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Comparative study of common methods for vision restoration

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Abstract:

Vision impairment is a major global health issue that affects more than two billion people and causes large economic losses. Therapies for it have been developed during the past few decades. This research summarizes common methods for vision restoration. Invasive methods include gene therapy, cell therapy, and retinal prostheses. They often show stronger effects while having problems like high cost and safety concerns. Non-invasive methods include electromagnetic stimulation and drug delivery. These approaches are generally safer, cheaper, and easier for patients to accept, but their efficiency is usually limited, especially in late stages. The paper reviews the mechanism of each method, evidence of effectiveness, remaining challenges, and potential improvements in the future. It highlights that no single method can fully restore natural vision; combining approaches may offer better solutions in the future. It also stresses the importance of comparing these methods to guide future research. By doing this, the paper provides a clear picture of current progress and possible directions for vision restoration.

Keywords: Vision restoration; Retinal degeneration; Invasive; non-Invasive.

1. Introduction

People perceive the world through their senses, including hearing, vision, touch, taste, and smell. Losing any of the senses will cause problems. Harm and loss of vision specifically will make people lose an important way to interact with people and the world. For example, it will cause people not be unable to see indications for danger and cause inconvenience in everyday life. Loss of sight is now a worldwide problem. According to the World Health Organization, at least 2.2 billion people have a vision impairment. Vision impairment is causing 411$ billion in losses in global productivity[1]. There is an urgency for efficient treatments.

Treatment of vision impairment is hard to solve because there are various causes, including age-related macular degeneration, retinitis pigmentosa, glaucoma, diabetic retinopathy, traumatic injuries, macular degeneration, cataract, and so on. Each cause may require a different method to detect and resolve. Over the past three decades, research into vision restoration has expanded from preventive care and supportive devices to active biological and technological

repair of the visual system. Strategies can be broadly classified into invasive methods—involving surgical intervention or direct modification of ocular or neural tissue—and non-invasive methods, which rely on external stimulation or systemic treatment without major surgery.

The common methods used can be generally classified as invasive and non-invasive methods. Invasive methods require the injection of one or more kinds of biocompatible chemicals or electrical, mechanical implants. Major invasive methods include gene therapy, cell therapy, and retinal prostheses. Gene therapy delivers functional genetic material to replace or repair defective genes. Cell therapy transplants retinal pigment epithelial (RPE) cells, photoreceptors, or stem cell–derived tissues to cure or replace harmed cells. Retinal prostheses are electronic devices that bypass damaged photoreceptors and stimulate remaining retinal neurons.

Non-invasive means the therapy doesn't require implants into the body, so it is generally safer, cheaper, and easier for patients to accept. It is broadly used on early-stage patients, which accounts for a large proportion of all patients. However, non-invasive methods are often less effective, especially on late-stage patients. Non-invasive methods also may require a longer time of intervention before the effect shows, though invasive methods, generally faster in effect, may require months of recovery due to the implant. Common non-invasive methods include electromagnetic stimulation and drug delivery. Electromagnetic stimulation uses electrical or magnetic stimulation to modulate retinal or cortical activity. Drug delivery means pharmacological interventions via topical, systemic, or targeted intraocular release systems.

Although each method has demonstrated some degree of clinical success, no single approach fully restores natural vision. Existing reviews don't analyze all major methods comparatively, leaving a gap for integrated analysis that examines biological efficacy, safety, cost, and patient experience. This review introduces and compares common methods from principles, applications, limitations, safety, efficacy, cost, patient experience, future challenges, and directions.

2. Common methods

2.1 Gene Therapy

Gene Therapy includes gene-replacement therapy, genome editing, and optogenetic therapy. The article will introduce and analyze the three therapies in order, provide ongoing examples, and propose possible future improvement directions.

2.1.1 Gene Replacement Therapy

Gene-replacement therapy delivers a healthy copy of a mutated gene by delivery factors into the nucleus of the host cell, enabling further modulation by promoters and enhancers. For retinal diseases, AAV vectors are used most frequently due to their high tropism for the photoreceptors, though their limited cargo capacity (5.7kb) requires customized delivery strategies like using dual AAV vectors or alternative viral vectors. AAV-based gene therapy drug LUXTURNA received the FDA (2017) and the EMA (2018) approval for RPE65-related retinal disease 20 years after its first successful experiment[2]. This approval is meaningful for two reasons: first, it used a multi-luminance mobility test instead of traditional acuity tests for the trial endpoint; second, the effect of therapy was shown in a relatively short period, saving time for proving the slowing of degeneration. A test based on VR showed positive effects, like reducing at least 5 log units of dark-adapted sensitivities by full-field sensitivity test (FST), and all test subjects finished the maze independently after surgery[3]. Although it is a monumental advance in gene therapy, most studies failed to prove its efficiency. It is also priced at 850000$, which is far too expensive for most patients. It is also shown that when including social costs, the actual price even exceeds 850000$[4]. The safety concerns are also evident. Over a long term, 27 out of 41 (66%) patients met one or more ocular problems, which is mostly due to the surgical procedure instead of the drug[4]. This still makes taking the therapy undesirable. Furthermore, this can only treat RPE65 gene mutation, which is just one out of more than 220 genes that may lead to inherited retinal dystrophies, 2% cases out of all reasons. AAV-based gene replacement cannot handle many retinal diseases, so endogenous gene editing may be more useful. Also, Optogenetic therapy is often a better solution for late-stage situations[2]. To sum up, gene replacement is a possible and valuable solution, but it is still developing and has various drawbacks. New solutions for other forms of inherited retinal dystrophy, including choroideremia, X-linked retinitis pigmentosa, Leber hereditary optic neuropathy, X-linked retinoschisis, and achromatopsia, are in progress and may lead to new advances and provide a better solution to patients[2].

2.1.2 Gene Editing Therapy

Genome editing directly modifies patients' own copy of gene using genome editing instead of injecting a healthy copy. Techniques like CRISPR-Cas9 show effects on modulating pathogenic missense changes in the genome or silencing a disease-causing gene, which traditional gene replacement has found challenging to solve[2]. Also, modulating a mutated gene is often more natural since

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the gene undergoes normal transcriptional regulation and epigenetic environment. CRISPR-Cas (the most commonly used genome editing technique) utilizes carefully designed and screened RNA-guided endonuclease. The endonuclease is then delivered into the target cell, and it will and only will cut the target DNA with a specific sequence of the genome. This triggers the DNA repair mechanism of the cell: homology-directed repair or non-homologous. Homology-directed repair can be controlled by inserting a specific DNA sequence, enabling modulations to be predictable. This makes precise human intervention to correct a genome mutation possible. For non-homologous repair, it may introduce random genome sequence to repair the gap, thus leading to stop codons or frameshift that creates a malfunctioning protein. If the RNA endonuclease only targets the mutated allele in a dominant inherited disease, it can help inhibit the mutated part[2]. EDIT-101 is a representative gene editing therapy approved by the FDA (2018) that's based on CRISPR-Cas-9 and targets CEP-290-related inherited retinal degeneration, which leads to vision loss during early ages. An in vivo study of 12 adult participants and two children (ages 9 and 14) with CEP-290 showed that 6 (42%) participants had a meaningful improvement from baseline cone-mediated vision as assessed with the use of FST. This study also addresses the safety issues of EDIT-101 since no serious adverse events or dose-limiting toxic effects were recorded after taking EDIT-101[5]. However, several problems are left to be resolved before further applications become clinically accessible. First, the delivery of RNA guides to most systems exceeds the capability of AAV vectors (a common and reliable vector used)[2]. This is important for on-target therapy and safety issues related to off-target effects, which is important to cure further diseases. Another thing is that although CRISPR-Cas9 is a commonly used gene editing method, it still has some safety concerns and potential long-term effects of genome chromosomal translocations. Therefore, further research and studies can improve the method used to improve safety, efficacy, and cost since current methods still have not completely solved these problems.

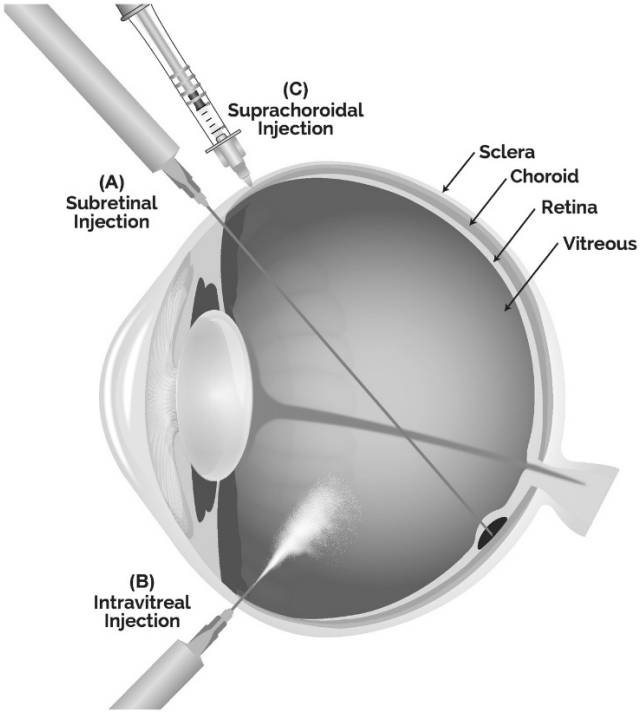

Fig. 1 Routes of injection of vectors in optogenetic therapy through a microneedle[6].

2.1.3 Optogenetic Therapy

Optogenetic therapy, compared to gene-replacement therapy and genome editing therapy, can be used to treat late-stage retinal degeneration. Optogenetic therapy introduces light-sensitive proteins into non-light-sensitive neurons, introducing the ability to respond to light signals. Ideally, an optogenetic tool should work at natural light intensity and be safe for the human body, and the kinetics should be around the level of natural retinal opsins to avoid further harm to the patient's eye. Currently, there are generally two types of optogenetic tools: microbial-based opsins based on light-gated ion channels, and human opsins and genetically engineered photo switches activating local receptors or ion channels. A study has shown partial recovery of vision in primates and one human participant[7]. However, as a developing technique, optogenetic therapy has concerns about adverse immune responses caused by microbial opsins. Microbial opsins also require light out of the physiological scope, which may be risky and requires an externally implanted device as a light source that is potentially dangerous for the patient. The method based on human opsins, in contrast, is generally safer since it is natural to the human body, avoiding adverse immune responses. It only requires less intense light that is inside the scope of physiological light, safer for the patient. Genetically engineering photo switches has also been devel-

oped in recent years and improved. But currently it still uses ultraviolet light for excitation, which is potentially dangerous and may raise problems. Exploring safer light sources can be a future direction for improvement. Another important aspect is the delivery method of optogene. As shown in Fig. 1, Potential methods for delivery include intravitreal, subretinal, suprachoroidal, and sub-inner-limiting-membrane administration of viral vectors. Intravitreal delivery is technically less challenging and may have more widespread retinal gene expression, though it is likely to require higher doses in humans compared to primates and mice[2]. The potential of high doses of vector may cause inflammation intraocular, limiting the application of intravitreal delivery. For subretinal gene delivery, it directly contacts high viral concentration with the target organ at a lower dosage, avoiding potential risk related to high dosage. One drawback of subretinal delivery is that it requires temporary retinal detachment, which may still be risky.

2.2 Gene Therapy

There are two main strategies for cell therapy: paracrine therapy and cell-replacement therapy. Paracrine therapy excites existing cells' function, while cell-replacement therapy directly replaces dead cells with embryonic stem cells.

2.2.1 Paracrine Therapy

Paracrine therapy is based on the concept that transplanted cells excite existing photoreceptor cells' viability by releasing diffusible chemical factors. This method should be applied before the onset of widespread retinal atrophy to be effective. Paracrine actions require diffusible factors to take effect. Therefore, treatment often takes place on a site some distance from diseased cells. Clinical-stage paracrine strategies employ mesenchymal-derived cells, such as umbilical tissue or bone marrow cells. When delivered intraocularly, these cells may exert effects through the release of trophic factors that support retinal cell survival. Experimental evidence suggests they may take effect by attenuating glial activation, preserving synaptic integrity, and promoting neurite growth in degenerating retinae. Mesenchymal cells are also known to secrete extracellular vesicles, which may contribute to immune modulation and molecular signaling, though their precise role in retinal therapy remains to be fully elucidated[2].

2.2.2 Cell-replacement Therapy

Cell-replacement therapy replaces either RPE or photoreceptors. It replaces diseased native RPE cells obtained from human embryonic stem cells (hESCs) into the subretinal space at or around damaged sites. RPE cells itself doesn't individually react to light; they don't mediate visual functions directly. Instead, it supports photoreceptor cells with nutritional and metabolic support. If RPE completely dies and photoreceptors degenerates, additional photoreceptors transplant is required[2].

To date, no cell-based therapeutic approach has yet demonstrated definitive clinical efficacy. Nonetheless, a growing number of clinical trials are trying diverse strategies, encompassing different surgical techniques, cell lineages, and immunosuppressive regimens. While these early-phase studies have generally reported acceptable safety profiles, robust evidence regarding both therapeutic benefit and long-term outcomes remains insufficient. Despite the absence of regulatory approval or confirmatory efficacy data, certain commercial "cell therapy" providers continue to promote non-FDA-approved interventions directly to patients, which may cause severe adverse events like irreversible vision loss[8].

2.3 Gene Therapy

Retinal prostheses are implantable biomedical devices designed to substitute degenerated photoreceptors. Retinal prostheses stimulate the residual retinal neural circuitry of human cells. It aims to restore vision of patients with severe photoreceptor loss by converting external visual input into patterned electrical activity that can be understood by normal cells.

During the 18th century, early experiments demonstrated that residual retinal neurons could still generate visual images, providing the theoretical support for retinal prostheses. But further development was restricted by the level of technology. Therefore, the idea of retinal prostheses came into reality by the mid-20th century. In 1956, Tassicker implanted a subretinal selenium-coated disc in rabbits. He realized that damaged photoreceptors can still be stimulated to produce visual percepts called "phosphenes"[9]. With subsequent progress in electronics, biomaterials, and microsurgical methods, clinical prototypes became feasible. In the early 2000s, Argus I, having 16 electrodes, helped blind patients detect simple motion and light patterns.

Currently, three devices have been legally approved: Argus II, Alpha IMS/AMS, and IRIS II. They introduced higher electrode counts and various methods of placements, although the most effective approach is left unknown[9]. Clinical trials demonstrated partial restoration of vision, including improvements in tasks such as object localization, navigation, and recognition of simple patterns. Preclinical work in animal models established safety parameters and optimized surgical techniques.

Despite achieving regulatory milestones, none of the

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three devices remains commercially available now. Clinical outcomes confirmed that patients could improve in mobility and daily living tasks. Nevertheless, all three devices ceased production in 2017, largely due to stricter regulations on innovation, high development and surgical costs, and limited product performance[9]. The market size is limited compared to cochlear implants, further limiting research and commercialization. Currently, only two groups—one in Japan (developing a 49-channel STS system) and another in Australia (pursuing a suprachoroidal approach)—continue clinical trials and aim for commercialization[9].

Retinal prostheses face persistent biological, technological, and economic obstacles. Retinal remodeling in degenerative diseases such as retinitis pigmentosa alters neural architecture, leading to unpredictable phosphene perception and complicated stimulation effects. Limited electrode density restricts resolution and visual field coverage. The high cost of devices further narrows patients' accessibility. Despite the challenges, opportunities for innovations still remain. Advances in image-processing algorithms like facial recognition can improve functional outcomes. Improvements in materials, wireless power delivery, and minimally invasive surgical techniques can increase the safety and durability of the device. Furthermore, withdrawal of earlier commercial players reduces competition, opening up market for new research groups or companies. Retinal prostheses may also complement emerging gene and cell therapies to create a new treatment pathway for late-stage disease when regenerative approaches alone are insufficient. Although economic and technical challenges remain, the combination of technical innovation and interdisciplinary research pursuits for more effective and accessible vision restoration solutions.

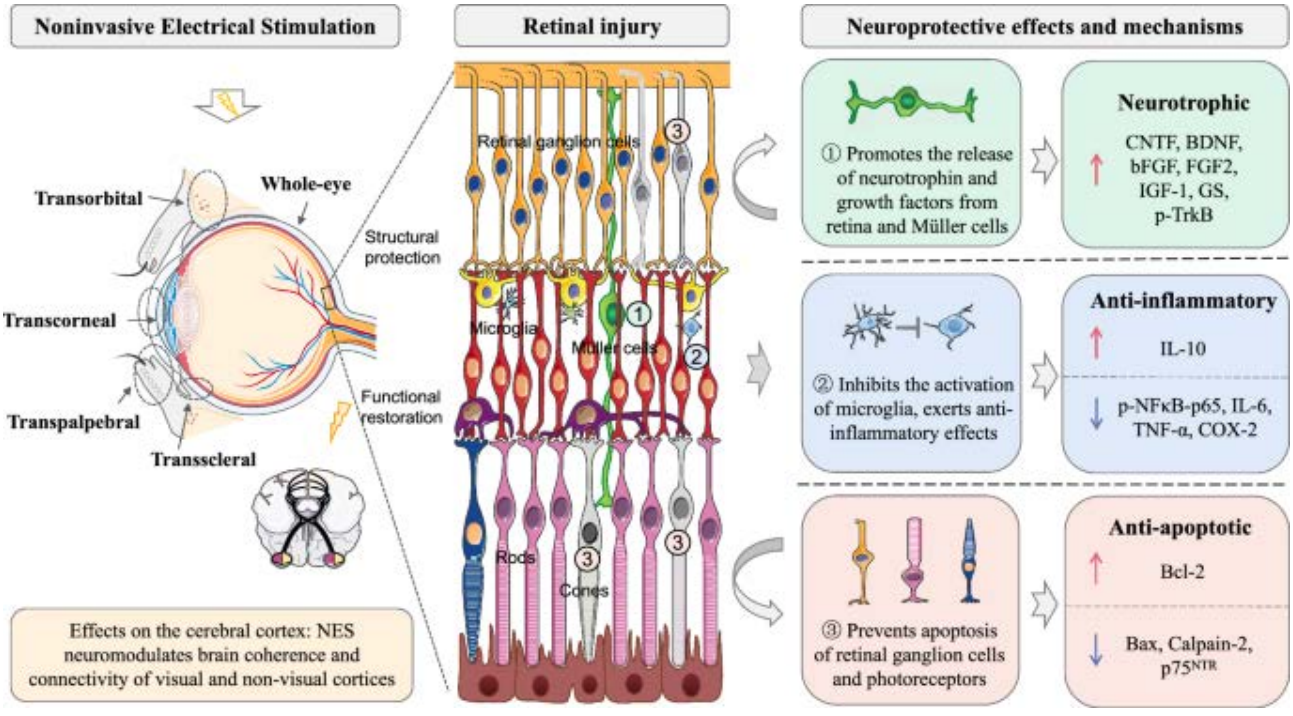

Fig. 2 An overview of the cellular structure of the retina[10].

2.4 Electromagnetic Stimulation

Electromagnetic stimulation, or non-invasive electromagnetic stimulation (NES), is also based on the fact that electrical stimulation can create a visual image in the brain. It differs from retinal prostheses by its non-invasiveness. It uses weak currents of less than 4mA to modulate neural activity. This current reduces the membrane potential, resulting in hyperpolarization or depolarization, which depends on the direction of the current relative to axonal orientation. This mechanism is shown on Fig. 2 above. Researchers used various methods to achieve this, including transcorneal electrical stimulation, transpalpebral stimulation, and high-frequency transcranial random noise stimulation. These techniques aim to boost residual visual function by promoting neuroplasticity, enhancing retinal perfusion, or reactivating partially preserved neuronal networks.

The first reported attempt to treat visual impairment with current stimulation was by Shinoda and her colleagues in 2008. They demonstrated that patients with age-related macular degeneration (AMD) improved their vision after transcorneal electrical stimulation. Following this, numer-

ous studies started applying NES on other diseases such as retinitis pigmentosa, glaucoma, retinal artery occlusion, and optic neuropathies. Investigations indicated that electrical currents could influence neuronal excitability, synaptic transmission, and oscillatory brain activity, leading an improvement in visual acuity, visual fields, contrast sensitivity, and color discrimination.

As techniques advanced, researchers classified common stimulations according to anatomical targets: pre-chiasmatic stimulation (e.g., tcES, tpES, rtACS) for retinal and optic nerve disorders, and post-chiasmatic stimulation (hf-tRNS, tDCS) for cortical visual conditions such as amblyopia or hemianopia. Over time, safety data accumulated, confirming that adverse events were generally mild and transient, including tingling, mild headache, or ocular surface irritation.

Systematic reviews and meta-analyses of NES applications reveal that evidence levels vary with techniques and pathologies. According to Perin et al., rtACS currently holds the strongest clinical evidence, reaching a Level A recommendation for certain optic nerve disorders, while tcES has achieved Level B evidence for retinitis pigmentosa. In contrast, hf-tRNS and tDCS are supported only by Level C evidence and thus may be less reliable[11].

Clinical trials report that rtACS can induce significant visual field improvements, in some cases persisting for months after treatment. For example, randomized controlled studies involving patients with glaucoma and ischemic optic neuropathy demonstrated mean visual field improvements of around 20–25% compared to minimal changes in sham groups. TcES has shown promise in enhancing retinal blood flow and preserving photoreceptor function, though results remain inconsistent. Post-chiasmatic protocols such as tDCS and hf-tRNS have been used to complement rehabilitative training for amblyopia and hemianopia, showing potential to accelerate recovery of visual fields or stereopsis.

Despite promising outcomes, NES doesn't have standardized guidelines for clinical implementation, and is not yet widely adopted in routine ophthalmological practice. Nevertheless, the relatively strong safety profile, the non-invasive nature of the techniques, and the absence of severe adverse events across more than 1,000 patients suggest NES is a feasible adjunctive strategy in vision rehabilitation[11].

The future of electromagnetic stimulation in vision recovery faces several important challenges. First, a standardized and widely accepted protocol is urgently needed: differences in electrodes, current amplitude, frequency, and duration make it difficult to compare results across studies. Second, heterogeneity in patients and disease stages causes variable outcomes. Thus, A more stratified trial design should be conducted to ensure. Third, the mechanisms of action remain incompletely understood. While residual vision activation theory suggests that NES reactivates dead photoreceptors and promotes neuroplasticity, the underlying mechanism between stimulation and functional outcomes remains unknown.

Opportunities lie in combining ES with other therapeutic strategies. Pairing NES with visual training appears to enhance treatment efficacy[11]. Integration with pharmacological or gene-based therapies could also offer benefits. Advances in personalized treatment may enable portable, patient-specific stimulation protocols, increasing accessibility. Furthermore, future studies leveraging neuroimaging and machine learning could refine biomarker-based assessments, providing a better comparison between results of different studies.

In summary, electromagnetic stimulation represents a promising, safe, and adaptable modality for vision restoration. While rtACS and tcES demonstrate the most encouraging evidence, further large-scale randomized controlled trials, mechanistic investigations, and standardized clinical protocols are essential before NES can be considered a mainstream therapeutic option.

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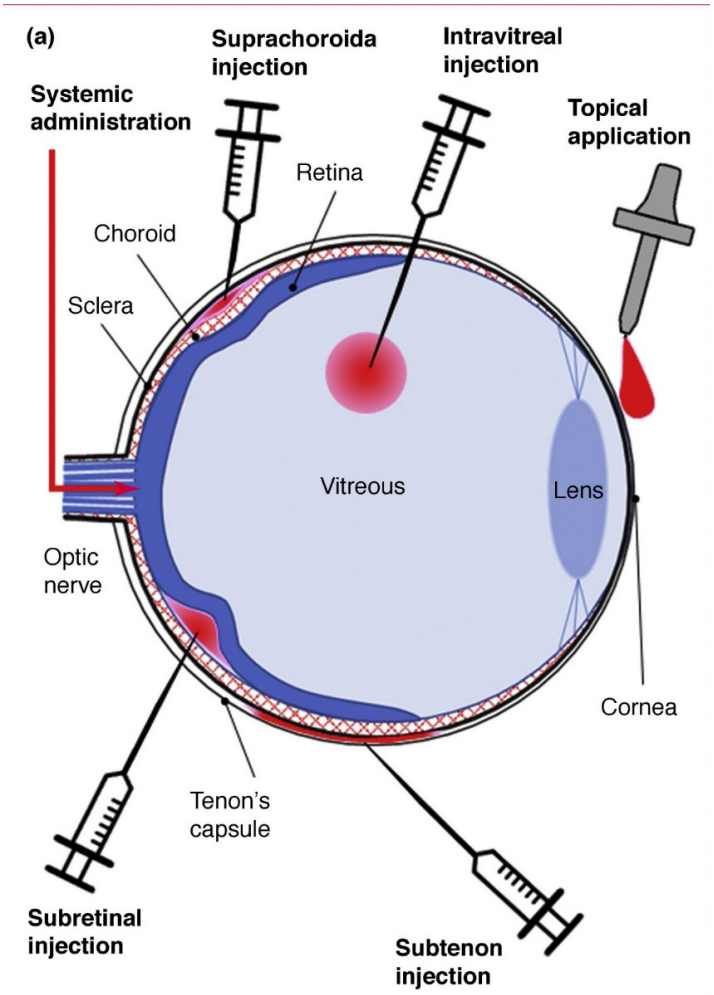

Fig. 3 Routes of administration for drugs targeted to the photoreceptors of the retina[12].

2.5 Drug delivery

Drug delivery directly injects therapeutic compounds into the highly vulnerable photoreceptor cells. A central barrier to effective therapy is the blood–retinal barrier (BRB), which is analogous to the blood–brain barrier and restricts access of circulating molecules to the neuroretina. While this barrier protects retinal tissue from toxins, it complicates pharmacological intervention. Consequently, retinal drug delivery has various routes of administration to inject drugs, including systemic administration with specialized carriers, local ocular injections, topical applications, and advanced formulations such as nanoparticles and liposomes, as shown in Fig. 3.

Initial approaches to retinal pharmacotherapy largely involved systemic administration of small molecules. However, the recognition that the BRB severely limits penetration to the posterior eye segment led to the development of local administration methods. Early intravitreal injections demonstrated that direct delivery could bypass vascular barriers, but these carried risks of intraocular inflammation and infection. Subretinal injections, later established as the preferred route for gene therapy, provided localized access to photoreceptors but also induced retinal detachment, limiting their use for chronic drug delivery.

Over time, alternative routes such as subtenon and suprachoroidal injections were explored to provide more targeted yet less invasive delivery. Suprachoroidal injections, in particular, emerged as a promising compromise by depositing drugs close to the choroid and retina while minimizing intraocular trauma. Parallel advances in material sciences introduced nanoparticles and liposomal carriers, which not only protect drugs from degradation but also enable controlled release and improved tissue penetration[12]. These innovations expanded the possibilities of delivering both small molecules and macromolecules to photoreceptor cells.

Today, retinal drug delivery research is characterized by the diversification of administration routes and drug delivery systems (DDS). Intravitreal injection remains the most

widely used method for anti-VEGF therapies in AMD and diabetic macular edema, despite its associated risks. Subretinal injection is reserved for gene therapy and certain experimental protein deliveries, while suprachoroidal injections are being tested clinically for corticosteroids and nanoparticle formulations. Subtenon administration offers an intermediate approach, though its efficacy for posterior segment diseases remains under evaluation[12].

Nanoparticle-based DDS are central to current innovation. Polymeric nanoparticles, solid lipid nanoparticles, niosomes, and liposomes have been engineered to enhance stability, sustain release, and target specific retinal layers. For example, light-responsive polymeric nanoparticles can release cargo in response to external triggers, maintaining functionality up to 30 weeks post-injection. Cyclodextrin-based nanoparticles improve solubility and prolong retention on the ocular surface, thereby increasing bioavailability. Furthermore, liposomes have demonstrated the ability to deliver both small molecules and recombinant proteins directly to photoreceptors, illustrating their versatility.

Topical administration remains an appealing but challenging strategy. Barriers such as tear turnover, corneal and conjunctival lipophilicity, and limited contact time restrict posterior segment delivery. Nonetheless, solubilizing nanoparticles and chemical penetration enhancers are under development to overcome these obstacles. Rabbit studies suggest that the scleral route may be more effective than the corneal route for certain drugs, highlighting the importance of mechanistic understanding in optimizing topical strategies.

Another line of research focuses on transient modulation of the BRB. Techniques such as siRNA-mediated knockdown of tight junction proteins (e.g., claudin-5) have been shown to temporarily increase retinal permeability, enabling larger molecules to enter. While effective in animal models, this strategy raises concerns about infection risks and long-term safety, particularly in chronic diseases requiring repeated administration[12].

Despite significant progress, drug delivery to the retina faces persistent challenges. Safety concerns remain central, particularly with repeated intraocular procedures that may induce cumulative damage. The BRB continues to limit systemic therapies, and strategies for its temporary modulation carry inherent risks. Moreover, the translation of experimental DDS into clinical practice is complicated by manufacturing, scalability, and regulatory hurdles, particularly for complex nanomaterials.

Opportunities lie in integrating drug delivery with emerging therapeutic modalities. For instance, nanoparticle carriers can enhance the stability and uptake of DNA or RNA-based therapeutics, supporting the growth of gene and cell therapies. Light-responsive and stimuli-sensitive materials provide promising avenues for controlled release, potentially reducing treatment burden by prolonging dosing intervals. In addition, personalized drug delivery systems that take into account patient-specific anatomical and physiological differences may increase therapeutic efficacy.

Converging nanotechnology, pharmacology, and ophthalmic surgery thus opens new frontiers. However, systematic evaluation of long-term safety and standardization are necessary. Academic-industry collaborations will be critical to overcome the translational gap and to establish drug delivery as a central component in the development of next-generation retinal therapies.

3. Conclusion

This study examines common invasive and non-invasive methods for vision restoration. Invasive strategies, including gene therapy, cell therapy, and retinal prostheses, are often more effective but sometimes face safety concerns. In contrast, non-invasive methods, including electromagnetic stimulation and drug delivery, are generally safer, less costly, and more acceptable to patients. Even though their therapeutic effects remain comparatively limited. Overall, while each method has some benefit, none of them achieves full restoration of natural vision for all patients.

Gene therapy offers a paradigm-shifting solution for monogenic disorders but is limited by cost, safety, and disease specificity. Cell therapy and prosthetic devices can treat late-stage patients but face integration and performance constraints. Non-invasive therapies such as electromagnetic stimulation and drug delivery are effective mostly during the early stages, though durability is sometimes limited.

In a broader scope, this research clarifies the complementary roles of invasive and non-invasive approaches. By outlining strengths and limitations, this work provides a foundation for interdisciplinary research to achieve more effective and accessible vision restoration methods in the future.

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