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The Mechanisms and Recent Advances in Research on Iron-Mediated Cell Death in Diabetic Cataracts

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Abstract: ***Diabetic cataract (DC) is one of the most common and blinding ocular complications of diabetes mellitus (DM). Its pathogenesis is complex; traditional theories suggest that hyperglycaemia-induced oxidative stress, the accumulation of advanced glycation end products (AGEs) and the activation of the polyol pathway are key mechanisms. However, these mechanisms do not fully account for the rapid progression and unique characteristics of DC. In recent years, a novel, iron-dependent form of programmed cell death—ferroptosis—has attracted significant attention in the pathogenesis of various diseases due to its unique biochemical characteristics. This article aims to provide a systematic review of the core molecular mechanisms of ferroptosis, explore its key role in the pathophysiology of DC, and focus on elucidating the molecular networks underlying iron metabolism imbalance, lipid peroxidation accumulation, and the collapse of the antioxidant defence system in lens epithelial cells under hyperglycaemic conditions. The article provides a detailed analysis of the roles of key signalling pathways such as Nrf2/HO-1, p53, GPX4, in regulating ferroptosis in DCs, and integrates newly identified regulatory axes, such as LCN2 and VDAC3-USP47, discovered in recent studies. Furthermore, this paper outlines the immense potential of targeting ferroptosis as a novel strategy for the prevention and treatment of DC, with the aim of providing a theoretical basis and future direction for elucidating the complete pathological mechanisms of DC and developing innovative drugs.***

Keywords: Diabetic cataract, Ferroptosis, Research progress**.**

1. Introduction

Cataracts are the leading reversible cause of blindness worldwide, characterised by a decrease in lens transparency or a change in colour, which can lead to progressive visual impairment. Diabetic cataracts (DC), as one of the severe microvascular complications of diabetes, are characterised by early onset and rapid progression, and are one of the primary causes of visual impairment and even blindness in diabetic patients [1]. With the global prevalence of diabetes continuing to rise, the prevention and treatment of DC has become a major public health issue requiring urgent attention.

Traditional research has confirmed that the pathophysiological process of DC primarily involves three mechanisms: activation of the polyol pathway leading to sorbitol accumulation and osmotic imbalance; non-enzymatic glycation reactions producing advanced glycation end products (AGEs), which damage lens proteins and cellular structures; and persistent oxidative stress induced by mitochondrial dysfunction under hyperglycaemic conditions [2]. Among these, oxidative stress is the central driver of the onset and progression of DC; by generating excessive reactive oxygen species (ROS) that attack cell membranes, proteins and DNA, it ultimately leads to the apoptosis of lens epithelial cells (LECs) and the degeneration of lens fibre cells, resulting in opacification [3]. Although research into these pathways has made some progress, no specific drugs capable of effectively delaying or reversing the progression of DC have yet emerged in clinical practice. The fundamental reason for this lies in our still incomplete and superficial understanding of the pathogenesis of DC.

For a long time, apoptosis was considered the primary mechanism leading to LEC depletion. However, a growing body of research has found that apoptosis does not occur significantly in LECs associated with age-related cataract (ARC) and DC, suggesting that other forms of programmed cell death may be involved in the pathological process of DC [4]. Ferroptosis is a novel, regulated, iron-dependent mode of cell death first defined in 2012 by Brent R. Dixon and colleagues [2]. In recent years, a large body of research has confirmed that ferroptosis plays a key role in a variety of major diseases, including cancer, neurodegenerative diseases, ischaemia-reperfusion injury and diabetes [5]. In particular, ferroptosis is closely associated with the development of diabetic microvascular complications, making it a highly promising new therapeutic target [6].

Given the prevalence of oxidative stress and abnormal iron metabolism in the pathological progression of diabetic foot ulcers (DFUs), the role of ferroptosis in the development and progression of DFUs is gradually becoming a research focus. Preliminary evidence suggests that in a hyperglycaemic environment, endothelial cells (LECs) are more susceptible to ferroptosis, and that inhibiting ferroptosis can effectively delay the progression of DFUs [1], [7]. This paper aims to systematically review and synthesise current research findings on the mechanisms of ferroptosis in DFUs. Firstly, we will provide a detailed introduction to the core molecular mechanisms of ferroptosis, including the regulatory networks of the three major modules: iron metabolism, lipid peroxidation, and the antioxidant defence system. Secondly, we will explore in depth how a high-glucose environment induces ferroptosis in LECs via multiple signalling pathways (such as Nrf2/HO-1 and p53), and analyse the complex interactions between this process and traditional DC pathological mechanisms (such as oxidative stress and

apoptosis). Finally, this paper will summarise therapeutic strategies targeting ferroptosis and their prospects for application in the prevention and treatment of DC, with the aim of providing new perspectives and insights for both basic research and clinical treatment of DC.

2. Core Molecular Mechanisms of Ferroptosis

Ferroptosis is a form of regulated cell death driven by the excessive accumulation of iron-dependent lipid peroxides and reactive oxygen species (ROS) [8]. Its biochemical processes are subject to fine-tuned and complex regulation by intracellular iron homeostasis, lipid metabolism, and antioxidant defence systems.

2.1 Imbalance in Iron Metabolism and Expansion of the 'active iron pool' (LIP)

Iron is an essential trace element for life, participating in numerous core physiological processes such as oxygen transport, electron transfer and DNA synthesis. However, iron is highly chemically reactive; excessive levels of free iron (particularly Fe^{2+}) within cells can catalyse the Fenton reaction, converting H_2O_2 into highly destructive hydroxyl radicals (·OH), thereby triggering widespread oxidative damage [9]. The pool of free, reactive iron ions within the cell is termed the 'labile iron pool' (LIP). The occurrence of ferroptosis is closely associated with the excessive expansion of the LIP. Under physiological conditions, cells maintain iron homeostasis through a finely tuned regulatory network, which encompasses iron uptake, storage, utilisation and efflux. Iron uptake is primarily achieved through endocytosis mediated by transferrin receptor 1 (TFRC/CD71). Once iron enters the cell, a portion is utilised for the synthesis of iron-containing proteins (such as haemoglobin and cytochromes), whilst the remainder is stored in a non-toxic form within ferritin, which consists of a heavy chain (FTPH) and a light chain (FTPL). When the cell requires iron, ferritin is degraded via ferritinophagy to release iron ions; this process is mediated by nuclear co-activator 4 (NCOA4) [10]. Iron efflux is facilitated by iron transporter 1 (FPN1/SLC40A1). During ferroptosis, this equilibrium is disrupted. Various stimuli can upregulate TFRC expression, enhancing iron uptake; simultaneously, NCOA4-mediated ferritinophagy is activated, leading to ferritin degradation and the massive release of iron ions. These events collectively result in a dramatic expansion of intracellular LIP, providing ample substrates for the Fenton reaction, thereby initiating and amplifying the ferroptotic signal [11].

2.2 Uncontrolled Lipid Peroxidation

Lipid peroxidation is the most critical biochemical event in ferroptosis. Cell membranes, particularly those rich in polyunsaturated fatty acids (PUFAs) such as arachidonic acid (AA) and adrenic acid (AdA), are highly susceptible to attack by ROS. Under the catalysis of iron ions, ROS oxidise the side chains of PUFAs to form lipid radicals (L·), which can further attack neighbouring PUFA molecules, triggering a chain reaction that generates large amounts of lipid peroxides (LPO) and reactive lipid aldehydes (such as MDA and 4-HNE) [2]. The accumulation of LPOs severely disrupts the fluidity and integrity of cell membranes (including organelle membranes), leading to the formation of membrane pores, the collapse of ion gradients and the leakage of cellular contents, ultimately triggering cell death. Studies have found that cytosolic phospholipase A2 (cPLA2) and acyl-CoA synthase long-chain family member 4 (ACSL4) play the role of 'igniters' in ferroptosis. ACSL4 is responsible for activating free PUFAs in the cytoplasm into PUFA-CoA, which serves as a substrate for the synthesis of PUFA-containing phospholipids. Inhibiting ACSL4 expression significantly reduces the content of PUFAs in the membrane, thereby effectively suppressing the occurrence of lipid peroxidation and ferroptosis. Consequently, the content of PUFA-PLs (i. e. phospholipids containing PUFAs) is one of the key factors determining a cell's susceptibility to ferroptosis.

2.3 Collapse of the Antioxidant Defence System

Under normal physiological conditions, cells possess a robust antioxidant defence system to scavenge ROS and repair lipid peroxidation damage, thereby preventing the onset of ferroptosis. Among these, the cysteine/glutamate antiporter (System Xc^-), glutathione (GSH) and glutathione peroxidase 4 (GPX4) constitute the core defence line against ferroptosis. System Xc^- is a heterodimer composed of two subunits, SLC7A11 and SLC3A2, responsible for exchanging intracellular glutamate with extracellular cysteine. Cysteine is an essential precursor for the synthesis of GSH. GSH is the most important non-enzymatic antioxidant within the cell; acting as a coenzyme for GPX4, it participates in the catalytic reduction of lipid peroxides, converting them into non-toxic hydroxy compounds, thereby protecting the cell from damage caused by lipid peroxidation [12]. Consequently, disruption at any stage may trigger ferroptosis.

3. Mechanisms of Ferroptosis in Diabetic Cataracts

3.1 Direct Evidence That a Hyperglycaemic Environment Induces Ferroptosis in Lens Epithelial Cells

Multiple studies across various models have directly confirmed that hyperglycaemia can induce ferroptosis in LECs. In vitro, treatment of human LEC lines (e.g., SRA01/04, HLE-B3) with high concentrations of glucose (e. g., 25–150 mM) to mimic diabetic conditions has revealed characteristic features of ferroptosis. These features include: a significant increase in intracellular Fe^{2+} and total iron content, a sharp rise in lipid peroxidation levels (as measured by MDA and the lipid peroxidation marker 4-HNE), and a significant decrease in GSH levels [1], [13]. Transmission electron microscopy (TEM) analysis is the 'gold standard' for identifying ferroptosis; in hyperglycaemically treated LECs, typical morphological changes such as reduced mitochondrial volume, increased membrane density, and reduced or absent cristae can be observed, which contrasts sharply with the condensed nuclei and intact mitochondria of apoptotic cells [1]. More importantly, the use of specific ferroptosis inhibitors, such as Fer-1 (Ferrostatin-1) and Lip-1 (Liproxstatin-1), effectively reverses high-glucose-induced cell death, lipid peroxidation and mitochondrial dysfunction, thereby functionally confirming the occurrence of ferroptosis [13].

3.2 The Role of Key Signalling Pathways in Regulating Ferroptosis

How hyperglycaemia triggers ferroptosis signalling in LECs, and the upstream molecular regulatory network involved, is currently a key focus of research. Studies indicate that multiple classical signalling pathways are activated by hyperglycaemia and ultimately converge at the core regulatory nodes of ferroptosis.

Firstly, the p53 pathway plays a dual role in ferroptosis in DCs. As the most important tumour suppressor, p53 has recently been found to regulate ferroptosis. In DCs, high glucose levels can induce the expression of p53. Activated p53 protein can bind directly to the promoter region of the SLC7A11 gene, thereby inhibiting its transcription. SLC7A11 is a key subunit of System Xc^-; its downregulation directly leads to impaired intracellular GSH synthesis, weakening the antioxidant capacity of GPX4 and consequently rendering cells highly susceptible to ferroptosis [14]. Studies have shown that emodin can lift the inhibition of SLC7A11 by directly binding to and inhibiting p53 activity, thereby protecting LECs from high-glucose-induced ferroptosis damage [14].

Secondly, the Nrf2/HO-1 pathway constitutes a core defence mechanism for cells against oxidative stress and ferroptosis. When cells are subjected to stimuli such as oxidative stress, Nrf2 dissociates from Keap1, enters the cell nucleus, and initiates the transcription of a series of downstream antioxidant enzyme genes, including haem oxygenase-1 (HO-1), NAD(P)H quinone oxidoreductase 1 (NQO1) and glutamate-cysteine ligase catalytic subunit (GCLC). HO-1 catalyzes the breakdown of haem into CO, Fe^{2+} and biliverdin, with Fe^{2+} being rapidly captured and stored by ferritin, thereby preventing iron overload. Consequently, activation of the Nrf2/HO-1 pathway can inhibit ferroptosis by enhancing antioxidant capacity and promoting iron storage. Studies have shown that polysaccharides (SSP) extracted from Sagittaria sagittifolia can activate the Nrf2 signalling pathway in LECs, upregulating the expression of proteins such as HO-1, GPX4 and ferritin, thereby simultaneously inhibiting apoptosis and ferroptosis in a high-glucose environment and exerting a protective effect [15]. Furthermore, certain synthetic small-molecule compounds, such as aryl piperazine, have also been shown to inhibit the development of DCs by activating the Nrf2/HO-1 pathway [16].

Finally, several newly discovered regulatory axes provide a more nuanced perspective on understanding ferroptosis in LECs. For instance, studies on the LCN2 (lipid carrier protein 2) pathway have revealed that high glucose levels significantly upregulate LCN2 expression in LECs. LCN2 is a secretory protein capable of binding and transporting iron carriers, internalising them into the cell, thereby increasing intracellular iron uptake and exacerbating ferroptosis. Knocking down LCN2 effectively protects LECs from high-glucose-induced ferroptosis and delays the progression of cataracts in diabetic rats [1]. Another study revealed the crucial role of the VDAC3-USP47 axis. Voltage-dependent anion channel 3 (VDAC3) is a protein located on the outer mitochondrial membrane that participates in the regulation of mitochondrial metabolism and ROS production. Under high-glucose conditions, the expression level of the deubiquitinating enzyme USP47 decreases, leading to the ubiquitination and degradation of VDAC3. The loss of VDAC3 disrupts mitochondrial function, leading to excessive ROS production and ultimately triggering ferroptosis. This finding directly links mitochondrial dysfunction to ferroptosis [7]. This suggests that hyperglycaemia may regulate the expression of ferroptosis-related genes by altering RNA modifications, thereby contributing to the development of DC [17].

3.3 Crosstalk Between Ferroptosis and Other Forms of Cell Death

Within the pathological microenvironment of DC, ferroptosis does not occur in isolation but is involved in complex crosstalk with other forms of cell death, particularly apoptosis. Apoptosis is a cell death process mediated by the caspase protease family, morphologically characterised by cellular shrinkage, chromatin condensation and the formation of apoptotic bodies. Although early studies suggested that apoptosis was the primary mechanism leading to a reduction in LEC numbers, subsequent findings indicate that apoptosis may not play a dominant role in DC and ARC [4]. Ferroptosis and apoptosis share common points of intersection in their signalling pathways. For example, the p53 protein can induce ferroptosis by inhibiting SLC7A11, whilst also inducing apoptosis by upregulating pro-apoptotic proteins such as Bax. Furthermore, certain apoptosis-related proteins, such as Caspase-3, may be activated during ferroptosis, although their precise role remains unclear. Studies have shown that inhibiting ferroptosis may simultaneously reduce the incidence of apoptosis [13], suggesting that the two processes may reside on different branches of the same death signalling pathway or may be mutually reinforcing. For instance, H_2O_2 and ultraviolet light (UVB) have been shown to induce both ferroptosis and apoptosis in LECs, suggesting that different stressors may activate distinct death programmes, or that both programmes may be activated simultaneously [18].

4. Intervening in Ferroptosis as a Therapeutic Strategy for Diabetic Cataracts

4.1 The Protective Effects of Ferroptosis Inhibitors

The use of specific ferroptosis inhibitors is a direct method for validating their effects in dendritic cells (DCs) and exploring their therapeutic potential. Fer-1, as the first ferroptosis inhibitor to be discovered, has demonstrated potent protective effects in numerous DC studies. For example, in studies on LCN2, Fer-1 was able to effectively inhibit ferroptosis induced by high glucose or LCN2 overexpression, thereby protecting the viability of liver endothelial cells (LECs) [1]. Similarly, in studies on Sagittaria sagittifolia polysaccharide (SSP), although SSP itself inhibits ferroptosis by activating the Nrf2 pathway, the use of Fer-1 as a positive control was also effective in alleviating high-glucose-induced cellular damage [13]. These studies provide strong evidence that directly blocking the ferroptosis pathway is a viable and effective approach in the treatment of DC. Although the clinical application of Fer-1 is limited by its unfavourable pharmacokinetic properties, it serves as an important lead compound and validation tool for the development of more potent and safer ferroptosis inhibitors.

4.2 The Potential of Natural Compounds and Active Ingredients from Traditional Chinese Medicine

Traditional Chinese medicine and natural products constitute a rich treasure trove for new drug discovery; many natural compounds, owing to their multi-targeted and low-toxicity characteristics, demonstrate immense potential in the prevention and treatment of DC. Recent studies have begun to re-evaluate the pharmacological effects of these compounds from the perspective of ferroptosis inhibition.

Gypenosides (GPs): These are the primary active components extracted from the traditional Chinese medicinal plant Gynostemma pentaphyllum and are known to possess various effects, including hypoglycaemic and antioxidant properties. A study combining network pharmacology and experimental validation demonstrated that GPs can significantly improve lens opacity in diabetic rats; this mechanism is associated with the regulation of the p53/SLC7A11/GPX4 signalling pathway and the inhibition of ferroptosis in LECs [15].

Astaxanthin (ATX): A carotenoid with potent antioxidant activity. Studies have found that ATX can upregulate GPX4 expression in LECs from aged mice, delaying age-induced ferroptosis and thereby preserving lens transparency [19]. In a diabetic environment, ATX has also been shown to mitigate oxidative stress damage by regulating GPX4 and ferroptosis [20].

4.3 Challenges and Prospects for Clinical Translation

Although therapeutic strategies targeting ferroptosis have yielded encouraging results in animal models of DC, their translation into clinical practice still faces numerous challenges. Firstly, the pathological mechanisms of DC are extremely complex, and ferroptosis is merely one important component. Future therapeutic strategies may require combination therapy, simultaneously targeting multiple pathological pathways such as ferroptosis, oxidative stress and glycation, in order to achieve optimal efficacy. Secondly, most of the drugs (e. g. Fer-1) and natural compounds (e. g. GPs) currently under investigation suffer from issues such as low bioavailability and poor intraocular permeability; suitable drug delivery systems (e. g. nanocarriers, ophthalmic formulations) need to be developed to enhance their concentration and efficacy in target tissues [23]. Developing therapeutic regimens capable of efficient and stable intraocular release by integrating modern drug delivery technologies. As these issues are progressively resolved, targeting ferroptosis is expected to open up entirely new avenues for the pharmacological prevention and treatment of diabetic cataracts.

5. Conclusion

Diabetic cataract (DC), a highly blinding complication of diabetes, has a complex pathogenesis that remains incompletely understood. Although traditional pathophysiological theories—such as oxidative stress, the accumulation of advanced glycation end products (AGEs) and the activation of the polyol pathway—provide a foundational framework for understanding DC, they struggle to fully account for its unique clinical features and rapid progression. This review systematically elucidates how ferroptosis, as a novel, iron-dependent form of programmed cell death, plays a crucial role in the development of DC, offering a fresh perspective on the pathological mechanisms of the condition.

Synthesising the existing research evidence, we can draw the following core conclusions: Firstly, ferroptosis is a key and independent mechanism underlying the damage and death of lens epithelial cells (LECs) in a hyperglycaemic environment, accompanied by intracellular iron overload, massive accumulation of lipid peroxides, and the collapse of key antioxidant defence systems (particularly the GPX4-SLC7A11 axis). Secondly, hyperglycaemia induces ferroptosis by activating multiple signalling pathways, including upregulating p53 expression to inhibit System Xc^-, suppressing the Nrf2/HO-1 antioxidant pathway, and exacerbating oxidative stress and iron metabolism imbalance through the regulation of newly identified molecular axes such as LCN2 and VDAC3-USP47. Finally, targeted inhibition of ferroptosis pathways, whether through specific inhibitors or natural compounds with antioxidant activity, has been shown to effectively protect LECs and delay lens opacification, opening up promising new avenues for the prevention and treatment of DC.

Looking ahead, targeted ferroptosis therapies hold immense potential for clinical intervention in DC, yet they are accompanied by challenges. Future research needs to explore in greater depth the fine-tuned regulatory networks of ferroptosis in DC and its complex crosstalk with other forms of cell death, whilst also focusing on the development of ferroptosis inhibitors that can be efficiently and specifically delivered to the eye. Through continued in-depth research in this emerging field, we hope to provide DC patients with effective pharmacological preventive and therapeutic options that go beyond traditional surgery, thereby better preserving and restoring their vision.

References

[1] Jiayue Zhang, Liyao Sun, Xiaohan Yu, Chen Yang, Qi An, Chaoqun Wei, and Hongyan Ge. “LCN2 Aggravates Diabetic Cataracts by Promoting Ferroptosis in Lens Epithelial Cells”, Frigid Zone Medicine 4. 3 (2024): 177-192.

[2] Guo Z, Ma X, Zhang R X, et al. Oxidative stress, epigenetic regulation and pathological processes of lens epithelial cells underlying diabetic cataract[J]. Advances in ophthalmology practice and research, 2023, 3(4): 180-186.

[3] Giglio R, Milan S, Inferrera L, et al. Nutrient-driven antioxidant interventions for prevention of age-related and diabetic cataracts[J]. Nutrients, 2025, 17(11): 1885.

[4] Zongbo Wei, Caili Hao, Jingru Huangfu, Ramkumar Srinivasagan, Xiang Zhang, and Xingjun Fan. “Aging Lens Epithelium is Susceptible to Ferroptosis. “, Free Radical Biology and Medicine 167 (2021): 94-108.

[5] ZHANG Yaoxia, and ZHOU Mingsheng. “Ferroptosis and its role in diabetic complication”, Journal of Shenyang Medical College 24. 2 (2022): 113-117.

[6] YOU Ziyi, and YANG Lina. “Research progress in the relationship between ferroptosis and diabetic

microvascular complications", Chinese Journal of Diabetes 31. 7 (2023).
[7] Haoran Xu, Wenjing Wang, Jianqiao Li, Xiaofei Zhao, and Han Zhang. "Methodological Approaches for Dissecting Ferroptosis in Lens Epithelial Cells and Its Role in Diabetic Cataract Progression.", Methods (San Diego, Calif) (2026).
[8] Liu Kexin, Li Huazhang, Wang Feng, and Su Ying. "Ferroptosis: Mechanisms and Advances in Ocular Diseases", Molecular and Cellular Biochemistry 478. 9 (2023): 2081-2095.
[9] Michael S. Petronek, Douglas R. Spitz, Garry R. Buettner, and Bryan G. Allen. "Linking Cancer Metabolic Dysfunction and Genetic Instability Through the Lens of Iron Metabolism", Cancers 11. 8 (2019).
[10] Jiahui He, Zhangwang Li, Panpan Xia, Ao Shi, Xinxi FuChen, Jing Zhang, and Peng Yu. "Ferroptosis and Ferritinophagy in Diabetes Complications. ", Molecular metabolism 60 (2022).
[11] Feiyin Sheng, Yuzhou Gu, Shengjie Hao, Ye Liu, Shuying Chen, Bing Lu, Lu Chen, Wei Zhao, Di Wu, Yili Xu, Rongrong Chen, Yu Han, Xiaoming Lou, Xiaofeng Wang, Zhijian Chen, Ke Yao, and Qiuli Fu. "Ferroptosis is Involved in the Damage of Ocular Lens under Long-Term PM2. 5 Exposure in Rat Models and Humans", Ecotoxicology and environmental safety 288 (2024): 117397.
[12] Zongbo Wei, Caili Hao, Kazi Rafsan Radeen, Zheng Hao, Kavitha Kettimuthu, Kristal Maner-Smith, Shinya Toyokuni, and Xingjun Fan. "Deficiency in Glutathione Peroxidase 4 (GPX4) Results in Abnormal Lens Development and Newborn Cataract. ", Proceedings of the National Academy of Sciences of the United States of America 121. 48 (2024).
[13] Man-Yu Zhou, Yang Jiang, Jia-Zhen Ding, Yao Liang, Jian-Bin Sun, Bing-Qing Liu, and Yan Liao. "Sagittaria Sagittifolia Polysaccharide Regulates Nrf2-mediated Antioxidant to Improve Apoptosis and Ferroptosis in High Glucose-Induced Lens Epithelial Cells", Experimental Cell Research 454. 2 (2025): 114841.
[14] Xiangrong Zuo, Xiuxian Wang, Jing Xie, and Yuhong Jia. "Emodin Alleviates the Damage to Lens Epithelial Cells in Diabetic Cataract by Repressing the P53-Mediated Ferroptosis Pathway", International Ophthalmology 45. 1 (2025): 1-15.
[15] Anqi Pei, Ying Ma, Mingjun Gao, and Qi Zhao. "Protective Efficacy and Mechanism of Gypenosides Against Diabetic Cataracts Via the Ferroptosis Pathway: A Study Based on Network Pharmacology. ", European Journal of Pharmacology 1007 (2025): 178175.
[16] Xiu Wang, Congcong Tian, and Chunning Zhao. "Aryl Piperazine Suppresses Cataractogenesis in a Diabetic Rat Model Via Nrf2/HO-1 Signaling Pathway", Tropical Journal of Pharmaceutical Research 19. 11 (2020): 2317-2322.
[17] Lei Cai, Xiao-Yan Han, Dan Li, Dong-Mei Ma, Yu-Meng Shi, Yi Lu, and Jin Yang. "Analysis of N6-methyladenosine-modified Mrnas in Diabetic Cataract", World journal of diabetes 14. 7 (2023): 1077-1090.
[18] JING Ruihua, QIAO Xinli, and XUE Fei. "Ferroptosis of lens epithelial cells induced by oxidative stress injury of cata-ract patients", Recent Advances in Ophthalmology 43. 4 (2023).
[19] Yong Wang, Pengfei Li, Congyu Wang, Sijie Bao, Siwen Wang, Guowei Zhang, Xi Zou, Jian Wu, Yu Guan, Min Ji, and Huaijin Guan. "Lens Epithelium Cell Ferroptosis Mediated by M 6 A-lncRNA and GPX4 Expression in Lens Tissue of Age-Related Cataract", BMC ophthalmology 23. 1 (2023).
[20] Deqian Kong, Yue Liu, Li, Huajun Wang, Ke Li, and Guangying Zheng. "Astaxanthin Ameliorates Oxidative Stress in Lens Epithelial Cells by Regulating GPX4 and Ferroptosis. ", Chemico-Biological Interactions 383 (2023).
[21] CHEN Lihua, LI Aiyan, WU Xiaotao, and LING Ziyun. "Protective effect of total flavonoids from Rosa on lens in diabetic cataract rats and its mechanism", China Medicine and Pharmacy 12. 11 (2022): 80-83.
[22] CUI Cui, ZHAO Junbo, WANG Min, HAN You, and LI Jiajia. "Influence of pterostilbene on oxidative stress and inflammatory reaction in diabetic cataract rats and its mechanism", Shandong Medical Journal 62. 31 (2022).
[23] Bowen Wang, Hao Zeng, Xuan Zhao, Xin Zuo, Xue Yang, Li Wang, and Jin Yuan. "D609-polymer-based Delivery Strategy Targeting Ferroptosis in Treatment of Dry Eye Disease", Chemical Engineering Journal 503 (2025).