Genome Publications
https://doi.org/10.61096/978-81-981372-2-7_14

Chapter 14

Ophthalmic and Otologic Pharmacology

Deepan natarajan
JKKMMRF'S – Annai JKK Sampoorani Ammal College of Pharmacy,
Komarapalayam, Namakkal, Tamilnadu. 638 183
Kannan Raman
St. Johns College of Pharmaceutical Sciences & Research, Kattappana, Idukki-685515, Kerala
Habeeb Rahiman Mubarak
JKKMMRF'S – Annai JKK Sampoorani Ammal College of Pharmacy,
Komarapalayam, Namakkal, Tamilnadu. 638 183

Abstract: Ophthalmic and otologic pharmacology represents a unique intersection of systemic and localized therapeutic interventions aimed at preserving two of the most vital sensory modalities vision and hearing. Drug delivery to the eye and ear faces formidable physiologic barriers, including tear turnover, corneal epithelium, blood–retinal barriers, cerumen, and limited vascularity of the inner ear. Recent advances have led to innovative approaches such as sustained-release intraocular implants, nanoparticle carriers, and targeted intratympanic therapies, revolutionizing management paradigms. Glaucoma pharmacotherapy remains a cornerstone of ocular therapeutics, with prostaglandin analogs, beta-blockers, carbonic anhydrase inhibitors, and rho kinase inhibitors forming the therapeutic backbone. Similarly, anti-VEGF agents have transformed retinal disease treatment, particularly in diabetic retinopathy and neovascular age-related macular degeneration. Anti-inflammatory regimens with corticosteroids and NSAIDs remain indispensable in managing uveitis, allergic conjunctivitis, and post-surgical inflammation. In diagnostic and therapeutic ophthalmology, mydriatics and cycloplegics continue to serve critical roles. On the otologic side, antibiotics, antifungals, corticosteroids, and antivertigo drugs such as betahistine dominate the pharmacotherapeutic spectrum. However, both ocular and otologic agents can cause sensory toxicities, necessitating vigilant monitoring. Pharmacogenomics introduces precision medicine into ophthalmology, offering tailored therapy based on genetic determinants of drug response. This chapter integrates current evidence, clinical applications, and translational advances in ophthalmic and otologic pharmacology, underscoring evolving trends and future possibilities.

Keywords: Ophthalmic pharmacology, Otologic pharmacology, Anti-VEGF, Glaucoma therapy, Sensory toxicity.

Citation: Deepan Natarajan, Kannan Raman, Habeeb Rahiman Mubarak. Ophthalmic and Otologic Pharmacology. *Precision Therapeutics Across Organ Systems. Genome Publications.* 2025; Pp144-153. https://doi.org/10.61096/978-81-981372-2-7_14

14.0 INTRODUCTION

The fields of ophthalmic and otologic pharmacology are integral to modern clinical practice, encompassing a diverse range of drugs aimed at preserving sensory function and improving quality of life. Pharmacological treatment of eye and ear diseases presents unique challenges due to the anatomical, physiological, and biochemical barriers that limit drug penetration. The eye, though accessible externally, is highly protected by structural barriers such as the corneal epithelium, conjunctiva, sclera, and dynamic barriers such as tear turnover and blinking. Similarly, systemic barriers such as the blood–aqueous and blood–retinal barriers restrict intraocular drug availability. These limitations necessitate higher drug concentrations, frequent dosing, or innovative delivery systems to achieve therapeutic efficacy [1].

In the case of the ear, drug delivery faces challenges due to cerumen accumulation, epithelial barriers of the external auditory canal, and limited vascularity of the cochlea and vestibular apparatus. Oral and parenteral therapies often fail to achieve adequate concentrations in the inner ear, leading to the increasing use of intratympanic and intracochlear delivery systems [2]. The demand for safe, effective, and targeted approaches has driven the development of nanocarriers, microparticle-based delivery, and sustained-release implants that provide prolonged therapeutic exposure while minimizing systemic side effects.

Pharmacological management of ocular diseases such as glaucoma, uveitis, diabetic retinopathy, and age-related macular degeneration has undergone a paradigm shift in recent decades. Likewise, otologic pharmacology continues to evolve with the emergence of advanced antibiotics, anti-vertigo agents, and corticosteroid-based therapies. However, sensory toxicity remains a persistent concern, with aminoglycosides and certain antimalarial drugs known to cause irreversible ototoxicity and chloroquine derivatives implicated in retinal toxicity [3].

The convergence of nanotechnology, regenerative medicine, and pharmacogenomics promises transformative changes in this domain. Gene therapy for inherited retinal dystrophies, stem cell transplantation for macular degeneration, and CRISPR-mediated correction of genetic mutations represent the cutting edge of future interventions [4]. In otology, experimental therapies exploring cochlear hair cell regeneration and auditory nerve repair are moving closer to clinical applicability.

Thus, ophthalmic and otologic pharmacology represents a dynamic and multidisciplinary arena, blending classical pharmacotherapy with next-generation biologics, devices, and personalized medicine. This chapter provides a structured overview of the key drug classes, therapeutic strategies, limitations, and innovations in this rapidly evolving field.

14.0.1 Barriers to Ocular and Otic Drug Delivery

The eye and ear possess specialized defense mechanisms designed to protect delicate sensory tissues from external insults, but these same barriers pose challenges to pharmacotherapy. The ocular barriers include static anatomical structures and dynamic physiological processes. The corneal epithelium, with its tight junctions, is lipophilic in nature and restricts penetration of hydrophilic drugs. Conversely, the stroma is hydrophilic and impedes lipophilic molecules, creating a biphasic challenge for drug permeation. In addition, tear turnover (approximately 1 µL/min) dilutes and clears topically applied medications, leading to bioavailability of less than 5% for most ophthalmic drugs [5]. The nasolacrimal drainage system further contributes to systemic absorption, potentially leading to unwanted systemic effects such as bradycardia with topical beta-blockers.

Posterior segment drug delivery is even more complex due to the presence of the blood–retinal barrier, which is analogous to the blood–brain barrier. This barrier consists of tight junctions between retinal capillary endothelial cells and retinal pigment epithelium, severely restricting drug penetration. Traditional topical and systemic therapies often fail to reach therapeutic levels in the posterior chamber, necessitating invasive strategies such as intravitreal injections or implants [6].

Similarly, the ear demonstrates both external and internal barriers. The external auditory canal, lined by stratified squamous epithelium and protected by cerumen, restricts penetration of many topical agents. In the middle ear, the tympanic membrane serves as a physical barrier, though permeable to lipophilic drugs under certain conditions. The inner ear poses the greatest challenge due to its sequestration within the bony labyrinth and the limited vascular supply of the cochlea and vestibular structures. The blood–labyrinth barrier, analogous to the blood–retinal barrier, restricts systemic drug penetration. Consequently, high systemic doses are often required, increasing the risk of toxicity [7].

Innovative strategies have been developed to circumvent these barriers. In ophthalmology, nanoparticle-based carriers, liposomes, and dendrimers are being employed to enhance corneal penetration and sustain drug release. Hydrogels and in situ gelling systems prolong residence time on the ocular surface. In otology, intratympanic injections deliver drugs directly across the round window membrane into the cochlea, bypassing systemic circulation and achieving higher local concentrations. Emerging technologies such as microneedle arrays, biodegradable intracochlear implants, and targeted nanocarriers hold promise in overcoming the current limitations [8].

These barriers illustrate why ophthalmic and otologic pharmacology must adopt specialized approaches distinct from systemic pharmacology. Understanding these obstacles has informed the design of novel delivery systems that improve therapeutic outcomes while reducing systemic adverse effects.

14.1 Glaucoma Pharmacology

Glaucoma is one of the leading causes of irreversible blindness worldwide, characterized by progressive optic neuropathy often associated with elevated intraocular pressure (IOP). The primary therapeutic goal in glaucoma management is reduction of IOP, which can be achieved by decreasing aqueous humor production, increasing its outflow, or both. The main classes of drugs include prostaglandin analogs, beta-adrenergic antagonists, alpha-adrenergic agonists, carbonic anhydrase inhibitors, and the newer rho kinase inhibitors [9].

Prostaglandin analogs, such as latanoprost, travoprost, and bimatoprost, are first-line therapies due to their potent IOP-lowering efficacy, once-daily dosing, and relatively favorable safety profile. They enhance uveoscleral outflow and lower IOP by 25–35%. However, side effects include conjunctival hyperemia, eyelash growth, iris pigmentation, and periocular skin darkening. Comparative studies demonstrate superior efficacy of prostaglandin analogs over beta-blockers in long-term IOP control [10].

Beta-blockers, including timolol and betaxolol, reduce aqueous humor production by inhibiting ciliary body beta-adrenergic receptors. Timolol, in particular, has been widely used but is associated with systemic side effects such as bradycardia, hypotension, and bronchospasm due to systemic absorption via the nasolacrimal duct. Selective beta-1 blockers such as betaxolol may reduce pulmonary risks but are less potent in IOP reduction [11].

Alpha-adrenergic agonists, such as brimonidine, reduce aqueous humor production and enhance uveoscleral outflow. Brimonidine also exhibits neuroprotective properties, though allergic conjunctivitis is a common adverse effect. Carbonic anhydrase inhibitors (CAIs), available as topical formulations like dorzolamide and brinzolamide or systemic agents like acetazolamide, reduce aqueous humor secretion by inhibiting carbonic anhydrase in the ciliary processes. Systemic CAIs are reserved for acute management due to metabolic side effects such as acidosis and hypokalemia [12].

The newer rho kinase (ROCK) inhibitors, exemplified by netarsudil, act by relaxing the trabecular meshwork and enhancing aqueous humor outflow through conventional pathways. Clinical trials demonstrate significant additive effects when combined with prostaglandin analogs. Combination therapies, including fixed-dose prostaglandin analog–beta blocker or prostaglandin analog–ROCK inhibitor combinations, enhance compliance and efficacy [13].

Despite the availability of multiple pharmacological options, adherence to glaucoma therapy remains a challenge due to chronicity, side effects, and complex regimens. Sustained-release implants, nanocarriers, and drug-eluting contact lenses represent novel delivery strategies designed to improve adherence and long-term outcomes.

14.2 Anti-VEGF Therapies

The introduction of anti-vascular endothelial growth factor (VEGF) therapies revolutionized the treatment of retinal vascular diseases, particularly neovascular age-related macular degeneration (AMD), diabetic retinopathy, and retinal vein occlusions. VEGF plays a central role in promoting abnormal neovascularization and increased vascular permeability, leading to vision-threatening complications such as macular edema and hemorrhage. Pharmacologic inhibition of VEGF has demonstrated profound benefits in halting disease progression and restoring visual acuity [14].

Ranibizumab, a monoclonal antibody fragment, was among the first anti-VEGF agents approved for intravitreal use. Clinical trials such as MARINA and ANCHOR established its efficacy in stabilizing and improving vision in neovascular AMD. Aflibercept, a fusion protein acting as a VEGF trap, offers extended dosing intervals with comparable or superior outcomes, making it suitable for patients requiring reduced injection frequency [15]. Bevacizumab, though initially developed for oncology, has been widely repurposed off-label for ocular use due to its cost-effectiveness, with numerous head-to-head trials confirming its non-inferiority to ranibizumab [16].

While intravitreal injections provide high local drug concentrations, they carry procedural risks such as endophthalmitis, retinal detachment, and intraocular hemorrhage. Furthermore, the need for repeated injections imposes a significant treatment burden. Sustained-release formulations, port delivery systems, and gene therapy approaches aimed at long-term VEGF suppression are under active investigation to overcome these limitations [17].

14.3 Anti-Inflammatory Agents

Inflammation contributes to a spectrum of ocular diseases, including uveitis, allergic conjunctivitis, and post-surgical complications such as cystoid macular edema. Pharmacologic interventions include corticosteroids, non-steroidal anti-inflammatory drugs (NSAIDs), and emerging biologics targeting inflammatory pathways.

Corticosteroids, such as prednisolone acetate, dexamethasone, and loteprednol, remain the gold standard for suppressing ocular inflammation. They act by downregulating pro-inflammatory cytokines, inhibiting leukocyte infiltration, and stabilizing lysosomal membranes. However, prolonged use is associated with adverse effects including cataract formation, ocular hypertension, and secondary infections [18]. The development of dexamethasone intravitreal implants (Ozurdex) has improved drug delivery for chronic posterior uveitis, providing sustained effects with reduced dosing frequency.

NSAIDs, including ketorolac and nepafenac, inhibit cyclooxygenase enzymes, thereby reducing prostaglandin-mediated inflammation. They are commonly used for pain and inflammation following cataract surgery and for prevention of cystoid macular edema. Comparative studies demonstrate that NSAIDs are less potent than corticosteroids but provide an additive effect when used in combination [19]. Recent advances include biologic agents targeting TNF-α, IL-6, and other cytokines implicated in refractory uveitis. Agents such as adalimumab have gained approval for non-infectious uveitis, marking the transition toward more targeted immunomodulatory strategies in ophthalmology [20].

14.4 Mydriatics and Cycloplegics

Mydriatic and cycloplegic agents are essential tools in ophthalmology for diagnostic, therapeutic, and surgical applications. By inducing pupil dilation and ciliary muscle paralysis, these agents facilitate fundus examination, refraction assessment, and treatment of certain ocular conditions. Mydriatics, such as phenylephrine, are sympathomimetic agents that stimulate the iris dilator muscle, producing rapid pupil dilation. They are commonly used in routine ophthalmic examinations and surgical procedures. Cycloplegics, such as atropine, cyclopentolate, and tropicamide, block muscarinic receptors in the ciliary muscle and iris sphincter, leading to paralysis of accommodation and dilation of the pupil [21].

Atropine remains the most potent cycloplegic, with prolonged duration of action, making it useful in penalization therapy for amblyopia. Cyclopentolate offers shorter action, suitable for pediatric refraction. Tropicamide provides rapid, short-term dilation, preferred for diagnostic purposes. Potential side effects include photophobia, blurred vision, and systemic anticholinergic effects such as tachycardia and dry mouth, especially in children [22]. Emerging formulations aim to provide reversible and adjustable dilation with reduced systemic absorption. Nanocarriers and novel delivery systems may minimize adverse effects while enhancing efficacy in pediatric and geriatric populations.

14.5 Otologic Treatments

Pharmacologic management of ear disorders includes treatment of infections, inflammation, and vestibular dysfunction. Otitis externa and otitis media are commonly managed with topical antibiotics such as aminoglycosides (gentamicin, neomycin), fluoroquinolones (ciprofloxacin), or combination preparations with corticosteroids. Fluoroquinolones are favored for their safety in cases with tympanic membrane perforation due to their minimal ototoxicity [23].

Systemic antibiotics are required for severe infections, with amoxicillin or cephalosporins often employed. Fungal otitis externa is managed with topical antifungals such as clotrimazole or nystatin. Corticosteroid-containing drops alleviate inflammation and edema, enhancing drug penetration. Management of vestibular disorders, including Ménière's disease and vertigo, often involves betahistine, which improves cochlear blood flow by acting as a histamine H1 agonist and H3 antagonist. Vestibular suppressants such as meclizine and dimenhydrinate are used for acute episodes, though prolonged use may hinder central compensation [24].

In recent years, intratympanic therapy has emerged as a promising approach, particularly for sudden sensorineural hearing loss and refractory Ménière's disease. Intratympanic corticosteroids and aminoglycosides provide high local concentrations while minimizing systemic side effects. However, careful monitoring is required to avoid ototoxic complications.

14.6 Sensory Toxicity

A major challenge in ophthalmic and otologic pharmacology is the potential for drug-induced sensory toxicity. Ototoxicity is commonly associated with aminoglycosides, loop diuretics, and chemotherapeutic agents such as cisplatin. These agents damage cochlear hair cells and vestibular structures, leading to irreversible hearing loss or balance disturbances [25]. Similarly, ocular toxicities arise from chloroquine and hydroxychloroquine (retinopathy), ethambutol (optic neuropathy), and corticosteroids (cataracts and glaucoma). The increasing use of biologics has introduced rare but serious ocular immune-mediated adverse effects. Preventive strategies include baseline and periodic ophthalmic or audiometric monitoring, dose adjustments, and the use of protective adjuvants under investigation [26].

14.7 Intraocular Delivery Systems

The limitations of conventional topical and systemic therapies have driven the development of advanced intraocular delivery systems. Sustained-release implants, biodegradable microparticles, and nanocarriers are designed to prolong drug exposure while reducing treatment burden. Dexamethasone intravitreal implant and fluocinolone acetonide implant exemplify corticosteroid delivery systems providing months to years of anti-inflammatory effect. Port delivery systems for anti-VEGF agents are undergoing clinical trials, offering refillable reservoirs for long-term retinal disease management [27].

Microparticles and nanoparticles encapsulating anti-glaucoma agents or anti-inflammatories are under study for targeted release in the anterior or posterior chamber. These approaches have the potential to transform chronic disease management by improving adherence, minimizing invasive procedures, and reducing systemic side effects.

14.8 Pharmacogenomics in Ophthalmology

Pharmacogenomics introduces personalized medicine into ophthalmology, tailoring drug therapy based on genetic polymorphisms influencing drug metabolism and response. For example, polymorphisms in CYP2D6 affect beta-blocker metabolism, influencing timolol response and systemic side effects. Genetic variants in prostaglandin receptor genes may determine the efficacy of prostaglandin analogs in lowering IOP [28]. In retinal pharmacotherapy, genetic markers predicting response to anti-VEGF agents are under active investigation, potentially guiding individualized dosing regimens. Pharmacogenomics also offers opportunities to identify patients at risk for drug-induced toxicities, enabling preventive strategies.

Table 1: Pharmacological Agents for Glaucoma Management

Drug Class	Examples	Mechanism of Action	Advantages	Limitations/Adverse Effects
Prostaglandin Analogs	Latanoprost, Bimatoprost, Travoprost	Increase uveoscleral outflow	Once-daily dosing, strong IOP reduction	Iris pigmentation, eyelash growth, conjunctival hyperemia
Beta-Blockers	Timolol, Betaxolol	Decrease aqueous humor production	Cost-effective, rapid onset	Systemic effects: bradycardia, bronchospasm
Alpha-Agonists	Brimonidine	Reduce	Neuroprotective	Allergic conjunctivitis,

		aqueous production, increase uveoscleral outflow	potential	dry mouth
Carbonic Anhydrase Inhibitors	Dorzolamide, Acetazolamide	Reduce aqueous humor secretion	Useful as adjunct	Topical: stinging; systemic: acidosis, kidney stones
Rho Kinase Inhibitors	Netarsudil	Enhance trabecular meshwork outflow	Novel mechanism, additive with prostaglandins	Conjunctival hyperemia, corneal verticillata

Table 2: Common Ophthalmic and Otologic Drug-Induced Toxicities

Drug	**Target Organ**	**Mechanism of Toxicity**	**Clinical Manifestations**	**Monitoring Strategies**
Aminoglycosides (Gentamicin, Amikacin)	Ear	Cochlear hair cell damage	Sensorineural hearing loss, vertigo	Audiometry, serum drug levels
Cisplatin	Ear	ROS generation, apoptosis in cochlea	Irreversible hearing loss	Regular hearing tests
Chloroquine, Hydroxychloroquine	Eye	Retinal pigment epithelium binding	Bull's-eye maculopathy, visual field defects	Baseline and annual retinal screening
Ethambutol	Eye	Optic nerve toxicity	Optic neuropathy, loss of color vision	Visual acuity and color vision testing
Corticosteroids	Eye	Increased IOP, lens protein alteration	Glaucoma, posterior subcapsular cataracts	IOP monitoring, slit-lamp exams
Loop Diuretics (Furosemide)	Ear	Disruption of endolymph homeostasis	Reversible hearing loss	Dose adjustment, hearing monitoring

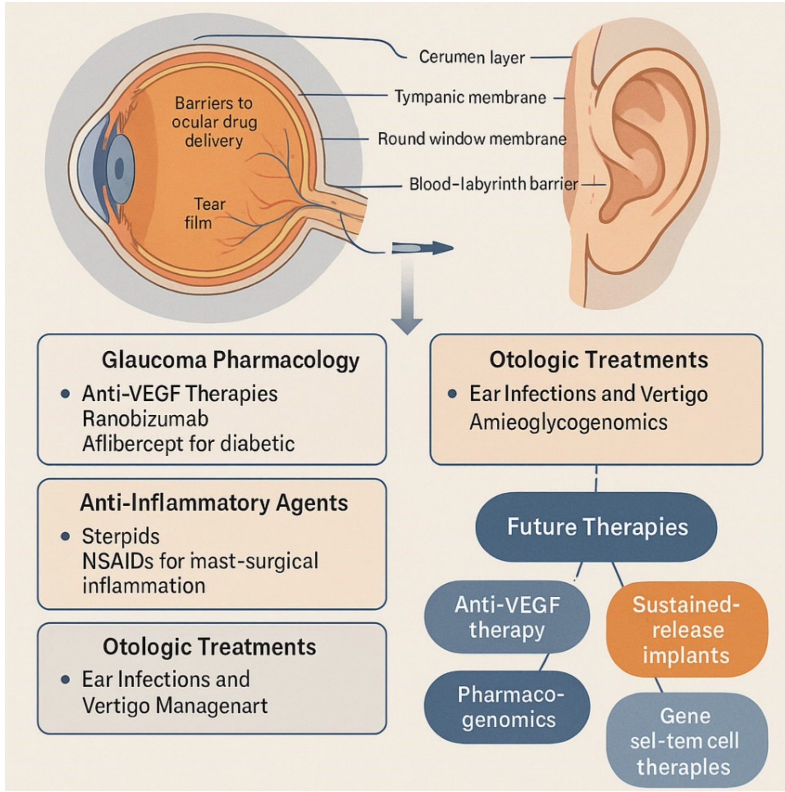

Figure 1: Ophthalmic & Otologic Pharmacology

14.9 Future Therapies

The future of ophthalmic and otologic pharmacology lies in regenerative and genetic approaches. Gene therapy has already entered clinical practice with voretigene neparvovec, approved for RPE65-associated inherited retinal dystrophy. Ongoing trials are exploring CRISPR-based therapies for retinitis pigmentosa and Leber congenital amaurosis [29]. Stem cell therapies, particularly using induced pluripotent stem cells (iPSCs), are being developed to restore retinal pigment epithelium and photoreceptors. In otology, regenerative strategies focus on cochlear hair cell regeneration using gene delivery and molecular modulation of signaling pathways such as Notch and Wnt [30]. Exosome-based delivery systems, optogenetic therapies, and artificial vision prostheses further expand the therapeutic horizon. The integration of bioengineering, nanomedicine, and precision genetics heralds a new era in sensory restoration.

14.10 CONCLUSION

Ophthalmic and otologic pharmacology embodies a rapidly advancing field that balances traditional drug therapy with cutting-edge innovations. From the management of glaucoma and retinal vascular diseases to the treatment of otitis media and vestibular dysfunction, pharmacologic interventions have improved quality of life for millions. However, unique anatomical and physiological barriers continue to challenge effective drug delivery, necessitating specialized systems such as implants, intravitreal injections, and intratympanic therapies. The emergence of anti-VEGF therapies has redefined retinal disease management, while prostaglandin analogs remain the cornerstone of glaucoma pharmacotherapy. Anti-inflammatory strategies continue to evolve, with biologics and implants providing more sustained and targeted effects. Yet, drug-induced sensory toxicities remain a critical concern, underscoring the need for careful monitoring and pharmacogenomic guidance.

Looking ahead, regenerative medicine and gene-based interventions promise transformative solutions for conditions once deemed incurable, such as retinitis pigmentosa and sensorineural hearing loss. The integration of pharmacogenomics, nanotechnology, and precision medicine into routine care will further personalize therapy, reduce systemic risks, and optimize outcomes. Ultimately, the field is moving toward a future where vision and hearing preservation are achievable goals for a broader patient population, supported by multidisciplinary innovations and continuous clinical refinement.

REFERENCES

1. Gaudana R, Ananthula HK, Parenky A, Mitra AK. Ocular drug delivery. AAPS J. 2010;12(3):348–360.
2. Salt AN, Plontke SK. Local inner-ear drug delivery and pharmacokinetics. Drug Discov Today. 2005;10(19):1299–1306.
3. Marmor MF, Kellner U, Lai TY, et al. Recommendations on screening for chloroquine and hydroxychloroquine retinopathy. Ophthalmology. 2016;123(6):1386–1394.
4. Bennett J. Gene therapy for ocular diseases. Nat Rev Genet. 2017;18(2):133–146.
5. Mitra AK, Mikkelson TJ, Kannan R. Barriers to ocular drug delivery. J Ocul Pharmacol Ther. 2018;34(2):101–111.
6. del Amo EM, Urtti A. Current and future ophthalmic drug delivery systems. Adv Drug Deliv Rev. 2017;126:1–2.
7. Juhn SK, Rybak LP, Prado S. Nature of blood–labyrinth barrier in experimental animals. Acta Otolaryngol. 1981;91(1–6):529–534.
8. Xu Q, Boylan NJ, Suk JS, et al. Nanoparticle-based ocular drug delivery: State of the art. Drug Discov Today. 2013;18(7–8):337–343.
9. Weinreb RN, Khaw PT. Primary open-angle glaucoma. Lancet. 2004;363(9422):1711–1720.
10. Parrish RK, Palmberg P, Sheu WP. A comparison of latanoprost, bimatoprost, and travoprost in patients with elevated IOP. Am J Ophthalmol. 2003;135(5):688–703.
11. Zimmerman TJ, Kaufman HE. Timolol: A beta-adrenergic blocking agent for the treatment of glaucoma. Arch Ophthalmol. 1977;95(4):601–604.
12. Serle JB. Medical treatment of glaucoma: A review. Drugs Aging. 1996;8(6):459–478.
13. Bacharach J, Dubiner HB, Levy B, et al. Double-masked, randomized, dose–response study of AR-13324 vs latanoprost in patients with elevated IOP. Am J Ophthalmol. 2015;160(3):511–519.
14. Rosenfeld PJ, Brown DM, Heier JS, et al. Ranibizumab for neovascular AMD. N Engl J Med. 2006;355(14):1419–1431.
15. Heier JS, Brown DM, Chong V, et al. Intravitreal aflibercept (VEGF trap-eye) in wet AMD. Ophthalmology. 2012;119(12):2537–2548.
16. Martin DF, Maguire MG, Ying GS, et al. Ranibizumab and bevacizumab for neovascular AMD. N Engl J Med. 2011;364(20):1897–1908.
17. Campochiaro PA, Wykoff CC, Brown DM, et al. Port delivery system with ranibizumab for neovascular AMD. Ophthalmology. 2019;126(8):1141–1154.

18. Jabs DA, Nussenblatt RB, Rosenbaum JT. Standardization of uveitis nomenclature. Am J Ophthalmol. 2005;140(3):509–516.
19. Kim SJ, Flach AJ, Jampol LM. Nonsteroidal anti-inflammatory drugs in ophthalmology. Surv Ophthalmol. 2010;55(2):108–133.
20. Nguyen QD, Merrill PT, Jaffe GJ, et al. Adalimumab for prevention of uveitic flare. Ophthalmology. 2016;123(5):1005–1015.
21. Fraunfelder FW. Drug-induced ocular side effects. Expert Opin Drug Saf. 2004;3(3):267–280.
22. Milder E, Rubin M. Ocular pharmacology. In: Yanoff M, Duker JS, editors. Ophthalmology. 5th ed. Elsevier; 2019.
23. Roland PS, Stroman DW. Microbiology of acute otitis externa. Laryngoscope. 2002;112(7):1166–1177.
24. Adrion C, Fischer CS, Wagner J, et al. Efficacy and safety of betahistine in Ménière's disease. Cochrane Database Syst Rev. 2016;2016(6):CD010696.
25. Rybak LP, Whitworth CA. Ototoxicity: Therapeutic opportunities. Drug Discov Today. 2005;10(19):1313–1321.
26. Fraunfelder FW, Fraunfelder FT. Drug-induced ocular side effects: Clinical ocular toxicology. Saunders; 2008.
27. Holekamp NM, Campochiaro PA, Chang MA, et al. Arch Ophthalmol. 2012;130(3):332–340.
28. Michelessi M, Lindsley K. Pharmacogenomics of glaucoma therapy. Cochrane Database Syst Rev. 2016;2016(11):CD011342.
29. Russell S, Bennett J, Wellman JA, et al. Efficacy and safety of voretigene neparvovec. Lancet. 2017;390(10097):849–860.
30. Atkinson PJ, Huarcaya Najarro E, Sayyid ZN, Cheng AG. Sensory hair cell development and regeneration. Development. 2015;142(20):3561–3571.