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Status and Trends in Glioblastoma Lipid Metabolism Research: A Bibliometric Analysis

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Abstract

Glioblastoma multiforme (GBM), which is the most lethal brain tumor, exhibits high aggressiveness, recurrence, and poor treatment response. The progression of GBM and resistance to therapy is attenuated by metabolic reprogramming and lipid metabolism. In this study, we conducted a bibliometric analysis of GBM lipid metabolism studies starting from the mid-1990s based on 798 articles from Web of Science Core Collection. In particular, publication trends, geographic and institutional distribution, authorship networks, keyword co-occurrence, and citation bursts, using CiteSpace, VOSviewer, and R-bibliometrix, were examined. Studies published in the US and China increased substantially after 2010. *De novo* lipogenesis, lipid droplet biology, and treatment targeting of lipid regulatory processes, such as fatty-acid oxidation, were the most frequently studied topics. Recent interest in lipid-immune interactions, ferroptosis, and lipomics suggests that further research is required. This bibliometric overview provides a solid foundation for future studies of GBM lipid metabolism and its clinical translation.

1. Introduction

Glioblastoma multiforme (GBM) is the most commonly occurring primary malignant brain tumor, and accounts for 13.7% of all primary brain and other CNS tumors and 52.2% of malignant tumors (all ages, U.S.) based on the 2024 CBTRUS reports [1,2]. It is characterized by microvascular proliferation and/or necrosis. WHO CNS5 defines it as “glioblastoma, IDH-wildtype, CNS WHO grade 4,” which is an IDH-wildtype and H3-wildtype diffuse astrocytic glioma, thus distinguishing it from other grade 4 astrocytoma-spectrum tumors [3-6]. Despite considerable progress in surgical resection, radiotherapy, and chemotherapy, particularly with temozolomide, the median overall survival for patients remains poor, ranging from 14 to 16 months [3,7,8]. This poor prognosis primarily results from diffuse infiltration, resistance to standard treatment, marked intratumoral heterogeneity, and extensive metabolic reprogramming [9,10].

Metabolic reprogramming is a well-known feature of cancer. It supports tumors to meet the high bioenergetic and biosynthetic demands required by uncontrolled growth. The Warburg effect and glucose metabolism have long been a topic of interest, and lipid metabolism has recently become a relevant, but well-studied area of GBM [11]. Fernández-García et al. exploited the unique lipid metabolic dependencies of pediatric brain tumors for biomarker discovery. Targeted intervention holds greater promise compared with adapting conventional cancer therapies [12]. Lipids are essential for membrane production, energy storage, signaling, and cell fate decisions [13]. In the unique microenvironment of the central nervous system, GBM cells are highly adaptable and regulate lipid synthesis, uptake, oxidation, and storage to promote tumor growth [14].

Recent studies have examined metabolic pathways, such as de novo fatty-acid synthesis, fatty-acid oxidation (FAO), lipid droplets, cholesterol homeostasis, and lipase signaling [15-20]. These are regulated by transcriptional factors, such as sterol regulatory element-binding proteins, peroxisome proliferator-activated receptors, and hypoxia-inducible factors. Enzymes, such as fatty-acid synthase (FASN), acetyl-CoA carboxylase (ACC), diacylglycerol O-acyltransferase 1 (DGAT1), and sterol O-acyltransferase 1 (SOAT1), enable GBM to survive in nutrient-restricted hypoxic environments [11,17,21,22]. These lipid modifications also enhance membrane biogenesis and energy storage.

Lipid metabolism is also important in immune resistance and tumor invasion [23]. Recent studies have also emphasized the connection between lipid metabolism and the immune microenvironment [24]. Lipid-rich tumor-

associated macrophages, dysregulated arachidonic acid metabolism, and altered lipid antigen presentation have been observed [25,26]. Moreover, lipid peroxidation and resistance to ferroptosis (a regulated iron-dependent cell death) are of interest in understanding treatment failure in GBM [14]. Therefore, lipids are both indicators of aggressive tumor activity and potential modulators of the immune response and therapy.

Although a mechanistic understanding of lipid metabolism in glioblastoma has progressed, a comprehensive picture of the research trends, collaborations, and future areas is not available. Bibliometric analysis provides a method to quantitatively analyze the development of a scientific research field over time. The analysis of publication trends, citation patterns, co-authorship patterns, and keyword evolution can detect influential authors, research hotspots, and knowledge gaps [27].

In this study, we present a bibliometric and qualitative description of GBM lipid metabolism research spanning nearly three decades. We applied data collected from the Web of Science Core Collection and used CiteSpace, VOSviewer, and R to quantify the intellectual structure and the dynamics of this research field. Our results may guide future studies by highlighting emerging ideas, translational challenges, and open challenges in glioblastoma lipid metabolism.

2. Materials and Methods

2.1 Data Source and Search Strategy

Bibliographic data were retrieved from the Web of Science Core Collection (WoSCC), which is a highly regarded and widely used database for bibliometric research because of its curated indexing and standardized metadata. For each study, we extracted bibliographic and analytical information, including the title, authors, author affiliations, publication year, journal, document type, abstract, keywords, cited references, total citation counts, and country/region information. The search was performed on July 31, 2025, and included publications from January 1, 1996, to July 31, 2025, to capture a complete longitudinal perspective of research in this field.

Lipid metabolism-related terms were selected to ensure comprehensive coverage of key lipid metabolic processes associated with glioblastoma biology, including lipid synthesis, FAO, lipid storage, and lipid signaling pathways, and encompassing both classical and emerging research themes. The query used canonical pathway/process terms to ensure broad coverage

of lipid metabolism mechanisms in GBM, while avoiding narrow, intervention-specific phrasing that is inconsistently indexed among publications. The search strategy was formulated using topic terms (TS) to ensure the inclusion of the relevant literature at the intersection of glioblastoma and lipid metabolism. The final search query was as follows:

TS = ((glioblastoma* OR glioblastoma multiform OR glioblastoma multiforme OR grade IV glioma OR GBM) AND ((lipid metabolism) OR (fatty acid metabolism) OR (cholesterol metabolism) OR (lipid droplet*) OR phospholipid metabolism) OR (sphingolipid metabolism) OR (lipogenesis) OR (lipolysis) OR (fatty acid oxidation) OR (FAO) OR (acetyl-CoA))

Only English-language publications were included. Eligible document types were limited to original research articles and reviews. Editorials, meeting abstracts, letters, book chapters, and other non-peer-reviewed formats were excluded.

2.2 Inclusion and Exclusion Criteria

Studies were included if they were focused on lipid metabolism in glioblastoma, whether through a mechanistic, translational, preclinical, or clinical perspective. Articles that discussed tumor metabolism more broadly, without specifically focusing on lipid-related pathways, were excluded.

2.3 Bibliometric Tools and Analytical Framework

Three validated software tools were used to conduct complementary layers of bibliometric analysis: CiteSpace (version 6.1.R6) was used for co-citation network analysis, citation burst detection, and cluster mapping to identify emerging research frontiers and thematic evolution [28]. VOSviewer (version 1.6.19, <https://www.vosviewer.com>) facilitated the construction of visual networks illustrating author collaboration, institutional productivity, country-level contributions, and keyword co-occurrence [29]. The Bibliometrix R-package (version 4.1.2, <https://www.bibliometrix.org/>), operating within the R statistical environment, was used for the descriptive analytics of annual publication trends, prolific journals, author productivity, and source impact [30]. Figures were generated using ggplot2 for data visualization. The workflow of the bibliometric analysis is shown in Figure 1.

3. Results

3.1 Annual Scientific Output

Following manual screening and validation by two independent reviewers, 798 publications were retrieved, which consisted of 635 original research articles and 163 review articles. The full list of articles considered for inclusion is available in Supplementary File S1. The temporal trajectory of publications on glioblastoma lipid metabolism revealed an upward trend beginning in 1997. The initial period (1997–2009) was characterized by sporadic, low-volume output, which is indicative of nascent interest in lipid metabolic pathways in GBM. Around 2010, the annual number of publications began to steadily increase, paralleling the recognition of cancer metabolism as a therapeutic target. From 2017 onward, research activity markedly accelerated, reaching a peak in 2022 and 2023. This growth reflects a robust and expanding scientific community dedicated to uncovering the metabolic basis of GBM (Figure 2).

3.2 Country and Institutional Contributions

According to the contributing countries, the United States continues to provide important work in glioblastoma lipid metabolism. China has published a substantial amount in recent years, which has contributed to its leading position. Other contributing countries include Germany, the United Kingdom, and Italy.

As shown in Figure 3A, the global collaboration map depicts a large co-authorship between the United States and other European countries because of strong transatlantic collaborations; however, Figure 3B shows that although China published more frequently than Western countries in the Asian-Pacific region, the research collaborations are more concentrated in Asia-Pacific countries, and lack international relationships when compared to Western countries. Interestingly, the overlay suggests that publications from China and several Central/South American countries have a later average publication year, indicating that their contributions are more concentrated in recent years, consistent with a late growth phase in this subfield.

At the institutional level (Figure 4), major academic research centers, such as the University of California, Los Angeles, Ohio State University, Northwestern University, and MD Anderson Cancer Center, have formed strong collaborations. In China, Capital Medical University, Zhejiang University, and Sun Yat-sen University form tightly-knit clusters, which suggests that domestic research collaborations play active roles. These patterns of institutional collaboration highlight the growing structural

complexity and international research interest in glioblastoma lipid metabolism.

3.3 Journal and Citation Analysis

Cancers (MDPI), Biomedicines, Cell Metabolism, Nature Reviews Cancer, and Cancer Research have emerged as prominent publications for studies on glioblastoma lipid metabolism. These journals differ in their thematic focus. For example, Cancers and Biomedicines frequently publish both clinical and translational research, whereas Cell Metabolism and Cancer Research tend to emphasize molecular and biochemical studies.

As shown by a journal coupling analysis (Figure 5A), publication venues were grouped into clusters based on shared citation patterns, suggesting disciplinary convergence among oncology, metabolism, and molecular biology. The co-citation map (Figure 5B) reveals the central role of high-impact journals, such as Nature, Cell, Journal of Biological Chemistry, and Cancer Cell, in shaping the intellectual landscape. These patterns reflect a multidisciplinary foundation and highlight the growing integration of metabolic research into neuro-oncology literature.

3.4 Overlay Visualization of Disciplinary Structure

Figure 6 provides an overlay analysis showing the disciplinary and thematic structure of lipid metabolism studies in glioblastoma. The map integrates citation flows and topic evolution among the Web of Science subject categories. The field is anchored primarily in molecular biology, biochemistry, oncology, and immunology, as indicated by the dense clustering and strong linkages in these areas. These foundational disciplines serve as central hubs for cross-disciplinary integration.

The most prominent citation pathways were observed in molecular and cellular biology toward clinical medicine, suggesting a translational trajectory from mechanistic insights to applied therapeutic studies. Additional overlays extend into pharmacology, neuroscience, and bioengineering, which reflect the adoption of advanced tools, such as lipidomics, nanotechnology, and systems biology.

Emerging interdisciplinary linkages connect the core field to health sciences, environmental toxicology, and computational biology, thus revealing the expanding influence of glioblastoma lipid metabolism research into adjacent biomedical domains. This underscores the dynamic and integrative nature of

the field, from its evolution as a molecular niche to a broad-based, translational research area.

3.5 Author Analysis

The co-authorship network highlights influential researchers, such as Deliang Guo, whose contributions to the sterol regulatory element-binding protein 1 (SREBP1) and Sterol O-Acyltransferase 1 (SOAT1) pathways have provided a foundation for further mechanistic studies. Other key figures include Yongjun Kou and Zsolt Balogi, who have examined lipid droplet dynamics and lipidomic alterations in GBM tissues. A collaborative analysis revealed that most productive author clusters are present within institutional networks, suggesting that dedicated research groups drive much of the innovation in this field (Figure 7).

Co-authorship Network Analysis (Figure 7A)

A co-authorship analysis revealed prominent collaborative relationships among researchers in the field of GBM lipid metabolism. Two major clusters emerged. The first was centered around Guo Deliang, Geng Feng, and Chakravarti Arnab, representing a highly interconnected group with frequent collaborations, particularly among researchers based in the United States and China. The second cluster was led by Cloughesy Timothy F., Cavenee Webster K., and Yong William H., and reflects a cohesive research team focusing on translational and clinical GBM studies. The strong intra-cluster links and several inter-cluster connections suggest a moderately collaborative global research environment, with cross-institutional and cross-national cooperation shaping the progress in this field.

Co-citation Network Analysis (Figure 7B)

The co-citation analysis highlights the intellectual structure of GBM lipid metabolism studies. Guo D.L., Stupp R., Louis D.N., DeBerardinis R.J., Warburg O., and Ostrom Q.T. rank among the most highly co-cited and most strongly connected authors, indicating their influence in shaping our current understanding of lipid metabolism in GBM. Notably, Stupp R. is strongly associated with clinical advances in standard-of-care treatment, whereas DeBerardinis R.J. and Guo D.L. have contributed significantly to the elucidation of metabolic reprogramming in gliomas. The interconnections among these highly cited authors suggest a shared reliance on landmark studies and an emerging consensus on mechanistic and therapeutic targets within the field.

3.6 Reference Co-Citation Analysis

Figure 8 presents a reference co-citation network, highlighting the foundational literature in the GBM lipid metabolism field. Notably, Stupp et al. (2005, *New Engl J Med*) is the most frequently co-cited article, reflecting its pivotal role in establishing the standard of care for GBM. Other highly co-cited studies include Louis et al. (2016, *Acta Neuropathologica*), which examines the molecular classification of gliomas, and Hanahan and Weinberg (2011, *Cell*), which examines hallmark cancer biology. Metabolism-focused articles, such as Heiden et al. (2009, *Science*) and Guo D.L. (2009, *Sci Signal*; 2011, *Cancer Discovery*), also feature prominently, thus highlighting the growing recognition of metabolic reprogramming in glioma pathogenesis. The dense clustering and cross-linking among references indicate a strong thematic coherence among molecular pathology, metabolism, and clinical therapeutics. Taken together, these citations represent the intellectual backbone of current GBM lipid metabolism research.

3.7 Citation Bursts and Knowledge Evolution

Figure 9 shows the top 15 references with the strongest citation bursts from 1997 to 2025, which reflect influential studies that garnered rapid attention during specific periods. Of these, Louis et al. (2021, *Neuro-Oncology*) had the highest burst strength (13.85), followed by Lin et al. (2017) and Heiden et al. (2009, *Science*), indicating their contributions to shaping the research trends in GBM lipid metabolism. Recent bursts (2022–2025) are dominated by publications, such as Cheng et al. (2020, *Cell Metab*), Miska et al. (2023, *J Clin Invest*), and Kou et al. (2022, *Biomedicines*), which demonstrate a growing emphasis on metabolic regulation and lipid biology in glioma research. Earlier high-impact studies, including Parsons et al. (2008, *Science*) and Yan et al. (2009, *NEJM*), provide a molecular foundation for glioma classification and mutation profiling.

3.8 Keyword Co-occurrence and Thematic Clustering

Keyword co-occurrence analysis revealed several interconnected thematic clusters in glioblastoma lipid metabolism research (Figure 10A). Cluster 1 was anchored by terms, such as glioblastoma, temozolomide, and apoptosis, which reflect foundational interest in GBM pathogenesis and response to chemotherapy. Cluster 2 highlighted metabolic themes, including lipid droplets, metabolic reprogramming, ceramide, and mitochondria, which indicate a focus on altered lipid metabolism and organelle function in glioma. Cluster 3 featured tumor microenvironment, angiogenesis, Warburg effect,

and hypoxia, representing studies on tumor ecology and metabolic adaptation. Cluster 4 included broader metabolic and disease-related terms, such as cancer metabolism, glycolysis, and oxidative stress. Temporal trend analysis (Figure 10B) revealed the recent interest in nanoparticles, temozolomide, glioblastoma, and mitochondria, indicating evolving research aimed at nanotechnology-based delivery and mitochondrial targeting. In a GBM-restricted corpus, the trend-topic analysis was dominated by pathology/therapy and study-design terms (e.g., glioblastoma, temozolomide, nanoparticles, *in vivo/in vitro*), indicating that lipid metabolism research revolves around tumor biology and translational endpoints, rather than lipid biochemistry alone. After filtering generic disease/method terms, lipid-focused signals (e.g., choline, phospholipid metabolism, fatty-acid synthase, mitochondria) remained prominent, which provide a clearer view of lipid-metabolism-specific research trends. A keyword word cloud summarizing term frequency in the dataset is provided in Supplementary Figure S1.

4. Discussion

GBM is one of the most aggressive and lethal forms of brain cancer, with a disproportionately high burden of cancer-related mortality [2,31,32]. Its resilience stems not only from its invasive nature, but also from its remarkable metabolic plasticity [33]. Our bibliometric and scientific analysis delineated the evolution and prospective trajectory of lipid metabolism studies in glioblastoma. In addition to highlighting the significance of lipid metabolism in GBM biology, the results indicated a shift of this research area from a specialized niche to a prominent focus within neuro-oncology. This progression is significant given the context of GBM as one of the most aggressive and lethal brain tumors, hallmarked by its invasiveness and notable metabolic plasticity [34].

Since approximately 2017, there has been a notable increase in publications, likely attributable to the growing recognition of metabolic vulnerabilities in GBM and the advent of enabling technologies, such as lipidomics, single-cell profiling, and advanced imaging techniques that have facilitated lipidomics research. Conceptually, the field has progressed beyond a glucose-centric perspective of cancer metabolism and has revealed lipids as central regulators [35,36], further emphasizing lipid pathways as promising targets for therapeutic intervention [37-40].

Geographic and institutional patterns reveal a field that is characterized by global distribution alongside structural concentration. The United States

retains a leadership position, particularly as a central node for international collaboration, whereas China demonstrates rapid growth in research output with networks that are comparatively more regionally concentrated. This asymmetry suggests that future advancements may be enhanced through more extensive cross-regional integration. The clustering of institutions around major academic centers further suggests that GBM lipid metabolism studies are driven by specialized groups with a sustained focus. Although this may accelerate progress, it also poses the risk of thematic silos if there is limited exchange between groups.

Journal and co-citation analyses revealed that lipid metabolism in GBM is inherently multidisciplinary and positioned at the intersection of oncology, metabolism, and molecular biology. The availability of clinically oriented and mechanistic journals has provided a reciprocal exchange flow between laboratory research and clinical application.

Keyword co-occurrence and clustering analysis (Figure 10) indicated that the predominant themes continue to focus on general GBM biology, chemoresistance, and metabolic reprogramming. This indicates that lipid metabolism is primarily examined within the broader context of tumor adaptation, rather than as an independent field. Conversely, highly specialized topics, such as ferroptosis and immunometabolism, despite their biological potential, are less prominent in the bibliometric landscape, suggesting that they are emerging, rather than established themes.

The analysis of citation bursts (Figure 9) corroborates this interpretation. Seminal publications on glioma classification and core metabolic reprogramming represent the most pronounced citation bursts, highlighting the enduring influence of foundational oncogenic and metabolic paradigms in guiding lipid-centric research. Recent citation bursts pertaining to metabolic regulation and mitochondrial function suggest a progressive refinement toward studies that are organelle-specific and pathway-focused. This progression indicates a shift in the field from broad metabolic descriptions to a more mechanistic precision.

The integration of overlay visualization and keyword clustering indicates a translational trajectory, with citation flows transitioning from molecular biology to clinical medicine. The emergence of terms such as nanoparticles and mitochondria in the recent literature suggests a shift from descriptive metabolic studies to therapeutic applications. This trend is consistent with the ongoing challenge of drug delivery across the blood-brain barrier (BBB).

Strategies such as lipid nanoparticle carriers, focused ultrasound-mediated BBB disruption, and receptor-mediated transcytosis are being explored to improve central nervous system bioavailability [41]. Consequently, studies on lipid metabolism in GBM not only target metabolic pathways, but also explore delivery mechanisms for lipophilic or lipid-based interventions, which are otherwise constrained by pharmacokinetic challenges.

At the biological level, the literature reviewed here indicates that GBM relies on metabolic plasticity rather than a singular dominant lipid pathway. The SREBP1-driven *de novo* lipogenesis pathway is consistently highlighted, with downstream enzymes, such as FASN and ACC, playing important roles in membrane biogenesis and signal transduction [15]. Preclinical studies indicate that the pharmacological inhibition of SREBP1 or its downstream effectors may disrupt glioma stem cell maintenance and enhance sensitivity to therapy, thus demonstrating the translational significance of this pathway [11].

FAO, particularly under hypoxic conditions, plays a complementary role in supporting adenosine triphosphate (ATP) production and indirectly contributes to maintaining redox balance through pathways associated with nicotinamide adenine dinucleotide phosphate NADPH homeostasis [42]. Carnitine palmitoyltransferase 1 (CPT1) has been identified as an important regulatory node in this process [43]. Massalha et al. reported that Minerval induces cancer-selective toxicity in glioblastoma by uncoupling mitochondrial oxidative phosphorylation and exploiting tumor fatty-acid dependence [44]. In the literature, the roles of lipogenesis and FAO in GBM cells currently include anabolic and catabolic lipid pathways [11], suggesting that therapeutic strategies targeting various metabolic states may be more effective than focusing on a single pathway.

Another prominent theme is the accumulation of lipid droplets, which buffer excess fatty acids and enable metabolic flexibility during stress [45], whereas lipophagy facilitates rapid lipid mobilization [46,47]. Regulators, such as adipose triglyceride lipase and perilipin-2 (PLIN2), are considered potential therapeutic targets [48,49], thereby linking organelle biology to therapeutic sensitivity. For example, Lladó et al. found that pharmacologically remodeling glioma cell membrane lipid composition suppresses oncogenic signaling, which supports lipids as therapeutic targets [50].

In addition to well-established regulators, such as FASN and SREBP1, new therapeutic targets have been identified, such as stearoyl-CoA desaturase-1

(SCD1), lysophosphatidylcholine acyltransferase (LPCAT), and those involved in cardiolipin remodeling, which play important roles in modulating membrane fluidity, maintaining mitochondrial integrity, and regulating apoptosis. These relatively unexplored components present novel therapeutic opportunities [51].

Despite these findings, bibliometric analysis indicates that the translation of mechanistic discoveries into clinical applications remains insufficient. Although lipid-targeted strategies, such as inhibiting SREBP1-driven lipogenesis, carnitine palmitoyltransferase 1 (CPT1)-mediated FAO, or the induction of ferroptosis, are being evaluated in experimental settings, they are underrepresented in high-impact or frequently occurring keyword domains. This discrepancy indicates a translational gap between experimental potential and clinical implementation.

Ferroptosis, which is a non-apoptotic form of cell death characterized by iron-dependent lipid peroxidation, has gained prominence as a significant area of research, particularly concerning treatment-resistant, stem-like GBM cells [52]. Key regulators, including ACSL4, GPX4, and FSP1, are potential therapeutic targets [53]. Although ferroptosis is not yet a predominant term in bibliometric analyses, its mechanistic association with lipid peroxidation and therapy resistance highlights its importance as a significant emerging field [15]. Its relatively modest bibliometric presence likely indicates that it is an emerging, rather than a fully developed research theme.

The endocannabinoid system, which includes bioactive lipids such as anandamide and 2-arachidonoylglycerol, also plays a role in GBM lipid metabolism [54]. These lipid-derived signals attenuate glioma proliferation, immune cell polarization, and inflammatory responses through CB1 and CB2 receptors. Despite being relatively underexplored, this pathway presents significant translational potential, particularly in modulating tumor-immune interactions [55].

Immune-related aspects of lipid metabolism were also less prominently represented in the keyword trends, despite their strong biological rationale. This suggests that the study of immunometabolism in GBM remains at a relatively early stage of investigation, despite its considerable translational potential. Tumor-associated macrophages and microglia can accumulate lipids and adopt an immunosuppressive M2-like phenotype [24]. Lipid mediators, such as prostaglandins, oxidized phospholipids, and endocannabinoids, stabilize PD-L1, impair antigen presentation, and induce

T-cell exhaustion. These mechanisms, in aggregate, may hinder the effectiveness of immunotherapy, thereby positioning immunometabolism as an important area of research [56].

4.1 Limitations and Future Directions

Although this study has notable strengths, several limitations should be acknowledged. The exclusive reliance on the WoSCC may have resulted in the omission of pertinent studies contained in other databases. In addition, bibliometric indicators are susceptible to a citation lag, potentially leading to an underrepresentation of recent influential work. Keyword-based analyses are dependent on author-defined terms, which may introduce bias. Moreover, because bibliometric term extraction is frequency/co-occurrence driven, less standardized intervention phrases (e.g., “lipid therapy/replacement” or “membrane composition”) may not emerge as dominant topic labels, even when mechanistically relevant. Finally, our reliance on English-language publications and selected databases may introduce selection bias, as non-English studies, although few, were excluded and tend to receive fewer citations.

To address these challenges, future bibliometric analyses should incorporate datasets from PubMed, Scopus, and Embase. The integration of machine learning tools for semantic analysis can also assist in identifying latent thematic structures. Longitudinal tracking of co-citation networks may facilitate the prediction of emerging research hotspots. From a translational standpoint, further studies are needed to validate lipid-targeted therapies in preclinical and clinical settings. The use of patient-derived xenografts, 3D organoids, and immune-competent models will be important for translating laboratory findings into clinical applications. Furthermore, the identification of reliable biomarkers for patient stratification is an essential area of study.

4.2 Implications for Research and Practice

Our review supports lipid metabolism as a pivotal element in the biology of GBM and its resistance to therapy. Incorporating lipidomic profiling into conventional molecular diagnostics may reveal metabolic dependencies that can be targeted, particularly in recurrent or treatment-resistant disease. Policymakers and funding agencies should consider prioritizing interdisciplinary initiatives that connect lipid biology, pharmacology, and neuro-oncology.

The field has progressed from observational studies to a comprehensive discipline that integrates molecular biology, systems-level omics, and translational science. The effect of lipid-targeted strategies will depend on sustained interdisciplinary collaboration and technological advancements. By addressing mechanistic gaps, enhancing drug delivery, and stratifying patients, we may unlock the therapeutic potential of targeting lipid metabolism in glioblastoma.

5. Conclusion

This bibliometric study reveals the evolving research landscape of lipid metabolism in glioblastoma since the mid-1990s, with a marked increase after 2010 and a shift from descriptive metabolic profiling to mechanistic and early translational studies. Core research themes center on *de novo* lipogenesis, FAO, lipid droplet dynamics, immunometabolic regulation, and emerging hotspots, such as ferroptosis, endocannabinoid signaling, and lipid storage regulators. Despite strong preclinical evidence, clinical translation remains limited because of tumor heterogeneity, metabolic plasticity, and therapeutic delivery challenges. Future progress will likely depend on integrating lipidomics with spatial transcriptomics, immune profiling, and patient stratification, while underexplored areas, such as sex-specific lipid signatures and lipid-immune interactions, may reveal therapeutic opportunities. Overall, this analysis provides a structured overview of the current field and emerging directions in GBM lipid metabolism research, which will inform future studies and therapeutic advances in neuro-oncology.

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Conflicts of interest: The authors declare that the research was conducted in the absence of any commercial or financial relationships.

Authors Contribution: W.W. and Y.Y. conceived the study. W.W, Y.Y. conducted the literature search and analyses. Y.Y. and W.W. drafted the manuscript; M.K.S., P.A.B and S.N.B. provided critical revisions. R.S., F.X., and Y.C. prepared figures/tables. L.C. and W.W. supervised and approved the final version. All authors reviewed and approved the manuscript.

Declarations.

Ethical Approval: This research did not involve human subjects or personal data, using only published bibliometric records. Therefore, the ethical approval was not necessary.

Consent to participate: Not applicable. This study did not involve direct participation of any individuals.

Consent to publish: Not applicable. This study does not contain any individual person's data in any form.

Data Availability Statement: Not applicable.

Figures and Tables:

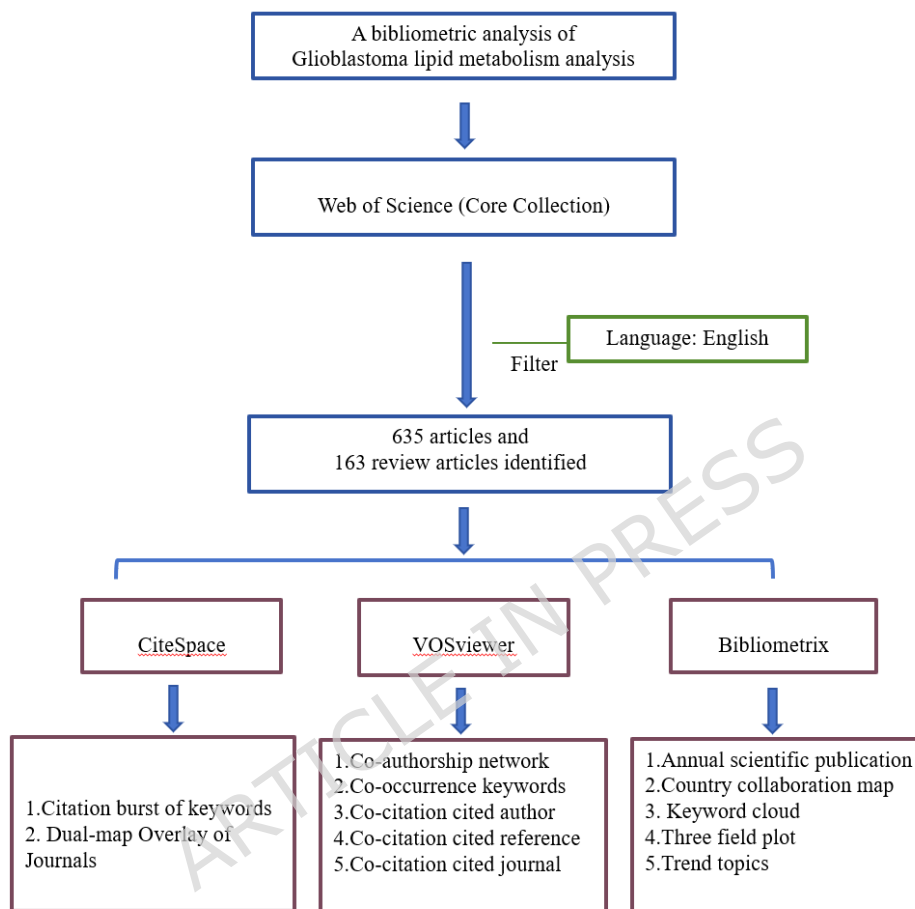


Figure 1. Workflow of the bibliometric analysis.

A schematic diagram outlining the study design and methodology. The process included literature retrieval from the Web of Science Core Collection (WoSCC), data cleaning and selection, and subsequent analysis using CiteSpace, VOSviewer, and R-bibliometrix.

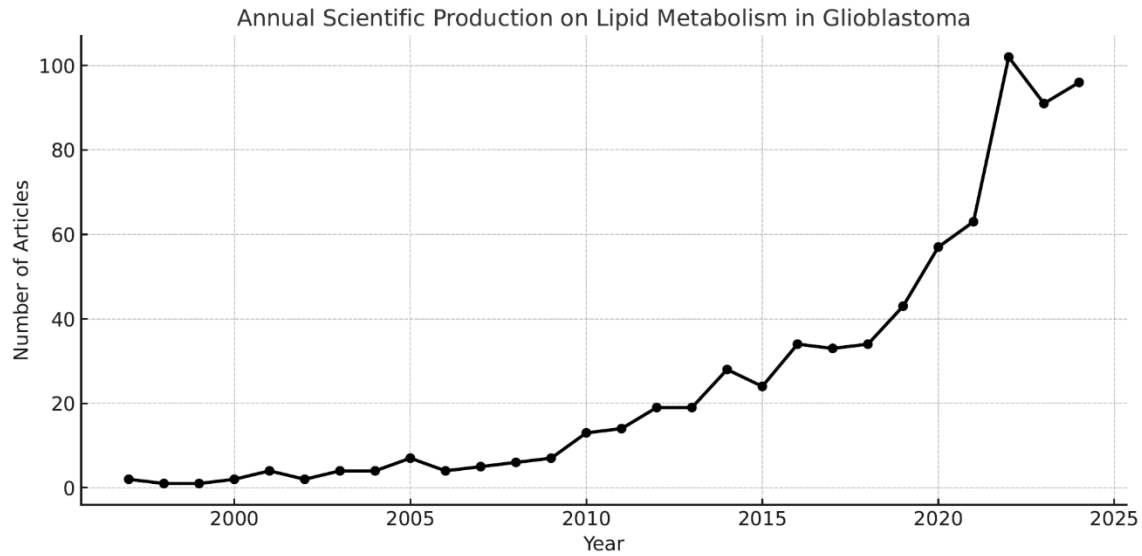
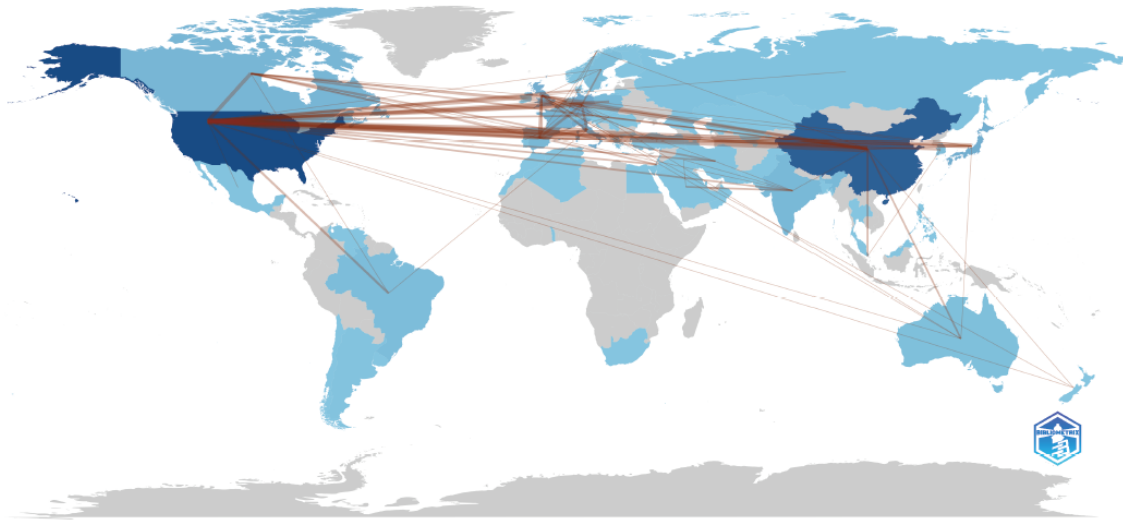


Figure 2. Annual publication trends on lipid metabolism in glioblastoma. The number of publications per year demonstrates a significant upward trajectory, particularly after 2010. The trend indicates the research attention on lipid-related mechanisms in GBM.

A



B

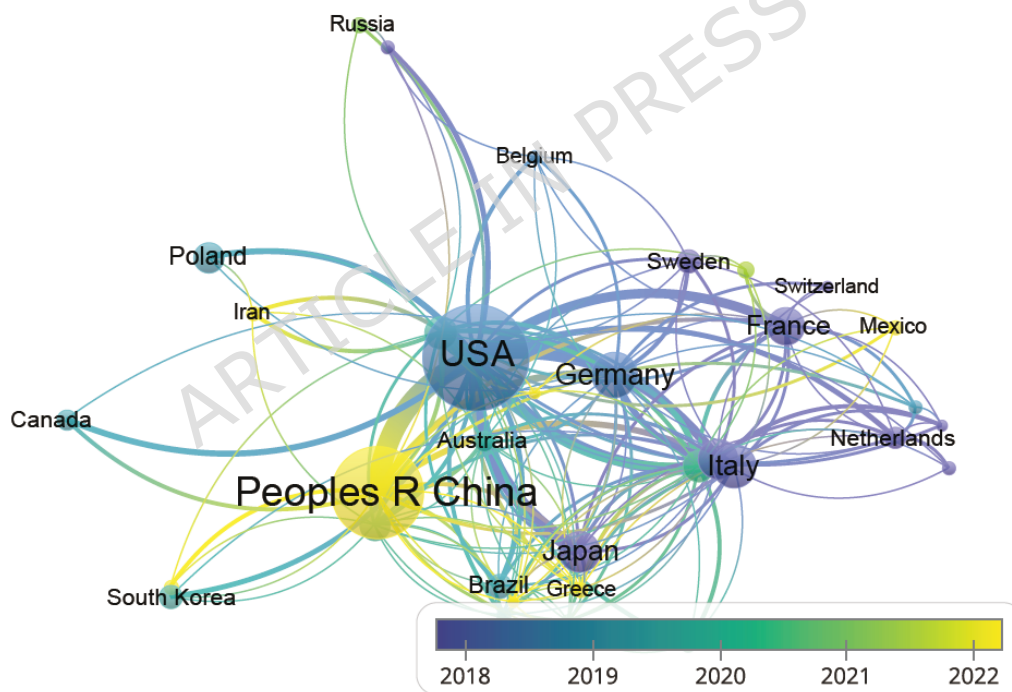


Figure 3. Global contributions and international collaboration network.

(A) Geographical distribution of publications. The United States and China are leading contributors, followed by Germany, the United Kingdom, and Italy. Node size represents publication count; line thickness reflects collaboration strength. (B) Co-authorship network map showing inter-country collaboration in GBM lipid metabolism research. Strong collaborative ties are evident between the United States, European nations, and East Asia.

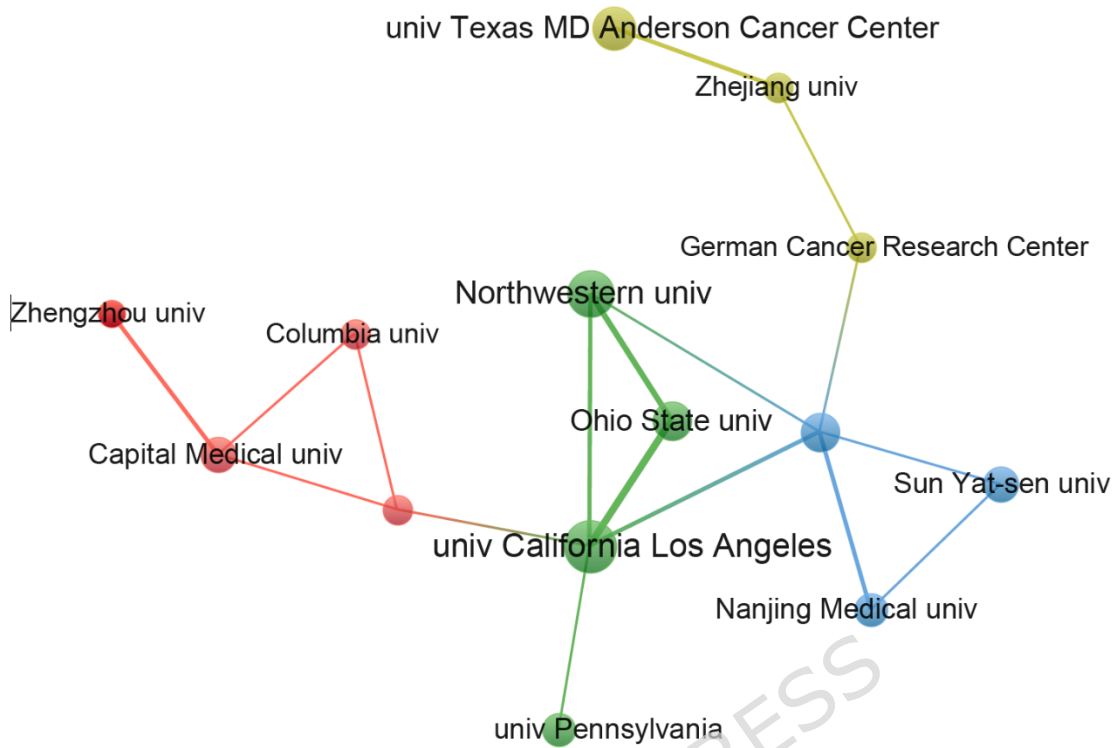
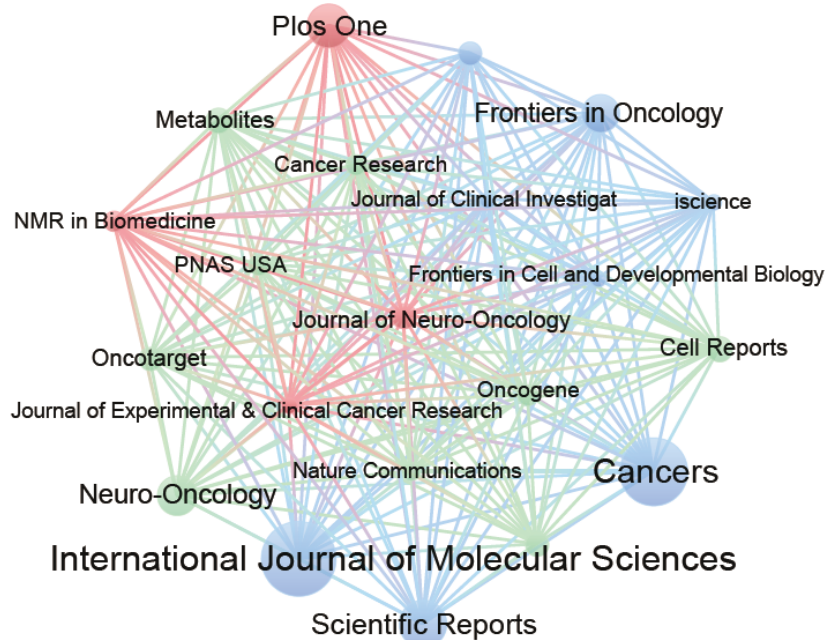


Figure 4. Institutional collaboration network.

Network visualization of top contributing institutions. The Ohio State University, Fudan University, and Pécs Medical School form key hubs in the collaborative landscape.

A



B

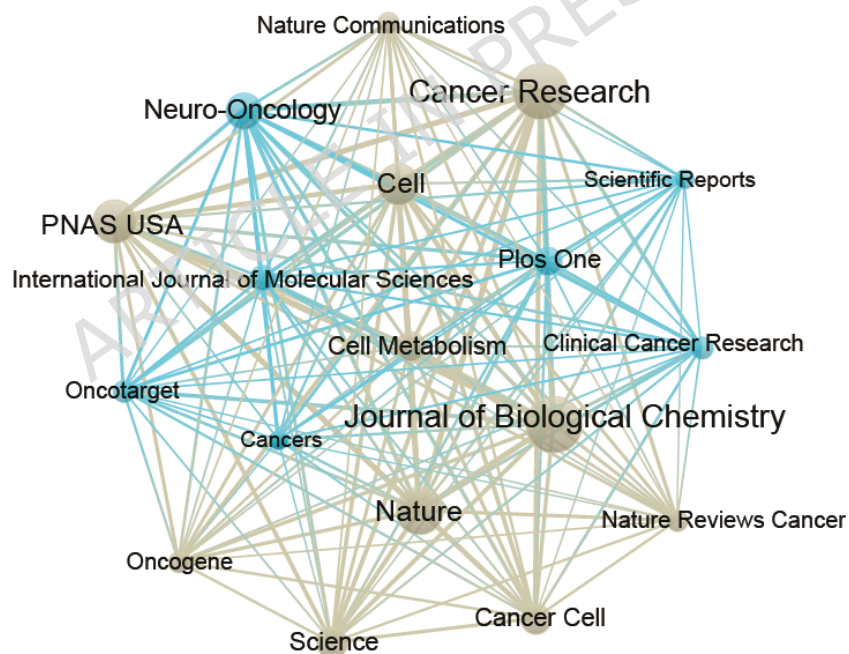


Figure 5. Journal analysis based on bibliographic coupling and co-citation.

(A) Bibliographic coupling map generated by VOSviewer showing clusters of journals with similar citation behavior. Node size reflects the total number of citations, and node proximity represents similarity in citation patterns.

(B) Co-citation network analysis identifying journals that are frequently cited together. Larger nodes indicate higher co-citation frequency.

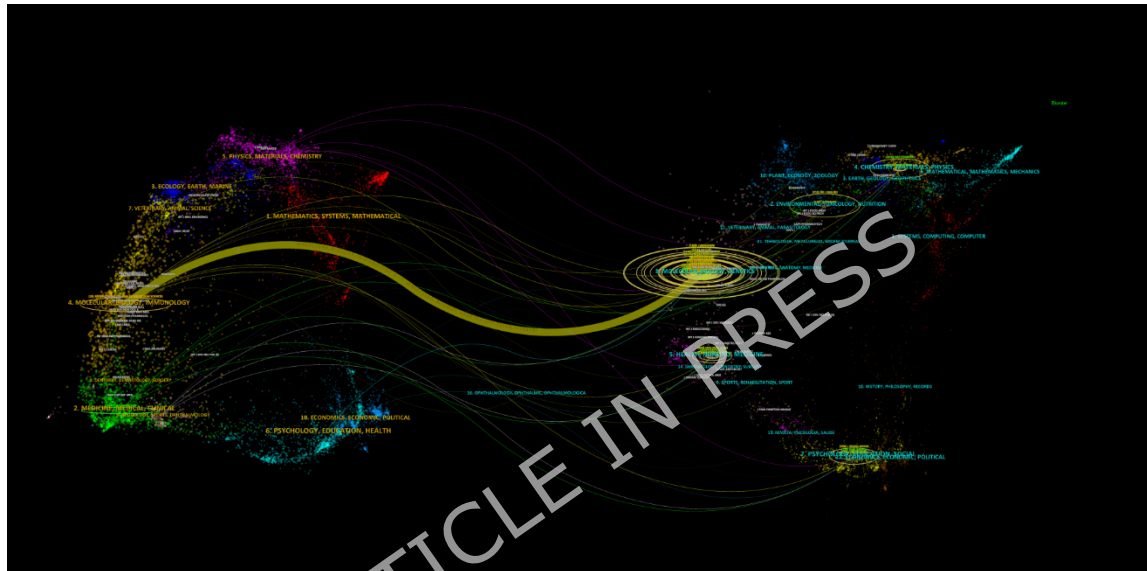
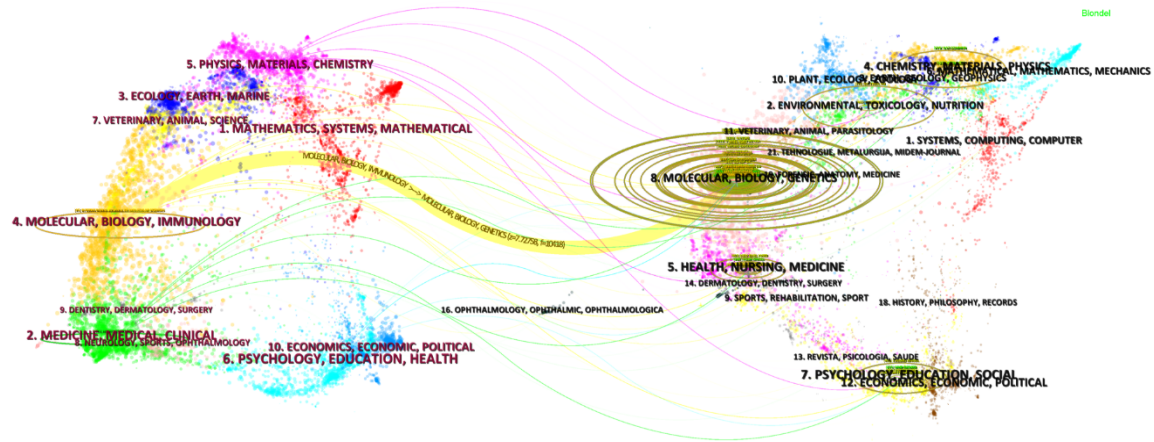
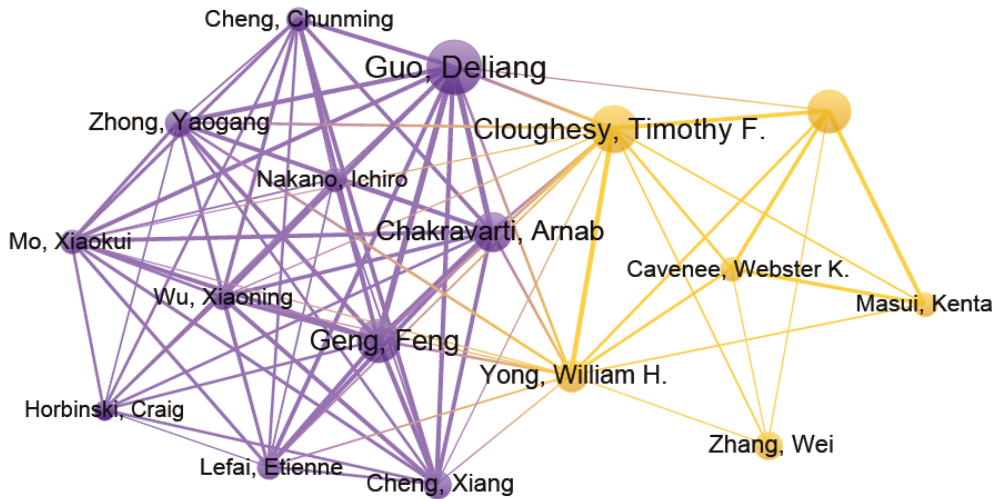


Figure 6. Dual-map overlay of citing and cited journals.

A global citation map showing disciplinary cross-talk. Arrows point from citing journals (left) to cited journals (right), with major flows between oncology, molecular biology, and neurology fields.

A



B

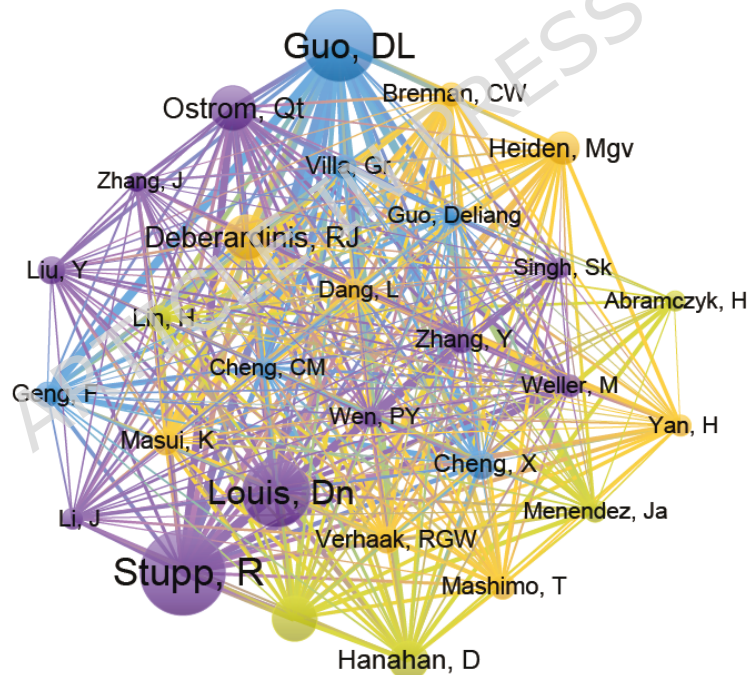


Figure 7. Author collaboration and co-citation networks.

(A) Graph showing top authors and their co-authorship patterns. Prominent contributors include Deliang Guo, Zsolt Balogi, and Yongjun Kou. Denser clusters represent close-knit research groups.

(B) Network of authors who are frequently co-cited, indicating shared thematic influence. Core nodes signify foundational figures in lipid metabolism and GBM.

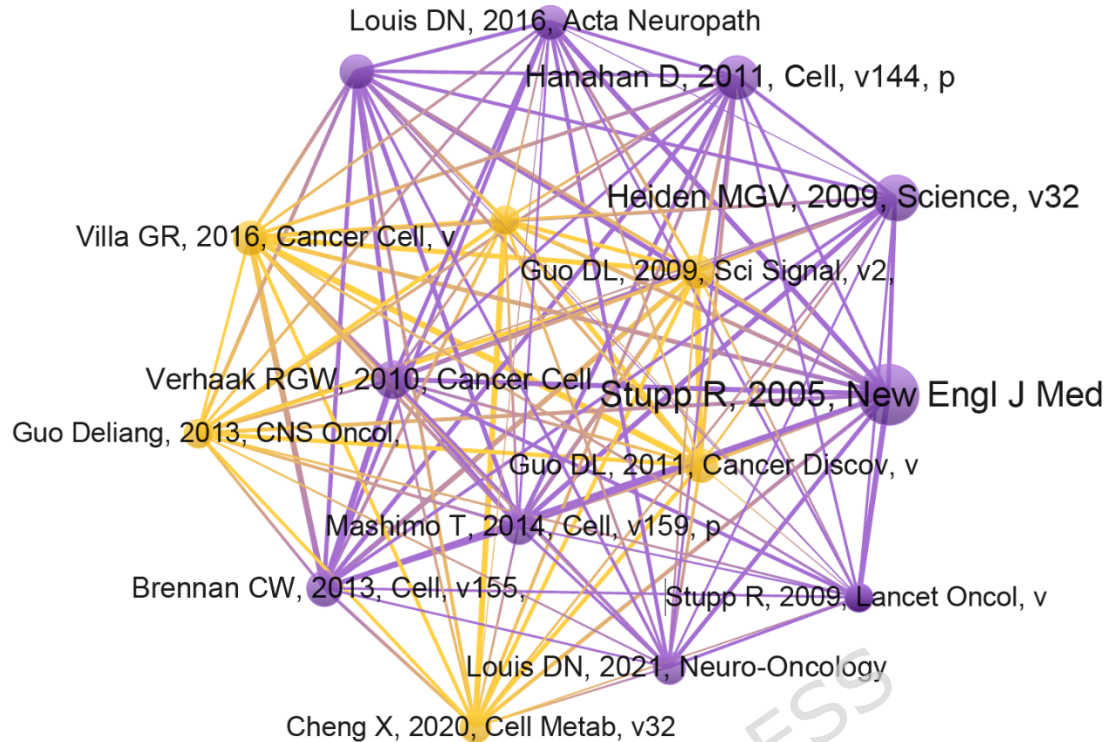


Figure 8. Co-citation network of references.

Visualization of highly co-cited references, grouped into distinct clusters representing shared thematic relevance. Each node represents a frequently cited publication, and the size of the node reflects its citation frequency. Edges indicate co-citation relationships.

Top 15 References with the Strongest Citation Bursts

References	Year	Strength	Begin	End	1997 - 2025
Heiden MG, 2009, SCIENCE, V324, P1029, DOI 10.1126/science.1160809, DOI	2009	12.79	2010	2014	
Parsons DW, 2008, SCIENCE, V321, P1307, DOI 10.1126/science.1164382, DOI	2008	9.83	2010	2013	
Yan H, 2009, NEW ENGL J MED, V360, P765, DOI 10.1056/NEJMoa0808710, DOI	2009	8.11	2010	2014	
Brennan CW, 2013, CELL, V155, P462, DOI 10.1016/j.cell.2013.09.034, DOI	2013	8.1	2014	2018	
Mashimo T, 2014, CELL, V159, P1603, DOI 10.1016/j.cell.2014.11.025, DOI	2014	8.95	2016	2019	
Louis DN, 2016, ACTA NEUROPATHOL, V131, P803, DOI 10.1007/s00401-016-1545-1, DOI	2016	11.15	2017	2021	
Lin H, 2017, NEURO-ONCOLOGY, V19, P43, DOI 10.1093/neuonc/nov128, DOI	2017	13.53	2018	2022	
Villa GR, 2016, CANCER CELL, V30, P683, DOI 10.1016/j.ccell.2016.09.008, DOI	2016	11.16	2018	2021	
Agnihotri S, 2016, NEURO-ONCOLOGY, V18, P160, DOI 10.1093/neuonc/nov125, DOI	2016	9.69	2018	2021	
Pavlova NN, 2016, CELL METAB, V23, P27, DOI 10.1016/j.cmet.2015.12.006, DOI	2016	8.29	2018	2021	
Taib B, 2019, SCI REP-UK, V9, P0, DOI 10.1038/s41598-019-55985-z, DOI	2019	9.54	2021	2025	
Louis DN, 2021, NEURO-ONCOLOGY, V23, P1231, DOI 10.1093/neuonc/noab106, DOI	2021	13.85	2022	2025	
Cheng X, 2020, CELL METAB, V32, P229, DOI 10.1016/j.cmet.2020.06.002, DOI	2020	10.07	2023	2025	
Miska J, 2023, J CLIN INVEST, V133, P0, DOI 10.1172/JCI163448, DOI	2023	8.04	2023	2025	
Kou YJ, 2022, BIOMEDICINES, V10, P0, DOI 10.3390/biomedicines10081943, DOI	2022	7.67	2023	2025	

Figure 9. Citation burst detection for top keywords and references.

Timeline of citation bursts showing emergent and declining research focuses.

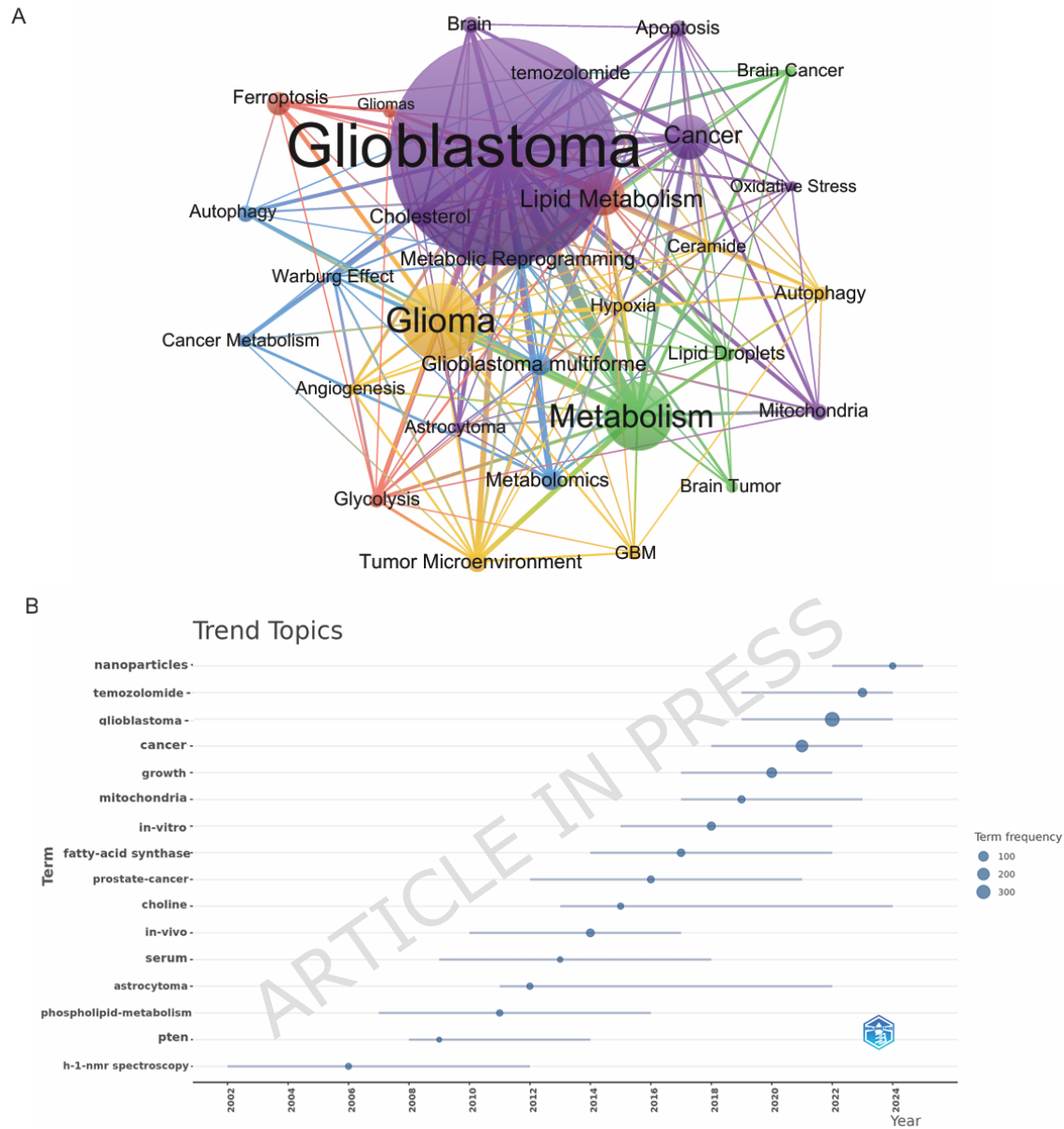


Figure 10. Author keyword co-occurrence network.

Clustering map of frequently used author keywords. Major themes include “lipogenesis,” “lipid droplet,” “fatty acid oxidation,” and “immune regulation,” reflecting conceptual structure in the field.

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