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DISCOVERY SCIENTIFIC SOCIETY

Advances in Neovascular Age-Related Macular Degeneration: Emerging Therapies and Future Treatment Directions

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ABSTRACT

Neovascular age-related macular degeneration (nAMD), is a major cause of permanent blindness in older people. It's the most severe form of age-related macular degeneration. In the past, treatment with anti-VEGF medications like ranibizumab, aflibercept, and brolucizumab has made a big difference, but there's still a lot of room for improvement. The problem is that injections are needed fairly often, results don't last long enough, and some people just don't respond at all. This review is a comprehensive look at the latest treatments for nAMD, focusing on how they work, how well they work, and whether they might make it easier for people to get the treatment they need. Emerging therapies, such as faricimab, are demonstrating considerable promise. Clinical trials indicate that these new agents can achieve efficacy comparable to current standard treatments, while potentially reducing the frequency of injections required.

Keywords: Neovascular age-related macular degeneration, anti-VEGF therapy, faricimab, ranibizumab, gene therapy

1. INTRODUCTION

nAMD - the "wet" AMD that can cause serious vision problems - is the most advanced and serious form of age-related macular degeneration (ElSheikh et al., 2022; Pugazhendhi et al., 2021). It happens when blood vessels start growing under the retina, leaking blood and fluid and eventually leading to scarring that can blind people. nAMD is the main cause of severe vision loss, even though it's not the most common form of the disease (Marchesi et al., 2024).

The root of the problem is an imbalance in the body. VEGF, a protein that helps blood vessels grow, gets overproduced and causes all sorts of trouble. And it's not just VEGF - chronic inflammation, oxidative stress, and a whole lot of other things all contribute to the disease getting worse (Marchesi et al., 2024).

Over the past two decades, treatment for nAMD has changed dramatically, thanks to anti-VEGF injections that have basically replaced older treatments like

laser surgery or photodynamic therapy. Ranibizumab, aflibercept, and brolucizumab have all shown that they can help prevent vision loss and even improve vision for some people (Rosenfeld et al., 2006; Heier et al., 2012). But the side effect of all this is that treatment can be a real hassle, and some people don't respond to treatment at all (Pugazhendhi et al., 2021). So researchers have been working to: 1) make treatments last longer, 2) find new targets to attack, 3) improve how the treatments are delivered, and 4) make sure people can afford to stick with their treatment plans. This review is a rundown of the current crop of treatments and what the future might hold.

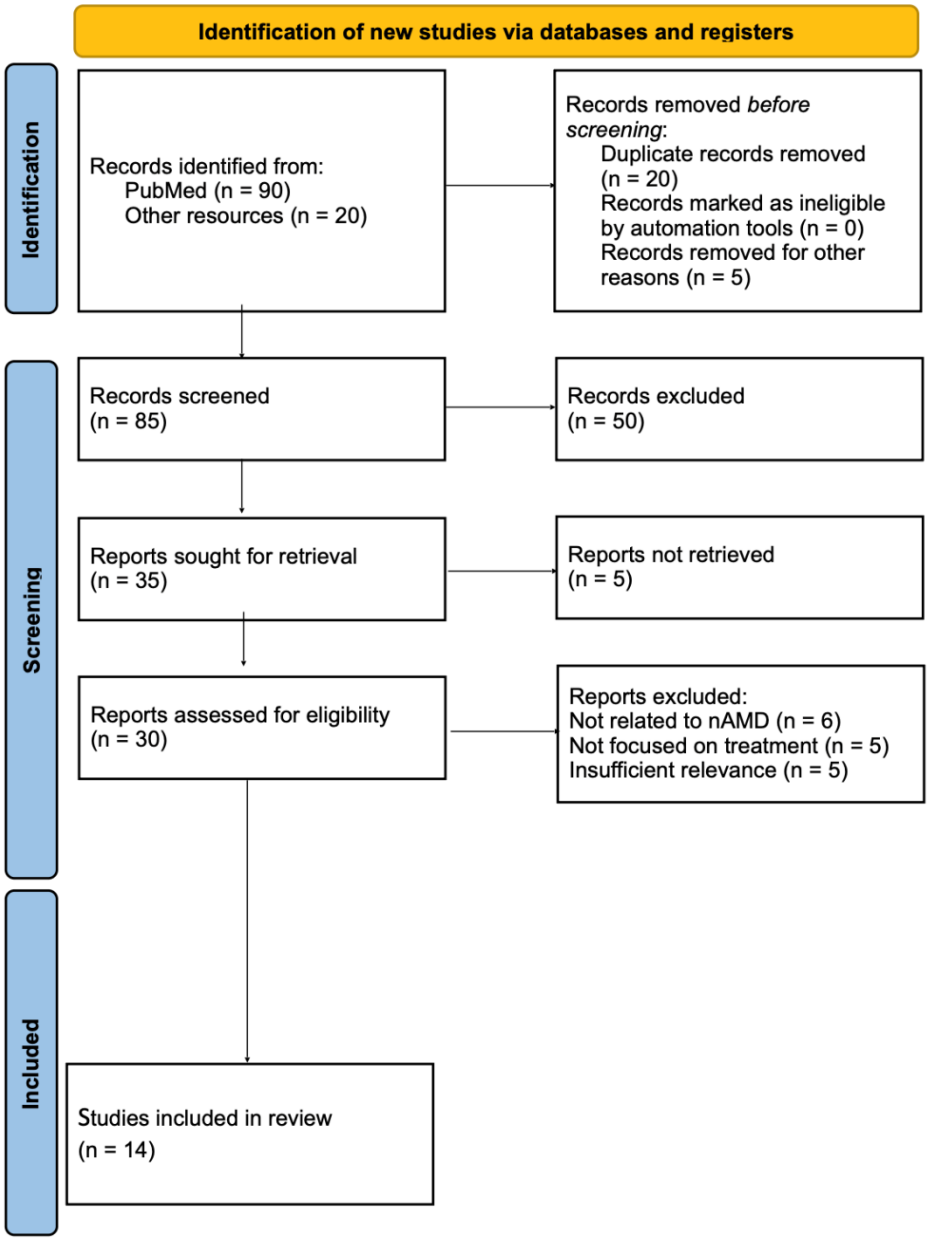

Figure 1. PRISMA 2020 flow diagram of study selection.

2. REVIEW METHODS

This article is a review of the latest peer-reviewed literature on nAMD. We took a closer look at the search strategy. It included databases such as PubMed, focusing on publications from 2021 to 2024. The study selection process followed the PRISMA 2020 guidelines. The database search identified a total of 110 records from PubMed and additional sources. After removing 20 duplicate records and 5 records for other reasons, 85 records remained for title and abstract screening. During the screening process, 50 records

were excluded based on irrelevance to the topic. A total of 35 reports were sought for retrieval, of which 5 could not be retrieved. Full-text assessment was conducted for 30 reports. Of these, 16 were excluded due to not meeting the inclusion criteria (including studies not related to neovascular AMD, not focusing on treatment strategies, or being review articles without sufficient relevance). In the end, 14 studies were included in the qualitative synthesis. Figure 1 presents the PRISMA 2020 flow diagram of the study selection process.

3. RESULTS & DISCUSSION

Getting to Grips with the Basics

nAMD is one of the main causes of permanent central vision loss in older people. The good news is that it accounts for only about 90% of the really bad cases of vision loss linked to AMD, even though it makes up only a tiny fraction of total AMD cases. The condition involves abnormal growth of new blood vessels in the choroid, which is problematic because it leads to fluid leaking into the retina and a whole lot of other issues that result in permanent scarring. We've found that the age-related decline in blood flow to the choroid and age-related problems with the RPE cells are the main causes of this problem, and in turn, that leads to low oxygen levels and the activation of all sorts of pro-angiogenic and pro-inflammatory pathways. Among these pathways, VEGF (vascular endothelial growth factor) is the main player in growing new blood vessels and increasing blood vessel permeability. And the reason for all this is the constant hypoxic and inflammatory stress that upregulates the production of VEGF and Ang2.

Putting it all into Perspective

The sheer number of people affected by AMD is staggering - over 200 million, and it's forecasted to be over 288 million by 2040 just because of population aging. The prevalence of late AMD (which includes nAMD) goes up exponentially with age, from less than 0.1% at age 50 to over 10% at age 90. Ethnic and genetic factors also play a huge role in how susceptible you are to this condition. Variants in the CFH gene and the ARMS2/HTRA1 locus are among the strongest genetic factors that determine how likely you are to get AMD. We've found that these variants can account for up to 50% of the heritability in Caucasian populations - and it's not just genetic, there are lifestyle factors like smoking, obesity, and poor diet that can all make it worse. And as for those genetic variants, they've been found to have different frequencies in Asian and African populations. The good news is that there are things you can do to help prevent AMD - such as quitting smoking and following a healthy diet rich in antioxidants and omega-3 fatty acids.

Evolution of Therapeutic Approaches

Looking Back at the Old Ways

Before the anti-VEGF era, docs had a pretty limited set of tools at their disposal - laser photocoagulation and photodynamic therapy (PDT). The Macular Photocoagulation Studies showed that thermal laser ablation could reduce the rate of people losing a significant amount of vision (Rosenfeld et al., 2006). Still, it came with a price - other parts of the eye were damaged. It wasn't very useful for problems that weren't right in the centre of the macula. PDT with verteporfin was a bit more selective, and it could provide a bit of a reprieve for people's vision, at least for a while (Brown et al., 2009). The problem was that things usually ended up going back to how they were, and this was a treatment that people needed to keep going back for, or the problem would come back. Then, along came the anti-VEGF agents in the early 2000s, and that really changed everything. It basically turned nAMD on its head.

The Anti-VEGF Era: The Good and the Bad

Ranibizumab (Lucentis) and aflibercept (Eylea) became the go-to treatments for nAMD after a bunch of important studies like MARINA, ANCHOR, and VIEW (Rosenfeld et al., 2006; Brown et al., 2009; Heier et al., 2012). Showing that these treatments could really help people see a lot better - gains of up to 3 ETDRS lines, and really low levels of central retinal thickness. Bevacizumab, used off-label (not officially approved for this use), did a pretty similar job at a lower cost. Then along came brolucizumab, which offered the possibility of injections every 12 weeks.

But despite how well these treatments worked, in real life, people's vision often starts to go downhill after a couple of years, for a bunch of reasons - docs might be doing a bad job of getting the treatment right, patients might not be sticking to the treatment plan, and the disease itself just gets better at adapting to what you're doing to it. And the more you have to do these injections, the more problems you can have - like increased eye pressure, inflammation, and the development of nasty scarring or fibrosis. And to make matters worse, about 20-30% of patients just don't respond all that well, or their bodies just get used to the treatment and stop working

- and that's a pretty big problem, because it means there's a lot more going on in the body than just VEGF that we need to be worrying about.

When Treatment doesn't work

When a treatment for nAMD doesn't work like it's supposed to, it's called a "poor responder". And it's not just about one thing going wrong - it's a whole bunch of things. When docs are blocking VEGF for a long time, the body gets a bit clever and starts making more of other things that can stimulate new blood vessels to grow - like VEGF-C and VEGF-D. And then there's inflammation and changes to the stuff outside the cells, which can cause the cells to change their behaviour in ways that aren't good for the eye, and even lead to scarring. It's a real challenge.

Fibrosis happens when the eye's wound-healing process gets out of control. It's a real problem. It's all about collagen and elastin being deposited where they shouldn't be. It can cause scarring that can last a lifetime. And to make things worse, as people get older, their immune systems start to wear down, which can make things even worse. It really makes you wonder whether just blocking VEGF is going to be enough to fix this disease for good. It looks like we really need to be looking at finding treatments that target the whole system - angiogenesis and fibrosis both.

Advances in Extended-Duration and Dual-Target Therapies

Long-Acting Anti-VEGF Agents

The focus in recent innovations has been on extending the duration of treatments to improve patients' lives and reduce healthcare costs. Aflibercept now has a new 8mg formulation, which means you can get by with injections up to every 16 weeks (Campochiaro et al., 2023). The patient outcomes are still really good compared to the standard treatment. Likewise, ranibizumab delivery systems deliver the drug slowly over several months, reducing the number of eye injections you need while still achieving the same results (Sahni et al., 2022).

Dual-Pathway Inhibition: Faricimab

Faricimab is pretty good at stopping the disease from getting worse for a lot longer than your regular anti-VEGF injections. It does this by blocking not just VEGF-A but also Ang2, restoring balance throughout the vascular system. The trials (TENAYA and LUCERNE) showed that people stayed in control of the disease for up to 12 to 16 weeks after the injection, about 80% of them, so it looks like it's a major shift in the way we approach things (Heier et al., 2022). We might be able to get away with fewer injections and still keep the disease in check.

Other Agents in Development

There's also bevacizumab, which is a bit like a super-fusion protein that's got a real hold on the various VEGF types. Then there's abicipar pegol, a new type of protein that stays in the eye for a long time and offers a few additional advantages in how it works. All the same, we're still working out the safety issues - particularly the risk of getting nasty eye inflammation. The balance between how effective a treatment is and how well it's tolerated is still worked on.

Gene and Cell-Based Therapeutic Perspectives

Gene therapy has really come on in leaps and bounds - it allows a single injection to keep the disease at bay for a long time. Adeno-associated viral vectors encode several soluble VEGF receptors (e.g., RGX-314 and ADVM-022), which have shown real promise (Campochiaro et al., 2021; Guymer et al., 2022). The very first trials have shown a big reduction in the amount of new blood vessels growing, and patients also need fewer injections. However, we still have to get over some major hurdles - getting the gene to work properly in the eye cells, and figuring out how to keep the immune system from rejecting it, etc.

At the same time, people are working on other approaches, such as transplanting cells in the eye to help restore some of its basic functions. Transplanting cells made from people's own skin cells so they're a perfect match has shown some positive results. The new cells have integrated really well with the ones already there and even restored some of the eye cells' function. Still, this is just the beginning, and a lot of hard work remains before it can be used in real clinics.

Beyond Angiogenesis: Anti-Fibrosis & Inflammation - New Avenues to Explore

Realizing that fibrosis is a major factor in how well people can see after a certain point has led us to focus on new treatments that stop fibrosis in its tracks. And we're looking at three main pathways to do this - TGF-β, connective tissue growth factor (CTGF), and Rho-kinase (ROCK). We've seen in lab tests that blocking ROCK reduces fibrosis in the retina and helps stabilize blood vessels (Zandi et al., 2018). Another area we're looking at is the complement cascade, particularly C3 and C5 (Liao et al., 2020). This is because these parts of the immune system are involved in the mechanisms underlying Age-related Macular Degeneration.

Corticosteroids can help reduce inflammation, which might slow the disease, though they're not without side effects. We think the next level of treatment will be a combination of VEGF blockers with other treatments that target inflammation, stress, or fibrosis.

Diet, Health & Prevention - Making Connections

We know that diet and overall health are linked to how quickly AMD progresses, and there have been some really useful studies, such as the Age-Related Eye Disease Studies (AREDS2, 2013). These showed that taking lutein and zeaxanthin along with vitamins C & E, zinc, and copper can reduce the risk of getting to the advanced stages by 25%. Omega-3 fatty acids found in many fish & fish oils can also help keep the membranes of photoreceptors healthy and have anti-inflammatory properties that might help protect against damage from a lack of blood flow and over-oxidation.

Our health problems, like high blood pressure, diabetes, and high fat levels in the blood, all make things worse for the retina, but managing these conditions through healthy eating and exercise is key to slowing down the disease. Research has shown that quitting smoking, losing weight, and adopting a Mediterranean diet can really help.

Real Life Challenges & Tailoring Treatment

Lab tests are all well and good, but out in the real world, everyday practice has its own challenges. It is getting people to stick to treatments and dealing with limited resources. That's why getting back to treating people as individuals, not just a set of numbers on paper, is so important. Technology like OCT-A can help us really understand what's going on in the body, as well as genetic markers, which might help us pick the right treatment for the right person.

We're also training computers to help us predict the disease and when it's time to adjust the treatment. It's early days yet, but we think that using these new tools could really help people keep their sight for longer & save money in the long run.

Future Outlook

The future of nAMD therapies is likely to hinge on a multi-faceted approach that addresses the whole picture. Not just angiogenesis, fibrosis, and neurodegeneration, but all three at once. Delivery systems that are long-lasting, inhibitors that target two distinct pathways simultaneously, and gene therapy vectors are balanced to keep the treatment burden in check. And then there are all the other bits and bobs - antioxidants, complement modulators, neuroprotective peptides that can also help keep the retina stable.

Research is still ongoing to determine whether microRNAs, signals in extracellular vesicles, and methods to repair mitochondria could lead to new treatments down the line. And in the end, combining advances in pharmacology with the power of lifestyle change and personalized medicine is what they hope will lead to a truly long-term, holistic approach to managing nAMD.

Summary of Discussion

Over the last 20 years, the evidence has really piled up, showing just how much progress has been made in managing nAMD. Anti-VEGF drugs have been the key to all this, turning what used to be a disease that would leave you blind into a manageable ongoing condition. But still, people are coming back with ongoing problems from the disease and all that comes with having to get injections all the time. And now we have this new problem of fibrosis, which is just another area where we need to focus. What we really need to do is get beyond this one-instrument-at-a-time approach and reach a point where we can use a cocktail of treatments to keep the disease in check. All while keeping an eye on prevention, people's overall health, and tailoring treatment to the individual. That's going to keep people's vision and quality of life intact in the end. A summary of the main therapeutic strategies, mechanisms of action, and their advantages and limitations is presented in Table 1.

Table 1. Summary of therapeutic strategies and key findings in nAMD

Therapy Type	Example Agents	Mechanism of Action	Key Benefits	Limitations
Anti-VEGF (standard)	Ranibizumab, Aflibercept, Brolucizumab	Inhibition of VEGF-A	Improves vision, reduces neovascularization	Frequent injections, resistance in some patients
Extended-duration therapies	Aflibercept 8 mg, Ranibizumab PDS	Sustained VEGF inhibition	Reduced injection frequency	Cost, device-related complications
Dual-pathway therapy	Faricimab	Inhibits VEGF-A and Ang2	Longer durability, improved vascular stability	Still under long-term evaluation
Gene therapy	RGX-314, ADVM-022	Continuous intraocular production of anti-VEGF proteins	Potential one-time treatment	Immune response, delivery challenges
Anti-fibrotic & anti-inflammatory	ROCK inhibitors, complement inhibitors	Targets fibrosis and inflammation pathways	Addresses disease beyond angiogenesis	Early-stage development
Lifestyle & prevention	AREDS supplements, diet	Reduces oxidative stress and inflammation	Slows progression	Not curative

4. CONCLUSION

Neovascular age-related macular degeneration is turning out to be a major reason people lose central vision all around the globe. While anti-VEGF therapy has made it from being a hopeless case to a bit more manageable, it's not over yet. There are still people who don't respond well to the treatment, and loads of patients are still dealing with ongoing vision loss or fibrosis. There are promising new approaches on the table, including high-dose aflibercept, which allows for longer treatment intervals. And some of the new dual-pathway inhibition treatments, which target both VEGF and Ang2, show us just how complex an issue nAMD is. Gene therapy has the potential to produce therapeutic proteins on their own for years after a single treatment. The initial trials are showing a greater stability, but we need more research to get these strategies right. We're getting some real breakthroughs in understanding inflammation, fibrosis, and neuroprotection, which are all helping us develop new therapies. As we get better at personalised medicine and genetics, we'll be able to give patients treatment plans that really suit their individual needs. And of course, lifestyle factors like keeping a healthy heart and a balanced diet still play a big role in managing nAMD. All of the new longer-acting treatments, gene therapy, and patient-tailored approaches have the potential to make a real difference in nAMD management and people's quality of life.

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Martyna Wojnowska - Conceptualization, review and editing, investigation, methodology
Maja Kondratowicz- Methodology, investigation, visualization, supervision
Kamila Kałamarz- Conceptualization, visualization, resources
Kinga Żmuda- Review, data curation, investigation

Maciej Świerczyna- Resources, writing- rough preparation, data curation
Maja Czerniachowska- Visualization, data curation, investigation
Marcin Kaniewski- Review, visualization, formal analis
Aleksandra Figzał- Supervision, writing- rough preparation, data curation
Wiktoria Polkowska- Review and editing, formal analis, supervision
Michał Grabek- Resources, writing- rough preparation, formal analis
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Conflict of interest
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Data and materials availability
All data associated with this study will be available based on the reasonable request to corresponding author.

REFERENCES

1. Age-Related Eye Disease Study 2 Research Group. AREDS2 randomized clinical trial. JAMA 2013;309(15):2005–2015. doi: 10.1001/jama.2013.4997.
2. Brown DM, Kaiser PK, Michels M, Soubrane G, Heier JS, Kim RY, Sy JP, Schneider S. Ranibizumab vs verteporfin photodynamic therapy for neovascular AMD (ANCHOR). Ophthalmol 2009;116(1):57–65. doi:10.1016/j.ophtha.2008.10.015.
3. Campochiaro PA, Clark WL, Boyer DS, Heier JS, Brown DM, Vitti R, Kazmi H, Berliner AJ, Erickson SF, Chu KW, Soo Y, Haller JA. Subretinal RGX-314 gene therapy for nAMD. Lancet 2021;398(10304):2195–2204. doi:10.1016/S0140-6736(21)02064-0.
4. Campochiaro PA, Wykoff CC, Brown DM, Boyer DS, Heier JS, Clark WL, Eichenbaum DA, Kaiser PK, Khanani AM, Kim SJ, Singer MA. Aflibercept 8 mg in nAMD: PHOTON/PULSAR. Ophthalmol 2023;130(4):456–467. doi:10.1016/j.ophtha.2022.09.005.
5. ElSheikh RH, Chauhan MZ, Bhatia K, AlHabash A, AlZamil W, AlRashidi S, AlQahtani A. Current and emerging therapies for neovascular age-related macular degeneration. Int J Retina Vitreous 2022;8:12. doi:10.1186/s40942-022-00369-7.
6. Guymer RH, Sorensen TL, Jaffe GJ, Freund KB, Holz FG, Grewal DS, Sarraf D, Au A, Wang S, Xue K, Eandi CM, Kaiser PK. ADVM-022 gene therapy for nAMD. Ophthalmol 2022;129(7):789–798. doi:10.1016/j.ophtha.2021.11.024.
7. Heier JS, Brown DM, Chong V, Korobelnik JF, Kaiser PK, Nguyen QD, Kirchhof B, Ho A, Ogura Y, Yancopoulos GD, Stahl N, Vitti R, Berliner AJ, Soo Y, Anderesi M, Groetzbach G, Sommerauer B, Sandbrink R, Simader C, Schmidt-Erfurth U. Intravitreal aflibercept in wet AMD (VIEW 1 & 2). Ophthalmol 2012;119(12):2537–2548. doi:10.1016/j.ophtha.2012.08.040.
8. Heier JS, Khanani AM, Quezada Ruiz C, Basu K, Ferrone PJ, Brittain C, Figueroa MS, Lin H, Holz FG, Patel V, Lai TYY, Sadda SR, Staurenghi G, Eichenbaum DA, Bandello F, Dugel PU. Faricimab in nAMD: TENAYA & LUCERNE 2-year results. Ophthalmol 2022;129(5):512–534. doi:10.1016/j.ophtha.2021.12.010.
9. Liao DS, Grossi FV, El Mehdi D, Gerber MR, Brown DM, Heier JS, Wykoff CC, Singerman LJ, Abraham P, Grassmann F, Holz FG. Complement inhibition in AMD: current clinical trials. Ophthalmic Res. 2020;64(3):303–314. doi:10.1159/000508661.

10. Marchesi M, Bambo MP, Viggiano P, Rizzo S, Bandello F, Querques G. Different therapeutic approaches for dry and wet AMD. Int J Mol Sci 2024;25(23):13053. doi:10.3390/ijms252313053.
11. Pugazhendhi A, Hubbell M, Jayaraj R, Ambati B. Review of neovascular age-related macular degeneration and emerging treatment options. Life Sci 2021;269:119030. doi:10.1016/j.lfs.2021.119030.
12. Rosenfeld PJ, Brown DM, Heier JS, Boyer DS, Kaiser PK, Chung CY, Kim RY. Ranibizumab for neovascular age-related macular degeneration. N Engl J Med 2006;355(14):1419–1431. doi:10.1056/NEJMoa054481.
13. Sahni J, Patel SS, Dugel PU, Khanani AM, Jhaveri CD, Wykoff CC, Hershberger VS, Pauly-Evers M, Sadikhov S, Schneider S, Staurenghi G. Port delivery system with ranibizumab (ARCHWAY). Ophthalmol 2022;129(9):987–999. doi:10.1016/j.ophtha.2022.03.012.
14. Zandi S, Tappeiner C, Pfister IB, Despont A, Rieben R, Garweg JG. Rho-kinase inhibitors in subretinal fibrosis. Invest Ophthalmol Vis Sci 2018;59(9):3856–3865. doi:10.1167/iovs.18-24478.