William Blair

Equity Research
Healthcare | Biotechnology

April 8, 2025
Industry Report

Lachlan Hanbury-Brown
+1 312 364 8125
lhanbury-brown@williamblair.com

Dryer for Longer

Neovascular AMD Market Set for Continued Growth With New Therapies, Aging Populations

Contents

Executive Summary

Over the past two decades, the management of retinal vascular diseases such as neovascular age-related macular degeneration (nAMD)—the leading cause of blindness globally among people over 50 years of age—has been transformed by the introduction of anti-VEGF therapies. The profound, vision-saving efficacy of these therapies has led to their broad adoption, driving global sales of branded anti-VEGF therapies to over $15 billion in 2024. While this reflects use across multiple retinal vascular disease beyond nAMD, nAMD accounts for a majority of sales and is often the entry point for new therapies in development for these indications.

While the market has grown from nothing to over $15 billion in the past two decades, we see a robust growth trajectory for several years ahead. First, the introduction of new therapies that require less frequent injections should lower the burden of treatment, thereby improving adherence and long-term outcomes. Up to an estimated 40% of patients discontinue therapy within the first year because of the burden of regular intravitreal injections—simply reducing this discontinuation rate could meaningfully increase the number of treated patients. Second, the prevalence of nAMD increases exponentially with age, so aging populations across much of the Western world are expected to lead to an increased prevalence of nAMD. Beyond nAMD, these new therapies may also drive growth in other retinal vascular diseases, such as diabetic retinopathy (DR), where the treatment burden of current anti-VEGF therapies has limited their adoption despite demonstrating efficacy.

Given the large and growing commercial opportunity, relatively concentrated prescriber base, and significant remaining unmet need, there is a robust pipeline of assets in development for nAMD, almost all of which are focused on extending durability to reduce the treatment burden. Indeed, the commercial dynamics in the anti-VEGF market demonstrate that new products with even modest increases in durability on the order of days to weeks rapidly gain market share, as that translates into fewer injections per year. Technologies being developed include new biologics, bioerodible or biodegradable implants that slowly release tyrosine kinase inhibitors (TKIs), and gene therapies that seek to make a "biofactory" of anti-VEGF proteins in the back of the eye to alleviate the need for regular injections of anti-VEGF proteins.

In this report, we review the epidemiology, pathophysiology, and commercial landscape for nAMD and briefly review products in clinical development in the indication. In conjunction with this report, we are also initiating coverage on Ocular Therapeutix and Kalaris Therapeutics, which we believe are developing best-in-class products to meaningfully improve the treatment burden and outcomes in nAMD. For more information on these companies, see our initiation reports on Kalaris Therapeutics and Ocular Therapeutix.

Neovascular Age-Related Macular Degeneration

Age-related macular degeneration (AMD) is a chronic eye disease characterized by progressive degeneration in the central retina (macula). It is a leading cause of permanent vision loss worldwide in people over the age of 50 and the most common cause of legal blindness in the U.S. The number of individuals affected globally is projected to rise from 196 million in 2020 to 288 million in 2040, and it is estimated to affect 10%-13% of people over age 65 in North America, Europe, and Australia. Risk factors include increased age, being female, genetic factors, Caucasian race, light iris color, smoking, increased BMI, alcohol intake, and other dietary habits.

It is typically categorized into three stages: early, intermediate, or late. Early disease is characterized by numerous small, hard drusen (<63 μm diameter) or intermediate, soft drusen (≥63 μm to <125 μm diameter) and is asymptomatic. Intermediate disease is characterized by either

extensive drusen of small to intermediate size or any drusen that is ≥125 μm in diameter (the average diameter of the retinal vein at the optic disc margin is 124 μm) and may be associated with some relatively minor visual impairment. Advanced disease comes in two forms: it can either remain in the dry form as geographic atrophy (GA) or transform into the wet form, known as wet AMD or neovascular AMD (nAMD). GA is characterized by the steady death of photoreceptors and retinal pigment epithelial (RPE) cells over several years; nAMD is characterized by the formation of abnormal blood vessels (i.e., neovascularization) developing from the choriocapillaris or neurosensory retina. These newly formed vessels have an increased likelihood to leak blood and serum (exudation), causing separation of Bruch's membrane, RPE, and retina from each other, and resulting in the accumulation of sub-RPE, subretinal, or intraretinal fluid. This results in photoreceptor misalignment and degeneration, cell loss, and over time, fibrosis and scar tissue formation.

This damage to the retina leads to progressive, severe vision loss, metamorphopsia, scotoma, photopsia, and impaired dark adaptation. While neovascular AMD accounts for only about 10% to 15% of AMD, it accounted for roughly 90% of the severe vision loss caused by AMD before the advent of anti-VEGF therapies. Without treatment, most eyes will have very poor central vision by 12 months, and many much sooner.

Complex Pathophysiology of AMD

The pathophysiology of AMD is complex, although advances in treatment and imaging have informed our understanding. AMD is a multifactorial disease related to aging, genetic susceptibility, oxidative stress, vascular dysfunction, and environmental risks, that develops as a consequence of disruption of the normal homeostatic mechanisms of the retina. Although the pathophysiology is not completely understood, it is believed that normal aging-related changes cause increasing resistance in blood vessels and reduction of choriocapillaris density, lipid and lipoprotein deposition in Bruch's membrane, and reduction in photoreceptor density. Combined with chronic inflammation, altered lipid and lipoprotein deposition, increased oxidative stress, and impaired extracellular matrix maintenance, this leads to extracellular deposits in the neurosensory retina, RPE, and Bruch's membrane. These extracellular deposits, known as drusen, comprise lipids, minerals, and proteins, and are implicated in the development and progression of AMD. For example, progression from early to intermediate AMD is characterized by increasing drusen size and pigmentary changes in the retina, reflecting migration of RPE cells from their normal place attached to Bruch's membrane into the more inner layers of the retina. Histopathological studies suggest that loss of choriocapillaris is the most pronounced effect of aging in the retina-RPE-Bruch's membrane complex, suggesting that AMD develops secondarily to vascular changes. This in turn diminishes the clearance of lipoprotein and cellular debris from the RPE, which ultimately accumulate in the RPE and Bruch's membrane, further impairing RPE-choriocapillary clearance and natural responses to oxidative stress and triggering inflammatory responses.

Late stages of AMD can manifest in two ways: GA involves the development of confluent areas of atrophy involving photoreceptors and RPE, while neovascular AMD involves the growth of abnormal blood vessels in the macula region. Neovascularization is thought to be induced by increased expression of hypoxia-driven vascular endothelial growth factor A (VEGF-A), which is released in response to stimuli such as oxidative stress and complement activation. VEGF promotes angiogenesis by binding to VEGF receptor 2 (VEGFR2) and activating downstream pathways that promote endothelial cell proliferation and vascular permeability. Leakage of these new blood vessels, known as exudative neovascular AMD, can result in accumulation of subretinal or intraretinal fluid, hemorrhages, and fibrosis, causing visual changes.

While typically referred to as a single entity, nAMD is actually a highly heterogenous disease. Though the extent of the heterogeneity is not yet fully understood, a commonly used classification system is based on the location of the fluid in the retina. The terminology used to describe the anatomy of nAMD was revised in 2020, when the term choroidal neovascularization (CNV)

was replaced with macular neovascularization (MNV) to reflect the fact that neovascularization does not necessarily originate from the choroid (as is the case with type 3 MNV). We detail the major subtypes in exhibit 1 and include diagrams of type 1, 2, and 3 MNV in exhibit 2. While these distinctions do not explain all of the heterogeneity of nAMD, they can help inform treatment decisions and prognosis. For example, type 3 lesions are known to be highly sensitive to anti-VEGF therapy—more so than others—while PCV is known to be relatively less responsive to current anti-VEGF therapies.

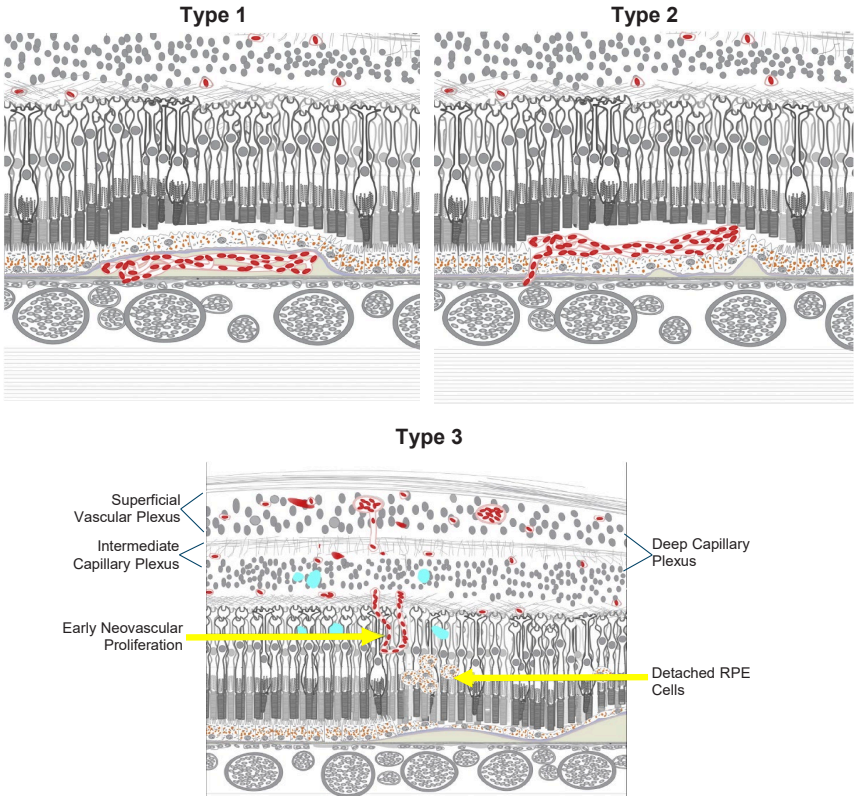
Early symptoms of AMD include difficulty performing tasks under low light conditions and in low-contrast situations, though patients typically do not seek help until they start experiencing more pronounced symptoms. As patients progress to late-stage AMD, the most common symptoms include visual distortion (e.g., straight lines appearing curved) and/or a decline in vision (blurred vision, loss of visual acuity, or difficulty focusing). While these develop relatively slowly in GA, they can develop rapidly in patients with nAMD, triggering their presentation to an ophthalmologist. Diagnosis is made through imaging, of which there are several different options, though spectral-domain optical coherence tomography (SD-OCT) is increasingly the most widely used approach as it allows for 3-dimensional visualization of ocular structures and facilitates detection and monitoring of IRF and SRF with a fast, noninvasive imaging procedure.

Exhibit 1
Consensus AMD Nomenclature

New Term	Old Term	Definition
Type 1 MNV	Occult CNV	Ingrowth of vessels initially from the choriocapillaris into and within the sub-RPE space. Leads to varying types of PEDs (pigment epithelial detachments).
Polypoidal choroidal vasculopathy (PCV)	Polypoidal choroidal vasculopathy (PCV)	A variant of type 1 MNV commonly seen in Asian persons. Indocyanine green angiography imaging shows a branching vascular network and aneurysmal dilations of varying number at the outer edge of the expanding lesion. The internal structure of the aneurysmal structures, often termed polyps, is controversial.
Type 2 MNV	Classic CNV	Neovascularization that originates from the choroid that traverses Bruch's membrane and the RPE monolayer and then proliferates in the subretinal space.
Mixed type 1 and type 2 MNV	Minimally classic CNV	OCT findings of both type 1 and type 2 MNV together. OCT angiography demonstrates neovascularization in the subretinal pigment epithelial and subretinal compartments.
Type 3 MNV	Retinal angiomatous proliferation	Neovascularization that originates from the retinal circulation, typically the deep capillary plexus, and grows toward the outer retina.
Retinal-choroidal anastomosis	Retinal-choroidal anastomosis	Aberrant connection from the retinal to the choroidal circulation.
Intraretinal fluid	Cystoid edema	Leakage in excess of the local capability of removal leading to accumulation of the fluid in retinal thickening and formation of cystoid spaces. The fluid in the retina may come from retinal vessels or a subretinal source if the external limiting membrane is not intact.
Subretinal fluid	Subretinal fluid	Leakage in excess of the local capability of removal leading to accumulation of the fluid under the retina that separates the neurosensory retina from the RPE. The fluid source generally is from underlying neovascularization in AMD in the context of an intact external limiting membrane.

Source: Spaide et al., Ophthalmology 2020;127:616-636; Adapted by William Blair Equity Research

Exhibit 2
Diagrams of Type 1, 2, and 3 Macular Neovascularization



Type 1: Ingrowth of vessels arises from the choriocapillaris and extends up to and under the retinal pigment epithelium.
Type 2: Ingrowth of vessels arises from the choriocapillaris and extends up through the RPE monolayer to proliferate in the subretinal space. To arrive in the subretinal space, the blood flow must traverse the sub-RPE space to reach the plane of neovascularization.
Type 3: When the regional proangiogenic-antiangiogenic balances shift in favor of neovascularization, proliferation of vessels occurs along a vector along the VEGF concentration gradient. The new vessels originate from and invade into tissues below the plane of the deep capillary plexus. Elevated cytokines, particularly VEGF levels, can induce vascular leakage and intraretinal hemorrhage in addition to stimulating angiogenesis.
Source: Spaide et al., Ophthalmology 2020;127:616-636; Adapted by William Blair Equity Research

Other Drivers of nAMD

While VEGF is a primary driver of nAMD, the complex pathophysiology suggests other pathways may also be involved. The retina is a complex neurovascular tissue made up of multiple cell types and a rich network of endothelial cells precisely layered in capillaries at various levels within the retina providing tightly regulated levels of oxygen. This has been clearly elucidated in models of retinopathy of prematurity, wherein hyperoxia results in obliteration of the retinal vasculature, and hypoxia promotes vascular growth and proliferation. VEGF was found to be spatially and temporally localized to the areas of vascular development, providing strong evidence that it is a driving force in the development of angiogenesis and in ischemic/hypoxic regulation of the retinal vasculature.

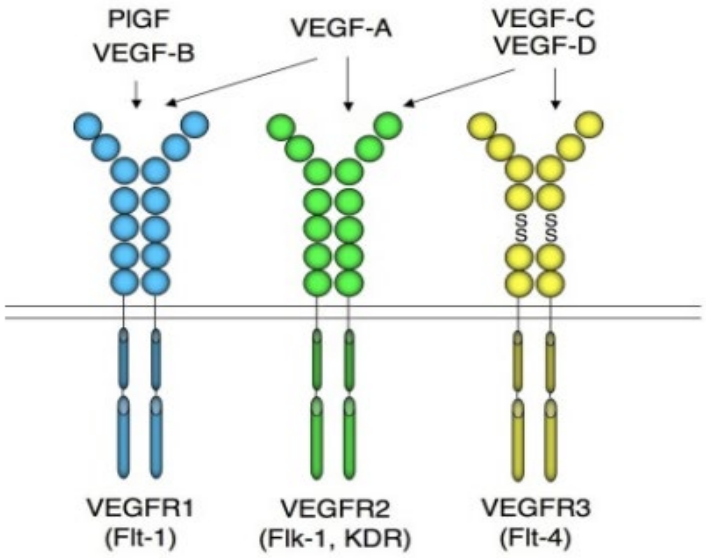
The retina is one of the most metabolically demanding tissues in the body, and as such, requires tightly regulated levels of oxygen. This high consumption of oxygen, combined with a high proportion of polyunsaturated fatty acids and exposure to visible light, makes it particularly susceptible to oxidative stress due to excessive production of reactive oxygen species (ROS), especially as our

natural systems to process ROS decline with age. Photoreceptors shed roughly 10% of their volume, and their outer segments (OS) basally regenerate roughly the same volume of cellular material, each day. This shed volume is phagocytosed by the RPE and metabolically processed into waste products that are ideally secreted through Bruch's membrane into the choroidal circulation. However, some of these products are more taxing to break down, leading to accumulation over time. Indeed, the presence of lipofuscin, a fluorescent material with significant phototoxic potential, in the RPE is one of the hallmarks of aging and is believed to accumulate due to the incomplete digestion of the shed OS in the RPE. Accumulation of lipofuscin and other toxic products in the RPE lysosomes including the bis-retinoid N-retinylidene-N-retinylethanolamine (A2E) inhibits phagolysosomal degradation of the OS by the RPE, and the fluorophores within these deposits sensitize lysosomes to the visible light spectrum, leading to cellular instability. In vitro and in vivo studies of ARPE-19 cells have demonstrated that treatment with A2E increases their expression of angiogenic factors and decreases the expression of anti-angiogenic factors, leading to increased CNV activity.

The accumulation of drusen between the RPE and Bruch's membrane is also an important step in the development of neovascular AMD (indeed, it is a defining characteristic of AMD). Acting as a mechanical barrier, it displaces RPE and photoreceptor cells, which can lead to distortion of vision, and reduces perfusion between the RPE and choroid. The accumulation of drusen is also believed to act as a stimulus for local activation of the complement system, driving inflammation, and contributes to local ischemia of RPE cells. Given the tight regulation of oxygen within the retina, RPE cells respond to this ischemic stress by releasing angiogenic stimuli such as hypoxia inducible factor 1 alpha (HIF-1α), which ultimately leads to neovascularization. In fact, under normal conditions, the basolateral RPE secretes VEGF in a polarized manner that is indispensable for the health of choroidal endothelial cells (CECs). In animal models, loss of VEGF secreted by basolateral RPE leads to CEC atrophy and significant thinning of the choriocapillaris. Whether anti-VEGF therapies lead to the same CEC atrophy is unclear. Aging RPE cells may also lose their polarity, leading them to secrete VEGF from the apical aspects of the cell surface, which could stimulate neovascularization.

Ultimately, these degenerative changes in the RPE, Bruch's membrane, and choriocapillaris create a proangiogenic and pro-inflammatory environment. Among the many proangiogenic factors expressed, the most studied, and likely most important, is VEGF. The VEGF signaling family plays an important role in vasculogenesis, angiogenesis, and vascular homeostasis in a wide range of tissues. It consists of six ligands (VEGF-A, VEGF-B, VEGF-C, VEGF-D, placental growth factor [PlGF], and the virally encoded VEGF-E) and three tyrosine kinase receptors (VEGFR-1, VEGFR-2, VEGFR-3), which are expressed on numerous cell types including the vascular endothelium. As shown in exhibit 3, the different ligands activate different receptors, which in turn lead to different downstream effects.

Exhibit 3
VEGF Ligands and Their Cognate Receptors



Source: Kawasaki and Miyazawa. Embryonic Stem Cells - Differentiation and Pluripotent Alternatives, 2011. William Blair Equity Research

The first component of the family to be identified (isolated and cloned in 1989), and the most widely studied, is VEGF-A, which is often referred to as simply VEGF unless further specification is required. Multiple isoforms of VEGF resulting from alternative splicing of mRNA from a single VEGF gene have been observed in humans. The most widely expressed in tissues is VEGF-$A_{165,}$ which plays a crucial role in pathologic angiogenesis and is believed to be the most physiologically relevant; however, other isoforms can have notably different properties, such as VEGF-A_{121}, which is highly diffusible, and VEGF-A_{189}, which is bound to the extracellular matrix (ECM) by heparan binding domains. Proteolysis plays an important part in regulating the biological activity of VEGF-A proteins, with proteolytic cleavage at the carboxyl terminus giving rise to biologically active isoforms and perhaps also inhibitory isoforms.

The expression of the VEGF-A gene is primarily stimulated by hypoxia, mediated by the hypoxia-inducible factor (HIF), but it is also mediated by other factors such as epidermal growth factor (EGF), platelet-derived growth factor (PDGF), basic fibroblast growth factor (bFGF), and hypoxia-independent pathways driven primarily by inflammation. VEGF-A binds to VEGFR1 (also known as FLT1) and VEGFR2 (also known as KDR and FLK1). While VEGFR2 is the main mediator of the roles of VEGF-A in cell proliferation, angiogenesis, and vessel permeabilization, it actually binds to VEGFR1 with about tenfold higher affinity, but a lack of consistent mitogenic effects suggests that at least in some cases it may be a decoy receptor. VEGF-A also interacts with the neuropilin co-receptors (NRP1 and NRP2), which can signal independently of VEGFRs and further influence VEGFR2 signaling (e.g., heparin-binding VEGF-A and PlGF binding to NRP-1 increases their binding affinity to VEGFR2). VEGF-A binding to VEGFR2 receptors on endothelial cell surfaces leads to dimerization and autophosphorylation, which activates multiple downstream cascades involved in proliferation, filopodial extension, chemotaxis, and ECM degradation.

While VEGF-A is the primary ligand for VEGFR2 and thus likely the most important driving factor in angiogenesis, several other signaling pathways may be implicated. First, while the primary receptor for VEGF-C and VEGF-D is VEGFR3, they can also activate VEGFR2. Though typically expressed at relatively low levels in the eye, studies have shown that VEGF-C/D can be upregulated

following treatment with anti-VEGF-A therapies, which could result in incomplete blockade of the pathway (though recent Phase III results with the VEGF-C/D trap sozinibercept may raise questions about the potential therapeutic benefits of blocking VEGF-C).

Beyond the VEGF family, several other pathways have been hypothesized to be important in angiogenesis. Among these, the angiopoietin/Tie2 pathway has received the most attention because of its role in vascular development, maintenance, and stability. Briefly, Ang-2 binding to Tie2 leads to the shedding of pericytes from endothelial cells, sensitizing the vasculature to VEGF and other proinflammatory factors. While there is good theoretical rationale for targeting the pathway, supported by preclinical data suggesting efficacy, and evidence of Ang-2 upregulation in the eyes of patients with neovascular retinal diseases, clinical studies targeting the Ang-2/Tie2 pathway have yet to conclusively show significant additive efficacy to VEGF inhibitors in nAMD. A multitude of other pathways have been hypothesized to contribute to nAMD, including PDGF/PDGFR2, TGF-β, bFGF, semaphorins, ANGPTL4, and the Wnt pathway; however, these have not shown any incremental clinical benefit to VEGF inhibition, have not yet made it to the clinic, or are still relatively early in clinical development.

Current Treatment Landscape in Neovascular AMD

Given the central role of VEGF in angiogenesis, anti-VEGF therapies have become the mainstay of treatment of nAMD and have transformed the disease since the approval of Macugen (pegaptanib) in 2004 and Lucentis (ranibizumab) in 2006. While the introduction of anti-VEGF-A therapies revolutionized the management of neovascular AMD starting in 2006, only modest advances have been made in the subsequent years; the introduction of additional therapies (exhibit 4) offered better convenience by extending dosing intervals, but has not improved on visual acuity outcomes beyond that achieved with Lucentis (exhibit 5). Specifically, up to 60% of patients experience a suboptimal vision recovery (defined as not reaching 20/40 vision), while 50% experience progressive disease activity (defined as unresolved fluid, hemorrhage, or progressive fibrosis) after one year of therapy. Even among those who achieve a good response, the treatment burden drives a high discontinuation rate, reported to be up to 30% after one year with further increases in subsequent years. Moreover, even among those who remain on therapy, visual acuity still declines with treatment over several years (exhibit 6).

Exhibit 4
Characteristics of Anti-VEGF Therapies for Retinal Diseases

	Bevacizumab*	Lucentis (ranibizumab)	Eylea (aflibercept)	Beovu (brolucizumab)	Vabysmo (faricimab)
First FDA approval	2004	2006	2011	2019	2022
Design	Humanized antibody	Fab fragment	Fc fusion protein	Single chain variable fragment	Bispecific monoclonal antibody
Targets	VEGF-A	VEGF-A	VEGF-A/B, PlGF	VEGF-A	VEGF-A/Ang-2
Molecular weight (kDa)	149	48	115	26	150
Dissociation constant (pM)	58	46	0.49	28.4	3
Vitreous half life (days)	4.9	9	9.1	3.1	7.5
Clinical dose for nAMD	1.25 mg	0.5 mg	2 mg / 8 mg	6 mg	6 mg

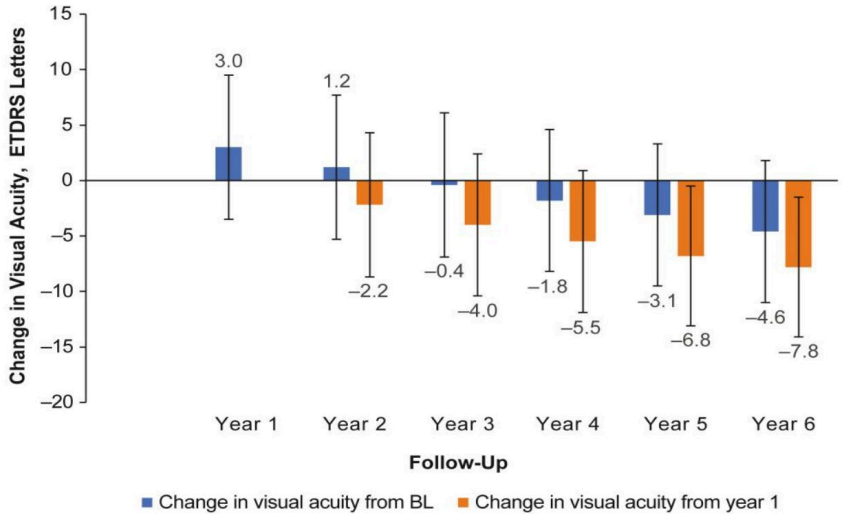
*Bevacizumab is not approved for retinal indications, but is widely used off label
Sources: FDA labels and review documents; William Blair Equity Research

Exhibit 5
Despite Several Product Approvals, None Have Shown Superior Visual Benefits Over Lucentis or Eylea

Product	Trial	Control arm	Difference (95% CI) in BCVA change vs. control at 1 year*
Eylea	VIEW 1	Lucentis	0.3 (-2.0, 2.5)
	VIEW 2	Lucentis	-0.9 (3.1, 1.3)
Beovu	HAWK	Eylea	-0.2 (-2.1, 1.8)
	HARRIER	Eylea	-0.7 (-2.4, 1.0)
Susvimo	ARCHWAY	Lucentis	-0.3 (-1.7, 1.1)**
Vabysmo	TENAYA	Eylea	0.7 (-1.1, 2.5)
	LUCERNE	Eylea	0.0 (-1.7, 1.8)
Eylea HD	PULSAR (Q12W)	Eylea	-1.0 (-2.9, 0.9)
	PULSAR (Q16W)	Eylea	-1.1 (-3.0, 0.7)

*From FDA labels; differences in mean or least-squares means. **Average of weeks 36 and 40
Source: FDA labels; William Blair Equity Research

Exhibit 6
Real-World Data Shows Declining Visual Acuity Over Time Even With Anti-VEGF Treatment



Mean change in visual acuity from baseline and from year 1 over time. Analysis based on a cohort of 160,423 eyes in the AAO IRIS registry with a first anti-VEGF injection and at least 2 years of follow-up with visual acuity data within the time frame of January 1, 2013, and June 6, 2020.

Source: Wykoff et al., Ophthalmol Sci, 2023 Oct 32;4(2):100421. Licensed under creative commons CC BY-NC-ND 4.0.

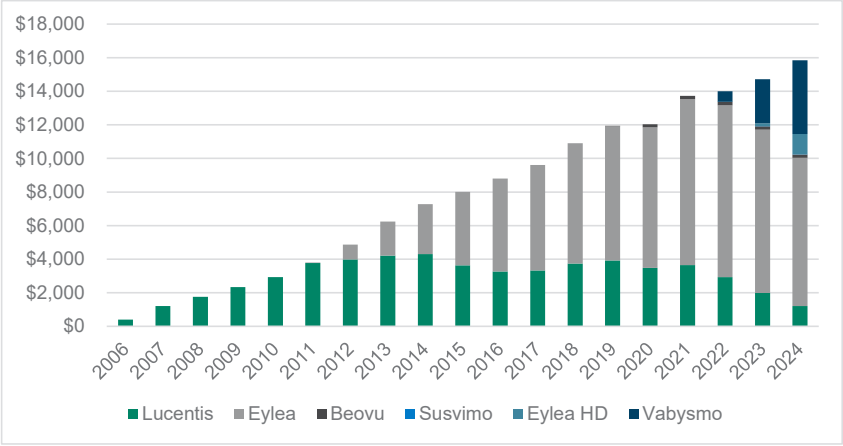
While many factors likely contribute to the decline in VA over time, one of the biggest reasons is believed to be undertreatment as patients and physicians continue to extend treatment intervals over time to minimize treatment burden. Perhaps not surprisingly, visiting the doctor every one to three months (for most patients) for an intravitreal injection is quite a burden, especially for elderly patients who often need assistance getting to the doctor (e.g., from a relative). This leads many physicians and patients to employ a "treat and extend" dosing paradigm, where they slowly increase the time between injections until they reach the maximum interval at which a given patient can be dosed without a recurrence of fluid in their retina. While this preserves much of the efficacy observed in clinical trials, real-world evidence suggests that visual acuity ultimately declines over several years of treatment, likely reflecting the inadequate suppression of VEGF in the retina with a treat-and-extend protocol and resulting fibrosis and scarring from the pulsatile

nature VEGF-suppression with such a treatment schedule. In some patients, this may also be driven by resistance to anti-VEGF therapies, perhaps driven by upregulation of other pathways in response to treatment.

While none of the anti-VEGF therapies approved after Lucentis demonstrated better visual acuity outcomes, the longer durability allowed for extended dosing intervals; as seen in exhibit 7, even modest improvements in durability on the order of days or weeks were enough for a new product to capture significant market share. Therefore, extending the durability of dosing intervals has been the focus of most new drug development in wet AMD since the approval of Lucentis in 2006. That largely remains the case today, with most development focused on extending durability to reduce the injection burden, although there is increasing recognition of the need for better treatments for patients who do not respond as well to anti-VEGF therapies, those "frequent flyers" who can barely extend treatment intervals even with the newest therapies (and a way to identify both of these populations a priori), and the negative consequences (e.g., fibrosis and scarring) from the pulsatile nature of existing therapies.

Exhibit 7

Global Sales of Branded Anti-VEGF Products Show That Increased Durability Is Rewarded With Market Share



Note: reflects revenue from all indications, not only nAMD
Sources: EvaluatePharma; William Blair Equity Research

Introduction of Biosimilars Adds to the Changing Commercial Dynamics

For most of the past two decades, all approved therapies for the treatment of nAMD have been branded therapies. While off-label Avastin (bevacizumab) has been widely used—either by cost-conscious physicians or as a forced step-through by insurers—there have been relatively few insurance hurdles to getting coverage for an anti-VEGF therapy.

This started to change in late 2021 with the approval of the first biosimilar to Lucentis, Byooviz (ranibizumab-nuna), followed by a second in Cimerli (ranibizumab-eqrn) in 2022, with list prices roughly 30%-40% below that of Lucentis. While these rapidly took market share (over 50% share of ranibizumab use after two years, according to Samsung Bioepis) and drove down average sales prices (ASPs), Lucentis had largely been supplanted by Eylea given the longer dosing interval of two months versus one month, so did not have a dramatic effect on the overall anti-VEGF market dynamics. In February 2025, Formycon announced that Sandoz, its commercialization partner for Cimerli, was in discussions with its licensing partner Bioeq about the future commercialization strategy due to increasing price discounts offered by ranibizumab providers in the U.S. Because of

the pricing situation, Sandoz temporarily suspended marketing activities for approximately one year with the aim of commercially realigning the product and tapping into new customer segments after reintroduction.

The introduction of Eylea biosimilars may lead to greater changes in the market given its position as the standard of care, although the market is already shifting away from Eylea toward Vabysmo and Eylea HD because of the longer durability. While five Eylea biosimilars have been approved by the FDA, ongoing litigation has prevented four of the five manufacturers from launching them to date. The sole risk-taker here is Amgen, which launched Pavblu (aflibercept-ayyh) in late 2024 at-risk. The company reported $31 million in revenue from nine weeks of sales in the fourth quarter of 2024, but it remains too early to gauge how the market will evolve in response to its launch.

In sum, the anti-VEGF market is set to see an additional layer of changing competitive dynamics over the coming years, driven by not only potential new entrants, but also the introduction of biosimilars and likely potential for increased payer management of the space. While the launch of biosimilars will likely accelerate the erosion of Eylea, we do not see it meaningfully impacting the trend of continuing to move patients to more durable therapies over time. To be sure, payers may implement additional barriers to getting patients on these longer-acting branded therapies like step-edits that may prevent some patients from progressing to newer therapies, but we ultimately believe retina specialists will be able to get patients on their preferred therapy in most cases, even if it takes a bit longer to get there. Indeed, many payers currently require step-through of Avastin before branded therapies, yet it holds only about 30% market share. It is conceivable that longer-acting therapies may actually offer a pharmacoeconomic benefit as they offer better outcomes and/or reduce the ancillary costs associated with physician visits and injections, though this of course could raise concerns about practice economics given that intravitreal injections have become an important part of many retina practices.

Development Pipeline in Neovascular AMD

The development pipeline for nAMD is active, with many companies seeking to develop therapies in the indication given the large, validated commercial opportunity and remaining unmet needs, and the potential to expand into other retinal vascular diseases. We include a selection of assets in clinical development in exhibit 8 and highlight some private companies developing new technologies in preclinical development below.

RevOpsis Therapeutics
RevOpsis is developing its lead asset RO-104, a fully human trispecific antibody with a unique format that lacks the Fc region targeting VEGF-A, VEGF-C, and Ang-2 for the treatment of nAMD. The company advanced RO-104 into IND-enabling studies in January 2025 and expects to move into the clinic in the second half of 2026. RO-104 was designed on the company's RevMod platform, which employs a modular plug-and-play approach to streamline and expedite the efficient discovery and development of multispecific biologics. The platform has a library of nearly 30 billion fully human antibody components in a structured phage display system that allows for rapid identification and assembly of multispecific product candidates.

Preclinically, RO-104 has shown similar inhibitory potency to aflibercept in a HUVEC assay of wound density and confluence using 4 nM VEGF-A. Analysis of wound density at 8, 10, and 12 hours post-treatment revealed an IC_{50} for VEGF-A of 5.65 nM for RO-104 compared to 14.97 nM for aflibercept, while analysis of wound confluence inhibition revealed an IC_{50} of 1.44 nM for RO-104 and 1.37 nM for aflibercept, suggesting RO-104 was highly effective at inhibiting VEGF-A.

Exhibit 8
Select Products in Clinical Development for Neovascular AMD

Company	Asset	Mechanism of Action	Modality	Route of Admin.	Status
Biologics					
Outlook Therapeutics	Lytenava (ONS-5010; bevacizumab-vikg)	Anti-VEGF	Antibody	IVT	Regulatory
Bio-Thera Solutions	BAT5906	Anti-VEGF	Antibody	IVT	Phase III
Kodiak Sciences	Tarcocimab tedromer (KSI-301)	Anti-VEGF	Antibody biopolymer conjugate	IVT	Phase III
Kodiak Sciences	Tabirafusp tedromer (KSI-501)	Anti-IL-6, VEGF trap	Antibody biopolymer conjugate	IVT	Phase III
RemeGen	RC28-E	Anti-VEGF/FGF	Fusion protein	IVT	Phase III
Innovent Biologics	Efdamrofusp alfa (IBI302)	Anti-VEGF / anti-C3b/C4b	Fusion protein	IVT	Phase III
Merck	Tiespectus (MK-8748; EYE201)	Undisclosed	Undisclosed	IVT	Phase I/II
Roche	Zifibancimig (RG6120)	Anti-VEGF/Ang-2	Dutafab	Port delivery system	Phase I/II
AffaMed	AM712 (ASKG712)	Anti-VEGF/ANG-2	Antibody	IVT	Phase I
PharmAbcine	PMC-403	Tie2 agonist	Antibody	IVT	Phase I
Kalaris	TH103	Anti-VEGF (trap)	Fusion protein	IVT	Phase I
Innovent Biologics	IBI333	Anti-VEGF-A/C	Fusion protein	IVT	Phase I
Small molecules					
Ocular Therapeutix	Axpaxli (OTX-TKI)	Anti-VEGFR/PDGFR	TKI	IVT	Phase III
Eyepoint	Duravyu (EYP-1901)	Anti-VEGFR/PDGFR	TKI	IVT	Phase III
Clearside	CLS-AX	Anti-VEGFR/PDGFR	TKI	Suprachoroidal	Phase III
Sylentis	SYL1801	Anti-NRARP receptor	siRNA	Topical	Phase II
TheratOcular Biotek	TO-O-1002 (MG-O-1002)	Anti-VEGFR	TKI	Topical	Phase II
Ashvattha Therapeutics	Migaldendranib (D-4517.2)	Anti-VEGFR/PDGFR/cKIT/CSF1R	TKI	Subcutaneous	Phase II
Kyowa Kirin	KHK4951	Anti-VEGFR	TKI	Topical	Phase II
Alcon	AR-14034 SR	Anti-VEGFR/PDGFR	TKI	IVT	Phase I/II
Caregen	CG-P5	Anti-VEGFR2	Peptide	Topical	Phase I
Olix Pharmaceuticals	OLX301A (OLX10212)	Undisclosed	siRNA	IVT	Phase I
Ocugenix Corporation	OCU-10-C-110	CXCR3 activator	TKI	IVT	Phase I
AiViva BioPharma	AIV007	Anti-VEGFR/PDGFR/FGFR/TGFβ	TKI	Periocular injection	Phase I
Gene therapies					
Regenxbio	RGX-ABBV-314	Anti-VEGF (trap)	AAV gene therapy	Subretinal / suprachoroidal	Phase III
Adverum	Ixoberogene soroparvovec (Ixo-vec; ADVM-022)	Anti-VEGF (trap)	AAV gene therapy	IVT	Phase III
Innostellar Biotherapeutics	LX102	Anti-VEGF (trap)	AAV gene therapy	Subretinal	Phase II
4D Molecular Therapeutics	4D-150	Anti-VEGF (trap) and anti-VEGF-C	AAV gene therapy	IVT	Phase II
Frontera Therapeutics	FT-003	anti-VEGF (trap)	AAV gene therapy	IVT	Phase II
Avirmax Biopharma	ABI-110 (AAV2.N54-VEGF Trap)	Anti-VEGF (trap)	AAV gene therapy	IVT	Phase I/II
Exegenesis Bio	EXG102-031	Anti-VEGF/Ang-2	AAV gene therapy	Subretinal	Phase I/II
Chengdu Origen Biotechnology	KH658	Anti-VEGF	AAV gene therapy	Suprachoroidal	Phase I/II
Avirmax Biopharma	ABI-110 (AAV2.N54-VEGF Trap)	Anti-VEGF (trap)	AAV gene therapy	IVT	Phase I/II
Neuracle Genetics	NG101	Anti-VEGF (trap)	AAV gene therapy	Subretinal	Phase I/II
Skyline Therapeutics	SKG0106	Anti-VEGF	AAV gene therapy	IVT	Phase I
Chengdu Genevector Biotechnology	JWK001	Anti-VEGF	AAV gene therapy	Subretinal	Phase I
Vanotech	KH631 / VAN-2201	Anti-VEGF (trap)	AAV gene therapy	Subretinal	Phase I

Sources: BioCentury; company websites; clinicaltrials.gov; William Blair Equity Research

In a separate in vitro study, the company used ELISA to evaluate the EC_{50} of RO-104 for each of its three targets (VEGF-A, VEGF-C, and Ang-2) starting at 10 nM using threefold dilutions to evaluate dose response. The results demonstrated an EC_{50} of RO-104 binding to VEGF-A of 17.6 pM, compared with 19.1 pM for aflibercept. The EC_{50} for VEGF-C was 92.9 pM and for Ang-2 was 26.5 pM, further supporting the similar potency against VEGF-A as aflibercept, while also demonstrating potent inhibition of its other targets. Then the same assay was run using faricimab as the control; the results demonstrated an EC_{50} for VEGF-A of 5.0 pM for RO-104 and 22.8 pM for faricimab, while the EC_{50} for Ang-2 was 10.7 pM compared with 676.4 pM for faricimab. As would be expected, faricimab showed no affinity for VEGF-C, while RO-104 demonstrated an EC_{50} for VEGF-C of 19.2 pM.

A separate ELISA study evaluating competitive binding of RO-104 to its ligands showed similar results when compared with faricimab as the control. Target-receptor competition showed RO-104 had slightly better IC_{50} for VEGF-A than faricimab (17.94 nM vs. 26.11 nM), a markedly better IC_{50} for Ang-2 (2.098 nM vs. 38.46 nM), and an IC_{50} for VEGF-C of 3.775 nM, while faricimab showed no measurable VEGF-C inhibition at concentrations up to 100 nM.

Given the promising in vitro results, the company evaluated the efficacy of RO-104 in a rat laser-induced CNV model using aflibercept and faricimab for the control. On day 1, an 810 nm diode laser was used to create four lesions of the Bruch's membrane in each eye, which cause progressive CNV that is readily quantifiable in one week by fluorescence angiography (FA). After the laser on day 1, all rats received 5 µL IVT injection. The four study arms (n=6 rats in each) received PBS vehicle (group 1), 0.066 mg aflibercept (group 2), 0.1 mg faricimab (group 3), or 0.1 mg RO-104 (group 4), giving the same molar concentration of each drug. Analysis of FA on day 8 determined mean wound areas for all reliably observable lesions. Compared with the control group, faricimab reduced the mean lesion area by 19% (p=0.2201) and aflibercept by 29% (p=0.2036), while RO-104 reduced the mean area by 48% (p=0.0061). While the low efficacy with faricimab may reflect poor selectivity for rat VEGF, the 29% with aflibercept is in line with historical results, so the superior efficacy observed with RO-104 suggests the potential for significant therapeutic efficacy with RO-104. Whether this is because of its inhibition on VEGF-C, greater potency for Ang-2, or other drug attributes is not clear, but the results nonetheless suggest that RO-104 has the potential to offer meaningful efficacy in neovascular retinal diseases.

The company has also completed a laser-induced CNV study in nonhuman primates (NHPs) evaluating two doses of RO-104 (0.27 mg and 1.09 mg; molar equivalent to Lucentis 0.5 mg and Eylea 2 mg), compared with vehicle (n=2 in vehicle group and n=3 in each RO-104 group). The laser injury was induced on day 0, and baselines were assessed on day 14 before treatment on day 15 and assessment of efficacy on day 28. Lesions were graded on a scale of 1-4, and the high-dose RO-104 achieved complete regression of all grade 3 and 4 lesions to grade 1 or 2 (no leakage), while the vehicle arm saw no meaningful changes in the number of grade 3 or 4 lesions. Overall, the high dose of RO-104 significantly reduced the severity of clinically assessed CNV compared with both the low dose and vehicle ($p<0.001$ each), and also significantly reduced the CNV damage area compared with vehicle (n=0.038). There were no notable changes in intraocular pressure, no intraocular inflammation with the high dose but one case of low-grade inflammation observed in a single animal with the low dose that self-resolved, and no other untoward clinical observations. While the study did not include Eylea or Vabysmo, the results look competitive with what has previously been reported for both of those products in a similar model.

In addition, the company has reported a half-life of 3.72 days (Lucentis and Eylea have been reported as 2.88 and 3.92 days, respectively) and has suggested it believes it can reach dosing intervals up to six months after loading in a significant portion of patients.

While still early in development, the preclinical data to date suggest RO-104 could offer a differentiated efficacy profile in the treatment of neovascular retinal disease such as nAMD. The company has composition-of-matter patents through 2044 with opportunities to extend beyond 2044 and can manufacture RO-104 with high yields and purity.

Valitor

Valitor is developing a novel approach to creating long-acting anti-VEGF therapies by leveraging its multivalent polymer (MVP) technology platform. Briefly, the platform is based on proprietary multivalent biopolymers that can be loaded with multiple copies of bioactive molecules. While most efforts to increase tissue retention add polymers to a single antibody to increase its size, Valitor takes the opposite approach, adding multiple active moieties to each polymer, offering the increased retention of a larger molecule but with anticipated potency benefits that come from having multiple conjugated antibodies. Both the biopolymers and bioactive molecules are interchangeable, allowing the company to create novel macromolecular entities with independent control over multiple drug attributes.

The company's lead asset is VLTR-599, a long-acting treatment in preclinical development for nAMD. VLTR-599 is built on a backbone of hyaluronic acid (HA), a biocompatible polymer that is the primary constituent of the vitreous matrix (and that has a history of safe use in the eye), to which multiple single-domain anti-VEGF antibodies are covalently linked. While the overall size of VLTR-599 is large, it is ultimately a linear molecule, which the company believes should allow for good tissue penetration as it can still thread through small pore sizes, and it has a similar anti-VEGF potency to Eylea and Vabysmo.

In pilot preclinical toxicology studies, anti-VEGF MVPs showed minimal immune response after repeat dosing in NHPs, with results on the Semiquantitative Preclinical Ocular Toxicity Score (SPOTS) over 125 days that were below the historically reported levels for Lucentis in the literature. In addition, antidrug antibodies were undetectable in the aqueous humor, and only detectable in the serum at low titers and periodically over the duration of the study, though they appear to be increasing toward the end of the 125-day study. In a laser-induced mouse model of CNV, VLTR-599 inhibited CNV lesions to a similar degree as Eylea, with both achieving reductions between 30% and 40% compared with vehicle at equimolar doses, supporting its similar potency to current agents and therapeutic potential.

Ocular PK studies in rabbits showed that anti-VEGF MVPs had a half-life following IVT injection ranging from 12-16 days, with VLTR-599 having a half-life of about 12.5 days. Though not a head-to-head study, this represents a marked increase over prior reports of ocular PK in rabbits of Vabysmo (4.3 days), Lucentis (4.5 days) and Eylea (4.5 days). Translating this into humans using established models to predict clinical efficacy would suggest the vast majority of patients could achieve a six-month treatment interval with VLTR-599, with some going even further between injections.

Valitor aims to initiate IND-enabling studies in 2026 with the goal of entering the clinic as early as late 2026. Beyond VLTR-599, the company already has two early-stage collaborations with ophthalmology pharmaceutical companies with two distinct APIs. It has demonstrated that the chemistry is compatible to use these APIs on its MVP platform and retain the right potency, and is conducting early preclinical work with them ahead of potential licensing decisions. While Valitor is initially focusing on clinically validated mechanisms, it is also looking at other mechanisms that are in the process of clinical validation, as the interchangeable nature of the platform could set the company up to be a fast follower to any newly approved mechanisms in ophthalmology.

The prices of the common stock of other public companies mentioned in this report follow:

4D Molecular Therapeutics, Inc.	$2.77
Adverum Biotechnologies, Inc.	$3.86
Amgen Inc. (Outperform)	$294.39
Clearside Biomedical, Inc.	$0.86
EyePoint Pharmaceuticals, Inc.	$4.61
Kalaris Therapeutics, Inc. (Outperform)	$7.08
Kodiak Sciences, Inc.	$2.35
Novartis AG	$105.85
Ocular Therapeutix Inc. (Outperform)	$6.36
Regeneron Pharmaceuticals, Inc.	$573.45
Regenxbio, Inc,	$6.29
Roche Holding Ltd	$37.65
Sandoz Group Ltd.	$38.00

IMPORTANT DISCLOSURES

William Blair or an affiliate is a market maker in the security of Ocular Therapeutix, Inc. and Kalaris Therapeutics, Inc.

William Blair or an affiliate expects to receive or intends to seek compensation for investment banking services from Ocular Therapeutix, Inc. and Kalaris Therapeutics, Inc. or an affiliate within the next three months.

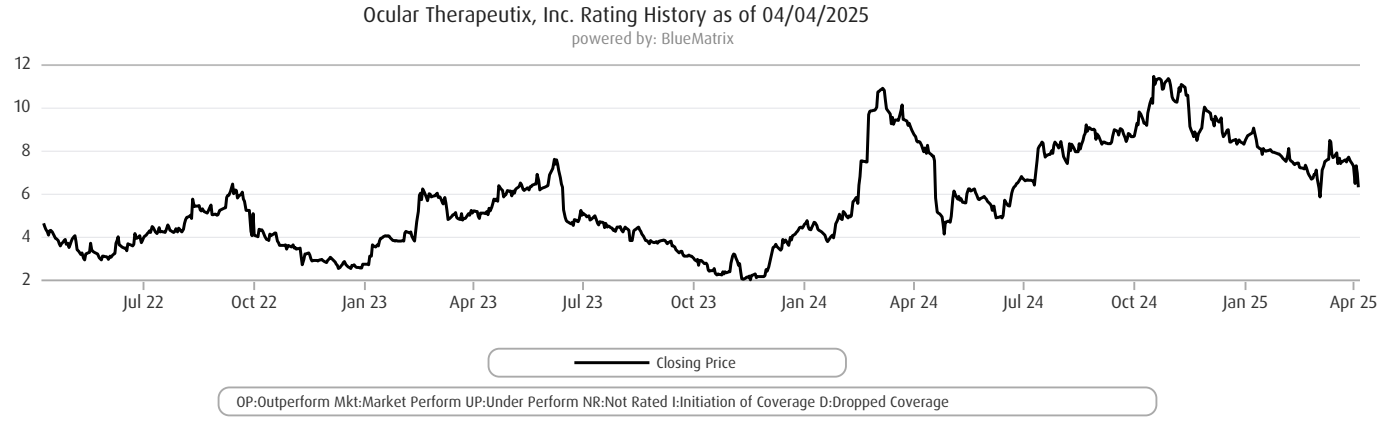
Officers and employees of William Blair or its affiliates (other than research analysts) may have a financial interest in the securities of Ocular Therapeutix, Inc. and Kalaris Therapeutics, Inc.

This report is available in electronic form to registered users via R*Docs™ at https://williamblairlibrary.bluematrix.com or www.williamblair.com.

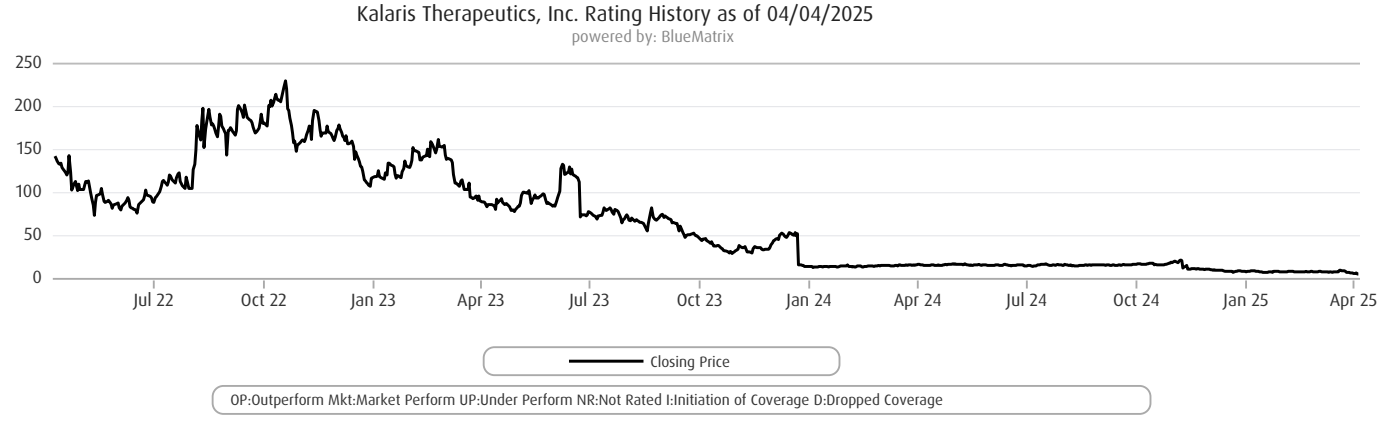
Please contact us at +1 800 621 0687 or consult https://www.williamblair.com/equity-research/coverage for all disclosures.

Lachlan Hanbury-Brown attests that 1) all of the views expressed in this research report accurately reflect his/her personal views about any and all of the securities and companies covered by this report, and 2) no part of his/her compensation was, is, or will be related, directly or indirectly, to the specific recommendations or views expressed by him/her in this report. We seek to update our research as appropriate. Other than certain periodical industry reports, the majority of reports are published at irregular intervals as deemed appropriate by the research analyst.

DOW JONES: 37965.60
S&P 500: 5062.25
NASDAQ: 15603.30



Source: FactSet & William Blair



Source: FactSet & William Blair

Additional information is available upon request.

Current Rating Distribution (as of April 8, 2025):

Coverage Universe	Percent	Inv. Banking Relationships *	Percent
Outperform (Buy)	71	Outperform (Buy)	10
Market Perform (Hold)	29	Market Perform (Hold)	1
Underperform (Sell)	1	Underperform (Sell)	0

*Percentage of companies in each rating category that are investment banking clients, defined as companies for which William Blair has received compensation for investment banking services within the past 12 months.

The compensation of the research analyst is based on a variety of factors, including performance of his or her stock recommendations; contributions to all of the firm's departments, including asset management, corporate finance, institutional sales, and retail brokerage; firm profitability; and competitive factors.

OTHER IMPORTANT DISCLOSURES

Stock ratings and valuation methodologies: William Blair & Company, L.L.C. uses a three-point system to rate stocks. Individual ratings reflect the expected performance of the stock relative to the broader market (generally the S&P 500, unless otherwise indicated) over the next 12 months. The assessment of expected performance is a function of near-, intermediate-, and long-term company fundamentals, industry outlook, confidence in earnings estimates, valuation (and our valuation methodology), and other factors. Outperform (O) - stock expected to outperform the broader market over the next 12 months; Market Perform (M) - stock expected to perform approximately in line with the broader market over the next 12 months; Underperform (U) - stock expected to underperform the broader market over the next 12 months; not rated (NR) - the stock is not currently rated. The valuation methodologies include (but are not limited to) price-to-earnings multiple (P/E), relative P/E (compared with the relevant market), P/E-to-growth-rate (PEG) ratio, market capitalization/revenue multiple, enterprise value/EBITDA ratio, discounted cash flow, and others. Stock ratings and valuation methodologies should not be used or relied upon as investment advice. Past performance is not necessarily a guide to future performance.

The ratings and valuation methodologies reflect the opinion of the individual analyst and are subject to change at any time.

Our salespeople, traders, and other professionals may provide oral or written market commentary, short-term trade ideas, or trading strategies-to our clients, prospective clients, and our trading desks-that are contrary to opinions expressed in this research report. Certain outstanding research reports may contain discussions or investment opinions relating to securities, financial instruments and/or issuers that are no longer current. Investing in securities involves risks. This report does not contain all the material information necessary for an investment decision. Always refer to the most recent report on a company or issuer. Our asset management and trading desks may make investment decisions that are inconsistent with recommendations or views expressed in this report. We will from time to time have long or short positions in, act as principal in, and buy or sell the securities referred to in this report. Our research is disseminated primarily electronically, and in some instances in printed form. Research is simultaneously available to all clients. This research report is for our clients only. No part of this material may be copied or duplicated in any form by any means or redistributed without the prior written consent of William Blair & Company, L.L.C.

This is not in any sense an offer or solicitation for the purchase or sale of a security or financial instrument.

The factual statements herein have been taken from sources we believe to be reliable, but such statements are made without any representation as to accuracy or completeness or otherwise, except with respect to any disclosures relative to William Blair or its research analysts. Opinions expressed are our own unless otherwise stated and are subject to change without notice. Prices shown are approximate.

This report or any portion hereof may not be copied, reprinted, sold, or redistributed or disclosed by the recipient to any third party, by content scraping or extraction, automated processing, or any other form or means, without the prior written consent of William Blair. Any unauthorized use is prohibited.

If the recipient received this research report pursuant to terms of service for, or a contract with William Blair for, the provision of research services for a separate fee, and in connection with the delivery of such research services we may be deemed to be acting as an investment adviser, then such investment adviser status relates, if at all, only to the recipient with whom we have contracted directly and does not extend beyond the delivery of this report (unless otherwise agreed specifically in writing). If such recipient uses these research services in connection with the sale or purchase of a security referred to herein, William Blair may act as principal for our own account or as riskless principal or agent for another party. William Blair is and continues to act solely as a broker-dealer in connection with the execution of any transactions, including transactions in any securities referred to herein.

For important disclosures, please visit our website at williamblair.com.

This material is distributed in the United Kingdom and the European Economic Area (EEA) by William Blair International, Ltd., authorised and regulated by the Financial Conduct Authority (FCA). William Blair International, Limited is a limited liability company registered in England and Wales with company number 03619027. This material is only directed and issued to persons regarded as Professional investors or equivalent in their home jurisdiction, or persons falling within articles 19 (5), 38, 47, and 49 of the Financial Services and Markets Act of 2000 (Financial Promotion) Order 2005 (all such persons being referred to as "relevant persons"). This document must not be acted on or relied on by persons who are not "relevant persons."

This report is being furnished in Brazil on a confidential basis and is addressed to the addressee personally, and for its sole benefit. This does not constitute an offer or solicitation for the purchase or sale of a security by any means that would constitute a public offering in Brazil under the regulations of the Brazilian Securities and Exchange Commission (*Comissão de Valores Mobiliários*) or an unauthorized distribution under Brazilian laws and regulations. The securities are authorized for trading on non-Brazilian securities markets, and this report and all the information herein is intended solely for professional investors (as defined by the applicable Brazilian regulation) who may only acquire these securities through a non-Brazilian account, with settlement outside Brazil in a non-Brazilian currency.

William Blair & Company, L.L.C. licenses and applies the SASB Materiality Map® and SICSTM in our work.

William Blair

Equity Research Directory

John Kreger, Partner Director of Research +1 312 364 8612
Kyle Harris, CFA, Partner Operations Manager +1 312 364 8230

Scott Hansen, Partner Associate Director of Research +1 212 245 6526

CONSUMER

Sharon Zackfia, CFA, Partner +1 312 364 5386
Group Head–Consumer
Lifestyle and Leisure Brands, Restaurants, Automotive/E-commerce

Jon Andersen, CFA, Partner +1 312 364 8697
Consumer Products

Phillip Blee, CPA +1 312 801 7874
Home and Outdoor, Automotive Parts and Services, Discount and Convenience

Dylan Carden +1 312 801 7857
E-commerce, Specialty Retail

ECONOMICS

Richard de Chazal, CFA +44 20 7868 4489

ENERGY AND SUSTAINABILITY

Jed Dorsheimer +1 617 235 7555
Group Head–Energy and Sustainability
Generation, Efficiency, Storage

Tim Mulrooney, Partner +1 312 364 8123
Sustainability Services

FINANCIAL SERVICES AND TECHNOLOGY

Adam Klauber, CFA, Partner +1 312 364 8232
Group Head–Financial Services and Technology
Financial Analytic Service Providers, Insurance Brokers, Property & Casualty Insurance

Andrew W. Jeffrey, CFA +1 415 796 6896
Fintech

Cristopher Kennedy, CFA +1 312 364 8596
Fintech, Specialty Finance

Jeff Schmitt +1 312 364 8106
Wealthtech, Wealth Management, Capital Markets Technology

GLOBAL SERVICES

Tim Mulrooney, Partner +1 312 364 8123
Group Head–Global Services
Commercial and Residential Services

Andrew Nicholas, CPA +1 312 364 8689
Consulting, HR Technology, Information Services

Trevor Romeo, CFA +1 312 801 7854
Staffing, Waste and Recycling

HEALTHCARE

Biotechnology

Matt Phipps, Ph.D., Partner +1 312 364 8602
Group Head–Biotechnology

Sami Corwin, Ph.D. +1 312 801 7783

Lachlan Hanbury-Brown +1 312 364 8125

Andy T. Hsieh, Ph.D., Partner +1 312 364 5051

Myles R. Minter, Ph.D., Partner +1 617 235 7534

Sarah Schram, Ph.D. +1 312 364 5464

Healthcare Technology and Services

Ryan S. Daniels, CFA, Partner +1 312 364 8418
Group Head–Healthcare Technology and Services
Healthcare Technology, Healthcare Services

Margaret Kaczor Andrew, CFA, Partner +1 312 364 8608
Medical Technology

Brandon Vazquez, CFA +1 212 237 2776
Dental, Animal Health, Medical Technology

Life Sciences

Matt Larew, Partner +1 312 801 7795
Life Science Tools, Bioprocessing, Healthcare Delivery

Andrew F. Brackmann, CFA +1 312 364 8776
Diagnostics

Max Smock, CFA +1 312 364 8336
Pharmaceutical Outsourcing and Services

INDUSTRIALS

Brian Drab, CFA, Partner +1 312 364 8280
Co-Group Head–Industrials
Advanced Manufacturing, Industrial Technology

Ryan Merkel, CFA , Partner +1 312 364 8603
Co-Group Head–Industrials
Building Products, Specialty Distribution

Louie DiPalma, CFA +1 312 364 5437
Aerospace and Defense, Smart Cities

Ross Sparenblek +1 312 364 8361
Diversified Industrials, Robotics, and Automation

TECHNOLOGY, MEDIA, AND COMMUNICATIONS

Jason Ader, CFA, Partner +1 617 235 7519
Co-Group Head–Technology, Media, and Communications
Infrastructure Software

Arjun Bhatia, Partner +1 312 364 5696
Co-Group Head–Technology, Media, and Communications
Software

Dylan Becker, CFA +1 312 364 8938
Software

Louie DiPalma, CFA +1 312 364 5437
Government Technology

Jonathan Ho, Partner +1 312 364 8276
Cybersecurity, Security Technology

Sebastien Naji +1 212 245 6508
Infrastructure Software, Semiconductor and Infrastructure Systems

Maggie Nolan, CPA, Partner +1 312 364 5090
IT Services

Jake Roberge +1 312 364 8056
Software

Ralph Schackart III, CFA, Partner +1 312 364 8753
Internet and Digital Media

Stephen Sheldon, CFA, CPA, Partner +1 312 364 5167
Vertical Technology – Real Estate, Education, Restaurant/Hospitality

EDITORIAL AND SUPERVISORY ANALYSTS

Steve Goldsmith, Head Editor and SA +1 312 364 8540

Audrey Majors, Editor and SA +1 312 364 8992

Beth Pekol Porto, Editor and SA +1 312 364 8924

Lisa Zurcher, Editor and SA +44 20 7868 4549