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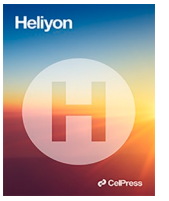

Review article

Knowledge mapping and emerging trends of ferroptosis in ischemia reperfusion injury research: A bibliometric analysis (2013–2022)

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ABSTRACT

Objective: Ischemia/reperfusion (I/R) injury is an inevitable dilemma when previously ischemic multiple organs and tissues are returned to a state of blood flow, with confirming a critical role of ferroptosis in molecular, pathway mechanisms, subcellular structure. Discovering the potential relationship may provide useful approaches for the clinical treatment and prognosis of the pathophysiological status of IRI. Therefore, a comprehensive visualization and scientometric analysis were conducted to systematically summarize and discuss the "ferroptosis in ischemia reperfusion injury" research to demonstrate directions for scholars in this field.

Methods: We retrieved all publications focusing on I/R injury and ferroptosis from the Web of Science Core Collection (WoSCC), published from 2013 to October 2022. Next, scientometric analysis of different items was performed using various bibliometrics softwares to explore the annual trends, countries/regions, institutions, journals, authors and their multi-dimensional relationship pointing to current hotspots and future advancement in this field.

Results: We included a total of 421 English articles in set timespan. The number of publications increased steadily annually. China produced the highest number of publications, followed by the United States. Most publications were from Central South University, followed by Sichuan University and Wuhan University. The most authoritative academic journal was Oxidative Medicine and Cellular Longevity. Cell occupied the first rank of co-cited journal list. Andreas Linkermann and Scott J Dixon may have the highest influence in this intersected field with the highest number of citations and co-cited references respectively. The essential biological reactions such as oxidative stress response, lipid peroxidation metabolism, anti-inflammmatory and pro-inflammatory procedure, and related molecular pathways were knowledge base and current hotspots. Molecules pathways exploration, effective inhibition of I/R injury and promising strategy of improving allografts may become future trends and focuses.

Conclusions: Research on ferroptosis in I/R injury had aroused great interest recently. This first bibliometric study comprehensively analyzed the research landscape of ferroptosis and I/R injury,

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and also provided a reliable reference for related scholars to facilitate further advancement in this field.

1. Introduction

Ischemia reperfusion injury (IRI) is a complex inflammatory status with multi-factors including hypoxia, metabolic stress, vascular leakage, cellular death pathways, and activating a series of immune response [1,2]. It usually occurs when previously ischemic tissues are returned to a state of blood flow, leading to the paradoxical further damage of cellular dysfunction disorders and death [3]. And then extensive tissue and organs are affected including kidney, cardiac, brain, lung, liver, skeletal muscle, gut, testis, spinal cord and the transplanted organs (especially renal allografts, liver transplantation *etc*.) [4–8]. Furthermore, collateral damage maybe also induced to strike distant organs, with server injury and potential multi-system organ failure culminating in inflammation storm and, at worst, death during surgical interventions [7,9]. The global disease burden of tissue and organs damage associated with IRI is exceedingly high and the economic costs are significant, the study reports, illustrating the importance and urgency of related researches [10–12].

Ferroptosis is a unique modality of programmed non-apoptotic cell death, characterized by iron-dependent lipid peroxidation, newly-discovered by Dixon et al. [13] in 2012 [14]. Since then, the number of researches in fields of ferroptosis has exponentially break out over the recent years [15]. And recent studies have reported that ferroptosis is closely related to the occurrence, progress and prognosis of multiple system diseases, such as nerve disorders [16], heart [17], liver [18], gastrointestinal [19], pulmonary [20], kidney [21] and pancreatic diseases [22] associated with the toxicity of iron and lipid peroxidation. And there is a closely connected relationship between ferroptosis and IRI in aforementioned multiple organs in molecular, pathway mechanisms, subcellular structure *etc.* [23–25], which provides potential approaches for the clinical treatment and prognosis of the pathophysiological status of IRI. However, up to now, some comprehensive reports on the research trends, related institutions and scholars, influential researches and the field hotspots of "ferroptosis in ischemia-reperfusion injury" are still lacking.

The aim of this work is to systematically summarize and discuss the "ferroptosis in ischemia reperfusion injury" researches from 2013 to October 2022 by visualization and scientometric analysis. A variety of software and algorithms have been combined into bibliometrics analysis [26], resulting in multidata presentation for the annual trends, countries/regions, institutions, journals, authors and their multi-dimensional relationship to indicate current hotspots and future "ferroptosis in IRI" research directions for scholars in this field.

2. Material and methods

2.1. Source database and data collection

Data were extracted from Science Citation Index Expanded (SCIE) of the Web of Science Core Collection (WoSCC) database on Nov 1, 2022. The search strategy included TS like ferroptosis, ischemic reperfusion injury *etc*, with details in Appendix. The study types were limited to "articles" or "reviews", publication language was restricted to English and timespan was from 2013 to Oct 31, 2022. A total of 442 publications met the search criteria, which were included in this study in the first step.

2.2. Data extraction and analysis

Documents were screened to meet the further criteria for exclusion of publication type including editorial material, meeting abstract, proceedings paper, non-English paper, retracted study, book chapter, early access, correction, monograph, news item, reprint and letter *etc*. [27]. Finally, a total of 421 documents were identified to perform bibliometric analysis and visualization, with 21 early access articles, 1 proceedings paper article, 1 book chapter review and 19 early access reviews excluded. And then we used several softwares including CiteSpace 6.1. R3 (Chaomei Chen, 2022), VOSviewer 1.6.17.0 (Nees Jan van Eck and Ludo Waltman, 2010), Scimago Graphica 1.0.24 and Microsoft Excel 2019 to perform further bibliometric analysis and visualization [28]. All associated data with annual publishment trends, countries/regions, journals and prolific institutions were imported into Excel for quantitative and dynamics trends. Meanwhile, VOSviewer was used to analyze the cooperation among countries, institutions, authors and keywords. While, CiteSpace was aimed to definite the dual-map overlay of journals, knowledge map and timeline view of references, top references and keywords with the strongest citation bursts. And the parameters were set as: link retaining factor (LRF = 3), e for top N (e = 1), time interval (2013–2022), years per slice (1), look backyears (LBY = 5), links (strength: cosine, scope: within slices), selection criteria (g-index: k = 10), and minimum duration (MD = 1) [29]. Different journals impact factor and Journal Citation Reports division were collected based on Web of Science website. The first stage was synonym merging, nonsense terms deletion, data cleaning, for instance, "ischemia-reperfusion", "reperfusion", "reperfusion injury", "ischemia", "ischemia/reperfusion injury" and "ischemia-reperfusion injury" were merged as "ischemia reperfusion injury", "gpx 4" was redefined as "glutathione-peroxidase 4", and "animals", "experiments" were deleted [28] (see Fig. 1).

3. Results

3.1. Annual growth trend of publications

A total of 421 papers were obtained for further analysis. In Fig. 2, since the first related research was published in 2013 [30], the crossed documents of ferroptosis and ischemia reperfusion injury (IRI) has increased manyfold in recent five years. The recent year 2022 only included the publications of previous 10 months, with 153 papers (38.69%), ranking first and the year 2021 had seen the fastest growth in the number of articles published. Meanwhile, the polynomial fitting line predicted future publication trends in this field. The trendline equation is $y = 3.8674x^2 - 25.596x + 36.083$ ($R^2 = 0.956$), with y representing the study volume and x representing the corresponding publication years. This simulation curve showed a positive trend of documents and predicted that the number will reach 222 documents next year.

3.2. Countries/regions and institutions analysis

The bibliometric analysis included 421 papers from 43 different countries/regions and 575 institutions. In Fig. 3A, a geographic visualization map generated from a geographic visualization map showed the relationships among all countries in this field. The red lines between different circles show strength relationship, the color of circles and the line width represent the intensity of collaboration, and the sizes of circles reveal the publications number [31]. It was shown that China (N = 281, 66.7%), the United States (N = 78, 18.5%) and Germany (N = 40, 95%) published the top three documents, representing the larger size circles. And they also had actively collaborated with other countries, showing wider link lines and more red circles. Meanwhile, in Fig. 3B, per citations of countries were analyzed based on VOSviewer 1.6.17.0 statistics among top 10 prolific countries. It could be seen that the United Kingdom (303.25 per citations), Australia (268.27 per citations) and Belgium (260 per citations) ranked first three. Moreover, many developed countries distributed in Japan, European and North American area published higher citied researches.

A detailed list of publications, citations, and cooperation strength of representative institutions was displayed in Fig. 4. As is shown in Fig. 4A, among all 575 institutions, top 10 prolific institutions (N = 13) were included to analyze the documents volume and per citations items. These 13 institutions were distributed in three countries/regions, with more than 10 researches published. Among them, 11 institutions were from China, while remaining 2 institutions were from Germany and the United States. Central South University was the most prolific institution (N = 24), followed by Sichuan University (N = 16) and Wuhan University (N = 13), all from China. However, Columbia University (608.30 per citations), Dresden University of Technology (308.25 per citations) and Shanghai Jiaotong University (123.33 per citations) ranked the top three in the list of per citations. And the VOSviewer map of institutions in Fig. 4B showed the co-authorship and publications. Each node reflected an independent institution. The size of the nodes was represented by the volume of documents (The larger nodes, the more publications). The links revealed the cooperation relationship between institutions (The stronger co-authorship, the wider link) [29]. The publications result in Fig. 4A was consistent with the

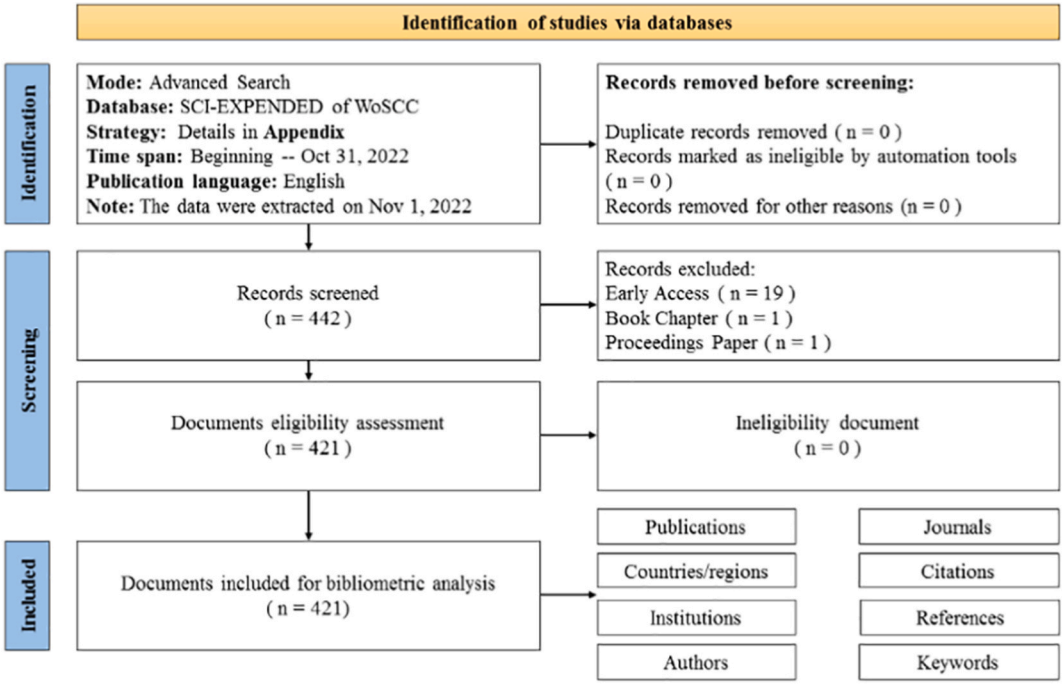

Fig. 1. Flow diagram of data identification and analysis.

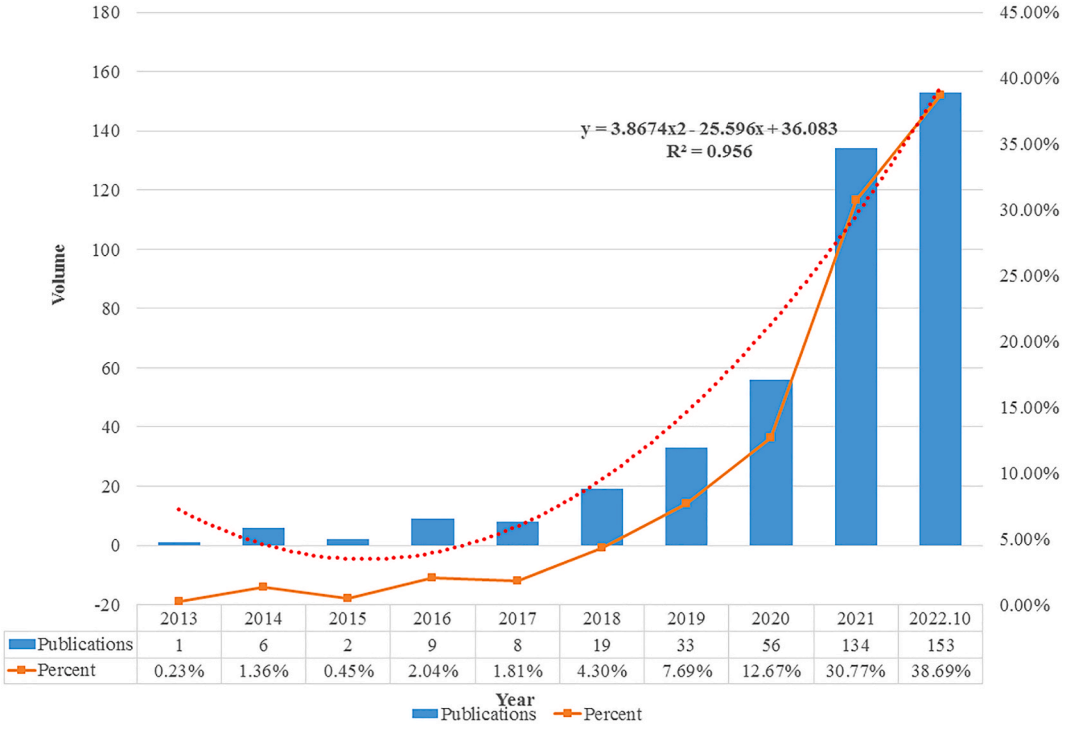

Fig. 2. Trends of ferroptosis and IRI documents over the past decade.

previous document results in Fig. 4A. The total co-authorship strength is represented by lines width between nodes. In this visualized map, 35 active cooperation institutions were included and the top three institutions with the largest co-authorship were listed as follows: Dresden University of Technology, Columbia University and Shanghai Jiaotong University.

3.3. Authors and Co-cited authors

A total of 2584 authors drafted the 421 publications in "ferroptosis in ischemia reperfusion injury". All co-authors could be divided into seven clusters roughly (Fig. 5A) and Andreas Linkermann was located in the center of the map, showing more active relationship. Meanwhile, the larger three nodes were as following: Andreas Linkermann from Dresden University of Technology was the most productive author (N = 16), followed by Mardus Conrad (N = 7) from Helmholtz Zentrum München and Stefan Krautwald (N = 7) from University Hospital Schleswig-Holstein, all from Germany, which was consistent with the results in Table 1. Among the top 10 prolific authors, eighteen authors from five countries were identified and most of them (13/18, 72.2%) were from developed countries, while China had most authors (5/18, 27.8%) in the list of top academics. In Fig. 5B, these co-cited authors (T ≥ 35, N = 79) from a total of 16,894 co-cited authors were selected to perform the visualized analysis with main three clusters. Scott J Dixon from Columbia University with the largest co-citations (N = 489) ranked first, followed by Wan Seok Yang (N = 442) from Columbia University and Andreas Linkermann (N = 275) from Dresden University of Technology representing the larger three nodes which was also consistent with the results in Table 1, among the top 10 co-cited authors from three countries, most of them (7/10) were from developed countries, and only one developing country China had three seats.

3.4. Journals and Co-cited academic journals

A total of 422 researches were published on 204 academic journals on ferroptosis and IRI. Table 2 showed the top 10 journals and co-cited journals ranked by the number of output and co-citations. Among them, *Oxidative Medicine and Cellular Longevity* (N = 22, 5.23%) had the most prolific papers, followed by *Frontiers in Cell and Developmental Biology* (N = 17, 4.04%) and *Cell Death & Disease* (N = 13, 3.09%). Most of them (8/12) were located in the JCR Q1 region with all 2021 IF more than five. Meanwhile, the most three co-cited journals, *Cell* (N = 1390) had the largest number of total co-citations, followed by *Nature* (N = 1126) and *Proceedings of the National Academy of Sciences of the United Stated of America* (N = 1048) with all 2021 IF more than ten and JCR Q1 region. In Fig. 6, the dual-map overlay of journals represented the topic distribution of different academic journals, with the map of the citing and cited journals lying on the left and right side. And the different colored links between bilateral sides reflect the citing and cited relationships between journals [32]. And there was only one main link with orange color representing that the studies published in molecular, biology or immunology journals are usually cited by molecular, biology or genetics journals.

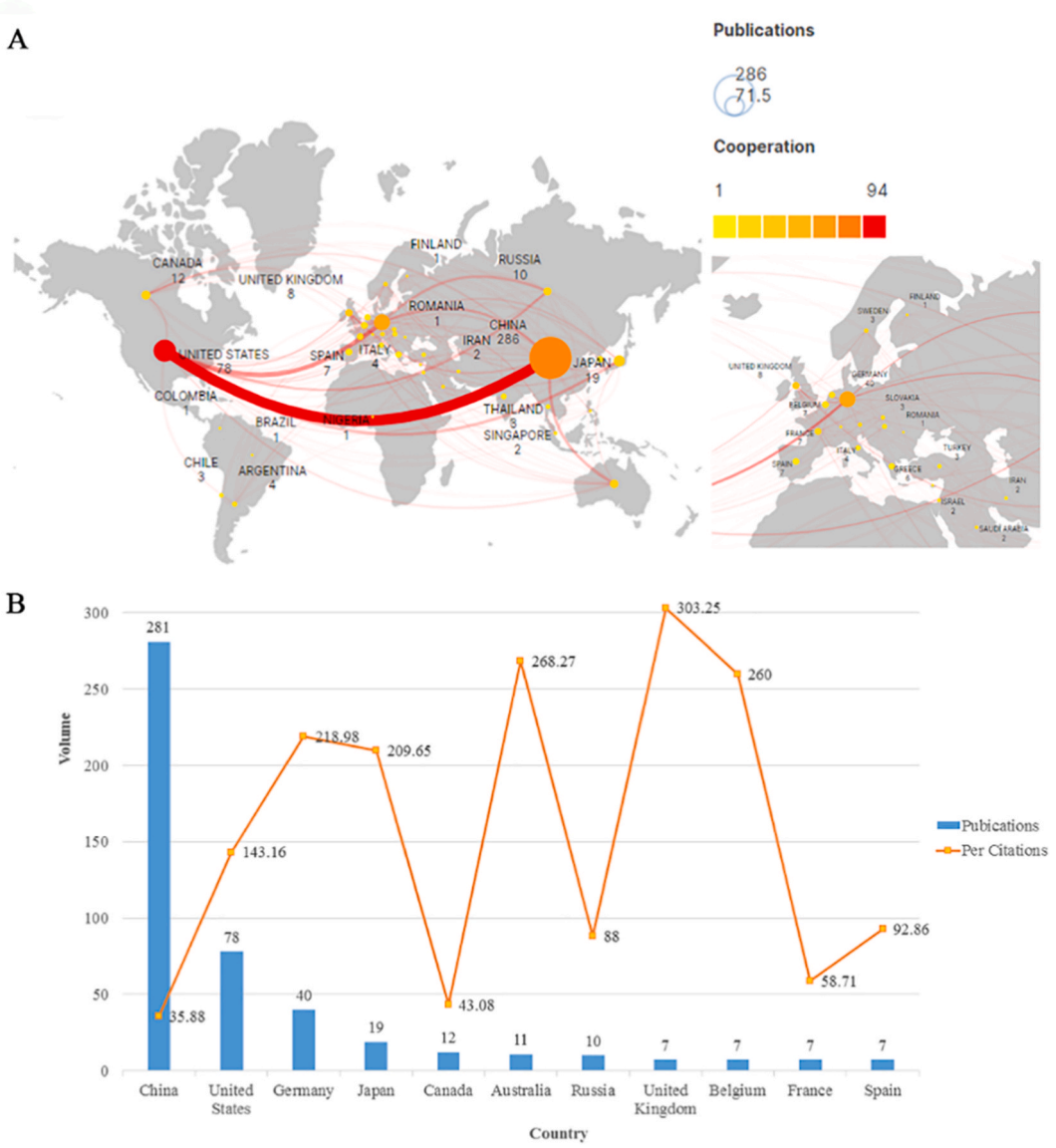

Fig. 3. (A) The prolific countries/regions distribution with related researches. The red lines between different countries/regions and the color of different circles represent the strength cooperation, more red, more closed. The sizes of different circles represent publication number. (B) Countries with the top 10 publications (11 countries). The blue bar chart represents the number of publications of top 10 countries. The yellow broken line shows the average citations per article.

3.5. Analysis of Co-cited references

Among a total of 421 documents related to the ferroptosis and IRI, there were 22,910 co-cited references, with top 20 references characteristic of the strongest citation bursts shown in Fig. 7. Most of them (15/20, 75%) began between 2014 and 2016 year and ended between 2018 and 2020 year. Additionally, there remained two researches with the strongest citation burst still lasting to data, titled "Activation of the p62-Keap1-NRF2 pathway protects against ferroptosis in hepatocellular carcinoma cells" [33] by XF Sun et al. on *Hepatology* and titled "Ferroptosis: process and function" [34] by Y Xie et al. on *Cell Death & Differentiation*. Among them, the strength of six documents were more than 15 (6/20, 30%). For example, JPF Angeli et al. published the document entitled "Inactivation of the ferroptosis regulator Gpx4 triggers acute renal failure in mice" [35] on *Nature Cell Biology* ranking first with the largest strength(Strength = 25.4), followed by WS Yang et al.(Strength = 24.28) [36] in *Cell* and A Linkermann et al.(Strength = 20.6) [37] in *PNAS*, with only three strength over 20. Interestingly, two authors named SJ Dixon [13,38–40] and A Linkermann [37,41,42] had published more than one top 20 references with the strongest citation bursts.

A total of 14 clusters of co-cited references were constructed to visualize and analyze the network between co-cited references by

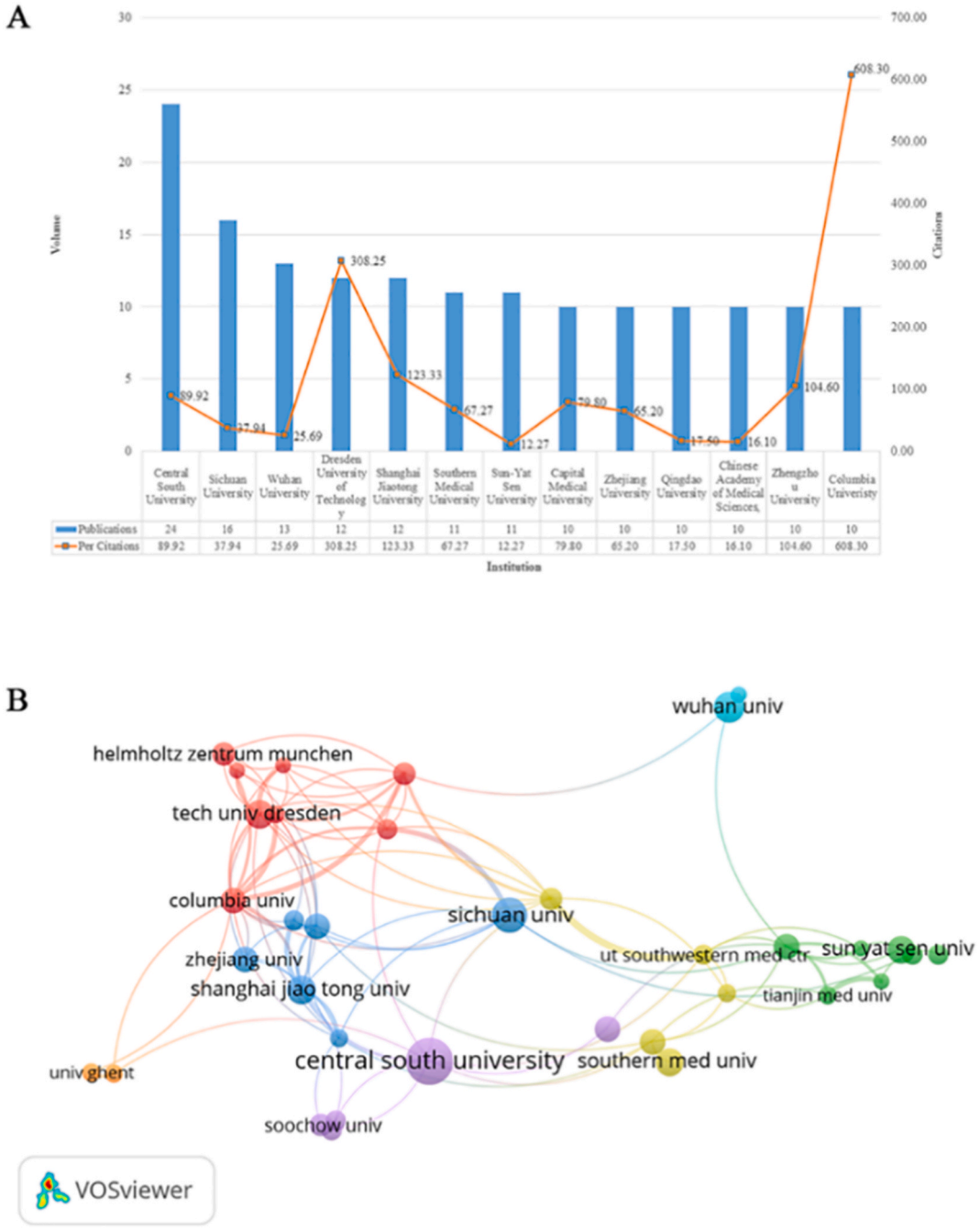

Fig. 4. (A) The top 10 prolific institutions (13 institutions) distribution with related researches. The blue bar chart represents the documents volume. The yellow broken line shows the average citations per article. (B) The VOSviewer visualization of collaborations among different institutions ($T \geq 5$, $N = 35$).

Citespace in Fig. 8A. Modularity Q (0.7197) and Mean Silhouette (0.8654) values were both greater than 0.7, suggesting the significant cluster structure and credible cluster results [43]. Generally, the constructed clusters were divided into two sub-groups: 1) Clinical diseases or physiopathologic mechanism, "#1 ischemic stroke", "#3 acute myocardial infarction", "#4 acute kidney injury", "#6 heart failure", "#13 myocardial ischemia reperfusion"; 2) molecular biological mechanisms, "#0 apoptosis", "#2 mitophagy", "#5 necroptosis", "#7 ripk3", "#8 regulated necrosis", "#9 transcription", "#10 signal transduction", "#11 transcription factor", "#12 Gasdermin D". The timeline view combined with cluster view and time slicing techniques was performed to visualize data related to references. Meanwhile, The size of nodes in a straight line represents the citation frequency of special topic [44]. The clusters on the timeline named "#0 apoptosis", "#1 ischemic stroke", "#2 mitophagy", "#3 acute myocardial infarction", "#4 acute kidney injury"

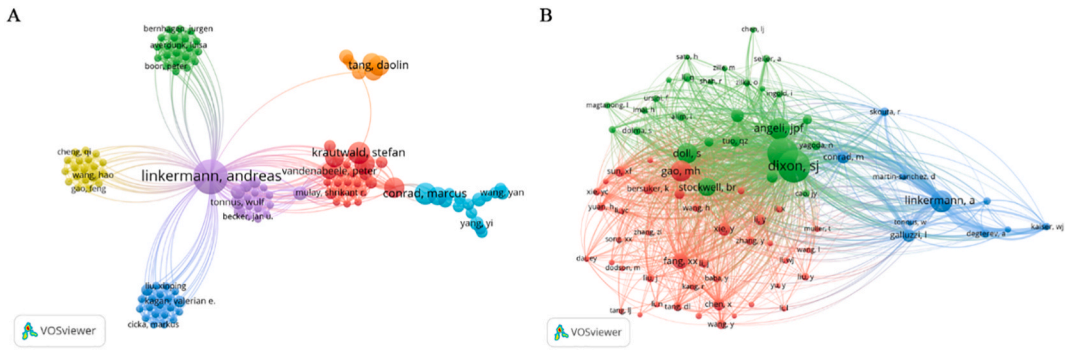

Fig. 5. (A) VOSviewer Analysis of co-authorship (A, T ≥ 1, N = 124) and co-cited authorship (B, T ≥ 35, N = 79) involved in ferroptosis and IRI.

Table 1
The top 10 authors and co-cited authors involved in research on Ferroptosis in Ischemia Reperfusion Injury.

Rank	Author	Organizations	Counts	Citations	Co-cited author	Organizations	Co-citations
1	Andreas Linkermann	Dresden University of Technology, Germany	16	3551	Scott J Dixon	Columbia University, USA	489
2	Stefan Krautwald	University Hospital Schleswig-Holstein, Germany	7	1199	Wan Seok Yang	Columbia University, USA	442
3	Marcus Conrad	Helmholtz Zentrum München, Germany	7	933	Andreas Linkermann	Dresden University of Technology, Germany	275
4	Ulrich Kunzendorf	Christian-Albrechts-University Kiel, Germany	6	1039	José Pedro Friedmann Angeli	University Of Wuerzburg, Germany	258
5	Brent R Stockwell	Columbia University, USA	6	1884	Minghui Gao	Harbin Institute of Technology, China	252
6	Daolin Tang	UT Southwestern Medical Center, USA	6	335	Sebastian Doll	Helmholtz Zentrum München, Germany	244
7	Lei Peng	Sichuan University, China	5	212	Brent R Stockwell	Columbia University, USA	201
8	Qingzhang Tuo	Sichuan University, China	5	212	Xuexian Fang	Zhejiang University, China	163
9	Tom Vanden Berghe	VIB Inflammation Research Center, Belgium	5	1704	Yangchun Xie	University of Pittsburgh Cancer Institute, China	126
10	Jun Peng[a]	Central South University, China	5	135	Lorenzo Galluzzi	Weill Cornell Medical College, USA	123

[a] Represents other authors who have also published 5 documents, including Theodoros Eleftheriadis (University of Thessaly, Greece), Vassilios Liakopoulos (University of Thessaly, Greece), Georgios Pissas (University of Thessaly, Greece), Ioannis Stefanidis (University of Thessaly, Greece), Yu Peng (Nanchang University, China), Xin Chen (Guangzhou Medical University, China), Rui Kang (UT Southwestern Medical Center, USA), Xuejun Jiang (Memorial Sloan Kettering Cancer Center, USA).

and "#13 myocardial ischemia reperfusion" were relatively closed and on the right side, representing more recent and related interrelationship references. Furthermore, their larger nodes showed the much higher citations, representing hot and emerging topics in this field in Fig. 8B.

3.6. Analysis of keywords

Table 3 showed the top 20 occurrences keywords involved in research on ferroptosis in IRI. Ferroptosis had the largest number of occurrences (N = 275) and total link strength (strength = 974), followed by Cell-death (N = 184, strength = 780) and Ischemia Reperfusion Injury (N = 179, strength = 646), showing larger size node in Fig. 9A. And keywords with strongest citation explosion were discerned through CiteSpace in Fig. 9B, showing top 12 related keywords. The timespan was set from 2013 to 2022, showing the green line, while the periods of keywords explosion are represented by the red line. The keywords with strongest citation bursts after 2017 were "glutathione peroxidase 4" (2017–2020, strength 2.9), "nonapoptotic cell death" (2017–2018, strength 2.9), "oxidative stress" (2017–2018, strength 2.59), "alzheimers disease" (2017–2019, strength 2.53) and "reactive oxygen specy" (2018–2020, strength 2.85).

4. Discussion

4.1. General information

First appearing in the views of scholars, Dolma et al. [45] discovered a new compound called Erastin could induce a new way of cell

Table 2
The top 10 journal and co-cited journals involved in research on ferroptosis in ischemia reperfusion injury.

RANK	Journal	Output	% of 421	JIF2021*	Q*	Co-cited Journal	Co-citation	JIF2021*	Q*
1	Oxidative Medicine and Cellular Longevity	22	5.23%	7.310	Q2	Cell	1390	66.850	Q1
2	Frontiers in Cell and Developmental Biology	17	4.04%	6.081	Q2	Nature	1123	69.504	Q1
3	Cell Death & Disease	13	3.09%	9.696	Q1	Proceedings of the National Academy of Sciences of the United States of America (PNAS)	1048	12.779	Q1
4	Antioxidants	12	2.85%	7.675	Q1	Cell Death & Differentiation	865	12.073	Q1
5	International Journal of Molecular Sciences	12	2.85%	6.208	Q1	Journal of Biological Chemistry	827	5.485	Q2
6	Frontiers in Pharmacology	10	2.38%	5.988	Q1	Free Radical Biology and Medicine	790	8.101	Q1
7	Free Radical Biology and Medicine	8	1.90%	8.101	Q1	Cell Death & Disease	654	9.696	Q1
8	Cell Death & Differentiation	8	1.90%	12.073	Q1	Nature Chemical Biology	585	16.284	Q1
9	Frontiers in Cellular Neuroscience[a]	7	1.66%	6.170	Q1	Biochemical and Biophysical Research Communications	585	3.322	Q3
10	Frontiers in Cardiovascular Medicine[a]	7	1.66%	5.848	Q2	Redox Biology	450	10.787	Q1
11	Biomedicine & Pharmacotherapy[a]	7	1.66%	7.419	Q1	–			
12	Cells[a]	7	1.66%	7.666	Q2	–			

[a] Equal Output, JIF2021*: Journal Impact Factor 2021, Q*: Quartile in Category (2021) .

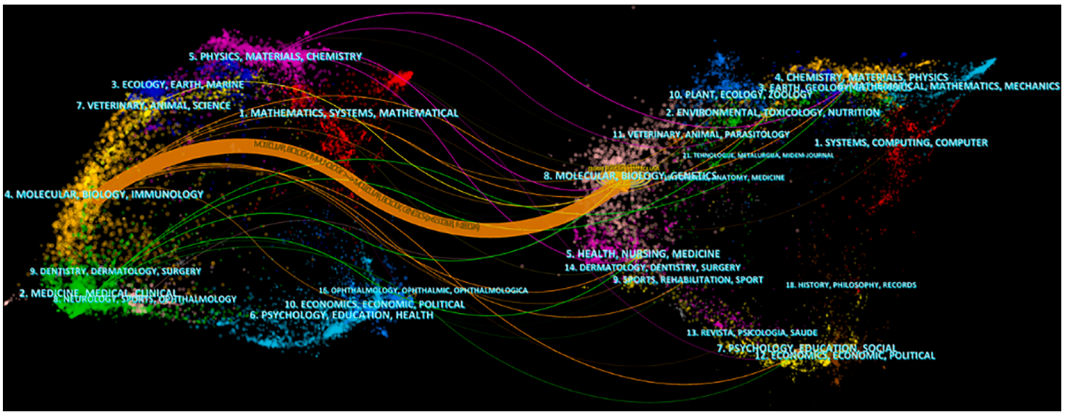

Fig. 6. The dual-map overlay of journals related to ferroptosis and IRI research.

death. Until 2012, Dixon et al. [13] formally named this novel type cell-death ferroptosis leading to further understanding of regulated cell death. In the past few years, the new progress had been made and the documents increased exponentially in the field of ferroptosis (Fig. 2), revealing the pivotal role in multiple systems diseases [15–22]. Meanwhile, as mentioned above, IRI was common in the secondary state after ischemia-induced injury in a variety of organs and was closely related to iron-dependent death [46,47]. However, with increasing researches on IRI and ferroptosis, the objective data analysis on the annual trends, prolific countries/institutions/scholars and their collaborations was still lacking, which posed great challenge on a systematic insight into the evolving research foci and hotspots, especially for those who were burst to this field. For more distinct understanding on development, this related research landscape of scientometrics analysis was conducted.

To our best of knowledge, several ferroptosis-related bibliometric studies were published focusing on the links with stroke [48], cancer [29,49], cardiovascular diseases [50], brain [32] or the whole field [51–53], lack of the critical investigation with IRI. In this study, a total of 421 articles and reviews on ferroptosis in IRI were discerned in WoSCC from 2013 to November 1, 2022. Interestingly, the number of publications published in the first ten months of 2022 accounted for almost 40% of the total published in the previous decade in Fig. 2, showing the increasing importance and popularity of this field.

In countries/regions and institutions analysis, the papers (N = 281) published from China ranked first accounting for 66.7%, showing the largest size circles in Fig. 3A. But developed countries, represented by the United Kingdom (N = 303.25), ranked the top list of per citations, showing more professional academic ability of them in Fig. 3B. As for representative institutions, universities or academics, represented by Central South University (N = 24) from China occupied 11 candidates (11/13) in the list of top 10 prolific institutions in Fig. 4A. Among them, Columbia University (N = 608.30) ranked the top in the institutions list of per citations, consistent

Top 20 References with the Strongest Citation Bursts

References	Year	Strength	Begin	End	2013 - 2022
Dixon SJ, 2012, CELL, V149, P1060, DOI 10.1016/j.cell.2012.03.042, DOI	2012	15.35	2013	2017	
Yang WS, 2014, CELL, V156, P317, DOI 10.1016/j.cell.2013.12.010, DOI	2014	24.28	2014	2019	
Skouta R, 2014, J AM CHEM SOC, V136, P4551, DOI 10.1021/ja411006a, DOI	2014	15.12	2014	2019	
Vanden Berghe T, 2014, NAT REV MOL CELL BIO, V15, P134, DOI 10.1038/nrm3737, DOI	2014	10.06	2014	2019	
Dixon SJ, 2014, NAT CHEM BIOL, V10, P9, DOI 10.1038/nchembio.1416, DOI	2014	10.06	2014	2019	
Linkermann A, 2013, P NATL ACAD SCI USA, V110, P12024, DOI 10.1073/pnas.1305538110, DOI	2013	9.36	2014	2018	
Kaiser WJ, 2013, J BIOL CHEM, V288, P31268, DOI 10.1074/jbc.M113.462341, DOI	2013	7.6	2014	2018	
Murphy JM, 2013, IMMUNITY, V39, P443, DOI 10.1016/j.immuni.2013.06.018, DOI	2013	6.43	2014	2018	
Angeli JPF, 2014, NAT CELL BIOL, V16, P1180, DOI 10.1038/ncb3064, DOI	2014	25.4	2015	2019	
Linkermann A, 2014, P NATL ACAD SCI USA, V111, P16836, DOI 10.1073/pnas.1415518111, DOI	2014	20.6	2015	2019	
Jiang L, 2015, NATURE, V520, P57, DOI 10.1038/nature14344, DOI	2015	13.75	2015	2020	
Gao MH, 2015, MOL CELL, V59, P298, DOI 10.1016/j.molcel.2015.06.011, DOI	2015	19.55	2016	2020	
Dixon SJ, 2014, ELIFE, V3, P0, DOI 10.7554/eLife.02523, DOI	2014	7.96	2016	2019	
Dixon SJ, 2015, ACS CHEM BIOL, V10, P1604, DOI 10.1021/acschembio.5b00245, DOI	2015	7.93	2016	2020	
Linkermann A, 2014, NAT REV IMMUNOL, V14, P759, DOI 10.1038/nri3743, DOI	2014	7.43	2016	2019	
Conrad M, 2016, NAT REV DRUG DISCOV, V15, P348, DOI 10.1038/nrd.2015.6, DOI	2016	7.07	2016	2019	
Chen LJ, 2015, J BIOL CHEM, V290, P28097, DOI 10.1074/jbc.M115.680090, DOI	2015	6.41	2017	2020	
Sun XF, 2016, HEPATOLOGY, V63, P173, DOI 10.1002/hep.28251, DOI	2016	6.47	2019	2022	
Kwon MY, 2015, ONCOTARGET, V6, P24393, DOI 10.18632/oncotarget.5162, DOI	2015	6.22	2019	2020	
Xie Y, 2016, CELL DEATH DIFFER, V23, P369, DOI 10.1038/cdd.2015.158, DOI	2016	7.52	2020	2022	

Fig. 7. Top 20 references with the strongest citation bursts. The red bars mean some references cited frequently; the blue bars were references cited infrequently.

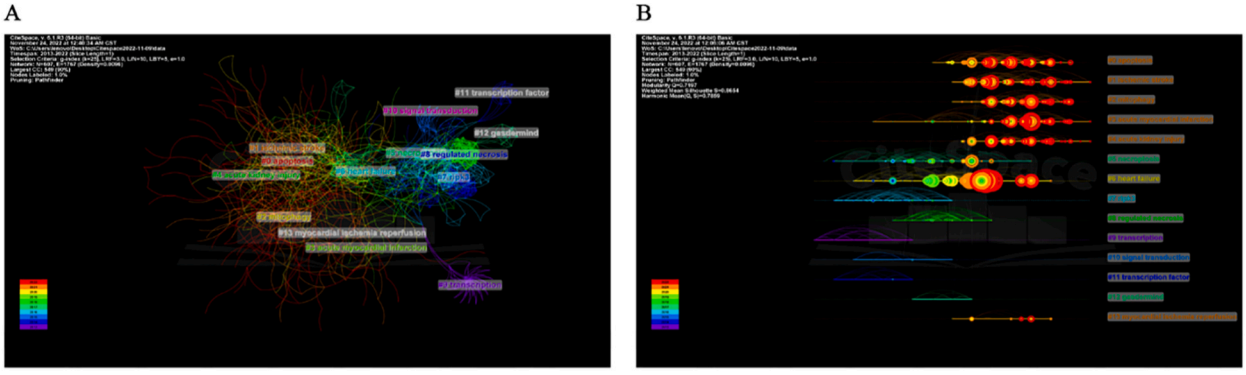

Fig. 8. The knowledge map (A) and timeline view (B) of references related to research between ferroptosis and IRI.

with the results of countries/regions' analysis. Meanwhile, the total link strength (data not shown) between Dresden University of Technology, Columbia University and Shanghai Jiaotong University was the highest, indicating their active cooperative relationship in Fig. 4B.

Among the analysis of authors and co-cited authors in Fig. 5 and Table 1, Andreas Linkermann from Dresden University of Technology, mainly focusing on researches between kidney injury and cell death, not only published the most related papers (N = 16) but also ranked first in the list of closed collaboration, and Scott J Dixon from Columbia University with the largest co-citations (N = 489) ranked first in the list of co-cited authorship, demonstrating the two authors' outstanding contributions to this field. Furthermore, as shown in Fig. 7, the mentioned two scholars were only authors publishing more than one top 20 references with the strongest citation bursts with three papers and four papers from Andreas Linkermann and Scott J Dixon respectively. Among them, the study published by Andreas Linkermann et al. [37]with more than 300 times citations and the third strongest citation bursts (Strength = 20.6) in this study demonstrated the insensitivity to necroptosis of renal tubules under Fas-associated protein with death domain (FADD) or caspase-8 and the failed protection of RIPK1 inhibitor necrostatin-1 (Nec-1) from hypoxic injury. Meanwhile, their group developed a novel third-generation ferrostatin (termed 16–86), with more stable and more effective compared with the first-in-class compound ferrostatin-1. And the landmark article published by Scott J Dixon et al. [13] in *Cell* with more than amazing 2400 times

Table 3
The top 20 occurrences keywords involved in research on ferroptosis in ischemia reperfusion injury.

Rank	Keyword	Occurrences	Total Link Strength
1	Ferroptosis	275	974
2	Cell-death	184	780
3	Ischemia Reperfusion Injury	179	646
4	Oxidative stress	142	529
5	Iron	105	451
6	Lipid-peroxidation	104	463
7	Mechanisms	96	372
8	Apoptosis	79	304
9	Glutathione-peroxidase 4	69	279
10	Metabolism	63	251
11	Activation	51	207
12	Necroptosis	51	192
13	Protects	42	172
14	Autophagy	41	189
15	Acute kidney injury	38	137
16	Disease	36	148
17	Inflammation	36	147
18	Cancer	29	142
19	Expression	28	120
20	Nrf2	27	132

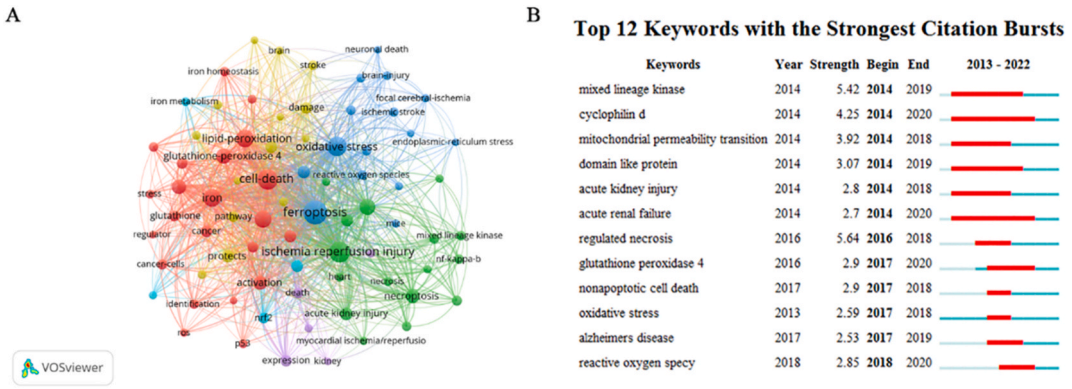

Fig. 9. (A) A network map of keywords by VOSviewer. (B) The keywords with the strong citation bursts in researches related to ferroptosis and IRI.

citations and the top 20 strongest citation bursts (Strength = 15.35) in this study proposed a new way of cell death called ferroptosis dependent on intracellular iron, and distinct from other cell-death patterns on morphology, biochemistry, and genetics.

In analysis of journals and co-cited journals, *Oxidative Medicine and Cellular Longevity* (JCR_{2021} = Q2, IF_{2021} = 7.31) published the most number related studies and *Cell* (JCR_{2021} = Q1, IF_{2021} = 66.85) had the most co-citations, followed by *Nature* and *PNAS*. Notably,

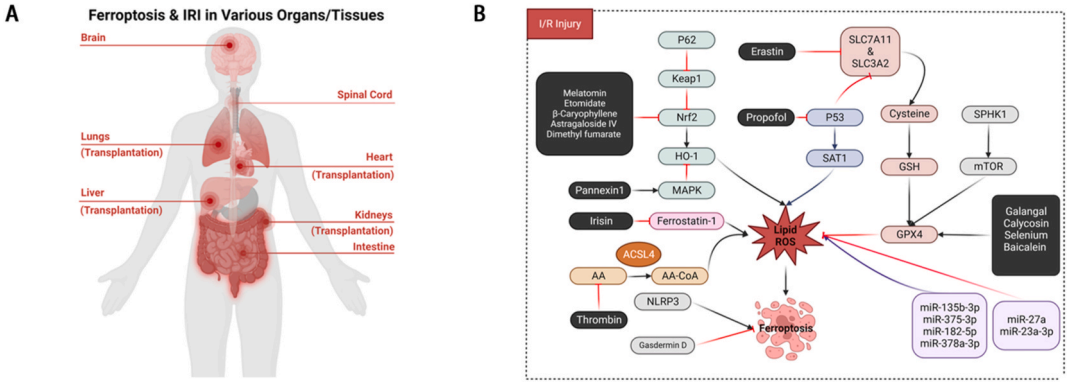

Fig. 10. (A) The brief summary of I/R injury related to ferroptosis in currently known various organs/tissues. (B) Cartoon depicting the main found popular mechanism and regulation of ferroptosis related to I/R injury.

the most journals (21/22) listed in Table 2 rank JCR Q1 and Q2 region, and 7/22 of them reached more than IF 10, showing their critical role in ferroptosis and IRI relatedly intersectional field and attracting higher quality attention and research enthusiasm. Furthermore, *Cell Death & Disease*, *Free Radical Biology and Medicine*, *Cell Death & Differentiation* were mentioned in both lists, indicating more published studies and submission direction for scholars. Interestingly, the aforementioned journals mainly belonged to the cell biology, immunology, and comprehensive fields, consistent with the analysis result in the dual-map analysis (Fig. 6), showing the representative main link among molecular, biology, immunology and genetics journals.

Among the analysis of co-cited references (Fig. 7), top 20 references of the strongest citation bursts were confirmed as fifteen articles and five reviews, representing partial knowledge base in this field. Molecular mechanisms or genes regulations that induce and inhibit various types of cell death were frequently mentioned in above articles and considered as the promising potentials to alleviate IRI and cell-death, including GPX4 [35,36,54], CypD [41], RIP1 [41], TLR3 [55], TRIF [55], RIP3 [55,56], MLKL [55,56], SLC7A11 [39,57], ACSL4 [40], LPCAT3 [40], HO-1 [33,58], P53 [57], NRF2 [33], RIPK3, Gasdermin-D *etc*. Meanwhile, some essential biological reactions such as oxidative stress response, lipid peroxidation metabolism, calcium overload, endoplasmic-reticulum stress, glutaminolysis, cystine-glutamate exchange, microvascular dysfunction, anti-inflammmatory and pro-inflammatory procedure played significant roles which were consistent with the keywords stated in Table 3 and Fig. 9. Notably, two references with strongest citation bursts were still in hotspots: one was newly found factor Nrf2 determining the therapeutic response to ferroptosis-targeted therapies, and another was to summarize the regulation mechanisms and signaling pathways of ferroptosis and discuss the role of ferroptosis in various disease. Based on the references and keywords, we briefly summarized all the currently known IRI organs or tissues in Fig. 10A.

4.2. The hotspots and trending

Keeping abreast of the frontiers and hot topics is essential for scholars in special research field. Luckily, a great deal of domain information was presented with the visualization of annual trends, countries/regions, institutions, journals and authors, especially the top references with the strongest citation bursts (Fig. 7), knowledge map and timeline view of references (Fig. 8), top occurrences keywords (Table 3), network map of keywords and keywords with the strong citation bursts (Fig. 9). Furthermore, these references and keywords relatively overall reflected the urgently needed information about hot issues and development tendency. On this basis, we had interconnected these often mentioned stars to partially represent the applications of ferroptosis in IRI.

4.3. Potential role of ferroptosis in various IRI organs/tissues

Programmed cell death was a general term of a set of cell death modes such as apoptosis, necroptosis, autophagy, pyroptosis, and ferroptosis, activated by signal transduction modules and regulated by genes and pharmacology molecules [59]. Intriguingly, ferroptosis initially attracted the interest of scholars in an anonymous way by Dolma et al. [45], which activated nonapoptotic cell death process different from other modes encountered before. While, until to 2012, Dixon et al. [13] confirmed the unique cell death characterized by iron-dependent lipid peroxidation. Notably, these cell death modes are not independent among each other in diseases, especially ischemia reperfusion injury, but mutually intersect with others via molecule regulation and gene expression mentioned in Figs. 7 and 9. For example, pyroptosis could be activated by the NLRP3 inflammasome under cellular ion homeostasis changes, while this could also be initiated by MLKL from the terminal effector necroptosis in response to membrane destroy [58]. And ferroptosis was confirmed to exacerbate RIPK1 phosphorylation and induce necroptosis by disrupting mitochondrial membrane in cardiac I/R Injury [60]. Meanwhile, RIPK1 phosphorylation activation and GPX4 inhibition could be promoted by heat shock protein 90, contributing to necroptosis and ferroptosis in kidney I/R injury [61]. Herein, the simplified and popular role of ferroptosis in IRI was summarized in Fig. 10B and discussed as follows.

4.3.1. Ferroptosis and cerebral I/R injury

Ischemic stroke posed a huge barrier to human health, especially the elderly. In the early stage of disease development, inhibiting ferroptosis, improving perfusion and attenuating inflammatory damage had been confirmed as essential roles in currently known treatment. Although the detailed mechanisms of ferroptosis in IRI were still unclear, ferroptosis could be regulated by protein, nuclear factor, enzyme and gene expression which had been investigated as promising targets. So far, a variety of compounds, like Galangal [62], Selenium Compound [63] and Calycosin [64] had been revealed to inhibit this process via GXP4/SLC7A11 axis, key nod of mediating ferroptosis. In addition, P62/Keap1/Nrf2 pathway-mediated ferroptosis was mitigated by Astragaloside IV [65], and Nrf2/HO-1 signaling pathway was activated by β-Caryophyllene to alleviate ferroptosis induced by cerebral IRI [66], showing intersected regulating network axis. Meanwhile, non-long chain coding RNA (miR-27a) played a positive role to aggravate cell death [67], and NLRP3 inflammasome also participated in the same procedure [68]. Some pathways such as SPHK1/mTOR axis [69] and arachidonic acid/ACSL4 regulation [70] were uncovered to indicate the potential target of ferroptosis for cerebral IRI treatment.

4.3.2. Ferroptosis and myocardial I/R injury

Myocardial IRI was an inevitable dilemma for acute myocardial infarction, ascribed to suggested percutaneous coronary intervention and drug thrombolytic therapy [71]. A variety of studies revealed that sedative or anesthetic drugs played important roles in inhibiting myocardial ferroptosis. For example, IRI could be alleviated by Dexmedetomidine activating AMPK/GSK-3β/Nrf2 channel [72], Propofol inhibiting AKT/P53 axis [73] and Etomidate regulating Nrf2/HO-1 signal pathways [74]. Coincidentally, in I/R rat model and secondary cardiac fibrosis cell model, miR-375–3p was revealed to directly target GPX4-an inhibitor of the ferroptosis pathway [75]. And in cardiomyocyte injury and IRI model, miR-135 b-3p was also found to aggravate ferroptosis and worsen IRI by

downregulating GPX4 expression [76]. The findings demonstrated that miRNAs played an important role in cardiocerebral system in the last few decades. However, in end-stage of heart disease, heart transplantation remained the first-line treatment with unclear precise inflammatory mechanisms. Li et al. [77] revealed the possible TLR4/Trif-dependent signaling way to initiate inflammation outburst and activiate neutrophil recruitment, leading to injured myocardium which provided potential target for the survival of transplanted heart.

4.3.3. Ferroptosis and kidney I/R injury

Renal IRI was still a puzzling trouble for the management of perioperative period and postoperative functional rehabilitation, which was related to oxidative stress and inflammatory storms, and often leaded to acute kidney injury. Recently, the role of ferroptosis in renal ischemia-reperfusion was discovered and a series of molecules were confirmed to be involved in regulating this pattern of death. For example, injury could be mitigated by followed processes of compounds via ROS inhibition: Quercetin and Irisin up-regulated SLC7A11/GPX4 axis [78,79]; Pannexin 1 activated MAPK/ERK to inhibit HO-1 [80]; Melatomin promoted Nrf2 into nucleus and restrained SLC711A [81]. Meanwhile, miRNAs such as miR-182–5p and miR-378a-3p, and Gasdermin-D played roles in promoting and suppressing lipid peroxidation [82,83]. Another special status of kidney IRI, kidney transplantation, has made substantial progress attributed to pharmacological development and advanced technology, while short and long-term complications still bothered patients and scholars. The modulation of ferroptosis had been discerned to be a promising tool of downregulating the deterioration of the renal I/R injury. Furthermore, lots of intermediate and terminal molecules may be exploited to predict transplanted complications [84].

4.3.4. Ferroptosis and others I/R injuries

In hepatic I/R injury, it usually occurred after transplantation and threatened postoperative organ survival and liver function recovery. Yamada et al. [85]revealed the existence of ferroptosis: iron overload aggravated liver I/R injury in liver grafts and the predictor of High ferritin levels. Meanwhile, Dimethyl fumarate (DMF) initiating the NRF2/SLC7A11/HO-1 axis and targeting Malic Enzyme 1 activation had been also ensured to prevent hepatic damage and oxidative stress reaction [86,87].

In vivo rats model of lung IRI, Xia et al. [88] also showed the inevitable role of ferroptosis that lung tissue mitochondrial oxidative stress and organelle deterioration could be reduced by Human bovine pyridine apelin-13 to inhibit ROS and edema. Especially, irisin, a known role in Renal IRI, the effective inhibitor of ferrostatin-1, down-regulated ROS and Fe2+ and promoted Nrf2/HO-1 signal axis to prevent ferroptosis [89]. Meanwhile, during the end-stage lung disease, IRI still damaged the allografts survival and function recovery after transplantation. Luckily, the found role of ferroptosis could provide the therapeutics for IRI and related molecules with cell-death also could be used to assess the academic status for grafts [90].

Intestinal tissue IRI was also associated with ferroptosis. Li et al. [47]demonstarated that ACSL4/Sp1 promoted lipid reactive oxygen species to execute ferroptosis. Furthermore, Apigenin-7-*O*-β-D-(-6″-*p*-coumaroyl)-glucopyranoside (APG), a new flavonoid glycoside was revealed to improve intestinal edema, attenuated ROS generation and restrained Fe2+ accumulation to inhibit ferroptosis [91]. In the cardiopulmonary resuscitation pig models, the aldehyde dehydrogenase 2 (ALDH2) agonist, Alda-1 was ensured to alleviate intestinal injuries [92].

Spinal cord ischemia-reperfusion injury (SCIRI) usually occurred when previously ischemic tissues were returned to a state of blood flow, leading to loss of sensory and motor function. In mice model, Rong et al. [8]demonstrated the associated link between ferroptosis and SCIRI, and that ubiquitin-specific protease 11 (USP11) inhibition may prevent autophagy-dependent ferroptosis pointing to a potential target for treatment of SCIRI.

4.3.5. The prospect of ferroptosis and vascularized composite allografts

Interestingly, the role of ferroptosis in organs was no single and liner, but mutual, networked and constantly explored. Meanwhile, the organs transplantation in ferroptosis made prominent progress in recent years, like heart, lungs, kidneys and liver etc., lack of related vascularized composite allografts (VCA) researches and potential effect of ferroptosis in donor lifetime had been confirmed recently. A compared review [93] on VCA and solid organ transplants showed the common and urgent barriers: short and long-term immune rejection and ischemia reperfusion injury. With more discovered signal pathways, ferroptosis may also provide promising therapeutic targets in IRI of VCA, contributing to essential promotion for VCA future.

4.4. Strength and limitations

Our research has several advantages. Firstly, to our knowledge, this is the first bibliometrics study on ferroptosis and integral body of ischemia reperfusion injury. Secondly, annual trends were presented in an accurate and objective manner to provide a comprehensive information to guide scholars working in this field. Several bibliometrics softwares were used to explore the relationships among different items including countries, authors, institutions, journals, fund agencies, keywords, citations/co-citations and references in multiple dimensions. This increased the strength of our results. Notably, based on the keywords and references indexed by bibliometrics, we conducted a detailed review of the landscape between ferroptosis, and IRI and VCA in various organs/tissues, pointing to the potential therapeutic and hotspots in the future. However, the following limitations should be noted. Firstly, we retrieved data from only WoSCC except Embase, Scopus and PubMed, increasing the risk of literature selection bias. But we needed to consider that WoSCC is the most uesd database for scientometric analyses [32]. Furthermore, current scientometric tools had extremely difficult time simultaneously analyzing data from multiple databases. Secondly, only publications in English were retrieved, and therefore some linguistic bias may exist in this study. Thirdly, we only included literature from limited years from 2012 to October

2022, which may not reflect the full landscape of changes in this subject. Fourthly, the literature review was based on the hot keywords of bibliometrics and partially burst references due to the limited content. And hotspots were developed based on a wide range of studies. However, RCTs, guidelines, recommendations and clinical trials may have greater impacts on the field than other types of studies, based on the conclusions they draw. Therefore, future research may benefit from data analysis and visualization for different types of studies. Finally, all information was extracted by scientometric tools rather than authors manually in meta-analysis or overview of systematic review, resulting in bias of our results. By developing machine learning, natural language processing, and data science, these problems may be solved in the future. 5. Conclusion.

The comprehensive and bibliometric analysis between ferroptosis and I/R injury was conducted in multidimensional form including the annual trends, countries/regions, institutions, journals, authors and their multi-dimensional relationship, hotspot topics, potential directions and detailed literature review. Ferroptosis has aroused great adulation in the management and treatment of IRI field, such as triggering mechanism, therapeutic target and even tissue engineering researches. This comprehensive bibliometric analysis had given the hotspots, key researches and potential development trends for scholars to expand and explore the relationships to effectively improve and inhibit I/R injury. Despite of early period exploration, further investigation of molecules pathways, effective inhibition of I/R injury and promising strategy of improving allografts via ferroptosis still remain riveting routes. We believed that useful information could be offered by this study for researchers in future research trends.

Author contribution statement

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Data availability statement

Data included in article/supp. material/referenced in article.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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The partial figure was created with https://biorender.com/with an academic license.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2023.e20363.

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