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LETTER TO THE EDITOR

Targeting nuclear factor erythroid 2-related factor 2-regulated ferroptosis to treat nervous system diseases

Ye-Qi Huang, Zheng-Wei Huang, Xue-Juan Zhang

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Abstract

By critically examining the work, we conducted a comprehensive bibliometric analysis on the role of nuclear factor erythroid 2-related factor 2 (NRF2) in nervous system diseases. We also proposed suggestions for future bibliometric studies, including the integration of multiple websites, analytical tools, and analytical approaches, The findings presented provide compelling evidence that ferroptosis is closely associated with the therapeutic challenges of nervous system diseases. Targeted modulation of NRF2 to regulate ferroptosis holds substantial potential for effectively treating these diseases. Future NRF2-related research should not only focus on discovering new drugs but also on designing rational drug delivery systems. In particular, nanocarriers offer substantial potential for facilitating the clinical translation of NRF2 research and addressing existing issues related to NRF2-related drugs.

Key Words: Bibliometric; Nervous system diseases; Nuclear factor erythroid 2-related factor 2; Ferroptosis; Target

Core Tip: Based on the original paper, this study highlights two primary points: Bibliometric methodologies and future perspectives for nuclear factor erythroid 2-related factor 2 (NRF2) research. We emphasize the need for broader literature databases and diversified analysis methods in bibliometric research. Additionally, we highlight the importance of NRF2 modulation in regulating ferroptosis for treating nervous system diseases and discuss how nanoparticle drug delivery systems may overcome challenges associated with NRF2-related drugs.

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TO THE EDITOR

We have thoroughly examined the high-quality bibliometric study by Chang *et al*[1], published in the high-impact journal, *World Journal of Clinical Cases*. In this study, a bibliometric analysis of the literature was conducted to provide a comprehensive overview of trends in evolutionary research trends on nuclear factor erythroid 2-related factor 2 (NRF2) in the nervous system disease, including focal points, frontiers, and potential future directions. The main content of the study is summarized in Table 1.

Herein, we critically analyze the paper by Chang *et al*[1] focusing on two aspects, *i.e.*, bibliometric methodologies and future perspectives, to stimulate constructive discussion in clinical and basic research communities. It will be our great pleasure to receive a response from the author team soon.

BIBLIOMETRIC METHODOLOGIES

The conceptualization and design of the bibliometric study were acceptable. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses guideline-followed recruitment approach was illustrated. The analyzed results were relatively convincing and the discussion was appealing. The obtained findings can serve as a reference for future research on neurological diseases.

Chang *et al*[1] commented on the limitations of their study. Based on this, we suggest the following for future bibliometric studies.

Incorporating data from a broader spectrum of literature

First, it is suggested to expand the range of literature databases used. Although the Web of Science Core Collection is highly trusted, other major databases such as Scopus, PubMed, and Google Scholar are recommended for cross-comparison. Second, the diversity of the TS can be improved including core clinical topics from the published literature on nervous system disorders. Using these approaches will provide access to a wider range of sources and diverse literature, enhancing the comprehensiveness and credibility of bibliometric analyses.

Acquiring more bibliometric information

Additional bibliometric tools and various bibliometric analytical methods can be used. Besides CiteSpace, other important software or online platforms like VOSviewer, SATI, HistCite, can be utilized for indicator analysis and visual representation. Additionally, statistical analysis and co-word analysis can be conducted alongside citation and cluster analyses to gain deeper insights.

Enhancing the dimensions of the discussion on the results

The integration of strengths, weaknesses, opportunities, and threats (SWOT) analysis is an effective approach. SWOT analysis includes problem diagnosis and prescription. Through a comprehensive analysis of internal and external factors, SWOT analysis assists in identifying the research object's current capabilities and potential for further development[2]. This analysis has been comprehensively applied in medical and biomedical sciences[3,4]. The inclusion of SWOT analysis can offer more valuable information and implications to the status quo and future directions in these fields of research.

By incorporating a broader spectrum of literature, acquiring more bibliometric information, and enhancing the dimensions of the discussion on the results, bibliometric investigations will become more informative.

FUTURE PERSPECTIVES

Importantly, as revealed by the study focus and frontiers in the original paper, the regulation of ferroptosis by NRF2 in nervous system cells has been a research hotspot in this field. NRF2 was first reported to participate in ferroptosis regulation by Sun *et al*[5] in 2016. In the study, the p62-Keap1-NRF2 antioxidative signaling pathway was identified as a crucial negative regulator of ferroptosis in hepatocellular carcinoma cells, exerting its effect through the transcriptional activation of genes involved in reactive oxygen species and iron metabolism. Up to now, ferroptosis has been shown to play an important role not only in the treatment of nervous system diseases but also in various cancer types. The regulatory pathways associated with NRF2 in diverse diseases have been widely examined and can be summarized as follows.

Table 1 Main content of the original paper

Item	Details
Background	NRF2, a pivotal transcription factor in the antioxidant network, has been reported to play a crucial role in nervous system diseases. susceptible to oxidative damage. Consequently, the regulatory capacity of NRF2 and its potential clinical applications in these diseases have generated increasing interest in the scientific community
Methods	Data were gathered through a systematic review of the literature on NRF2 and nervous system diseases from the Web of Science Core Collection database and used the bibliometric tool CiteSpace for analysis
Results	Recent years have seen a rise in NRF2-related literature on nervous system diseases, with China holding a dominant position in terms of article publications, financial investments, and the number of authors with the highest publication records. NRF2 research hotspots have evolved from focusing solely on antioxidants to include anti-apoptosis and active ferroptosis
Conclusion	China should prioritize enhancing the quality and impact of research articles. Research on NRF2 in nervous system diseases has expanded to encompass various cell death mechanisms. Further clinical investigations of NRF2-related medications are necessary

NRF2: Nuclear factor erythroid 2-related factor 2.

Antioxidative activities

The NRF2 signaling pathway is intricately associated with multiple pathways involved in oxidoreductase synthesis, which plays an overlooked role in the process of ferroptosis. Specifically, the downstream targets of NRF2 encompass nicotinamide adenine dinucleotide phosphate [NAD(P)H] quinone oxidoreductase 1, heme oxygenase-1 (HO-1), solute carrier family 7 membrane 11, NAD(P)H quinone oxidoreductase 1, thioredoxin 1, phase II detoxifying enzymes, and various multidrug resistance-associated transporters[6,7].

Iron homeostasis

Notably, NRF2 plays a crucial role in maintaining iron homeostasis, thereby influencing cell susceptibility to ferroptosis through its impact on free radical generation and lipid peroxidation[8]. For instance, NRF2 has been reported to control HERC2 (E3 ubiquitin ligase for nuclear receptor coactivator 4 and F-box/LRR-repeat protein 5), and vesicle-associated membrane proteins 8 (which mediates autophagosome-lysosome fusion) for the maintenance of iron homeostasis[9]. Additionally, various iron-related proteins such as ferritin, transferrin receptor, ferroportin, and HO-1 have been shown to be with NRF2[10,11]. In a nutshell, NRF2 is an inhibitor of ferroptosis.

The elucidation of the above-mentioned regulatory mechanisms contributes to a deeper understanding of NRF2's role in nervous system diseases. It is well documented that multiple nervous system diseases, such as central nervous system injury (ischemic stroke, hemorrhage, and spinal cord injury)[12], neurodegenerative disease, such as Huntington's disease, Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis[13], and the brain cancers[14] are closely related to upregulated ferroptosis processes. For instance, the induction of ferroptosis has been shown to effectively suppress glioblastoma cells through the downregulation of the protein kinase B/mammalian target of rapamycin complex 1/glutathione peroxidase 4 signaling pathway in the study by Cai *et al*[15] in 2023. Suppressing the ferroptosis level in damaged cells is a reasonable and promising strategy to manage the above-mentioned nervous system diseases.

From this point of view, to upregulate NRF2 expression, by downregulating the expression of NRF2 competition factors like bric-a-brac and cap-n-collar homology 1[16], or activate the NRF2-involved signaling pathways, *e.g.* vitamin D receptor/NRF2/HO-1 pathway[17], Keap1-NRF2-ARE pathway[18], will become a ferroptosis-targeted therapy for nervous system diseases. Numerous NRF2 activators have entered clinical trials, highlighting their potential therapeutic applications[19]. Dimethyl fumarate (DMF), which has demonstrated the ability to induce NRF2 activation within the central nervous system, has been approved for psoriasis treatment[20]. Additionally, another NRF2 agonist, cyanoenone triterpenoid RTA-408 (omaveloxolone), was reported in a phase 2 randomized clinical trial to be well tolerated and to enhance neurological function when administered at a dosage of 160 mg/day over 12 weeks[21]. These findings underscore the significant clinical value of targeted NRF2 therapy and its growing prominence in research. To achieve the above targeted therapy, the utilization of various drugs, consistent with the findings of Chang *et al*[1], particularly, bioactive compounds from traditional Chinese medicine has been documented in relevant research. For instance, Salidroside[22], Forsythoside A[23], DMFs[24], Trehalose[25], Berberine[26], *etc.*, and exerted promising activities.

Furthermore, sophisticated nanocarriers can be designed to deliver the corresponding therapeutics to enhance the efficacy of ferroptosis-targeted therapy for nervous system diseases. Nanocarriers, *e.g.*, lipid nanoparticles (*e.g.* liposomes), polymeric nanoparticles (*e.g.* polymeric micelles), and protein nanoparticles (*e.g.* albumin-based nanoparticles) (Figure 1), are versatile vesicles that are attracting attention from the clinic, industry, and laboratory. Appropriately designed nanocarriers can enhance the stability[27], bioavailability[28], and targetability[29] of encapsulated therapeutic agents, which are largely beneficial for treating nervous system diseases. For instance, Liu *et al* [30] designed a quercetin-modified ultrasmall $Cu_{2-x}Se$ antioxidative nanoparticles (CSPQ) to boost Parkinson's disease therapy by activating NRF2. Moreover, previous research on CSPQ by the same authors demonstrated the successful targeting of microglia by coating the nanoparticle with a dopaminergic neuron cell membrane[31]. Gai *et al*[32] prepared folate-modified liposome nanoparticles for the targeted co-delivery of erastin and putative metallothionein (to inhibit NRF2 expression) to enhance the bioavailability and efficiency of the drug/gene combination. These studies demonstrate the potential of nanoparticle drug delivery systems for precise and effective modulation of NRF2 signal pathways.

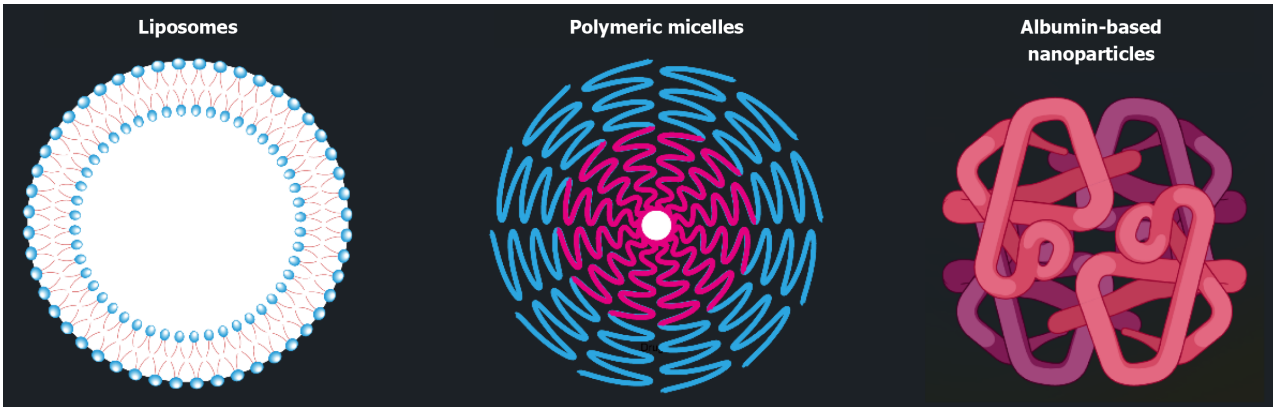

Figure 1 Illustration of representative lipid nanoparticles, polymeric nanoparticles, and protein nanoparticles.

CONCLUSION

We predict that the NRF2 regulation can effectively modulate ferroptosis levels, which are closely associated with nervous system diseases, thereby facilitating the treatment of related disorders. Nanoparticle drug delivery systems are expected to enhance the bioavailability, targetability, and stability of NRF2-related drugs, thereby facilitating the clinical translation of relevant research. In our ongoing studies examining the involvement of NRF2 in a wide range of critical signaling pathways within the human body, we plan to explore the potential of the co-delivery of NRF2-related drugs in combination with other ferroptosis-regulating drugs. We hypothesize that this strategy will enhance cellular drug sensitivity, which exhibits limited efficacy across various tumor cell types. Additionally, NRF2 regulation has demonstrated potential therapeutic efficacy against neurological disorders; however, the treatment of these diseases inevitably encounters delivery challenges such as the blood brain barrier. We envision achieving precise and efficient regulation of NRF2 at specific sites by employing rational bioengineering design strategies for delivery systems, including the utilization of bionic "hitchhiking" mechanisms and charge-mediated penetration capabilities.

FOOTNOTES

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