Kalaris Therapeutics

Kalaris Company Overview

April 2026

Forward-Looking Statements & Disclaimer

This presentation contains "forward-looking statements" within the meaning of the U.S. Private Securities Litigation Reform Act of 1995 and Section 21E of the Securities Exchange Act of 1934, as amended, that involve substantial risk and uncertainties.

All statements, other than statements of historical fact, contained in this presentation, including statements regarding the strategy, future operations, future financial position, projected costs, prospects, plans and objectives of management of Kalaris, the therapeutic potential of TH103 for neovascular Age-related Macular Degeneration and other exudative and neovascular retinal diseases, the anticipated timeline for reporting data from the ongoing Phase 1a clinical trial of TH103 and the ongoing Phase 1b/2 clinical trial of TH103, plans to advance TH103 into Phase 3 clinical trials and to develop TH103 for additional indications, plans to improve the manufacturing process for TH103 and the sufficiency of Kalaris' cash resources for the period anticipated, are forward-looking statements. The words "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "might," "plan," "potential," "predict," "project," "should," "target," "would" and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. These statements are based on current expectations and beliefs of the management of Kalaris as well as assumptions made by, and information currently available to, the management of Kalaris and are subject to risks and uncertainties. There can be no assurance that future developments affecting Kalaris will be those that it has anticipated. Actual results or events could differ materially from the plans, intentions and expectations disclosed in these forward-looking statements as a result of various important factors, including: risks associated with the clinical development and regulatory approval of its product candidate, including potential delays in the completion of clinical trials; expectations regarding the therapeutic benefits, clinical potential and clinical development of TH103; the timing of and Kalaris' ability to enroll patients in clinical trials; whether results from preclinical studies and initial data from early clinical trials will be predictive of the final results of the clinical trials or future trials; dependence on third parties for the development and manufacture of TH103; risks related to the inability of Kalaris to obtain sufficient additional capital to continue to advance its product candidate; uncertainties in obtaining successful clinical results for product candidates and unexpected costs that may result therefrom; risks related to the failure to realize any value from any product candidates being developed and anticipated to be developed in light of inherent risks and difficulties involved in successfully bringing product candidates to market; the ability to obtain, maintain, and protect intellectual property rights related to product candidates; changes in regulatory requirements and government incentives; Kalaris' competitive position and expectations regarding developments and projections relating to its competitors and any competing therapies that are or become available; the risk of involvement in current and future litigation; and such other factors as are set forth in Kalaris' public filings with the U.S. Securities and Exchange Commission, including, but not limited to, those described under the heading "Risk Factors".

Kalaris may not actually achieve the plans, intentions or expectations disclosed in its forward-looking statements, and you should not place undue reliance on its forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements Kalaris makes. The forward-looking statements contained in this presentation are made as of the date of this presentation, and Kalaris does not assume any obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by applicable law.

This presentation also contains estimates, projections and other statistical data made by independent parties and by Kalaris relating to market size and growth and other data about Kalaris' industry and business. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. Kalaris has not independently verified the accuracy and completeness of the information obtained by third parties included in this presentation. In addition, projections, assumptions and estimates of Kalaris' future performance and the future performance of the market in which Kalaris operates are necessarily subject to high degree of uncertainty and risk.

Your Vision
Our Mission

Kalaris is a clinical stage biopharmaceutical company dedicated to the development and commercialization of treatments for prevalent retinal diseases

Our lead asset, TH103, was **invented by Dr. Napoleone Ferrara**, whose pioneering research established the anti-VEGF class of therapies for retinal and oncology diseases, to address major remaining unmet needs

TH103 is an anti-VEGF therapeutic specifically engineered to achieve extended intraocular retention with enhanced VEGF inhibition

Kalaris Therapeutics

VEGF = Vascular Endothelial Growth Factor

Potential best in class, dual-action, anti-VEGF therapeutic

TH103 was engineered by Dr. Napoleone Ferrara for **extended intraocular retention** with **enhanced VEGF inhibition**

Early clinical data from Phase 1a SAD study **validates molecular design**

Ongoing Phase 1b/2 multi-dose trial

Preliminary **Phase 1b/2 data expected in 1H 2027**

$50MM oversubscribed private placement[1] in December 2025 fortified balance sheet

Sufficient **cash runway to fund company into Q4 2027**

Kalaris Therapeutics

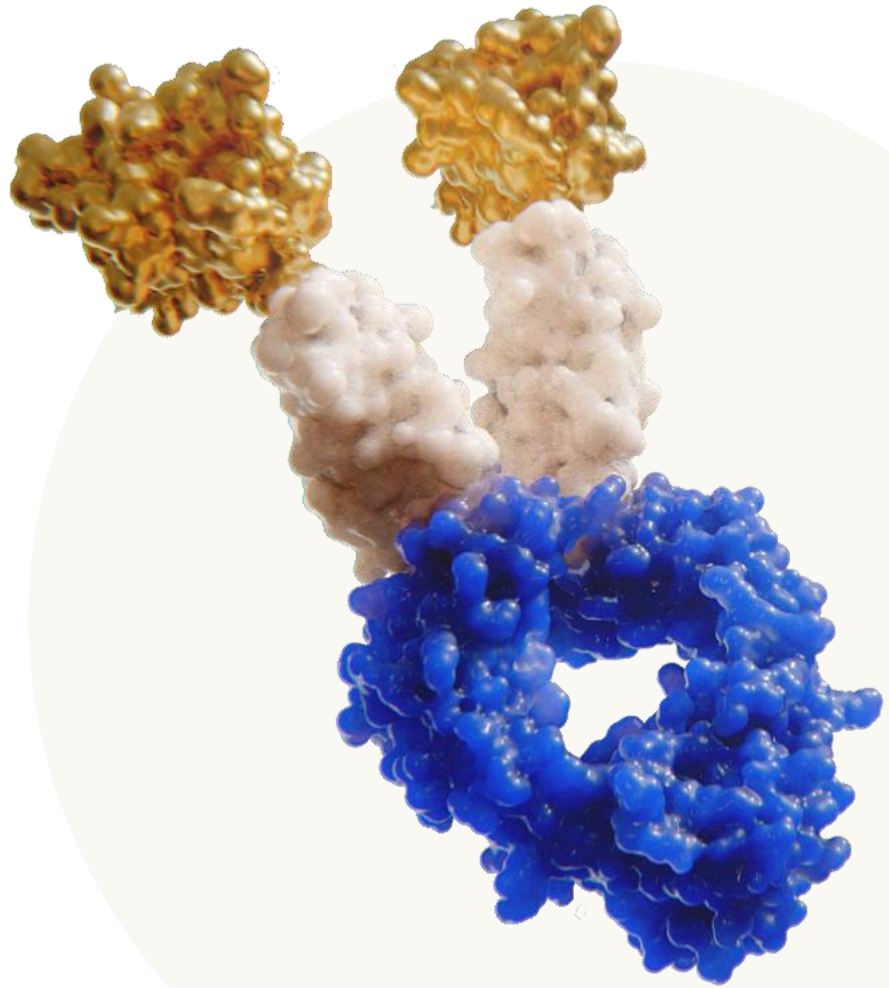
1) Private placement included participation from both new and existing investors, including ADAR1 Capital Management, Coastlands Capital, Invus, RTW Investments, Samsara BioCapital, Woodline Partners LP and others.

TH103

Engineered to improve upon a drug class that has helped millions of patients, with **optimization built directly into the molecule itself.**

Fully humanized, recombinant fusion protein designed for intravitreal delivery, with **potential to be best-in-class for neovascular and exudative retinal diseases.**

Targets VEGF as a soluble decoy receptor with high affinity for both VEGF and HSPG, **engineered for increased and longer-acting activity.**

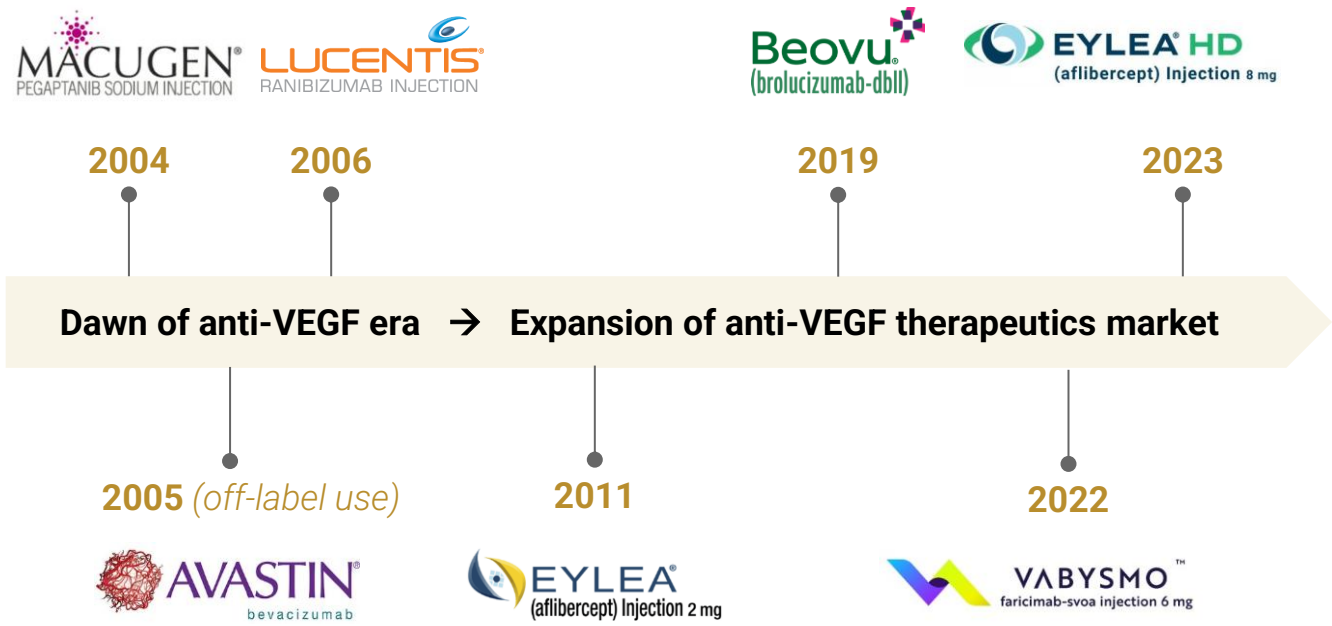


HSPG = Heparan Sulfate Proteoglycans

Kalaris Therapeutics

Anti-VEGF Therapeutics Background

Anti-VEGF therapy has revolutionized treatment for major retinal diseases, a global market projected to grow to over $18B by 2029



Kalaris Therapeutics

NEXT-GEN ANTI-VEGF

TH103

Kalaris is focused on driving the next wave of innovation for **retinal neovascular / exudative disease**

Sources: 2025 Retinal Pharmaceuticals Market Report, Market Scope October 2025; FDA Approvals

Unmet need remains high, with suboptimal real-world outcomes

Onerous visit frequency

Best outcomes may require **clinic visits as frequently as every 1-2 months** for monitoring and injections.

Current Solution

Physicians attempt to **extend the time between patient visits,** reducing injection frequency.

Suboptimal Outcomes

Reduced injection frequency can lead to **undertreatment and reduced efficacy.**

"Although multiple anti-VEGF therapies exist, unmet need remains high owing to treatment underutilization…"[1]

…regular treatment and monitoring requires substantial time commitment and may contribute to poor compliance. This treatment burden has been recognized by ophthalmologists; consequently, personalized treatment strategies attempt to balance the treatment burden against potentially reduced efficacy"[1]

Kalaris Therapeutics

Sources: 1) Mulligan, K., Seabury, S. A., Dugel, P. U., Blim, J. F., Goldman, D. P., & Humayun, M. S. (2020). Economic value of anti–vascular endothelial growth factor treatment for patients with wet age-related macular degeneration in the United States. JAMA ophthalmology, 138(1), 40-47.

Recognizing persistent unmet need, our lead asset was developed by VEGF pioneering scientist, Napoleone Ferrara

- **Co-discoverer of VEGF and VEGF isoforms** while at Genentech
- **Scientist** behind Anti-VEGF Agents:

AVASTIN® bevacizumab LUCENTIS® RANIBIZUMAB INJECTION **TH103**

- **Winner** of Major Awards including Lasker Award, Champalimaud Vision Award and Breakthrough Prize in Life Sciences

Napoleone Ferrara, MD

Kalaris Co-Founder

Genentech Fellow | Professor, UCSD

Our Solution: TH103

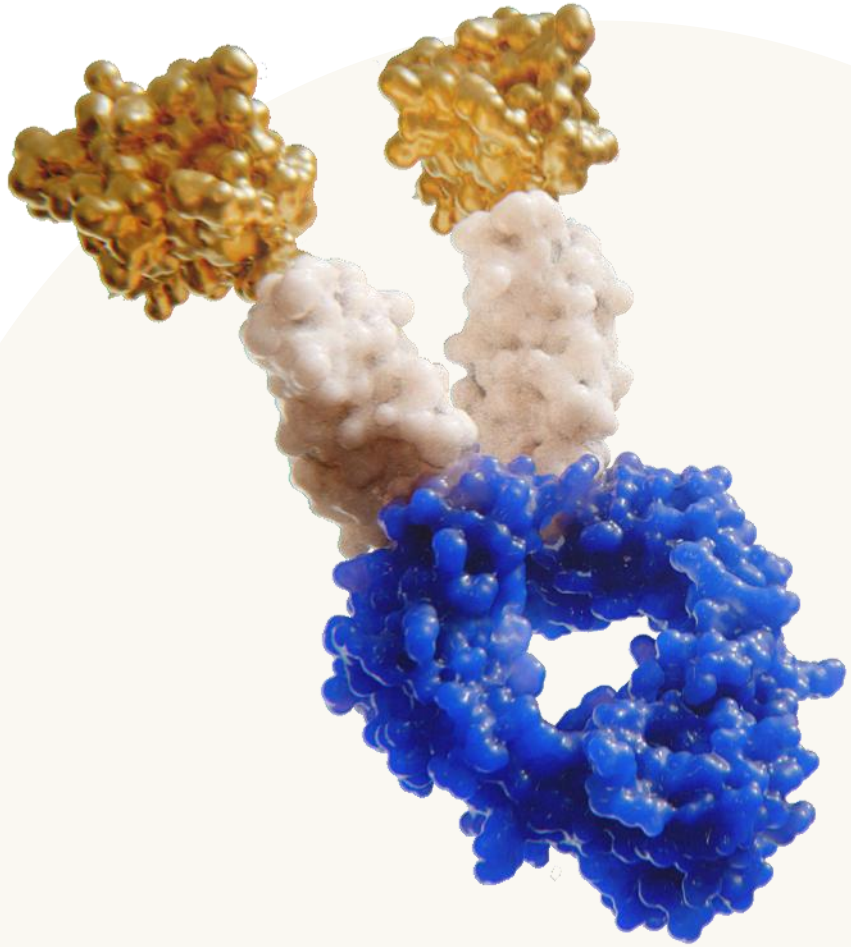
TH103: Dual-targeting, next generation drug engineered to address major unmet needs in retina disease

Optimized VEGF Binding:

Leverages higher-affinity VEGFR1[1], potentially leading to enhanced VEGF inhibition

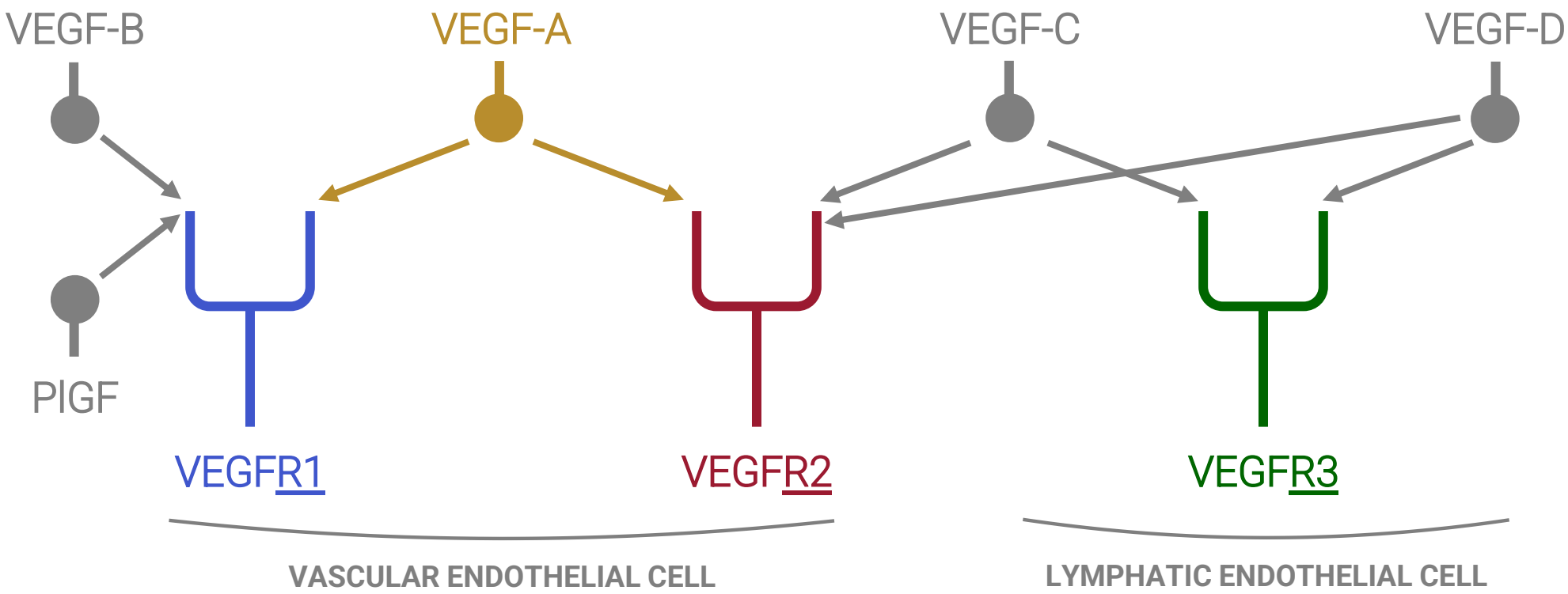
Extended Ocular Retention:

Leverages high-affinity binding to HSPG[2], potentially providing prolonged retinal retention and driving enhanced efficacy and/or durability



Source: 1) Holash, J., Davis, S., Papadopoulos, N., Croll, S. D., Ho, L., Russell, M., ... & Rudge, J. S. (2002). VEGF-Trap: a VEGF blocker with potent antitumor effects. Proceedings of the National Academy of Sciences, 99(17), 11393-11398. 2) Xin H, Biswas N, Li P, et al. 2021. 'Heparin-binding VEGFR1 variants as long-acting VEGF inhibitors for treatment of intraocular neovascular disorders', Proc Natl Acad Sci U S A, 118.

VEGF-A is the primary mediator of neovascularization and exudation

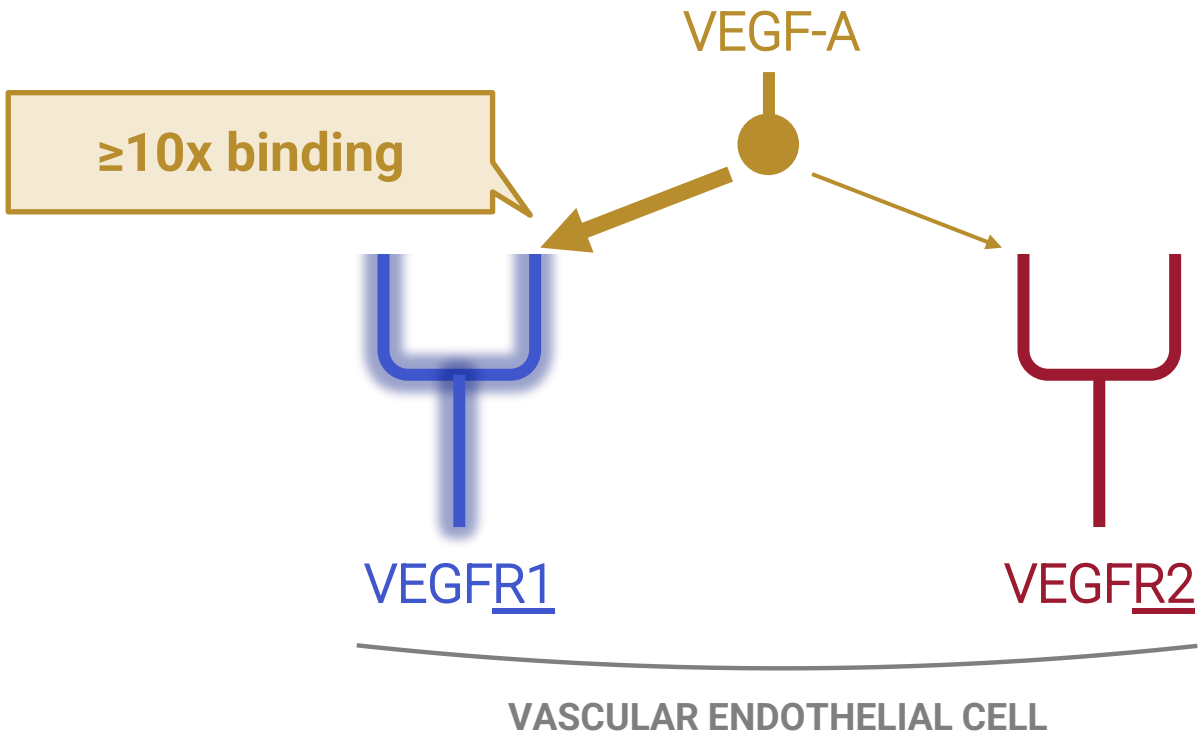


R# = Receptor 1, 2, and 3

Source: Karkkainen, M. J., & Petrova, T. V. (2000). Vascular endothelial growth factor receptors in the regulation of angiogenesis and lymphangiogenesis. Oncogene, 19(49), 5598-5605.

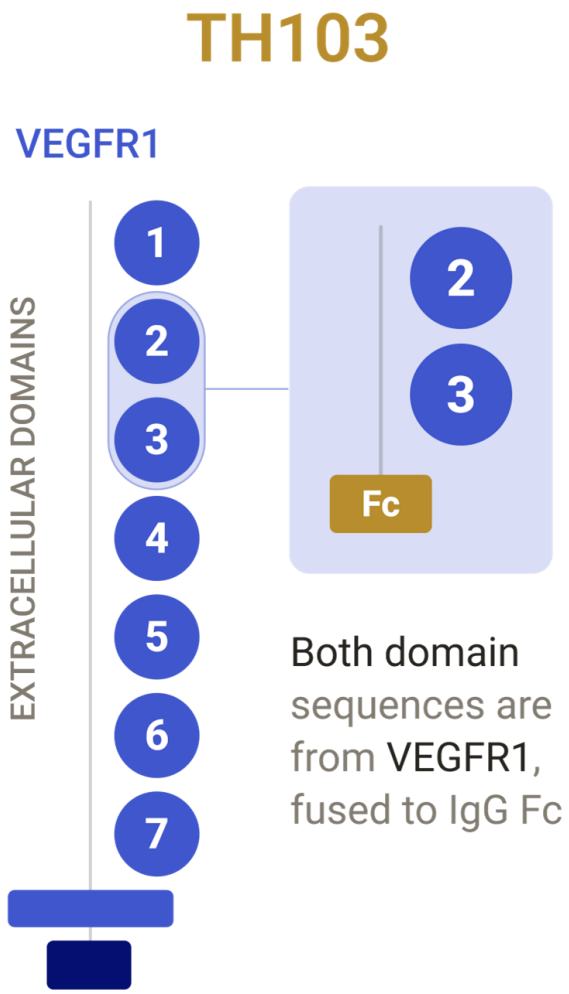
Kalaris Therapeutics

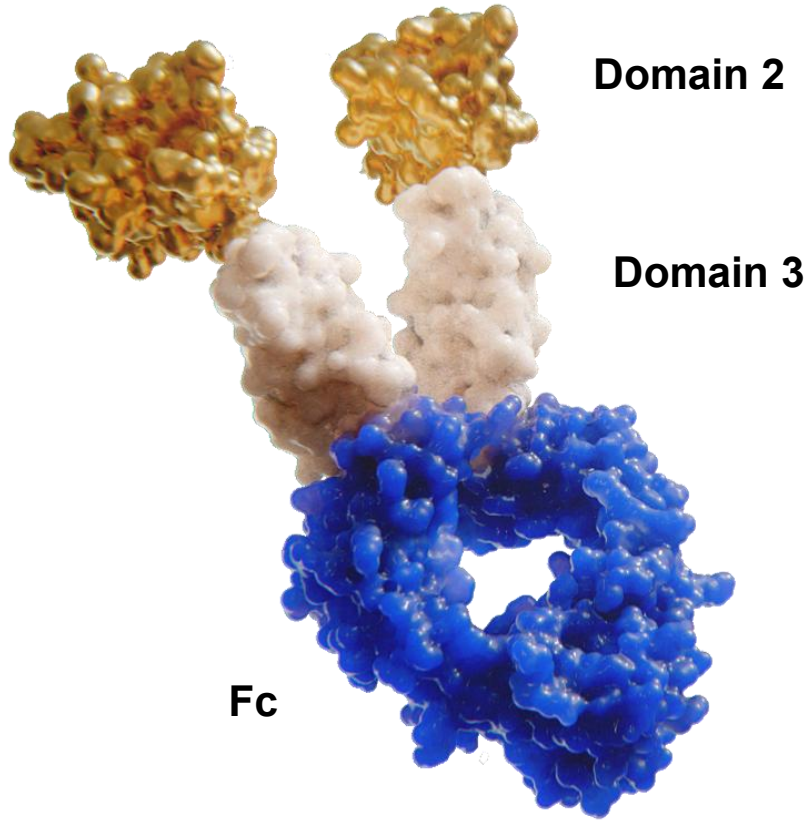
VEGF binds to Receptor 1 with ≥10-fold greater affinity than Receptor 2



Sources: Karkkainen, M. J., & Petrova, T. V. (2000). Vascular endothelial growth factor receptors in the regulation of angiogenesis and lymphangiogenesis. Oncogene, 19(49), 5598-5605; Cudmore et al., Scientific Reports (2020); Wiesmann et al., Cell (1997); Ferrara et al., Nature Medicine (2003)

TH103 leverages the key binding domains from VEGFR1

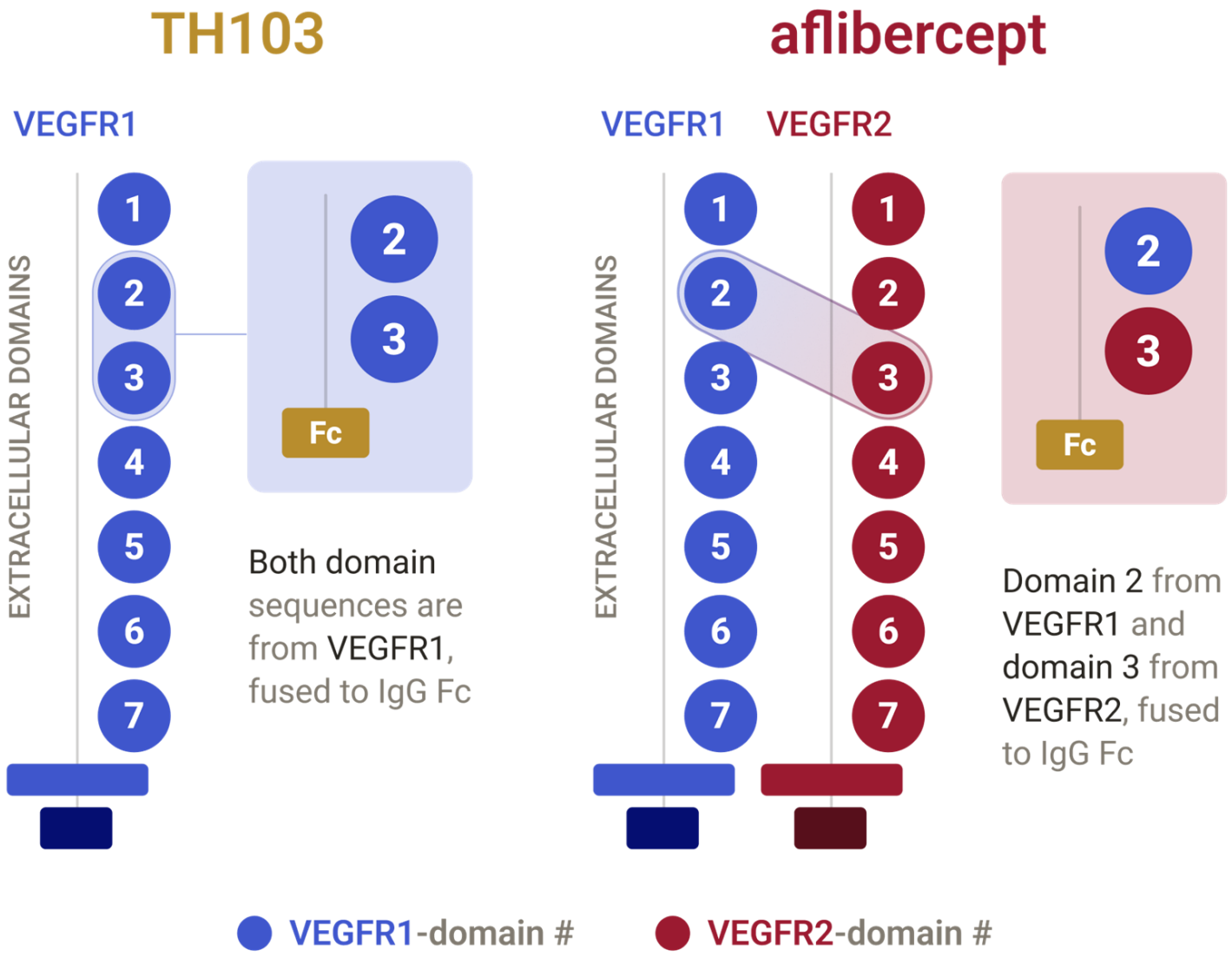




IgG= Immunoglobulin G; Fc =Fragment Crystallizable Region

Source: Xin, H., Biswas, N., Li, P., Zhong, C., Chan, T. C., Nudleman, E., & Ferrara, N. (2021). Heparin-binding VEGFR1 variants as long-acting VEGF inhibitors for treatment of intraocular neovascular disorders. Proceedings of the National Academy of Sciences, 118(21), e1921252118.

Using higher affinity VEGFR1 may confer enhanced VEGF inhibition



Source: Xin, H., Biswas, N., Li, P., Zhong, C., Chan, T. C., Nudleman, E., & Ferrara, N. (2021). Heparin-binding VEGFR1 variants as long-acting VEGF inhibitors for treatment of intraocular neovascular disorders. Proceedings of the National Academy of Sciences, 118(21), e1921252118.

VEGFR1 domain 3 enables potential TH103 binding to retina

Domain 3 from VEGFR1:

Binds **strongly to HSPG** which are present in all retinal layers, thereby sequesteringTH103 in the eye

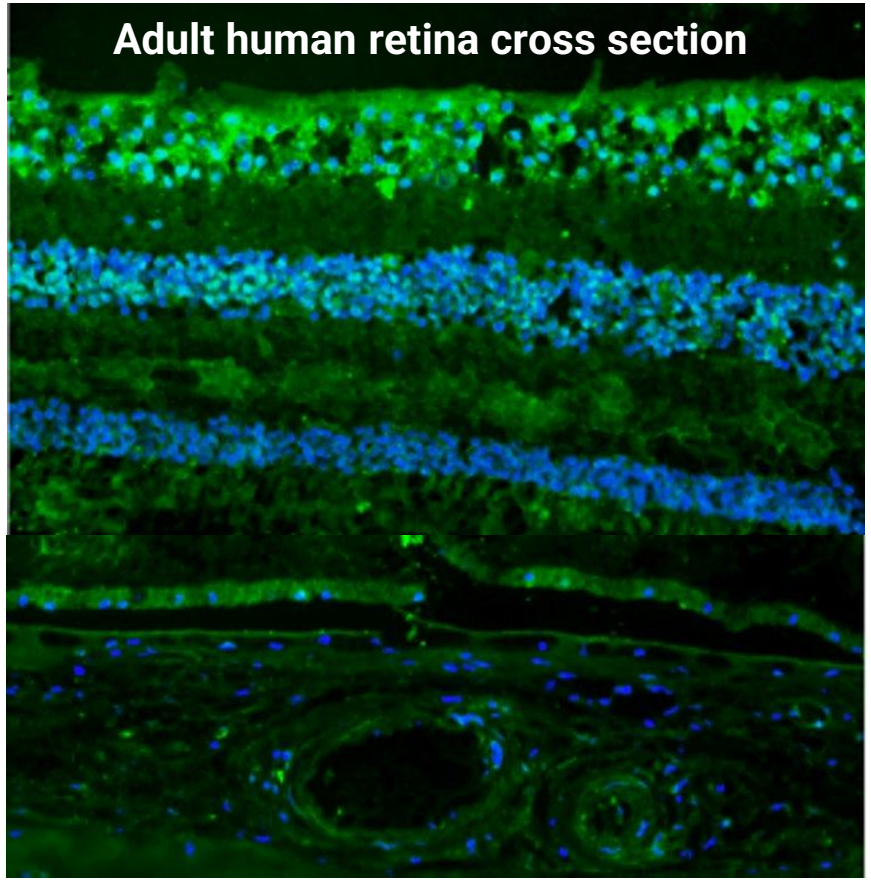
In contrast, domain 3 from VEGFR2:

Binds less strongly to HSPG, leading to reduced tissue sequestration (preferred for systemic circulation, e.g., ZALTRAP®, but suboptimal for ocular retention)[2]

VEGFR1-domain # VEGFR2-domain #

Source: 1) Xin H, Biswas N, Li P, et al. 2021. 'Heparin-binding VEGFR1 variants as long-acting VEGF inhibitors for treatment of intraocular neovascular disorders', Proc Natl Acad Sci U S A, 118.; 2) Holash, J., Davis, S., Papadopoulos, N., Croll, S. D., Ho, L., Russell, M., ... & Rudge, J. S. (2002). VEGF-Trap: a VEGF blocker with potent antitumor effects. Proceedings of the National Academy of Sciences, 99(17), 11393-11398.

HSPG is present throughout the retinal layers[1]



HSPG has been shown to be highly concentrated near CNV lesions[2], **potentially prolonging ocular retention precisely at the site of disease activity**

Green: Heparan sulfate antibody

Blue: DAPI staining of cell nuclei

Sources: 1) Clark SJ, Keenan TD, Fielder HL, et al. 2011. 'Mapping the differential distribution of glycosaminoglycans in the adult human retina, choroid, and sclera', Invest Ophthalmol Vis Sci, 52: 6511-21; 2) Regatieri, C. V., Dreyfuss, J. L., Melo, G. B., Lavinsky, D., Hossaka, S. K., Rodrigues, E. B., & Nader, H. B. (2010). Quantitative evaluation of experimental choroidal neovascularization by confocal scanning laser ophthalmoscopy: fluorescein angiogram parallels heparan sulfate proteoglycan expression. Brazilian Journal of Medical and Biological Research, 43, 627-633.

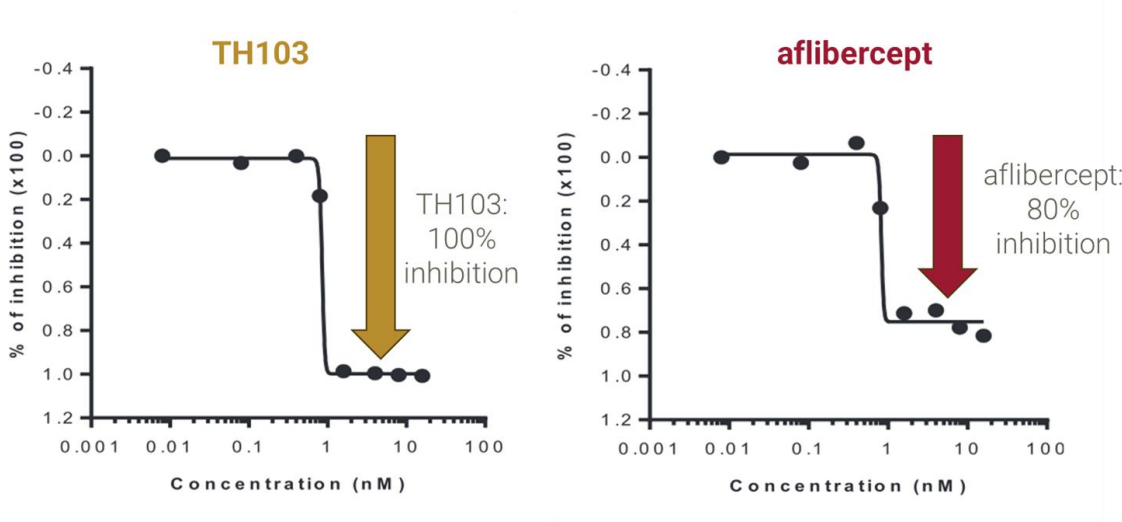
Preclinical Data Review & Initial Phase 1a Clinical Data

TH103: Increased VEGF-inhibitory activity vs. aflibercept in preclinical studies

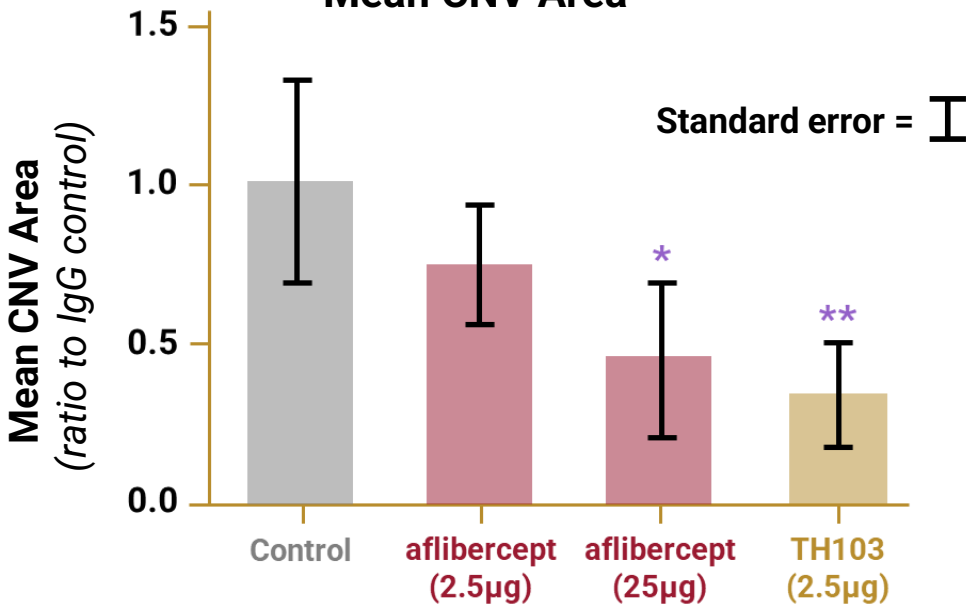
TH103 achieved 100% inhibition vs. aflibercept 80% inhibition of VEGF-induced endothelial cell proliferation
(in vitro, bovine choroidal endothelial cell proliferation assay[1])

TH103 increased reduction in mean choroidal neovascularization (CNV) area after administration at Day -1[2]
(in vivo, murine model)

Concentration Dependent VEGF Inhibition



Mean CNV Area



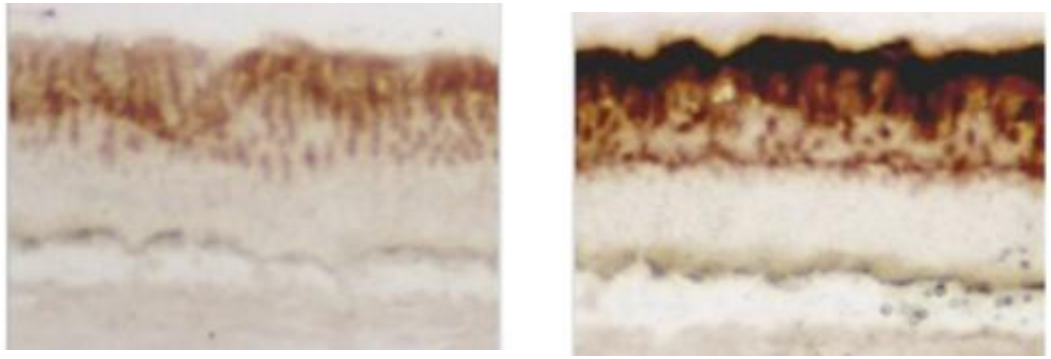
Notes: 1) human choroidal endothelial cells proliferate in nAMD pathologic angiogenesis; 2) The rodent laser-induced CNV model is the most widely used animal model to study the effects of anti-VEGFs in inhibiting CNV; Data are based on three independent experiments with at least five mice per group; Asterisks denote significant differences (Student's t test) compared to the appropriate IgG control groups (**P < 0.01, *P < 0.05); Source: Adapted from Xin H, Biswas N, Li P, et al. 2021. 'Heparin-binding VEGFR1 variants as long-acting VEGF inhibitors for treatment of intraocular neovascular disorders', Proc Natl Acad Sci U S A, 118.

TH103: Demonstrated prolonged retinal retention vs. aflibercept in preclinical studies

TH103 demonstrated increased retention in the retina as compared to aflibercept at two weeks post-injection
(in vivo, rabbit model)

TH103 demonstrated reduced systemic exposure after intravitreal administration[1]
(in vivo, murine model)

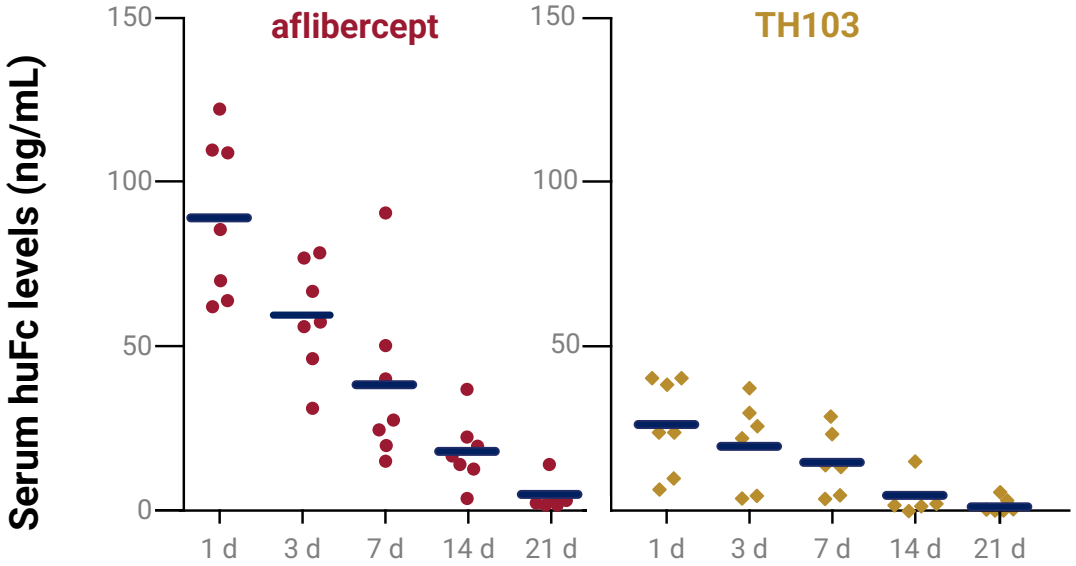
Rabbit Retina Cross-Sections at Day 14



aflibercept TH103

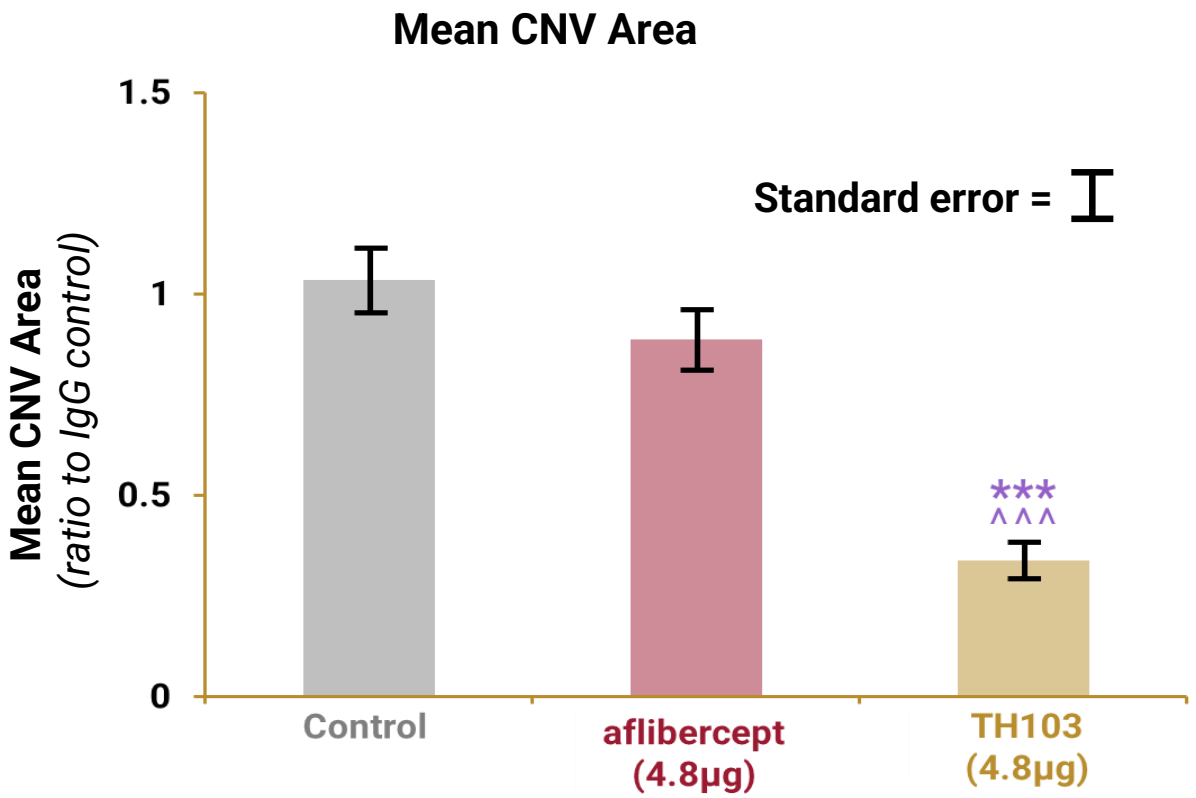
Equimolar dose administrated; Darker immuno-histochemistry staining indicates higher drug levels present

Serum Levels of TH103 Compared to Aflibercept After Bilateral Intravitreal Injection



Note: 1) Serum levels of aflibercept and TH103 in mice at different time points after intravitreal injection. Each molecule was injected in both eyes in equimolar amounts (2.4 µg). After 1, 3, 7, 14, and 21 d, peripheral blood was collected from the tail vein. Human Fc levels were measured by ELISA. Values shown are means ± SEM. n = 8 per point;
Source: Adapted from Xin H, Biswas N, Li P, et al. 2021. 'Heparin-binding VEGFR1 variants as long-acting VEGF inhibitors for treatment of intraocular neovascular disorders', Proc Natl Acad Sci U S A, 118.

TH103: Demonstrated prolonged bioactivity vs. aflibercept in an animal model



In a second murine experiment, rather than at Day -1, TH103 and aflibercept were administered at Day -14 prior to laser injury to assess durability of treatment effect. In this model, **TH103 showed smaller mean CNV area compared to equimolar aflibercept 21 days after injection.**

Note: TH103 and aflibercept administered 14 days prior to laser injury; CNV measurement at Day 7 post-laser; Symbols denote significant differences (Student's t test) between TH103 and control (***P < 0.001) and between TH103 and aflibercept (^^^P < 0.001). Source: Adapted from Xin H, Biswas N, Li P, et al. 2021. 'Heparin-binding VEGFR1 variants as long-acting VEGF inhibitors for treatment of intraocular neovascular disorders', Proc Natl Acad Sci U S A, 118.

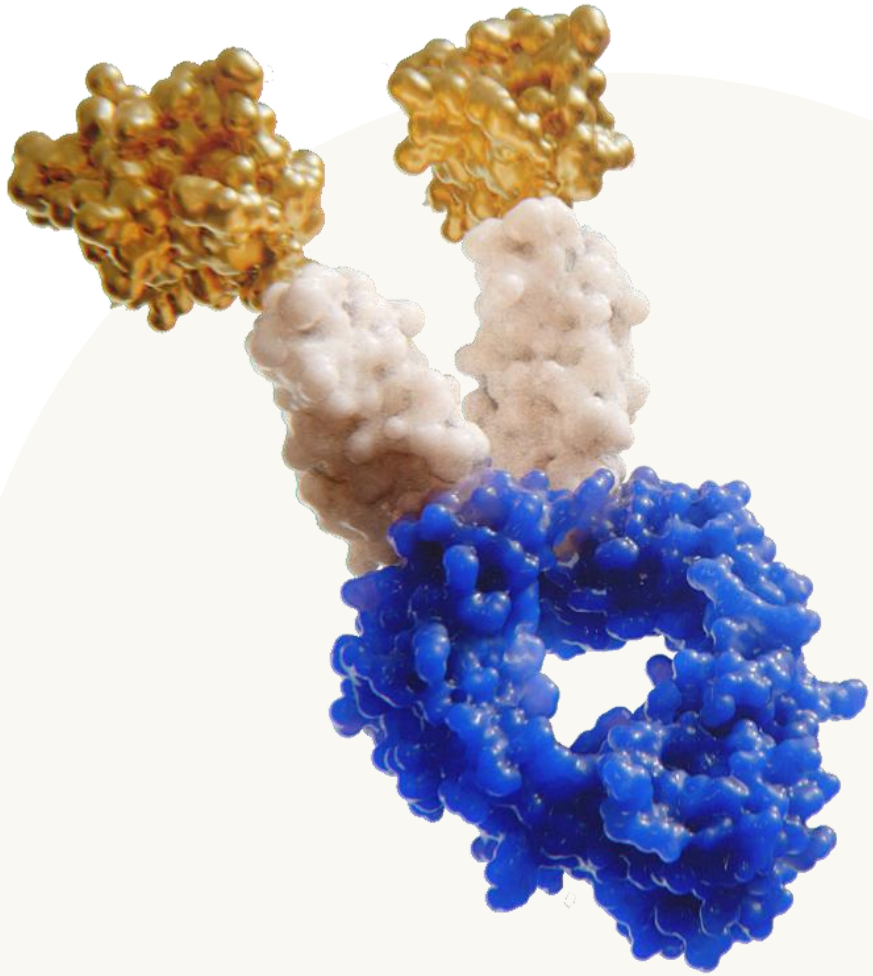
Phase 1a initial data summary

✓ **Efficacy:** rapid, robust response on BCVA and OCT parameters observed across dose levels at one month

✓ **Safety:** TH103 generally well tolerated, supporting exploration of further dose-escalation

✓ **Durability:**

- PK analysis consistent with greater TH103 intraocular retention vs. other leading agents
- Single-dose durability signal suggests potential for stronger durability outcomes after standard four-dose loading regimen

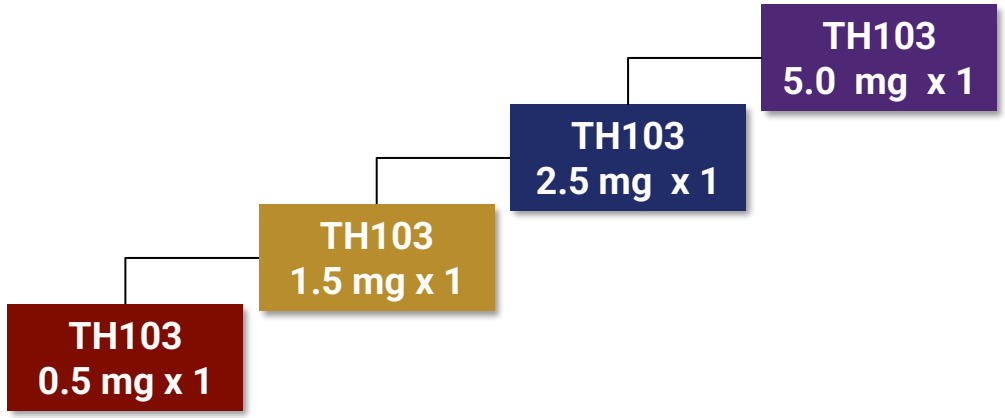


Kalaris Therapeutics

BCVA = Best Corrected Visual Acuity; CST = Central Subfield Thickness; PK = Pharmacokinetics

Phase 1a Single Ascending Dose (SAD) Study in Treatment-Naïve nAMD

Multi-center U.S. study to evaluate safety, tolerability, pharmacokinetics, and anti-VEGF activity following a single injection of TH103



Study Details

- Primary timepoint for analysis at Month 1
- Frequent follow-up visits within the first month; patients then followed monthly out to Month 6

Criteria for retreatment with aflibercept

- Increase of > 50 μm thickness in CST on SD-OCT compared to the lowest previously measured CST
- New macular hemorrhage due to nAMD

SD-OCT = Spectral-Domain Optical Coherence Tomography

Note: Data safety monitoring oversight occurred before dose-escalations

Key baseline characteristics of patients in Phase 1a trial of TH103 who have reached study completion

		Study Cohort			
		0.5 mg (n=3)	1.5 mg (n=7)	2.5 mg (n=3)	All Patients (n=13) [1]
Age (mean)		78	77	82	79
Sex (female / male)		3 / 0	5 / 2	1 / 2	9 / 4
BCVA (ETDRS letters, mean, range)		58 (44-71)	59 (35-73)	49 (36-63)	57 (35-73)
Lesion Type	Type 1	1	3	1	5 (38%)
	Type 2	-	1	-	1 (8%)
	Type 3[2]	1	3	2	6 (46%)
	Ungradable	1	-	-	1 (8%)
CST (µm, mean, range)		483 (421-550)	442 (329-611)	485 (440-554)	470 (329-611)

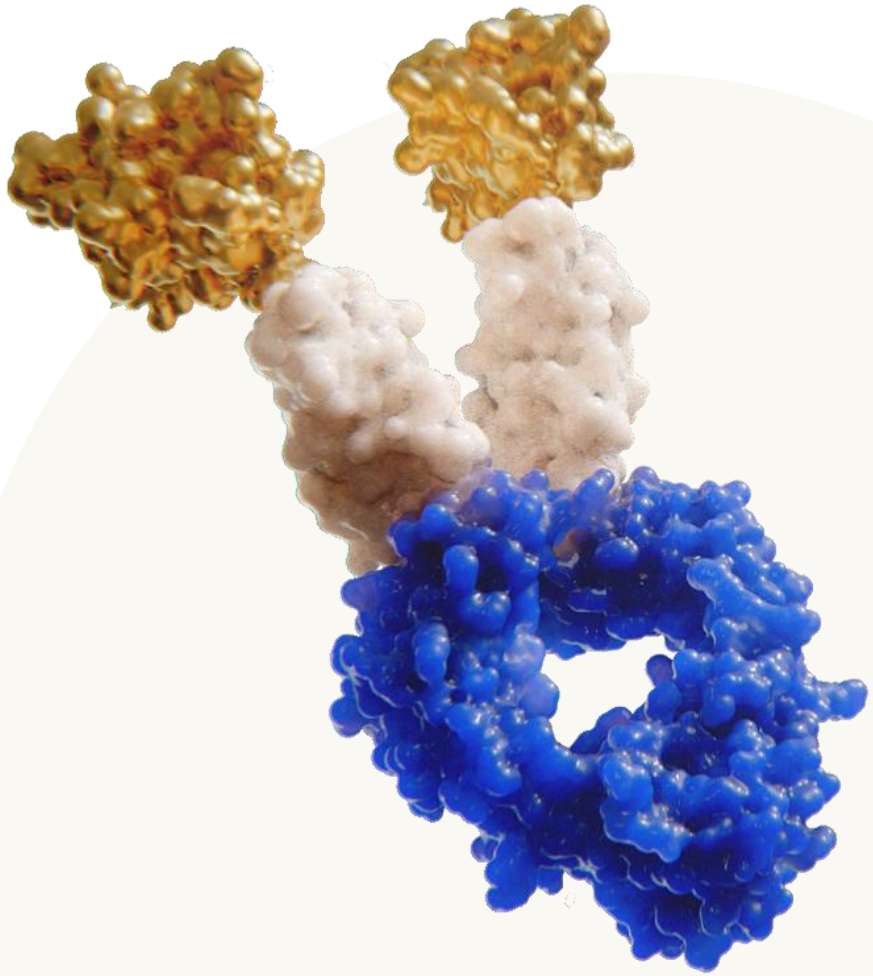
No study dropouts and 100% protocol adherence

Kalaris Therapeutics

1) Includes all patients who completed the entire 6-month follow-up period, excludes patients dosed with additionally purified material (6 dosed at the 2.5mg dose level and 1 patient dosed at the 5.0mg level subsequent to the December 2025 disclosure)
2) Also called retinal angiomatous proliferation, or RAP; all Type 3 lesions were determined to be Stage 3

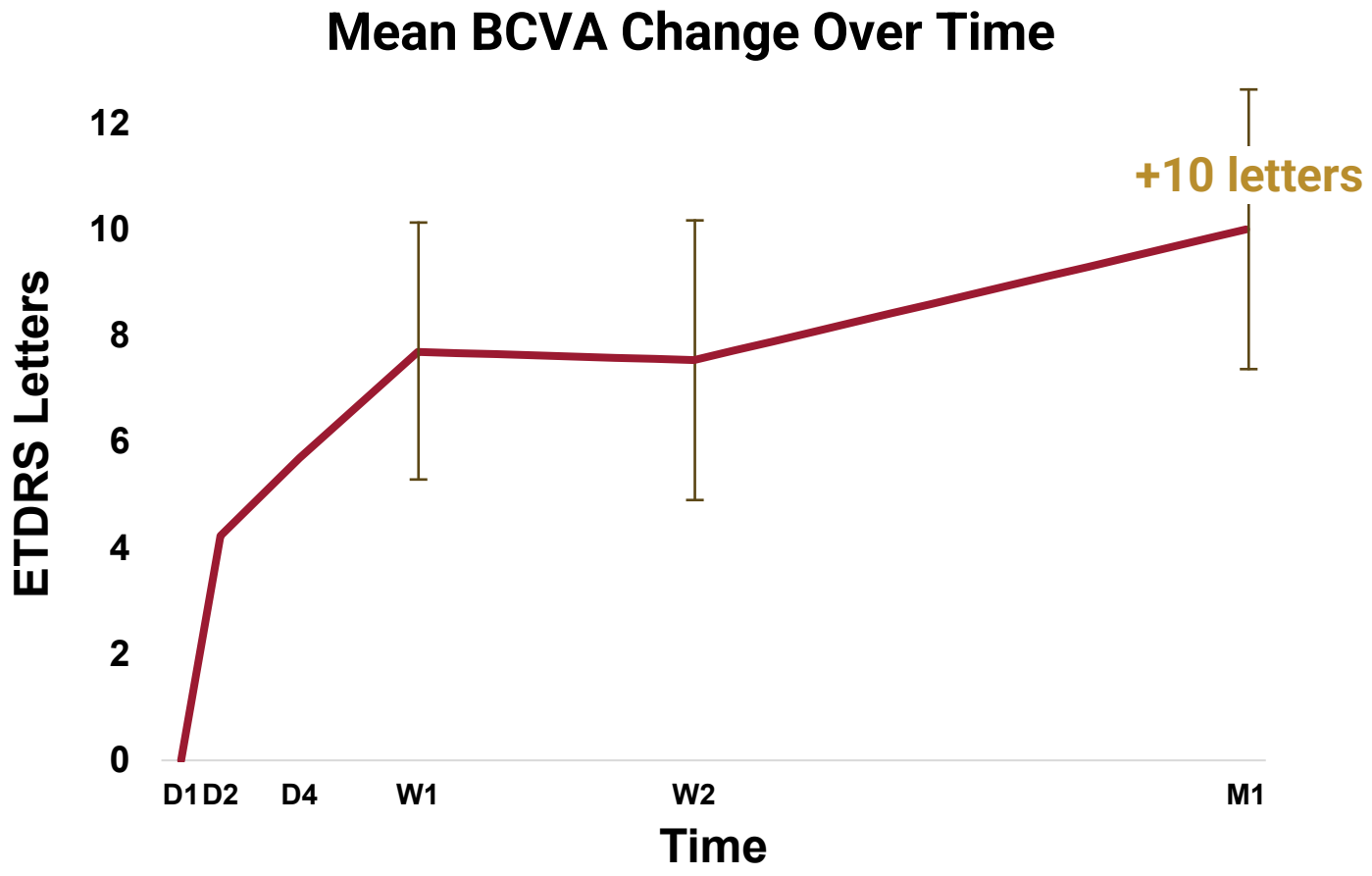
Phase 1a initial data summary

✓ **Efficacy:** rapid, robust response on BCVA and OCT parameters observed across dose levels at one month



Kalaris Therapeutics

Mean 10 letter gain in BCVA letter score after a single TH103 injection at Month 1

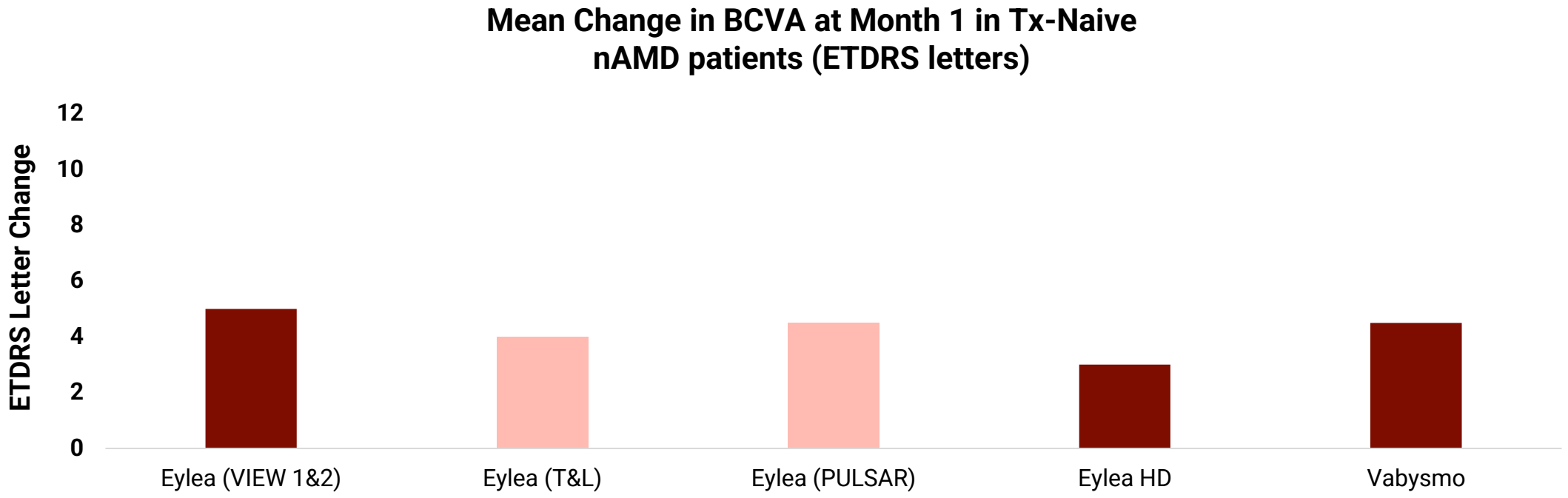


54%
(7/13 patients)
Gained ≥ 10 letters at Month 1

23%
(3/13 patients)
Gained ≥ 20 letters at Month 1

ETDRS = Early Treatment Diabetic Retinopathy Study
Note: n = 13 at all timepoints except Month 1, where n =12; one patient in the 0.5 mg cohort was treated with aflibercept at Week 2 and therefore the Month 1 data point is censored. Patients dosed at 2.5 mg with additionally purified material (n=6) are excluded from efficacy & PK analyses due to limited follow-up.; Brackets indicate standard error.

Change in mean visual acuity in treatment naïve nAMD patients for current market-leading agents at Month 1



Eylea (VIEW 1&2) BCVA Baseline Mean: ~54, n= 607
Eylea (PULSAR) BCVA Baseline Mean: ~59, n=336
Eylea (T&L) BCVA Baseline Mean: ~60, n = 664

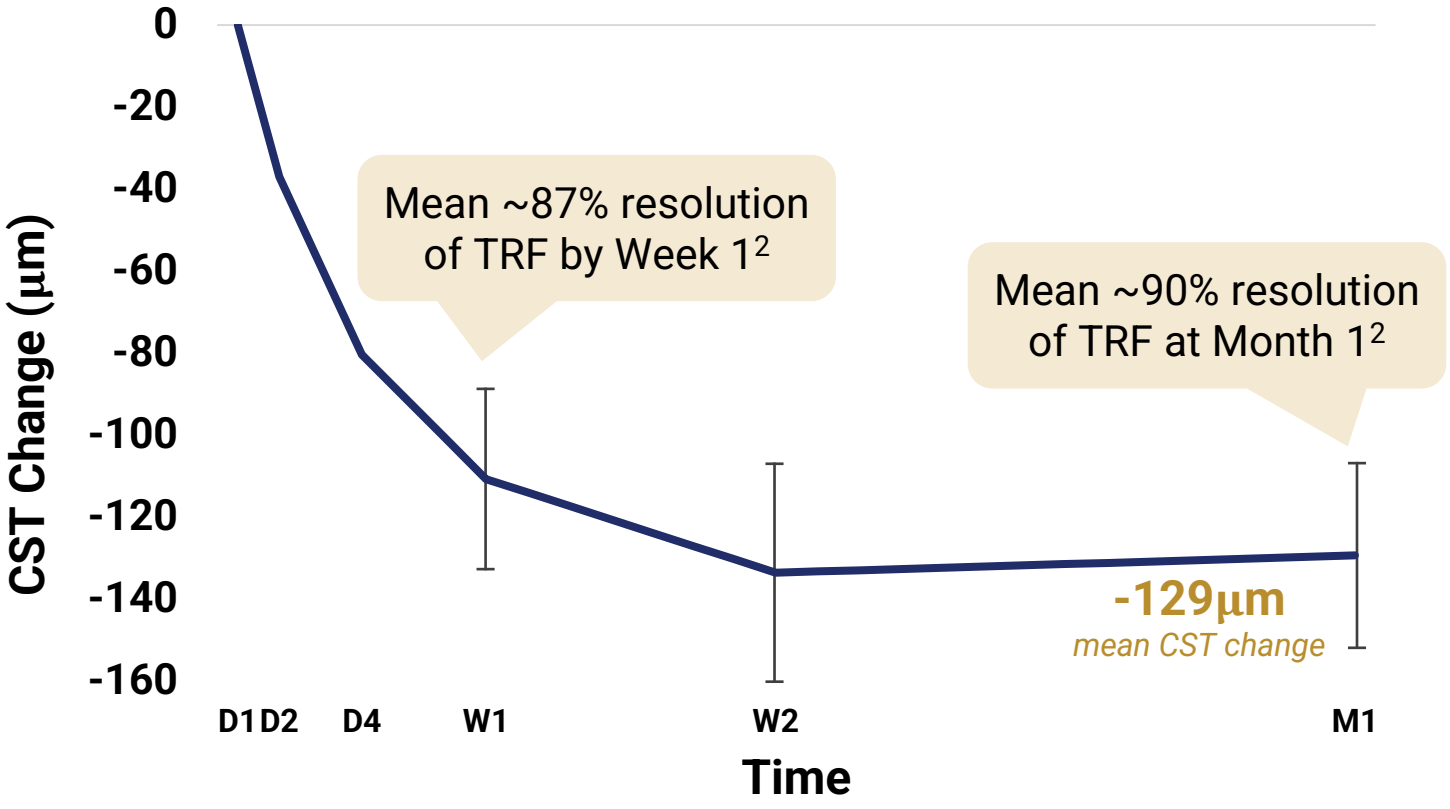
Eylea HD BCVA Baseline Mean: ~60, n=673
Vabysmo BCVA Baseline Mean: ~60, n=665

The above competitor data is approximate and reflects multiple Phase 3 studies. No head-to-head trials have been conducted comparing TH103 to any approved agents for nAMD. Such data may not be directly comparable due to differences in trial protocols, dosing regimens and patient populations. Accordingly, these cross-trial comparisons may not be reliable.

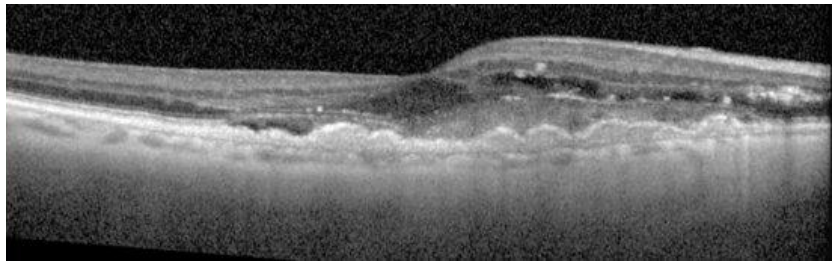
Sources: Khanani, Arshad M., et al. "TENAYA and LUCERNE: Two-Year Results from the Phase 3 Neovascular Age-Related Macular Degeneration Trials of Faricimab with Treat-and-Extend Dosing in Year 2." Ophthalmology, vol. 131, no. 8, 2024, pp. 914–926; Lanzetta, P., et al. "Intravitreal Aflibercept 8 mg in Neovascular Age-Related Macular Degeneration (PULSAR): 48-Week Results from a Randomised, Double-Masked, Non-Inferiority, Phase 3 Trial." The Lancet, vol. 403, no. 10344, 2024, pp. 1141-1152; Heier, J., et al. "Intravitreal Aflibercept (VEGF Trap-Eye) in Wet Age-Related Macular Degeneration." Ophthalmology, vol. 119, no. 12, 2012, pp. 2537-2548.

Rapid, robust improvement in CST and total retinal fluid (TRF) volume at Week 1 and Month 1

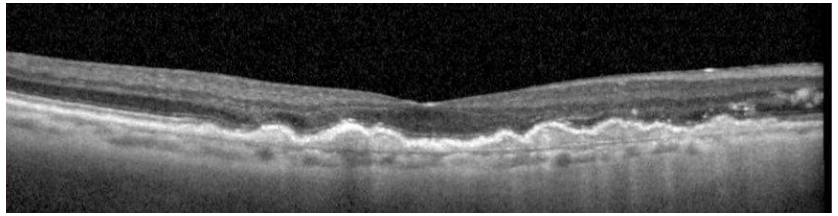
Mean CST Change Over Time[1]



Case Example (1.5 mg)



Single Dose TH103

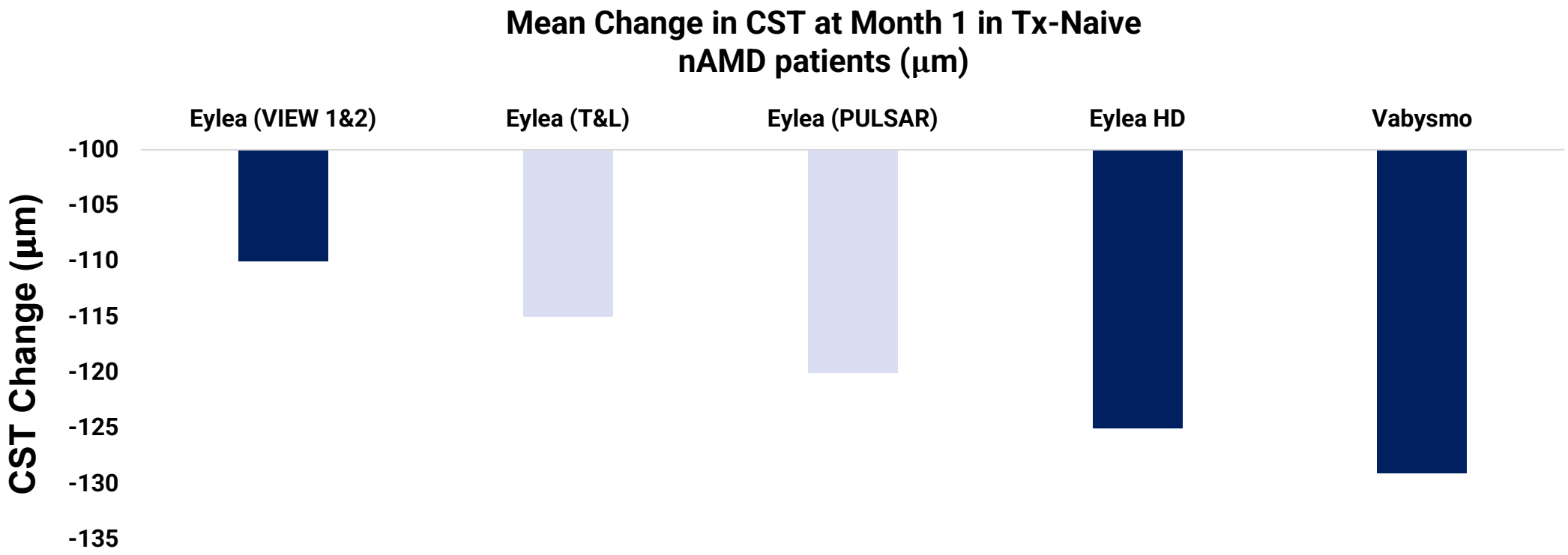


Month 1

Note: n = 13 at all timepoints except Month 1, where n =12; one patient in the 0.5 mg cohort was treated with aflibercept at Week 2 and therefore the Month 1 data point is censored. Patients dosed at 2.5 mg with further purified material (n=6) are excluded from efficacy & PK analyses due to limited follow-up; Brackets indicate standard error.
Sources: 1) As measured by independent reading center; 2) Data from automated fluid measurement software, Notal Vision Inc.; percentage change in mean central subfield TRF volume (subretinal fluid + intraretinal fluid in the central subfield, measured in nanoliters) from Day 1 to Week 1 & Month 1

Kalaris Therapeutics

Change in mean CST in treatment naïve nAMD patients for current market-leading agents at Month 1



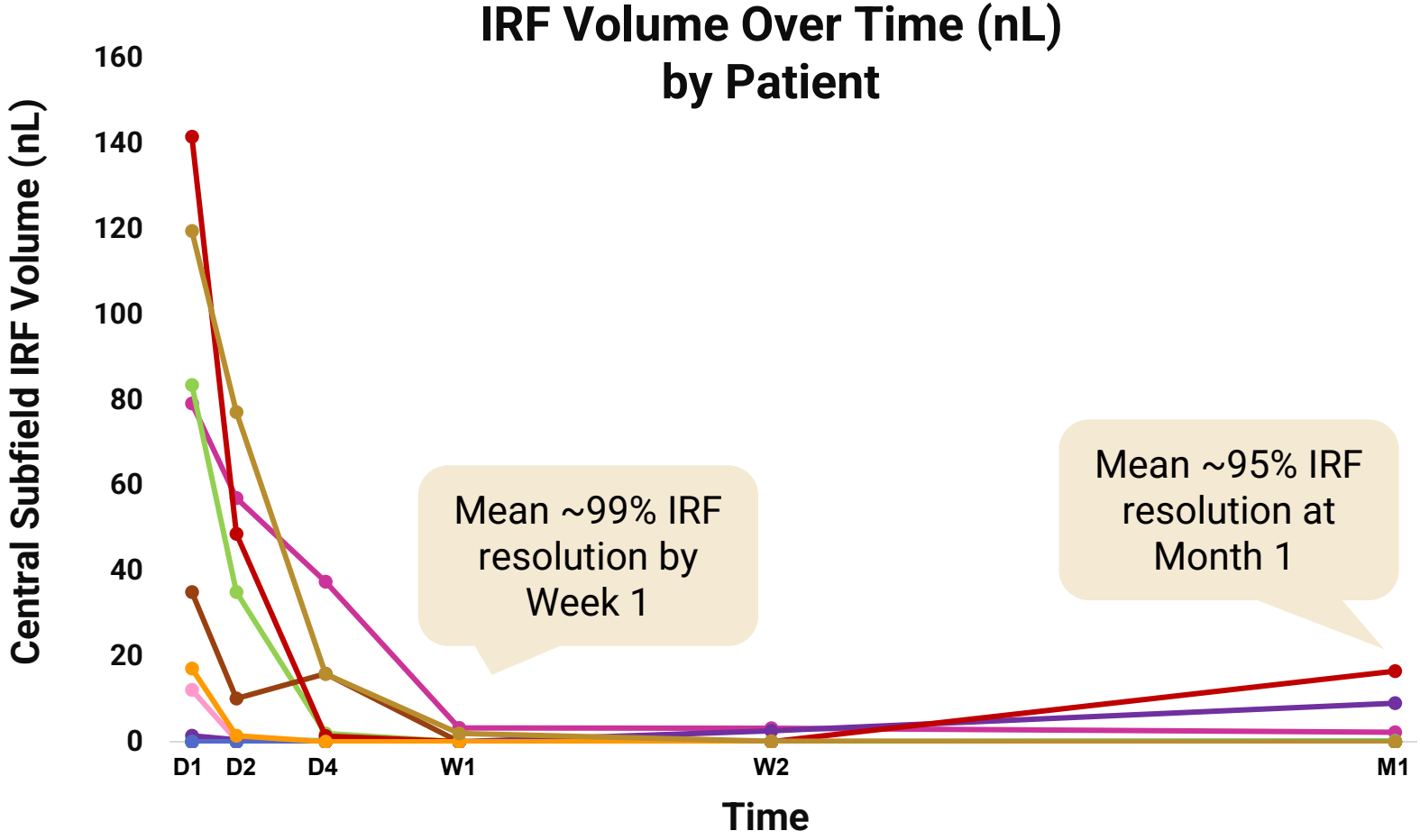
Eylea (VIEW 1&2) CST Baseline Mean: ~324, n=607
Eylea (PULSAR) CST Baseline Mean: ~367, n=336
Eylea (T&L) CST Baseline Mean: ~358, n = 664

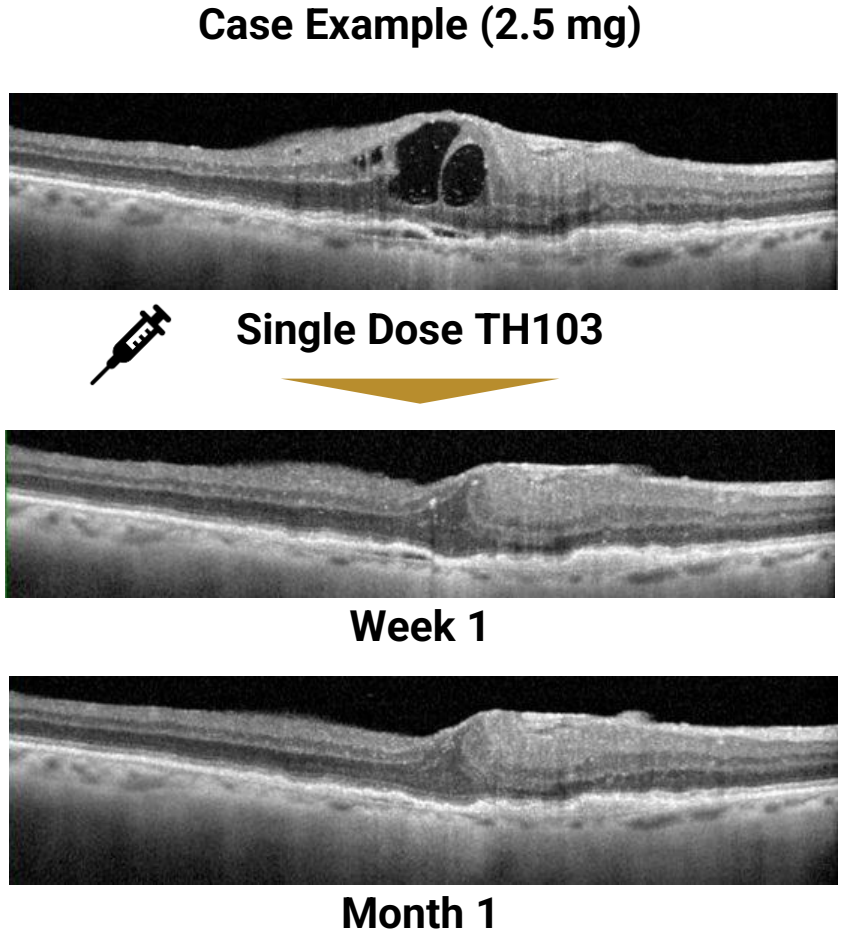
Eylea HD CST Baseline Mean: ~370, n=673
Vabysmo CST Baseline Mean: ~357, n=665

The above competitor data is approximate and reflects multiple Phase 3 studies. No head-to-head trials have been conducted comparing TH103 to any approved agents for nAMD. Such data may not be directly comparable due to differences in trial protocols, dosing regimens and patient populations. Accordingly, these cross-trial comparisons may not be reliable.

Sources: Khanani, Arshad M., et al. "TENAYA and LUCERNE: Two-Year Results from the Phase 3 Neovascular Age-Related Macular Degeneration Trials of Faricimab with Treat-and-Extend Dosing in Year 2." Ophthalmology, vol. 131, no. 8, 2024, pp. 914–926; Lanzetta, P., et al. “Intravitreal Aflibercept 8 mg in Neovascular Age-Related Macular Degeneration (PULSAR): 48-Week Results from a Randomised, Double-Masked, Non-Inferiority, Phase 3 Trial.” The Lancet, vol. 403, no. 10344, 2024, pp. 1141-1152; Heier, J., et al. “Intravitreal Aflibercept (VEGF Trap-Eye) in Wet Age-Related Macular Degeneration.” Ophthalmology, vol. 119, no. 12, 2012, pp. 2537-2548.

Rapid and consistent resolution of intraretinal fluid (IRF) volume observed across doses



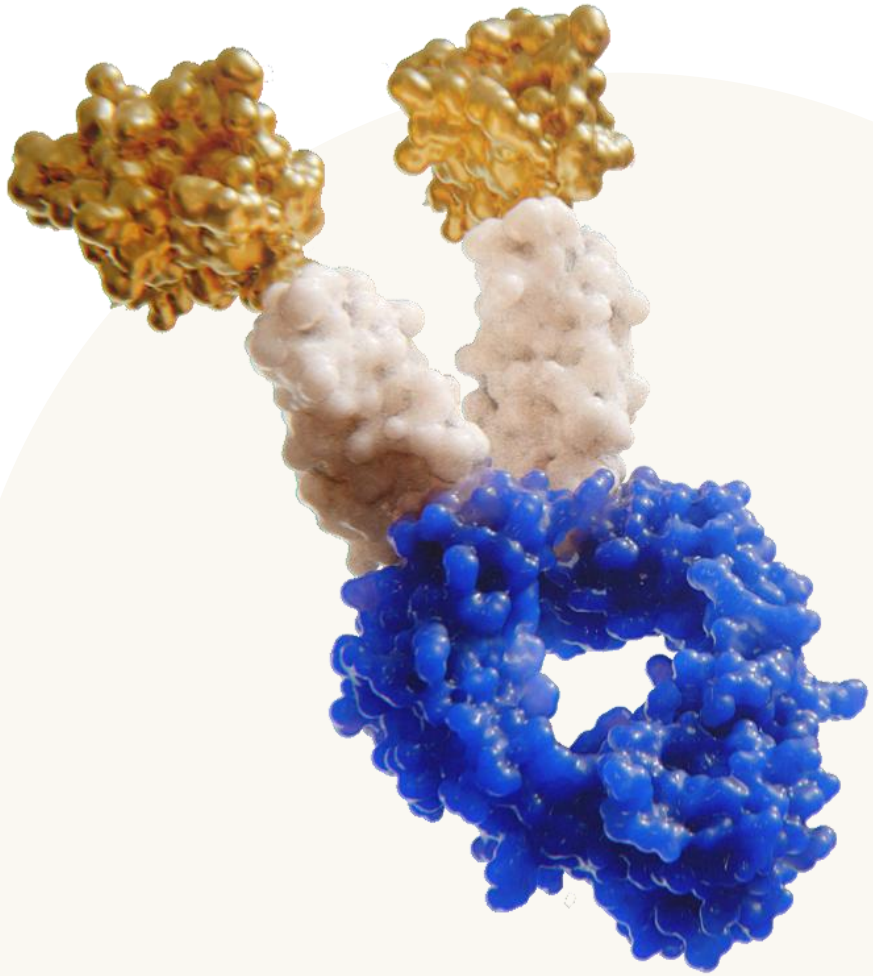


Note: Measurement of intraretinal fluid volume (nL) in the central subfield, depicting individual patients (n = 12; one patient in the 0.5 mg cohort was treated with aflibercept 2mg at Week 2 and excluded from the analysis); 3 patients had zero measured IRF throughout depicted timeframe and appear as overlapping lines on the x-axis; Patients dosed at 2.5 mg with additionally purified material (n=6) are excluded from efficacy & PK analyses due to limited follow-up.
Source: Data from automated fluid measurement software, Notal Vision Inc.; percentage change in mean central subfield IRF volume (nL) from Day 1 to Week 1 / Month 1 (n = 12)

Phase 1a initial data summary

✓ **Efficacy:** rapid, robust response on BCVA and OCT parameters observed across dose levels at one month

✓ **Safety:** TH103 generally well tolerated, supporting exploration of further dose-escalation



Kalaris Therapeutics

Safety Summary from Phase 1a Trial

Phase 1a Single-Ascending Dose Trial[1]

- No dose limiting toxicity (DLT) or serious adverse events (SAEs) observed
- Transient, mild-moderate intraocular inflammation (IOI) presented at Day 4 in 2 patients dosed at 2.5mg, attributed to product host cell protein (HCP) levels
- Further processing steps added to manufacturing process reduced host cell protein levels significantly

Update Since Dec. 2025 Data Disclosure[2]

- 6 patients[3] dosed at 2.5mg with the new process material have completed at least 3-months follow-up
  - No dose limiting toxicity (DLT) or serious adverse events (SAEs) observed
  - No reported cases of intraocular inflammation (IOI)
- Transient, moderate IOI presented at Day 2 in 1 patient dosed at 5.0mg

No reported TH103-related adverse events of retinal vascular occlusive disease, retinal vasculitis, cataracts, or elevated intraocular pressure

1) Data as of safety cut-off date Dec 15, 2025
2) Update after December 17, 2025, data release; patients are not included in efficacy analyses
3) Includes treatment-experienced patients

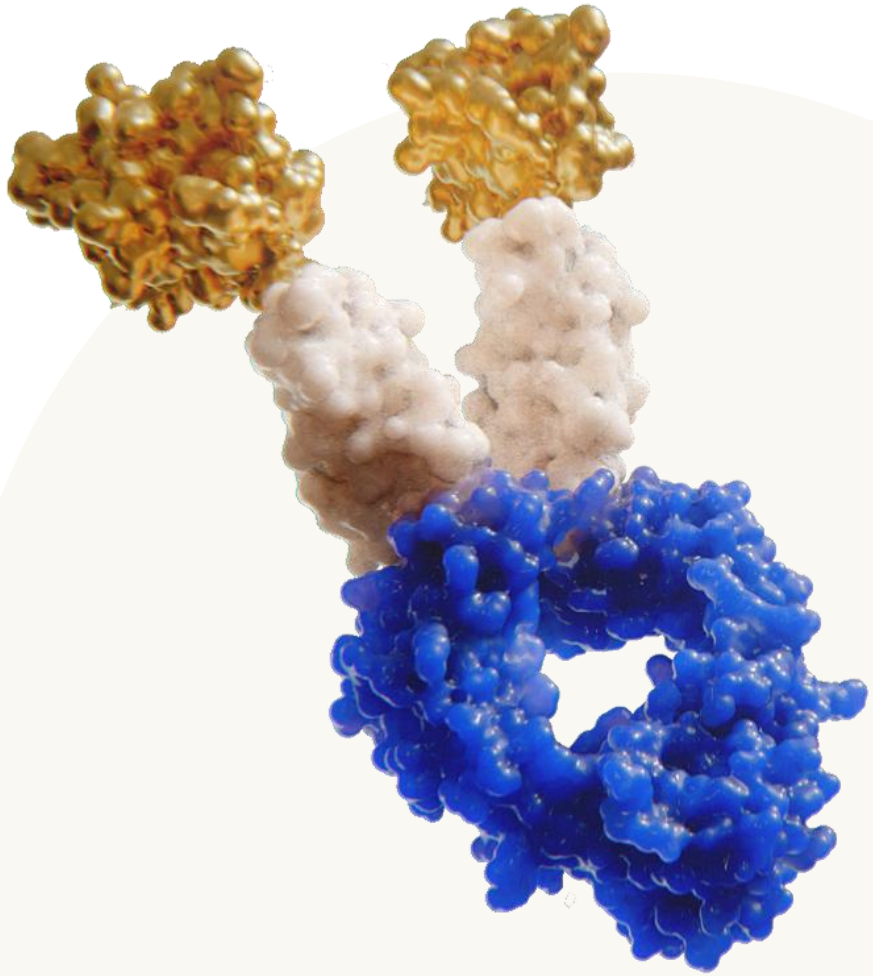
Phase 1a initial data summary

✓ **Efficacy:** rapid, robust response on BCVA and OCT parameters observed across dose levels at one month

✓ **Safety:** TH103 generally well tolerated, supporting exploration of further dose-escalation

✓ **Durability:**

- PK analysis consistent with greater TH103 intraocular retention vs. other leading agents
- Single-dose durability signal suggests potential for stronger durability outcomes after standard four-dose loading regimen



Kalaris Therapeutics

Initial SAD plasma PK data is consistent with greater TH103 intraocular retention

Plasma Drug Levels

Treatment	Cmax* (ng/mL)	Cmax/Dose* (nM/mmol)
Eylea 2 mg[1]	40.5	20.6
Eylea HD 8 mg[2]	247	31.2
Vabysmo 6 mg[3]	234	39.0
TH103 0.5 mg[4]	Not detected	n/a
TH103 1.5 mg[4]	0.877	0.354
TH103 2.5 mg[4]	1.87	0.762

TH103 2.5 mg Plasma Levels (Cmax/Dose):

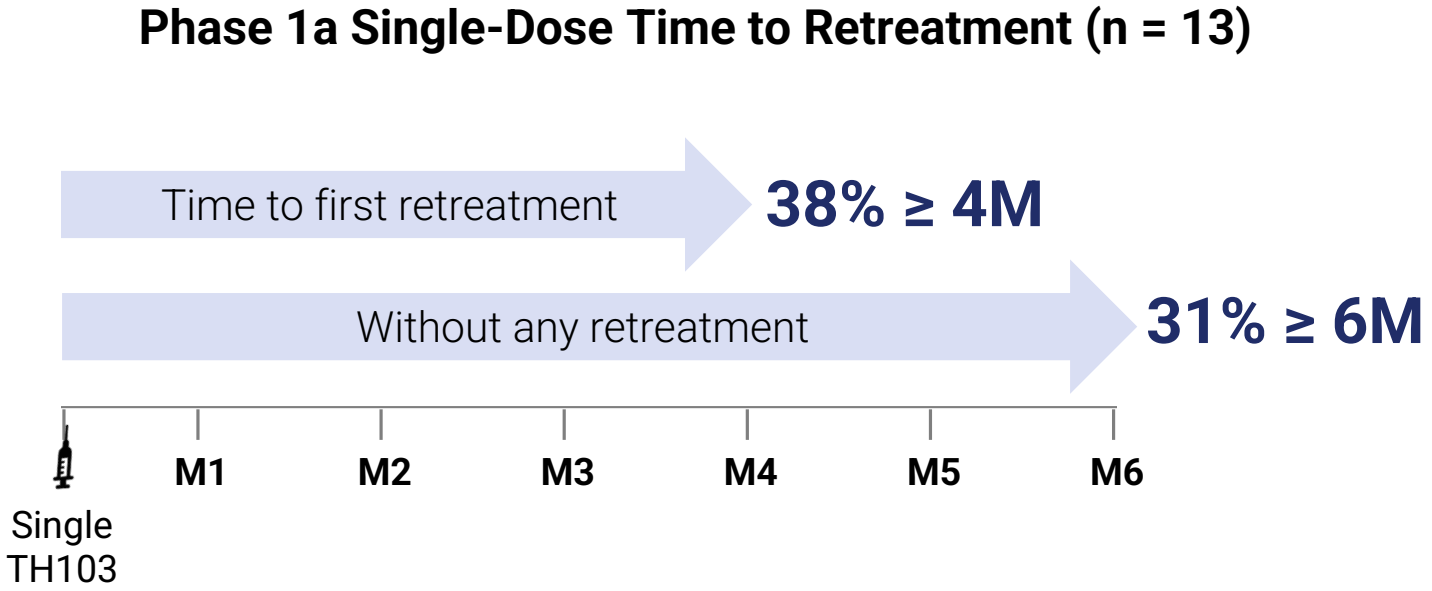
27x lower than Eylea 2mg

41x lower than Eylea HD

51x lower than Vabysmo

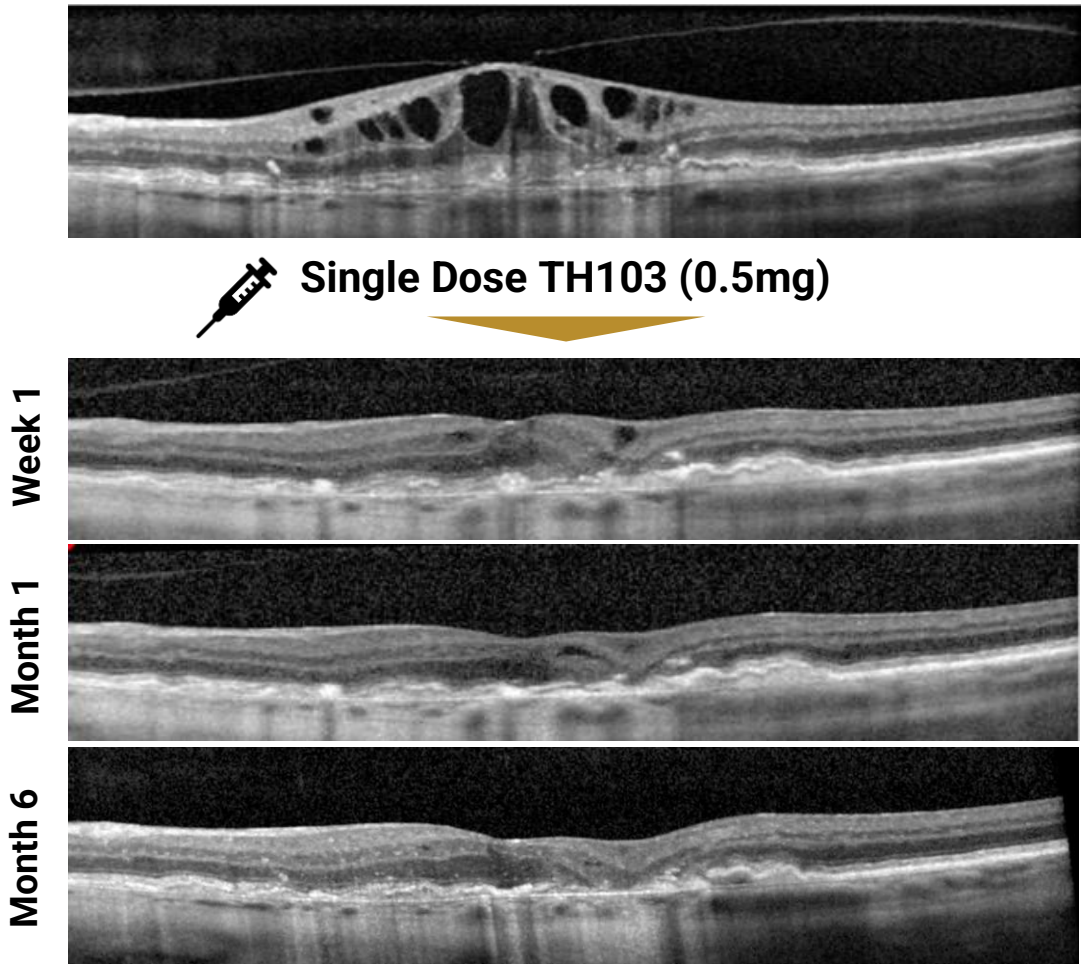
*Mean, except for Vabysmo which is median
Sources: 1) Data from BLA761355 and published studies; 2) Data from BLA761355 ; 3) Data from BLA761235; 4) Data from KLRS-100 Clinical Trial
Notes: Dose normalization of a parameter involves converting the mg dose to its molar dose and dividing it by the molar concentration of the administered dose

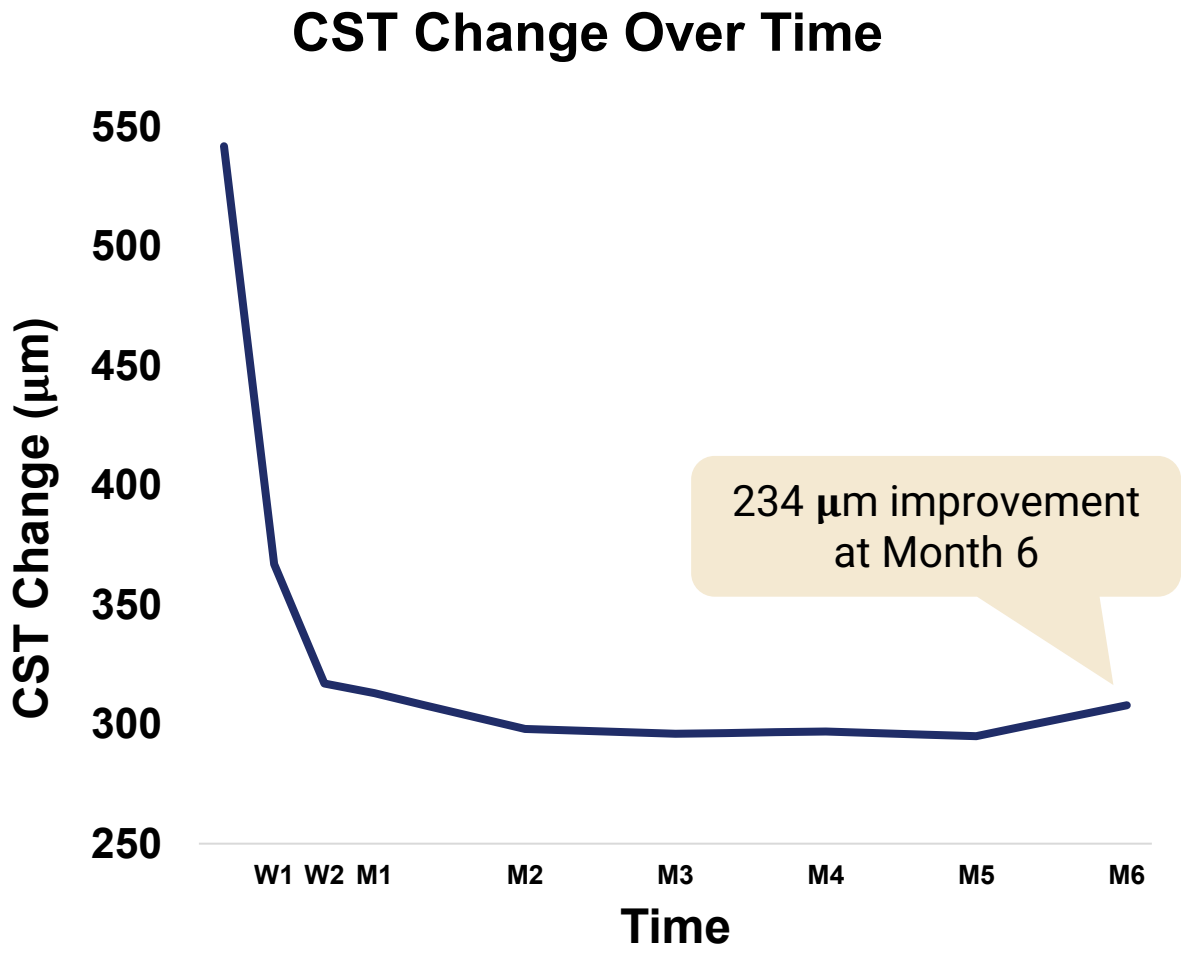
Single-dose durability signal suggests potential for stronger durability outcomes after standard four-dose loading regimen



Ongoing **Phase 1b/2 study** designed to further explore durability signal following a standard four-dose loading regimen

Case Example:
TH103 single-injection durable response past Month 6





Source: As measured by independent reading center

First-in-Human data support TH103's potential to be best-in-class, first-line treatment for prevalent retinal diseases

✓ **Efficacy:** rapid, robust response on BCVA and OCT parameters observed across dose levels at one month

- Mean 10-letter BCVA improvement at Month 1
- Mean 129µm improvement in mean CST and mean 95% resolution in CSF intraretinal fluid at Month 1

✓ **Safety:** TH103 generally well tolerated, supporting exploration of further dose-escalation

- No dose-limiting toxicities or TH103-related SAEs observed
- 2 cases of mild/moderate IOI at 2.5mg dose level with original process material[1]
- No cases of IOI at 2.5mg dose level (n=6) observed through ≥3 months with new process material[2]
- Single case of moderate IOI at 5.0mg dose level with new process material[2]

✓ **Durability:**

- PK analysis consistent with greater TH103 intraocular retention vs. other leading agents
- Single-dose durability signal suggests potential for stronger durability outcomes after standard four-dose loading regimen

Kalaris Therapeutics

1) Data as of safety cut-off date Dec 15, 2025
2) Update after December 17, 2025, data release; patients are not included in efficacy analyses
CSF = Central Subfield

TH103 Ongoing Development Program

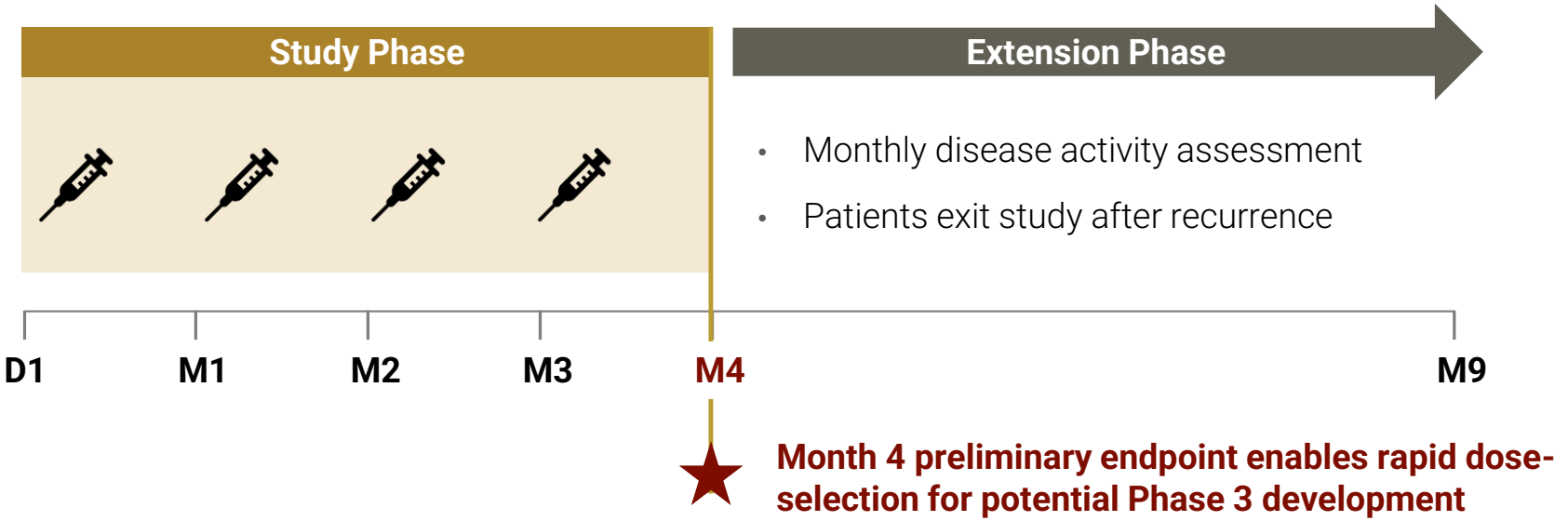
Ongoing Phase 1b/2 Trial in nAMD; preliminary endpoint data expected 1H 2027

Open label, multiple ascending dose design followed by randomized, masked, multi-dose cohort-expansion phase

Potential dose levels range from 0.5mg up to 5.0mg

Patient Population

- Age 50+
- Tx-naïve nAMD
- > 325 microns CST*
- BCVA: 20/32 to 20/200



*Confirmed by independent reading center

Kalaris Therapeutics

Ongoing manufacturing process refinements continue to drive down levels of product host cell protein

Original Clinical Batch

- Fusion protein produced through CHO cell culture, with process impurities (HCP) removed through downstream steps to levels acceptable to FDA
- Batch used for original 13 patients in Phase 1a

Current Clinical Batch

- Added reagents to manufacturing process to dissociate and separate HCPs from TH103
- Overall HCP appreciably lower than original clinical batch

Future Clinical Batches

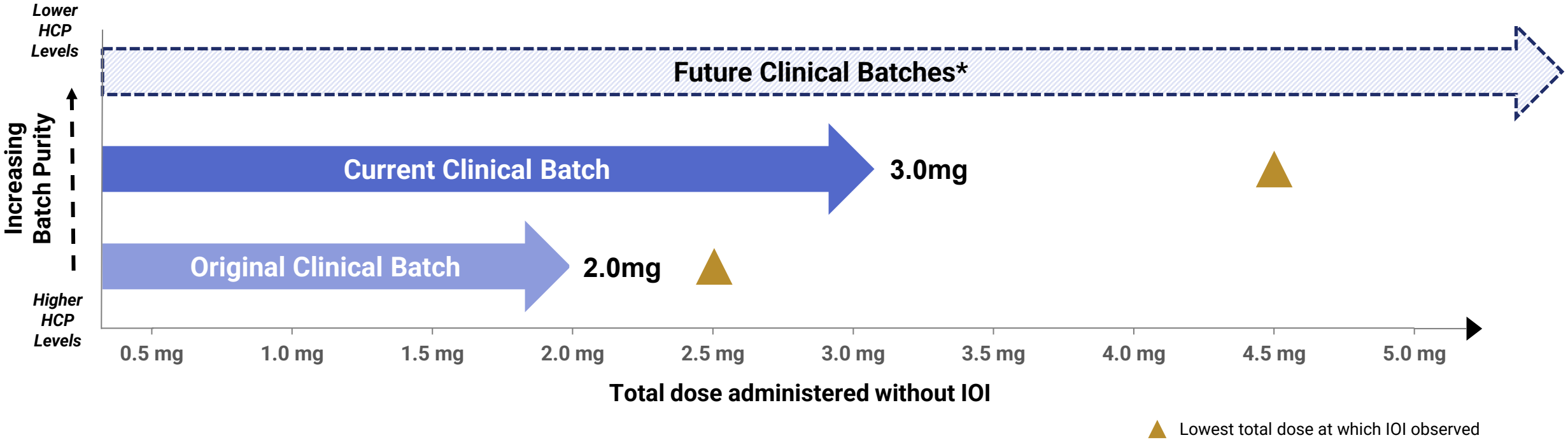
- Advanced analytical methods provided a granular identification of the specific remaining constituent HCP sub-types
- Ongoing process refinements focused on eliminating all remaining sub-HCPs
- Future clinical batches with further reduced levels of HCP vs. current clinical batch available in Q2 2026
  - All further patient dosing will be with future clinical batches

We have now identified specific process modifications aimed at eliminating all remaining HCP subtypes to below levels of detection

Kalaris Therapeutics

CHO = Chinese Hamster Ovaries
HCP = Host Cell Protein

Continued progress in reducing HCP levels in manufacturing is consistent with clinical findings



Original Clinical Batch: SAD: 0.5mg with no IOI (n=3); 1.5mg with no IOI (n=7); 2.5mg with IOI (n=2[a] of 3); MAD: 0.5mg x 4 injections with no IOI (n=6pts, 24 injections); No DLT identified

Current Clinical Batch: SAD: 2.5mg with no IOI (n=6); 5.0mg with IOI (n=1[b]); MAD: 1.5mg (n =10) with no IOI x 1 or 2 injections (n=6, 2); IOI after 3 injections (n=1[c] of 2); No DLT identified

IOI = Intraocular inflammation
*Goal for future clinical batches
Notes: a) Mild-moderate, resolved; b) Moderate, resolved; c) Moderate anterior vitreous cell and mild arteriolar abnormalities without leak or occlusion; asymptomatic; improved BCVA/CST from baseline; resolving

Kalaris Therapeutics

TH103 Clinical Development Program & Anticipated Milestones

- Preliminary data from ongoing **Phase 1b/2 MAD study expected in 1H 2027**
- Pending the results from the Phase 1b/2 trial, potential **Phase 3 trial initiation in nAMD planned by year-end 2027**
- Cash runway **extended and expected to fund company into Q4 2027**

Kalaris Therapeutics

Planned expansions beyond nAMD into other prevalent VEGF-mediated diseases such as DME, DR, and RVO

Product Candidate	Indication	Discovery	IND Enabling	Phase 1	Phase 2	Phase 3
TH103	nAMD*			→		
TH103	DME / DR**			→		
TH103	RVO**			→		

DME = Diabetic Macular Edema
DR = Diabetic Retinopathy
RVO = Retinal Vein Occlusion

*Two ongoing nAMD studies – Phase 1a and Phase 1b/2
**Subject to IND submission and clearance

Kalaris Therapeutics

Kalaris Therapeutics

Corporate

Intellectual Property

1

TH103 Compositions of Matter

- Issued/allowed in United States, Japan, China, Australia, Colombia, and Eurasia
- Pending in Europe, China, Korea, India, Brazil, Mexico, Singapore, New Zealand, Hong Kong, and Israel

2

TH103 Methods of Use

- Issued/allowed in United States, Europe, Japan, Australia, Israel, and Eurasia
- Pending in Canada, China, Korea, India, Brazil, Mexico, Singapore, New Zealand, and Hong Kong

3

US Exclusivity through early 2040s

- Later of US patent expiry (Q4 2040) or 12-year post-approval biologics exclusivity period
- Ex-US geographies vary, with coverage expected through 2039

Experienced Board & Management Team

Board of Directors

David Hallal
Board Chairman

Anthony Adamis, MD
Director

Srinivas Akkaraju, MD, PhD
Director & Co-founder

Mike Dybbs, PhD
Director & Co-founder

Napoleone Ferrara, MD
Director & Co-Founder

Morana Jovan-Embiricos, PhD
Director

Leone Patterson
Director

Management Team

Andrew Oxtoby
CEO & Director

Matthew Feinsod, MD
CMO

Kristine Curtiss
SVP Clinical

Brett Hagen, CPA
SVP Finance & CAO

Jill Porter, PhD
SVP CMC

Nancy Davis
VP Clinical Ops

Select Key Accomplishments

Discoverer of **VEGF, VEGF receptors, VEGF isoforms**

Leadership involved in developing first two **anti-VEGF agents ever FDA approved**

Extensive experience in anti-VEGF therapeutic development

Investment firm with track record in funding **successful retina therapeutic development** to FDA approval

Extensive experience in preclinical through commercial stage

TH103 Advantage: Established yet Differentiated

ESTABLISHED
Part of a Proven Class[1]

- ✓ Targets VEGF-A, primary mediator of disease activity in the retina
- ✓ Soluble decoy receptor MoA2
- ✓ Biologic (recombinant fusion protein); simple protein structure
- ✓ Drug class with well-established path to approval
- ✓ Drug administration would align with current clinical practice workflows

DIFFERENTIATED
TH103 Innovation

- \+ Optimized dual target (VEGF + HSPG), with novel HSPG pathway disruption
- \+ Leverages native higher-affinity VEGFR1 for optimized VEGF binding
- \+ Designed for clinical differentiation and potentially improved:
  - \+ Disease control via optimized VEGF inhibition
  - \+ Durability via extended ocular retention

TH103 builds on the proven success factors of leading agents, with molecular innovation to address persistent unmet needs

Kalaris Therapeutics

1) ranibizumab, aflibercept, faricimab
2) aflibercept

Potential best in class, dual-action, anti-VEGF therapeutic

TH103 was engineered by Dr. Napoleone Ferrara for **extended intraocular retention** with **enhanced VEGF inhibition**

Early clinical data from Phase 1a SAD study **validates molecular design**

Ongoing Phase 1b/2 multi-dose trial

Preliminary **Phase 1b/2 data expected in 1H 2027**

$50MM oversubscribed private placement[1] in December 2025 fortified balance sheet

Sufficient **cash runway to fund company into Q4 2027**

Kalaris Therapeutics

1) Private placement included participation from both new and existing investors, including ADAR1 Capital Management, Coastlands Capital, Invus, RTW Investments, Samsara BioCapital, Woodline Partners LP and others.


Kalaris
Therapeutics

Glossary

Glossary

BCVA: Best Corrected Visual Acuity

Cmax: Maximum Plasma Concentration

CNV: choroidal Neovascularization

CST: Central Subfield Thickness

DLT: Dose Limiting Toxicity

DME: Diabetic Macular Edema

DR: Diabetic Retinopathy

ETDRS: Early Treatment Diabetic Retinopathy Study

HCP: Host Cell Protein

HSPG: Heparan Sulfate Proteoglycans

IOI: Intraocular Inflammation

IOP: Intraocular Pressure

IRF: Intraretinal Fluid

nAMD: neovascular Age-related Macular Degeneration

OCT: Optical Coherence Tomography

PK: Pharmacokinetics

RVO: Retinal Vein Occlusion

SAD: Single Ascending Dose

SAE: Serious Adverse Events

SD-OCT: Spectral-Domain Optical Coherence Tomography

TRF: Total Retinal Fluid

Anti-VEGF Therapeutics Background

Lessons from over two decades of using Anti-VEGF to treat retinal disease

VEGF is the **primary mediator and the key target** for pathologic angiogenesis and exudation (permeability) in retinal disease[1]

Anti-VEGF therapy has **revolutionized treatment** for major retinal diseases[2]

VEGF has been the **primary target for neovascular / exudative retinal diseases** for over 20 years

Multiple anti-VEGF agents have become **blockbuster therapies,** treating millions of patients

>$15 Billion[3] global branded anti-VEGF market in 2024, projected to grow to approx. $18B by 2029[4]

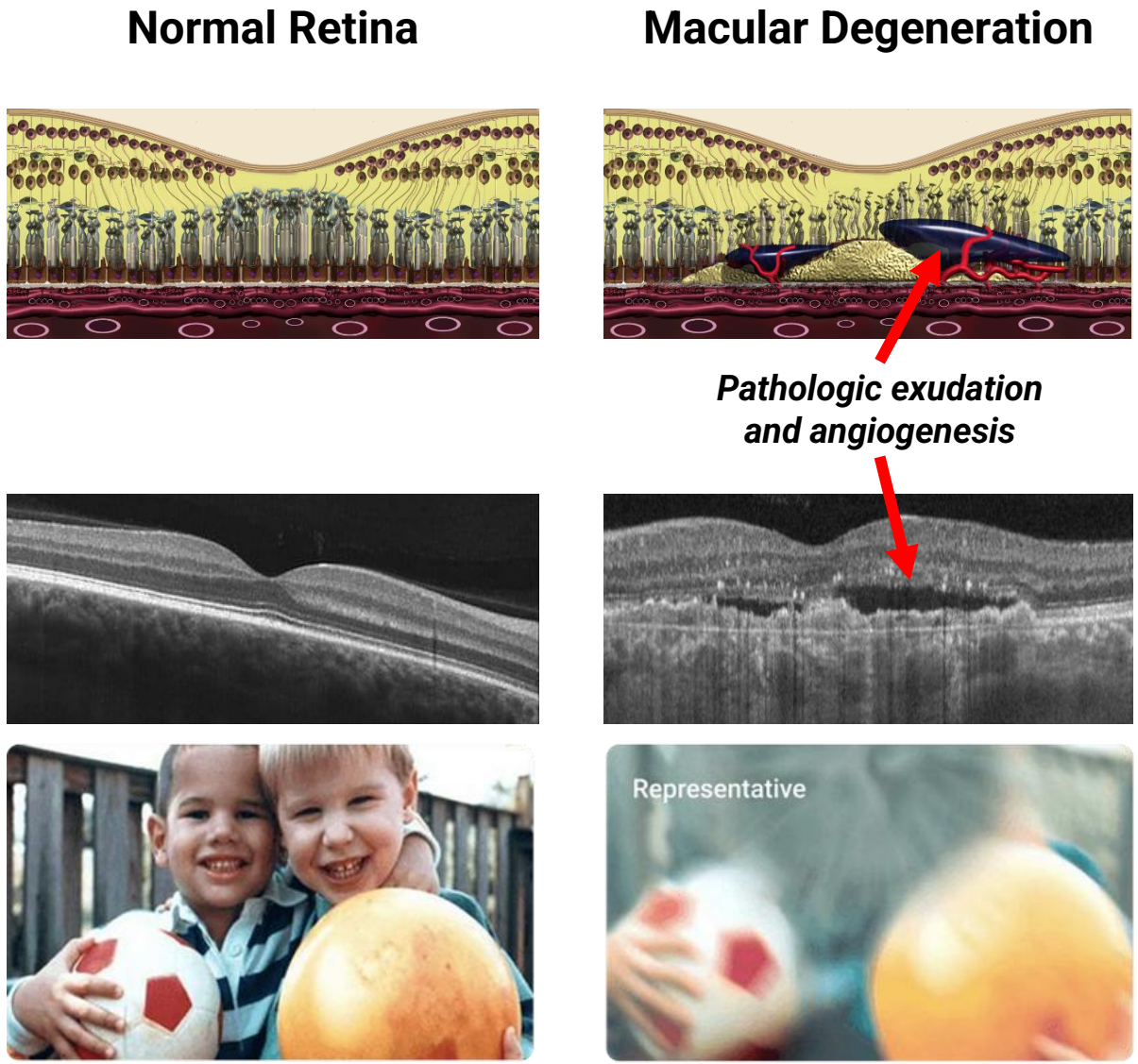
Unmet need remains high, with suboptimal real-world outcomes commonly explained by undertreatment due to onerous visit regimen[5,6,7,8,9]

Sources: 1) Witmer, A. N., Vrensen, G. F. J. M., Van Noorden, C. J. F., & Schlingemann, R. O. (2003). Vascular endothelial growth factors and angiogenesis in eye disease. Progress in retinal and eye research, 22(1), 1-29.2) Solomon, Sharon D., Kristina Lindsley, Satyanarayana S. Vedula, Magdalena G. Krzystolik, and Barbara S. Hawkins. "Anti-vascular endothelial growth factor for neovascular age-related macular degeneration." Cochrane Database of Systematic Reviews 8 (2014); 3) Based on publicly available sales data 2024; 4) 2024 Retinal Pharmaceuticals Market Report, Market Scope September 2024; 5) Prenner, J.L. · Halperin, L.S. · Rycroft, C., Am J Ophthalmol. 2015; 160:725-731.e1; 6) Varano, M. · Eter, N. · Winyard, S., Clin Ophthalmol. 2015; 9:2243-2250; 7) Monés, J. · Singh, R.P. · Bandello, F., Ophthalmologica. 2020; 243:1-8; 8) Gohil, R. · Crosby-Nwaobi, R. · Forbes, A., PLOS ONE. 2015; 10, e0129361; 9) MacCumber, M.W. · Yu, J.S. · Sagkriotis, A., Can J Ophthalmol. 2023; 58:252-261

Kalaris Therapeutics

VEGF is the primary mediator and the key target for pathologic angiogenesis and exudation (permeability) in retinal disease

Growth and leakage from abnormal vessels leads to visual impairment in diseases such as nAMD, DME, and RVO. VEGF is a primary mediator of this pathology.

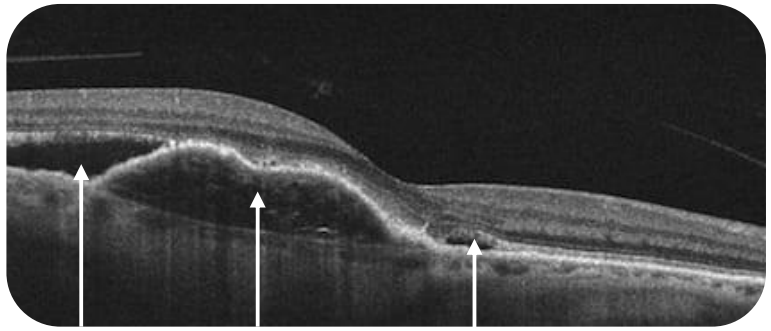


Source: Apte, R. S., Chen, D. S., & Ferrara, N. (2019). VEGF in signaling and disease: beyond discovery and development. Cell, 176(6), 1248-1264.

Kalaris Therapeutics

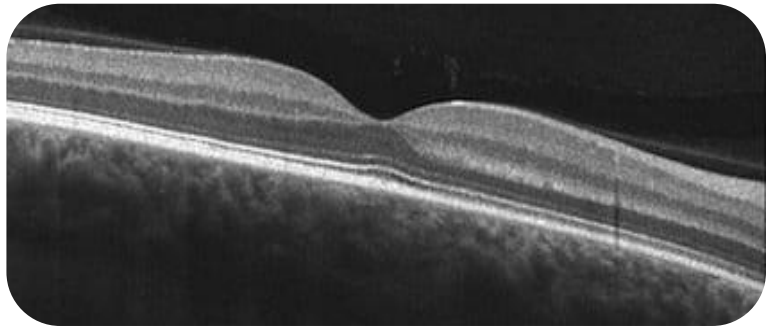
Anti-VEGF therapy has revolutionized treatment for major retinal diseases

Pre-Anti-VEGF Treatment



Pathological exudation

Post-Anti-VEGF Treatment



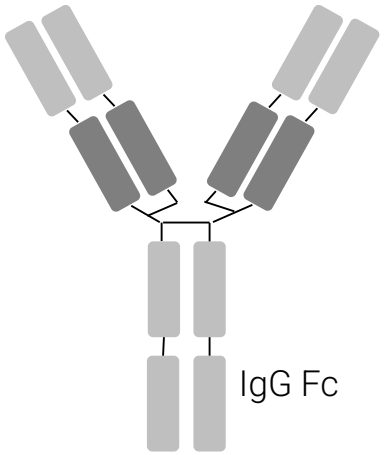
- Anti-VEGFs have a potent anti-permeability effect, causing reduction or resolution of pathological fluid, often leading to visual acuity improvements
- Retinal neovascular diseases treated with anti-VEGF as standard of care include:
  - **nAMD**: neovascular age-related macular degeneration
  - **DME**: diabetic macular edema
  - **DR**: diabetic retinopathy
  - **RVO**: retinal vein occlusion
- Optical coherence tomography (OCT) is the current standard for quantitatively detecting fluid presence across various retinal layers, along with other pathological features

Kalaris Therapeutics

Source: Solomon, S. D., Lindsley, K., Vedula, S. S., Krzystolik, M. G., & Hawkins, B. S. (2014). Anti-vascular endothelial growth factor for neovascular age-related macular degeneration. Cochrane Database of Systematic Reviews, (8).

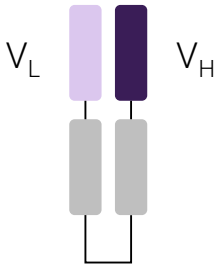
VEGF has been the primary target for neovascular / exudative retinal diseases for over 20 years

Bevacizumab



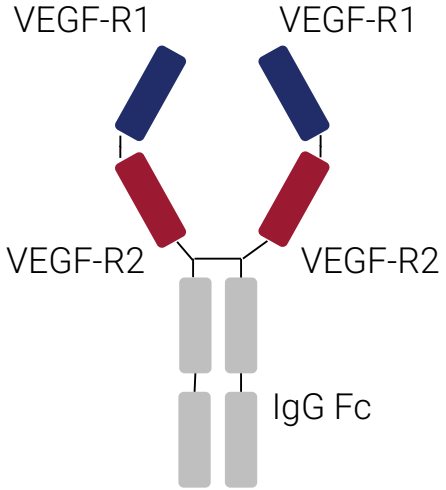
Type: **mAb targeting VEGF**
Licensed use: **Oncology**
Launch: **2005**

Ranibizumab



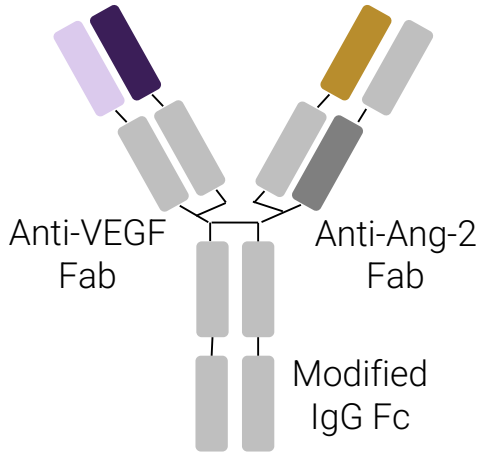
Type: **Fab fragment targeting VEGF**
Original use: **Ophthalmology**
Launch: **2006**

Aflibercept



Type: **Decoy Receptor binding to VEGF**
Original use: **Oncology**
Launch: **2011**

Faricimab

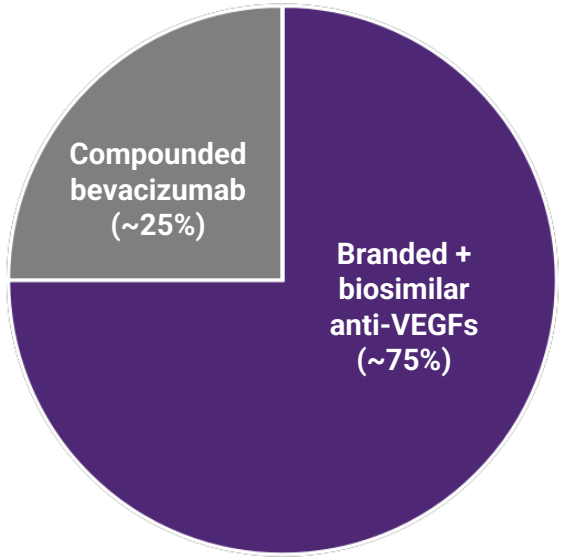


Type: **Bi-specific mAb VEGF & Ang-2***
Original use: **Ophthalmology**
Launch: **2022**

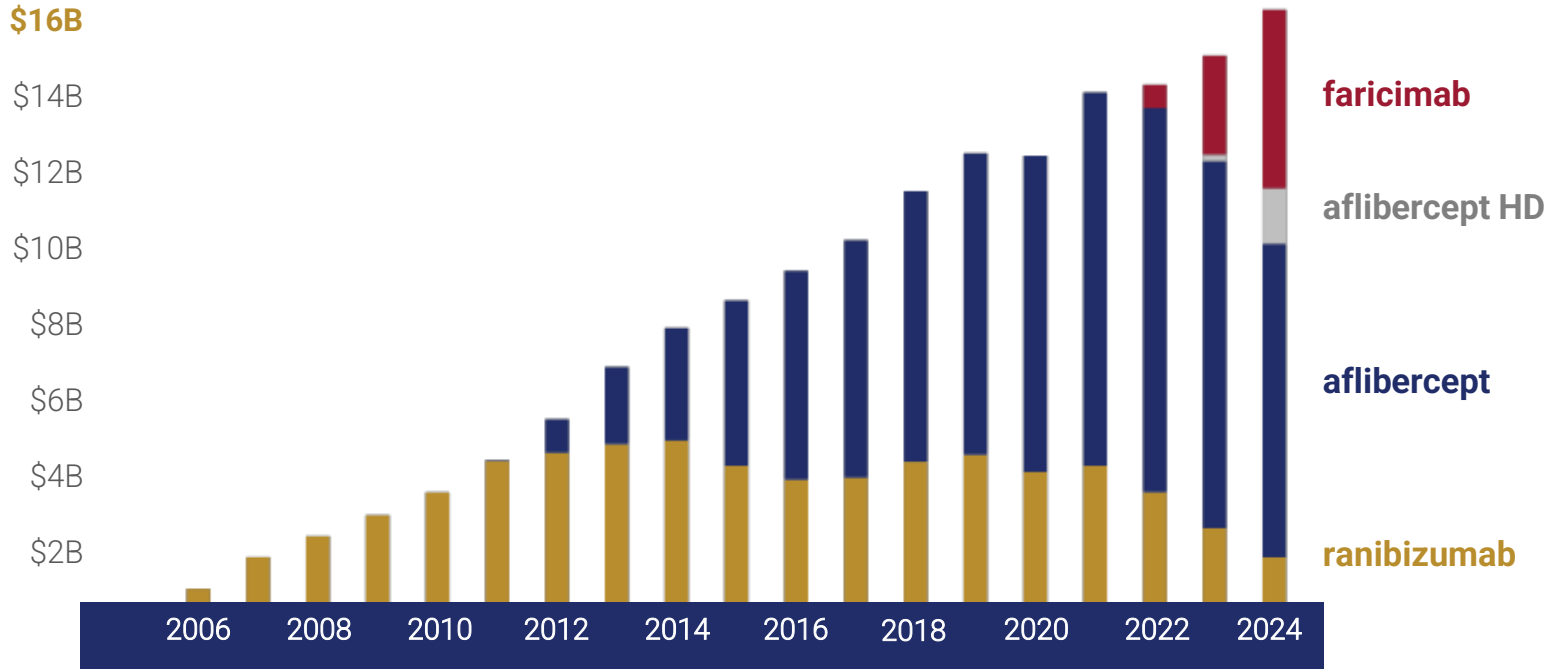
*According to the VABYSMO Prescribing Information, "The contribution of Ang-2 inhibition to the treatment effect and clinical response for nAMD, DME, and RVO has yet to be established."[1]
Source: 1) VABYSMO Prescribing Information accessed via AccessData.FDA.gov July 8, 2025
Additional Sources: Tatsumi, Tomoaki. (2023). Current Treatments for Diabetic Macular Edema. International Journal of Molecular Sciences. 24. 9591. 10.3390/ijms24119591; FDA Approval.

>$15B global branded anti-VEGF market in 2024, projected to grow to approximately $18B by 2029[1,2]

Global Anti-VEGF Units in Retinal Disease (2024)[1]



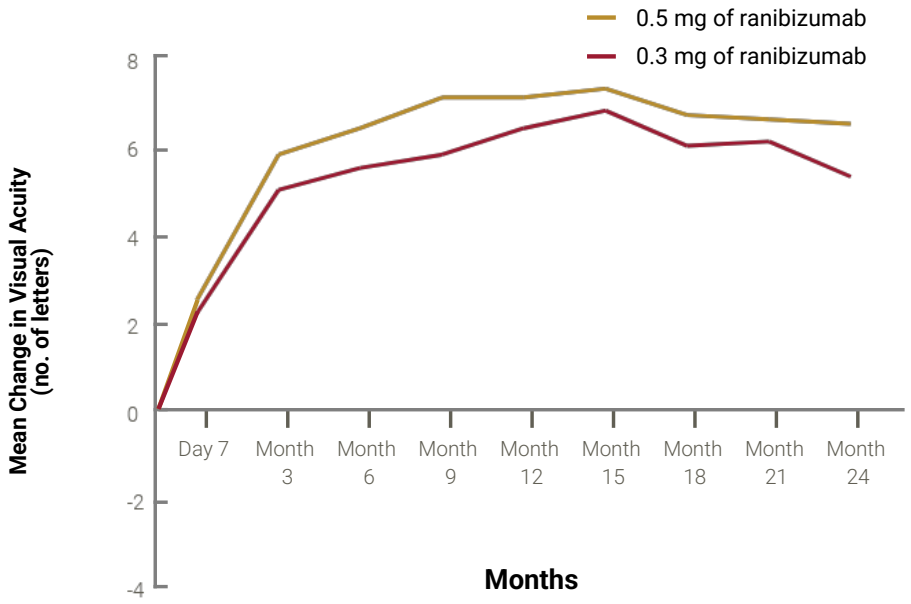
Branded Anti-VEGF Therapies 2024 Global Sales[2]



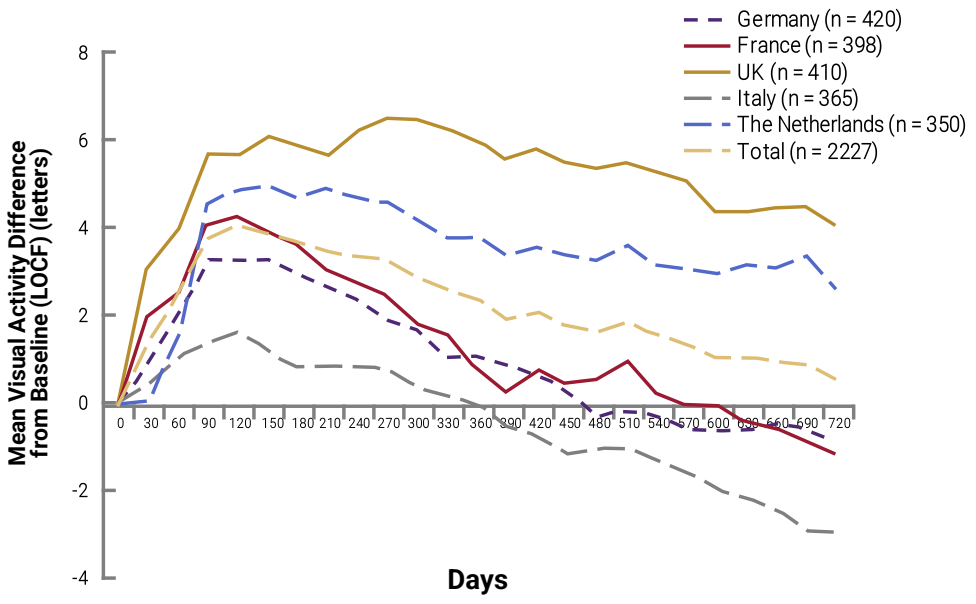
Sources: 1) 2025 Retinal Pharmaceuticals Market Report, Market Scope October 2025; 2) Based on publicly available sales data 2024

Suboptimal Real-World outcomes as compared to clinical trial results[1,2,3,4,5]

Registrational Clinical Trial[6]



Real World Study[7]



A major unmet need remains for a long-acting agent that preserves patient vision and reduces patient visit burden

Sources: 1) Prenner, J.L. · Halperin, L.S. · Rycroft, C., Am J Ophthalmol. 2015; 160:725-731.e1; 2) Varano, M. · Eter, N. · Winyard, S., Clin Ophthalmol. 2015; 9:2243-2250; 3) Monés, J. · Singh, R.P. · Bandello, F., Ophthalmologica. 2020; 243:1-8; 4) Gohil, R. · Crosby-Nwaobi, R. · Forbes, A., PLOS ONE. 2015; 10, e0129361; 5) MacCumber, M.W. · Yu, J.S. · Sagkriotis, A., Can J Ophthalmol. 2023; 58:252-261; 6) Rosenfeld PJ, Brown DM, Heier JS, Boyer DS, Kaiser PK, Chung CY, Kim RY; MARINA Study Group. Ranibizumab for neovascular age-related macular degeneration. N Engl J Med. 2006; 7) Holz FG, et al. Br J Ophthalmol 2015;99:220-226