Neurology Asia 2026; 31(2) : 723 – 742

Autophagy in neurodegenerative diseases: A double-edged sword in disease progression and therapy

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Abstract

Background: Autophagy constitutes a critical catabolic pathway that mediates the degradation and recycling of dysfunctional organelles and cytotoxic protein aggregates. Accumulating evidence indicates its pivotal involvement in the pathogenesis of major neurodegenerative disorders, including Alzheimer's disease (AD), Parkinson's disease (PD), Huntington's disease (HD), and amyotrophic lateral sclerosis (ALS). This study seeks to systematically investigate global research trends and the underlying intellectual structure pertaining to autophagy in the context of neurodegenerative diseases. *Methods:* A total of 5,034 articles published over 20 years were analyzed using CiteSpace (version 6.4.1R), VOSviewer (version 1.6.20), bibliometrix (R package), Scimago Graphica(version 1.0.46.0), and Microsoft Office Excel 2021(version 16.48). Analyses included co-citation, keyword evolution, and collaboration networks. *Results:* Over the past two decades, the field has experienced robust growth, with a 24.93% annual publication growth rate and over 224,000 cumulative references. China and the United States led in publication output, with significant international cooperation. Authors such as Rubinsztein DC and Nixon RA were highly cited. Influential journals included *Autophagy* and *Journal of Biological Chemistry*. Established research areas include autophagy, neurodegenerative diseases, Parkinson's disease, Alzheimer's disease, amyotrophic lateral sclerosis, oxidative stress and apoptosis. keywords analysis emphasizes mitophagy, oxidative stress, and neuroinflammation, with a focus on disease-specific mechanisms and cellular microdomains. The autophagy–mitochondria–lysosome axis has gained prominence, highlighting organelle quality control and its role in neuronal survival, paving the way for integrated, stage-specific therapeutic strategies.
Conclusion: Autophagy is now embedded within disease-specific networks, revealing new insights into disease mechanisms and therapeutic strategies. Its dual role in neuroinflammation, both protective and contributory, adds complexity to its involvement in disease progression. Moving forward, research should refine these insights, targeting disease stages and cellular networks to develop more effective therapies.

Keywords: Autophagy, neurodegeneration, Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis, bibliometric analysis

INTRODUCTION

Autophagy is a crucial cellular process that eliminates damaged organelles and aggregated proteins, playing a key role in the pathogenesis of neurodegenerative diseases (ND) such as Alzheimer's disease (AD), Parkinson's disease (PD), and Huntington's disease (HD). In AD, impaired autophagy leads to the accumulation of amyloid-beta plaques and tau tangles, while in PD, it is linked to the buildup of misfolded alpha-synuclein.[1,2] In HD, dysfunction in autophagy contributes to the aggregation of mutant huntingtin proteins.[3] Depending on its regulation, autophagy can either protect

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Date of Submission: 4 December 2025; Date of Acceptance: 8 February 2026

https://doi.org/10.54029/2026reh

neurons or exacerbate cell death, making it a potential therapeutic target. In HD, activating autophagy with DRD3 agonists promotes the clearance of toxic huntingtin proteins, offering neuroprotection.[4] In PD, exosomes sourced from human umbilical cord mesenchymal stem cells stimulate autophagy in dopaminergic neurons, which helps limit cell death by reducing apoptosis.[5] Similarly, in AD, cannabidiol (CBD) has demonstrated the ability to induce autophagy and apoptosis in amyloid-beta expressing cells, suggesting it as a promising therapeutic avenue.[6] When autophagy is impaired, it leads to inflammation, disrupted metabolism, and the accumulation of toxic proteins, accelerating disease progression.[7] Additionally, abnormal autophagy activation via the DLK/JNK pathway has been linked to cell death, indicating that inhibiting this pathway may help reduce neurodegenerative damage.[8] These discoveries reveal how autophagy plays a complicated but crucial role in neurodegenerative diseases and point to its potential for future treatments.

Bibliometrics is a quantitative analysis method that allows for the examination of literature to extract insightful statistics and analyses of citation relationships, authors, keywords, and other critical aspects.[9] This approach reveals research trends, hotspots, and correlations within specific fields, and it has been extensively applied across various disciplines, including medicine. The role of autophagy in neurodegenerative diseases has attracted growing interest in recent years.[10] However, a comprehensive analysis specifically focused on autophagy in neurodegenerative diseases has not yet been conducted. Our study provides an in-depth review of the past 20 years of global studies, focusing on the link between autophagy and neurodegenerative disorders. This analysis aims to uncover core research areas, identify current trends, and outline possible future directions in the field. Our study is intended to provide valuable insights for future research and offer a clearer understanding of the therapeutic potential of autophagy in treating neurodegenerative conditions.

METHODS

Database and search strategy

The Web of Science Core Collection (WoSCC) was selected as the data source for this bibliometric analysis due to its standardized indexing system and comprehensive coverage of high-quality scientific literature. The literature search was conducted on January 5, 2025, using the Science Citation Index Expanded (SCI-EXPANDED) within WoSCC. To comprehensively retrieve publications related to autophagy in neurodegenerative diseases, the search strategy was designed to query the Title (TI), Abstract (AB), and Author Keywords (AK) fields. The complete search query was as follows: ((AK=("Neurodegenerative Disease*" OR "Neurodegenerative Disorder" OR "Neuro-degenerative disease*" OR "Neuro-Degenerative Disorder"OR "cognitive dysfunction" OR "Alzheimer* Disease*" OR "Parkinson* disease*" OR "Huntington* Disease*" OR "Amyotrophic Lateral Sclerosis" OR "Gehrig* Disease*" OR "multiple sclerosis*")) OR (AB=("Neurodegenerative Disease*" OR "Neurodegenerative Disorder" OR "Neuro-degenerative disease*" OR "Neuro-Degenerative Disorder"OR "cognitive dysfunction" OR "Alzheimer* Disease*" OR "Parkinson* disease*" OR "Huntington* Disease*" OR "Amyotrophic Lateral Sclerosis" OR "Gehrig* Disease*" OR "multiple sclerosis*")) OR (TI=("Neurodegenerative Disease*" OR "Neurodegenerative Disorder" OR "Neuro-degenerative disease*" OR "Neuro-Degenerative Disorder"OR "cognitive dysfunction" OR "Alzheimer* Disease*" OR "Parkinson* disease*" OR "Huntington* Disease*" OR "Amyotrophic Lateral Sclerosis" OR "Gehrig* Disease*" OR "multiple sclerosis*"))) AND ((AK=(Autophagy OR Autophagic)) OR (AB=(Autophagy OR Autophagic)) OR (TI=(Autophagy OR Autophagic))). This inclusive search strategy was adopted to ensure comprehensive coverage of autophagy-related studies within neurodegenerative contexts, while subsequent analyses focused on disease-specific patterns relevant to neurodegeneration.

The time span was restricted to publications published between January 1, 2004 and December 31, 2024. Only documents classified as Article or Review and written in English were included. Other document types, such as meeting abstracts, editorials, letters, and corrections, were excluded. After applying these inclusion criteria, a total of 5,035 records were initially retrieved from WoSCC. However, one record was excluded because complete metadata could not be exported from the Web of Science database at the time of retrieval. Consequently, 5,034 records were exported in plain text format with full records and cited references. This dataset

constituted the initial dataset for bibliometric analysis.

Data cleaning and processing

Prior to bibliometric analysis, data cleaning procedures were performed to ensure data consistency and reliability. Duplicate records were identified and removed using the built-in deduplication functions of the bibliometric analysis software. Records were further screened according to document type, language, and publication year to ensure consistency with the predefined inclusion criteria. During data processing with the bibliometrix R package, several early-access records indexed as 2025 publications were automatically included by the software. To strictly adhere to the predefined study period (2004–2024), these 2025 records were excluded from bibliometrix-based analyses. No additional manual exclusion based on content relevance was performed. As a result, the datasets used for analysis were tool-specific. The full set of 5,034 records was retained for analyses conducted using CiteSpace,VOSviewer and Scimago Graphica, while a cleaned dataset of 4,990 publications was used for bibliometrix-based analyses. The descriptive characteristics of the bibliometrix dataset are summarized in Supplementary Table 1. References published in 2025 are cited exclusively to contextualize bibliometric trends and were not included in quantitative analyses.

Bibliometric analysis tools and parameter settings

Multiple bibliometric tools were employed to ensure a comprehensive and robust analysis, includingCiteSpace (version 6.4.1R), VOSviewer (version 1.6.20), the bibliometrix R package (https://www.bibliometrix.org) and Microsoft Office Excel 2021(version 16.48). CiteSpace (version 6.4.1R)[11], developed by Professor Chaomei Chen, was used to conduct co-occurrence analysis (keywords), co-citation analysis (references), burst detection, and timeline visualization. The main parameter settings were as follows: time slicing from 2004 to 2024 with one year per slice, node types selected according to the analytical purpose (e.g., author, institution, country, keyword, and reference), pruning methods set to Pathfinder and Pruning sliced networks, and selection criteria based on the g-index (k = 25). VOSviewer (version 1.6.20), developed by Leiden University in the Netherlands, is particularly proficient in building and visualizing bibliometric networks, and supports co-citation, bibliographic coupling, and co-occurrence analyses.[12] The full counting method was used, and minimum thresholds for inclusion (e.g., number of documents or keyword occurrences) were set according to the specific type of network. Default layout and clustering algorithms were applied. The bibliometrix R package[13], was used to perform descriptive bibliometric analyses, historiographic mapping, thematic evolution analysis, and collaboration network visualization. Default parameters were applied unless otherwise specified. Scimago Graphica (version 1.0.46.0) was utilized to create geographic visualizations of national research output and collaboration patterns. Additionally, Microsoft Office Excel 2021(version 16.48) was used to estimate trends in publication and citation numbers.

RESULTS

Trends and academic contributions in publications

As illustrated in Figure 1, the study employed a structured search strategy based on titles (TI), abstracts (AB), and author keywords (AK), ensuring broad inclusion of relevant literature. The field has exhibited robust growth, with an annual publication growth rate of 24.93% and a cumulative reference count exceeding 224,000. As shown in Figure 2A, annual publication output increased from fewer than 50 in 2004 to over 400 by 2023, with average citations per document reaching 60.39. In the initial WoSCC dataset (n = 5,034), the literature was dominated by original research articles (n = 3,227) and review papers (n = 1,807), as shown in Figure 2B .The cumulative publication trajectory and Price's Law curve (Figures 2C and 2D) indicate a consistent and accelerating trend, underscoring the maturation and rising influence of this research domain.

Analysis of cooperation between countries

As illustrated in Figure 3A, the United States and China hold central positions within the global co-authorship network. Countries in Europe, East Asia, and North America also demonstrate close collaborative ties, indicating a high degree of international cooperation in this research field. Figure 3B illustrates patterns of bilateral and multilateral collaboration, with particularly strong links observed between

Table 1: Top 10 authors on autophagy research in neurodegenerative diseases

Rank	Author	Documents	Citations	Norm. citations	Avg. pub. yea	Avg. citations	Avg. norm. citations
1	Rubinsztein, Dc	54	13744	138.2724	2014.037	254.5185	2.5606
2	Li, Min	37	1703	44.2214	2020.1622	46.027	1.1952
3	Poletti, Angelo	29	1877	31.1524	2017.5172	64.7241	1.0742
4	Lu, Jia-Hong	27	1286	28.3598	2019.4444	47.6296	1.0504
5	Crippa, Valeria	24	1423	20.2719	2016.875	59.2917	0.8447
6	Fornai, Francesco	24	1272	12.5968	2014.3333	53	0.5249
7	Klionsky, Daniel J.	23	4723	54.1035	2016.5217	205.3478	2.3523
8	Song, Juxian	23	1207	30.0557	2019.7826	52.4783	1.3068
9	Nixon, Ralph A.	22	5193	61.0866	2015.8636	236.0455	2.7767
10	Rusmini, Paola	22	1325	18.524	2016.7273	60.2273	0.842

the USA and China, the USA and the UK, and China and Japan. Figure 3C reveals that China has experienced a rapid increase in publication output since 2015, surpassing the United States in recent years. The USA, in contrast, has maintained a consistent level of productivity, reflecting sustained leadership in the field. The United Kingdom and Japan have also contributed steadily. As shown in Figure 3D, the USA leads in total citation count, highlighting its strong research impact. Although China ranks first in publication volume, its average citation per article remains relatively low, suggesting that its global academic influence is still in development. Figure 3E further illustrates annual publication contributions by country, confirming China's recent dominance alongside the continued participation of the USA, the UK, and Japan. Figure 3F ranks the top ten most productive countries, led by China and the USA, with Italy, the UK, and Japan following. Supplementary Table 2 complements these figures by providing publication counts, collaboration ratios, and citation data. While China leads in volume, the USA exhibits stronger citation performance and more frequent international co-authorship.

Analysis of cooperation between institutions

Institutional analysis revealed a diverse and dynamic landscape of global contributors, with several key organizations playing a pivotal role in advancing autophagy research in neurodegenerative diseases. As shown in the institutional collaboration network (Figure 4A), research institutions were grouped into distinct regional and thematic clusters, reflecting both geographic proximity and shared research interests. Prominent institutions with the highest publication outputs included leading research universities and medical centers, particularly those located in China, the United States, and Europe. Among the top affiliations (Figure 4B), institutions such as University of California System (320 publications), University of London (218 publications), Institut National de la Santé et de la Recherche Médicale (INSERM) (201 publications), and Harvard University (180 publications) stood out, each contributing significantly to shaping the global research landscape. These institutions, alongside many others, have driven key advancements in understanding autophagy's role in neurodegenerative diseases. Temporal trends (Figure 4C) reveal a consistent rise in

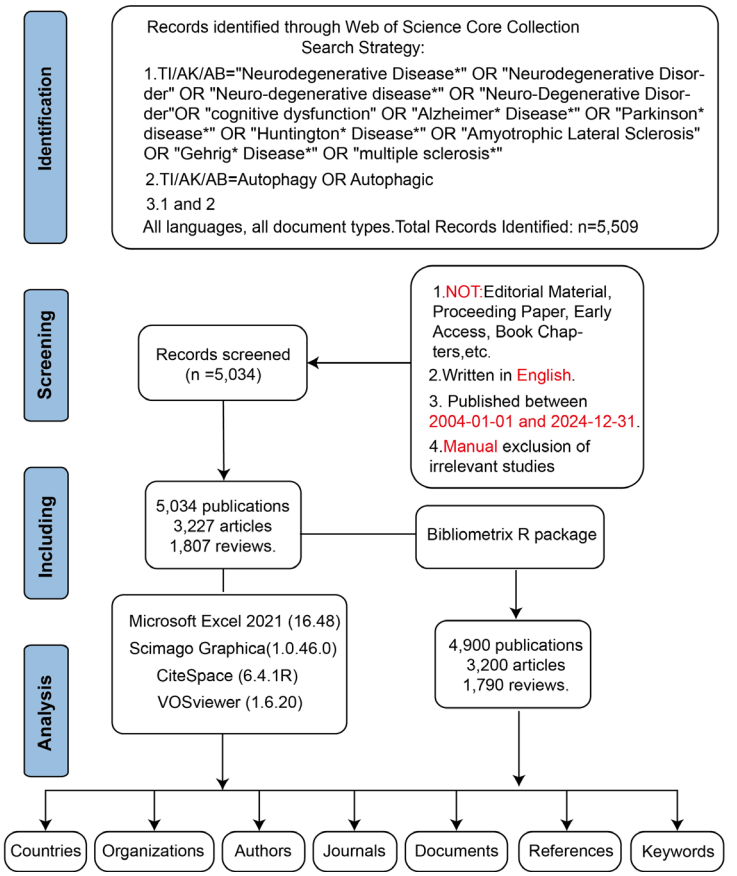

Figure 1. The flowchart for literature search, selection and analysis.
Abbreviations: TI:Title; AK: Author keywords; AB: Abstract.

institutional involvement over the past two decades, with a marked increase in publication output starting in 2015.

Authors and co-cited authors

The analysis of authorship patterns revealed a highly collaborative and influential scholarly community in the field of autophagy research in neurodegenerative diseases. As shown in Table 1 and Figure 5A, Rubinsztein DC stands out as the most productive author, with 54 publications and a total of 13,744 citations, reflecting both prolific output and substantial impact. Other leading contributors include Li Min, Poletti Angelo, and Nixon RA, each of whom has made notable contributions in recent years. Co-authorship network visualization revealed distinct collaboration clusters, indicating the presence of stable research teams with frequent intra-group cooperation. In the co-citation network (Figure 5B), Nixon RA, Mizushima N, and Ravikumar B emerged as the most co-cited authors, underscoring their foundational roles in shaping the theoretical and methodological development of the field.

Journal analysis

As presented in Figure 6A and Table 2, *Autophagy* ranks first in publication count and citation metrics, underscoring its central role in the field. Other influential journals, such as *Journal of Biological Chemistry*, *Human Molecular Genetics*, and *International Journal of Molecular Sciences*, also demonstrate strong citation metrics, reflecting their significance in autophagy and neurodegenerative disease research. Figure 6C shows a steady increase in publication activity, with *Autophagy* and *International Journal of Molecular Sciences* exhibiting notable growth, indicating their expanding influence. Figure 6B presents the top 10 journals by total citation count, with *Journal of Biological Chemistry* leading at 17,721

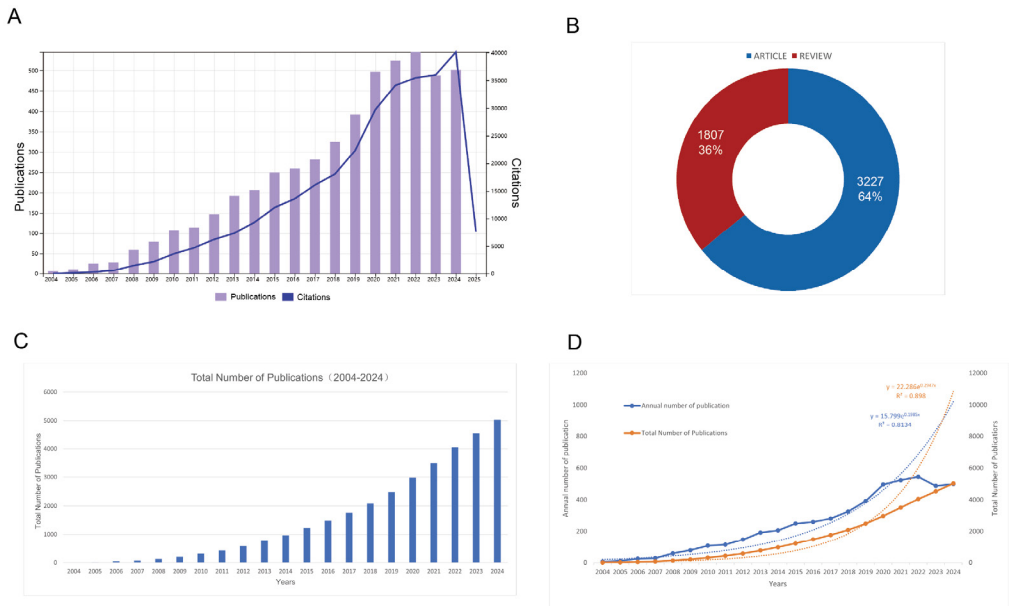

Figure 2. Overview of publications and citations on autophagy research in neurodegenerative diseases (2004–2024) (**A**) Annual number of publications and citations from 2004 to 2024. The bar graph (purple) represents the number of publications each year, while the line graph (blue) represents the corresponding citations. (**B**) Distribution of document types. (**C**) Cumulative number of publications. (**D**) Price's Law Growth Curve.

citations, followed by *Autophagy* (13,569) and *Proceedings of the National Academy of Sciences of the United States of America* (13,297). These journals are key contributors to advancing research in the field. Figure 6D's Bradford's Law analysis reveals that a small group of core journals contribute the majority of publications. Additionally, the citation and co-citation networks in Figures 6E and 6F identify tightly-linked citation communities, reinforcing the centrality of these journals in shaping the research landscape.

Publication analysis

As summarized in Table 3, the most cited publication in the field of autophagy and neurodegenerative diseases is the 2007 article by ELMORE S published in Toxicologic Pathology[14], which provides an extensive review of apoptosis and programmed cell death. This seminal work has accumulated 9965 citations to date, establishing it as a cornerstone in the study of cell death mechanisms and their implications for neurodegeneration. Other influential studies include those by Mizushima N (2011, Cell)[1] and Pankiv S (2007, Journal of Biological Chemistry)[15], each with over 3000 citations. These articles have contributed critical insights into the molecular processes of autophagy, including its role in cellular maintenance and the autophagic degradation of proteins, as well as the consequences of disrupted autophagic activity in neurodegenerative diseases. Together, these works not only represent foundational contributions but continue to shape ongoing research in the field. A more comprehensive grasp of the intellectual structure of this domain is achieved through the co-citation network visualization (Figure 7A), where references are classified into thematic groups depending on their modular properties. These clusters cover key topics such as mitochondrial dysfunction, neurodegenerative pathways, and cellular stress responses, illustrating the interdisciplinary nature of the research. The timeline view of co-citation clusters (Figure 7B) shows the evolution of these knowledge domains, with more recent clusters indicating a growing interest in the intersection of autophagy with mitochondrial dynamics and neuroinflammation. Additionally, Figure 8 identifies the top 25 references with the strongest citation bursts, highlighting periods of concentrated scholarly attention. Notably, several citation bursts have occurred in the last five years, reflecting the rapid development of the field and its responsiveness to recent scientific breakthroughs and clinical advancements.

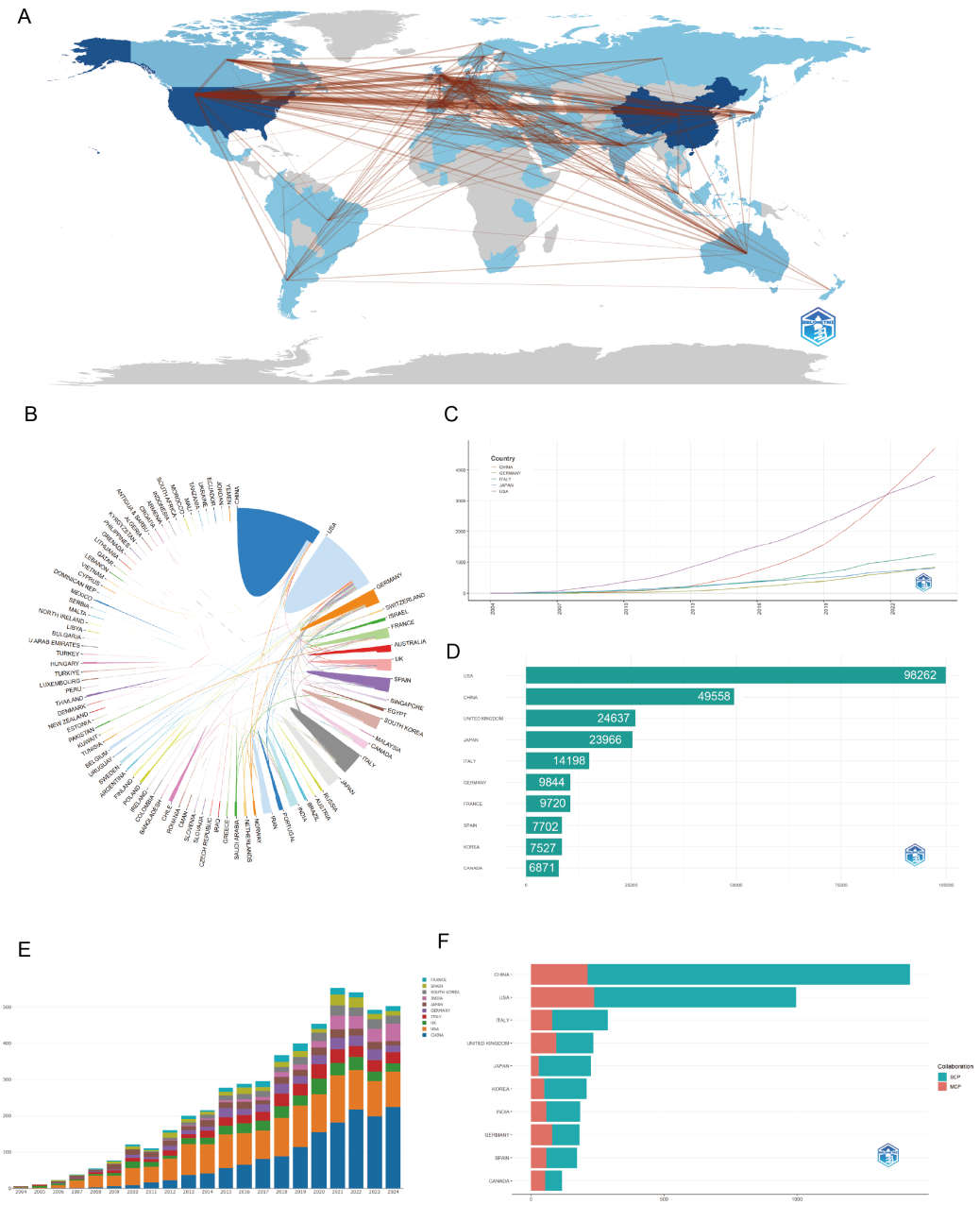

Figure 3. Global trends and collaboration networks in scientific publications on autophagy research in neurodegenerative diseases

(A) Global collaboration map of co-authorship networks. Darker blue shades reflect higher publication output, with lines depicting international collaborative ties. The United States and China exhibit the densest connections. (B) Circular plot of international collaboration. (C) Country publication trends. The x-axis spans years, and the y-axis shows citation counts. (D) Country influence by citation impact. (E) Stacked bar chart of annual publications. (F) The top 10 countries responsible for the number of studies.

Abbreviations: MCP: Multiple-Country Publications; SCP: Single-Country Publications;

Table 2: Top 10 influential journals on autophagy research in neurodegenerative diseases

Rank	Source	h_index	g_index	m_index	TC	NP	PY_start	IF	JCR
1	AUTOPHAGY	87	135	4.143	22073	280	2005	14.6	Q1
2	JOURNAL OF BIOLOGICAL CHEMISTRY	58	97	2.9	15018	97	2006	4.0	Q2
3	HUMAN MOLECULAR GENETICS	46	72	2.3	9856	72	2006	3.1	Q2
4	INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES	41	70	2.412	5980	177	2009	4.9	Q1
5	PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA	37	56	1.947	8190	56	2007	9.4	Q1
6	NEUROBIOLOGY OF DISEASE	34	60	2	3823	60	2009	5.1	Q1
7	PLOS ONE	32	51	1.778	2746	73	2008	2.9	Q1
8	FRONTIERS IN MOLECULAR NEUROSCIENCE	31	48	2.385	2382	62	2013	3.5	Q2
9	JOURNAL OF NEUROCHEMISTRY	30	51	1.429	2862	51	2005	4.2	Q2
10	CELL DEATH & DISEASE	29	45	2.071	2798	45	2012	8.1	Q1

Abbreviation: IF: Impact Factor; JCR: Journal Citation Reports

Keyword analysis

The keyword co-occurrence network (Figure 9A) highlights strong links between core terms such as "autophagy," "neurodegenerative diseases," "Parkinson's disease," and "Alzheimer's disease," reflecting a sustained focus on disease mechanisms and autophagy's role. As shown in Table 4, the most frequent keywords include "autophagy" (2267 times), "neurodegenerative diseases" (1038), "Parkinson's disease" (691), and "Alzheimer's disease" (618), confirming their central role in this field .Figure 9B illustrates a temporal shift from general terms to more specific topics like "mitophagy," "oxidative stress," and "neuroinflammation." Notably, "mitophagy" and "neuroinflammation" have emerged as key focuses in recent years, aligning with increased interest in mitochondrial health and inflammatory processes. Figure 9C further emphasizes the rise of these newer terms, while Figure 9D shows that keywords like "neuroinflammation" are more frequent in recent publications, reflecting their growing importance in current studies.

DISCUSSION

Over the past two decades, research into neurodegenerative diseases has increasingly incorporated autophagy as a central conceptual framework for understanding disease mechanisms. Autophagy has thus evolved from a relatively specialized cellular process into a key explanatory lens through which neurodegenerative pathology is investigated. The rapid expansion of publications observed in this bibliometric analysis reflects not only increasing recognition of autophagy's biological relevance in neurodegeneration, but also its methodological versatility across diverse neurodegenerative disease models. The rapid expansion of publications observed in this bibliometric analysis reflects not only increasing recognition of autophagy's biological relevance but also its methodological versatility as a research entry point across diverse disease models. Rather than converging on a single mechanistic or therapeutic consensus, the literature reveals a progressively diversified and integrative research landscape shaped by disease specificity, experimental accessibility, and emerging cellular paradigms. By synthesizing keyword co-occurrence, co-citation structures, and temporal trend analyses, the present discussion contextualizes how autophagy-related research in neurodegenerative

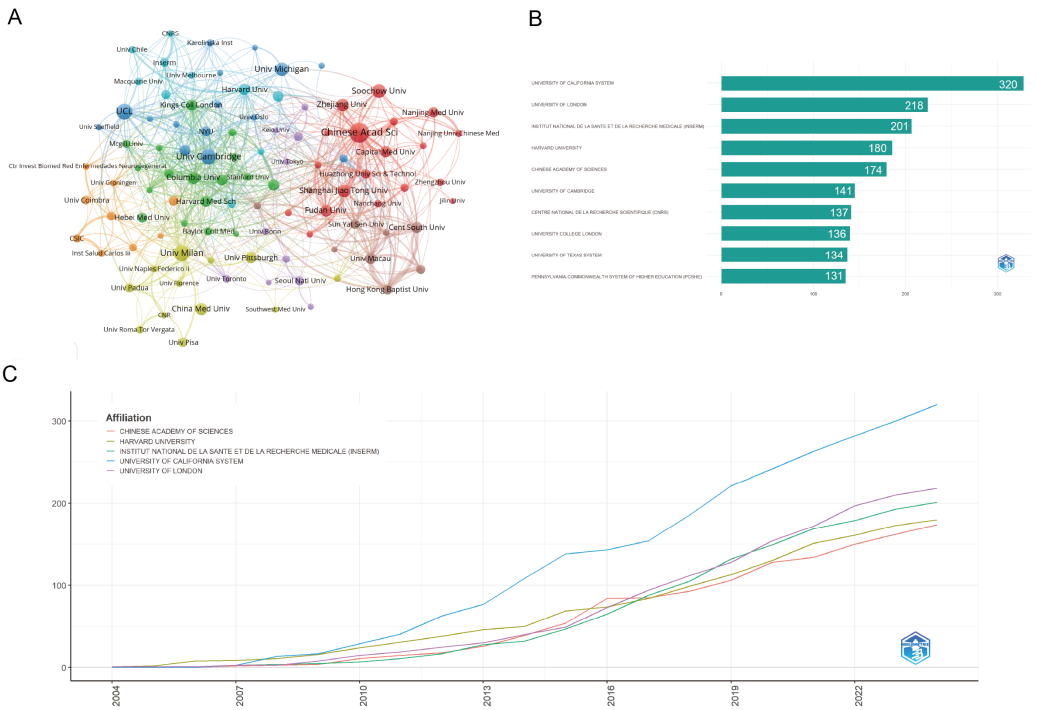

Figure 4. Institutional contributions and collaboration networks on autophagy research in neurodegenerative diseases.
(A) Institutional collaboration network (VOSviewer). Node size corresponds to collaboration extent, and colors denote distinct regional or institutional clusters. (B) Top affiliations by publication volume. (C) Institutional publication trends (Bibliometrix). The x-axis denotes years, and the y-axis indicates publication counts.

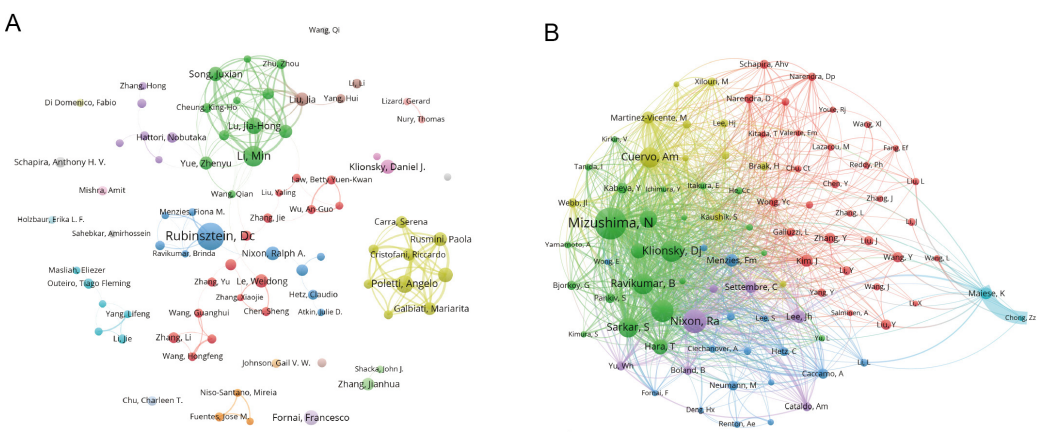

Figure 5. Co-authorship and Co-cited Authorship Networks on Autophagy Research in Neurodegenerative Diseases
(**A**) Co-authorship network: Nodes represent authors, sized by publication count. Colors indicate collaboration clusters. (**B**) Co-cited authorship network: Nodes, connected by collaborative publications, have edge thickness proportional to joint works. Clusters, marked by colors, reveal research groups.

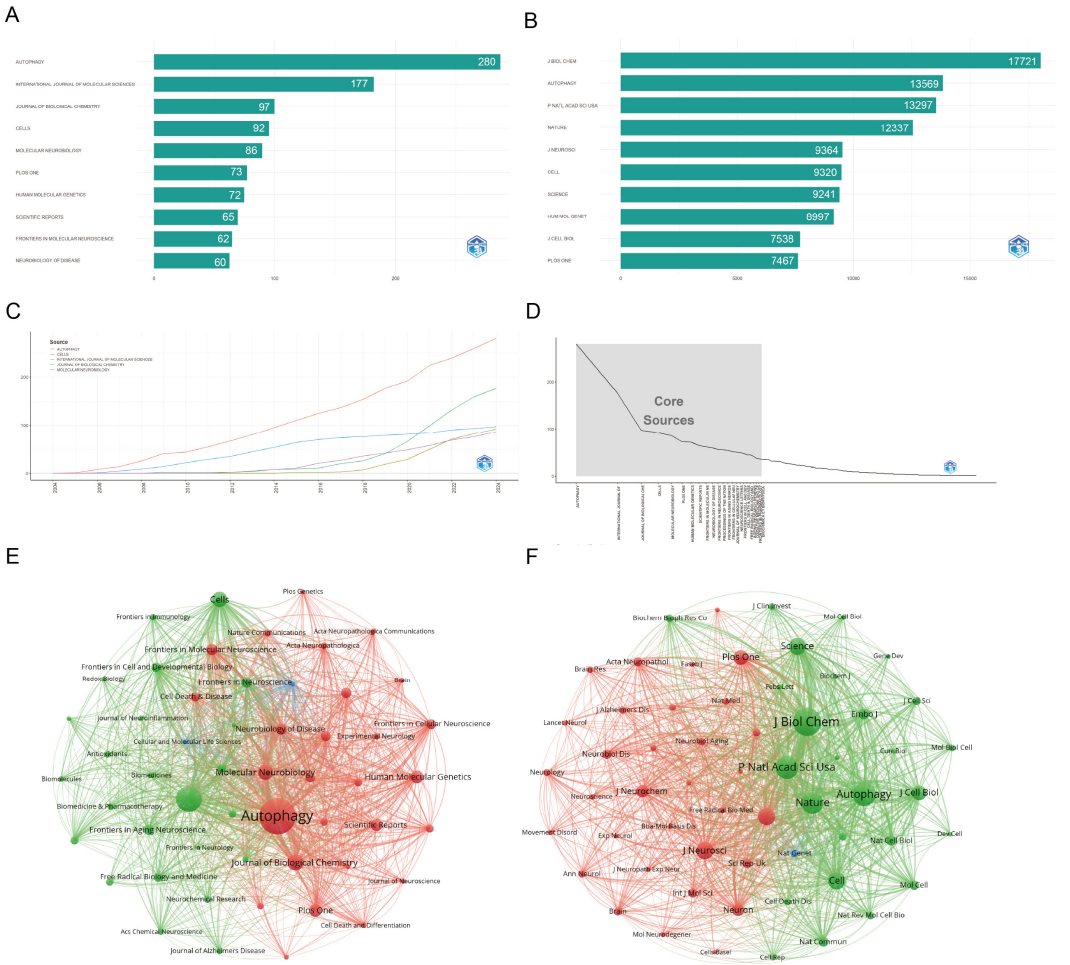

Figure 6. Comprehensive analysis of journals and citation networks related to autophagy research in neurodegenerative diseases
(A) Top journals by publication count: X-axis shows article numbers; y-axis lists journals. (B) Top 10 journals by total citation count. (C) Top 5 journals' publication trends. (D) Bradford's Law analysis: Identifies core journals based on publication distribution. (E) Journal citation network: Nodes represent journals, connected by citations. Colors (green, blue, red) indicate thematic clusters of frequent inter-citation. (F) Journal co-citation network: Nodes, sized by co-citation frequency, are linked by co-citation ties. Colors denote clusters of commonly co-cited journals.

diseases has been structured, prioritized, and transformed over time. Importantly, these patterns reflect collective research behavior and conceptual emphasis, rather than direct measures of clinical efficacy.

Core mechanisms and disease-oriented autophagy research

Bibliometric clustering demonstrates that mechanistic investigations of autophagy are deeply embedded within disease-oriented research frameworks, particularly for Alzheimer's disease (AD), Parkinson's disease (PD), amyotrophic lateral sclerosis (ALS), and Huntington's disease (HD). The prominence of these disease categories reflects their function as conceptual and experimental anchors for autophagy research, rather than uniform targets for therapeutic translation.

Across clusters, autophagy is rarely examined

Table 3: Top 25 most cited publications based on bibliometrix analysis

RANK	First Author	Year	Journal	Paper	DOI	Total Citations	TC per Year	Normalized TC
1	ELMORE S	2007	TOXICOL PATHOL	Apoptosis: a review of programmed cell death.	10.1080/01926230701320337	9965	524.47	12.17
2	MIZUSHIMA N	2011	CELL	Autophagy: renovation of cells and tissues.	10.1016/j.cell.2011.10.026	4899	326.6	33.42
3	PANKIV S	2007	J BIOL CHEM	p62/SQSTM1 binds directly to Atg8/LC3 to facilitate degradation of ubiquitinated protein aggregates by autophagy.	10.1074/jbc.M702824200	3657	192.47	4.47
4	HARA T	2006	NATURE	Suppression of basal autophagy in neural cells causes neurodegenerative disease in mice.	10.1038/nature04724	3222	161.1	13.32
5	KROEMER G	2007	PHYSIOL REV	Mitochondrial membrane permeabilization in cell death.	10.1152/physrev.00013.2006	2914	153.37	3.56
6	RAVIKUMAR B	2004	NAT GENET	Inhibition of mTOR induces autophagy and reduces toxicity of polyglutamine expansions in fly and mouse models of Huntington disease	10.1038/ng1362	1949	88.59	3.3
7	FENG YC	2014	CELL RES	The machinery of macroautophagy.	10.1038/cr.2013.168	1633	136.08	15.08
8	NIXON RA	2013	NAT MED	The role of autophagy in neurodegenerative disease.	10.1038/nm.3232	1556	119.69	13.71
9	MATSUDA N	2010	J CELL BIOL	PINK1 stabilized by mitochondrial depolarization recruits Parkin to damaged mitochondria and activates latent Parkin for mitophagy.	10.1083/jcb.200910140	1513	94.56	9.62

Table 3: (continued)

RANK	First Author	Year	Journal	Paper	DOI	Total Citations	TC per Year	Normalized TC
10	NIXON RA	2005	J NEUROPATH EXP NEUR	Extensive involvement of autophagy in Alzheimer disease: an immuno-electron microscopy study.	10.1093/jnen/64.2.113	1253	59.67	4.84
11	RUBINSZTEIN DC	2012	NAT REV DRUG DISCOV	Autophagy modulation as a potential therapeutic target for diverse diseases.	10.1038/nrd3802	1213	86.64	11.67
12	TANIDA I	2004	INT J BIOCHEM CELL B	LC3 conjugation system in mammalian autophagy.	10.1016/j.biocel.2004.05.009	1185	53.86	2.01
13	CHEN HC	2009	HUM MOL GENET	Mitochondrial dynamics--fusion, fission, movement, and mitophagy--in neurodegenerative diseases.	10.1093/hmg/ddp326	1143	67.24	9.25
14	PANDEY UB	2007	NATURE	HDAC6 rescues neurodegeneration and provides an essential link between autophagy and the UPS.	10.1038/nature05853	1017	53.53	1.24
15	PICKFORD F	2008	J CLIN INVEST	The autophagy-related protein beclin 1 shows reduced expression in early Alzheimer disease and regulates amyloid beta accumulation in mice.	10.1172/JCI33585	990	55	5.75
16	SARKAR S	2007	J BIOL CHEM	Trehalose, a novel mTOR-independent autophagy enhancer, accelerates the clearance of mutant huntingtin and alpha-synuclein.	10.1074/jbc.M609532200	921	48.47	1.12

Table 3: (continued)

RANK	First Author	Year	Journal	Paper	DOI	Total Citations	TC per Year	Normalized TC
17	HERAS-SANDOVAL D	2014	CELL SIGNAL	The role of PI3K/AKT/mTOR pathway in the modulation of autophagy and the clearance of protein aggregates in neurodegeneration.	10.1016/j.cellsig.2014.08.019	885	73.75	8.17
18	MENZIES FM	2017	NEURON	Autophagy and Neurodegeneration: Pathogenic Mechanisms and Therapeutic Opportunities.	10.1016/j.neuron.2017.01.022	821	91.22	11.59
19	GHAVAMI S	2014	PROG NEUROBIOL	Autophagy and apoptosis dysfunction in neurodegenerative disorders.	10.1016/j.pneurobio.2013.10.004	821	68.42	7.58
20	CHAN NC	2011	HUM MOL GENET	Broad activation of the ubiquitin-proteasome system by Parkin is critical for mitophagy.	10.1093/hmg/ddr048	802	53.47	5.47
21	SARKAR S	2005	J CELL BIOL	Lithium induces autophagy by inhibiting inositol monophosphatase.	10.1083/jcb.200504035	793	37.76	3.06
22	YAN HF	2021	SIGNAL TRANSDUCT TAR	Ferroptosis: mechanisms and links with diseases.	10.1038/s41392-020-00428-9	789	157.8	24.85
23	MOSTAFALOU S	2013	TOXICOL APPL PHARM	Pesticides and human chronic diseases: evidences, mechanisms, and perspectives.	10.1016/j.taap.2013.01.025	786	60.46	6.92
24	DAGDA RK	2009	J BIOL CHEM	Loss of PINK1 function promotes mitophagy through effects on oxidative stress and mitochondrial fission.	10.1074/jbc.M808515200	782	46	6.33
25	DING WX	2012	BIOL CHEM	Mitophagy: mechanisms, pathophysiological roles, and analysis.	10.1515/hsz-2012-0119	780	55.71	7.51

Abbreviation: DOI: Digital Object Identifiers

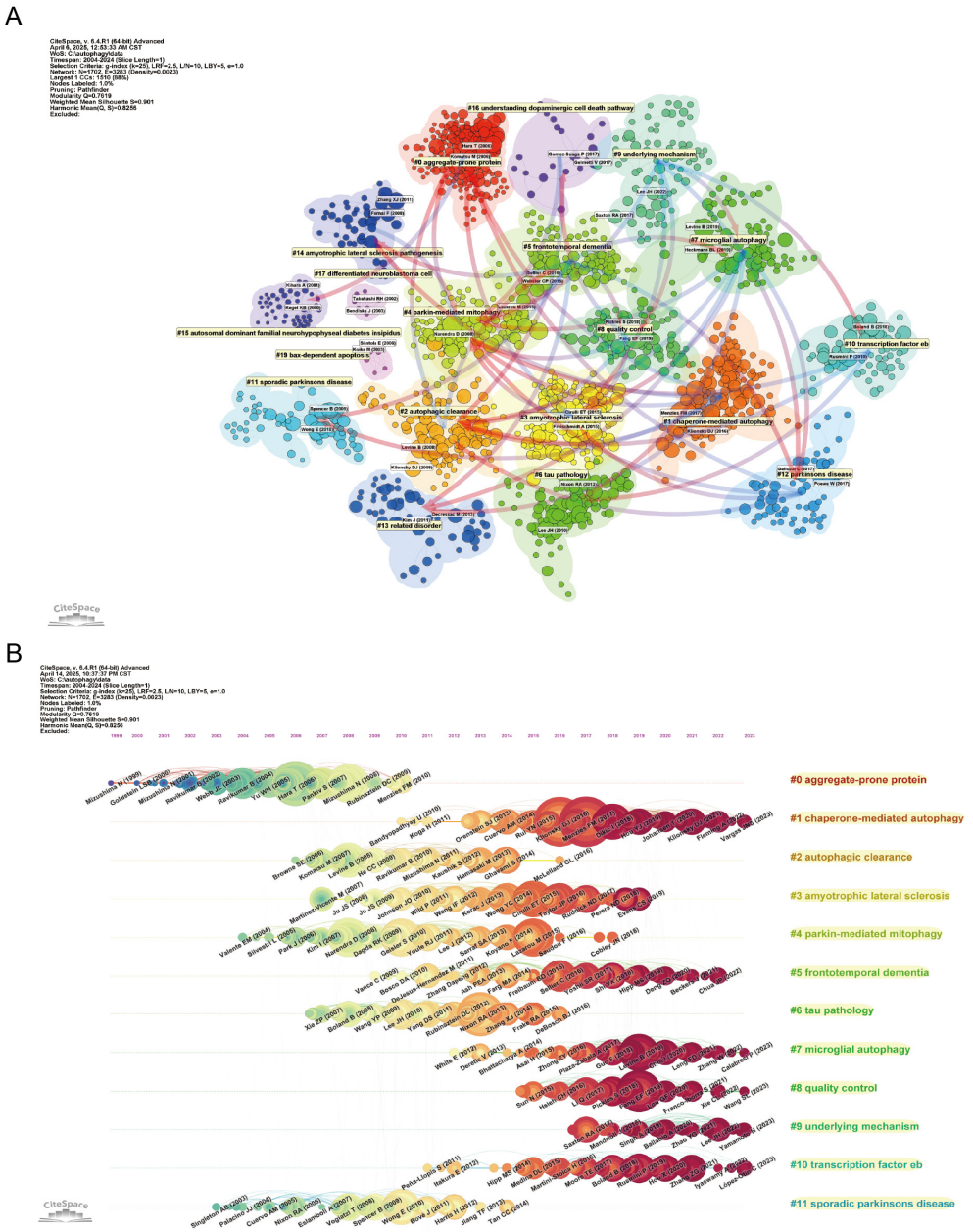

Figure 7. Network visualization of references on autophagy research in neurodegenerative diseases.
(A) Clustered co-citation network: Nodes, sized by citation frequency, are grouped by modularity with keyword labels for major clusters. Edge thickness reflects co-citation strength; colors highlight thematic diversity.
(B) Timeline view of co-citation clusters: Each horizontal line represents a research cluster labeled by key terms. Nodes correspond to highly cited references, with their positions reflecting the year of citation emergence. Node size indicates citation frequency, and color represents temporal distribution. This visualization illustrates the evolution and duration of key topics in autophagy-related neurodegenerative disease research.

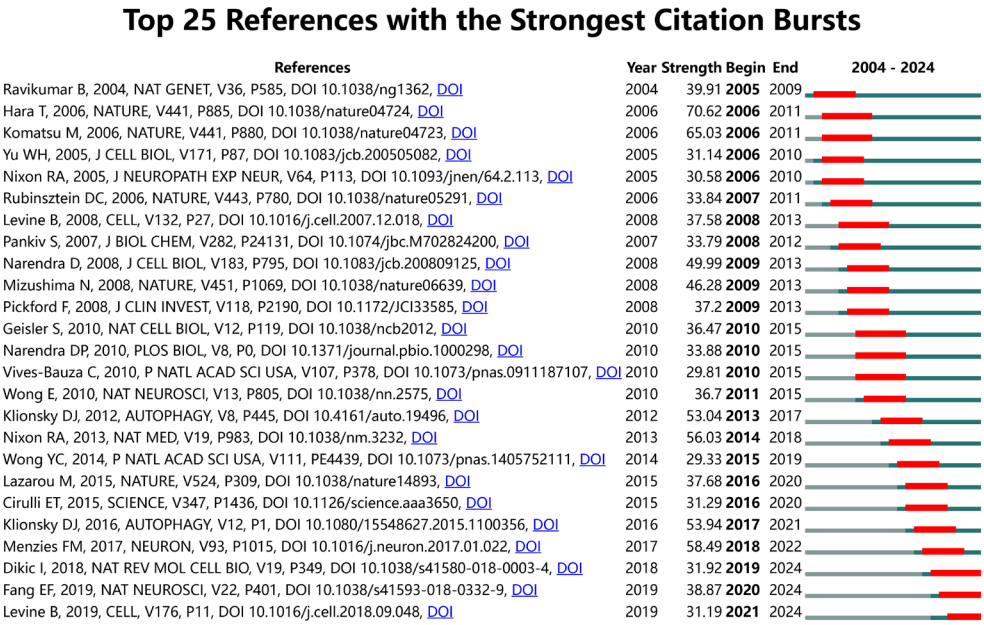

Figure 8. Top 25 references with strongest citation bursts on autophagy research in neurodegenerative diseases. The burst timeline illustrates the periods during which each reference experienced a surge in attention, with red segments marking the specific intervals of intensified citation activity.

Table 4: Top 15 author keyword on autophagy research in neurodegenerative diseases

Rank	Author Keyword	Occurrences	Avg. pub. year	Avg. citations	Avg. norm. citations
1	autophagy	2267	2018.3966	50.5664	0.837
2	neurodegenerative diseases	1038	2018.9152	47.6233	0.979
3	parkinson's disease	691	2018.5485	56.3357	1.0526
4	alzheimer's disease	618	2019.0906	48.9337	1.0133
5	amyotrophic lateral sclerosis	458	2018.4738	47.9017	0.8151
6	apoptosis	317	2017.9306	83.347	0.8503
7	oxidative stress	315	2019.127	47.6	1.061
8	mitophagy	299	2019.4415	65.2843	1.3633
9	mitochondria	265	2018.2377	56.7283	1.009
10	lysosomes	253	2018.1344	71.0514	1.0348
11	alpha-synuclein	197	2018.4619	41.5431	0.8082
12	neuroinflammation	187	2021.0963	34.9037	1.2826
13	ageing	178	2018.6404	52.2416	1.0723
14	huntington's disease	153	2017.3987	57.7124	0.9457
15	neuroprotection	138	2019.2754	41.1014	0.9892

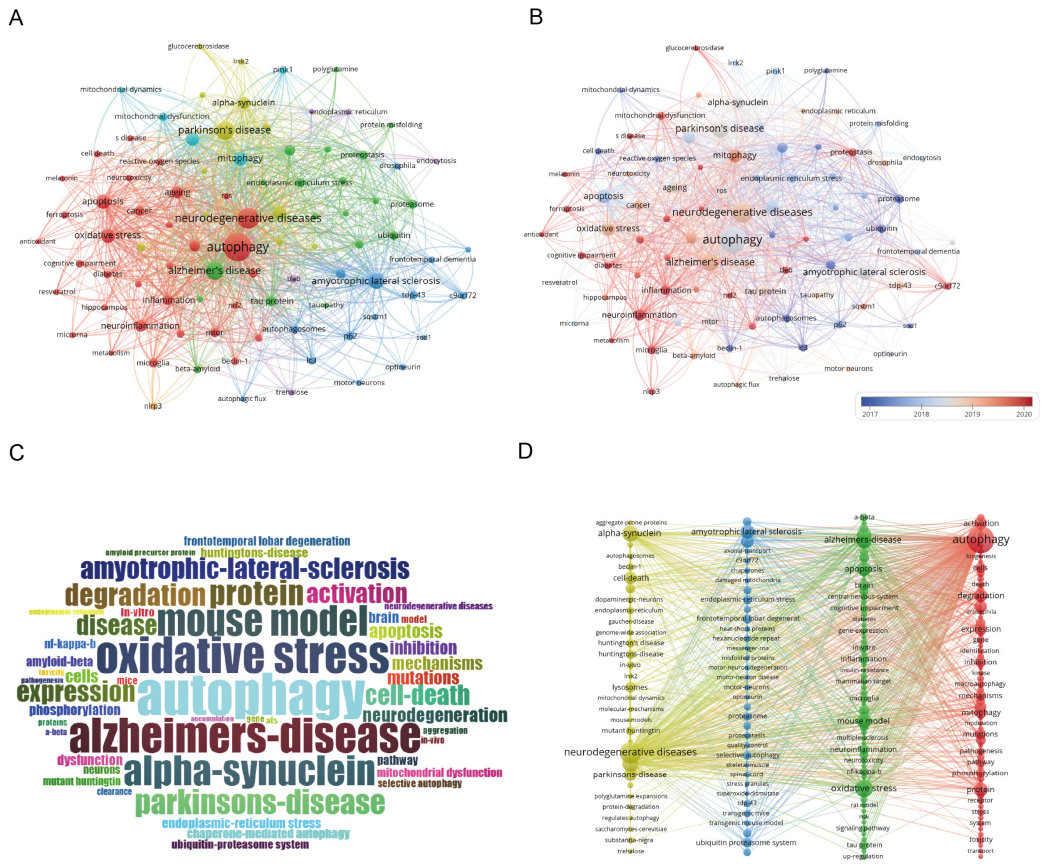

Figure 9. Keywords mapping of autophagy research in neurodegenerative diseases.
(A) Keywords co-occurrence network: Nodes represent keywords, sized by occurrence frequency (larger = more frequent), with edges reflecting co-occurrence and colors denoting thematic clusters. (B) Keywords timeline: Nodes, sized by frequency and colored by cluster, illustrate the temporal evolution of research topics. (C) Keywords Plus word cloud: Word size reflects frequency, emphasizing prevalent author-assigned terms. (D) Keywords overlay visualization: Keywords are plotted to highlight their relationships across various research themes. Node size reflects keyword frequency, and colors represent different thematic clusters, illustrating how keywords are interconnected within the research landscape.

as an isolated degradative pathway. Instead, it is repeatedly contextualized within broader networks involving protein aggregation, mitochondrial dysfunction, lysosomal impairment, and inflammatory signaling. This integrative orientation suggests that the field has largely moved beyond early descriptive studies toward systems-level interpretations of cellular homeostasis failure in neurodegeneration.

Alzheimer's disease (AD)

Within the neurodegenerative disease–focused autophagy research network, AD constitutes one of the most densely connected and influential clusters, reflecting sustained interest in its complex proteopathic and metabolic pathology. Bibliometric patterns indicate that AD-focused autophagy research has progressively shifted from general associations toward more refined investigations of autophagic flux, lysosomal degradation capacity, and pathway-specific regulation. Highly frequent and centrally positioned keywords related to amyloid-beta, tau pathology, mTOR signaling, and lysosomal dysfunction suggest that impaired autophagic clearance—rather than insufficient autophagy induction—has become a dominant conceptual focus. This trend aligns with an increasing number of studies emphasizing bottlenecks in

autophagosome maturation, cargo recognition, and lysosome competence rather than simple deficits in autophagy initiation.[16-20] Notably, recent AD-related literature increasingly embeds autophagy within structural and regulatory microdomains, such as mitochondria-associated membranes and non-coding RNA–mediated modulation, reflecting a transition toward pathway integration and intracellular spatial regulation.[16,21] From a bibliometric standpoint, this evolution indicates a maturation of the field, in which autophagy is no longer viewed as a standalone therapeutic lever but as a component of a tightly regulated cellular network whose modulation requires precision and disease-stage specificity.

Parkinson's disease (PD)

PD represents a second major pillar of autophagy-related neurodegenerative research, characterized by strong associations with selective autophagy, mitochondrial quality control, and inflammatory regulation. The prominence of α-synuclein, mitophagy, and oxidative stress–related keywords highlights PD as a preferred model for dissecting protein- and organelle-specific autophagic processes. Bibliometric patterns reveal that PD-focused research frequently converges on chaperone-mediated autophagy, PINK1–Parkin signaling, and lysosome-dependent degradation pathways, reflecting the suitability of PD models for mechanistic dissection of selective autophagy.[22-24] Importantly, the recurrent co-occurrence of neuroinflammation-related terms suggests that PD research increasingly conceptualizes autophagy not only as a proteostatic mechanism but also as a regulator of immune responses within the central nervous system. Unlike earlier narratives portraying autophagy as uniformly neuroprotective, the bibliometric landscape reflects a more nuanced perspective. Dysregulated or insufficient autophagy appears to contribute to disease progression, while targeted restoration of autophagic balance remains an experimental and context-dependent strategy.[22,23] This duality is mirrored in the heterogeneous and densely interconnected structure of PD-related clusters.

Amyotrophic lateral sclerosis (ALS)

ALS occupies a comparatively smaller yet conceptually coherent position within the autophagy research network. ALS-related studies are strongly linked to keywords associated with selective autophagy receptors, protein aggregation, and stress-response signaling, reflecting the genetic and molecular specificity of disease pathology. The prominence of p62/SQSTM1, C9ORF72, and mTOR-related terms suggests that ALS research emphasizes disruptions in cargo recognition, autophagic flux completion, and cellular stress adaptation rather than generalized autophagy modulation.[25-27] Unlike AD and PD, ALS-related autophagy studies often focus on late-stage failure of degradation systems and compensatory mechanisms, consistent with the rapid disease progression and limited neuronal plasticity characteristic of motor neuron disorders. From a structural perspective, ALS serves primarily as a model for interrogating the intersection of genetic mutations and autophagy dysfunction, rather than as a broad platform for therapeutic generalization. This positioning may explain its relatively lower publication volume but strong thematic cohesion within the bibliometric network.

Huntington's disease (HD)

HD-related autophagy research forms a distinct and historically influential cluster, particularly in establishing autophagy as a modifiable pathway in neurodegeneration. Early bibliometric clusters emphasize autophagy induction and mTOR inhibition as strategies for clearing polyglutamine aggregates, consistent with foundational experimental studies in HD models.[28] More recent keyword trends, however, indicate an expansion of conceptual focus toward mitochondrial dysfunction, immune signaling, and autophagy–inflammation crosstalk. The increasing association of HD research with cGAS–STING signaling and mitochondrial stress reflects a broader shift from aggregate-centric models toward integrated stress-response frameworks.[29,30] This transition underscores the evolving role of HD research within the field—from serving as a proof-of-concept system for autophagy modulation to contributing to more comprehensive models of neurodegenerative pathology that integrate metabolic, inflammatory, and degradative processes.

Neuroinflammation and neuroprotection: Autophagy's dual role

Neuroinflammation has emerged as one of the most prominent and temporally recent themes within the autophagy research landscape.

Bibliometric analyses demonstrate increasing co-occurrence between autophagy-related terms and markers of microglial activation, inflammasome signaling, and cytokine regulation, highlighting a growing recognition of autophagy as an immune-modulatory process.[20,29,31] These patterns support a dual-role framework in which autophagy constrains neuroinflammatory stress by facilitating the removal of damaged organelles and toxic aggregates, while chronic inflammation can, in turn, impair autophagic flux and degradation efficiency. This bidirectional relationship is consistently observed across disease-specific clusters, suggesting that autophagy operates within a dynamic feedback system rather than as a unidirectional protective pathway. From a translational perspective, the convergence of autophagy and neuroinflammation research implies that effective therapeutic strategies may need to target both processes simultaneously. The bibliometric prominence of this intersection highlights its growing importance as a research frontier rather than a resolved mechanism.

Autophagy–mitochondria–lysosome axis in neurodegeneration

One of the most significant findings of this bibliometric analysis is the increasing centrality of the autophagy–mitochondria–lysosome axis. Keywords related to mitophagy, lysosomal function, and oxidative stress exhibit strong connectivity and recent citation bursts, indicating a field-wide transition toward integrated organelle quality control models.[32,33] Rather than examining autophagy in isolation, contemporary studies increasingly frame it as part of a coordinated cellular stress-response network governing mitochondrial turnover, metabolic regulation, and neuronal survival.[18,32] The prominence of lysosomal dysfunction–related terms further underscores the recognition that impaired degradation capacity—rather than autophagy initiation alone—represents a critical bottleneck in neurodegenerative pathology.[16,20] This integrative perspective reflects the maturation of autophagy research in neurodegeneration, shifting from single-pathway hypotheses toward systems-level models that better capture the complexity of neuronal vulnerability and disease progression.

In conclusion, for major neurodegenerative diseases such as AD, PD, ALS, and HD, autophagy research has expanded to explore not only autophagic flux and lysosomal dysfunction but also the cellular contexts and microdomains regulating these processes. In particular, autophagy has become increasingly embedded within disease-specific networks, offering new insights into disease mechanisms and potential therapeutic strategies. Furthermore, the growing recognition of autophagy's role in modulating neuroinflammation and its dual role in both protecting against and contributing to disease progression underscores the complexity of its involvement in neurodegenerative diseases. The autophagy–mitochondria–lysosome axis has emerged as a particularly significant theme, with an increasing focus on organelle quality control and its impact on neuronal survival. These findings suggest that autophagy-related research is moving toward more comprehensive, integrated models that better capture the complexities of neurodegeneration. Importantly, while significant strides have been made in understanding the mechanisms underlying autophagy's role in neurodegeneration, future research must continue to refine these insights, targeting specific disease stages, cellular networks, and cross-talk between autophagy and other cellular processes for more effective therapeutic interventions.

ACKNOWLEDGEMENTS

We are grateful to the editors and anonymous reviewers for their thoughtful feedback and valuable suggestions, which greatly contributed to the refinement of this manuscript.

DISCLOSURE

Data availability: The data that support the findings of this study are available from the Web of Science (https://www.webofscience.com). For additional data, please contact the corresponding author.

Financial support: 1. Fund Program for the Scientific Activities of Selected Returned Overseas Professionals in Shanxi Province (20250046); 2. Research Project supported by Shanxi Scholarship Council of China (2023-188); 3. Shanxi Health Committee Foundation Project (2024057); 4. Fundamental Research Program of Shanxi Province (202403021222432).

Conflict of interests: None

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Supplementary Table 1: Bibliometric overview of autophagy research in neurodegenerative diseases

Description	Results
MAIN INFORMATION ABOUT DATA	
Timespan	2004:2024
Sources (Journals, Books, etc)	868
Documents	4990
Annual Growth Rate %	24.93
Document Average Age	6.49
Average citations per doc	60.39
References	224849
DOCUMENT CONTENTS	
Keywords Plus (ID)	8867
Author's Keywords (DE)	7513
AUTHORS	
Authors	25009
Authors of single-authored docs	96
AUTHORS COLLABORATION	
Single-authored docs	122
Co-Authors per Doc	6.82
International co-authorships %	26.41
DOCUMENT TYPES	
article	3200
review	1790

Supplementary Table 2: Most relevant countries by corresponding author contributions

Rank	**Country**	**Articles**	**Articles %**	**SCP**	**MCP**	**MCP %**
1	CHINA	1423	28.5	1211	212	14.9
2	USA	997	20	759	238	23.9
3	ITALY	289	5.8	210	79	27.3
4	UNITED KINGDOM	234	4.7	139	95	40.6
5	JAPAN	225	4.5	196	29	12.9
6	KOREA	209	4.2	159	50	23.9
7	INDIA	184	3.7	126	58	31.5
8	GERMANY	182	3.6	103	79	43.4
9	SPAIN	173	3.5	115	58	33.5
10	CANADA	116	2.3	63	53	45.7
11	FRANCE	115	2.3	61	54	47
12	AUSTRALIA	91	1.8	73	18	19.8
13	IRAN	74	1.5	42	32	43.2
14	PORTUGAL	49	1	34	15	30.6
15	BRAZIL	41	0.8	30	11	26.8
16	POLAND	36	0.7	31	5	13.9
17	ISRAEL	35	0.7	19	16	45.7
18	CHILE	33	0.7	11	22	66.7
19	BELGIUM	32	0.6	17	15	46.9
20	RUSSIA	31	0.6	27	4	12.9
21	SWEDEN	31	0.6	16	15	48.4
22	GREECE	29	0.6	20	9	31
23	EGYPT	28	0.6	15	13	46.4
24	NETHERLANDS	26	0.5	16	10	38.5