Journal of Inflammation Research

Dovepress
Taylor & Francis Group

Open Access Full Text Article

REVIEW

Lifting the Veil on Neuroinflammation in Parkinson's Disease: A Bibliometric Analysis

Shuo Dong[1,*], Xuefeng Li[2,*], Siyi Wang[1], Yubo Gong[1], Lingfeng Wu[1], Jinglin Hu[1], Xinhua Chen[2]

[1]College of Acupuncture and Massage, Changchun University of Chinese Medicine, Changchun, Jilin Province, 130117, People's Republic of China; [2]Department of Acupuncture, The Affiliated Hospital to Changchun University of Chinese Medicine, Changchun, Jilin Province, 130021, People's Republic of China

*These authors contributed equally to this work

Correspondence: Xinhua Chen, Department of Acupuncture and Moxibustion and Massage, The Affiliated Hospital to Changchun University of Chinese Medicine, No. 1035 Boshuo Road, Jingyue National High-Tech Industrial Development Zone, Changchun, Jilin Province, 130117, People's Republic of China, Email 187098190@qq.com

Abstract: Parkinson's disease (PD) is a chronic neurodegenerative disorder characterized by gradual progression. A significant pathophysiology of its development and evolution is neuroinflammation. A previous review assessed the neuroinflammation research in PD, but limitations in its search period and analytical methods restricted its ability to reflect the full landscape of the field and recent research trends. To solve this problem, we performed a bibliometric and visual review in order to describe the current state of research and outline the recent progress in PD-related neuroinflammation. The Web of Science Core Collection (WoSCC) and Scopus were searched to retrieve a total of 5,926 publications published between January 1, 2015 and September 23, 2025. CiteSpace, VOSviewer, and the bibliometrix R package were used to perform a comprehensive bibliometric and visualization analysis. The results showed that China produced the largest number of publications in this field, accounting for 30.75% of the total. At the institutional level, the University of California System and Shanghai Jiao Tong University were among the leading contributors. The International Journal of Molecular Sciences was the most productive journal in this field. Among individual authors, Wang, Q.S. Zhang, F. and Cuzzocrea, S. were the most prolific contributors, whereas McGeer, P.L. was the most frequently co-cited author. Keyword analysis further identified three major research themes: fundamental pathological mechanisms, clinical diagnosis, and intervention strategies. Overall, this study provides a systematic overview of the current research landscape, principal themes, and emerging directions in neuroinflammation research on PD by means of bibliometric analysis.

Keywords: Parkinson's disease, neuroinflammation, bibliometrics, CiteSpace, VOSviewer

Introduction

Parkinson's disease (PD) is one of the most rapidly growing neurodegenerative diseases in the world.[1] The clinical manifestations are mainly characterized by motor symptoms, such as progressive asymmetric bradykinesia, myotonia, and postural instability, as well as nonmotor symptoms, including hypoesthesia, cognitive impairment, and depression.[2,3] According to the Global Burden of Disease (GBD) analysis, the number of individuals with PD is projected to exceed 25.2 million by 2050, which is more than twice the number reported in 2021. This shows its growing public health burden.[4] Epidemiological studies have shown that the incidence and risk of PD increase clearly with age, are higher in males than in females, and differ across regions, ethnic groups, and socioeconomic groups.[5] Because of this, the rising prevalence of PD has placed a heavy burden on health-care systems and has raised social and economic costs around the world.

Although the exact cause of PD is still not fully understood, accumulating evidence shows that neuroinflammation is a main factor driving the disease-related pathological changes.[6–8] Aberrant expression of pro-inflammatory cytokines accompanies the activation of microglia and astrocytes throughout the central nervous system (CNS).[9] These inflammatory responses promote neuronal injury and apoptosis, thereby contributing to PD pathogenesis.[10] Within macrophage-

https://doi.org/10.2147/JIR.S607710
Received: 10 March 2026
Accepted: 6 May 2026
Published: 29 May 2026

and microglia-mediated inflammatory processes, activation of the NLRP3 inflammasome appears to be particularly important.[11] It has been implicated in modulating intracellular α-synuclein (α-syn) aggregation and dopaminergic neuronal survival.[12] Emerging evidence further suggests that peripheral factors, such as gut microbiota dysbiosis, may exacerbate inflammatory responses through activation of the gut–brain axis, thereby accelerating PD progression.[13] Together, these findings increasingly support a central role for neuroinflammation in PD pathogenesis. The emergence of single-cell sequencing, multi-omics approaches, and advanced imaging techniques has further accelerated knowledge generation in the field of PD-related neuroinflammation.[14–16] However, the limited sample sizes and lack of standardized protocols in many clinical studies constrain the generalizability of these findings.

Bibliometric analysis can effectively reveal the evolution of research topics, key contributors, collaborative networks, and emerging hotspots through the systematic analysis of large-scale scientific literature.[17] Compared with traditional systematic reviews, bibliometrics can objectively process large-scale data, reduce subjective bias, and help identify key authors, institutions, journals, and high-impact publications. In addition, it can reveal the clustering of research topics and their developmental trends.[18,19] Earlier bibliometric studies have provided preliminary insights into research trends on neuroinflammation in PD.[20] However, these studies mainly used the Web of Science Core Collection (WoSCC) for bibliometric analysis. Literature from PubMed was used only as supplementary clinical evidence and was not incorporated into the construction of the knowledge graph. As a result, the comprehensiveness of the study findings may have been limited. The period after 2023, furthermore, appears to mark a phase of rapid development, with increasing attention shifting from fundamental pathological mechanisms to clinical intervention strategies.

This study has three main contributions. First, by combining two databases, a more comprehensive depiction of the research landscape is provided. Second, the inclusion of recent literature enables a more timely identification of changes in research hotspots and emerging fields. Third, through multiple analytical dimensions, research in this field is categorized into three major areas: fundamental pathological mechanisms, diagnosis and biomarkers, and intervention strategies. This framework facilitates a clearer understanding of the knowledge structure of neuroinflammation research in PD and its potential translational directions.

Material and Methods

Data Retrieval and Search Strategy

This bibliometric study was based on publications retrieved from the WoSCC and Scopus, two widely used multidisciplinary databases.[21] We searched for literature published between January 1, 2015, and September 23, 2025. This period was selected because research on neuroinflammation in PD developed rapidly, and many influential studies were published during these years. The search strategy was as follows: TS = (“Parkinson’s disease” OR “Parkinson disease”) AND TS = (neuroinflammation). To ensure the accuracy of the study, we selected only those publications classified as “article” and “review”, included only English-language publications, and exported them in plain text format. After screening and deduplication across databases, 5,926 publications were included in the final analysis. The detailed search and screening workflow is shown in Figure 1. Literature screening and duplicate removal were performed independently by two researchers. Any disagreements were resolved by a third researcher. To minimize potential bias arising from ambiguity in author names, particularly common Chinese names, this study distinguished between authors using their institutional affiliations, Research IDs, and ORCIDs as provided by the WoSCC and Scopus databases.

Data Analysis and Visualization

Bibliometric analysis and visualizations were performed using CiteSpace (v6.3.3) and VOSviewer (v1.6.20), with additional support from Scimago Graphica, the bibliometrix R package (v4.4.3), GraphPad Prism (v10.4.1), and Microsoft Excel for data management. CiteSpace was used to construct collaboration networks among countries/regions, institutions, and authors. It was also used to identify co-citation structures, detect references with citation bursts, and perform keyword co-occurrence and clustering analysis to capture emerging themes and their evolution over time.[22] VOSviewer (v1.6.20) provides multiple visualization modes, including density view, cluster density view, and network view. The closely related nodes are divided into different colors for cluster analysis, which is used to generate a visual analysis of co-citation, such as co-citation of

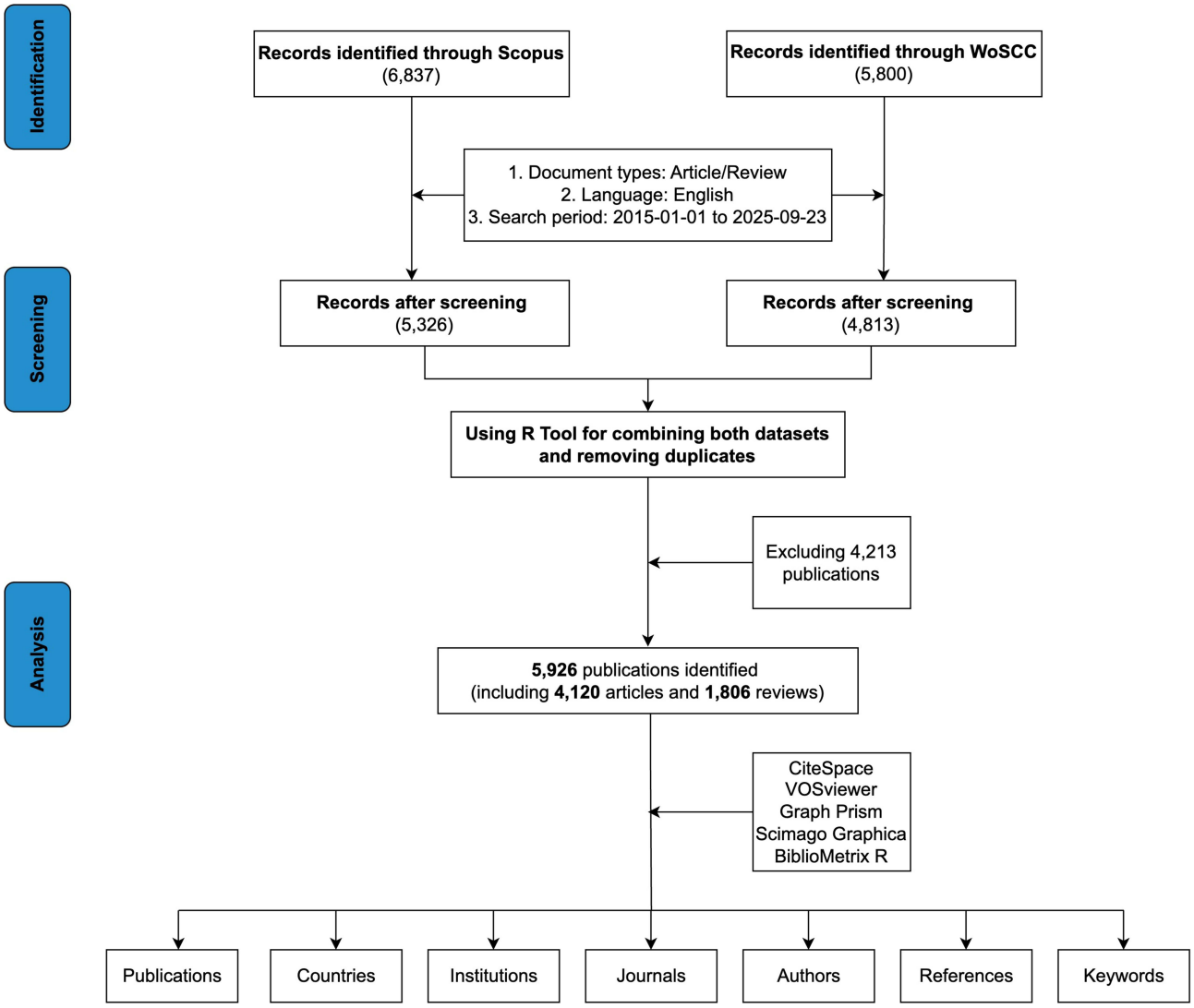

Figure 1 Flowchart illustrating the screening process for articles included in this study.

authors, references, and keyword frequency.[23] Scimago Graphica was employed to generate global maps illustrating international collaboration patterns. The bibliometrix package was utilized to summarize the principal bibliometric indicators, such as author productivity metrics and topic trend timelines.[24] GraphPad Prism was used to plot annual and cumulative publication trends and to present selected descriptive statistics; Excel was used for data cleaning and management.

Results

Analysis of Annual Publication Trends

A total of 5,926 publications on neuroinflammation in PD were included in the analysis, comprising 4,120 articles (69.5%) and 1,806 reviews (30.5%) (Figure 2A). As shown in Figure 2B, the annual publications of the relevant fields show a trend of steady growth. Publication output reached its highest level in 2022, with nearly 800 papers published, about 29.7% more than in 2021. This trend suggests that interest in the topic increased during this period. The cumulative number of publications also rose steadily over time. By September 23, 2025, the total number of publications had approached 6,000, showing continued interest in this research area.

https://doi.org/10.2147/JIR.S607710

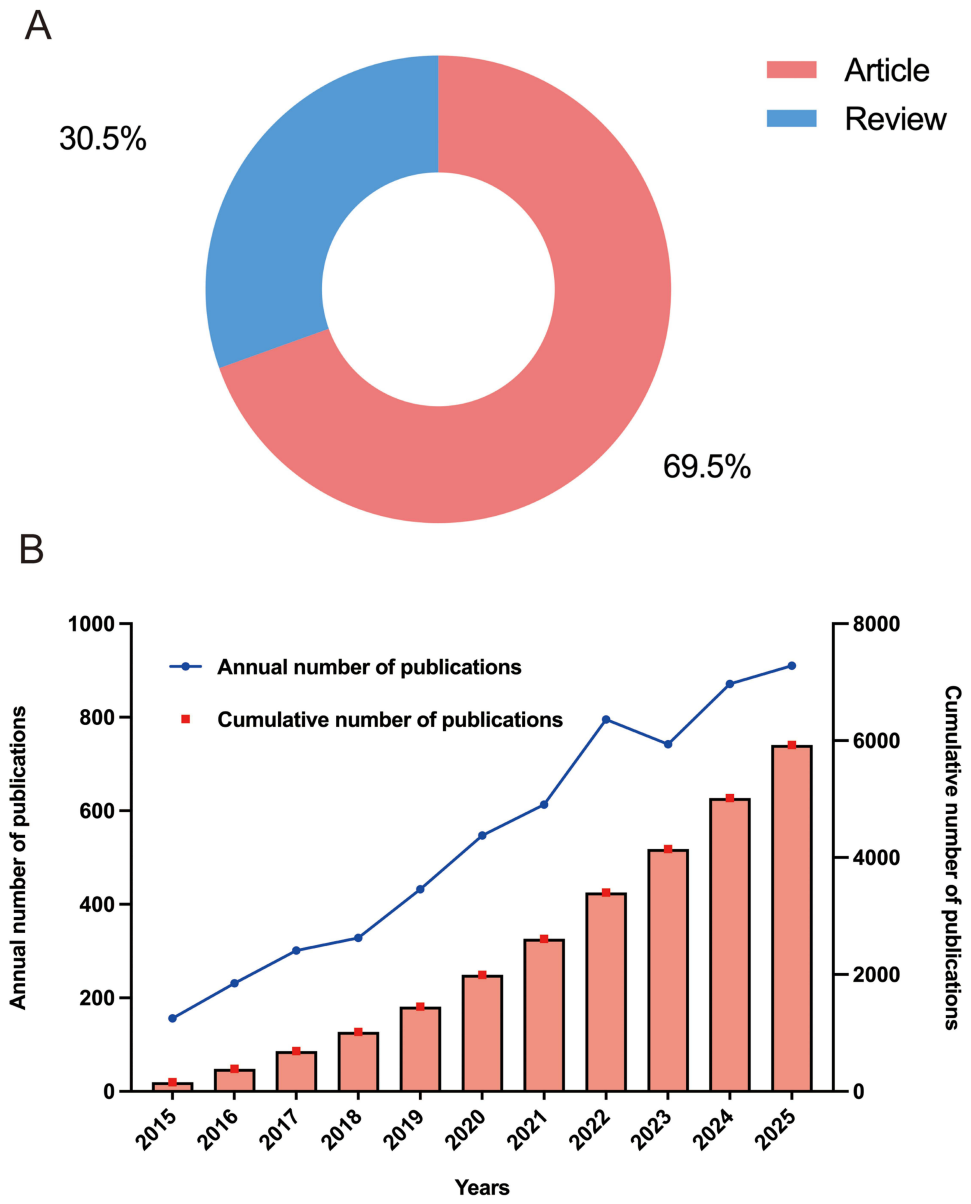

Figure 2 Annual and cumulative number of publications on neuroinflammation and PD research from 2015 to 2025. (**A**) Number of articles and reviews published per year. (**B**) Annual publications (blue line) and cumulative publications (Orange bars).

Analysis of Countries/Regions

Between 2015 and 2025, 80 countries and regions have put out relevant studies in this time frame. Table 1 shows the top 10 countries and regions in terms of the number of publications that they have generated. China was the first in terms of 1,822 publications (30.75%), then the United States with 1,258 publications (21.23%), and India with 619 publications (10.45%). Further examination of the geographical distribution in this field (Figure 3A), the shades of color indicate the intensity of the collaboration. The more the color is dark, the closer the cooperation of the country with others in this field of research. Among these, the United States and China show darker patches, which reflect their central position in the global partnership network. CiteSpace was also used to create a country/region collaboration network (Figure 3B). The size of each node stands for the publication output of that area. Larger nodes mean the corresponding region has higher research output and stronger influence in the field. The purple circles around the nodes of countries imply high centrality values (centrality > 0.1). The United States, Germany, and India had prominent nodes with purple rings, which is

Table 1 Top 10 Countries by Number of Publications in Neuroinflammation and PD Research from 2015 to 2025

Rank	Country	Counts	Centrality	H-Index
1	China	1822	0.00	30.70
2	USA	1258	0.19	59.20
3	India	619	0.38	21.00
4	Italy	463	0.11	42.70
5	South Korea	339	0.01	39.70
6	Germany	241	0.12	43.20
7	United Kingdom	234	0.03	36.10
8	Spain	207	0.03	49.90
9	Brazil	195	0.04	31.60
10	Australia	178	0.09	61.20

consistent with their active research participation and extensive global collaboration. In Figure 3C, the collaboration among countries is more intuitively visualized.

Analysis of Institutions

The top 10 institutions contributing to research on neuroinflammation in PD were listed in Table 2. In terms of publication output, the top three contributors are the University of California System (100 publications), Shanghai Jiao Tong University (73 publications), and Institut National de la Sante et de la Recherche Medicale (71 publications). Figure 4A lists the institutions with the highest publication output. The University of California system has the highest total number of citations (11,296), average citations per paper (108.62), and H-index (41). Harvard University has also demonstrated substantial academic influence, with an H-index of 33. Among the top ten institutions ranked by citation impact (Figure 4B), each institution has a citation intensity greater than 6, indicating periods of rapidly increasing scholarly attention. Since 2015, the U.S. Department of Veterans Affairs has shown the strongest citation burst, with a burst strength of 15.66.

Distribution of Journals

Figure 5A is a dual-map overlay of journals, which demonstrates the relationship and citation path that journals have with various subject areas. The citing journals on the left primarily come from fields such as clinical medicine, molecular biology, and immunology, reflecting the main channels through which research findings in this field are published. On the right side, the cited journals are mainly concentrated in molecular biology, genetics, and health sciences. The colored lines in the center represent the primary citation pathways between different disciplines, illustrating the flow of knowledge from the foundational base to the research frontiers. A thicker line usually indicates a stronger citation relationship along that path. Overall, research in this field exhibits distinct interdisciplinary characteristics, with research outputs primarily distributed across journals related to medicine and molecular biology, while the knowledge base primarily originates from research in molecular biology, genetics, and medicine, with some extensions into the fields of psychology and the social sciences. In addition, combined with the number of published papers, the total citation frequency and journal evaluation index for further analysis. Figure 5B and Table 3 list the top 10 journals in terms of published papers, mainly from the United States, the United Kingdom, Switzerland, and China, showing the country's strong scientific research capacity. The International Journal of Molecular Sciences has the highest number of papers (295 publications), and the Journal of Neuroinformation has the highest impact factor (IF=10.1, JCR Q1). This shows that the former has a strong research carrying capacity in this field, while the latter is more prominent in the impact index.

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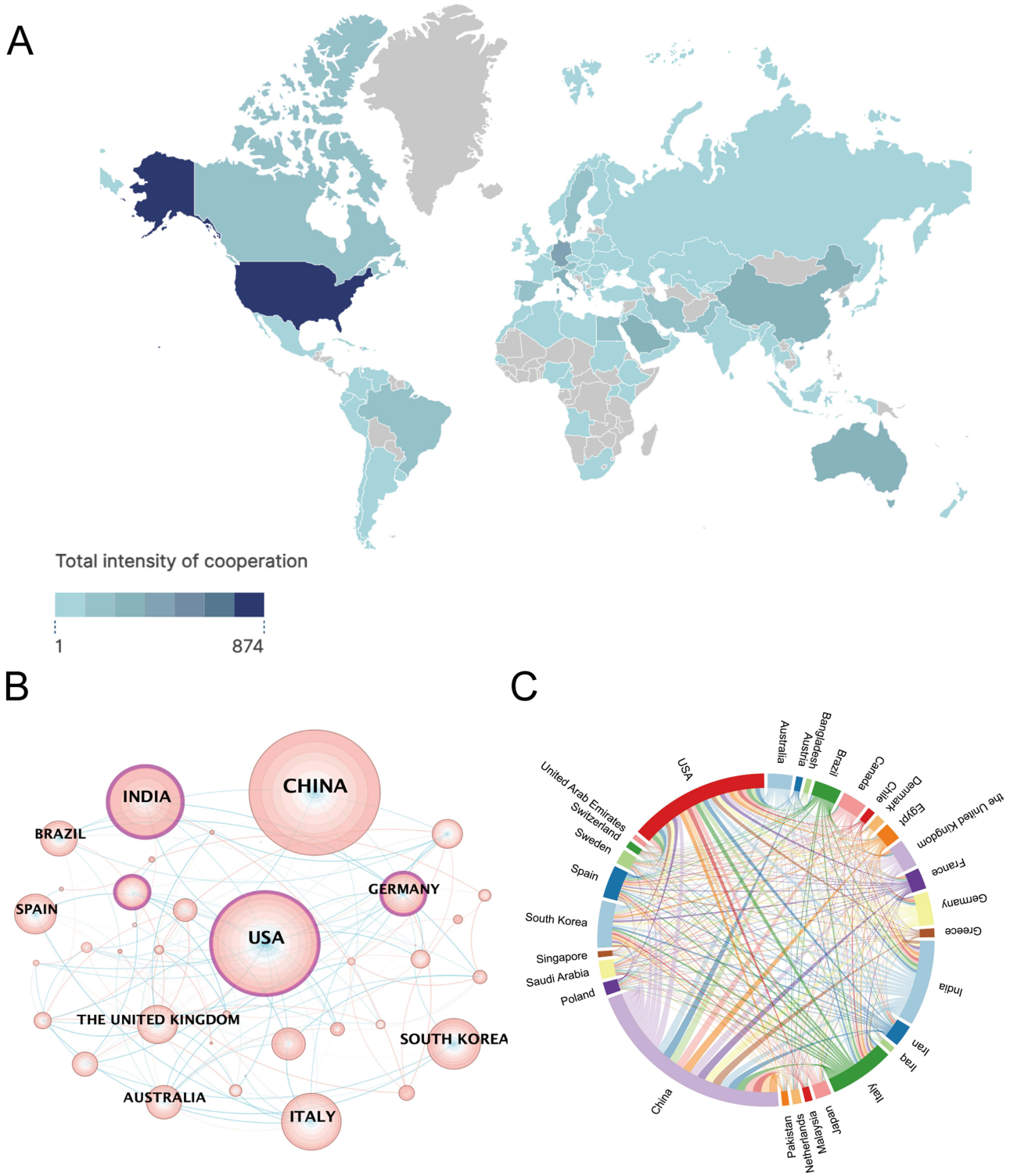

Figure 3 Geographic distribution and international collaboration in neuroinflammation and PD research from 2015 to 2025. (**A**) Global distribution of publications and collaboration intensity. Darker colors indicate stronger collaboration. (**B**) Visualization of publication networks by country. (**C**) Map of international research partnerships.

Analysis of Author Productivity and Co-Citation

The author collaboration network in Figure 6 illustrates the patterns of author collaboration and the distribution of academic influence in this field. It is drawn with CiteSpace, including 397 nodes and 474 connections, and the network density is 0.0006. Despite the overall limitation of collaboration in this field, authors within the same teams tend to work together more closely.

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Table 2 Top 10 Institutions Publishing Research on Neuroinflammation and PD from 2015 to 2025

Rank	Institutions	Country	Counts	Total Citations	Average Citations	H-Index
1	University of California System	USA	100	11,296	108.62	41
2	Shanghai Jiao Tong University	China	73	4876	61.72	29
3	Institut National de la Sante et de la Recherche Medicale	France	71	3133	38.68	32
4	CIBERNED	Spain	64	2669	38.68	27
5	Fudan University	China	63	1590	26.95	23
6	National Institutes of Health	China	63	4331	59.33	31
7	Nanjing Medical University	China	61	3194	47.67	31
8	Harvard University	USA	57	4446	63.51	33
9	Capital Medical University	China	54	1597	24.95	27
10	Jilin University	China	50	2079	41.58	24

Larger nodes represent greater publication output, while node colors correspond to the year of publication. Wang, Q.S., Zhang, F., and Cuzzocrea, S. were the top three authors, with 28, 25, and 24 publications, respectively. A relatively large collaborative team was observed around Wang, Q.S. in the network. Figure 6B provides a more intuitive visualization of Table 4 by evaluating author influence in terms of H-index, publication count, and citation count. Wang, Q.S. is the most prominent (n = 28), the reference count (n = 1786), and the H-index (n = 19) in this area. The author co-citation analysis shown in Figure 6C indicates that five authors were cited more than 600 times (Table 4). McGeer, P.L. ranked first with 1,024 citations, followed by Braak, H. (n = 949), Hirsch, E.C. (n = 736), Tansey, M.G. (n = 642), and Mogi, M. (n = 629). Therefore, these authors have played an important role in shaping the study of neuroinflammation in PD.

Analysis of Reference Bursts and Co-Citation References

A total of 253,818 references are cited in publications in this field, reflecting the extensive attention and theoretical accumulation in this direction. Based on the highly cited literature data in Figure 7A and Table 5, the most cited article is the one by McGeer, P.L. et al,[25] with 517 citations. This article was the first to clearly report HLA-DR-positive reactive microglia in the substantia nigra of PD patients with PD, which provided important evidence that neuroinflammation is involved in PD pathology. Hirsch, E.C. and Hunot, S.[26] published the second most frequently cited article, which has been cited 491 times. The authors showed that neuroinflammation plays an important part in the pathological process of PD on the basis of autopsy findings, in vivo studies, and animal models. They also proposed neuroinflammation as a potential target for neuroprotective therapy, shifting the field from pathological observation toward mechanistic investigation and therapeutic translation. Figure 7B shows 25 references with strong citation bursts, indicating their important role in shaping research hotspots in this area. Among them, the article "Parkinson's disease" by Kalia, L.V. and Lang, A.E.,[27] published in *The Lancet*, had the highest burst strength (57.5). This article describes PD as a neurodegenerative disease with substantial heterogeneity and complex clinical manifestations and treatment strategies, and it provides a relatively comprehensive conceptual framework for future research. The articles ranked second and third in impact score were those by Tansey, M.G. et al[35] and Wang, Q.Q. et al,[36] with strengths of 53.02 and 51.9, respectively. Both articles primarily explored the key roles of neuroinflammation and immune dysfunction, further emphasizing the significance of the gut-brain axis in mediating interactions between central and peripheral immune systems. These results are useful in conducting research in this area.

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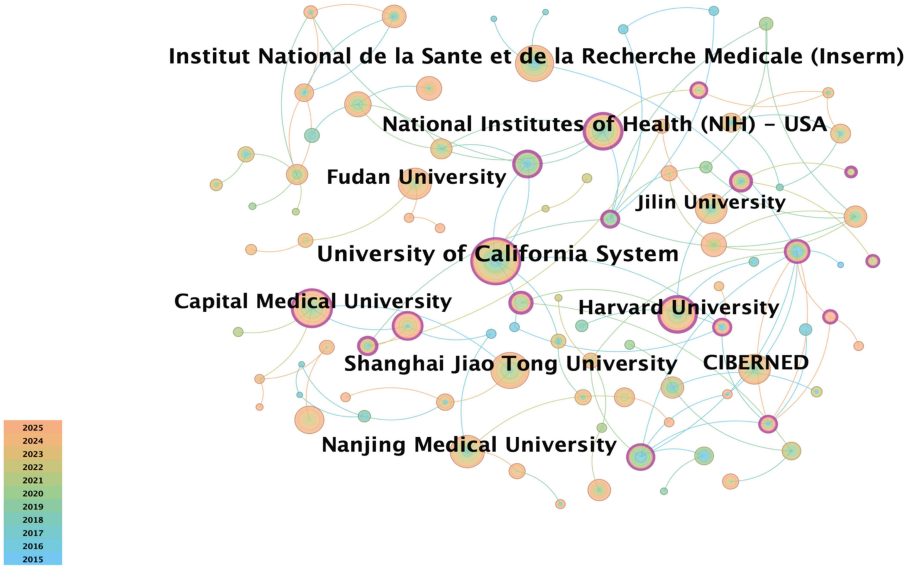

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Top 10 Institutions with the Strongest Citation Bursts

Institutions	Year	Strength	Begin	End	2015 – 2025
US Department of Veterans Affairs	2015	15.66	2015	2020	
Institut National de la Sante et de la Recherche Medicale (Inserm)	2015	7.11	2015	2017	
Institute of Materia Medica – CAMS	2017	8.53	2017	2019	
Veterans Health Administration (VHA)	2015	14.64	2018	2020	
State University System of Florida	2016	11.44	2019	2022	
NIH National Institute on Aging (NIA)	2019	6.75	2019	2020	
University of London	2016	6.27	2021	2023	
National Institute of Pharmaceutical Education & Research	2022	7.9	2022	2023	
Central South University	2023	9.04	2023	2025	
Prince Sattam Bin Abdulaziz University	2023	6.87	2023	2025	

Figure 4 Institutional contributions to neuroinflammation and PD research from 2015 to 2025. (**A**) Metrics of the top 10 institutions: number of publications, total citations, average citations per article, and H-index. (**B**) Top 10 institutions with the strongest citation bursts.

Analysis of Keywords

In terms of the thematic distribution and characteristics level, keyword analysis can better reveal the core themes of the research field and their evolving trends. We used VOSviewer to visualize the co-occurrence network of keywords in this field, as shown in Figure 8A. "Neuroinflammation" and "Parkinson's disease" occupy central positions in the co-occurrence network, forming the core of this research field. These terms are frequently associated with keywords such as "microglia", "α-synuclein" and "NLRP3" reflecting a strong focus on inflammation and protein aggregation mechanisms. In addition, there is significant interest in keywords such as "gut microbiota", "gastrointestinal microbiome", "blood-brain barrier" and "mitochondrial autophagy". Further keyword clustering analysis yielded a modularity value of $Q = 0.5586$ and an average silhouette coefficient of $S = 0.8079$, suggesting that the clustering structure was relatively stable and interpretable (Figure 8B). However, it should be noted that keyword clustering is based on algorithms that analyze co-occurrence relationships, and the resulting clusters are more suitable for identifying relatively concentrated research themes. Some degree of overlap and intersection may therefore exist between clusters. Despite this limitation, the analysis is still of great value for a comprehensive understanding of its knowledge structure. We summarize research in this domain into three major themes: pathophysiological mechanisms (#1 mitochondria, #3 neuroinflammation, #4 alpha-synuclein, #6 neurodegeneration, #8 NLRP3, #9 gut-brain axis), clinical diagnosis (#0 Parkinson's disease, #5 human, #7 neuroimaging, #11 COVID-19, #12 biomarkers), and drug intervention/therapy (#2 neuroprotective agents,

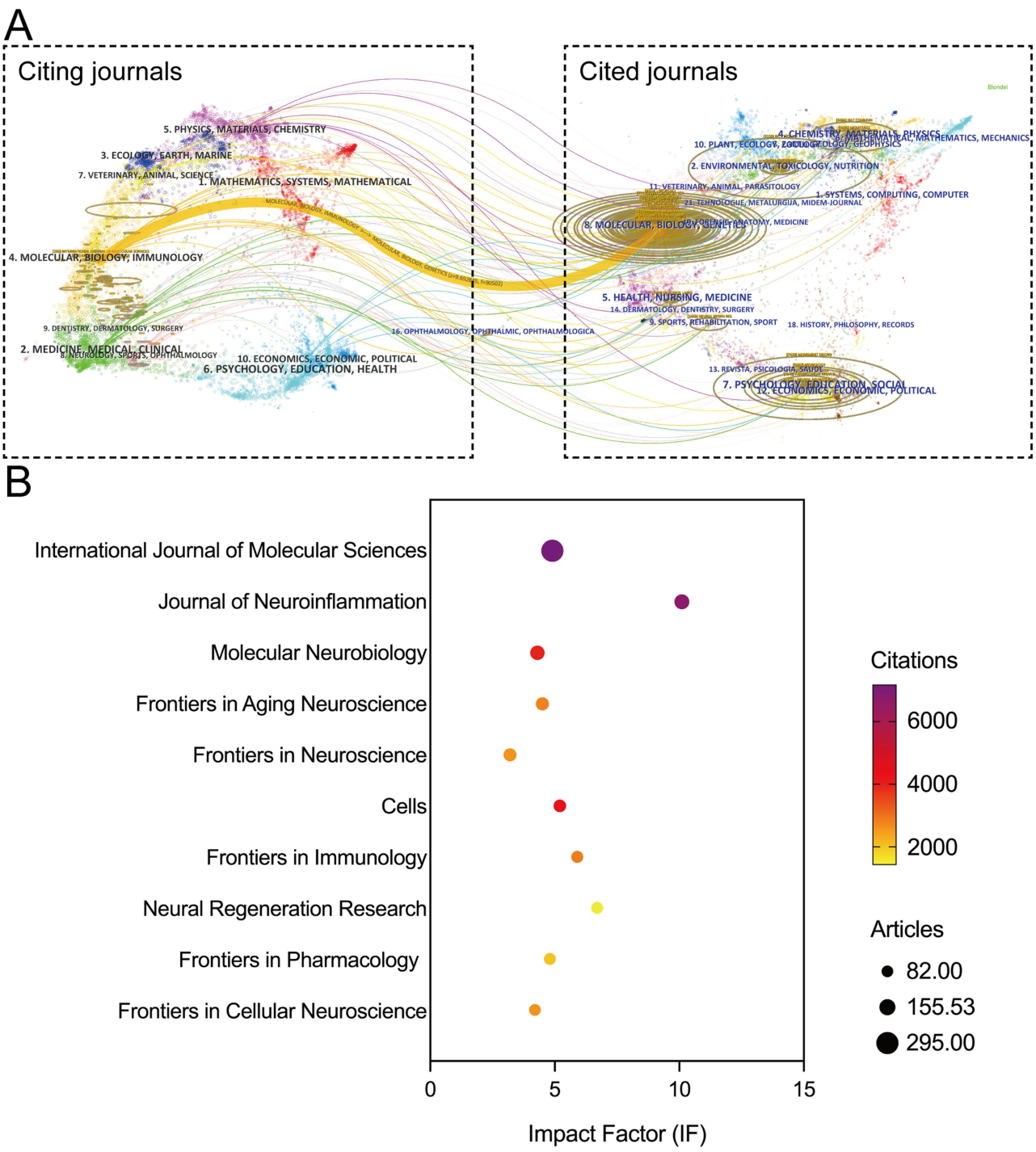

Figure 5 Journal impact in neuroinflammation and PD research from 2015 to 2025. (**A**) Dual-map overlay. Citing journals are on the left, and cited journals are on the right. (**B**) Top 10 journals by number of publications.

#10 nanoparticles and #13 extracellular vesicles). Moreover, the timeline chart of trend themes further indicates that research hotspots have shifted over time (Figure 8C). Research in this field no longer centers mainly on early preclinical topics such as neurotoxins, genetics, or cell and animal models. Greater attention is now being paid to the mechanisms that drive disease progression. In this context, ferroptosis has attracted increasing interest and is being discussed in relation to oxidative stress and inflammation. Meanwhile, the emergence of keywords such as "drug therapy" and "blood–brain barrier" suggests that research is shifting toward therapeutic strategies and clinical translation.

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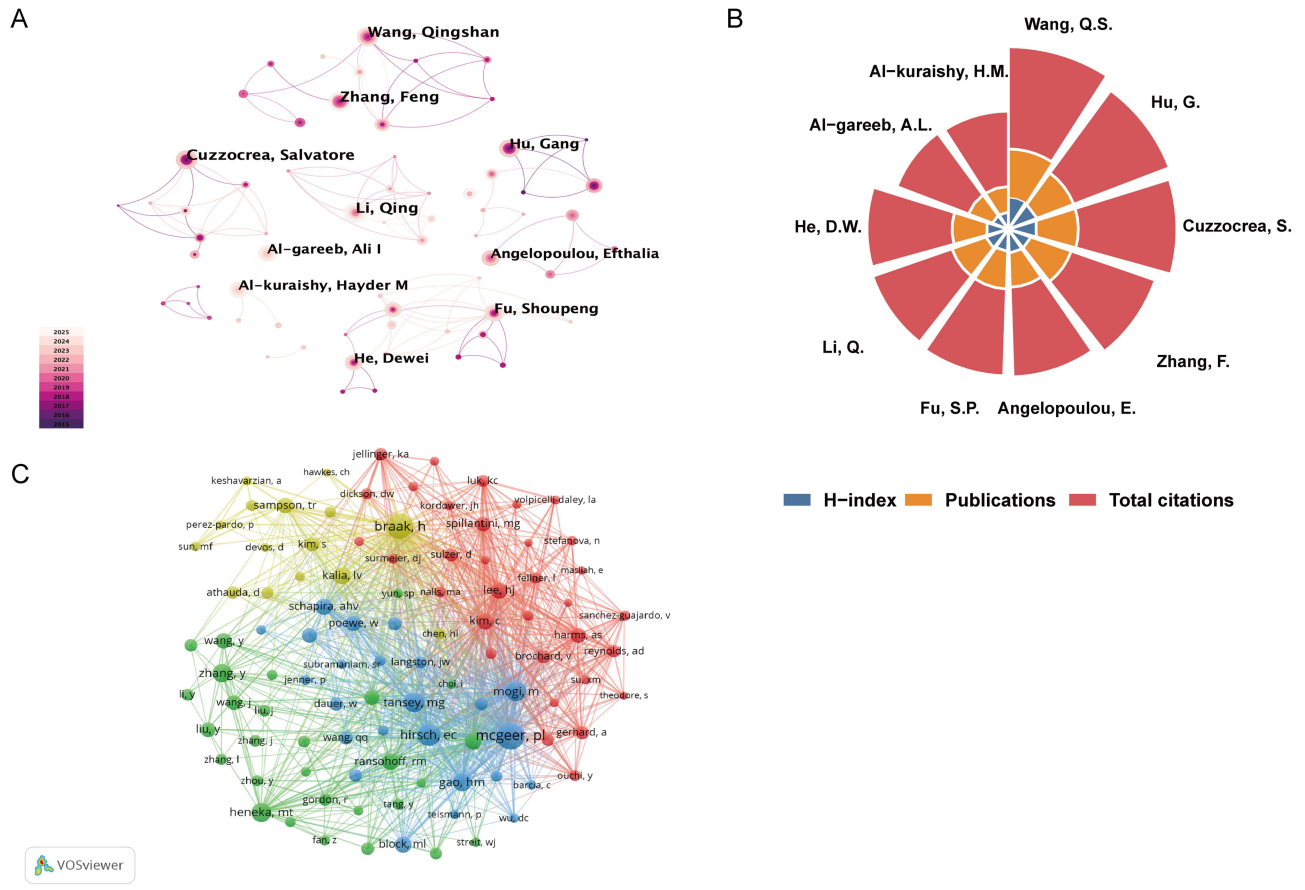

Figure 6 Authorship and co-authorship networks in neuroinflammation and PD research from 2015 to 2025. (**A**) Collaborative network of lead authors. (**B**) Top 10 authors ranked by publication count, total citations, and H-index. (**C**) Co-citation network of authors.

Discussion

General Overview

In this study, we performed a comprehensive literature search on neuroinflammation in PD using the WoSCC and Scopus databases. The objective is to gain a comprehensive understanding of the current state of research, major trends, and future directions in this field. In terms of publication output, the annual number of papers increased steadily overall, reaching a peak in 2022, followed by a modest decrease in 2023. This pattern may reflect several influences. During and after the COVID-19 pandemic, neuroinflammation and its relevance to neurological disorders received heightened

Table 3 Top 10 Journals Publishing Research on Neuroinflammation and PD from 2015 to 2025

Rank	Journals	Counts	Total Citations	IF	Country	JCR-c
1	International Journal of Molecular Sciences	295	7154	4.9	USA	Q1
2	Journal of Neuroinflammation	133	6745	10.1	UK	Q1
3	Molecular Neurobiology	124	4017	4.3	USA	Q1
4	Frontiers in Aging Neuroscience	103	2817	4.5	Switzerland	Q1
5	Frontiers in Neuroscience	101	2621	3.2	Switzerland	Q2
6	Cells	95	4358	5.2	Switzerland	Q2

(*Continued*)

Table 3 (Continued).

Rank	Journals	Counts	Total Citations	IF	Country	JCR-c
7	Frontiers in Immunology	86	2879	5.9	Switzerland	Q1
8	Neural Regeneration Research	84	1450	6.7	China	Q1
9	Frontiers in Pharmacology	83	2054	4.8	Switzerland	Q1
10	Frontiers in Cellular Neuroscience	82	2617	4.2	Switzerland	Q2

Table 4 Top 10 Most Productive and Most Co-Citation Authors in Neuroinflammation and PD Research from 2015 to 2025

Rank	Author	Counts	Total Citations	H-index	Co-Citation Authors	Citations
1	Wang, Q.S.	28	1786	19	Mcgeer, P.L.	1024
2	Zhang, F.	25	675	14	Braak, H.	949
3	Cuzzocrea, S.	24	1371	17	Hirsch, E.C.	736
4	Hu, G.	23	1826	18	Tansey, M.G.	642
5	Fu, S.P.	22	578	13	Mogi, M.	629
6	Li, Q.	21	467	14	Heneka, M.T.	599
7	Angelopoulou, E.	20	710	14	Gao, H.M.	559
8	He, D.W.	20	508	13	Zhang,Y.	533
9	Al-gareeb,A.L.	15	275	10	Kalia, L.V.	478
10	Al-kuraishy, H.M.	15	250	10	Ransohoff, R.M.	462

attention.[37] At the same time, rapidly advancing approaches, such as single-cell sequencing, transcriptomic profiling, and other high-throughput methods, have been increasingly adopted in this area.[38–40] Therefore, the slight decline in 2023 can be understood as a short-term adjustment after the peak in 2022, not a long-term trend of recession.

Further analysis shows that the United States and China have the highest publication output in this domain. Their strong economic strength and continuous investment in research facilities are conducive to producing high-level research results. The growing share of elderly individuals has increased the urgency of PD-related research and will also promote the funding and research activities of the two countries. Although China ranks first by publication volume, its network centrality remains relatively low. In China, research in this field is still primarily conducted by independent research teams, with relatively limited international collaboration. This also explains why Chinese academic papers have relatively limited international influence. It also suggests that China's research in this field is not sufficiently integrated into the global scientific discourse. Strengthening cross-regional collaboration and expanding international academic exchange could help increase both the visibility and influence of future research findings from China. Compared with others, the United States (H-index = 59.20) and Australia (H-index = 61.20), due to close collaboration among researchers and advanced research facilities, are better positioned to produce high-quality academic output and enjoy greater international recognition.

At the institutional level, the University of California system is among the leading contributors to PD research worldwide, reflecting its strong academic influence and extensive international collaboration. Studies associated with this system have advanced understanding of PD-related inflammation by suggesting that inflammatory responses occur not

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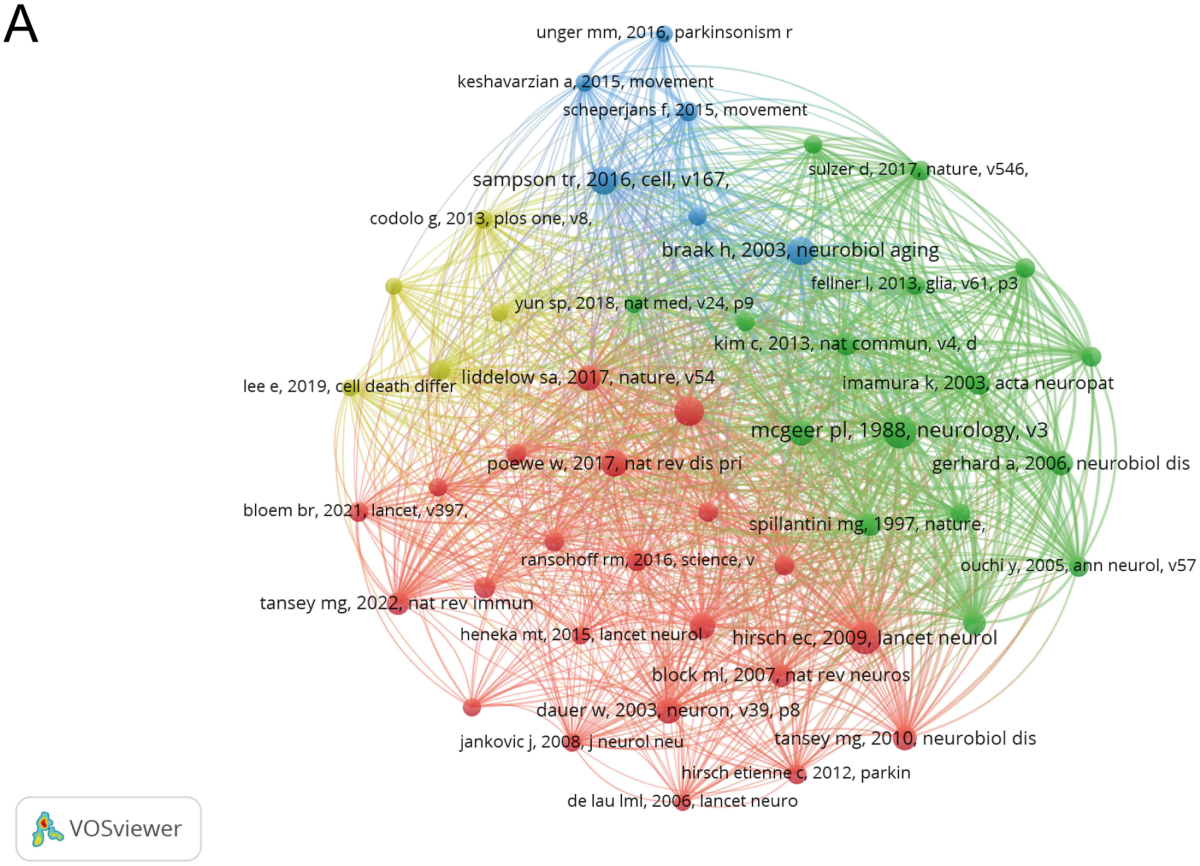

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Top 25 References with the Strongest Citation Bursts

References	Year	Strength	Begin	End	2015 – 2025
Kim C, 2013, NAT COMMUN, V4, P0, DOI 10.1038/ncomms2534, DOI	2013	34.13	**2015**	2018	
Fellner L, 2013, GLIA, V61, P349, DOI 10.1002/glia.22437, DOI	2013	28.06	**2015**	2018	
Harms AS, 2013, J NEUROSCI, V33, P9592, DOI 10.1523/JNEUROSCI.5610–12.2013, DOI	2013	21.44	**2015**	2018	
Hirsch Etienne C, 2012, PARKINSONISM RELAT DISORD, V18 Suppl 1, PS210, DOI 10.1016/S1353–8020(11)70065–7, DOI	2012	20.98	**2015**	2017	
Codolo G, 2013, PLOS ONE, V8, P0, DOI 10.1371/journal.pone.0055375, DOI	2013	20.34	**2015**	2018	
Wang QQ, 2015, TRANSL NEURODEGENER, V4, P0, DOI 10.1186/s40035–015–0042–0, DOI	2015	51.9	**2016**	2020	
Tang Y, 2016, MOL NEUROBIOL, V53, P1181, DOI 10.1007/s12035–014–9070–5, DOI	2016	22.33	**2016**	2021	
Heneka MT, 2015, LANCET NEUROL, V14, P388, DOI 10.1016/S1474–4422(15)70016–5, DOI	2015	22.21	**2016**	2020	
Yan YQ, 2015, CELL, V160, P62, DOI 10.1016/j.cell.2014.11.047, DOI	2015	19.64	**2016**	2020	
Sampson TR, 2016, CELL, V167, P1469, DOI 10.1016/j.cell.2016.11.018, DOI	2016	42.24	**2017**	2021	
Ransohoff RM, 2016, SCIENCE, V353, P777, DOI 10.1126/science.aag2590, DOI	2016	38.15	**2017**	2021	
Zhou Y, 2016, MOL NEURODEGENER, V11, P0, DOI 10.1186/s13024–016–0094–3, DOI	2016	21.54	**2017**	2021	
Qin XY, 2016, JAMA NEUROL, V73, P1316, DOI 10.1001/jamaneurol.2016.2742, DOI	2016	19.85	**2017**	2021	
Kalia LV, 2015, LANCET, V386, P896, DOI 10.1016/S0140–6736(14)61393–3, DOI	2015	57.5	**2018**	2020	
Liddelow SA, 2017, NATURE, V541, P481, DOI 10.1038/nature21029, DOI	2017	33.68	**2018**	2022	
Sulzer D, 2017, NATURE, V546, P656, DOI 10.1038/nature22815, DOI	2017	28.1	**2018**	2022	
Scheperjans F, 2015, MOVEMENT DISORD, V30, P350, DOI 10.1002/mds.26069, DOI	2015	26.45	**2018**	2020	
Unger MM, 2016, PARKINSONISM RELAT D, V32, P66, DOI 10.1016/j.parkreldis.2016.08.019, DOI	2016	21.61	**2018**	2021	
Keshavarzian A, 2015, MOVEMENT DISORD, V30, P1351, DOI 10.1002/mds.26307, DOI	2015	19.58	**2018**	2020	
Poewe W, 2017, NAT REV DIS PRIMERS, V3, P0, DOI 10.1038/nrdp.2017.13, DOI	2017	41.7	**2019**	2022	
Tysnes OB, 2017, J NEURAL TRANSM, V124, P901, DOI 10.1007/s00702–017–1686–y, DOI	2017	18.91	**2019**	2022	
Gordon R, 2018, SCI TRANSL MED, V10, P0, DOI 10.1126/scitranslmed.aah4066, DOI	2018	22.5	**2020**	2023	
Tansey MG, 2022, NAT REV IMMUNOL, V22, P657, DOI 10.1038/s41577–022–00684–6, DOI	2022	53.02	**2023**	2025	
Bloem BR, 2021, LANCET, V397, P2284, DOI 10.1016/S0140–6736(21)00218–X, DOI	2021	33.14	**2023**	2025	
Kwon HS, 2020, TRANSL NEURODEGENER, V9, P0, DOI 10.1186/s40035–020–00221–2, DOI	2020	27.16	**2023**	2025	

Figure 7 Reference co-cited analysis in neuroinflammation and PD research from 2015 to 2025. (**A**) Top 50 most frequently co-citation references (visualized using VOSviewer). (**B**) Top 25 references with the strongest citation bursts.

Table 5 Top 10 Most Frequently Co-Citation References in Neuroinflammation and PD Research from 2015 to 2025

Author	Title	Journal	Co-Citation	Year
McGeer, P.L. et al[25]	Reactive microglia are positive for HLA-DR in the substantia nigra of Parkinson's and Alzheimer's disease brains	Neurology	517	1988
Hirsch, E.C. et al[26]	Neuroinflammation in Parkinson's disease: a target for neuroprotection?	The Lancet Neurology	491	2009
Kalia, L. V. et al[27]	Parkinson's disease	The Lancet	397	2015
Braak, H. et al[28]	Staging of brain pathology related to sporadic Parkinson's disease	Neurobiology of aging	361	2003
Sampson, T.R. et al[29]	Gut microbiota regulate motor deficits and neuroinflammation in a model of Parkinson's disease	Cell	345	2016
Poewe, W. et al[30]	Parkinson disease	Nature reviews Disease primers	333	2017
Brochard,V. et al[31]	Infiltration of CD4+ lymphocytes into the brain contributes to neurodegeneration in a mouse model of Parkinson disease	The Journal of clinical investigation	307	2008
Liddelow, S.A. et al[32]	Neurotoxic reactive astrocytes are induced by activated microglia	Nature	303	2017
Dauer, W. et al[33]	Parkinson's disease: mechanisms and models	Neuron	296	2003
Glass, C.K. et al[34]	Mechanisms underlying inflammation in neurodegeneration	Cell	281	2010

only in the brain but also in peripheral organs such as the gut.[41] This process may be related to α-syn-triggered immune activation, which may help explain non-motor symptoms of PD, including constipation, dysphagia, and cognitive impairment.[42] This progress also demonstrates that long-term scientific research and first-rate resource allocation can promote knowledge sharing in relevant fields worldwide, thereby providing a solid foundation for the production of global scientific research outcomes. At the same time, the rise of a large scientific research country, especially in China, shows that the research on PD neuroinflammation is becoming more and more extensive under the continuous capital investment and international cooperation background.

The double-map overlay of the journal also shows the interdisciplinary characteristics and knowledge flow path. The result shows that the study of PD turns out to be about biological mechanisms, rather than simply relying on symptom management. The joint citation analysis shows that JCR Q1 journals account for about 70% of the ten most commonly cited institutions, indicating that high-quality journals have far-reaching influence. Among them, the open access (OA) publishing path, exemplified by the Journal of Neuroinflammation and the Frontiers series of journals in Switzerland, has enhanced the visibility of research results, thereby promoting the dissemination and sharing of global scientific knowledge. It should be noted that the total citation frequency exhibits a significant time-accumulation effect, and journals that published related research earlier may thus obtain higher cumulative citation counts. We used this metric as a descriptive indicator and interpreted it together with the IF and JCR classification to provide a comprehensive analysis of journal characteristics.

Within the author collaboration network, Wang Q.S.'s team has emerged as a rising force. However, this development has also exposed underlying issues. Current efforts are primarily focused on relatively independent, small-scale research collaborations, with a lack of large-scale, cross-team, and cross-border exchanges. This has, to some extent, hindered the production of innovative and high-quality research outcomes. The small-scale collaboration model can lead to rigid thinking among researchers, preventing breakthrough progress. In contrast, international collaboration can promote the dissemination of knowledge and technology through multiple perspectives and dimensions, address the issue of sample

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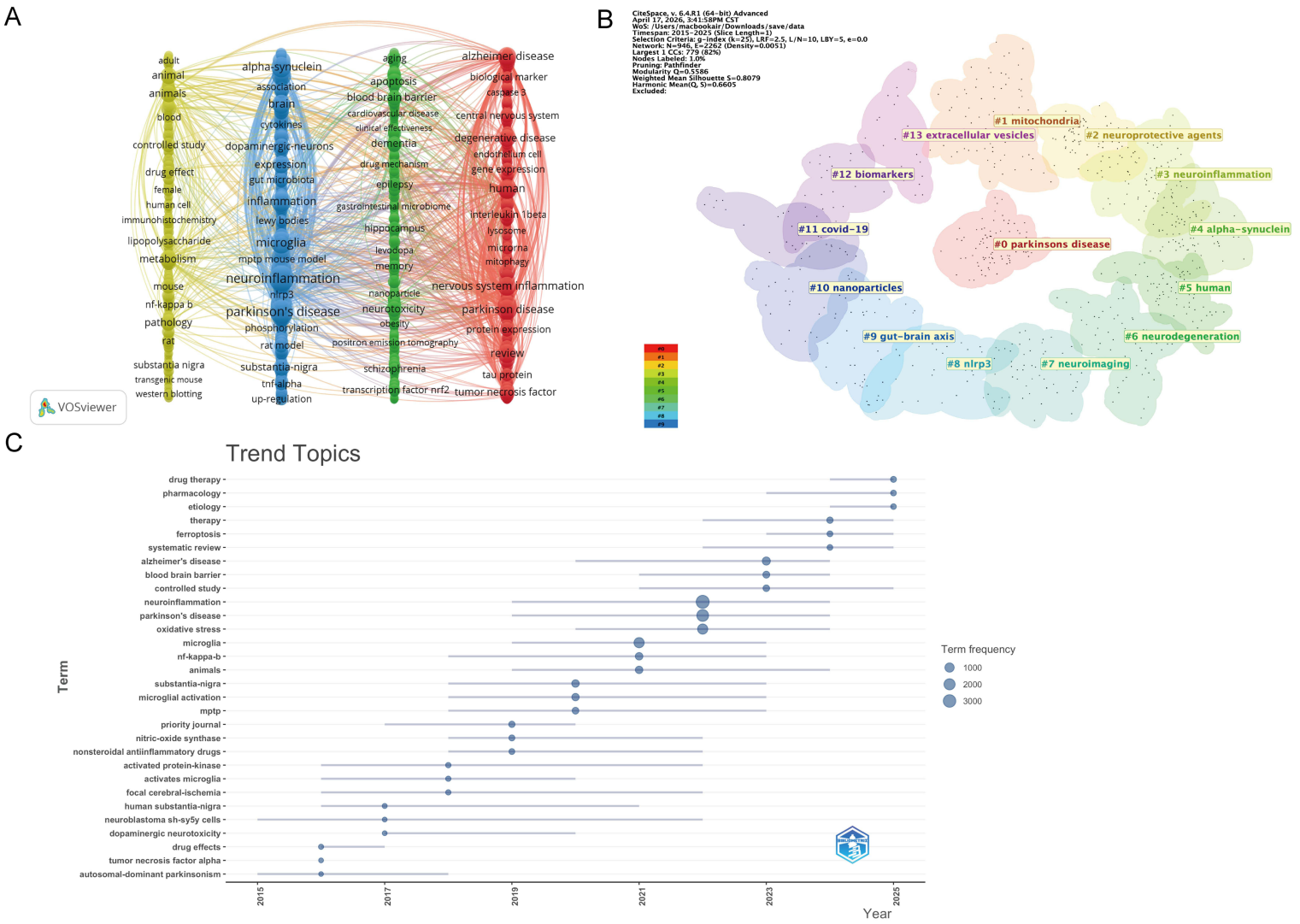

Figure 8 Keyword analysis in neuroinflammation and PD research from 2015 to 2025. (**A**) Keyword co-occurrence network visualization. (**B**) Cluster analysis of keywords. (**C**) Keyword trend timeline chart.

heterogeneity commonly found in multicenter clinical cohort studies, and enhance the generalizability and accuracy of research findings.

Research Contributions

When examining highly cited authors and references, McGeer et al provided early evidence for an inflammatory component in PD by reporting markedly increased HLA-DR–positive reactive microglia in the substantia nigra of PD brain tissue.[25] Braak et al proposed that the affected regions in PD patients do not originate in the substantia nigra, but may initially involve areas such as the glossopharyngeal nerve and the dorsal motor nucleus of the vagus nerve.[28] This hypothesis explains the clinical course of PD and extends the researchers' focus from the core mechanism to the peripheral mechanism. For this reason, numerous studies have been carried out on PD animal models, including toxin-based paradigms (eg., 6-hydroxydopamine, MPTP, paraquat, and rotenone) and genetic models involving PD-related genes (eg., synuclein, parkin, and ubiquitin C-terminal hydrolase-L1).[33] This development reflects a shift in PD research toward systematic and mechanistic validation, thereby providing essential groundwork for early detection and intervention of PD. Hirsch and Hunot identified neuroinflammation as a key pathological feature of PD.[26] They reported that microglial activation in the substantia nigra, immune-cell infiltration, and increased levels of pro-inflammatory mediators contribute to dopaminergic neurodegeneration. They also described immunomodulatory treatments, including PPARγ agonists, as potential neuroprotective strategies. Most of the available support still comes from animal models, while convincing clinical confirmation is only early stage. To advance translation, future work should prioritize developing models that better mirror neurodegeneration in human PD and identifying dependable neurobiological biomarkers. In recent years, increasing public recognition has been given to microbiome technology, notably around 2015. Scheperjans et al found that the intestinal microbiota of PD patients was different from that of the healthy control group and was related to the motor phenotype.[43] The subsequent work can be used to link flora disorders with PD through pathways related to abnormal α-syn aggregation and inflammatory signaling.[44] It can be seen that focusing attention on diseases with interaction between the gut-brain axis and peripheral immunity has become an important aspect of explaining the pathogenesis of PD.

The phenomenon of citation bursts highlights the research focus of this field. These scholars mainly pay attention to the individual pathological mechanism of neuroinflammation, explore the polarization of microglia cells and their impact on dopaminergic neurons, and then provide theoretical support for anti-inflammatory therapy targeting glial cells.[36] Notably, Tansey et al propose PD as a multisystem disorder involving interactions between central and peripheral immune systems.[35] Specifically, gut microbiota dysbiosis and intestinal inflammation may trigger peripheral inflammatory responses and promote monocyte and T-cell activation. As a consequence, inflammatory activity and neuronal injury within the brain may be intensified through increased permeability of the blood-brain barrier (BBB), which permits the migration of peripheral immune cells into the CNS. However, current clinical treatments for PD cannot reverse the neurodegenerative process. Further research is needed to identify useful biomarkers for diagnosing early precursor conditions. Additionally, personalized therapies tailored to different disease stages are required, alongside enhanced management of late-stage symptoms.[27]

Research Hotspots and Development Trends

Keyword cluster, co-occurrence analysis, and the temporal evolution of trending topics can provide a basis for identifying research hotspots and development trends. Based on the results of the keyword analysis described above, research in the field of neuroinflammation in PD between 2015 and 2025 has primarily focused on three areas: fundamental pathological mechanisms, clinical diagnosis and biomarkers, and intervention strategies.

Core Pathological Mechanism

As shown by the keyword analysis, it is evident that research into the mechanisms of PD-associated neuroinflammation has evolved from early studies that focused solely on whether microglia were "activated" to a comprehensive research framework involving the interplay of multiple mechanisms, including protein homeostasis imbalance, mitochondrial dysfunction, inflammasome activation, cell death, and the gut-brain axis. It should be noted that although keyword

clustering analysis identified "#1 mitochondria", "#3 neuroinflammation" and "#4 alpha-synuclein" as distinct themes, these themes are not completely independent at the biological level. There is a clear overlap between them. Neuroinflammation is now considered to be the main pathological feature of PD and one of the main factors of the degeneration of dopaminergic neurons.[45] A large amount of evidence has proved that these inflammatory processes are caused by microglia, and the innate immune response mediated by microglia plays a central role.[46–48] When over-activated, Microglia will secrete pro-inflammatory mediators, which will cause damage to dopaminergic neurons.[49] Recent studies have further found that simple M1 and M2 classifications cannot reflect the phenotype of microcolloids well.[50] Single-cell RNA sequencing reveals the obvious space-time heterogeneity in the brain region and microglial groups under the background of disease, providing a more refined angle for the inflammatory regulation of PD.[51]

Combining the results of keyword co-occurrence networks and cluster analysis, mitochondrial dysfunction and its associated inflammatory amplification pathways have emerged as a key research direction in PD neuroinflammation, with increasing attention focused on neuroinflammatory responses mediated by the "mitochondrial autophagy–NLRP3 inflammasome" axis.[52] In the co-occurrence network, "nlrp3" is closely associated with keywords such as "microglia", "alpha-synuclein" and "inflammation". The emergence of "#8 nlrp3" in the clustering analysis also suggests that the NLRP3 inflammasome has become a relatively independent and active mechanistic branch in this field. Research shows that defective mitophagy promotes the accumulation of oxidatively damaged mitochondrial genetic material, elevated intracellular oxidants, lipid components such as cardiolipin, and other mitochondria-associated danger signals (mtDAMPs), promoting sustained NLRP3 activation and enhancing IL-1β/IL-18 maturation, thereby driving inflammatory cascades.[53] Further studies confirm that hereditary PD gene mutations (eg., PINK1 and Parkin) impair mitochondrial turnover, thereby causing increased ROS buildup and subsequent neuronal death.[54] Notably, α-syn aggregation may modulate the NLRP3 inflammasome via mitochondrial and autophagic pathways, further exacerbating neuroinflammation and dopamine loss.[55] However, how the NLRP3 inflammasome and α-syn expression reciprocally interact remains unclear. The NLRP3 inflammasome not only participates in inflammatory cascades but also plays a key role in linking mitochondrial damage to immune-mediated inflammatory responses. Further research on this pathway will contribute to a deeper understanding of the pathological mechanisms underlying neuroinflammation in PD and provide a basis for identifying relevant therapeutic targets.

A closer look at the temporal evolution of trending topics reveals that ferroptosis has emerged as a topic of growing interest since 2023, suggesting that it has become one of the cellular damage mechanisms worthy of attention in neuroinflammation in PD.[56,57] Furthermore, mononuclear transcriptomic and spatial transcriptomic studies have further revealed that FGF signaling between oligodendrocytes and astrocytes is significantly impaired in PD, accompanied by elevated Ca^{2+} levels and inhibition of the NRF2/SLC7A11/GPX4 pathway.[58] This suggests that ferroptosis is not merely a downstream pathological consequence of neuronal dysfunction, but may serve as a critical link connecting glial imbalance to dopaminergic neuronal degeneration. Research has shown that ferroptosis in microglia itself can exacerbate α-syn deposition and impairments in voluntary motor function in PD mice. Research has shown that ferroptosis in microglia itself can exacerbate α-syn deposition and impairments in voluntary motor function in PD mice.[59] Under α-syn stimulation, activation of the p62/Keap1/Nrf2 signaling pathway can mitigate ferroptosis, thereby suppressing excessive microglial activation and neuronal apoptosis, which further demonstrates the association between ferroptosis and neuroinflammation in PD.[60] Through integrated bioanalytical approaches, researchers identified key genes associated with ferroptosis and lipid metabolism in PD, including CBS, PRKAR2B, and RELA. These genes are linked to neuroinflammation and immune dysregulation and hold significant value for the diagnosis and treatment of PD.[61] Consequently, research in this field may shift from single anti-inflammatory strategies toward combined intervention strategies that simultaneously target ferroptosis and neuroinflammation.

Furthermore, the connections between keywords such as "#9 gut-brain axis" in the cluster analysis and "gut microbiota" and "gastrointestinal microbiome" in the co-occurrence network indicate that research on the gut-brain axis is emerging as a key focus in mechanistic studies within this field. A human autopsy study suggests that the dissemination of α-syn–related lesions may originate in the enteric nervous system (ENS) and propagate to the CNS via the vagus nerve, a hypothesis thoroughly validated in multiple animal experiments.[62] For example, colonic injection of α-syn preformed fibrils (PFFs) in mice can trigger intestinal inflammation and activation of enteric glial cells, together with

detectable inflammatory responses in the brain and neurodegenerative changes. It can be seen from the results that the intestine is connected with the brain and participates in the pathological development related to PD.[63] In terms of mechanism, the disorder of intestinal microbiota can cause changes in the characteristics of metabolites, weaken the integrity of the mucosal barrier, and increase peripheral inflammatory signals, which further worsens dopaminergic neuron loss.[64] Therefore, the emerging field of gut-brain axis research believes that the mutual influence of peripheral immune imbalance and CNS inflammation is implicated in PD development, providing a new direction for the research of new biomarkers and intervention methods.

It should be noted that, although protein palmitoylation did not emerge as a prominent hotspot in the keyword analysis of the present study, recent evidence suggests that it may represent an emerging area of interest in the field of neuroinflammation in PD. Studies have indicated that aberrant palmitoylation may contribute to PD-related pathological processes by modulating NLRP3 inflammasome activation and regulating the involvement of proteins such as Syt11 in the abnormal aggregation of α-syn.[65,66] In addition, a Mendelian randomization analysis suggested a potential causal association between the palmitoylation-related gene ZDHHC8 and PD risk.[67] Furthermore, DNA methylation can reduce the overexpression of the ZDHHC13 gene, thereby lowering the risk of developing PD.[68] These findings demonstrate that abnormalities in palmitoylation hold promise as an emerging area of research warranting continued attention in this field and may serve as biomarkers for the early diagnosis and treatment of PD.

Clinical Diagnosis and Biomarkers

The keyword clustering analysis indicated that, within PD neuroinflammation research, clinical diagnosis is primarily centered on topics such as "#7 neuroimaging" and "#12 biomarkers". This indicates that the research is gradually shifting from the exploration of underlying mechanisms toward early diagnosis, disease stratification, and treatment efficacy assessment. The clinical diagnosis of PD is mainly determined by the symptoms, but these symptoms generally appear in the late stage of the disease, thus affecting early detection and timely treatment. Biomarkers may also serve as indicators for assessing the progression of PD pathology and therapeutic efficacy.[69] A case–control study using multiparametric flow cytometry to profile cerebrospinal fluid (CSF) from patients with PD reported a higher fraction of non-classical monocytes in the CSF, together with activation of both $CD4^+$ and $CD8^+$ T cells.[70] Furthermore, a meta-analysis confirmed that PD is associated with inflammatory responses in both peripheral blood and CSF: compared to healthy controls, PD patients exhibited significant differences in biomarker levels (TNF-α, IL-1β and MCP-1) in both peripheral blood and CSF. However, no significant differences were observed in blood levels of IL-4, IFN-γ and STNFR1.[71] Therefore, individual inflammatory markers still exhibit considerable heterogeneity across different populations. Furthermore, factors such as small sample sizes, testing platforms, and confounding variables further limit precise clinical diagnosis.

The "#7 neuroimaging" in the keyword clustering results indicates that neuroimaging has become a hot research area in the field of neuroinflammation in PD. Neuroimaging enables visualization and quantification of brain pathological processes before clinical symptom onset or during atypical stages, aiding in early PD diagnosis and stratified precision treatment.[72,73] Common imaging methods include dopaminergic imaging, substantia nigra imaging, and free water imaging.[74] Research indicates that enhanced microglia activation occurs in the brains of individuals in the preclinical stage of PD, accompanied by impaired dopaminergic neuron function. This provides compelling imaging evidence for the involvement of neuroinflammation in the early pathological processes of PD.[75] Targeting the 18 kDa translocator protein (TSPO) remains one of the most commonly used approaches for PET-based assessment of neuroinflammation in PD.[76] A meta-analysis reported increased TSPO binding across several regions—including the midbrain and striatum—when first-generation ligands were used, whereas with second-generation ligands, a significant increase was mainly detected in the midbrain. Taken together, these findings are consistent with the view that glia-associated inflammation in PD is closely linked to dopaminergic vulnerability and is particularly prominent in midbrain-related pathology.[77] At the same time, PET TSPO imaging still faces practical limitations, such as high tracer lipophilicity, potential off-target binding, and sensitivity to the rs6971 polymorphism. These issues highlight the need for improved third-generation TSPO tracers and more robust quantitative analysis pipelines.[78] The integrity of the BBB can also be examined using MRI.[79] Neuroinflammation is often accompanied by the destruction of the BBB or an increase in permeability.[80] Once

the BBB is damaged, microglia will be activated, and further blood-sourced neurotoxic factors will accumulate in the CNS, and pro-inflammatory signals will be amplified, thus causing neuronal dysfunction.[81] Further use of gadolinium-enhanced MRI reveals alterations in the BBB within the substantia nigra of PD rats, leading to increased free iron levels that further exacerbate neuroinflammatory responses.[82] Although these neuroimaging methods are of certain significance for early detection, most of the evidence is exploratory. The progress will be based on the multi-center longitudinal cohort of precursors or high-risk groups, the comprehensive multimodal imaging method, and the further standardisation of quantitative readings, so as to support early diagnosis, prognosis, and treatment response detection.

Intervention Strategy

The results of the keyword clustering analysis show that intervention studies in the field of PD neuroinflammation primarily involve topics such as "#2 neuroprotective agents", "#10 nanoparticles" and "#13 extracellular vesicles". This indicates that the research focus in this field is no longer limited to traditional anti-inflammatory drug therapy but has expanded to include the optimization of delivery systems and the development of novel therapeutic carriers. The trend map further corroborates this, showing that the focus has gradually shifted from "nonsteroidal anti-inflammatory drugs" (2017–2020) to topics such as "drug therapy", "etiology" and "therapy" (2023–2025).

Anti-inflammatory and disease-modifying drugs are rapidly emerging in the study of PD neuroinflammation. The candidate drugs involved mainly include NSAIDs, NLRP3 inflammasome inhibitors, and GLP-1 receptor agonists.[83] In preclinical experiments, MCC950 can improve the motor ability of MPTP-treated mice, which is owing to its down-regulation of IL-1β expression, and reduce inflammation caused by α-syn aggregation.[12] At the same time, after the GLP-1 receptor is activated, it will trigger a series of intracellular signaling pathways, showing a certain neuroprotective effect.[84] Recently, double GLP-1 and GIP receptor agonists, DA5-Ch, have shown good curative effects in animal models, which can cross the BBB, protect dopamine neurons and reduce chronic inflammatory reactions.[85] Despite these progresses, it is still difficult to convert them into clinical benefits. Key obstacles include low BBB permeability of those compounds, unknown long-term safety, differences in clinical trial results, etc.[86] Therefore, enhancing the efficacy of drugs in the brain through delivery strategies such as nanoparticles and extracellular vesicles has become a key focus of current intervention strategies.

There are more and more modification strategies for nanoparticles related to PD diseases, especially nanoparticles used as drug delivery carriers to improve the BBB. Use packaged small molecules, proteins, or nucleic acids to protect goods from degradation by the surrounding environment, thus prolonging the cycle time and providing continuous release to be achieved, which may promote drug access to the CNS. Such nanoparticles can also more accurately locate specific cell types and regulate related intracellular pathways.[87,88] At present, most research on PD uses polymer-based, lipid-based, and inorganic nanomaterials.[89] Yadav et al show that intranasal administration is more conducive to the brain delivery of biological agents such as TNF-α siRNA than the systemic route, increasing brain concentration and reducing the expression of TNF-α induced by LPS in macrophages.[90] However, intranasal conveying is still limited by practical problems, that is, low conveying efficiency and poor formula stability. To solve this problem, Li et al combined the nasal dose with nanoparticles excited by biomimetic microglia nanoparticles to improve the transport and bioavailability of metformin in the inflammatory brain area.[91]

As naturally secreted biological nanoparticles, extracellular vesicles (EVs) are the most promising next-generation drug delivery system and the potential treatment target of PD.[92,93] Preclinical studies confirm that human neural stem cell-derived EVs mitigate neuroinflammation and promote neuronal regeneration.[94] As naturally occurring nanoscale vesicles, exosomes can cross the BBB and function in intercellular communication.[95] However, their complex composition and high heterogeneity, coupled with unclear delivery efficiency and long-term safety, limit clinical translation.[96] Consequently, engineered exosomes are employed to enhance delivery capacity and stability, expanding their therapeutic potential.[97] For instance, engineered exosome-based delivery systems permit more accurate brain-targeted transport of catalase mRNA, alleviating neurotoxicity and neuroinflammation in a PD model.[98]

Existing Challenges

In summary, research in the field of neuroinflammation in PD has witnessed considerable advancement during the last ten years, yet it now faces a bottleneck in translating basic research into clinical applications. Firstly, existing evidence primarily focuses on correlation rather than sufficient validation of causality. Simultaneously, the limited replication in animal/cell models and inadequate stratification of clinical populations may lead to variability in intervention efficacy and reduce the credibility of research findings. Furthermore, the absence of specific humoral/imaging biomarkers and the BBB's obstruction to drug delivery constrain clinical translation potential. Future studies should integrate single-cell sequencing and multi-omics technologies to elucidate causal pathways in key mechanisms through multi-level cross-validation, combined with large-scale, multi-center, longitudinal cohort follow-ups and mechanism-based stratification. Earlier diagnosis and the therapeutic effect of reading that can be accurately read will depend on strong serum and imaging biomarkers. Meanwhile, the BBB delivery strategy should have clearer standards and better consistency. These two major parts are breakthroughs in clinical transformation.

Limitations

This study uses CiteSpace, VOSviewer, and other bibliometric tools to analyze and visualize the literature on PD neuroinflammation. The results outline the key issues in this area and indicate its changing research trends. Nevertheless, there are a few restrictions to consider.

1) This study only uses WoSCC and Scopus. These databases are widely used, but they are not comprehensive of all publications. Their regional representation is also uneven. Because of this, some database bias might be present in the results, particularly when it comes to the assessment of countries, institutions, and journals.
2) We included only the more common types of English-language literature, such as research articles and reviews, and excluded other types, such as conference papers and books. Such a selection of literature types may lead to some analytical bias.
3) Although the search terms used in this study covered a broad range of literature, their broad nature may have led to the inclusion of publications that only mentioned neuroinflammation without addressing it as a core topic, thereby affecting the accuracy of the bibliometric results to some extent.
4) Citation analysis is subject to a clear time-accumulation effect and citation lag. Recently published high-quality studies often require time to accumulate sufficient citations, and their academic impact may therefore have been underestimated in the present study.
5) Author name ambiguity may have affected the analysis results, especially for authors with common surnames. For highly productive authors such as "Wang, Q.S.," the possibility that authors with the same name have been merged cannot be entirely ruled out. Therefore, the results of author analysis metrics should be interpreted with caution.

Future studies may further optimize the search strategy and inclusion criteria by integrating additional databases and including non-English publications as well as other types of literature. Standardized methods may also be applied to reduce citation differences across publication years. In addition, more rigorous author disambiguation strategies are needed to further improve the accuracy of the results.

Conclusion

In the past ten years, PD neuroinflammation literature has been a trend of significant growth, with contributions from more than 80 countries and 253,818 cited references from 23,569 authors. Publication output in this area continues to increase. China and the United States have become the leaders in this area, and there is an urgent need to further strengthen international cooperation and strengthen academic exchanges with different countries and institutions. The research results reveal that the related research hotspots have gradually expanded from the traditional pathological mechanisms, including α-syn and mitochondrial dysfunction, to the emerging directions, such as NLRP3 inflammasome, ferroptosis, and the gut-brain axis, and have carried out more in-depth exploration in clinical manifestations and

transformation research, for example, body fluid/imaging biomarkers and drug delivery systems. This study lays the groundwork for clarifying future research directions and promoting interdisciplinary cooperation in this emerging field.

Data Sharing Statement

The datasets used and analysed during the current study available from the corresponding author on reasonable request.

Acknowledgments

We would like to express our gratitude to the developers of the relevant software used in this paper.

Author Contributions

Shuo Dong: Conceptualization, Data curation, Writing – review & editing, Writing – original draft, Xuefeng Li: Methodology, Formal analysis, Writing – original draft. Siyi Wang: Validation, Project administration, Writing – review & editing. Yubo Gong: Investigation, Software, Writing – review & editing. Lingfeng Wu: Formal analysis, Writing – review & editing. Jinglin Hu: Visualization, Supervision, Writing - review & editing. Xinhua Chen: Data curation, Funding acquisition, Supervision, Visualization, Resources, Writing – review & editing.

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.

Funding

This work is supported by the Jilin Provincial Department of Science and Technology (YDZJ202401140ZYTS).

Disclosure

The author(s) report no conflicts of interest in this work.

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