, Xuemei	14	Yamaoka Y	137
4	Wang, Ruilin	11	Peek RM	125
5	Wei, Shizhang	9	Backert S	121
6	Di sabatino, Antonio	8	Dixon MF	115
7	Lenti marco, Vincenzo	8	Blaser MJ	102
8	Yuan, Yuan	8	Fox JG	101
9	Li, Haotian	7	El-Omar EM	93
10	Jing, Manyi	7	Graham DY	92

threshold and were utilized for further analysis. As presented in Table 4, which listed the top 20 occurring keywords, these indicated the research hotspots of the CAG mechanism. Seven keywords appeared more than 100 times. By frequency, the keyword "Helicobacter pylori" topped the list with 372 occurrences, followed by "gastric cancer" (n = 176), "expression" (n = 162), "cancer" (n = 138), "infection" (n = 117), "chronic atrophic gastritis" (n = 113), "atrophic gastritis" (n = 106), "Helicobacter pylori infection" (n = 96), "intestinal metaplasia" (n = 87), and "inflammation" (n = 78). Additionally, these high-frequency keywords were visually represented in the density map (Figure 6A).

Additionally, Figure 6B depicted the results of a network cluster analysis on keywords. In this map, 7 clusters represented 7 distinct research directions and scopes. Cluster 1, colored in red, is the largest, followed by cluster 2 (green), cluster 3 (blue), cluster 4 (yellow), cluster 5 (purple), cluster 6 (light blue), and cluster 7 (orange). Specifically, cluster 1 contained 83 items, including Helicobacter pylori eradication, CDX-2, COX-2, gastric intestinal metaplasia, NF-kappa B, and others. Cluster 2 had 69 items, such as acid-secretion, antibiotic resistance, duodenal ulcer, dyspepsia, and Helicobacter pylori infection. Cluster 3 included 68 items, such as Helicobacter pylori, autoimmune gastritis, chronic inflammation, cytokines, and immune-responses. Cluster 4 consisted of 62 items, including Helicobacter pylori, CAGA, colonization, beta-catenin signal, e-cadherin, and more. Cluster 5 had 59 items, encompassing anti-inflammation, antioxidant, apoptosis, metabolomics, and oxidative stress. Cluster 6 contained 22 items, such as Helicobacter pylori, body-mass index, Chinese medicine, gut microbiota, and leptin. Lastly, cluster 7 had just 2 items, which were China and epidemiology.

The Keywords Timeline Viewer, constructed by CiteSpace, clustered keywords while considering their temporal evolution, effectively displaying the trajectory of high-frequency terms within each cluster. Additionally, the viewer aided in identifying the timeline of specific topics and the developmental path of our research field. As depicted in Figure 7, the focus and evolution of the mechanism of CAG at each stage were visually apparent.

Citing Article and Co-Cited References

In order to identify the major citing article and the most co-cited references related to the mechanism of CAG, CiteSpace [version 6.3.R1 (64-bit)] was utilized for bibliometric analysis. As depicted in Table 5, the results of clustering all the included references revealed five relevant clusters: A. Traditional Chinese Medicine, B. Macrophage Biology, C. Gastric Intestinal Metaplasia, D. Changing Face, and E. Antioxidant Systems Vitamin C. Specifically, cluster A primarily described the mechanism of action of Chinese medicine compounds in the treatment of CAG. Cluster B mainly elucidated the pathological changes of gastric mucosa following Hp infection and its eradication from the perspective of macrophage biology. Cluster C primarily addressed the progression of gastric mucosal intestinal metaplasia and the current diagnosis and treatment strategies. Cluster D primarily interpreted the changing face of chronic autoimmune atrophic gastritis. Lastly, cluster E primarily detailed the role of the antioxidant system in the progression of gastric cancer.

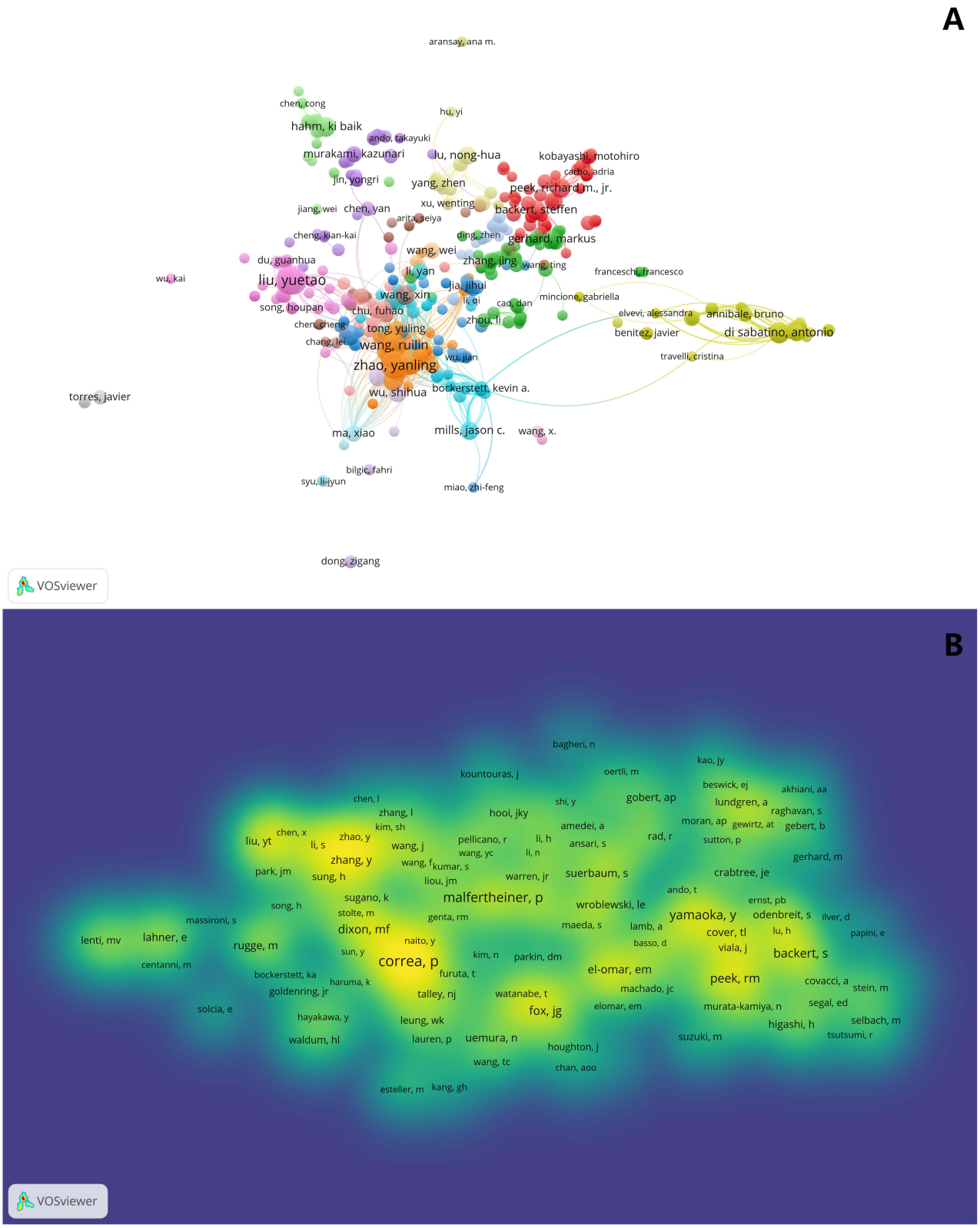

Figure 5 The co-occurrence maps in the mechanism of chronic atrophic gastritis (**A**) Authors; (**B**) Co-authors). Notes: The size of node indicates the author's co-occurrence frequencies while its different colors reflect different clusters, and the links reflect the co-occurrence relationship between authors (Map A). The size of word and round, and the opacity of yellow are positively associated with the co-cited frequency (Map B).

Table 4 The Top 20 Keywords Associated with the Mechanism of Chronic Atrophic Gastritis

Rank	Keyword	Count	Rank	Keyword	Count
1	Helicobacter pylori	372	11	Activation	74
2	Gastric cancer	176	12	Mechanisms	69
3	Expression	162	13	Risk	59
4	Cancer	138	14	NF-kappa b	54
5	Infection	117	15	Eradication	50
6	Chronic atrophic gastritis	113	16	Oxidative stress	46
7	Atrophic gastritis	106	17	Disease	46
8	Helicobacter pylori infection	96	18	Epithelial cells	45
9	Intestinal metaplasia	87	19	Association	44
10	Inflammation	78	20	Cells	42

Reference Burst

Reference bursts, characterized by frequent citation over a period of time, were detected using CiteSpace software. The selection criteria included a minimum burst duration of 2 years and the identification of the top 50 references with the strongest citation bursts. As shown in Figure 8, 14 references (28%) exhibited citation bursts in 2005, followed by 2017 and 2007, both with 5 references each, accounting for 10%. Furthermore, it was observed that 47 references, constituting 94%, experienced bursts until 2022. Regarding the strongest burst, a practice guideline titled "Management of Helicobacter pylori infection–the Maastricht IV/Florence Consensus Report," with a burst strength of 11.79, was published in GUT by Malfertheiner Pet al in 2012.[14] Additionally, its citation burst occurred from 2012 to 2022.

Discussion

General Information

From 2005 to 2024, a total of 954 papers on the mechanism of chronic atrophic gastritis (CAG) were published across 430 academic journals, involving 5686 authors from 3259 institutions in 177 countries/regions. Publication output demonstrated sustained growth over this period, reflecting consistent research engagement and advancing knowledge in CAG pathogenesis.

China led in productivity (41.51% of publications), followed by the USA (15.20%), Japan (9.01%), Italy (6.71%), and South Korea (6.50%). The USA exhibited high centrality (0.43), indicating its pivotal role in international collaborations. Institutional analysis revealed dominance by Chinese and American entities, such as Chengdu University of Traditional Chinese Medicine and Vanderbilt University, with intensified inter-institutional cooperation observed post-2018.

Journal analysis highlighted World Journal of Gastroenterology as the most productive journal, while Gastroenterology had the highest impact factor (IF = 25.7) and co-citation frequency. Author contributions were led by Liu Yuetao (16 publications), with CORREA P being the most co-cited author (n = 259), emphasizing foundational work on Helicobacter pylori and CAG pathogenesis.

Knowledge Base

Co-cited references, cited together by other publications, can measure the degree of relevance between papers. The knowledge base, characterized by the corresponding research community, is a collection of co-cited references. Furthermore, research papers with the highest co-citation frequency are an important research foundation in a certain field. In this bibliometric analysis, Table 5 further unraveled the multidisciplinary and evolving research framework underlying the mechanism of CAG, as evidenced by co-citation clustering of pivotal references. The five identified clusters (A-E) reflected both established and emerging paradigms in CAG research, bridging traditional therapeutic approaches, molecular pathology, and clinical translation.

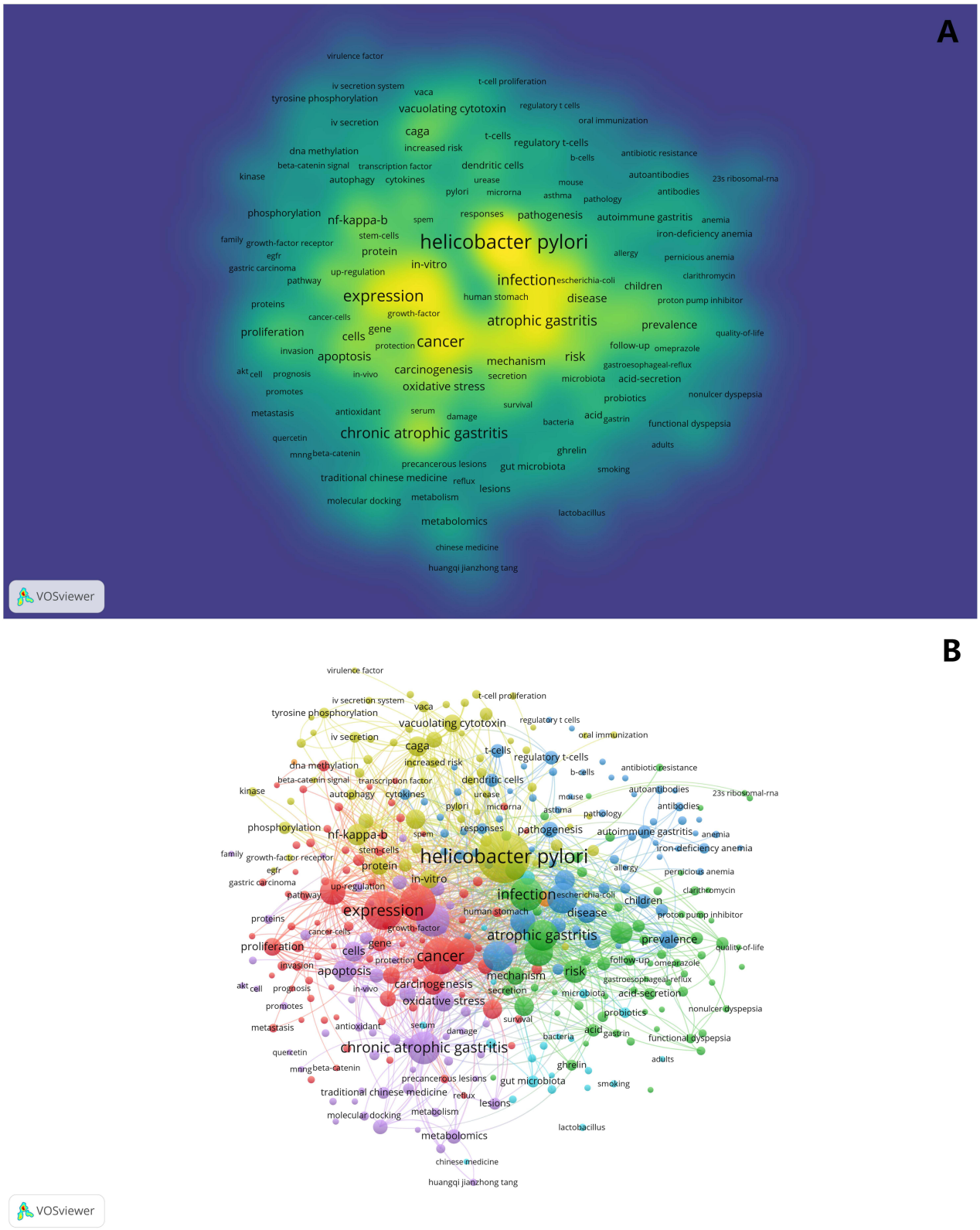

Figure 6 Maps of keywords in the mechanism of chronic atrophic gastritis (**A**) The density map; (**B**) Co-occurrence network and clusters). Notes: The size of word and round, and the opacity of yellow are positively associated with the co-cited frequency (Map A). The size of node and keyword indicates the co-occurrence frequencies while their different colors reflect different clusters, and the links reflect the co-occurrence relationship (Map B).

Figure 7 Keywords timeline viewer of the mechanism of chronic atrophic gastritis.

Cluster A (Traditional Chinese Medicine) highlighted the growing scientific interest in ethnopharmacology and holistic interventions for CAG. The focus on Chinese medicinal compounds suggested a shift toward integrating traditional knowledge with modern mechanistic studies, particularly in modulating inflammation, epithelial repair, and Hp-induced damage. This cluster underscored the need for rigorous validation of herbal therapies through molecular and clinical studies to reconcile traditional practices with evidence-based medicine.[15–18] For example, traditional drugs-particularly compounds derived from TCM (eg, Sijunzi Decoction, Xiangsha Liujunzi decoction)-highlighted their emerging role in modulating inflammation, apoptosis, and signaling pathways (eg, TLR/TNF-α/NF-κB).[19,20]

Cluster B (Macrophage Biology) emphasized the immunological dimension of CAG pathogenesis. By linking Hp infection to macrophage-driven mucosal remodeling, this cluster aligned with contemporary insights into host-microbe interactions, chronic inflammation, and immune-mediated atrophy. The prominence of macrophage biology reflected a broader trend in decoding the immune microenvironment's role in gastric carcinogenesis, offering potential targets for immunomodulatory therapies.[21–24]

Cluster C (Gastric Intestinal Metaplasia) reiterated the clinical urgency of addressing pre-neoplastic transitions, particularly the molecular and histopathological drivers of intestinal metaplasia (IM). The inclusion of diagnostic and therapeutic strategies in this cluster signaled translational efforts to intercept CAG progression, such as biomarker discovery and endoscopic surveillance protocols. However, gaps remained in understanding the reversibility of IM and its microenvironmental regulators.[25–28]

Cluster D (Changing Face) likely explored the evolving clinical and immunological understanding of autoimmune atrophic gastritis (AAG), a subtype of CAG. This cluster may address advancements in autoantibody detection, genetic predispositions, and the interplay between autoimmune responses and gastric microenvironmental changes. The "changing face" metaphor suggested a redefinition of diagnostic criteria or therapeutic paradigms for AAG over the past two decades.[2,29,30]

Cluster E (Antioxidant Systems Vitamin C) underscored oxidative stress as a critical mechanistic node in CAG progression. The focus on antioxidant defenses, particularly vitamin C, aligned with evidence linking Hp-induced

Table 5 Summary of the Largest 5 Clusters Including Citing Articles and Cited References

The Summary Highlights Major Clusters	Count	Title	First Author	Year	Journal	DOI
The largest cluster: TRADITIONAL CHINESE MEDICINE	50 members	/	/	/	/	/
The major citing article	/	Research on drug treatment and the novel signaling pathway of chronic atrophic gastritis.	Jinhao Jia	2024	Biomedicine & Pharmacotherapy	10.1016/j.biopha.2024.116912
The most cited references	22	Network pharmacology based investigation into the effect and mechanism of Modified Sijunzi Decoction against the subtypes of chronic atrophic gastritis	Tian GH	2019	Pharmacol Res	10.1016/j.phrs.2019.04.012
	21	Zuojin Pill ameliorates chronic atrophic gastritis induced by MNNG through TGF-β1/PI3K/Akt axis	Tong YL	2021	J Ethnopharmacol	10.1016/j.jep.2021.113893
	20	Network pharmacology to unveil the mechanism of Moluodan in the treatment of chronic atrophic gastritis	Zhou W	2022	Phytomedicine	10.1016/j.phymed.2021.153837
The second cluster: MACROPHAGE BIOLOGY	39 members	/	/	/	/	/
The major citing article	/	Helicobacter pylori infection	Malfertheiner, P	2023	Nature Reviews Disease Primers	10.1038/s41572-023-00431-8
The most cited references	13	Helicobacter pylori Therapy for the Prevention of Metachronous Gastric Cancer	Choi IJ	2018	New Engl J Med	10.1056/NEJMoa1708423
	13	Gastric microbes associated with gastric inflammation, atrophy and intestinal metaplasia 1 year after Helicobacter pylori eradication	Sung JJY	2020	Gut	10.1136/gutjnl-2019-319,826
	9	Helicobacter pylori infection and antibiotic resistance - from biology to clinical implications	Tshibangu-Kabamba E	2021	Nat Rev Gastro Hepat	10.1038/s41575-021-00449-x
The third cluster: GASTRIC INTESTINAL METAPLASIA	33 members	/	/	/	/	/
The major citing article	/	Gastric intestinal metaplasia: progress and remaining challenges.	Tong, Q	2024	Journal of Gastroenterology	10.1007/s00535-023-02073-9
The most cited references	20	AGA Clinical Practice Update on the Diagnosis and Management of Atrophic Gastritis: Expert Review	Shah SLC	2021	Gastroenterology	10.1053/j.gastro.2021.06.078
	15	Notoginsenoside R1 (NGR1) Attenuates Chronic Atrophic Gastritis in Rats	Luo C	2019	Med Sci Monitor	10.12659/MSM.911512
	13	Interferon-γ directly induces gastric epithelial cell death and is required for progression to metaplasia	Osaki LH	2019	J pathol	10.1002/path.5214

(*Continued*)

Table 5 (Continued).

The Summary Highlights Major Clusters	Count	Title	First Author	Year	Journal	DOI
The 4th cluster: CHANGING FACE	13 members	/	/	/	/	/
The major citing article	/	The changing face of chronic autoimmune atrophic gastritis: an updated comprehensive perspective	Massironi, S	2018	Autoimmunity Reviews	10.1016/j.autrev.2018.08.011
The most cited references	15	The changing face of chronic autoimmune atrophic gastritis: an updated comprehensive perspective	Massironi, S	2018	Autoimmun Rev	10.1016/j.autrev.2018.08.011
	12	Chronic atrophic gastritis: Natural history, diagnosis and therapeutic management. A position paper by the Italian Society of Hospital Gastroenterologists and Digestive Endoscopists [AIGO], the Italian Society of Digestive Endoscopy [SIED], the Italian Society of Gastroenterology [SIGE], and the Italian Society of Internal Medicine [SIMI]	Lahner E	2019	Digest Liver Dis	10.1016/j.dld.2019.09.016
	11	Autoimmune atrophic gastritis: current perspectives	Minalyan A	2017	Clin Exp Gastroenter	10.2147/CEG.S109123
The 5th cluster: ANTIOXIDANT SYSTEMS VITAMIN C	6 members	/	/	/	/	/
The major citing article	/	Pathways of gastric carcinogenesis, Helicobacter pylori virulence and interactions with antioxidant systems, vitamin c and phytochemicals	Toh, JWT	2020	International Journal of Molecular Sciences	10.3390/ijms21176451
The most cited references	9	Epidemiology of gastric cancer: global trends, risk factors and prevention	Rawla P	2019	Gastroenterol Rev	10.5114/pg.2018.80001
	7	From inflammation to gastric cancer: Role of Helicobacter pylori	Zhang XY	2017	Oncol Lett	10.3892/ol.2016.5506
	6	Interleukin-17A Promotes Parietal Cell Atrophy by Inducing Apoptosis	Bockerstett KA	2018	Cell Mol Gastroenter	10.1016/j.jcmgh.2017.12.012

Top 50 References with the Strongest Citation Bursts

References	Year	Strength	Begin	End	2005 - 2024
Malfertheiner P, 2012, GUT, V61, P646, DOI 10.1136/gutjnl-2012-302084, DOI	2012	11.79	**2012**	2022	
Hooi JKY, 2017, GASTROENTEROLOGY, V153, P420, DOI 10.1053/j.gastro.2017.04.022, DOI	2017	10.56	**2017**	2022	
Kwok T, 2007, NATURE, V449, P862, DOI 10.1038/nature06187, DOI	2007	7.32	**2007**	2022	
Malfertheiner P, 2017, GUT, V66, P6, DOI 10.1136/gutjnl-2016-312288, DOI	2017	7.06	**2017**	2022	
Sugano K, 2015, GUT, V64, P1353, DOI 10.1136/gutjnl-2015-309252, DOI	2015	6.7	**2015**	2022	
Gebert B, 2003, SCIENCE, V301, P1099, DOI 10.1126/science.1086871, DOI	2003	6.44	**2005**	2022	
Polk DB, 2010, NAT REV CANCER, V10, P403, DOI 10.1038/nrc2857, DOI	2010	6.43	**2010**	2022	
Sung H, 2021, CA-CANCER J CLIN, V71, P209, DOI 10.3322/caac.21660, DOI	2021	6.42	**2021**	2024	
Amieva MR, 2008, GASTROENTEROLOGY, V134, P306, DOI 10.1053/j.gastro.2007.11.009, DOI	2008	6.07	**2008**	2022	
Ohnishi N, 2008, P NATL ACAD SCI USA, V105, P1003, DOI 10.1073/pnas.0711183105, DOI	2008	6.07	**2008**	2022	
Suerbaum S, 2002, NEW ENGL J MED, V347, P1175, DOI 10.1056/NEJMra020542, DOI	2002	6.06	**2005**	2022	
Bass AJ, 2014, NATURE, V513, P202, DOI 10.1038/nature13480, DOI	2014	5.93	**2014**	2022	
Unknown -, 2020, CA CANCER J CLIN, V70, P313, DOI 10.3322/caac.21492, DOI	2020	5.64	**2020**	2022	
Viala J, 2004, NAT IMMUNOL, V5, P1166, DOI 10.1038/ni1131, DOI	2004	5.64	**2005**	2022	
Peek RM, 2002, NAT REV CANCER, V2, P28, DOI 10.1038/nrc703, DOI	2002	5.45	**2005**	2022	
Saadat I, 2007, NATURE, V447, P330, DOI 10.1038/nature05765, DOI	2007	5.24	**2007**	2022	
Harris PR, 2008, GASTROENTEROLOGY, V134, P491, DOI 10.1053/j.gastro.2007.11.006, DOI	2008	5.15	**2008**	2022	
Pimentel-Nunes P, 2019, ENDOSCOPY, V51, P365, DOI 10.1055/a-0859-1883, DOI	2019	5.08	**2019**	2022	
Blaser MJ, 2004, J CLIN INVEST, V113, P321, DOI 10.1172/JCI20925, DOI	2004	5.07	**2005**	2022	
Wilson KT, 2007, GASTROENTEROLOGY, V133, P288, DOI 10.1053/j.gastro.2007.05.008, DOI	2007	5.05	**2007**	2022	
Boncristiano M, 2003, J EXP MED, V198, P1887, DOI 10.1084/jem.20030621, DOI	2003	4.84	**2005**	2022	
Yamaoka Y, 2010, NAT REV GASTRO HEPAT, V7, P629, DOI 10.1038/nrgastro.2010.154, DOI	2010	4.82	**2010**	2022	
Cui JJ, 2017, J PHARMACEUT BIOMED, V132, P77, DOI 10.1016/j.jpba.2016.09.044, DOI	2017	4.72	**2017**	2022	
Neumann WL, 2013, NAT REV GASTRO HEPAT, V10, P529, DOI 10.1038/nrgastro.2013.101, DOI	2013	4.63	**2013**	2022	
Xu JY, 2018, BIOMED PHARMACOTHER, V108, P95, DOI 10.1016/j.biopha.2018.09.012, DOI	2018	4.63	**2018**	2022	
Tong YL, 2021, J ETHNOPHARMACOL, V271, P0, DOI 10.1016/j.jep.2021.113893, DOI	2021	4.57	**2021**	2024	
Jiménez-Soto LF, 2009, PLOS PATHOG, V5, P0, DOI 10.1371/journal.ppat.1000684, DOI	2009	4.57	**2009**	2022	
Hatakeyama M, 2004, NAT REV CANCER, V4, P688, DOI 10.1038/nrc1433, DOI	2004	4.51	**2005**	2022	
Park Yo Han, 2015, J CANCER PREV, V20, P25, DOI 10.15430/JCP.2015.20.1.25, DOI	2015	4.47	**2015**	2022	
Ernst PB, 2006, GASTROENTEROLOGY, V130, P188, DOI 10.1053/j.gastro.2005.06.032, DOI	2006	4.45	**2006**	2022	
Fukase K, 2008, LANCET, V372, P392, DOI 10.1016/S0140-6736(08)61159-9, DOI	2008	4.4	**2008**	2022	
Tian GH, 2019, PHARMACOL RES, V144, P158, DOI 10.1016/j.phrs.2019.04.012, DOI	2019	4.37	**2019**	2022	
Malfertheiner P, 2007, GUT, V56, P772, DOI 10.1136/gut.2006.101634, DOI	2007	4.36	**2007**	2022	
Tong YL, 2021, FRONT PHARMACOL, V12, P0, DOI 10.3389/fphar.2021.644638, DOI	2021	4.32	**2021**	2024	
Ferlay J, 2015, INT J CANCER, V136, PE359, DOI 10.1002/ijc.29210, DOI	2015	4.31	**2015**	2022	
Minalyan A, 2017, CLIN EXP GASTROENTER, V10, P19, DOI 10.2147/CEG.S109123, DOI	2017	4.3	**2017**	2022	
Xu JJ, 2017, SCI REP-UK, V7, P0, DOI 10.1038/srep45580, DOI	2017	4.29	**2017**	2022	
Lundgren A, 2003, INFECT IMMUN, V71, P1755, DOI 10.1128/IAI.71.4.1755-1762.2003, DOI	2003	4.23	**2005**	2022	
Sugano K, 2015, BEST PRACT RES CL GA, V29, P895, DOI 10.1016/j.bpg.2015.09.013, DOI	2015	4.1	**2015**	2022	
Matsushima K, 2011, INT J CANCER, V128, P361, DOI 10.1002/ijc.25348, DOI	2011	4.1	**2011**	2022	
Machado JC, 2003, GASTROENTEROLOGY, V125, P364, DOI 10.1016/S0016-5085(03)00899-0, DOI	2003	4.09	**2005**	2022	
Gewirtz AT, 2004, J INFECT DIS, V189, P1914, DOI 10.1086/386289, DOI	2004	4.09	**2005**	2022	
Amieva MR, 2003, SCIENCE, V300, P1430, DOI 10.1126/science.1081919, DOI	2003	4.09	**2005**	2022	
El-Omar EM, 2003, GASTROENTEROLOGY, V124, P1193, DOI 10.1016/S0016-5085(03)00157-4, DOI	2003	4.09	**2005**	2022	
Backert S, 2008, CELL MICROBIOL, V10, P1573, DOI 10.1111/j.1462-5822.2008.01156.x, DOI	2008	4.07	**2008**	2022	
Salama NR, 2013, NAT REV MICROBIOL, V11, P385, DOI 10.1038/nrmicro3016, DOI	2013	4.05	**2013**	2022	
Sundrud MS, 2004, P NATL ACAD SCI USA, V101, P7727, DOI 10.1073/pnas.0401528101, DOI	2004	3.94	**2005**	2022	
Wong BCY, 2004, JAMA-J AM MED ASSOC, V291, P187, DOI 10.1001/jama.291.2.187, DOI	2004	3.94	**2005**	2022	
Amieva M, 2016, GASTROENTEROLOGY, V150, P64, DOI 10.1053/j.gastro.2015.09.004, DOI	2016	3.93	**2016**	2022	
Tammer I, 2007, GASTROENTEROLOGY, V132, P1309, DOI 10.1053/j.gastro.2007.01.050, DOI	2007	3.92	**2007**	2022	

Figure 8 View Citation Burst History.
Notes: The blue bars mean the reference had been published; The red bars mean citation burstness.

oxidative DNA damage to gastric carcinogenesis. This cluster may highlight therapeutic strategies to restore redox balance, potentially synergizing with eradication therapies to mitigate atrophy and metaplasia.[31–34]

The Analysis of Hotspots Evolution, Knowledge Structure, and Emerging Topics

Keywords co-occurrence, within the field of bibliometrics, can reflect the hotspots and trends of an academic discipline.[35] Table 4 elucidated the evolving research landscape and mechanistic focus of CAG, revealing key trends, hotspots, and interdisciplinary connections. The keyword co-occurrence analysis highlighted Helicobacter pylori as the most prominent keyword (372 occurrences), underscoring its central role in CAG pathogenesis. This aligned with decades of evidence linking Helicobacter pylori infection to chronic gastric inflammation, mucosal atrophy, and subsequent progression to gastric cancer – a trajectory further emphasized by high-frequency terms such as "gastric cancer" (176), "chronic atrophic gastritis" (113), "intestinal metaplasia" (87), and "inflammation" (78). The strong association between Helicobacter pylori infection and CAG-related carcinogenesis reflected the field's sustained focus on microbial-driven mechanisms and their clinical implications (Figure 6A). The network cluster analysis shown in Figure 6B identified seven distinct research directions, with Cluster 1 (red, 83 items) likely representing Hp-centric studies, including its virulence factors, host-pathogen interactions, and carcinogenic pathways. Clusters 2–7 may encompass diverse themes such as molecular mechanisms (eg, "expression", "apoptosis", "oxidative stress"), histopathological transitions (eg, "intestinal metaplasia", "dysplasia"), and clinical management (eg, "diagnosis", "biomarkers"). The smaller clusters, particularly Cluster 7 (2 items), suggested niche or emerging topics warranting further exploration. The temporal evolution mapped by CiteSpace (Figure 7) revealed shifting priorities over two decades. Early research likely focused on Hp eradication and histopathological staging (eg, "atrophic gastritis", "intestinal metaplasia"), while recent trends may emphasize molecular pathways (eg, "NF-κB", "microRNA"), immune regulation, and early cancer detection. This progression mirrored advancements in omics technologies and a growing emphasis on precision medicine in CAG management.

Emerging and research topics of a certain field could be also characterized by references with intense citation bursts.[36] The citation burst analysis derived from Figure 8 provided critical insights into the intellectual dynamics and evolving research priorities in the field of CAG over the past two decades. The pronounced citation bursts observed in 2005 (28% of top 50 references) likely reflected pivotal advancements during this period, potentially linked to emerging consensus on CAG pathogenesis, diagnostic criteria, or therapeutic strategies. Subsequent bursts in 2007 and 2017 (10% each) may correspond to milestone studies or updated guidelines that reinvigorated scholarly interest in molecular mechanisms, such as Hp-induced mucosal injury, autoimmune pathways, or the role of epigenetic modifications in gastric atrophy progression. Notably, the strongest citation burst (strength: 11.79) was attributed to the Maastricht IV/Florence Consensus Report,[14] published in GUT in 2012, which sustained its influence until 2022. This enduring impact underscored its foundational role in shaping clinical and research paradigms for Hp management – a key driver of CAG. Its prolonged burst duration aligned with ongoing debates about eradication protocols, virulence factors, and their interplay with host immune responses in CAG development. The finding that 94% of references exhibited citation bursts persisting until 2022 highlighted the sustained relevance of CAG-related research, particularly in elucidating mechanisms bridging chronic inflammation, metaplastic transformation, and carcinogenesis. However, the declining number of post-2017 bursts may signal a shift toward emerging themes (eg, microbiome dysbiosis, precision medicine) or methodological innovations (eg, single-cell sequencing, multi-omics integration) that warranted deeper exploration. Future studies should prioritize mechanistic investigations into the molecular cascades driving glandular atrophy and metaplasia, leveraging longitudinal bibliometric trends to identify underexplored pathways or therapeutic targets.

Limitations

There are some certain limitations inherent in our bibliometrics. First, data were sourced solely from the WoSCC, which may not uniformly cover all fields or journals, potentially leading to over- or underrepresentation of certain research areas. Second, the use of specific search terms and Boolean operators might have omitted relevant literature, introducing retrieval bias. Third, although CiteSpace and VOSviewer are robust tools, their outputs depend on data quality and completeness. Fourth, geographic inclusivity is limited, as Western and Chinese journals dominate the top rankings, potentially overlooking region-specific insights from high-prevalence areas like Latin America or Eastern Europe. Finally, bibliometric analysis prioritizes quantitative trends and may miss nuanced qualitative context. Additionally, while prolific authors were identified objectively, alternative metrics (eg, citation impact or clinical translation records) could complement productivity-based assessments. Meanwhile, though leading specialty journals are well-represented, the inclusion of guidelines among highly cited references highlights their

consolidating role in evidence-based research trajectories. Future efforts should encourage equitable visibility for studies addressing regionally distinct risk factors, such as dietary habits or genetic predispositions, to enrich global mechanistic models.

Conclusion

This bibliometric analysis provides a comprehensive overview of the research landscape on the mechanism of CAG over the past two decades. A concise summary of the meaningful findings is as follows:

Leading Contributors: China and the USA were the leading contributors to CAG mechanism research over the past two decades, with China producing the highest volume of publications (41.51%) and the USA playing a central role in international collaboration.

Interdisciplinary Integration: The integration of TCM with modern molecular approaches has emerged as a promising interdisciplinary strategy, offering novel insights for CAG management.

Research Focus: Key research themes included Hp pathogenesis, macrophage biology, gastric intestinal metaplasia, and oxidative stress mechanisms.

Future Directions: The study emphasized the need for enhanced global collaboration, application of advanced technologies (eg, multi-omics), and translation of basic research into clinical practices for early diagnosis and personalized therapies.

Data Sharing Statement

The datasets used during the current study are available from the corresponding author upon reasonable request.

Author Contributions

All authors meet the ICMJE and Dove Press authorship criteria. Individual contributions are specified using the CRediT taxonomy:

Yun-kai Dai: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Project administration, Resources, Software, Validation, Visualization, Writing – original draft, Writing – review & editing.

Dan-yan Li: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Validation, Writing – original draft.

Long-ling Cong: Data curation, Formal analysis, Investigation, Validation, Visualization.

Yuan Liao: Data curation, Formal analysis, Investigation, Validation.

Xue-chuan Wang: Formal analysis, Investigation, Validation.

Jia-wei Fan: Formal analysis, Investigation, Validation.

Wen-jian Chen: Formal analysis, Investigation, Validation.

Chun-hua Fan: Funding acquisition, Resources, Supervision.

Teng Ma: Conceptualization, Project administration, Resources, Supervision, Writing – review.

Yu-jin Wu: Conceptualization, Project administration, Resources, Supervision, Writing – review & editing.

Yun-kai Dai and Dan-yan Li are co-first authors. All authors took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

All authors disclosed no competing interests in any aspects.

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