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ORIGINAL RESEARCH

Inflammation in Obstructive Sleep Apnea: A Global Bibliometric Perspective

Haixia Fan[1,*], Huiyan Niu[2,*], Bomeng Zhao[2], Xiaoling Gao[3]

[1]Department of Sleep Center, First Hospital of Shanxi Medical University, Taiyuan, Shanxi Province, People's Republic of China; [2]Second College of Clinical Medicine, Shanxi Medical University, Taiyuan, Shanxi Province, People's Republic of China; [3]Department of Sleep Center, Second Hospital of Shanxi Medical University, Taiyuan, Shanxi Province, People's Republic of China

*These authors contributed equally to this work

Correspondence: Xiaoling Gao, Department of Sleep Center, Second Hospital of Shanxi Medical University, Taiyuan, Shanxi Province, People's Republic of China, Tel +86 13513607801, Email yihexiyuan@sxmu.edu.cn

Purpose: Obstructive sleep apnea (OSA) is increasingly recognized as a systemic disorder in which inflammation plays a pivotal role in its pathophysiology and comorbidities. This study aimed to conduct the first comprehensive bibliometric analysis of global research on inflammation in OSA, in order to map knowledge structures, identify influential contributors, and highlight emerging research trends.

Patients and Methods: Publications related to OSA and inflammation were retrieved from the Web of Science Core Collection (WoSCC) (1996–2025). After exclusions, 2,075 articles and reviews were analyzed using bibliometric tools including CiteSpace, VOSviewer, and the bibliometrix R package. Citation frequencies, co-citation networks, and collaboration patterns among countries, institutions, authors, and journals were examined.

Results: The number of publications has shown robust growth (annual rate 12.11%). China and the United States were the leading contributors, with Harvard University, the University of Chicago, and Institut National De La Sante Et De La Recherche Medicale (INSERM) among the most productive institutions. Influential authors included Gozal D, Kheirandish-Gozal L, and Khalyfa A. *Sleep and Breathing* was the most prolific journal, while *Circulation* and *American Journal of Respiratory and Critical Care Medicine* were highly co-cited sources. Highly cited works established the link between intermittent hypoxia, systemic inflammation, and cardiovascular/metabolic consequences. Thematic clusters revealed major research focuses on cardiovascular disease, metabolic syndrome, oxidative stress, neuroinflammation (including microglial activation), and therapeutic strategies such as continuous positive airway pressure (CPAP), adenotonsillectomy, and bariatric surgery. Emerging topics included gut microbiota, vitamin D, and neurodegenerative diseases.

Conclusion: Research on inflammation is evolving from mechanistic insights toward clinical management and interdisciplinary exploration. Looking ahead, priority should be given to biomarker discovery and the development of anti-inflammatory therapeutic strategies. These findings provide a roadmap for targeted research and clinical translation in the field of OSA-related inflammation, ultimately improving patient outcomes.

Keywords: obstructive sleep apnea, inflammation, bibliometric analysis, mechanisms, comorbidities, treatment

Introduction

Obstructive sleep apnea (OSA) is an increasingly common disorder of repeated upper airway collapse during sleep, which leads to oxygen desaturation and disrupted sleep.[1] Since the first description of OSA in the 1960s,[2] and with further understanding of the pathophysiology of upper airway collapse and the hemodynamic effects of OSA in the 1980s,[3] along with the introduction of continuous positive airway pressure (CPAP) as an effective treatment,[4] it is estimated that approximately 1 billion adults worldwide may suffer from OSA, with an estimated 425 million individuals having moderate to severe OSA up to now. The most affected population is in China, followed by the United States, Brazil, and India.[5] OSA is associated with an increased risk of work-related and traffic accidents, more frequent health-related absenteeism, and a decline in quality of life.[6] Therefore, research on OSA is of great significance.

Accumulating evidence supports that OSA is a chronic low-grade inflammatory condition, in which inflammation may play a pivotal role in its pathophysiology, comorbidities, and associated oxidative stress.[7] The literature by A.N. Vgontzas indicates that the hypoxic-ischemic response induced by OSA leads to elevated levels of various pro-

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inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which play critical roles in oxidative stress and endothelial dysfunction.[8] Takuya Yokoe's research further shows that inflammatory markers, such as C-reactive protein (CRP) and IL-6, are significantly elevated in OSA patients, and these elevated levels are closely linked to the occurrence of cardiovascular events.[9] Therefore, a comprehensive and up-to-date analysis of OSA and its relationship with inflammation is of great importance.

Bibliometric analysis, which makes use of citation data to quantitatively evaluate the spread and influence of scientific works across various sectors within a specific discipline, has become a highly useful approach for analyzing research trends and scholarly contributions.[10,11] This method plays a key role in providing a more precise and up-to-date perspective on scientific advancements related to OSA, thereby assisting in evidence-based decision-making and informing the development of research strategies and policies.[12] The primary aim of this study was to explore the progression of research on OSA and inflammation in recent decades, employing co-citation network analysis to pinpoint influential studies and identify emerging areas of inquiry. A secondary aim was to comprehensively map the research environment surrounding OSA and inflammation, quantifying the contributions of significant entities such as countries, institutions, authors, and journals. In addition, this analysis intends to uncover existing gaps in research, limitations, and unexplored avenues, providing valuable insights for shaping future research directions and formulating targeted policy measures in the OSA and inflammation field.

Materials and Methods

Database and Search Strategy

The Web of Science Core Collection (WoSCC), a widely used dataset in bibliometrics,[13] was retrieved for this study. Searches were performed on April 16, 2025, using terms related to inflammation[14] and OSA[15,16] in the title (TI), abstract (AB), and author keywords (AK). Detailed search terms are provided in Supplementary Table 1. The initial search yielded 1,294,829 inflammation-related records and 63,736 OSA-related records. Intersecting the two datasets resulted in 2,394 publications. After excluding proceeding papers, corrections, early access articles, news items, book chapters, retractions, reprints, biographical items, book reviews, meeting abstracts, editorial materials, and letters, and restricting to English-language articles and reviews, 2,075 studies remained for analysis (Figure 1).

Data Processing

The data covered the period from 1 January 1996 to 16 April 2025, with the data segmented into one-year time slices for detailed analysis. All records from the WoSCC were exported as "full records and cited references" in plain text format and Tab format.[17] We utilized Microsoft Excel 2019, Online Analysis Platform of Literature Metrology (OALM) (http://bibliometric.com/), CiteSpace (6.4.R1, 64-bit Advanced Edition), VOSviewer (1.6.20), and the bibliometrix R package for network and citation analysis, including the generation of knowledge maps, cluster analysis, network visualization, and historiographic tracking of trends in journals and authors.[18] Key metrics such as the g-index,[19] h-index,[20] number of citations (NC), and number of publications (NP) were also calculated.[21]

Results

Publication Trends and General Characteristics

Figure 2A displays the annual count of publications and corresponding citations from 1996 to 2025. The number of publications has increased steadily over the years, reaching over 158 by 2024. The number of citations, on the other hand, surged significantly starting in 2005, peaking at nearly 9,000 in 2023 and 2024. Notably, these documents exhibit an impressive annual growth rate of 12.11%, indicating a robust increase in scholarly outputs. The significant decline in the volume of documents in 2025 may be due to the fact that the data only included records up to April 16, 2025. As of the retrieval date, 2,075 papers sourced from 693 different sources were published over the last 40 years. Among them, articles account for the majority. There were 1,572 articles, accounting for 75.76%, and 503 reviews, accounting for 24.24% of the papers (Figure 2B and Supplementary Table 2). Additionally, Figure 2C shows the cumulative number of publications over the study period, depicting an exponential growth trend that is consistent with the general rise in scientific productivity. Figure 2D shows the annual publication count for each country. The stacked bars show how the

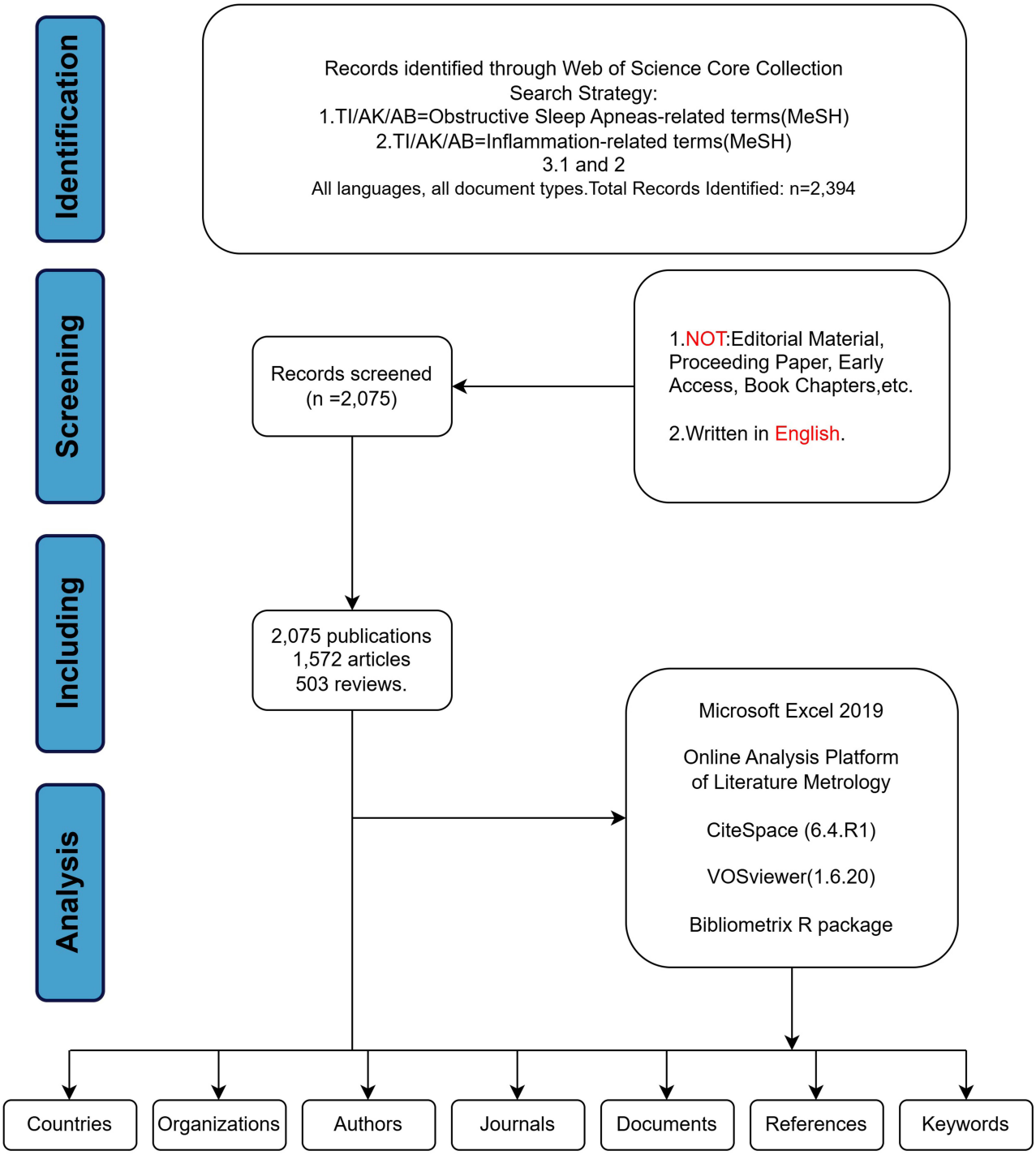

Figure 1 Flowchart of Literature Search, Screening, Inclusion, and Analysis.
Abbreviations: TI, Title; AK, Author keywords; AB, Abstract.

contributions from each country evolve over time. From 1996 to the early 2000s, the number of publications was relatively low, starting around 2010, there was a sharp rise in publications, indicating a growing research output. By 2020 and beyond, the total number of publications per year reached over 100. Overall, these findings demonstrate a steady growth of research activity with a marked acceleration in the past decade, highlighting the increasing scholarly attention to OSA and inflammation and its emerging significance as a sustained research hotspot.

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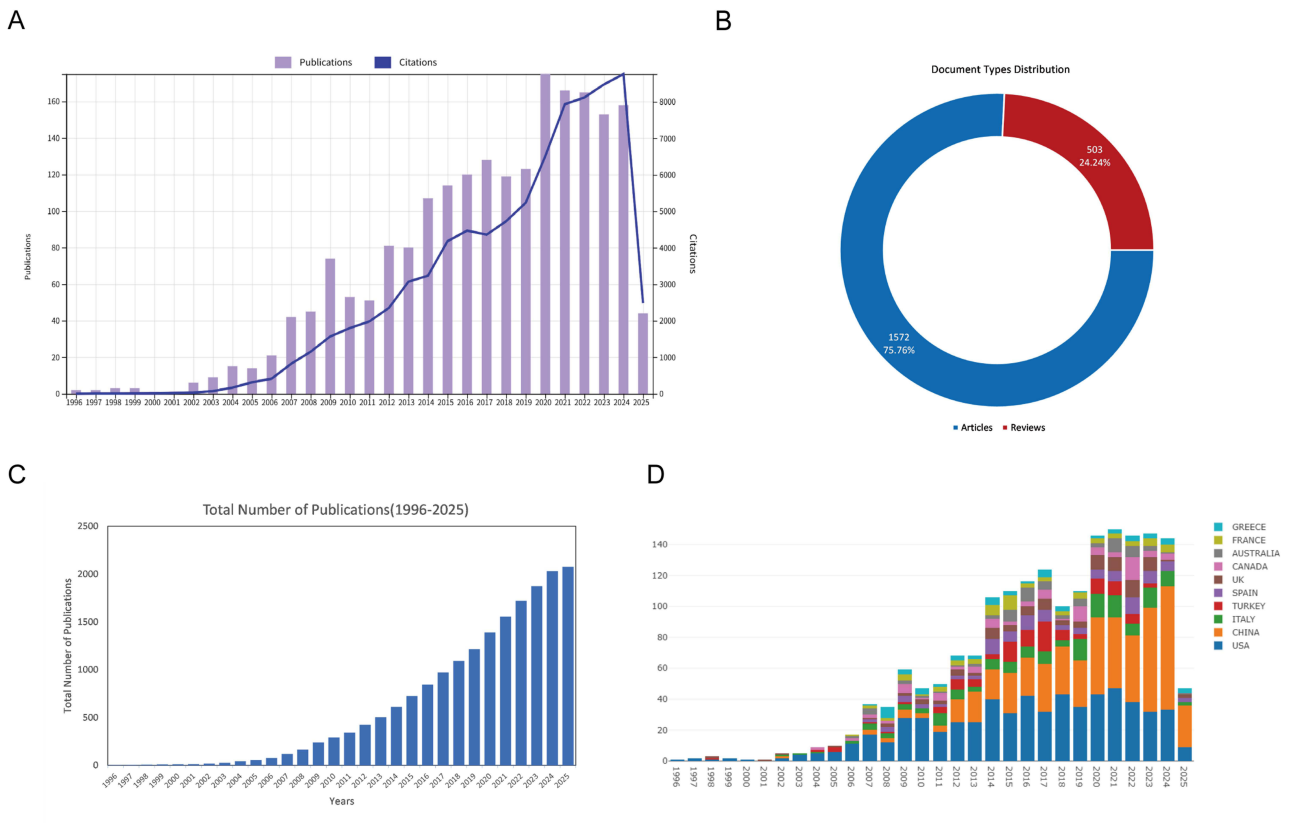


Figure 2 Overview of Publications and Citations on Inflammation in Obstructive Sleep Apneas (1996–2025). (**A**) Annual number of publications (purple bars) and citations (blue line). (**B**) Distribution of document types, including articles (blue) and reviews (red). (**C**) Cumulative number of publications from 1996 to 2025. (**D**) Annual number of publications by country, with different colors representing different countries.

Collaboration Among Countries/ Regions

Our analysis of WoSCC data reveals that, 2,075 publications originated from 73 countries, highlighting the collaborative nature of research worldwide. The country collaboration map (Figure 3A) illustrates strong international partnerships, particularly between the US, Europe, and Asia, with the US and China standing out as the dominant players. Figure 3B emphasizes the preeminent role of US and China, aligning with the network visualization in Figure 3A. China and the United States are global leaders in academic output. From 1996 to 2025, the two countries published 516 articles (24.6%) and 488 articles (23.5%), far outpacing other countries such as Italy and Turkey, which published 122 articles (15.9%) and 103 articles (5%), respectively (Figure 3C). The analysis of the most cited countries further emphasizes impact, with the US leading in both the number of publications and citations (28,361), followed by China (8,214) and the Canada (4,622) (Figure 3D). The trend toward multi-country collaborations is particularly evident, reflecting a global shift towards more integrated and cross-border research endeavors. Figure 3C distinguishes between single-country publications (SCP) and multiple-country publications (MCP). China has shown remarkable performance in single-country publications, highlighting its strength in both independent research and growing influence in the academic world. The United States dominates in multi-country publications, demonstrating its strength in international collaboration (Supplementary Table 3). In summary, the analysis reveals that China and the United States are not only leading contributors but also key drivers of both independent and collaborative research, underscoring the importance of international partnerships in shaping the global research landscape.

Institutional Collaboration and Author/Co-Author Networks

In addition to identifying leading institutions, our analysis explores their collaborative networks. Figure 4A highlight the top 5 institutions by research productivity over time: Harvard University (157,7.56%), University of California System

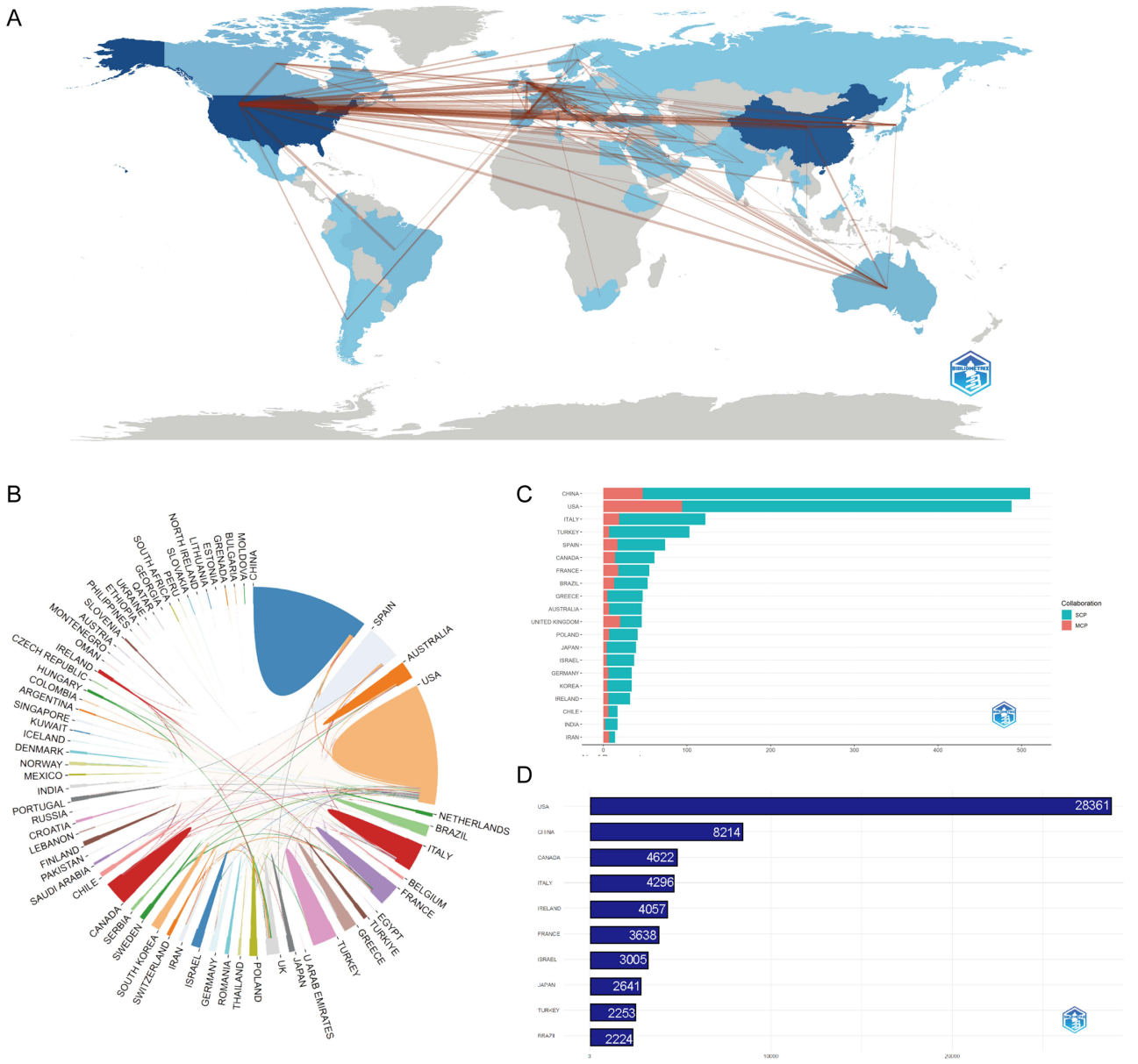


Figure 3 Global Trends and Collaboration Networks in Scientific Publications on Inflammation in Obstructive Sleep Apneas. (**A**) Global collaboration map of co-authorship networks, with darker blue indicating higher publication output and lines representing international collaboration. (**B**) Circular plot of international collaboration among countries. (**C**) Top 10 countries by number of publications, classified as single-country publications (SCP, red) or multiple-country publications (MCP, green). (**D**) Country influence measured by total citation counts.

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

(118,5.68%), Harvard University Medical Affiliates (103,4.96%), Chang Gung Memorial Hospital (95,4.57%), Institut National De La Sante Et De La Recherche Medicale (INSERM) (88,4.24%) and University of Chicago (88,4.24%) are tied for fifth place. Figure 4B further reveals a complex interaction network, with Harvard Medical School and the University of Chicago appearing as key nodes, highlighting their pivotal roles in global research collaboration. This collaboration trend is driven by the complex nature of modern scientific challenges, which require diverse expertise, and results in enhanced visibility, citation impact, and interdisciplinary innovation. Notably, European institutions display a stronger tendency toward multi-institutional partnerships, likely driven by European Union research funding policies that encourage international collaboration.

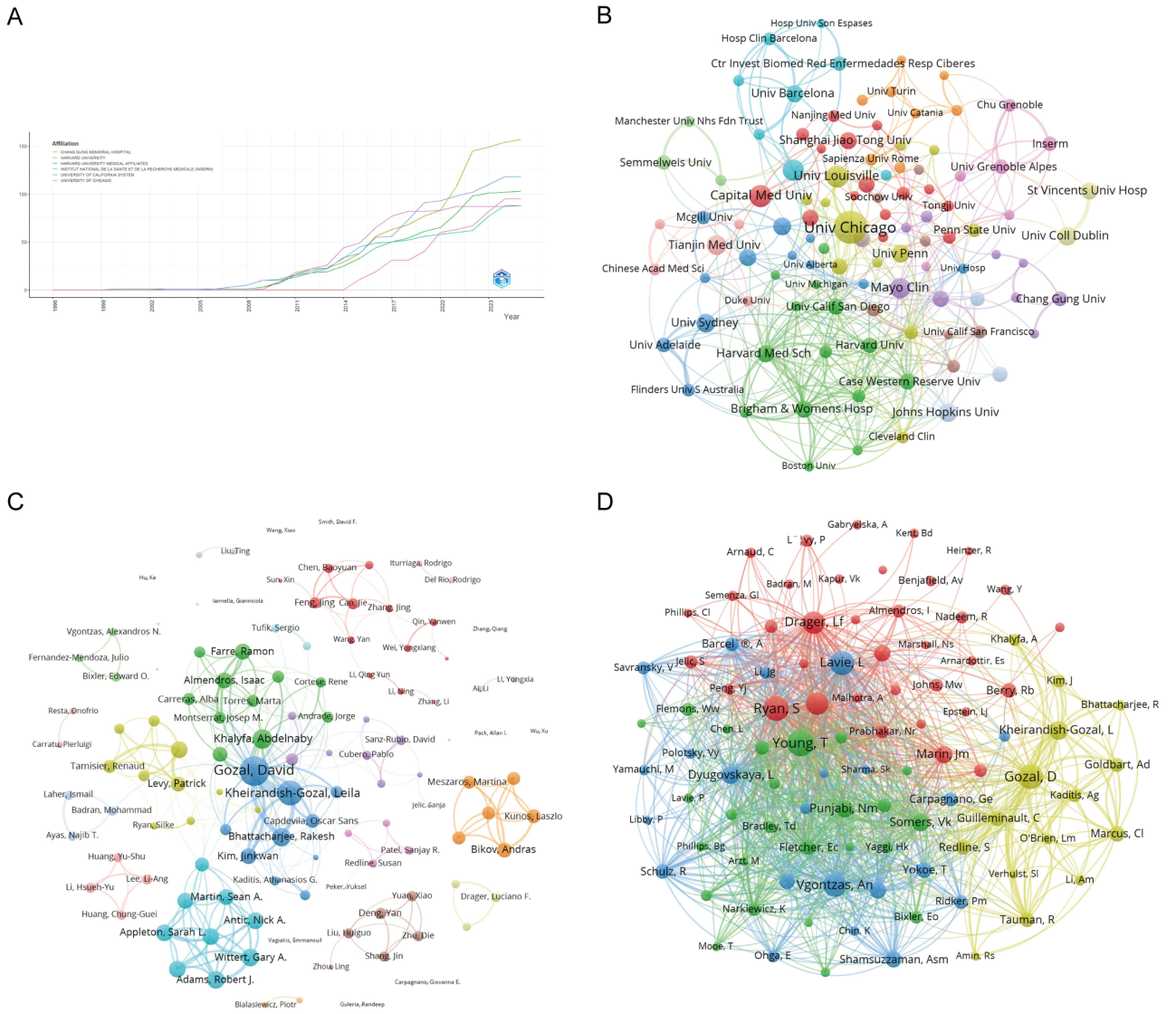


Figure 4 Institutional Contributions, Collaboration Networks and Authorship Analyses on Inflammation in Obstructive Sleep Apneas. (**A**) Institutional publication trends over time. The x-axis represents years, and the y-axis indicates publication counts. (**B**) Institutional collaboration network, where node size reflects collaboration extent and colors represent institutional clusters. (**C**) Co-authorship network: nodes represent authors (sized by publication count), and colors indicate collaboration clusters. (**D**) Co-cited authorship network: nodes represent authors who are frequently cited together; edge thickness reflects co-citation strength, and colors represent research groups.

The contributions of authors to the academic landscape are substantial, with 9,147 individuals contributing to these documents. Of these, 52 authors have produced single-authored works, resulting in a total of 66 single-authored documents. Collaboration is prominent, as evidenced by an average of 6.45 co-authors per document and 18.51% of documents involving international co-authorship (Supplementary Table 2).

In Figure 4C, distinct clusters represent specialized research communities, with authors like Gozal, David, Khalyfa, Abdelnaby, and Wittert, Gary A. at the center, indicating their significant roles. The proximity of these clusters suggests overlapping research areas and interdisciplinary connections. Figure 4D depicts the co-cited author network, emphasizing the collective influence of key figures such as Young, T, Lavie, L, and Gozal, D, who form foundational hubs with dense inter-author connections. Table 1 quantifies author influence using metrics such as publication count, h-index, g-index, and citation frequency. The h-index and g-index are both bibliometric indicators used to assess a researcher's productivity and citation impact. However, the h-index identifies the largest number h such that a researcher has h papers with at least h citations each, without giving extra weight to highly cited papers.[20] In contrast, the g-index is defined as the largest number g such that the total citations of the top g papers are at least g^2, giving more emphasis to highly cited

Table 1 Top 10 Authors on Inflammation in Obstructive Sleep Apneas Research (1996–2025)

Rank	Author	h_index	g_index	m_index	TC	NP	PY_start	Articles	Articles Fractionalized
1	Gozal D	46	81	2.091	6800	96	2004	96	21.19
2	Kheirandish-Gozal L	35	52	1.75	3680	52	2006	52	10.97
3	Mcnicholas WT	20	24	0.952	4066	24	2005	24	11.74
4	Bhattacharjee R	18	21	1.059	1114	21	2009	21	3.43
5	Wang Y	18	35	1.2	1295	35	2011	35	5.21
6	Khalyfa A	17	21	1	878	21	2009	21	3.35
7	Kim J	16	20	0.941	929	20	2009	20	3.42
8	Lévy P	16	17	0.842	2231	17	2007	17	2.80
9	Pépin JL	16	20	0.842	2138	20	2007	20	2.46
10	Redline S	14	21	0.667	1289	21	2005	21	3.59

Abbreviations: TC, Total Citations; NP, Number of Publications; PY_start, Year of First Publication.

works.[19] Table 1 shows that, Gozal D has an h-index of 46 and a g-index of 81, while Kheirandish-Gozal L has an h-index of 35 and a g-index of 52, reflecting their substantial impact. Mcnicholas WT's high co-authorship count, coupled with an h-index of 20 and g-index of 24, highlights his significant collaborative contributions. Bhattacharjee R and Wang Y, with an h-index of 18, have a high publication count and high citation frequencies, underscoring their influence, collaborative impact and roles as central figures in the literature. Collectively, these results demonstrate that institutional and author collaborations are highly clustered around a few influential centers and scholars, which drive the intellectual development and research impact of the field.

Top Journals and Co-Citation Patterns

Figure 5A ranks journals by the number of published articles, with the "Sleep and Breathing" leading (149 articles), followed by the " Sleep Medicine" (60 articles) and "Sleep" (46 articles), which both contribute significantly to the literature. Bradford's Law, illustrated in Figure 5B, reveals a skewed distribution of scholarly output: a small number of journals contribute disproportionately to the number of articles. Core sources, represented in the shaded region, include prominent journals like "Journal of Clinical Sleep Medicine" "Sleep Medicine Reviews" and "Journal of Sleep Research" marking these as primary venues for critical advancements and scholarly discussions in sleep science. Figure 5A and B both indicate that the "Sleep and Breathing" is a key source, in terms of the number of documents published and citation frequency. Figure 5C shows the cumulative number of articles over time, with consistent growth in output from leading journals. Notably, "Sleep and Breathing" has exhibited steady growth since 2010, "Sleep Medicine" has steadily increased since 2015, reaching nearly 50 articles by 2022. Both the "Journal of Clinical Sleep Medicine" and "Sleep" have shown slower growth, but still exhibit a notable accumulation of articles. Overall, the number of publications began to rise after 2000, with a significant acceleration after 2010, reflecting the expansion of research in the field. This trend indicates that the association between OSA and inflammation has become a research hotspot, likely driven by the clinical significance of OSA, the deeper exploration of inflammatory mechanisms, and advancements in related technologies (Table 2). The journal citation network (Figure 5D) identifies clusters of journals that are frequently cited together, indicating strong research communities around specific themes. The co-citation network (Figure 5E) further highlights the strength of these relationships, with prominent nodes like "Circulation" and "American Journal of Respiratory and Critical Care" acting as central hubs. Together, these findings suggest that a limited number of core journals dominate the dissemination of knowledge on OSA and inflammation, thereby shaping the primary avenues for scholarly communication in this field.

Highly Cited Studies and Thematic Clustering

In total, the dataset includes 2,075 documents and 71,34 references (Supplementary Table 2). The average age of these documents is 7.84 years, while each document has been cited 39.67 times, reflecting their substantial impact within the academic community. Table 3 display that the most frequently cited documents with major contributions from ground-breaking studies such as "Elevated Levels of C-Reactive Protein and Interleukin-6 in Patients With Obstructive Sleep

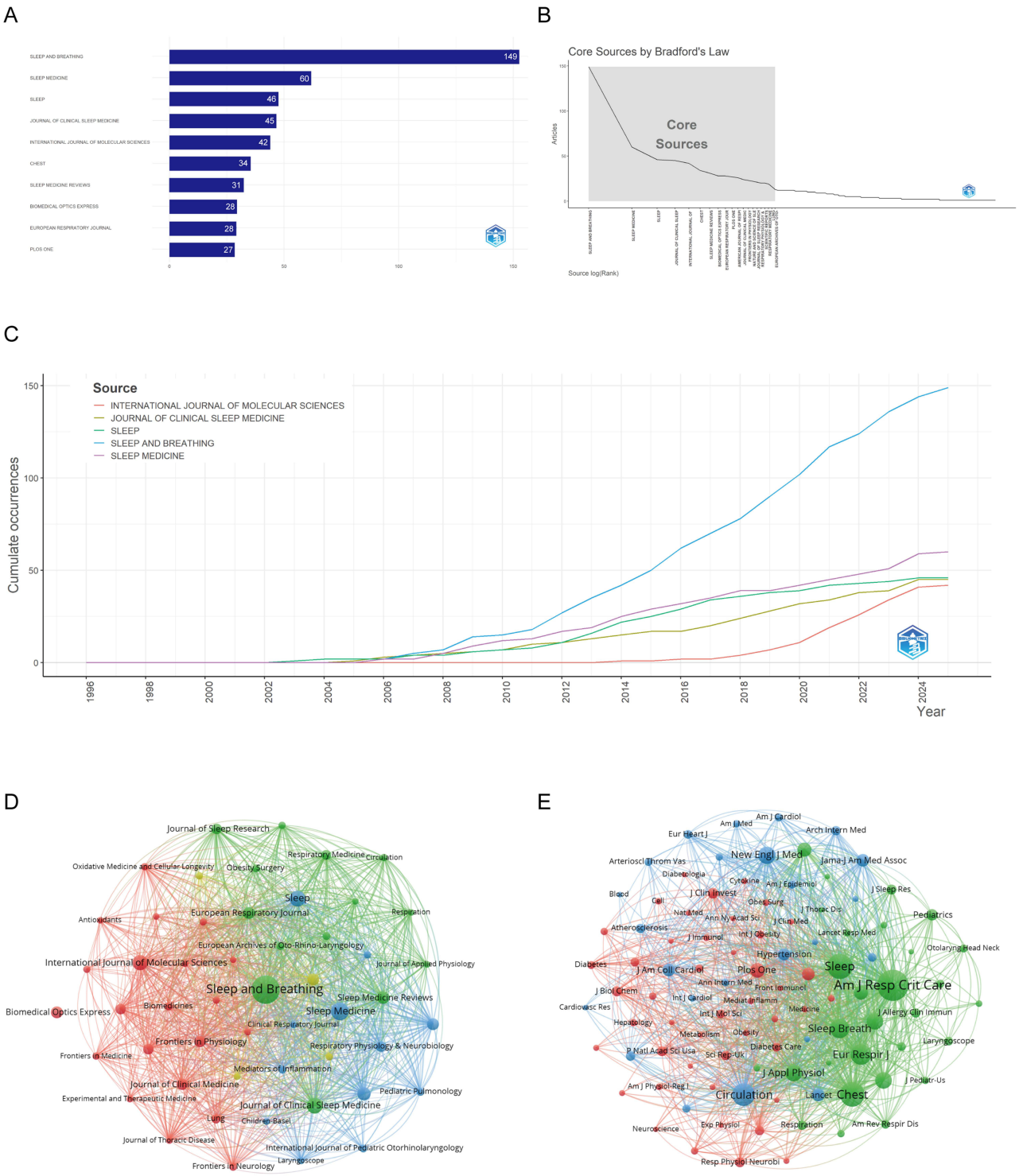


Figure 5 Analysis of Journals and Citation Networks Related to Inflammation in Obstructive Sleep Apneas. (**A**) Top journals ranked by publication count. (**B**) Bradford's Law analysis identifying core journals. (**C**) Publication trends of the top five journals over time. (**D**) Journal citation network, where nodes represent journals connected by citation links; colors indicate thematic clusters. (**E**) Journal co-citation network, where node size reflects co-citation frequency and colors represent clusters of commonly co-cited journals.

Apnea Syndrome Are Decreased by Nasal Continuous Positive Airway Pressure"[9] and "Selective Activation of Inflammatory Pathways by Intermittent Hypoxia in Obstructive Sleep Apnea Syndrome"[22] in Circulation, with 256 and 240 local citations respectively. The high yearly citation rates demonstrate their substantial impact on advancing the

Table 2 Top 10 Influential Journals on Inflammation in Obstructive Sleep Apneas Research (1996–2025)

Rank	Source	h_index	g_index	m_index	TC	NP	PY_start	IF	JCR
1	Sleep and Breathing	29	45	1.45	2978	149	2006	2	Q3
2	Sleep	28	46	1.217	2551	46	2003	4.9	Q1
3	Chest	26	34	0.897	3739	34	1997	8.6	Q1
4	American Journal of Respiratory and Critical Care Medicine	25	26	1.136	3705	26	2004	19.4	Q1
5	Sleep Medicine	25	42	1.25	1875	60	2006	3.4	Q2
6	European Respiratory Journal	24	28	1.143	3143	28	2005	21	Q1
7	Sleep Medicine Reviews	23	31	1	3145	31	2003	9.7	Q1
8	Journal of Clinical Sleep Medicine	20	41	0.952	1745	45	2005	2.9	Q2
9	PLOS ONE	18	27	1.059	1165	27	2009	2.6	Q2
10	International Journal of Molecular Sciences	15	26	1.25	753	42	2014	4.9	Q1

Abbreviations: IF, Impact Factor; JCR, Journal Citation Reports; TC, Total Citations; NP, Number of Publications; PY_start, Year of First Publication.

understanding of Obstructive Sleep Apneas and Inflammation. Other influential studies, such as "Elevated C-Reactive Protein in Patients With Obstructive Sleep Apnea"[23] and "Obstructive sleep apnoea syndrome–an oxidative stress disorder",[24] have also made significant contributions, each with over 170 local citations. These works have been pivotal in elucidating the mechanisms of activation of inflammatory pathways and the elevation of CRP levels in OSA.

Figure 6A reveals distinct thematic clusters in OSA and Inflammation research. Cluster 0 ("fat-laden hepatocyte") serves as a core theme, with dense connections to other clusters, highlighting the critical role of fat-laden hepatocytes in the inflammatory mechanisms associated with OSA and the overall pathology. Cluster 1 ("cardiovascular consequence") emphasizes the association between OSA-induced inflammation and cardiovascular complications such as hypertension and atherosclerosis, reflecting the pivotal role of inflammation in the long-term health consequences of OSA. Cluster 10 ("intermittent hypoxia") suggests that intermittent hypoxia (IH), a core pathological feature of OSA, accelerates disease progression by inducing oxidative stress and inflammatory responses. Other important clusters include Cluster 8 ("continuous positive airway pressure"), where CPAP provides significant evidence for the clinical treatment and management of inflammation in OSA by improving nocturnal intermittent hypoxia. Cluster 2 ("metabolic syndrome") highlights metabolic disorders such as insulin resistance, hypertension, and dyslipidemia as important comorbidities and inflammation-driving factors in OSA. Taken together, Figures 6A illustrates not only the major research hotspots but also the interconnections among pathophysiological mechanisms, clinical consequences, and therapeutic strategies. The clustering patterns highlight that OSA-related inflammation is a multifactorial process involving metabolic, cardiovascular, and respiratory pathways. These insights underscore the central role of inflammation in linking OSA to systemic comorbidities and point toward potential translational directions for targeted interventions.

Figure 6B has shown the top 25 references in the field, ranked by the strength of their citation bursts between 2003 and 2025. The citation burst strength is a dynamic measure that reflects the frequency and intensity of citations during a given period. For instance, Benjafield's 2019 article[5] stands out with a remarkable citation burst strength of 46.61 in 2021, suggesting its pivotal role in shaping subsequent research on OSA and inflammation. In contrast, more recent papers, such as those by Yeghiazarians[31] and Gottlieb,[32] show bursts in citation strength around 2022 and 2021 respectively, reflecting ongoing developments and growing interest in their work on OSA and inflammation. Overall, the most influential studies and thematic clusters converge on the mechanisms, comorbidities, and clinical management of OSA-related inflammation, reflecting both the historical foundations and evolving frontiers of the field.

Keyword Co-Occurrence and Research Themes

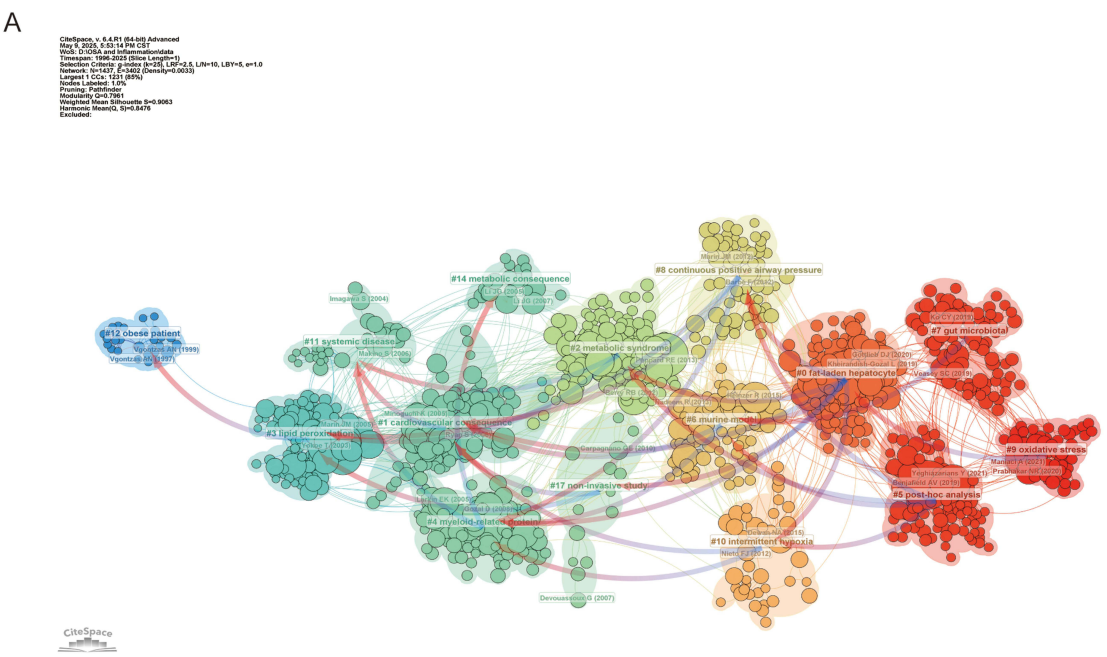
The keyword analysis reveals significant trends and focal points in OSA research. Specifically, we identified 3,894 Keywords plus alongside 3,250 author keywords, enhancing the effective indexing and retrieval of scholarly content (Supplementary Table 2).

Table 3 Top 10 Most Cited Publications Based on Bibliometrix Analysis (1996–2025)

Rank	First Author	Year	Journal	Paper	DOI	Local Citations	Global Citations	LC/GC Ratio (%)	Normalized Local Citations	Normalized Global Citations
1	Yokoe T[9]	2003	Circulation	Elevated Levels of C-Reactive Protein and Interleukin-6 in Patients With Obstructive Sleep Apnea Syndrome Are Decreased by Nasal Continuous Positive Airway Pressure	10.1161/01.CIR.0000052627.99976.18	256	750	34.13	3.10	2.03
2	Ryan S[22]	2005	Circulation	Selective Activation of Inflammatory Pathways by Intermittent Hypoxia in Obstructive Sleep Apnea Syndrome	10.1161/CIRCULATIONAHA.105.556746	240	729	32.92	5.61	4.48
3	Shamsuzzaman ASM[23]	2002	Circulation	Elevated C-Reactive Protein in Patients With Obstructive Sleep Apnea	10.1161/01.CIR.0000018948.95175.03	201	734	27.38	4.45	4.11
4	Lavie L[24]	2003	Sleep Med Rev	Obstructive sleep apnoea syndrome – an oxidative stress disorder	10.1053/smrv.2002.0261	174	779	22.34	2.11	2.10
5	Nadeem R[25]	2013	J Clin Sleep Med	Serum Inflammatory Markers in Obstructive Sleep Apnea: A Meta-Analysis	10.5664/jcsm.3070	135	373	36.19	13.71	6.49
6	Ryan S[26]	2006	Am J Resp Crit Care	Predictors of Elevated Nuclear Factor-κB–dependent Genes in Obstructive Sleep Apnea Syndrome	10.1164/rccm.200601–066OC	130	283	45.94	4.82	1.57
7	McNicholas WT[27]	2007	Eur Respir J	Sleep apnoea as an independent risk factor for cardiovascular disease: current evidence, basic mechanisms and research priorities	10.1183/09031936.00027406	104	705	14.75	5.79	7.85
8	Ohga E[28]	2003	J Appl Physiol	Effects of obstructive sleep apnea on circulating ICAM-1, IL-8, and MCP-1	10.1152/japplphysiol.00177.2002	101	263	38.40	1.22	0.71
9	Jelic S[29]	2008	Circulation	Inflammation, Oxidative Stress, and Repair Capacity of the Vascular Endothelium in Obstructive Sleep Apnea	10.1161/CIRCULATIONAHA.107.741512	96	458	20.96	5.22	5.30
10	Ciftci TU[30]	2004	Cytokine	The relationship between serum cytokine levels with obesity and obstructive sleep apnea syndrome	10.1016/j.cyto.2004.07.003	92	232	39.66	3.64	2.44

Abbreviations: DOI, Digital Object Identifier; LC/GC Ratio, Local Citation to Global Citation Ratio.

A



B

Top 25 References with the Strongest Citation Bursts

References	Year	Strength	Begin	End	1996 - 2025
Shamsuzzaman ASM, 2002, CIRCULATION, V105, P2462, DOI 10.1161/01.CIR.0000018948.95175.03, DOI	2002	21.51	**2003**	2007	
Dyugovskaya L, 2002, AM J RESP CRIT CARE, V165, P934, DOI 10.1164/ajrccm.165.7.2104126, DOI	2002	15.09	**2003**	2007	
Yokoe T, 2003, CIRCULATION, V107, P1129, DOI 10.1161/01.CIR.0000052627.99976.18, DOI	2003	29.87	**2004**	2008	
Lavie L, 2003, SLEEP MED REV, V7, P35, DOI 10.1053/smrv.2002.0261, DOI	2003	18.6	**2004**	2008	
Marin JM, 2005, LANCET, V365, P1046, DOI 10.1016/S0140-6736(05)71141-7, DOI	2005	24.45	**2006**	2010	
Ryan S, 2005, CIRCULATION, V112, P2660, DOI 10.1161/CIRCULATIONAHA.105.556746, DOI	2005	22.02	**2006**	2010	
Minoguchi K, 2005, AM J RESP CRIT CARE, V172, P625, DOI 10.1164/rccm.200412-1652OC, DOI	2005	16.78	**2006**	2010	
Minoguchi K, 2004, CHEST, V126, P1473, DOI 10.1378/chest.126.5.1473, DOI	2004	15.75	**2006**	2009	
Ryan S, 2006, AM J RESP CRIT CARE, V174, P824, DOI 10.1164/rccm.200601-066OC, DOI	2006	22.24	**2008**	2011	
Ryan S, 2009, THORAX, V64, P631, DOI 10.1136/thx.2008.105577, DOI	2009	14.91	**2010**	2014	
Gottlieb DJ, 2010, CIRCULATION, V122, P352, DOI 10.1161/CIRCULATIONAHA.109.901801, DOI	2010	15.8	**2011**	2015	
Redline S, 2010, AM J RESP CRIT CARE, V182, P269, DOI 10.1164/rccm.200911-1746OC, DOI	2010	14.84	**2011**	2015	
Peppard PE, 2013, AM J EPIDEMIOL, V177, P1006, DOI 10.1093/aje/kws342, DOI	2013	40.81	**2014**	2018	
Berry RB, 2012, J CLIN SLEEP MED, V8, P597, DOI 10.5664/jcsm.2172, DOI	2012	19.86	**2014**	2017	
Nadeem R, 2013, J CLIN SLEEP MED, V9, P1003, DOI 10.5664/jcsm.3070, DOI	2013	19.78	**2014**	2018	
Heinzer R, 2015, LANCET RESP MED, V3, P310, DOI 10.1016/S2213-2600(15)00043-0, DOI	2015	19.67	**2016**	2020	
Lavie L, 2015, SLEEP MED REV, V20, P27, DOI 10.1016/j.smrv.2014.07.003, DOI	2015	19.17	**2016**	2020	
McEvoy RD, 2016, NEW ENGL J MED, V375, P919, DOI 10.1056/NEJMoa1606599, DOI	2016	15.25	**2017**	2021	
Kapur VK, 2017, J CLIN SLEEP MED, V13, P479, DOI 10.5664/jcsm.6506, DOI	2017	20.28	**2018**	2022	
Javaheri S, 2017, J AM COLL CARDIOL, V69, P841, DOI 10.1016/j.jacc.2016.11.069, DOI	2017	16.96	**2018**	2022	
Senaratna CV, 2017, SLEEP MED REV, V34, P70, DOI 10.1016/j.smrv.2016.07.002, DOI	2017	16.49	**2018**	2022	
Kheirandish-Gozal L, 2019, INT J MOL SCI, V20, P0, DOI 10.3390/ijms20030459, DOI	2019	28.35	**2020**	2025	
Benjafield AV, 2019, LANCET RESP MED, V7, P687, DOI 10.1016/S2213-2600(19)30198-5, DOI	2019	46.61	**2021**	2025	
Gottlieb DJ, 2020, JAMA-J AM MED ASSOC, V323, P1389, DOI 10.1001/jama.2020.3514, DOI	2020	20.93	**2021**	2025	
Yeghiazarians Y, 2021, CIRCULATION, V144, PE56, DOI 10.1161/CIR.0000000000000988, DOI	2021	25.77	**2022**	2025	

Figure 6 Reference Co-citation Analysis and Citation Burst Detection on Inflammation in Obstructive Sleep Apnea. (**A**) Clustered co-citation network, where node size reflects citation frequency, edge thickness indicates co-citation strength, and colors represent major thematic clusters labeled by keywords. (**B**) Top 25 references with strongest citation bursts over time, with red segments indicating periods of heightened attention.

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Figure 7A depicts the overall keyword networks, with distinct clusters emerging around themes of OSA, inflammation, and intermittent hypoxia. Figure 7B, the word cloud, further highlights key themes, reflecting the network visualizations in Figure 7A. As Figure 7C show "obstructive sleep apnea" (1063 occurrences, total link strength = 2050) (Figure 7C, cluster 4 and 7) pathology is at the center of current sleep medicine. "inflammation" (475 occurrences, total link strength = 1195) and "oxidative stress" (150 occurrences, total link strength = 38) are central to the "pathophysiology" (15 occurrences, total link strength = 45) mechanisms of OSA. "Chronic intermittent hypoxia" (63 occurrences, total link strength = 125) induces an inflammatory response by activating the "nuclear factor-kappa b" (20

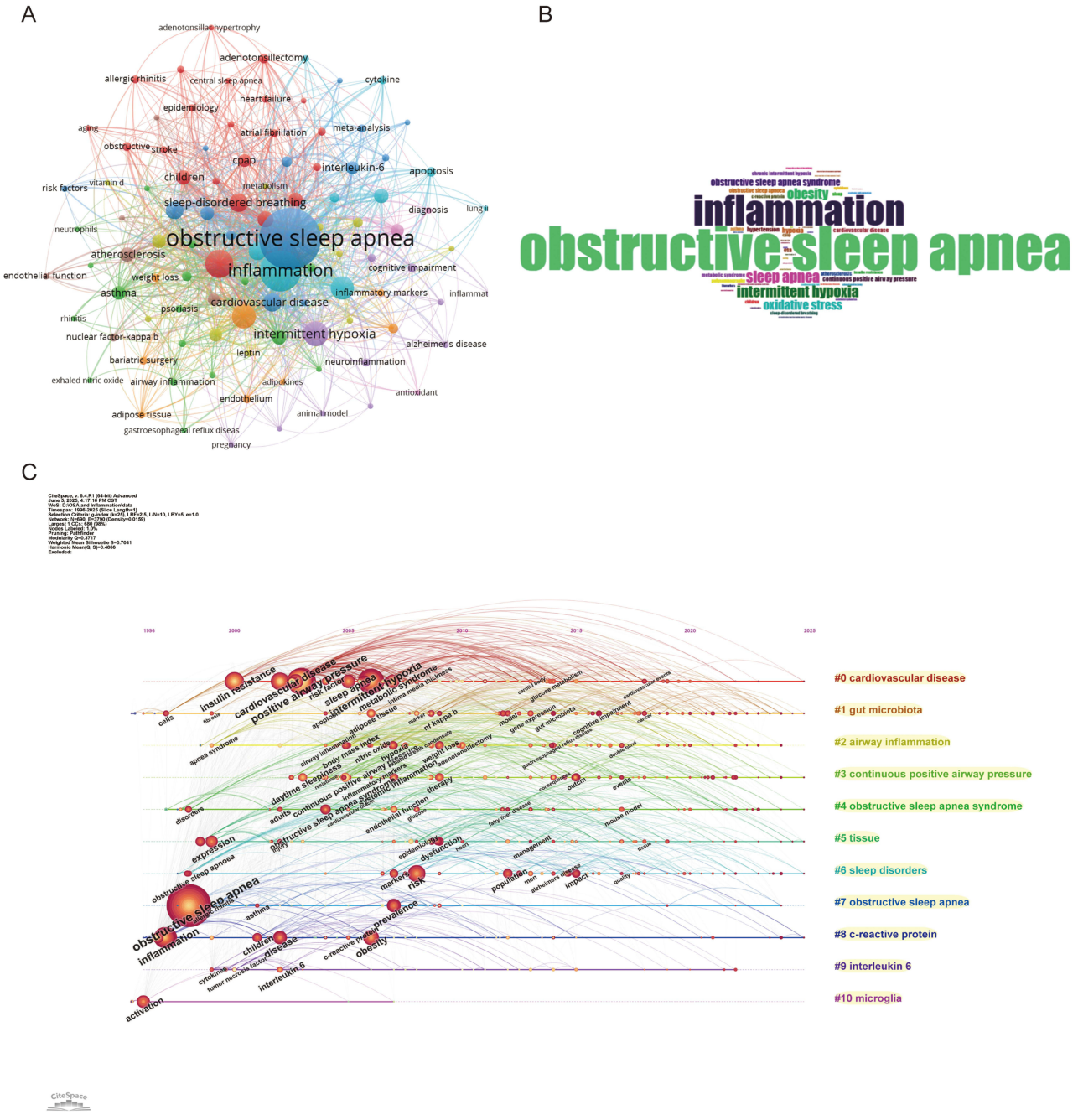

Figure 7 Keywords Mapping of Inflammation in Obstructive Sleep Apneas. (**A**) Keyword co-occurrence network, where nodes represent keywords (sized by frequency), edges indicate co-occurrence, and colors denote thematic clusters. (**B**) Word cloud of title keywords, with word size reflecting frequency. (**C**) Keyword timeline showing major clusters and their evolution over time; node size indicates frequency, node color represents time, and connections indicate co-occurrence relationships.

occurrences, total link strength = 48) signaling pathway, increasing the expression of systemic "inflammatory markers" (16 occurrences, total link strength = 35) such as "c-reactive protein" (56 occurrences, total link strength = 170) (Figure 7C, cluster 8), "interleukin-6" (46 occurrences, total link strength = 126) (Figure 7C, cluster 9), "tumor necrosis factor-alpha" (33 occurrences, total link strength = 97), thereby leading to "endothelial function" (14 occurrences, total link strength = 40) impairment and "tissue" (Figure 7C, cluster 5) damage. OSA can also lead to significant airway inflammation (Figure 7C, cluster 2).

"Hypertension" (73 occurrences, total link strength = 216), "atherosclerosis" (58 occurrences, total link strength = 163), "endothelial dysfunction" (27 occurrences, total link strength = 81), "atrial fibrillation" (21 occurrences, total link strength =60), "stroke" (17 occurrences, total link strength = 53), "coronary artery disease" (10 occurrences, total link strength = 38) suggest a close association between OSA and "cardiovascular disease" (95 occurrences, total link strength = 256) (Figure 7C, cluster 0). IH and inflammatory markers significantly increase "cardiovascular risk" (29occurrences, total link strength = 72) through inflammation and sympathetic nervous activation. "Metabolic syndrome" (64 occurrences, total link strength = 190), "diabetes" (18 occurrences, total link strength = 59), "adipose tissue" (15 occurrences, total link strength = 47), "adiponectin" (12 occurrences, total link strength = 28) and "non-alcoholic fatty liver disease" (28 occurrences, total link strength = 79) reflects the interaction between OSA and metabolic disorders. "Obesity" (167 occurrences, total link strength = 470) is a major risk factor for OSA and OSA exacerbates "insulin resistance" (47 occurrences, total link strength = 150) and "dyslipidemia" (12 occurrences, total link strength = 42) through inflammation and dysregulation of "adiponectin" (12 occurrences, total link strength = 28).

"Excessive daytime sleepiness" (11 occurrences, total link strength = 34), "insomnia" (14 occurrences, total link strength = 39) and "sleep disorders" (Figure 7C, cluster 6) highlights the disruption of sleep quality by OSA and its impact on cognitive function such as "cognitive impairment" (25 occurrences, total link strength = 64) and mental health such as "depression" (17 occurrences, total link strength = 52). "Polysomnography" (56 occurrences, total link strength = 133) and "bariatric surgery" (19 occurrences, total link strength = 38) suggest that research on the "diagnosis" (14 occurrences, total link strength = 40) and treatment of OSA is a key area of research. "Continuous positive airway pressure" (89 occurrences, total link strength =246) (Figure 7C, cluster 3), as the standard treatment, can significantly improve the "apnea-hypopnea index" (18 occurrences, total link strength = 44). Research on OSA in "children" (59 occurrences, total link strength = 168) and OSA during "pregnancy" (10 occurrences, total link strength = 23) have gradually gained attention. Adenotonsillectomy (30 occurrences, total link strength = 80) is the primary treatment for pediatric OSA.

Research on OSA is expanding into multidisciplinary fields, with a potential association between OSA and "Alzheimer's disease" (12 occurrences, total link strength = 26), "cancer" (9 occurrences, total link strength = 30), "gut microbiota" (17 occurrences, total link strength = 37) (Figure 7C, cluster 1) "microglia" (18 occurrences, total link strength =23) (Figure 7C, cluster 10), and "vitamin d" (11 occurrences, total link strength = 30).

In conclusion, the keyword analysis highlights that research on OSA and inflammation spans mechanisms, comorbidities, and treatments, while also expanding into emerging multidisciplinary domains, thereby underscoring its growing clinical and scientific relevance.

Discussion

The incidence and prevalence of OSA have increased substantially worldwide, posing a growing burden on healthcare systems and raising significant public health concerns. In this study, we conducted the first comprehensive bibliometric analysis of research on OSA-related inflammation. This bibliometric analysis is not intended to replace systematic reviews or mechanistic studies, but rather to provide a complementary perspective by mapping the knowledge structure and identifying underexplored research areas. Our results provide an integrated overview of publication trends, international collaborations, influential authors and institutions, landmark studies, and emerging research themes. Beyond presenting novel bibliometric patterns, these findings offer relevant insights into how the field has evolved, the intellectual structure that underpins it, and the potential directions for future exploration. Such insights are particularly valuable for guiding new research initiatives, fostering cross-disciplinary collaborations, and identifying underexplored areas with clinical and translational importance.

Placing these bibliometric findings into the clinical context is essential for understanding their significance. The increasing recognition of inflammation as a central mechanism in OSA pathophysiology helps explain why this research domain has become a sustained hotspot, bridging basic science with clinical practice. Current management strategies for OSA primarily aim to alleviate symptoms, with CPAP therapy being the most widely used intervention to mitigate the effects of intermittent hypoxia. Although such treatments improve sleep quality and provide short-term relief, they do not address the underlying inflammatory processes associated with OSA. As the link between OSA and systemic inflammation becomes clearer, there is growing interest in the role of inflammatory pathways in disease progression. In particular, the release of pro-inflammatory cytokines in response to hypoxic episodes is increasingly recognized as a central factor contributing to the long-term cardiovascular and metabolic complications observed in OSA patients. Understanding this intricate relationship between OSA and inflammation may pave the way for novel therapeutic strategies that target inflammatory mechanisms, potentially reducing the broader health impacts of OSA.

Research Highlights

The results of keyword and reference clustering analysis highlight the primary areas of interest for researchers. Combining these findings with our understanding of the field, we succinctly summarize several major research hotspots, including mechanism, comorbidities and treatment.

Mechanism

OSA is increasingly recognized as a systemic disorder linked to chronic inflammation and oxidative stress, with profound implications for both peripheral and central nervous system (CNS) health. The intermittent hypoxia caused by OSA activates various inflammatory pathways, primarily through the upregulation of nuclear factor kappa-B (NF-κB), which subsequently enhances the production of pro-inflammatory cytokines such as IL-6, IL-8, and TNF-α.[33] These cytokines are central to the development of systemic inflammation and tissue damage, which are implicated in the exacerbation of comorbidities, including cardiovascular diseases and cognitive impairment.[34,35] Additionally, OSA has been found to promote the polarization of M1 macrophages via NF-κB activation, further intensifying the inflammatory response and increasing susceptibility to disorders such as atrial fibrillation.[36] Moreover, the apnea index in OSA patients correlates positively with systemic inflammation markers, indicating that OSA may serve as a catalyst for chronic inflammatory states that further complicate metabolic health.[37] Evidence also suggests that CPAP therapy, a common treatment for OSA, can significantly reduce levels of inflammatory markers such as CRP, IL-6, and TNF-α, supporting the link between OSA and systemic inflammation.[38]

A particularly important aspect of OSA's neurobiological impact is its influence on microglial activation within the CNS. The blood-brain barrier (BBB), a vital protective structure, was once thought to shield the brain from peripheral inflammation and playing a crucial role in maintaining brain homeostasis.[39] However, emerging research indicates bidirectional communication between peripheral inflammation and neuroinflammation, challenging this notion.[40] Chronic peripheral inflammation has been shown to disrupt endothelial cell function, compromising the BBB's integrity and permeability, which triggers neuroinflammation.[41] In response to OSA-induced hypoxia, microglia, the brain's resident immune cells, are activated, leading to neuroinflammation. This activation is marked by increased levels of neuroinflammatory markers, which are associated with cognitive impairments.[42] Additionally, chronic intermittent hypoxia (CIH), a hallmark of OSA, results in changes to microglial function, including alterations in their numbers and gene expression, which may link OSA to neurodegenerative diseases like Alzheimer's disease (AD).[43] Furthermore, microglia exhibit a dual role in inflammation, acting as both promoters and regulators depending on the disease context. In neurodegenerative diseases, for instance, microglia switch between pro-inflammatory and anti-inflammatory states,[44,45] highlighting their complex involvement in the inflammatory response.

In conclusion, the complex interplay between OSA, systemic inflammation, and neuroinflammation, with microglia as central mediators, underscores the necessity of addressing these pathways in OSA management. Given the complexity of this network, further research into therapeutic strategies targeting inflammation and microglial activation may offer potential to alleviate the broader health risks associated with OSA.

Comorbidities

First, OSA and metabolic syndrome exhibit a strong interrelationship, where OSA may worsen the key components of metabolic syndrome, including obesity, insulin resistance, and dyslipidemia. The intermittent hypoxia and disrupted sleep patterns inherent in OSA may trigger an elevation in sympathetic nervous system activity and systemic inflammation, thereby further disrupting metabolic processes. Conversely, the presence of metabolic syndrome can exacerbate the occurrence and severity of OSA, resulting in a bidirectional interaction that complicates the clinical management and treatment of both disorders. Patients with obesity hypoventilation syndrome, a form of OSA, show significantly higher rates of metabolic syndrome, as evidenced by increased waist circumference and triglyceride levels, suggesting a strong association between the two conditions.[46] Two large cross-sectional epidemiological studies demonstrated that obstructive sleep apnea is independently associated with both metabolic abnormalities and an increased risk of developing diabetes, regardless of other risk factors.[47,48] Additionally, individuals with metabolic syndrome exhibit mixed treatment preferences for OSA, indicating a complex relationship, though not providing strong evidence of a direct association.[49]

Second, OSA is closely associated with cardiovascular disease (CVD), primarily through inflammatory mechanisms. Patients with OSA often present with elevated pulse wave velocity, a marker linked to increased all-cause and cardiovascular mortality, highlighting the significant cardiovascular risks posed by this condition.[50] Additionally, myocardial dysfunction, such as reduced left ventricular global longitudinal strain, is frequently observed in moderate-to-severe OSA cases, further emphasizing its cardiovascular impact.[51] Inflammation plays a crucial role in this connection, as it accelerates atherosclerosis and contributes to the progression of other cardiovascular diseases.[52] OSA is also strongly correlated with an increased risk of stroke, with a high prevalence of OSA observed in patients with ischemic stroke.[53] This relationship is further supported by the identification of shared differentially expressed genes, such as DUSP1, which are upregulated in both conditions.[54] Inflammation is a key mediator in this association, as systemic inflammation disrupts blood-brain barrier integrity and promotes thrombosis, thus increasing the likelihood of cerebrovascular events.[55] Moreover, inflammation following ischemic stroke exacerbates neural damage through mechanisms like pyroptosis, highlighting the bidirectional relationship between stroke and inflammation.[56]

Additionally, OSA is increasingly recognized for its complex interplay with systemic inflammation and comorbid respiratory diseases, particularly asthma and allergic rhinitis. Studies have demonstrated a significantly higher prevalence of asthma among individuals with OSA, suggesting a meaningful association between the two conditions.[57] The inflammatory pathophysiology underlying allergic rhinitis—driven primarily by Th2 immune responses—has been shown to exacerbate asthma symptoms, contributing to the heightened inflammatory burden observed in patients with OSA.[58] This systemic inflammation is corroborated by elevated levels of circulating inflammatory markers, which show a positive correlation with OSA severity.[37] Moreover, the relationship between OSA and chronic obstructive pulmonary disease (COPD) has emerged as a critical area of concern. OSA frequently coexists with stable COPD, and this overlap is associated with increased right ventricular systolic pressure, indicating adverse cardiovascular consequences.[59] In COPD, persistent inflammation characterized by elevated biomarkers such as C-reactive protein and interleukin-6 is strongly linked to disease progression and severity.[60] Asthma, a known risk factor for COPD, further supports the hypothesis of a shared inflammatory continuum bridging these respiratory conditions.[61] Together, these findings underscore the intricate pathophysiological connections among OSA, asthma, allergic rhinitis, and COPD, mediated through common inflammatory pathways.

What's more, OSA has emerged as a notable risk factor for the development and progression of several neurodegenerative diseases, most prominently AD. The hallmark feature of OSA—CIH—is strongly associated with enhanced neuroinflammatory responses, which are increasingly recognized as central to the pathophysiology of cognitive decline and AD. Experimental and clinical studies have demonstrated that CIH leads to upregulation of pro-inflammatory cytokines and elevated oxidative stress levels, both of which contribute to neuronal injury and impaired cognitive performance.[62] Moreover, cognitive dysfunction is frequently observed in OSA patients, with a clear correlation between OSA severity and amyloid-β deposition, as well as increased markers of neuroinflammation.[63] In addition, the prevalence studies indicate that there may be an increased prevalence of OSA in individuals with major depressive disorder. Studies of treatment of OSA indicate an improvement in both OSA and psychiatric symptoms.[64] Depression is commonly co-

morbid with OSA and has been shown to accelerate cognitive deterioration in AD, potentially through mechanisms involving amplified neuroinflammation and increased amyloid plaque burden.[65] Furthermore, elevated inflammatory biomarkers have been identified in individuals with depressive symptoms, and these markers are closely associated with worsening cognitive outcomes.[66] Collectively, these observations suggest that targeting neuroinflammatory processes may offer dual benefits—amelioration of depressive symptoms and attenuation of cognitive decline—in patients affected by OSA and related neuropsychiatric conditions.[67]

Treatment

CPAP therapy, a common treatment for OSA, has been shown to significantly reduce inflammatory markers, indicating its role in mitigating inflammation associated with OSA.[38] Adenotonsillectomy, a surgical intervention for OSA, also reduces systemic inflammation, as demonstrated by decreased levels of inflammatory biomarkers such as interleukin-8 and C-C chemokine ligand 5, which contribute to improvements in apnea-hypopnea index (AHI).[68] OSA is typically characterized by a low-grade inflammatory state, which contributes to its cardiovascular comorbidities. Although CPAP therapy remains the cornerstone of OSA management, adjunctive approaches such as weight loss and antioxidant-rich diets—including vitamins A, B, C, D, and E—have shown promise in alleviating the pathophysiology of OSA and mitigating cardiovascular risks[69] Weight loss, whether achieved through lifestyle changes or surgical means, is generally associated with a reduction in pro-inflammatory macrophages and an increase in anti-inflammatory cells, contributing to better management of OSA.[70] Moreover, bariatric surgery has a profound impact on systemic inflammation, with patients often experiencing reductions in inflammatory markers. These changes are typically associated with improvements in metabolic conditions.[71] However, it is important to note that weight loss may also trigger increased inflammation in certain contexts, such as during infections.[72] Thus, the relationship between weight loss and inflammation is complex, but overall, it appears to benefit OSA outcomes by reducing the respiratory burden and systemic inflammation.[53] In conclusion, the management of OSA requires a multifaceted approach that incorporates surgical interventions like adenotonsillectomy, the use of CPAP therapy, and lifestyle modifications, such as weight loss. These combined strategies not only help reduce inflammation but also significantly improve patient outcomes.

Future Research Directions

OSA has been increasingly recognized for its complex interactions with systemic and neuroinflammation, which play a crucial role in the pathogenesis of various comorbidities associated with OSA. As research in this field continues to evolve, there is growing interest in understanding how OSA-induced inflammation contributes to disease progression and the development of long-term health complications. Our findings suggest that investigating the molecular pathways through which OSA triggers inflammatory responses, and how these pathways affect various organs, could provide novel insights into potential therapeutic strategies. Future research should focus on identifying specific biomarkers and therapeutic targets within inflammatory pathways that are activated by OSA, which may lead to the development of more effective interventions for mitigating inflammation and preventing the chronic effects of OSA. Additionally, exploring the potential of anti-inflammatory treatments as adjuncts to existing OSA therapies could open new avenues for improving patient outcomes and reducing associated health risks.

Biomarker Discovery

Research should focus on identifying reliable biomarkers to improve early diagnosis, patient stratification, and treatment monitoring. The discovery of these biomarkers is gaining increasing attention, as recent studies suggest that these markers may not only improve the diagnosis of OSA but also provide deeper insights into its underlying mechanisms. For instance, Galectin-3 has been identified as a potential biomarker for sleep health, underscoring the role of inflammatory pathways in OSA.[73] Similarly, the chemokine Eotaxin has been linked to an increased risk of OSA, further highlighting the importance of inflammation in disease progression.[74] Genetic variants, such as those in the FTO gene, have also been associated with OSA, suggesting potential applications in both diagnostic and prognostic assessment.[75] Moreover, circulating non-coding RNAs (ncRNAs) have shown promise as diagnostic biomarkers for OSA hypoventilation syndrome, offering new opportunities for early detection and monitoring.[76] In addition, OSA-

related sleep fragmentation contributes to cardiovascular alterations, with elevated levels of matrix metalloproteinase-9 (MMP9) and oxidized low-density lipoprotein (ox-LDL) suggesting a role in cardiovascular remodeling.[77] Elevated levels of CHI3L1-Ab have also been observed in OSA patients compared to healthy controls, indicating its potential as an independent diagnostic biomarker.[78] Overall, these findings underscore the critical role of biomarker discovery in advancing the understanding and management of OSA.

Neuroinflammation-Targeted Therapy

Targeted therapeutic approaches that modulate neuroinflammation and microglial activity hold promise for improving cognitive function and mitigating long-term neurological complications in OSA. Ongoing research into neuroinflammation-targeted interventions seeks to address the underlying biological mechanisms, offering potential strategies to complement existing OSA treatments and reduce its neurocognitive burden. IH has been shown to elevate inflammatory markers and induce cognitive impairment.[79] Certain interventions can counteract these effects; for instance, melatonin reduces pro-inflammatory cytokines and enhances synaptic plasticity, thereby improving hypoxia-related cognitive deficits,[80] while the glucagon-like peptide-1 receptor (GLP-1R) agonist liraglutide alleviates cognitive dysfunction by activating the nuclear factor erythroid 2-related factor 2 (Nrf2)/ heme oxygenase-1 (HO-1) pathway and inhibiting mitogen-activated protein kinase (MAPK)/NF-κB signaling.[81] These findings suggest that neuroinflammation-targeted strategies may serve as valuable adjuncts to conventional treatments such as continuous positive airway pressure, potentially reducing the neurocognitive burden of OSA.[82]

Limitations

The analysis of inflammation in OSA presents several notable advantages. First, it is a groundbreaking attempt to systematically examine the research landscape of inflammation in OSA through bibliometric methods, facilitating a quantitative assessment of the field's development. The study's extensive coverage of OSA literature offers a well-rounded view of the subject. By utilizing various bibliometric tools, it delivers an in-depth understanding of the research landscape, highlighting key contributors, thematic trends, and existing gaps. The incorporation of software such as VOSviewer and CiteSpace bolsters the objectivity and reproducibility of the analysis, establishing a solid foundation for guiding future research.

Nevertheless, the study has limitations that should be recognized. A major concern is the potential bias from the choice of specific databases. Solely relying on sources like WoSCC may result in the omission of relevant studies indexed in other databases such as PubMed, Scopus, or the Cochrane Library. Additionally, the emphasis on English-language publications might lead to an underrepresentation of research conducted in other languages, distorting the understanding of the global OSA research landscape. Data consistency issues, including discrepancies in author names or institutional affiliations, could also compromise the accuracy of the analysis. Lastly, the coverage may not encompass the latest research due to the delay between data collection and publication.

Conclusion

Our study highlights the remarkable progress that has been made in understanding inflammation in OSA, underscoring the importance of international collaboration, the contributions of leading institutions and scholars, the influence of high-impact journals, and the evolution of research priorities. Future work should focus on elucidating the molecular mechanisms underlying OSA, improving the management of comorbidities, and developing novel therapeutic strategies. These advances will require sustained interdisciplinary collaboration among clinicians in sleep medicine, cardiology, and neurology, together with basic scientists, public health professionals, and data scientists. Equally crucial is the effective translation of research findings into clinical practice to enhance diagnostic approaches, refine therapeutic interventions, and ultimately improve long-term outcomes for patients with OSA.

Abbreviations

OSA, Obstructive Sleep Apnea; WoSCC, Web of Science Core Collection; INSERM, Institut National De La Sante Et De La Recherche Medicale; CPAP, Continuous Positive Airway Pressure; OSAS, Obstructive Sleep Apnea Syndrome; TNF-α,

Tumor Necrosis Factor-alpha; IL-6, Interleukin-6; CRP, C-reactive protein; TI, Title; AB, Abstract; AK, Author Keywords; OALM, Online Analysis Platform of Literature Metrology; NC, Number of Citations; NP, Number of Publications; SCP, Single-country Publications; MCP, Multiple-country Publications;; IH, Intermittent Hypoxia; NF-κB, Nuclear Factor Kappa-B; IL-8, Interleukin-8; BBB, Blood-brain Barrier; CNS, Central Nervous System; CIH, Chronic Intermittent Hypoxia; AD, Alzheimer's Disease; T2DM, Type 2 Diabetes Mellitus; CVD, Cardiovascular Disease; COPD, Chronic Obstructive Pulmonary Disease; AHI, Apnea-hypopnea Index; ncRNAs, Non-coding RNAs; MMP9, Matrix Metalloproteinase-9; ox-LDL, Oxidized Low-Density Lipoprotein; GLP-1R, Glucagon-Like Peptide-1 Receptor; Nrf2, Nuclear Factor Erythroid 2-Related Factor 2; HO-1, Heme Oxygenase-1; MAPK, Mitogen-Activated Protein Kinase.

Data Sharing Statement

The request for original data that support this paper's conclusions can be directed to the corresponding authors.

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Author Contributions

Haixia Fan: Writing – Review & Editing, Writing – Original Draft Preparation, Conceptualization, Supervision, Formal Analysis. Huiyan Niu: Writing – Review & Editing, Writing – Original Draft Preparation, Conceptualization, Formal Analysis. Bomeng Zhao: Writing – Review & Editing, Writing – Original Draft Preparation, Formal Analysis. Xiaoling Gao: Writing – Review & Editing, Conceptualization, Data curation, Methodology, Funding Acquisition, Supervision, Project Administration. All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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