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THE OPHTHALMOLOGY MEDICINES COMPANY

Corporate Presentation

March 2026

Forward-Looking Statements

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We are a precommercial, retina-focused biotech on the move

Wholly Owned

KSI-101

- Robust end-of-study results from Phase 1b APEX
- MOA validated by scientific community
- Phase 3 PEAK and PINNACLE topline data expected in 4Q 2026 and 2Q 2027
- Commercial opportunity of 150,000+ initial addressable patients with headroom

Tarcocimab & KSI-501

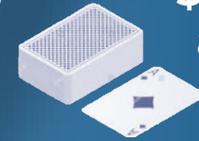
- Science-based “heavyweights”
- Tarcocimab: targeting BLA mid-2026 in wet AMD, RVO and diabetic retinopathy
- KSI-501: bispecific ABC[®] may be even better!

Pipeline, Digital Health, Manufacturing

- KSI-102, KSI-103: bispecifics for inflammation
- Duets for glaucoma and geographic atrophy
- VETi: AI headsets for commercial leadership
- URSUS: commercial manufacturing

A potent reason to believe in Kodiak

2 options in the \$15+ billion anti-VEGF market



Accelerating our technology and pipeline leadership

With 3 Phase 3 retina programs leveraging our 15-year history, Kodiak is at a decisive moment and represents a compelling investment opportunity

For patients with MESI

KSI-101

Anti-IL-6, VEGF trap
bispecific protein

- High-strength, dual inhibition of IL-6 and VEGF
- Safe and potent control of macular edema secondary to inflammation (MESI)

Greenfield market opportunity

For patients with retinal vascular diseases

Tarcocimab

Anti-VEGF **ABC® biologic**

- Strong immediacy
- Industry leading durability
- BLA filing expected 2026 in 3 indications (wet AMD, DR and RVO)

Quadrant of core unmet need

KSI-501

Anti-IL-6, VEGF trap
bispecific ABC® biologic

- Improved efficacy beyond anti-VEGF monotherapies
- Strong immediacy
- Industry leading durability

Quadrant of core unmet need

KEY CATALYSTS (expected)

Feb 2026

24-week data from Phase 1b APEX



Q4 2026

Topline data from Phase 3 PEAK (MESI)

Q2 2027

Topline data from Phase 3 PINNACLE (MESI)

Q1 2026

Topline data from Phase 3 GLOW2 (DR)

Q3 2026

Topline data from Phase 3 DAYBREAK (wet AMD)

BLA filing in 2026

Wet AMD, Diabetic Retinopathy, Retinal Vein Occlusion

Q3 2026

Topline data from Phase 3 DAYBREAK (wet AMD)

All three assets are on track for Phase 3 topline data readouts in 2026; a series of BLA filings possible in 2026 and 2027

	MOA	Indication	Phase 3 Study	Complete	Q1 '26	Q2 '26	Q3 '26	Q4 '26	1Q '27	2Q '27	3Q '27	4Q '27	
Tarcocimab	Anti-VEGF Antibody Biopolymer Conjugate (ABC)	RVO	BEACON	✓									
		DR	GLOW1	✓									
		Wet AMD	DAYLIGHT	✓									
		DR	GLOW2			🎯							
		Wet AMD	DAYBREAK					🎯					
				Enrollment Complete									
KSI-501	Anti-IL-6, VEGF Trap ABC	Wet AMD	DAYBREAK				🎯						
		TBD	2nd Pivotal								🎯	BLA*	
KSI-101	Anti-IL-6, VEGF Trap Protein	MESI	PEAK					🎯					
			PINNACLE								🎯	BLA*	
				Actively Enrolling									

ABC: antibody biopolymer conjugate; DR: diabetic retinopathy; AMD: age-related macular edema; RVO: retinal vein occlusion; MESI: macular edema secondary to inflammation; BLA: biologics license application; GLOW2: NCT06270836. DAYBREAK: NCT06556368; PEAK: NCT06990399; PINNACLE: NCT06996080

*Timing of topline data readouts reflect current expectations, subject to change

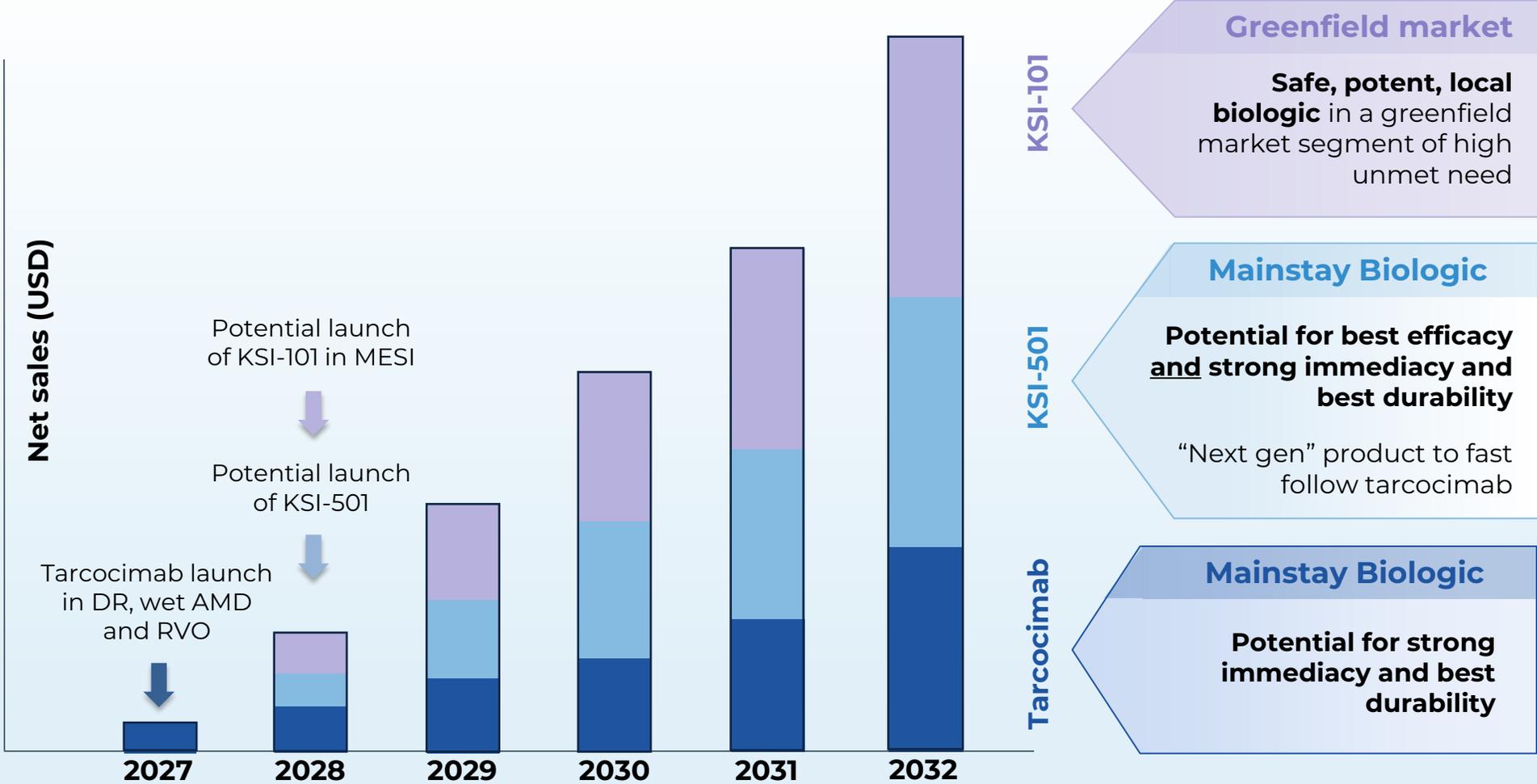
= active and ongoing Phase 3 study
🎯 = BLA-facing topline data readouts (expected)

Our portfolio has the potential for sustainable revenue beginning 2027, with built-in life cycle management, risk diversification and continued leadership in retinal innovation

Kodiak's assets are proprietary and wholly-owned.

We have the flexibility in our commercialization decisions to support adoption of our products in the marketplace.

Net Sales Potential of Kodiak Clinical Portfolio (Illustrative)



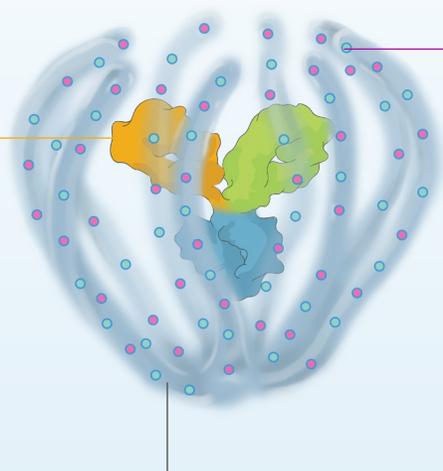
A growing pipeline of multi-functional investigational therapies is built in modular fashion on our ABC® Platform and is maturing rapidly

ANTIBODY BIOPOLYMER CONJUGATE DRUG (“ABCD”)

Designed to embed small molecules and other **Active Pharmaceutical Ingredients (“API”)** such as oligonucleotides and peptides into the biopolymer backbone for release over a designed-in time period

Antibody or Other Biologic

Engineered to exhibit high binding affinity and specificity. Any biologic can be conjugated to the biopolymer



Drug Cargo

Diverse APIs of varying biophysical properties including small molecules, macrocycles, peptides and oligonucleotides are embedded in the biopolymer and released over a designed-in time

Biopolymer

Combines multiple APIs and can be tailored to meet a specific therapeutic goal. It is optically clear and made of phosphorylcholine, the primary hydrophilic component of human cell membranes

1

Conjugates of Diverse APIs +/- a Biologic
Target both intracellular and extracellular pathways

2

High Drug Antibody Ratio (“DAR”)
Can include APIs with DAR of 10 up to >250

3

Tailored Release of APIs
Release of API payloads enabled by pH modulation or enzymatic cleavage of linkers

4

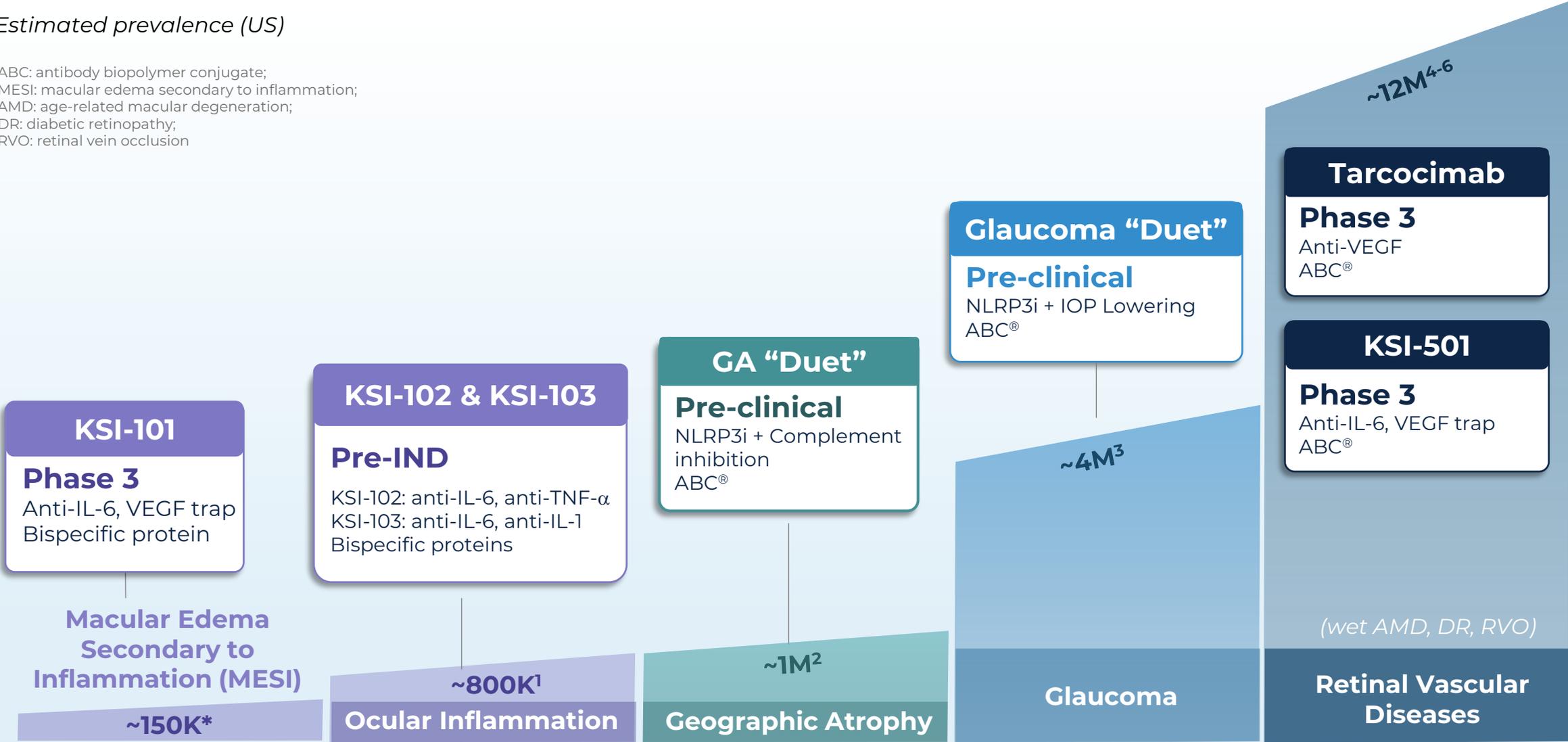
Proven Safety Record of the ABC Platform
>2,500 patient years of experience in patients

A new combination of targeting, high drug loading, mixed API formats and tailored drug release – applications in ophthalmic and systemic diseases

Our Phase 3 clinical programs (3) and pipeline assets (4) are designed to address the leading causes of vision loss

Estimated prevalence (US)

ABC: antibody biopolymer conjugate;
 MESI: macular edema secondary to inflammation;
 AMD: age-related macular degeneration;
 DR: diabetic retinopathy;
 RVO: retinal vein occlusion



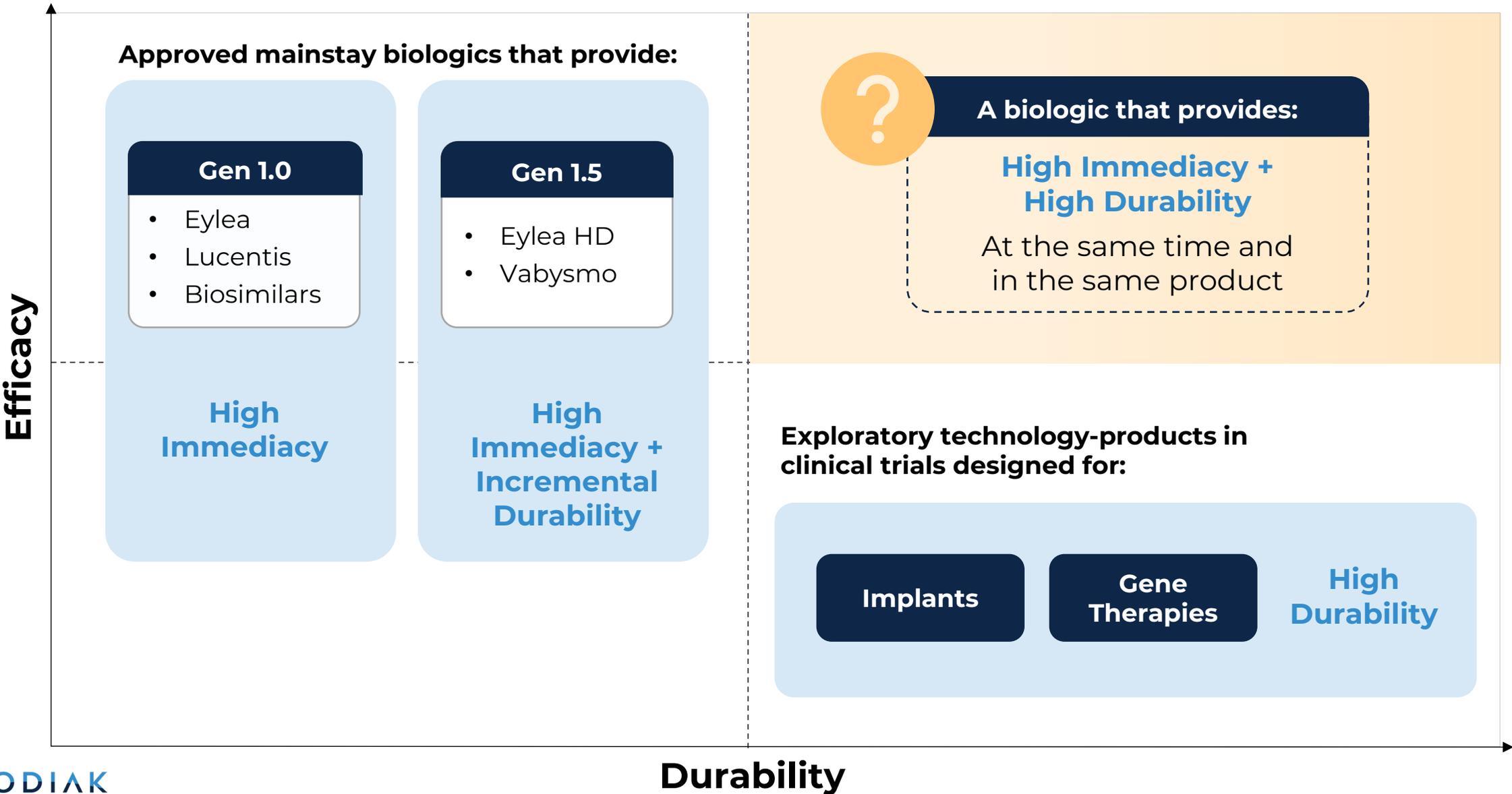
*KSI-101 initial addressable population in MESI. Kodiak Data on File and shared Investor R&D Day July 2025.

A vertical strip on the left side of the slide shows a microscopic view of cells, likely cancer cells, with a light blue and white color scheme. The cells are clustered and have irregular shapes, some with prominent nuclei.

Tarcocimab & KSI-501

Retinal Vascular Diseases and the Anti-VEGF Market

There remains valuable open space in the \$15 billion retinal vascular diseases market, despite the availability of approved biologics and clinical trials of new exploratory technologies

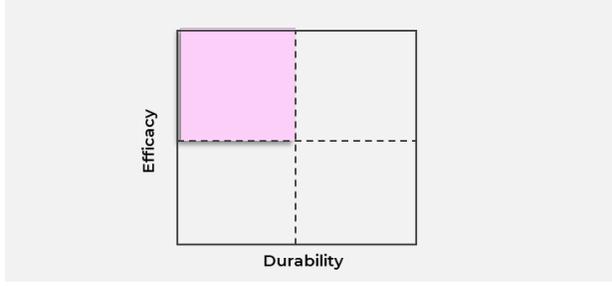
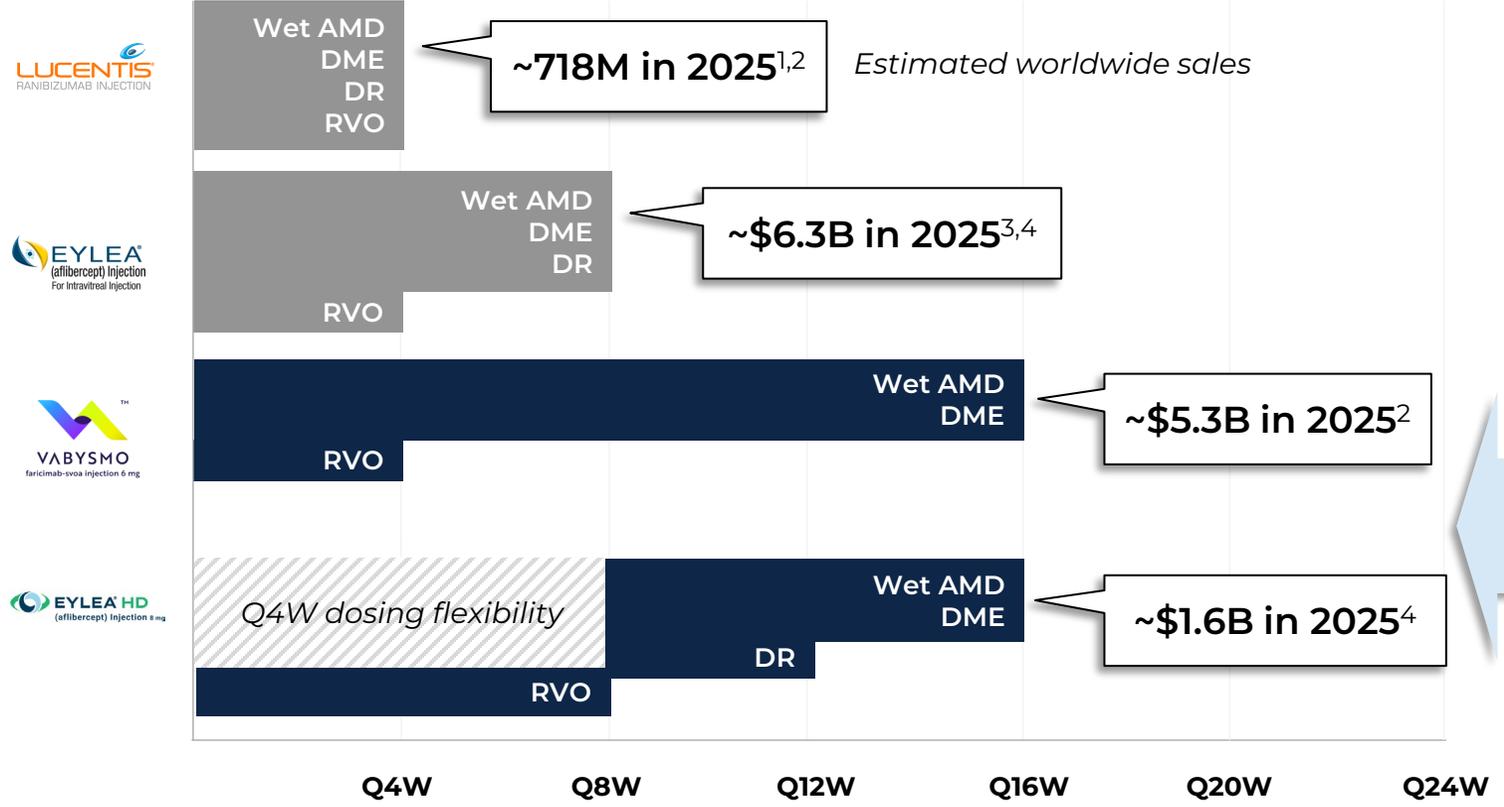


The Gen 1.0 biologics that provide **immediacy** and the Gen 1.5 biologics that provide **immediacy and incremental durability** are meaningful for patients and physicians

Dosing regimen per label for approved intravitreal biologics

Gen 1.0
Good efficacy, limited durability

Gen 1.5
Immediacy, incremental durability

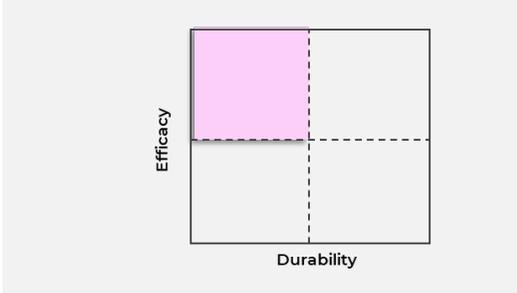


The commercial success of new branded therapies is a testament to:

- The **power of the intravitreal biologic** as the mainstay of therapy
- The **unmet need** that remains for patients

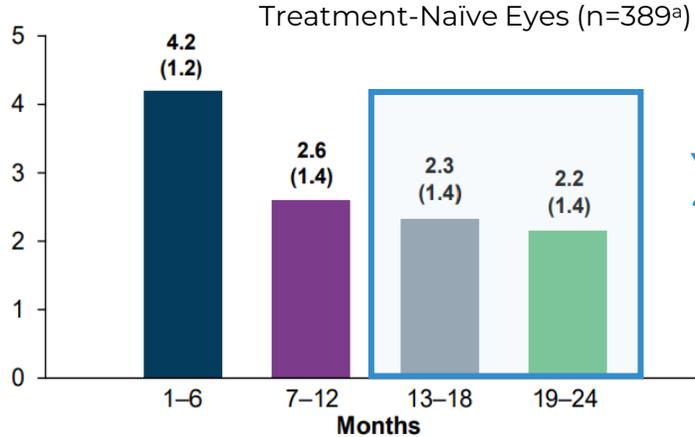
Despite their commercial success, real-world data in wet AMD show that most patients are falling short of the durability promise of the Gen 1.5 labels

Faricimab injections observed after the first 6 months of treatment through Year 2 in the FARENTINA study¹



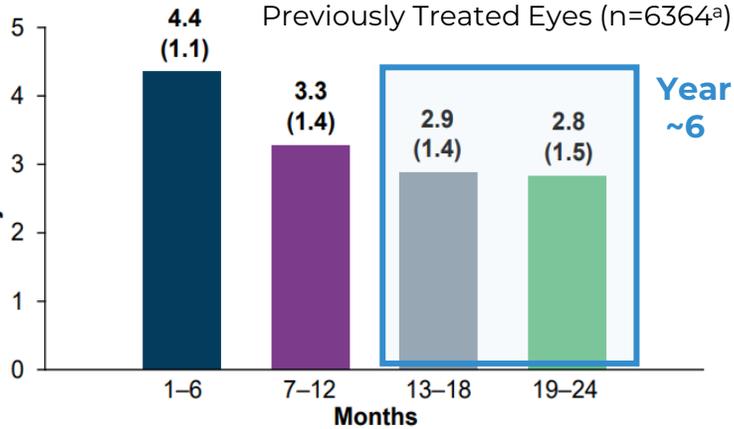
Most patients are on a Q8W-Q12W schedule in the real world in Year 2

^a Only eyes with ≥ 1 injection after index and prior to censoring were included.



**Year 2:
~4.5**

In Year 2, patients received 4.5 injections which is an average dosing interval of every 11.5 weeks



**Year 2:
~6**

In Year 2, patients received 6.0 injections which is an average dosing interval of every 8 weeks

Technologies being explored in clinical trials, including implants and gene therapies, are engineering for high durability but are lacking immediacy

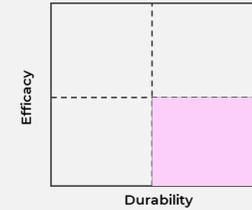
Implants

- Engineering for durability
- Long-term safety unknown

Gene Therapies

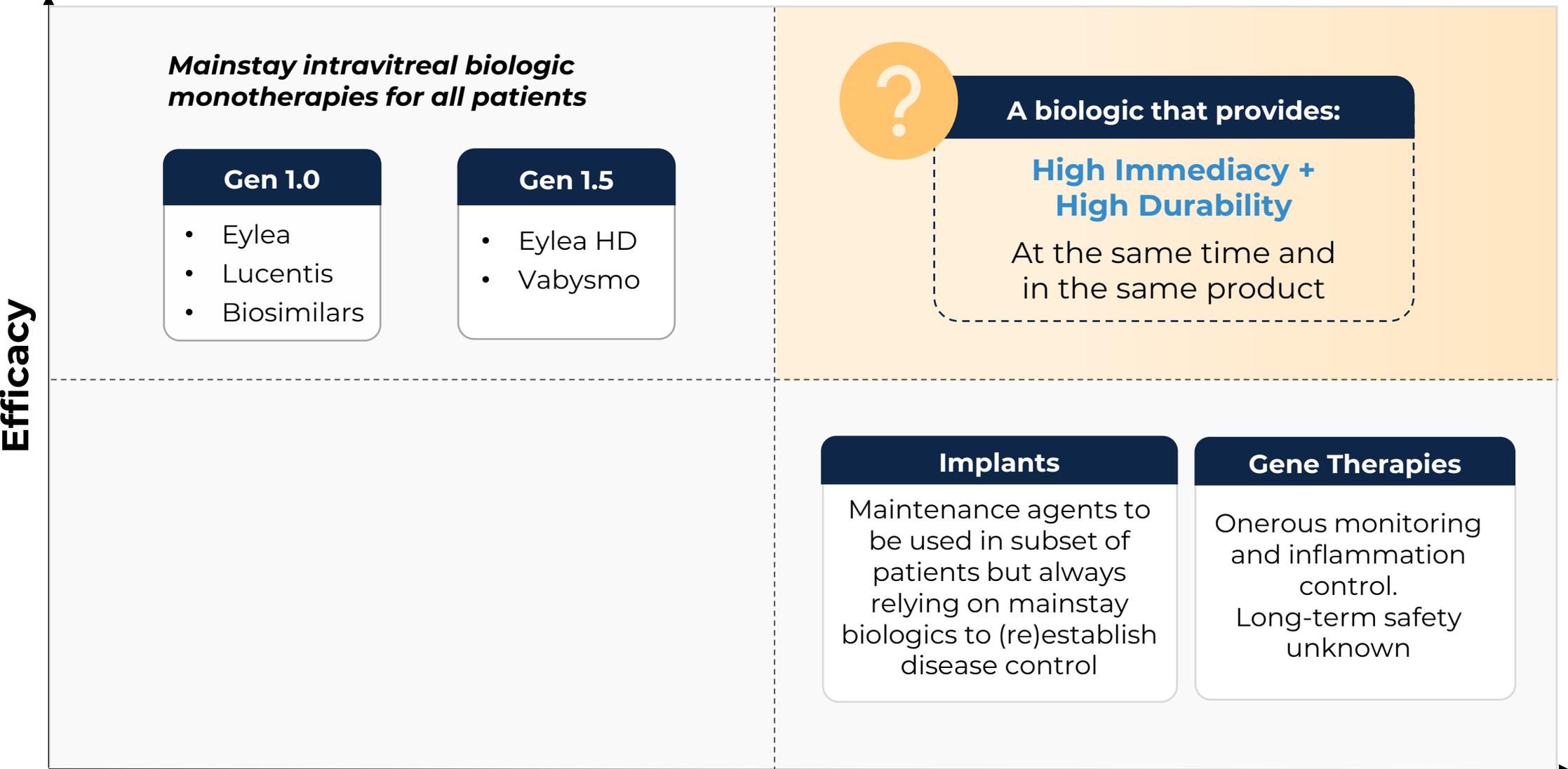
- Engineering for durability
- Onerous monitoring and inflammation control
- Long-term safety unknown

Implants and gene therapies are being tested **on top of mainstay biologics**, which are given during the **loading phase** and then as needed during the **maintenance phase**.



Maintenance agents to be used in subset of patients but always **relying on mainstay biologics to (re)establish disease control**

Therefore, the open space remains. A biologic that provides high immediacy and high durability *at the same time and in the same therapy* has the potential to be a heavy weight in this important \$15+ billion marketplace



Kodiak's ABC[®] Platform Science in Retina

The ABC Platform supports Kodiak's science of *immediacy and durability*



Designed-in Extended Tissue Residence Time

A proprietary phosphorylcholine-based polymer is conjugated to an antibody to increase molecular size which extends ocular half-life

High In-Vitro Potency

Both unconjugated protein and conjugated protein demonstrate high binding affinity and potency *in vitro*

Extended Ocular Half-Life in Animals

3x the ocular $t_{1/2}$ of approved intravitreal biologics when measured in rabbits following an intravitreal injection

Extended Ocular Half-Life in Humans

3x the ocular $t_{1/2}$ of faricimab when measured from aqueous humor in patients following an intravitreal injection



Powerful Immediacy via Unconjugated Antibody

The unconjugated protein delivers a strong “pulse” of anti-VEGF inhibition during the loading phase, or to recapture control of the disease in patients whose disease has reactivated

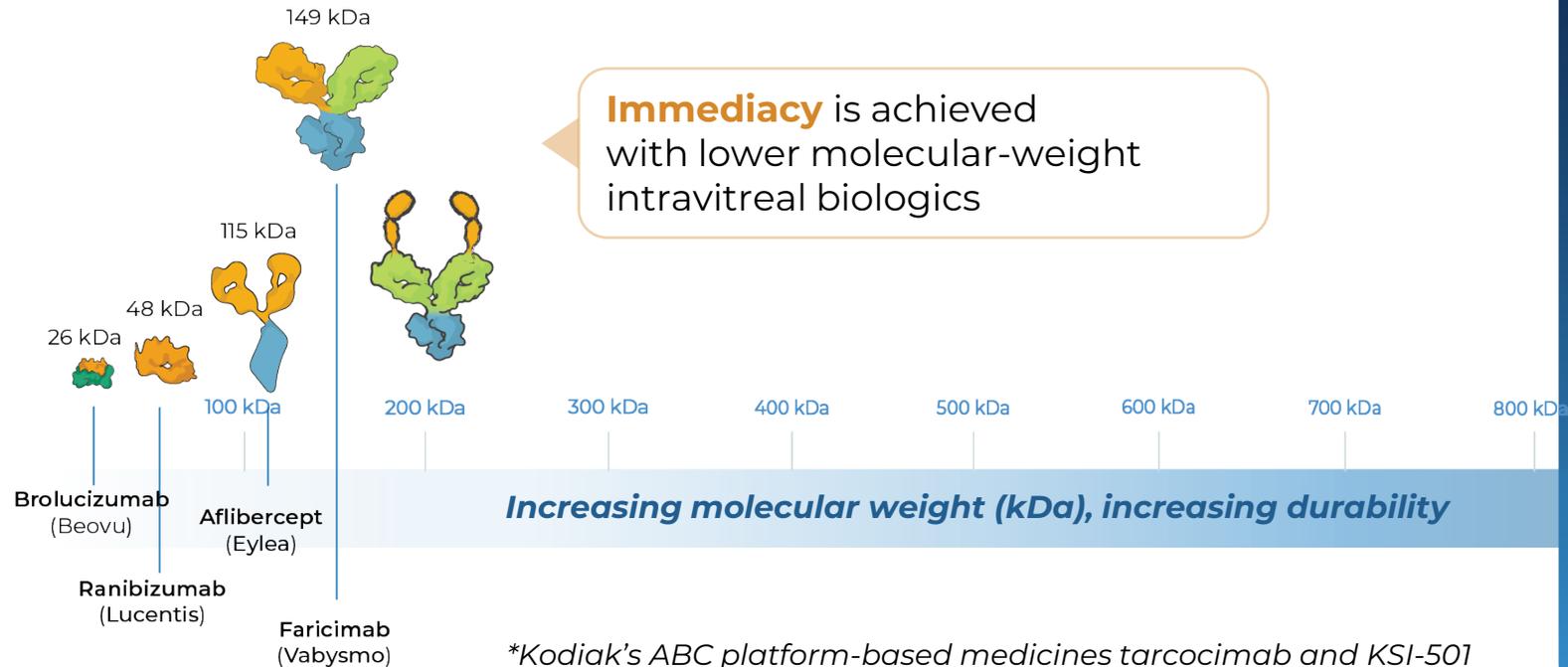
Extended Clinical Durability via Conjugated Antibody

The conjugated protein maintains the signature durability as seen in Kodiak's pivotal studies to date

ABC[®] Platform-based medicines have a high molecular weight which increases their ocular half-life compared to today's intravitreal biologics

Durability increases as molecular weight increases for intravitreal biologics

Immediacy is achieved with lower molecular-weight intravitreal biologics



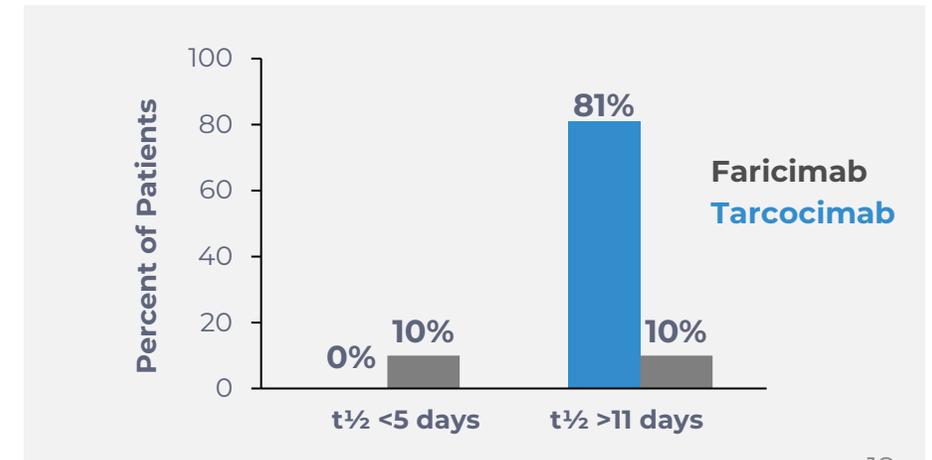
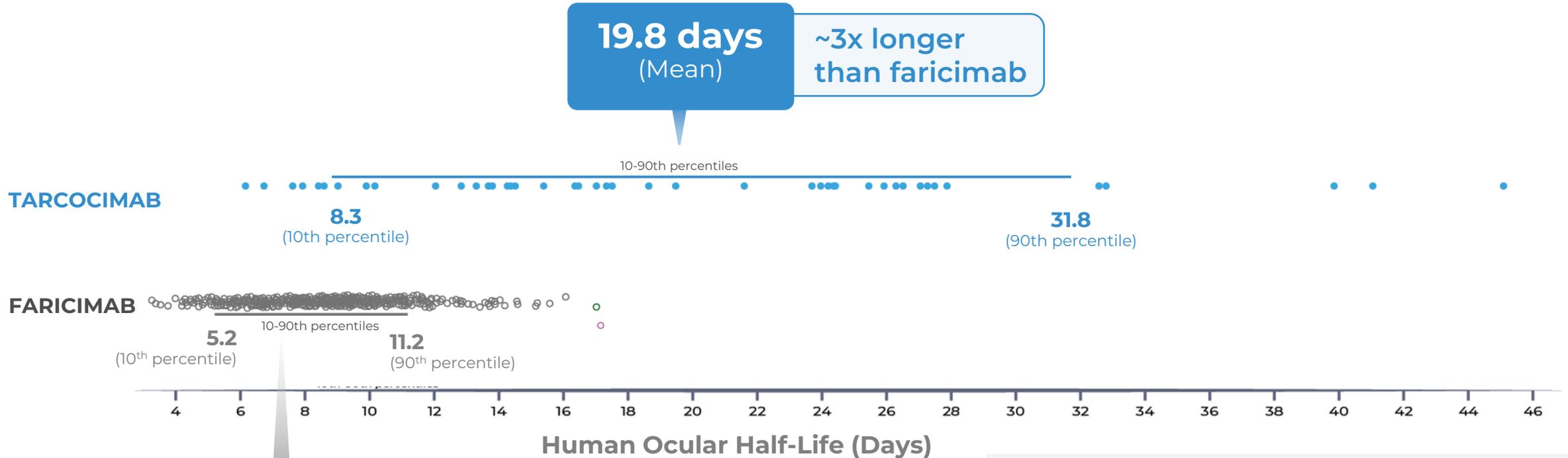
*Kodiak's ABC platform-based medicines tarcocimab and KSI-501 combine unconjugated and conjugated protein in a single biologic

Tarcocimab | 950 kDa

KSI-501 | 1000 kDa

Conjugated Portion of ABC[®] Platform-based medicines*

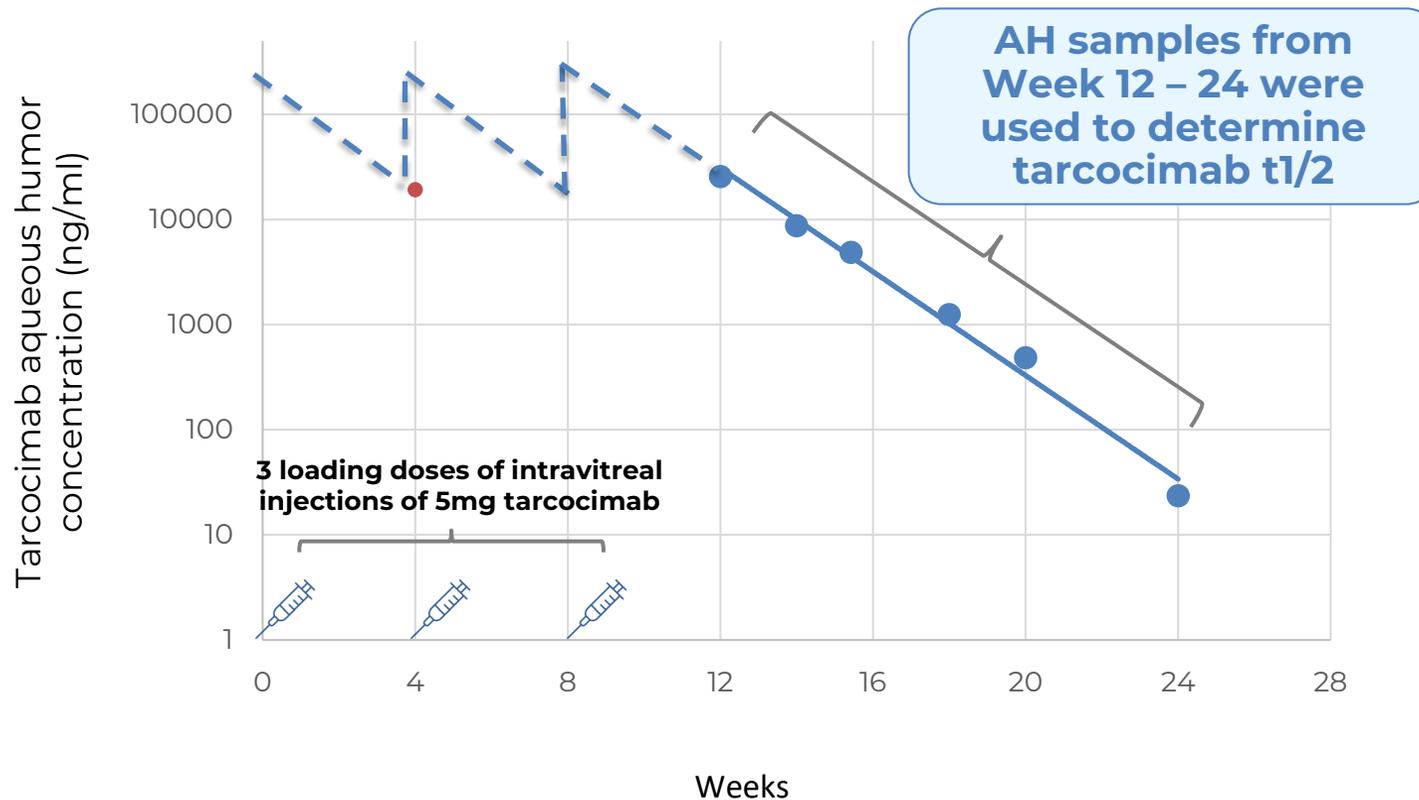
Tarcocimab has a mean ocular half-life in humans of 20 days, which is 3-fold longer than faricimab



Each dot represents the ocular half-life from one individual patient. Blue dots are tarcocimab from the Phase 1b study of tarcocimab in patients with wet AMD, DME and RVO. Gray dots are faricimab from Genentech, Inc. PK and ER of faricimab, Report # 1105763

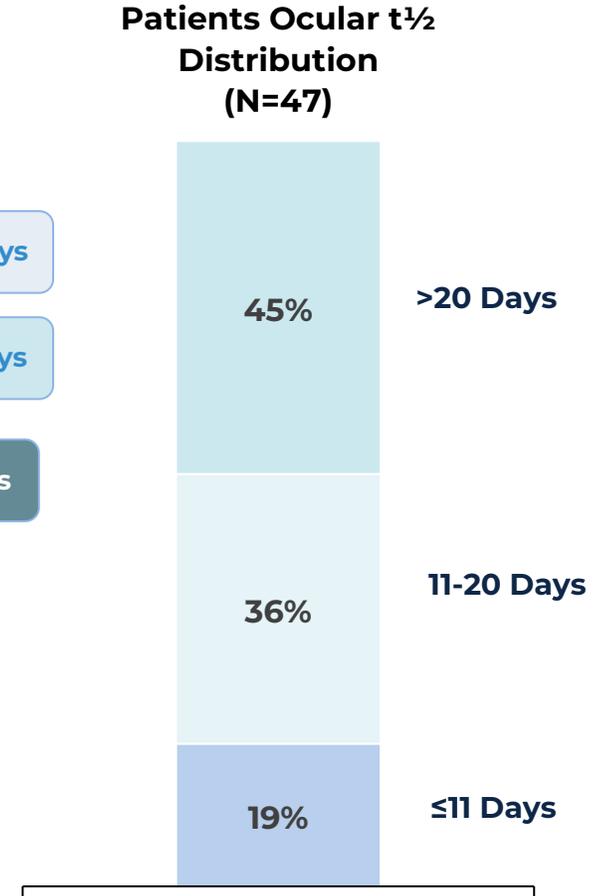
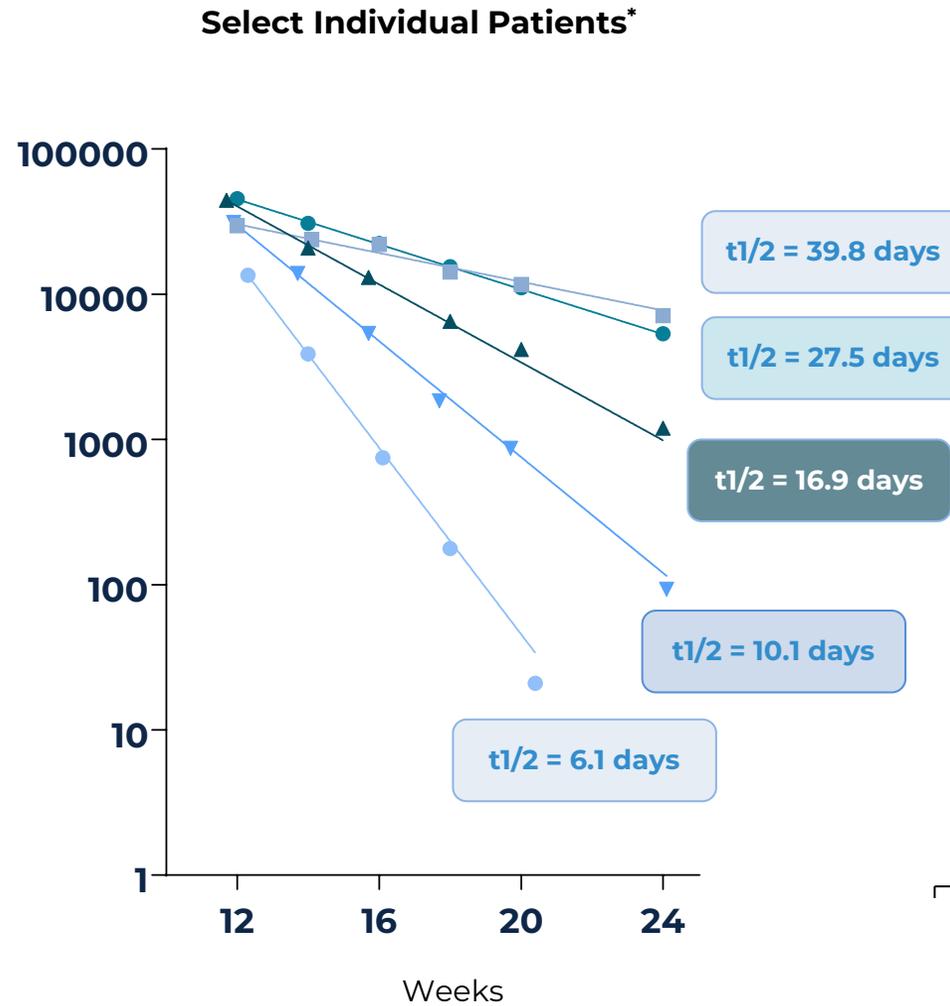
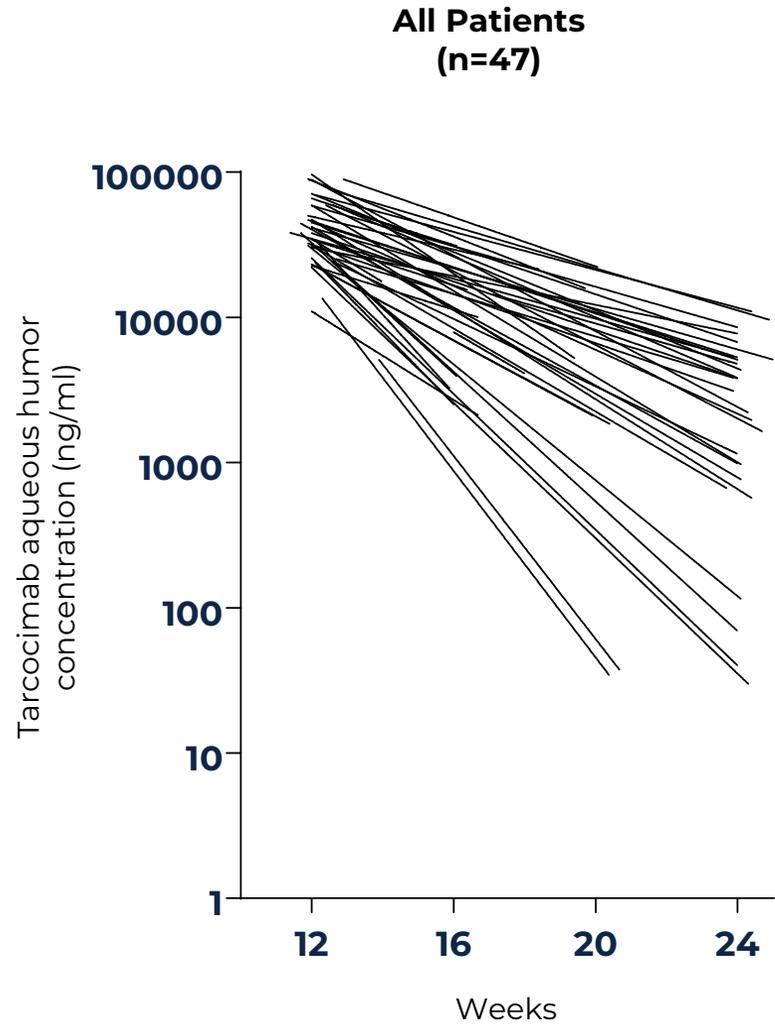
Tarcocimab's ocular half-life in humans was calculated by measuring aqueous humor concentrations over time from patients in the Phase 1b Study

Using Aqueous Humor Concentration of Tarcocimab to Determine Ocular $t_{1/2}$ in human

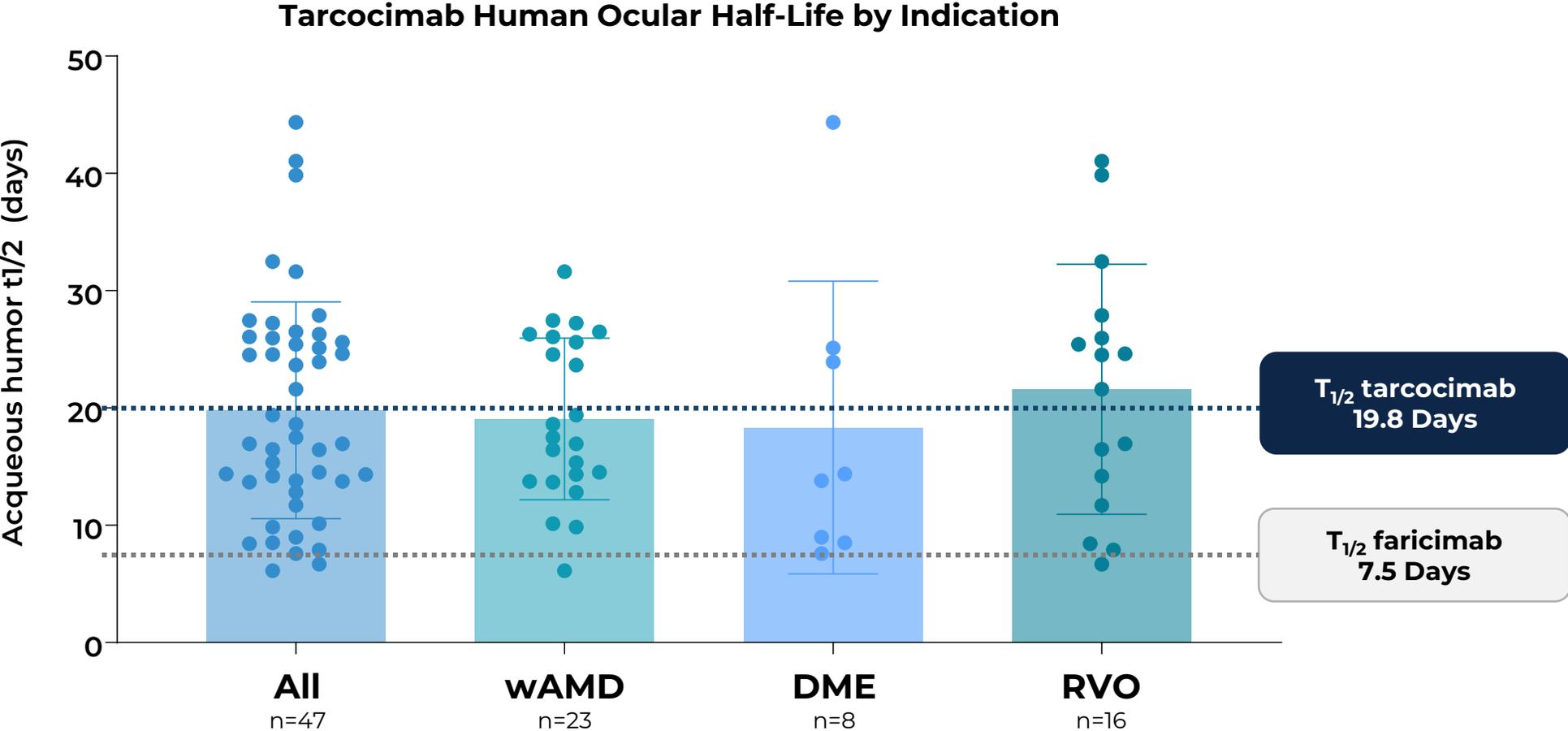


- Aqueous humor samples were collected from 47 subjects in the tarcocimab Phase 1b study in patients with wet AMD, DME and RVO and were used to evaluate tarcocimab ocular half life in patients
- Aqueous humor samples were collected at baseline and at Week 4, 12, 14, 16, 18, 20 and 24 and measured for tarcocimab concentrations
- Samples collected between the last loading dose and the next re-dose were used to determine ocular half-life of tarcocimab

Tarcocimab achieved an extended ocular half-life of >20 days in 45% of sampled patients from the Phase 1b Study



Tarcocimab demonstrated consistent 20-day ocular half-life across wet AMD, DME and RVO patients

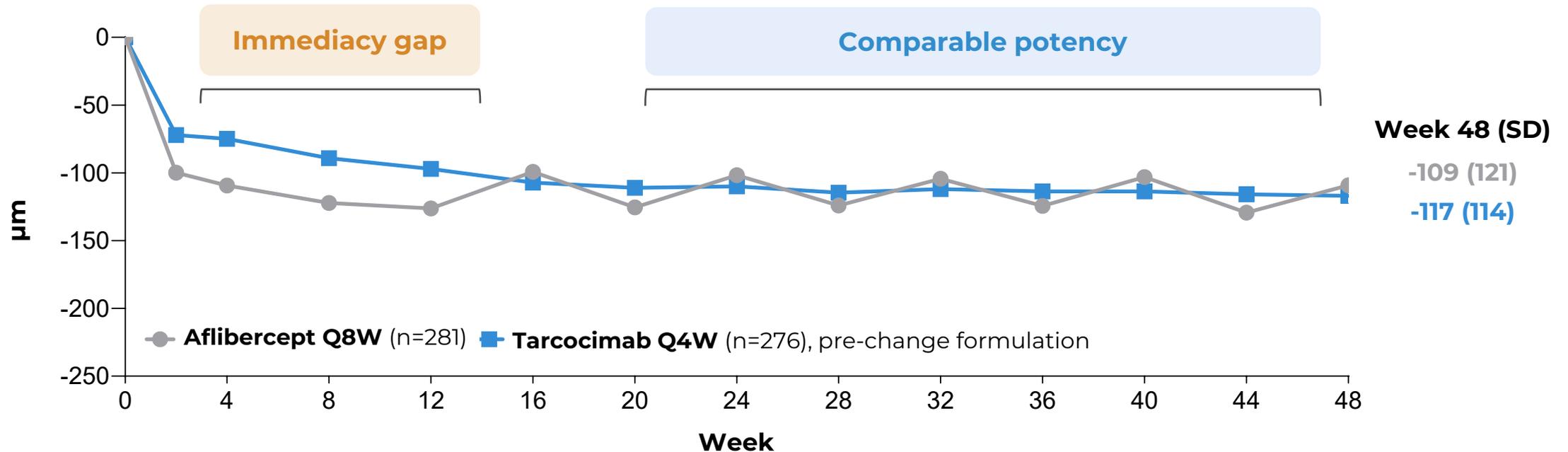


Height of the box represents the mean; error bars represent standard deviation.

Did this 3x-longer durability come at a cost?

- **Immediacy seemed to be the cost.** In the wet AMD DAYLIGHT Phase 3 study with the pre-change tarcocimab formulation, a deficit was seen in the loading phase, in the “immediacy” of the effect.
- After the loading phase, the drying potential or “potency” was comparable to aflibercept

Mean Change in OCT CST Over Time In the wet AMD DAYLIGHT Phase 3 Study

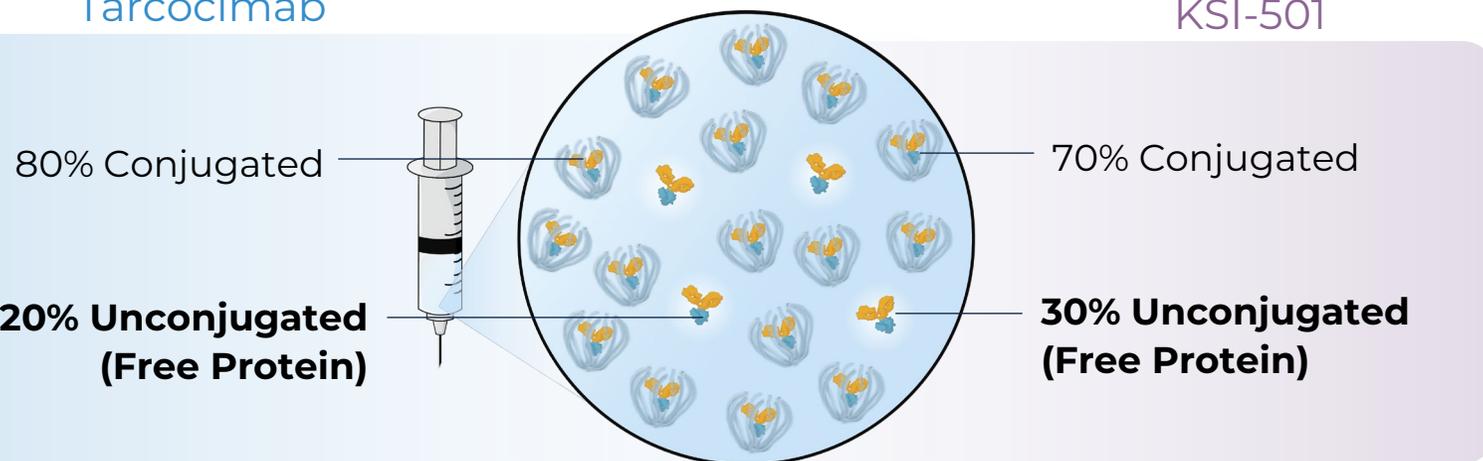


To fix the immediacy gap, we add **free protein** and **conjugated protein** to the commercial formulations for **tarcocimab** and **KSI-501**

Commercial Formulation

Tarcocimab

KSI-501



5 mg
4 mg
1 mg

Strength
(Total Anti-VEGF antibody)

Amount of **Conjugated Protein**

Amount of **Free Protein**

5 mg
3.5 mg
1.5 mg

The commercial formulation is designed to confer several key benefits:

- Improved **manufacturability**
- Increased **ease of dosing & safety**
- Maintenance of **dose & potency**
- Maintenance of **signature durability**

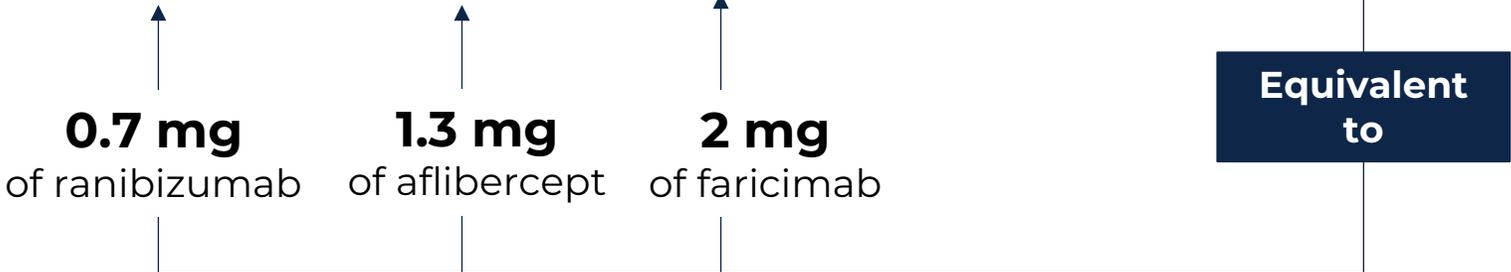
and

- Improved **immediacy**

The unconjugated portion of the commercial formulation for tarcocimab contains a high molar equivalent to approved intravitreal biologics

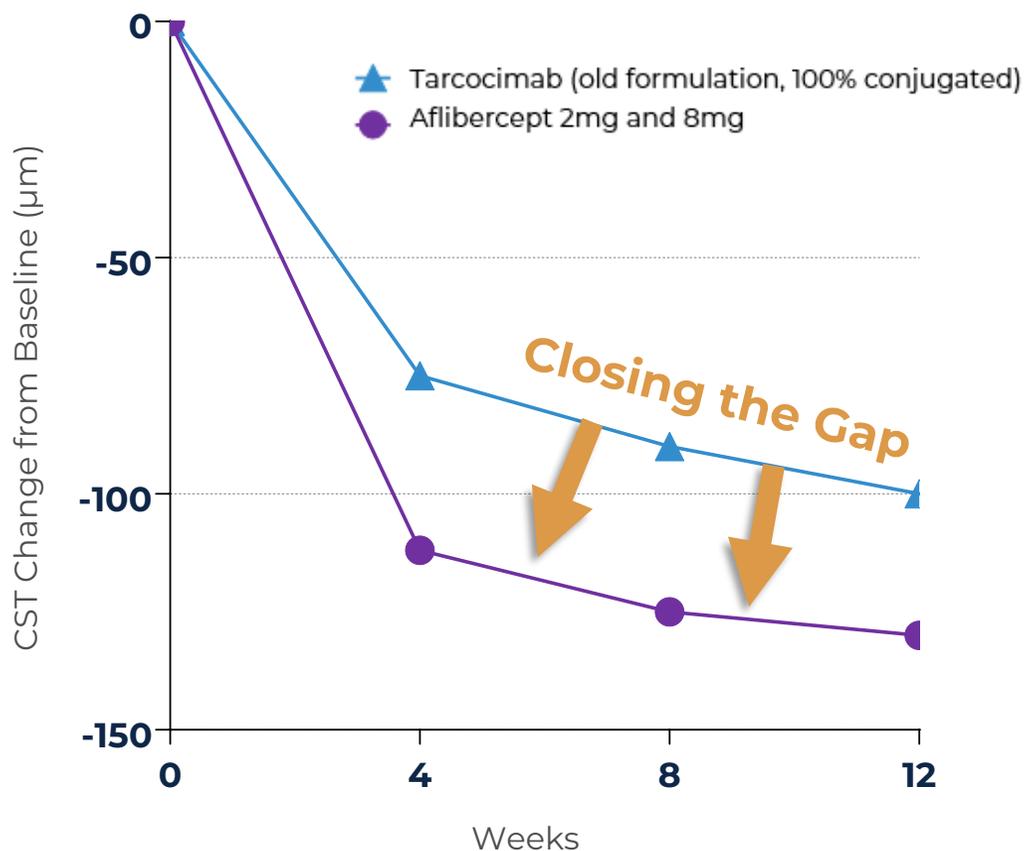
	Brolucizumab	Ranibizumab	Aflibercept	Faricimab	Tarcocimab Old Formulation	Tarcocimab Commercial Formulation	
Molecule Type	Single-Chain Antibody Fragment	Antibody Fragment	Fusion Protein	Antibody	Antibody Biopolymer Conjugate (ABC)	Unconjugated antibody + ABC	
Molecular Structure							
Molecular Weight	26 kDa	48 kDa	115 kDa	149 kDa	950 kDa	150 kDa	950 kDa
Clinical Dose	6 mg	0.3-0.5 mg	2 mg	6 mg	5 mg By weight of antibody	5 mg	
Equivalent Molar Dose	11	0.5	1.0	2	3.5	1 mg	4 mg
						0.7	2.8

Equivalent values are shown as fold changes relative to aflibercept. kDa = kilodalton



Free protein was added was to bring powerful and immediate disease control into the commercial formulation, while also improving manufacturability, dose administration and patient safety

Disease Control Through the Loading Dose Phase in Wet AMD*



Powerful and immediate disease control with the 20% unconjugated protein

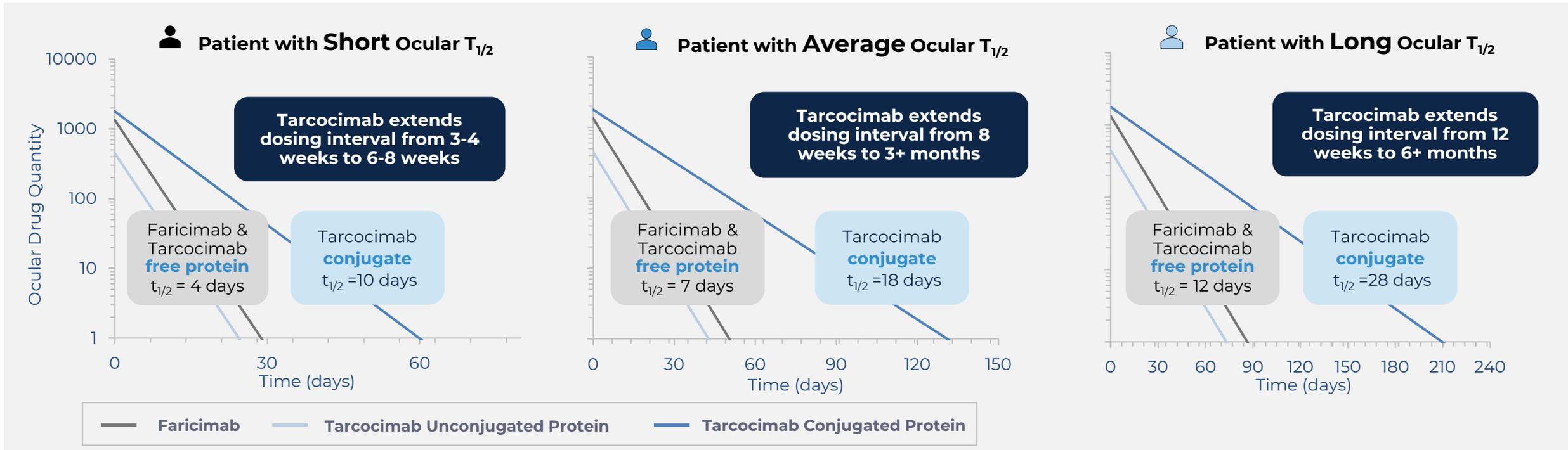
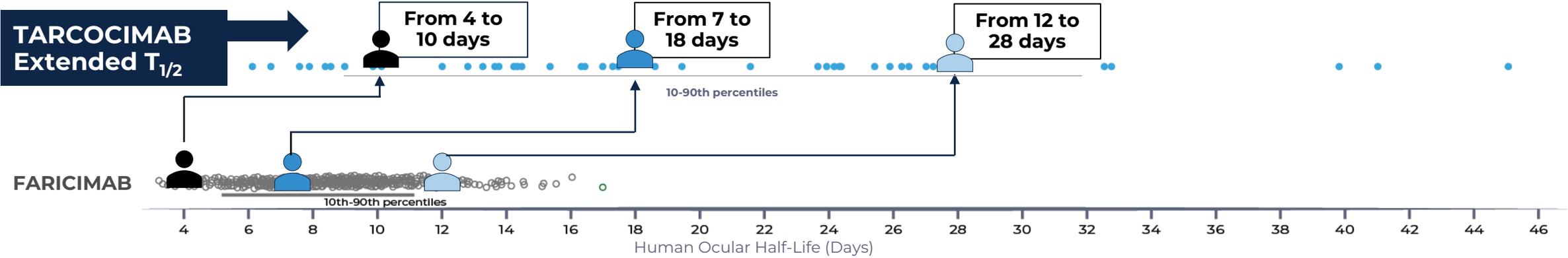
- The 20% unconjugated protein (1 mg) is designed to improve the immediacy of the drying effect during the loading dose phase, "closing the gap"

Maintain 6-month predominant durability with the 80% conjugated protein

- As seen in tarcocimab pivotal studies to date

*Approximate CST changes are plotted based on pivotal clinical studies of approved intravitreal biologics. CST changes for tarcocimab enhanced formulation is a projection

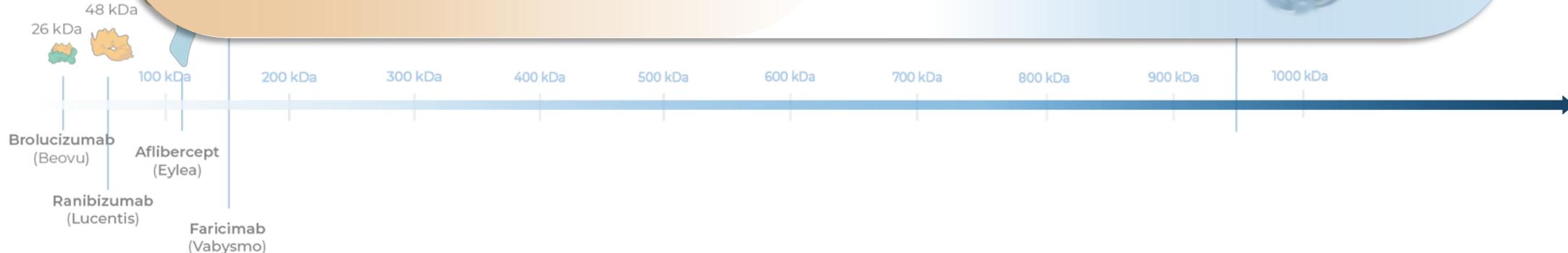
Modeling suggests tarcocimab may provide a strong immediacy of efficacy while meaningfully extending dosing intervals for all patient types



Tarcocimab and KSI-501 bring the best of both worlds – high immediacy and high durability – at the same time and in the same biologic therapy

Immediacy: Unconjugated Protein

High Durability: ABC (Conjugated) Protein



ABC® Enabled Mainstay Biologics Target Product Profiles

TARCOCIMAB

- Non-inferior Efficacy
- Strong Immediacy
- Industry-leading Durability

KSI-501

- Better Efficacy
- Strong Immediacy
- Industry-leading Durability

Ongoing and planned studies may support a tarcocimab BLA in 2026 and a potential KSI-501 BLA in 2027, while enabling complementary & potentially differentiated commercial profiles

	Indication	Phase 3 Study		Primary Endpoint	6-Month Durability	Results / Progress
Tarcocimab	Retinal Vein Occlusion	BEACON	Completed	✓	✓	<ul style="list-style-type: none"> Doubled treatment interval (Q8W) at PE (month 6) and ~50% of tarcocimab treated patients on 6-month dosing at Year 1
	Diabetic Retinopathy	GLOW1		✓	✓	<ul style="list-style-type: none"> 100% of patients on 6-month dosing at Year 1
	Wet AMD	DAYLIGHT		✓	Not Applicable	<ul style="list-style-type: none"> Monthly study of tarcocimab demonstrated favorable safety and non-inferior efficacy at Year 1
	Diabetic Retinopathy	GLOW2	Ongoing	Superiority	✓	<ul style="list-style-type: none"> Topline data on track for 1Q 2026 Design mirrors successful GLOW1 study
	Wet AMD	DAYBREAK		Non-Inferiority	✓	<ul style="list-style-type: none"> Enrollment complete Topline data expected 3Q 2026
KSI-501	Wet AMD	DAYBREAK	Ongoing	Non-Inferiority	Not Applicable	<ul style="list-style-type: none"> Enrollment complete Topline data expected 3Q 2026
	Not Specified	2nd Pivotal	Planned	TBD	TBD	<ul style="list-style-type: none"> In planning Planned start in 2Q 2026

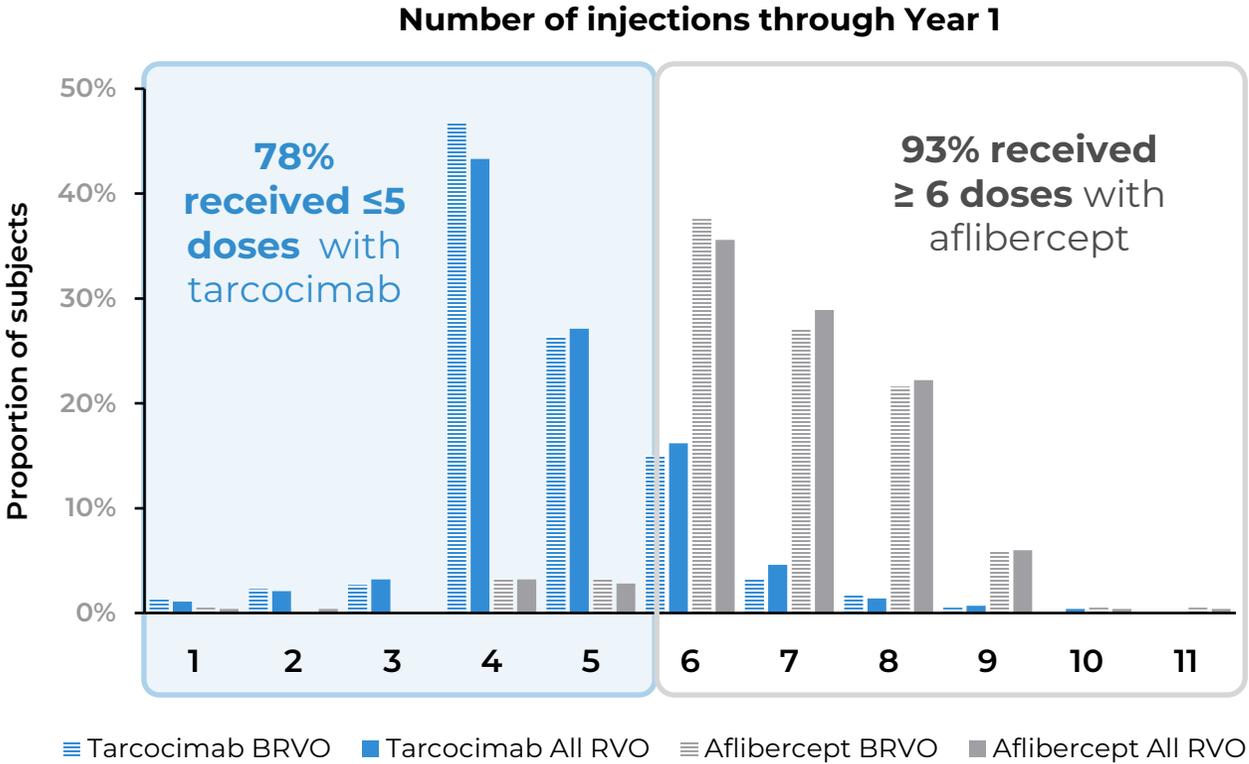
Tarcocimab demonstrated high efficacy and industry-leading durability in Retinal Vein Occlusion in the Phase 3 BEACON study

RVO
BEACON

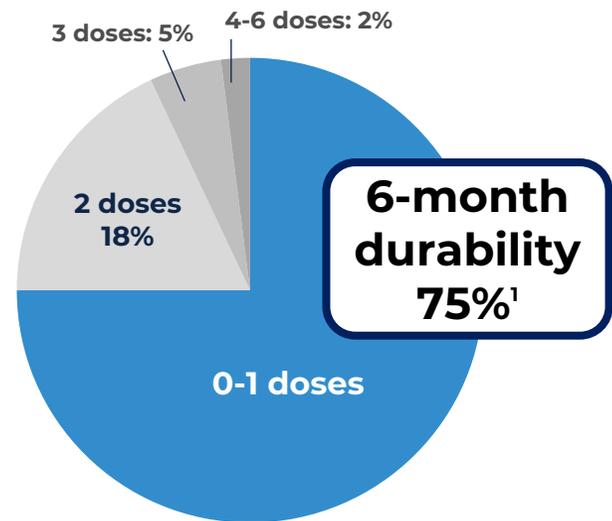
Primary endpoint ✓

6-month durability ✓

Quadrant of core unmet need



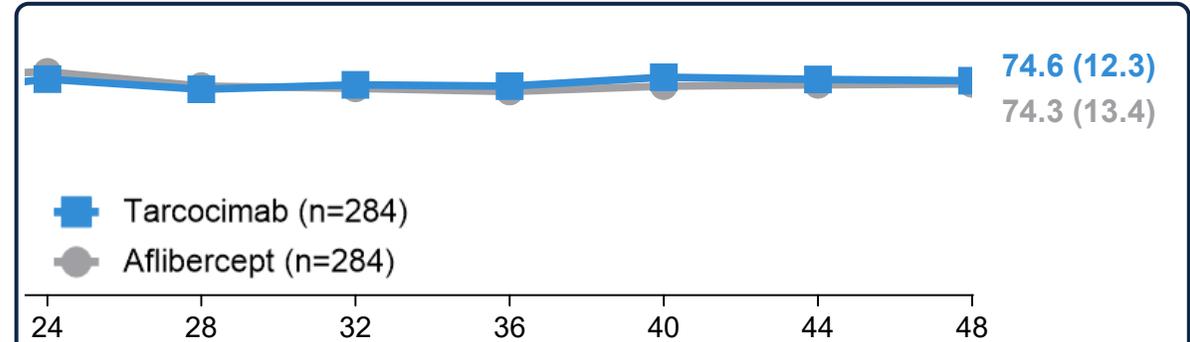
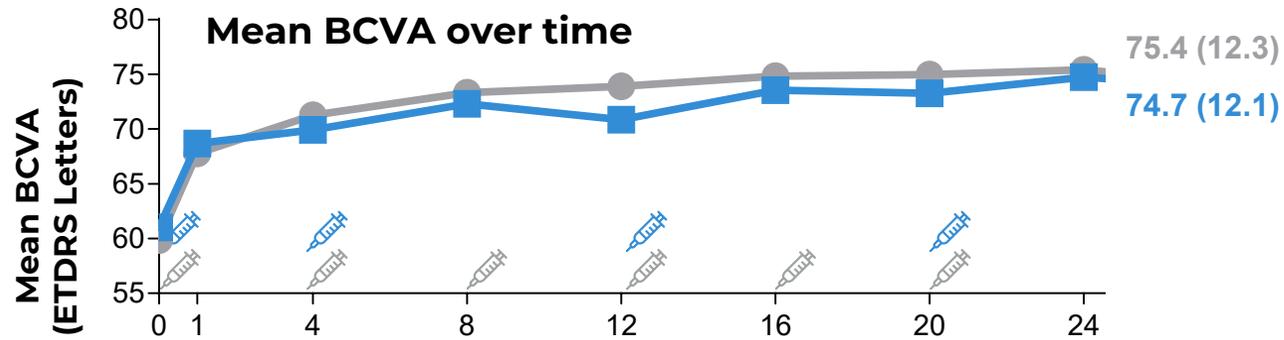
Tarcocimab: Number of doses in the second 6 months of Year 1



RVO is a ~\$3 billion market. Gen 1.5 agents have failed to address the key unmet need for high efficacy with better durability. Tarcocimab delivered 6-month durability in 75% of patients in BEACON and is uniquely poised to be a valuable medicine in this market

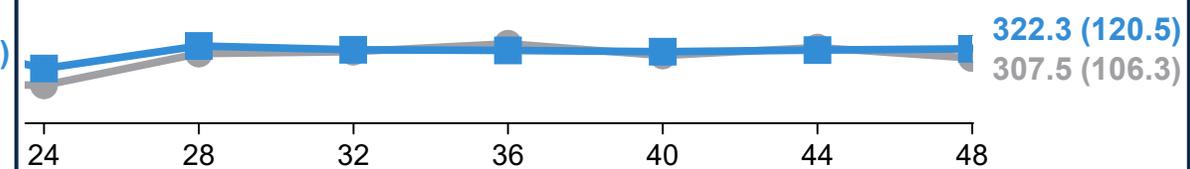
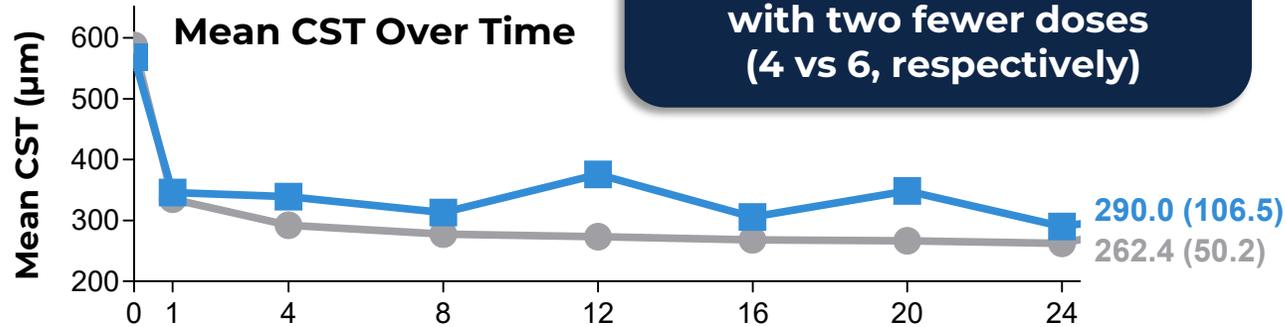
RVO: retinal vein occlusion; BRVO: branched retinal vein occlusion;
 1. Durability interval calculated based on patients that received no injections (46%) or 1 injection (29%) over the second 6 months of Year 1.
 2. RVO market size from imarc: RVO Market Size, Epidemiology, In-Market drug sales, pipeline therapies, and Regional Outlook

Tarcocimab achieved comparable visual and anatomical outcomes in all RVO patients, irrespective of the treatment paradigm used



Tarcocimab achieved similar visual and anatomical gains with two fewer doses (4 vs 6, respectively)

In 75% of patients, tarcocimab delivered 6-month durability with similar visual and anatomical gains from Week 24 to 48



Fixed Dosing (doubling of treatment interval)

Head-to-head Individualized Dosing of tarcocimab versus aflibercept

Mean observed data; Week 24 and 48 datapoints are Mean (Standard Deviation). Results for BCVA are based on a mixed model repeated measures (MMRM) analysis, with the change from baseline value as the dependent variable; treatment, visit (Week 1 through Week 48), and treatment by visit interaction as fixed effects; randomization stratification variables [baseline BCVA, disease duration, RVO type) and geographical location], as well as continuous covariates of baseline BCVA value and baseline OCT CMM value, as fixed effects; and subject as a random effect. a. Nominal p-value. Non-inferiority margin = 4.5 ETDRS letters.

	LSM change from BL BCVA at Week 48 (MMRM)	95% CI for LSM difference	P-value for non-inferiority ^a
Tarcocimab	11.7	-3.11, 0.94	p = 0.001
Aflibercept	12.8		

Tarcocimab demonstrated high efficacy and industry-leading durability in diabetic retinopathy in the Phase 3 GLOW1 study

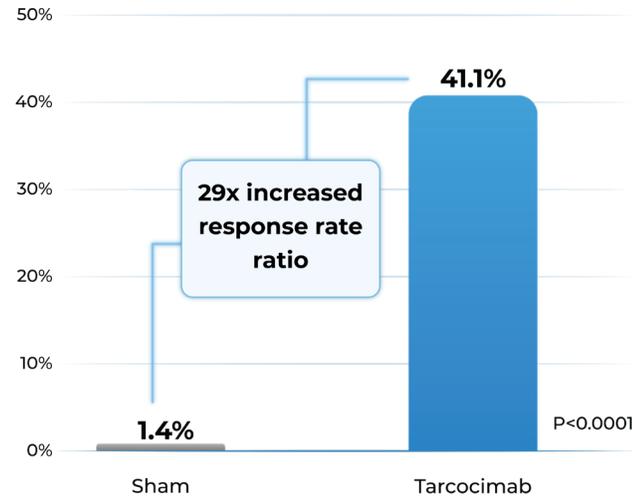
DR
GLOW1

Primary endpoint (superiority) ✓

6-month dosing in all patients ✓

Quadrant of core unmet need

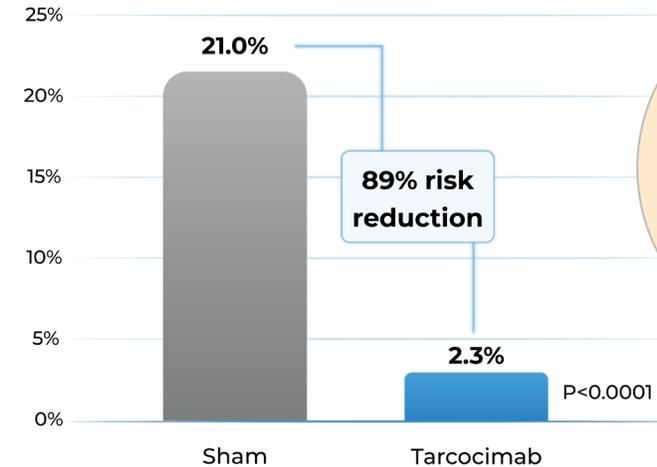
Proportion of patients with ≥ 2 -Step improvement in DRSS from Baseline to Week 48



Primary Endpoint

Tarcocimab **demonstrated superiority** in ≥ 2 -step and ≥ 3 -step improvement in DRSS

Proportion of patients developing any sight-threatening complication from Baseline to Week 48



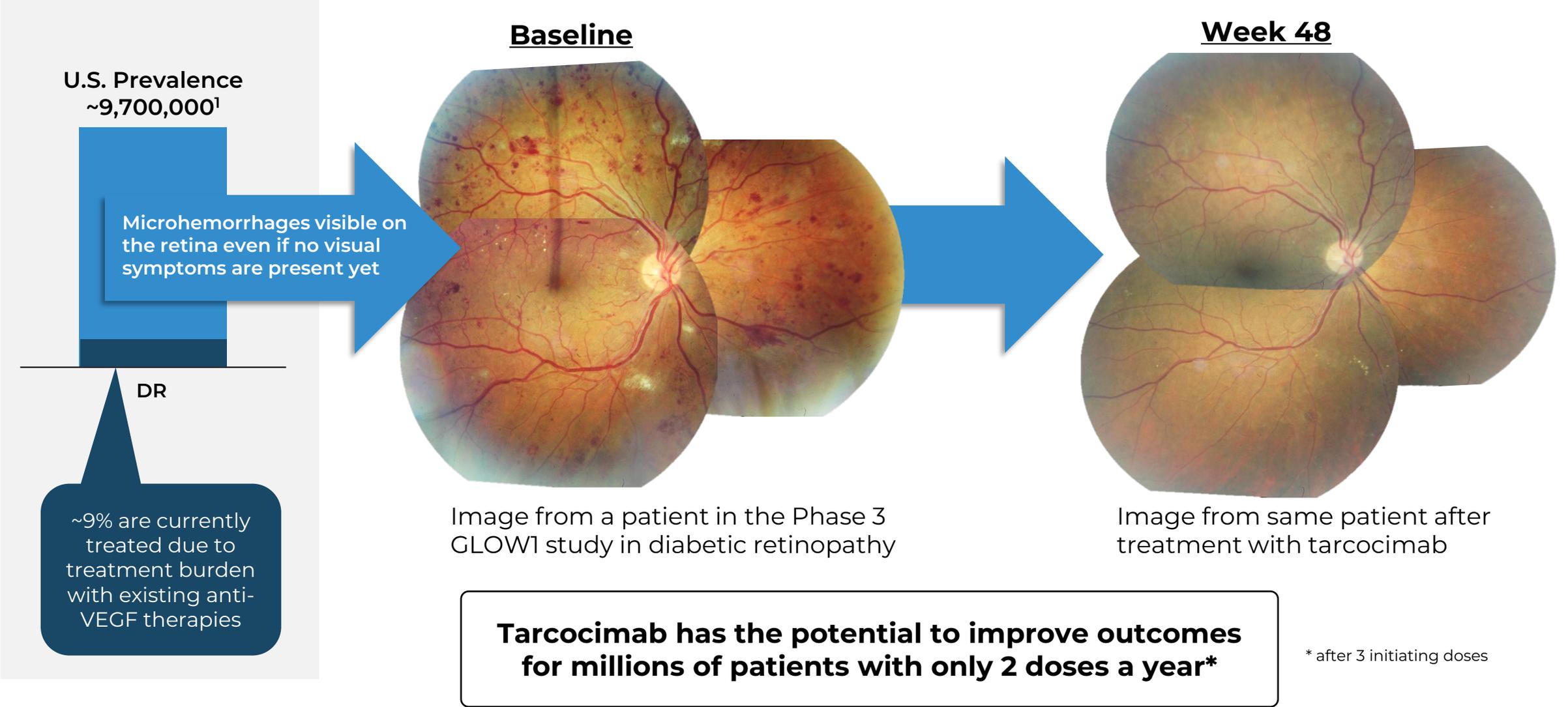
Key Secondary Endpoint

~90% risk reduction for developing a pre-specified sight-threatening complication with tarcocimab

100% of patients on 6-month dosing at Year 1

Approximately 855,000 patients with DR were treated with anti-VEGF agents in the US in 2025, indicating a higher use of anti-VEGF agents than previously estimated¹

The GLOW1 study shows that tarcocimab opens the door to earlier treatment with only 2 doses a year – a transformative potential for millions of patients



In the ongoing Phase 3 GLOW2 study in DR, what are the potential implications and upsides for tarcocimab?

DR
GLOW2

Superiority
study

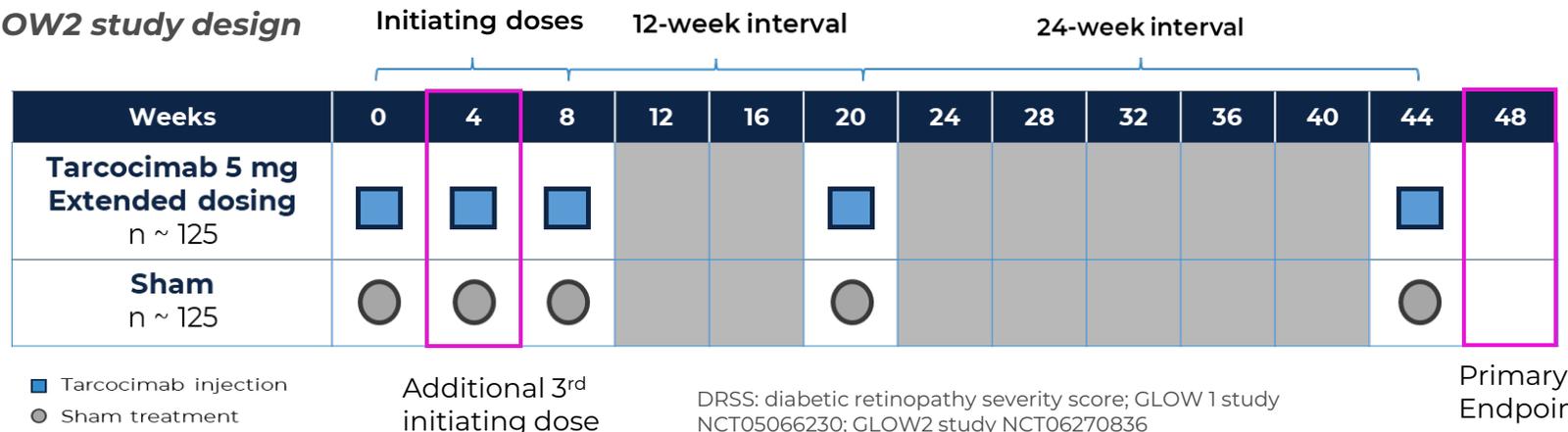
6-month
dosing in all
patients

Topline data:
Q1 2026

Quadrant
of core
unmet
need

- Topline data expected **Q1 2026**. Key implications of GLOW2:
 - **1st Phase 3 readout using the commercial formulation for tarcocimab**, providing important safety data
 - **2nd pivotal superiority study with 100% of patients on every 6-month (twice a year) dosing** versus sham – evaluating ≥ 2 -step improvement in DRSS (primary endpoint) and risk reduction in developing pre-specified sight-threatening complications (key secondary endpoint)
 - **Features a similar study design as the successful GLOW1 study**, with the benefit of an additional 3rd initiating dose
- If successful, **tarcocimab will have a BLA-ready profile** for a filing in Diabetic Retinopathy (based on GLOW1 and GLOW2 studies) and in Retinal Vein Occlusion (based on BEACON study)

GLOW2 study design



The ongoing Phase 3 DAYBREAK study in wet AMD explores in a definitive manner **immediacy through the loading phase, real-world durability**, and for KSI-501, the potential for **better efficacy**

Wet AMD
DAYBREAK

Noninferiority
study

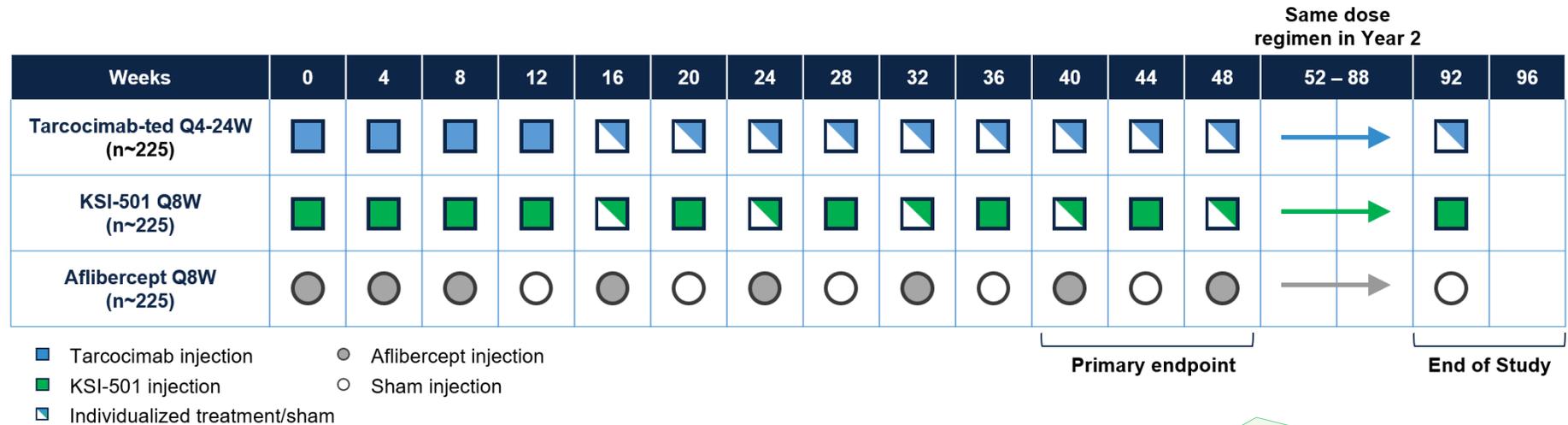
6-month
durability
(tarcocimab)

Better efficacy
(KSI-501)

Topline data:
Q3 2026

Quadrant
of core
unmet
need

- Registrational study for both tarcocimab and KSI-501. Explores in a definitive manner immediacy through the loading phase, real-world durability, and for KSI-501, the potential for better efficacy



Tarcocimab objective

Assess 6-month durability potential with individualized Q4W to Q24W dosing

KSI-501 objective

Explore the efficacy potential of bispecific IL-6 and VEGF inhibition in fixed Q8W dosing with additional individualized monthly dosing

- **Uses an AI-based tool to precisely measure fluid in the eye so treatment is optimized per patient**
 - **In high-need patients** treats until dry, enables monthly dosing and detects disease reactivation earlier
 - **In long-durability patients** allows patients without active disease to safely go to every 6-month dosing

In DAYBREAK for wet AMD, what are the potential implications and upsides for **tarcocimab**?

Wet AMD
DAYBREAK

Noninferiority
study

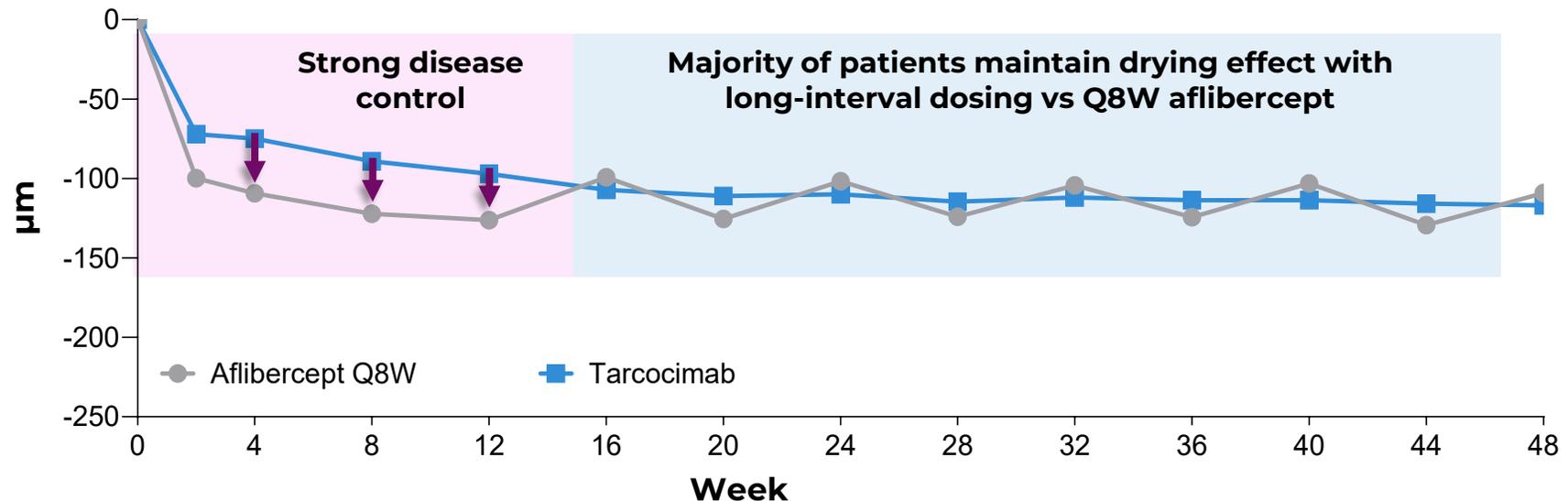
6-month
durability

Topline data:
Q3 2026

Quadrant
of core
unmet
need

- Topline data expected **Q3 2026**
- If successful, DAYBREAK will demonstrate tarcocimab's (commercial formulation) ability to provide:
 - **Strong and immediate disease control in the loading phase** (closing the 'immediacy gap'),
 - **Non-inferior vision gains to aflibercept Q8W**, and
 - **Long-interval dosing** with a flexible 1-month through 6-month label

Illustrative only: Highlighting the potential for the tarcocimab commercial formulation to demonstrate **high immediacy (in the loading phase) and high durability (in the maintenance phase)**



In DAYBREAK for wet AMD, what are the potential implications and upsides for KSI-501?

Wet AMD
DAYBREAK

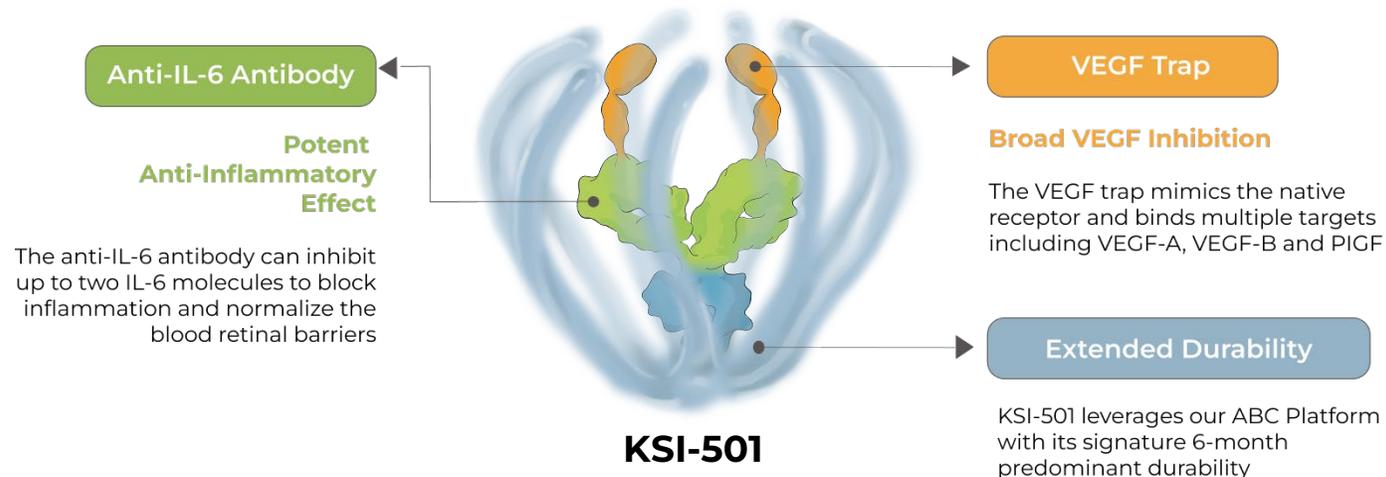
Noninferiority
study

Better
efficacy

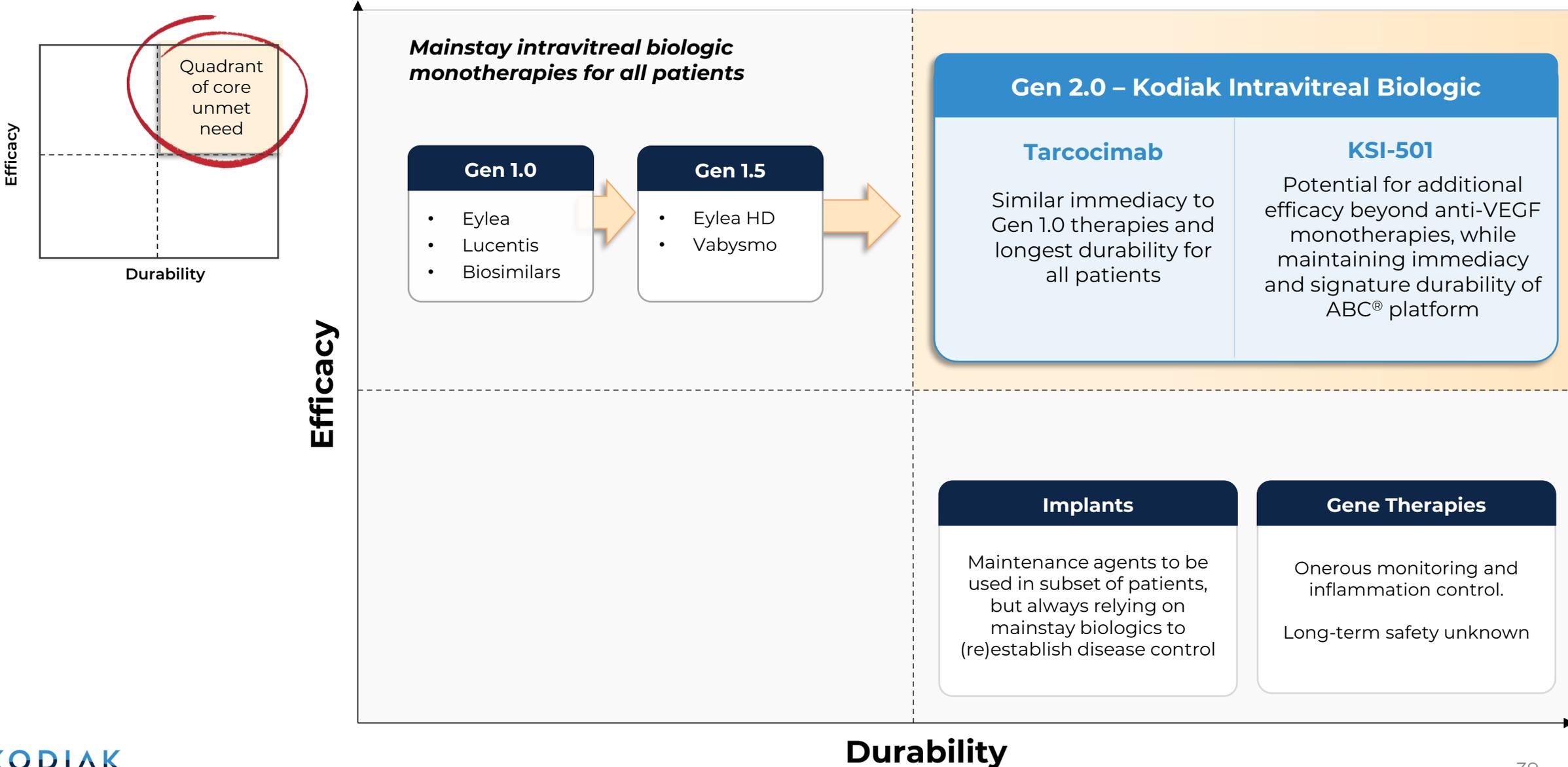
Topline data:
Q3 2026

Quadrant
of core
unmet
need

- **Q3 2026 – Year 1 topline data** evaluating Q4W/Q8W KSI-501 in wet AMD
- **Potential efficacy upside**
 - **Better vision gains** than anti-VEGF monotherapy aflibercept with good OCT control
 - Note that bispecific inhibition of IL-6 and VEGF in the APEX Phase 1b study has shown that KSI-101, a bispecific anti-IL-6, VEGF trap, provides rapid and meaningful vision gains and rapid and powerful anatomical improvement in very sick patients
 - **Strong and immediate disease control**
 - Immediate disease control in the loading phase (closing the ‘immediacy gap’*),



Revisiting the core unmet need: based on a science of **high immediacy** and **high durability**, tarcocimab and KSI-501 are poised to fill the 'golden quadrant'



Significant investment in commercial manufacturing has positioned Kodiak well for potential launch of multiple ABC[®] products into large and growing markets

News Release

Lonza
KODIAK

Grand Opening of Kodiak Sciences' Purpose-Built Bioconjugation Facility to Support Potential Commercial Manufacture of KSI-301, an Antibody Biopolymer Conjugate for Retinal Diseases

- Purpose-built bioconjugation facility to support the potential commercial manufacture of high-prevalence retinal disease therapies
- The opening ceremony took place at the facility in March 2022

Basel, Switzerland and Palo Alto (CA) Kodiak Sciences, a biopharmaceutical company committed to developing transformative therapeutics to treat rare diseases, announced the opening of a new, custom-built commercial manufacturing complex in Visp (CH).



Ursus, a premium commercial manufacturing facility

- A commercial scale facility dedicated to the manufacture of Kodiak's ABC medicines
- Custom designed for large scale **premium manufacturing of complex antibody conjugate biotherapies**
- Mechanical completion in 1H2022; commissioned as a cGMP facility for commercial supply in Jan 2023
- Successful cGMP manufacture and release commercial scale tarcocimab commercial formulation in Nov 2023
- **BLA-facing commercial-scale validation batches were manufactured and released in 2025 for antibody, biopolymer, and bioconjugate**

Macular
Edema Secondary
to Inflammation
(MESI)

KSI-101

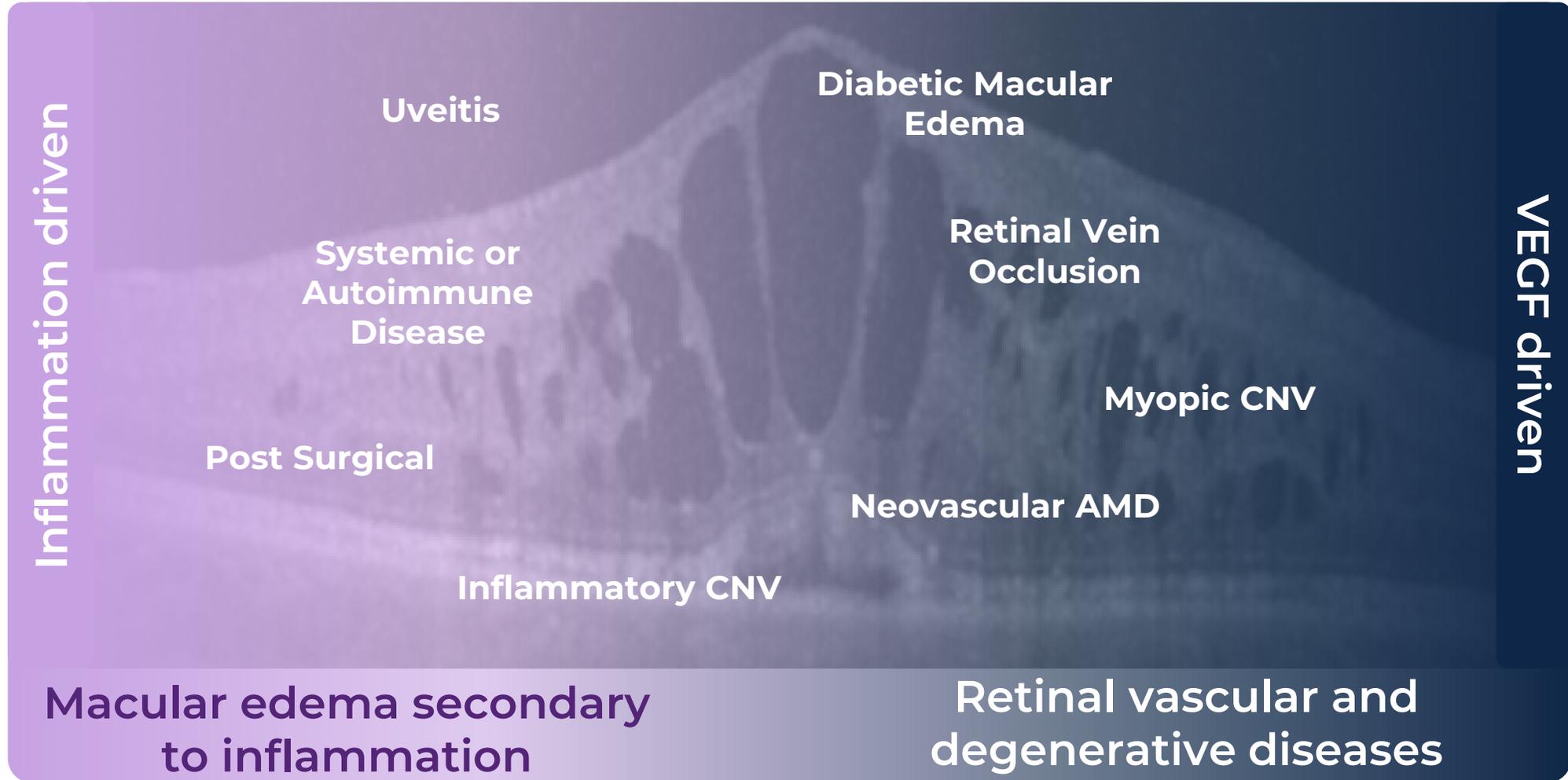
A grayscale, high-magnification microscopic image of retinal tissue, showing a dense field of cells with distinct nuclei and cytoplasmic structures, likely representing the macula.

Macular Edema Secondary
to Inflammation

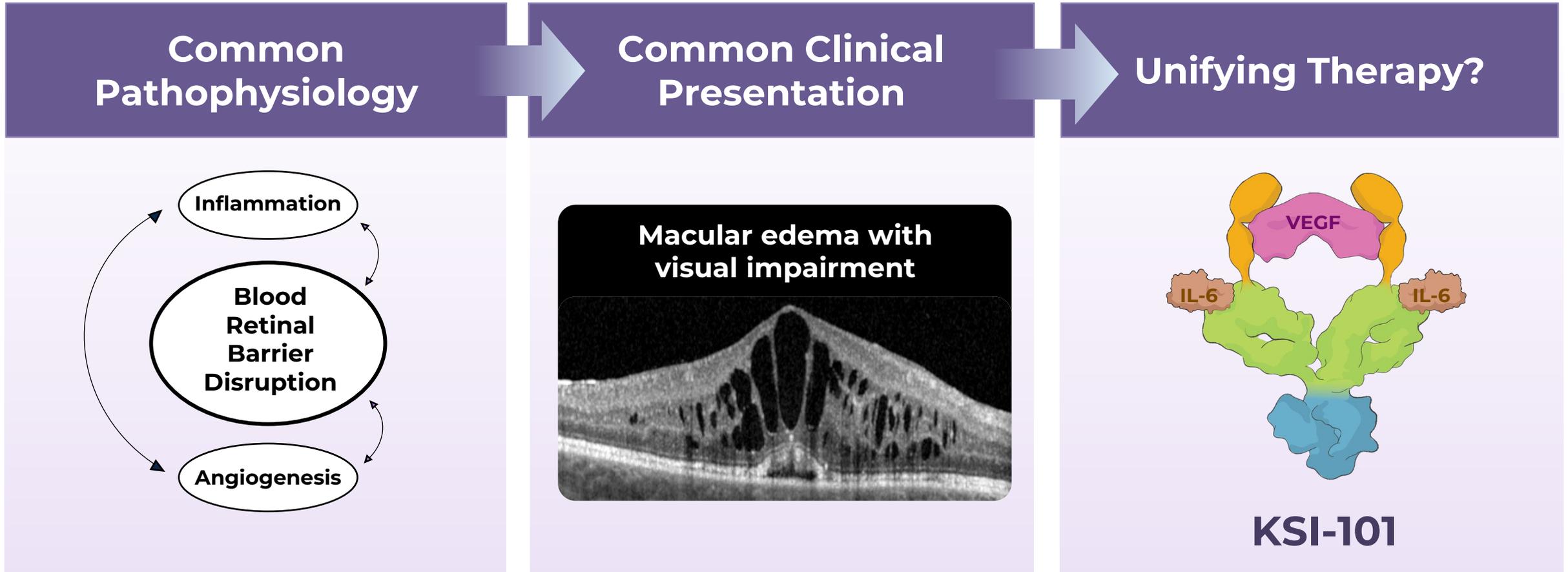
What is MESI?

Macular edema, the common clinical presentation of a wide spectrum of diseases, can be caused by inflammation and/or by VEGF over-expression

Macular Edema Spectrum of Diseases

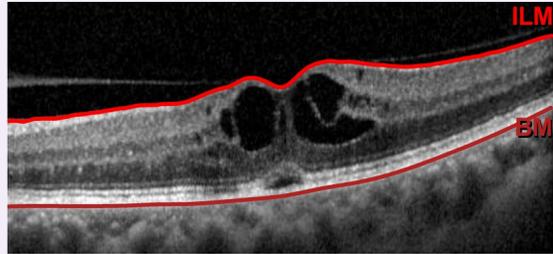


What is macular edema secondary to inflammation (MESI)?

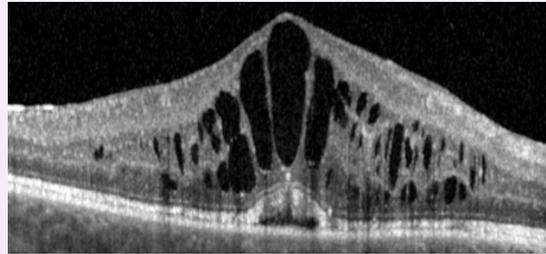


MESI is a heterogenous group of diseases that clinically present with macular edema and visual impairment, which are caused by a common pathophysiology: inflammation and blood retinal barrier disruption

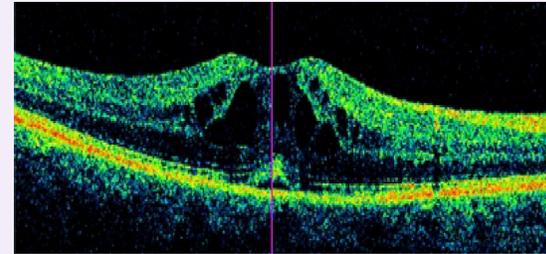
MESI comprises a heterogenous group of diseases with a common, readily identifiable clinical presentation: macular edema with visual impairment



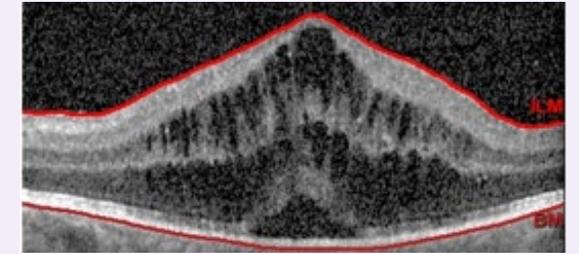
Anterior



Intermediate



Posterior



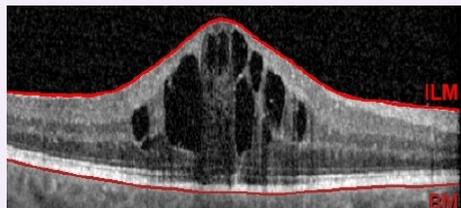
Panuveitis

Location of Inflammation

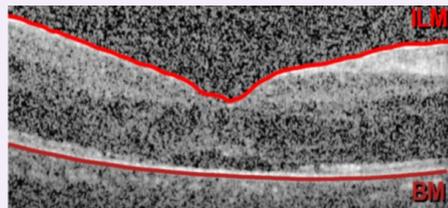
Irrespective of the anatomical location of the inflammation or the specific etiology, the clinical presentation is the same: macular edema

Specific Etiology

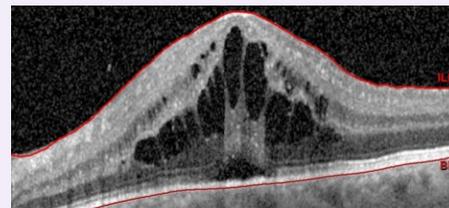
Idiopathic



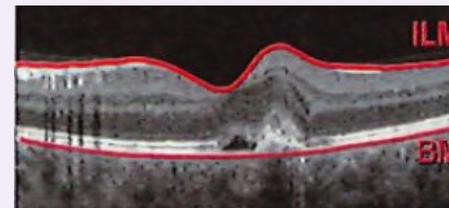
Juvenile Idiopathic Arthritis



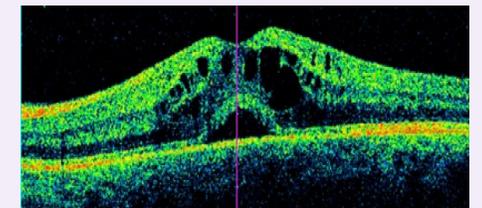
Focal Chorioretinal inflammation



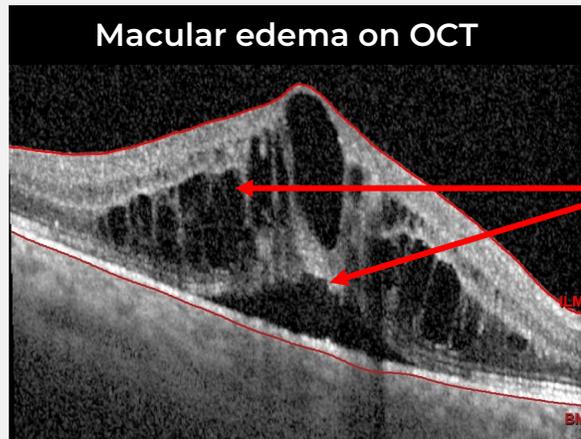
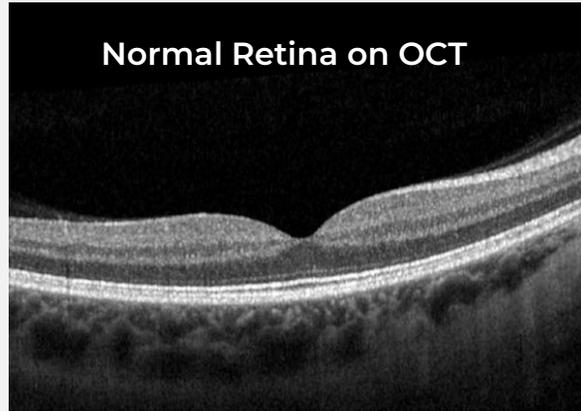
Punctate Inner Choroidopathy



Post-Operative Macular Edema



Macular edema is the leading cause of vision loss among patients with ocular inflammation



Fluid that leaked into the retina causing macular edema

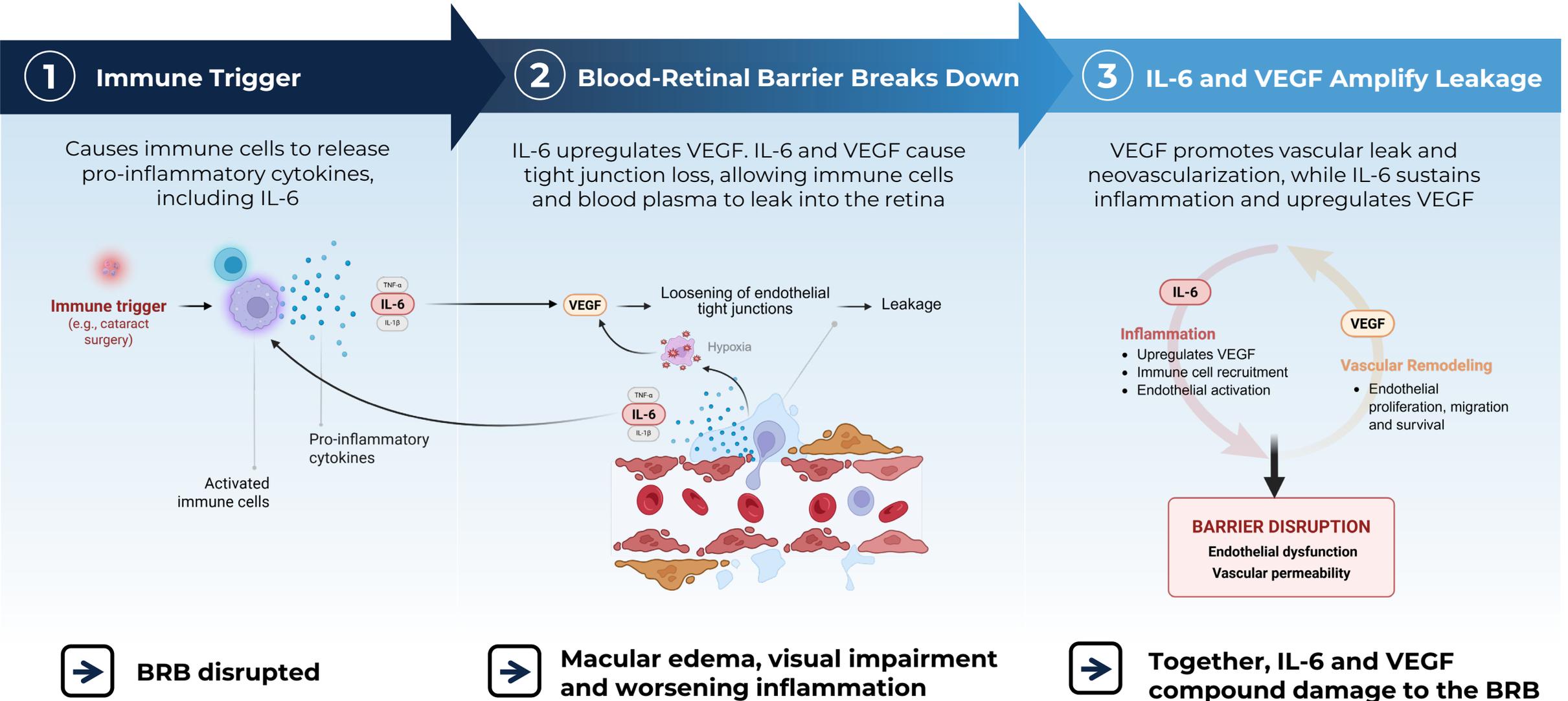
- Noninfectious MESI represents a set of serious ocular inflammatory conditions that cause significant vision loss.
- Ocular inflammation is the **4th leading cause of vision loss among working aged adults in the developed world¹**
- Approximately **1/3 of patients with ocular inflammation develop macular edema** in the U.S.²
- Symptoms at diagnosis typically include distorted central vision, vitreous floaters, reduced visual acuity, and decreased color and contrast sensitivity
- MESI leads to photoreceptor damage and **can result in permanent loss of visual acuity**

A grayscale, high-magnification microscopic image of retinal tissue, showing a dense field of cells with distinct nuclei and cytoplasmic structures, likely representing the macula.

Macular Edema Secondary
to Inflammation

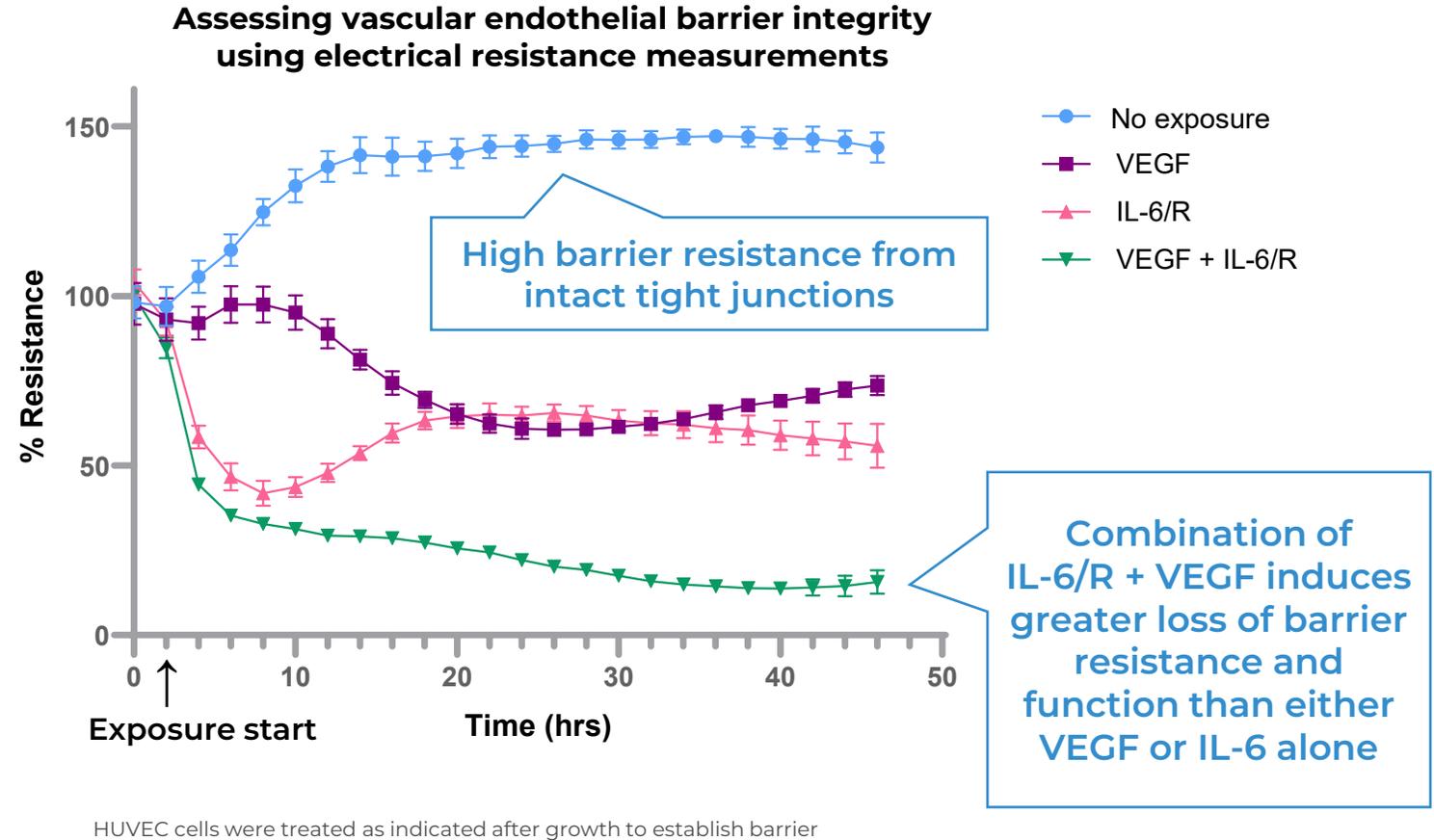
What causes MESI?

Dysregulation of the immune system causes a series of insults to the blood-ocular barrier, leading to breakdown of the barrier and release of inflammatory mediators



IL-6 and VEGF each disrupt the blood-retinal barrier independently. When combined, they cause an even greater loss of barrier integrity

- Ocular inflammation damages tight junctions between endothelial cells, compromising the integrity of the blood retina barrier. This increases vascular permeability
- The integrity of the blood-retinal barrier can be measured by barrier resistance
- Exposure to both IL-6/R and VEGF **additively induces greater loss in barrier function**, as measured by decreased barrier resistance, than either IL-6 or VEGF alone, in human umbilical vein endothelial cells (HUVEC)



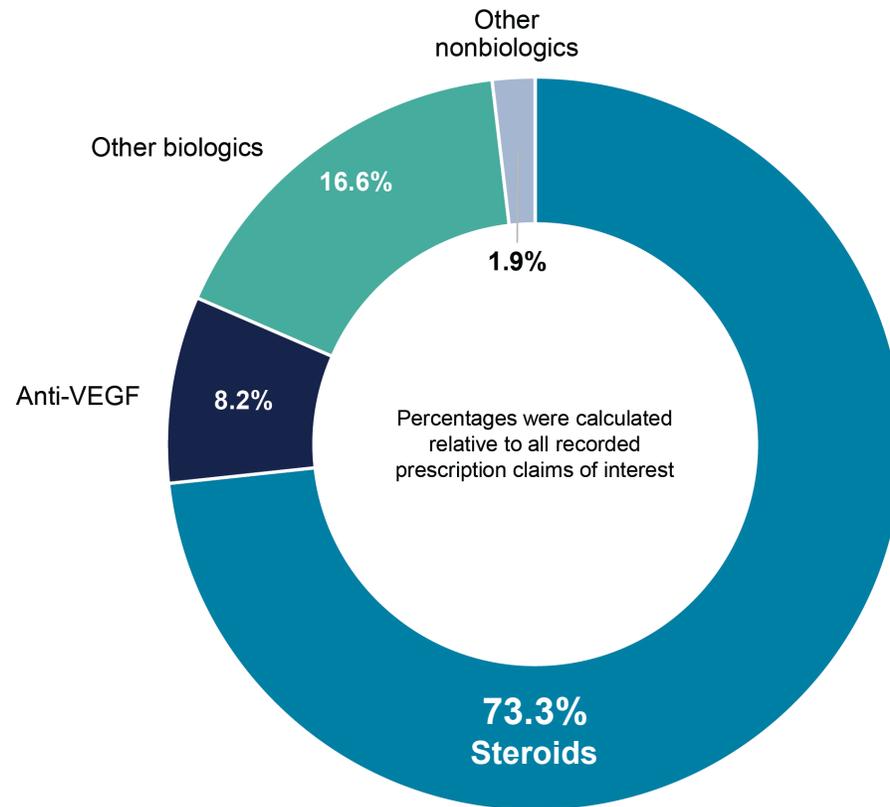
A grayscale, high-magnification microscopic image of retinal tissue, showing a dense layer of cells with distinct nuclei and cytoplasmic structures, likely representing the macula.

Macular Edema Secondary to Inflammation

**What is the
unmet need?**

Corticosteroids are used most often for MESI, but carry significant safety risks and efficacy can be limited

Most frequently prescribed medications in patients with macular edema due to noninfectious uveitis, across any line of therapy¹



- Steroids were the most common medication class and remain the standard of care¹
- XIPERE[®] (suprachoroidal triamcinolone acetonide) is the only approved local ocular treatment in the U.S.
- **Approximately 30-40% of patients do not fully respond to intraocular steroids^{2,3}**
- Intraocular steroids are avoided in the pediatric population and **used with caution in adults due to high risk of permanent glaucoma damage and cataract formation**

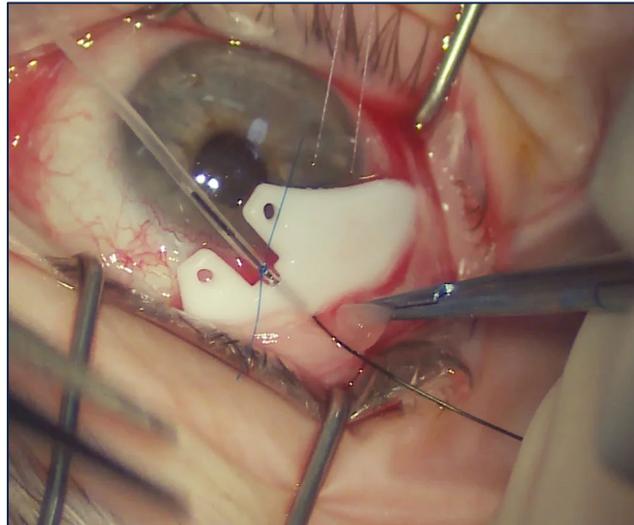
Adverse effects from today's treatments for MESI often require multiple surgical interventions, but surgeries are complicated in MESI patients and require complete control of inflammation before, during and after each procedure

Cataract repair in MESI patients is complex and can be contraindicated, leading to permanent vision loss



- Even with good inflammation control, surgeries can trigger intractable inflammation, often leaving the eyes with no intraocular lens implant and/or suboptimal results ((synechiae/scarring)

Visual damage from glaucoma can be irreversible and can lead to legal blindness



- Concerns about permanent IOP elevation limit steroid use and dose
- Supplemental topical drops to control elevated IOP are often insufficient, requiring invasive glaucoma surgeries
- **Once the optic nerve is damaged due to glaucoma (high eye pressure), the visual loss can be irreversible**

RETISERT® (fluocinolone acetonide intravitreal implant) label:

- **60% of patients will require chronic IOP lowering medications** to control intraocular pressure
- **37% will require filtering procedures** to control intraocular pressure
- Within an average post-implantation period of approximately 2 years, **nearly all phakic eyes are expected to develop cataracts and require cataract surgery**

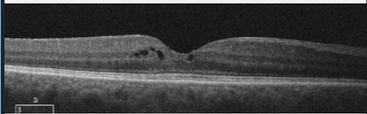
RETISERT® (fluocinolone acetonide intravitreal implant)
Prescribing Information.

Patient journey: steroids induced glaucoma with recurrent MESI over the course of approximately 2 years

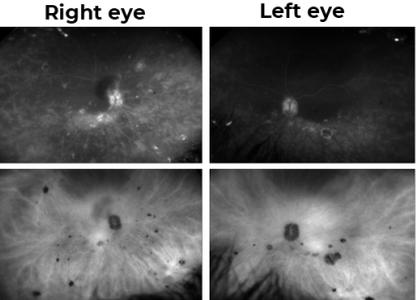
 Patient presented to uveitis specialist

1

Patient presented in August 2023 with a 12-year history of **panuveitis of both eyes** and **multiple failed treatments**



Minimum macular edema initially



- Failed treatments**
- Weekly adalimumab
 - Methotrexate
 - Cellcept
 - Azathioprine
 - Oral steroids
 - Difluprednate eyedrops

2

Developed worsening of chorioretinal lesions and **macular edema**

Intraocular pressure (IOP) spikes to 40s with any steroids (topical eyedrops and intravitreal)

Referred to a glaucoma specialist

 **Surgery #1:** A combined cataract surgery and glaucoma surgery (OMNI canaloplasty) of the right eye in Jan 2024

IOP continue to rise despite surgery

3

Worsening **macular edema** with taper of **difluprednate eyedrops**

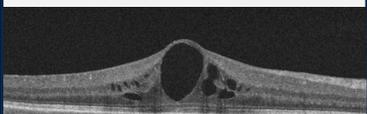
Referred to a rheumatologist

 For alternate **immunomodulatory therapy (IMT)**

Attempted authorization for tocilizumab but insurance denied twice. Unable to get authorization for infliximab either

4

Trial of bromfenac drops with initial improvement then worsening of **macular edema**



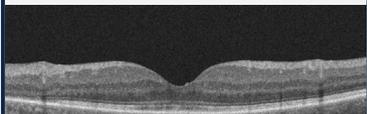
Place back on difluprednate drops QID with spike of IOP to 38

Referred to a glaucoma specialist

 **Surgery #2:** Glaucoma tube shunt surgery of the right eye in May 2025

IOP now stable

IOP stable and macular edema now resolved after **fluocinolone intravitreal injections**



Will evaluate further need for IMT next visit

There is a core unmet need in MESI for a potent, high-strength, locally administered and safe therapy

Illustrative



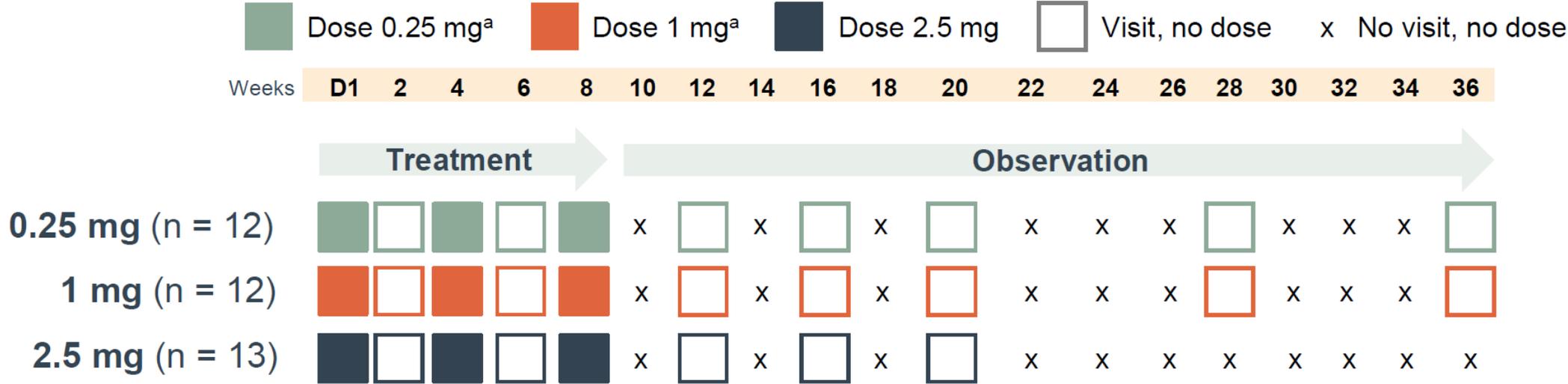
A large, light-colored, semi-transparent image of a microscopic view of cells, possibly retinal cells, occupies the left side of the slide. The cells are arranged in a somewhat regular pattern, with some showing distinct nuclei and cytoplasm.

IL-6 Inhibition in Inflammatory Macular Edema

**What have we
learned?**

DOVETAIL Study – intravitreal anti-IL-6 monotherapy (Roche, vamikibart) has been studied as a potential therapy for patients with inflammatory macular edema

- DOVETAIL is a phase 1, multipart, multicenter, nonrandomized, open-label, multiple ascending dose study of intravitreal vamikibart



Primary objective: Safety and tolerability

Secondary objectives: PK, ADA formation, efficacy



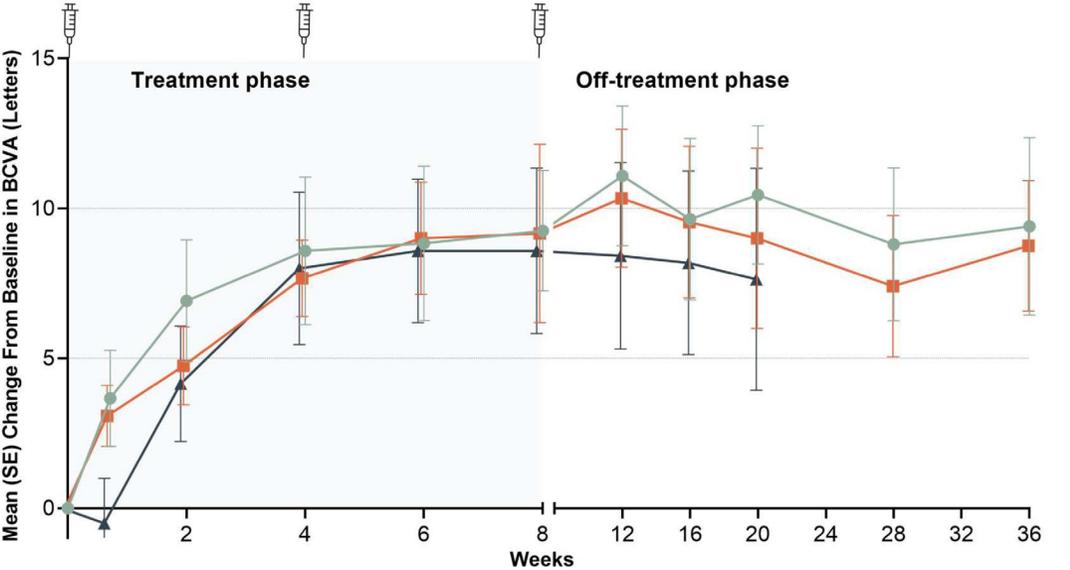
Age ≥ 18 years

Noninfectious uveitis and concurrent macular edema (CST ≥ 325 μm)

Vamikibart (Roche, anti-IL-6) has shown that anti-IL-6 monotherapy can provide visual and anatomical improvement in patients with inflammatory macular edema

DOVETAIL

Change from Baseline in BCVA

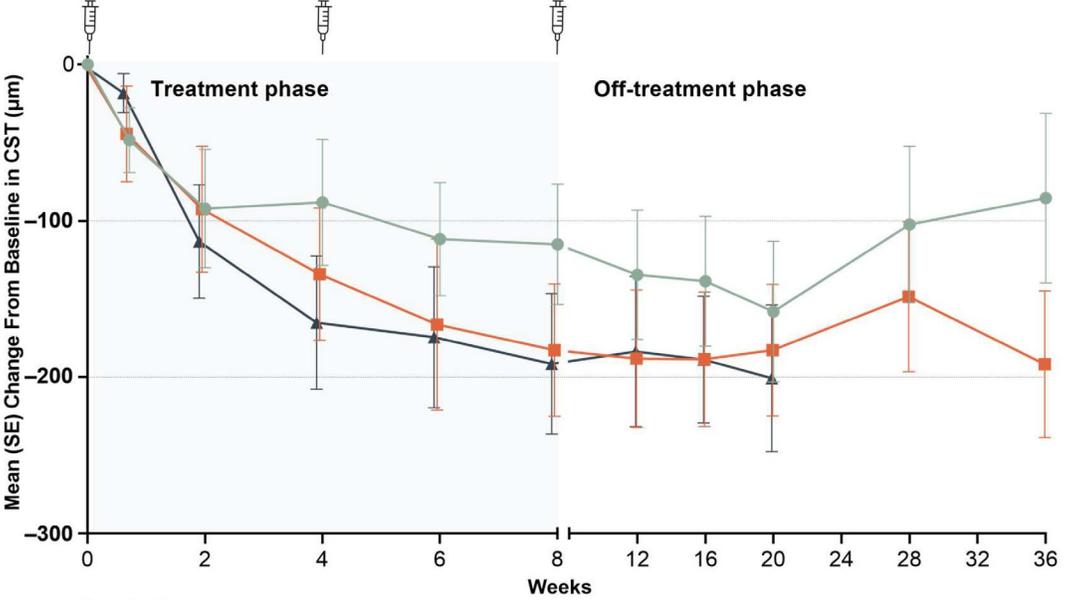


Sample size	0	2	4	6	8	12	16	20	28	36
0.25 mg (n)	12	12	12	12	12	12	11	11	10	10
1 mg (n)	12	12	12	12	11	12	11	11	10	8
2.5 mg (n)	13	12	13	12	12	12	11	11	10	8

2.5 mg cohort last F-up is W20

DOVETAIL

Change from Baseline in OCT CST



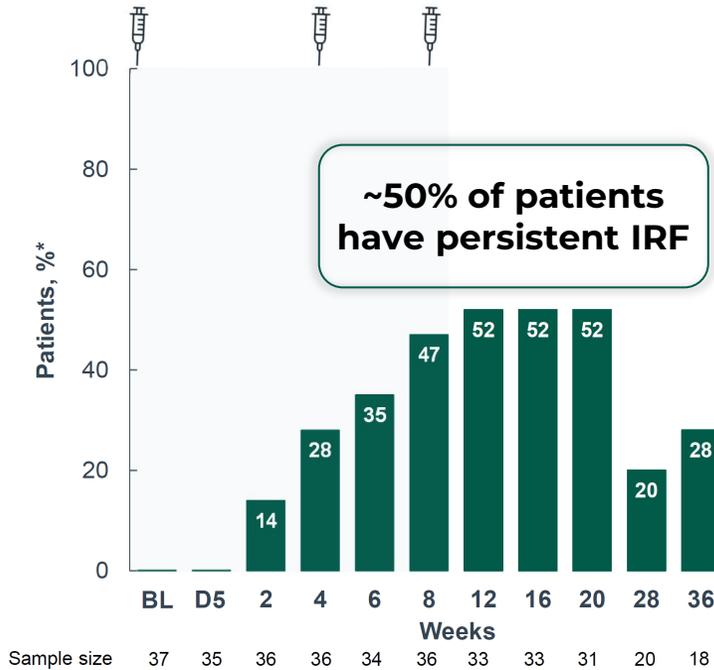
Sample size	0	2	4	6	8	12	16	20	28	36
0.25 mg (n)	12	12	12	11	12	12	11	11	10	10
1 mg (n)	12	12	12	12	11	12	12	11	11	10
2.5 mg (n)	13	11	12	12	12	12	11	11	11	8

2.5 mg cohort last F-up is W20

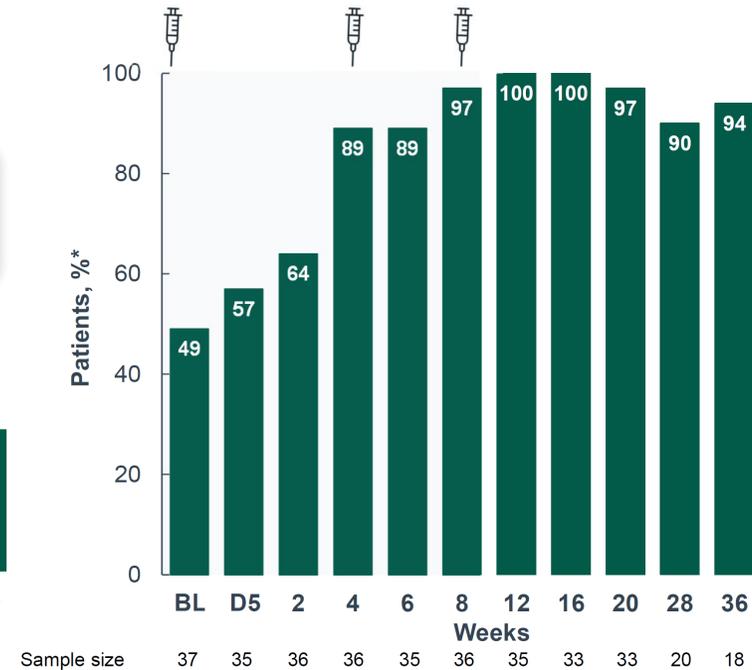
A clear dose response is seen with IL-6 monotherapy in patients with inflammatory macular edema

While intravitreal IL-6 monotherapy is helpful, 50% of patients have persistent IRF¹, similar to the overall failure rate of systemic adalimumab, leaving room for a more potent and/or broader spectrum biologic therapy

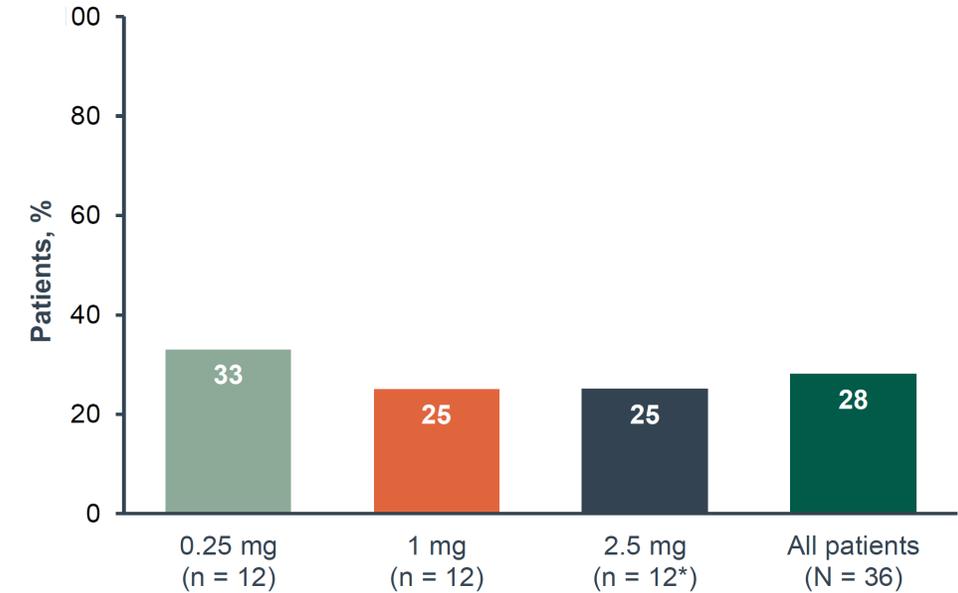
DOVETAIL
Absence of IRF



DOVETAIL
Absence of SRF



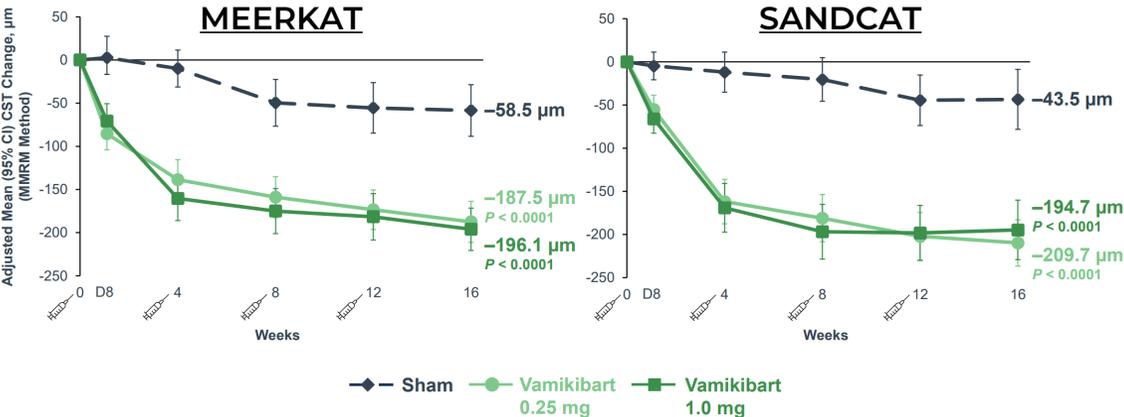
DOVETAIL
≥15-letter gainers at Week 12



Persistent intraretinal fluid (IRF) is known to cause permanent negative effects on visual function

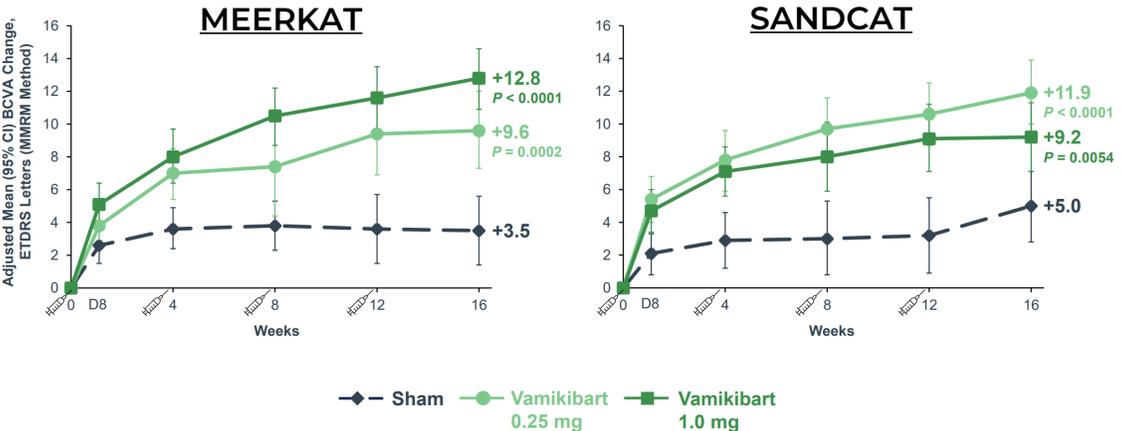
The IL-6 pathway has been further validated in recent pivotal studies as a key target for inhibition in inflammatory macular edema

Vamikibart Phase 3 Program
Change from Baseline in OCT CST



Clear anatomical improvement is seen with anti-IL-6 monotherapy

Vamikibart Phase 3 Program
Change from Baseline in BCVA



Visual acuity gains correlating with the anatomical improvement are observed

No on-target adverse effects were identified in pivotal studies, derisking intravitreal IL-6 inhibition in inflammatory macular edema

Vamikibart Phase 3 Program
Summary of Ocular (Study Eye) and non-ocular adverse events

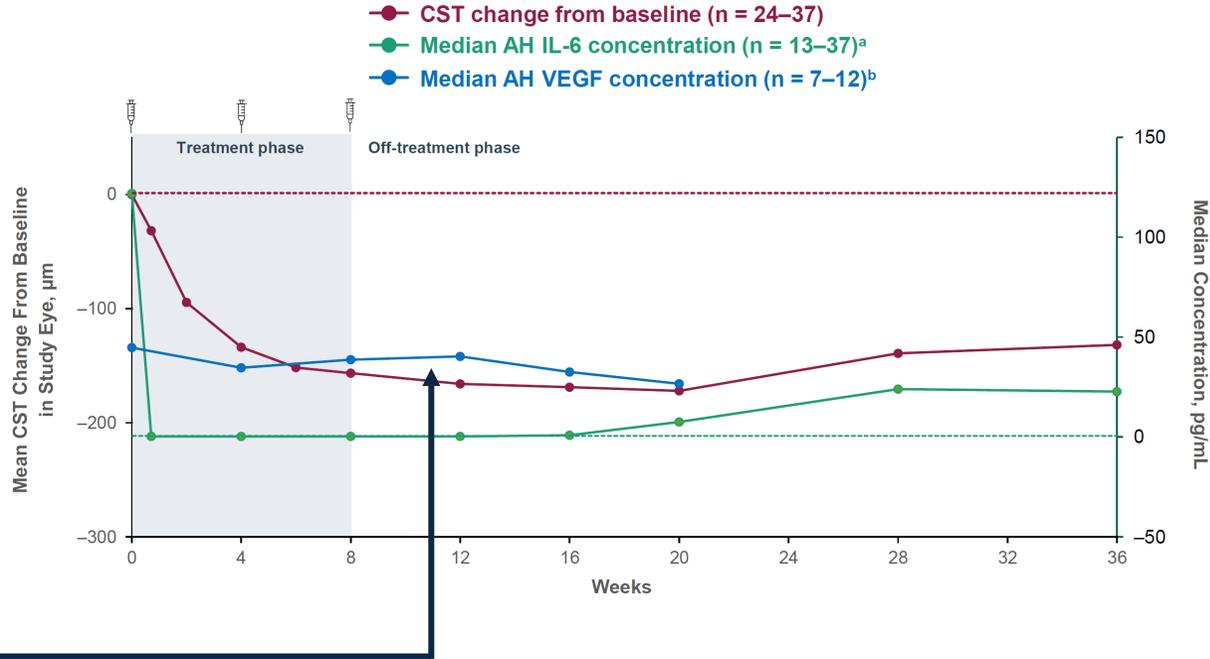
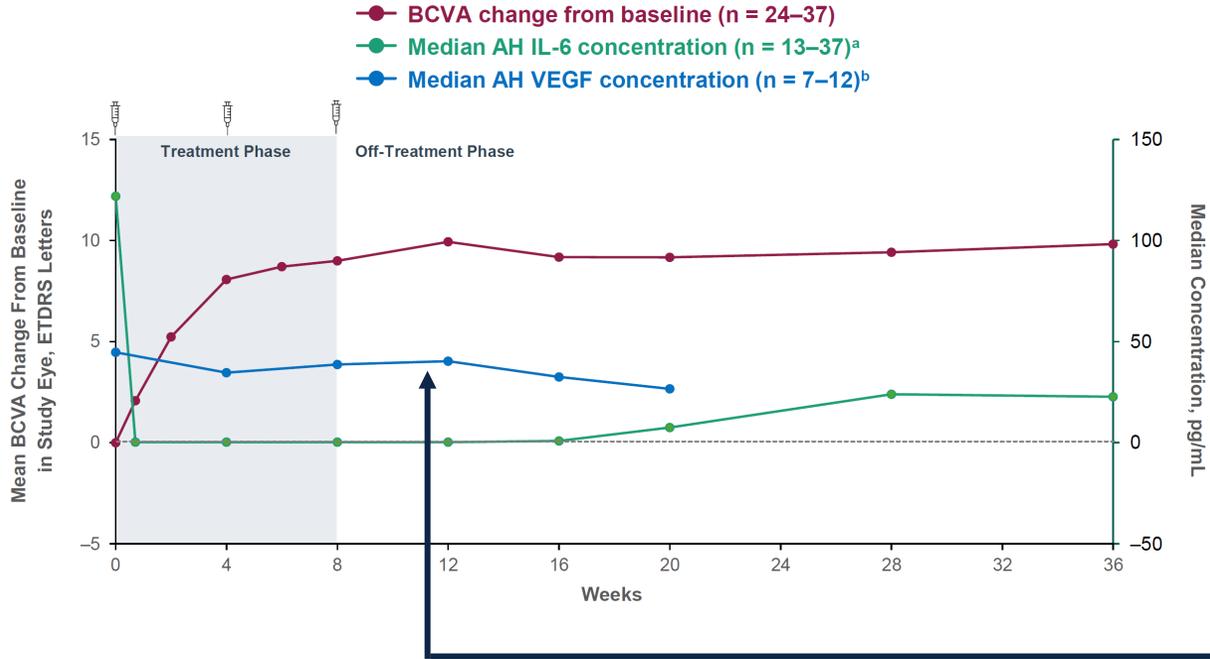
Patients Experiencing AEs Through Week 16, n (%)	MEERKAT			SANDCAT		
	Sham n = 80	Vami 0.25 mg n = 74	Vami 1.0 mg n = 78	Sham n = 82	Vami 0.25 mg n = 85	Vami 1.0 mg n = 86
Ocular AEs in the study eye						
≥ 1 AE	17 (21.3)	20 (27.0)	20 (25.6)	24 (29.3)	32 (37.6)	33 (38.4)
≥ 1 SAE	1 (1.3)	2 (2.7)	0	1 (1.2)	0	1 (1.2)
≥ 1 treatment-related AE	0	3 (4.1)	1 (1.3)	3 (3.7)	4 (4.7)	3 (3.5)
≥ 1 treatment-related SAE	0	1 (1.4)	0	0	0	0
≥ 1 ocular AE leading to treatment discontinuation	1 (1.3)	3 (4.1)	2 (2.6)	2 (2.4)	3 (3.5)	3 (3.5)
Nonocular AEs						
≥ 1 AE	20 (25.0)	24 (32.4)	24 (30.8)	27 (32.9)	30 (35.3)	31 (36.0)
≥ 1 SAE	3 (3.8)	1 (1.4)	2 (2.6)	5 (6.1)	5 (5.9)	1 (1.2)

Vamikibart Phase 3 Program
Selected ocular adverse events

Patients Experiencing Selected Ocular AEs Through Week 16, n (%) ^a	MEERKAT			SANDCAT		
	Sham n = 80	Vami 0.25 mg n = 74	Vami 1.0 mg n = 78	Sham n = 82	Vami 0.25 mg n = 85	Vami 1.0 mg n = 86
Intraocular inflammation (IOI)						
Anterior chamber inflammation	0	3 (4.1)	1 (1.3)	1 (1.2)	3 (3.5)	1 (1.2)
Eye inflammation	0	0	1 (1.3)	0	1 (1.2)	0
Iridocyclitis	0	0	1 (1.3)	0	0	1 (1.2)
Retinal occlusive vasculitis	0	1 (1.3)	0	1 (1.2)	0	0
Uveitis	0	0	0	0	0	0
Endophthalmitis	0	2 (2.7)	0	0	2 (2.4)	0
Cataract	0	0	0	0	0	0
Glaucoma/raised IOP	2 (2.5)	1 (1.4)	2 (2.6)	2 (2.4)	3 (3.5)	4 (4.7)
	4 (5.0)	4 (5.4)	5 (6.4)	4 (4.9)	7 (8.2)	6 (7.0)

A low rate of intraocular inflammation was observed, with no events of retinal vasculitis or vascular occlusion

Importantly, vamikibart was shown to have no effect on VEGF aqueous humor concentrations in DOVETAIL



Median Aqueous Humor VEGF concentration is not suppressed

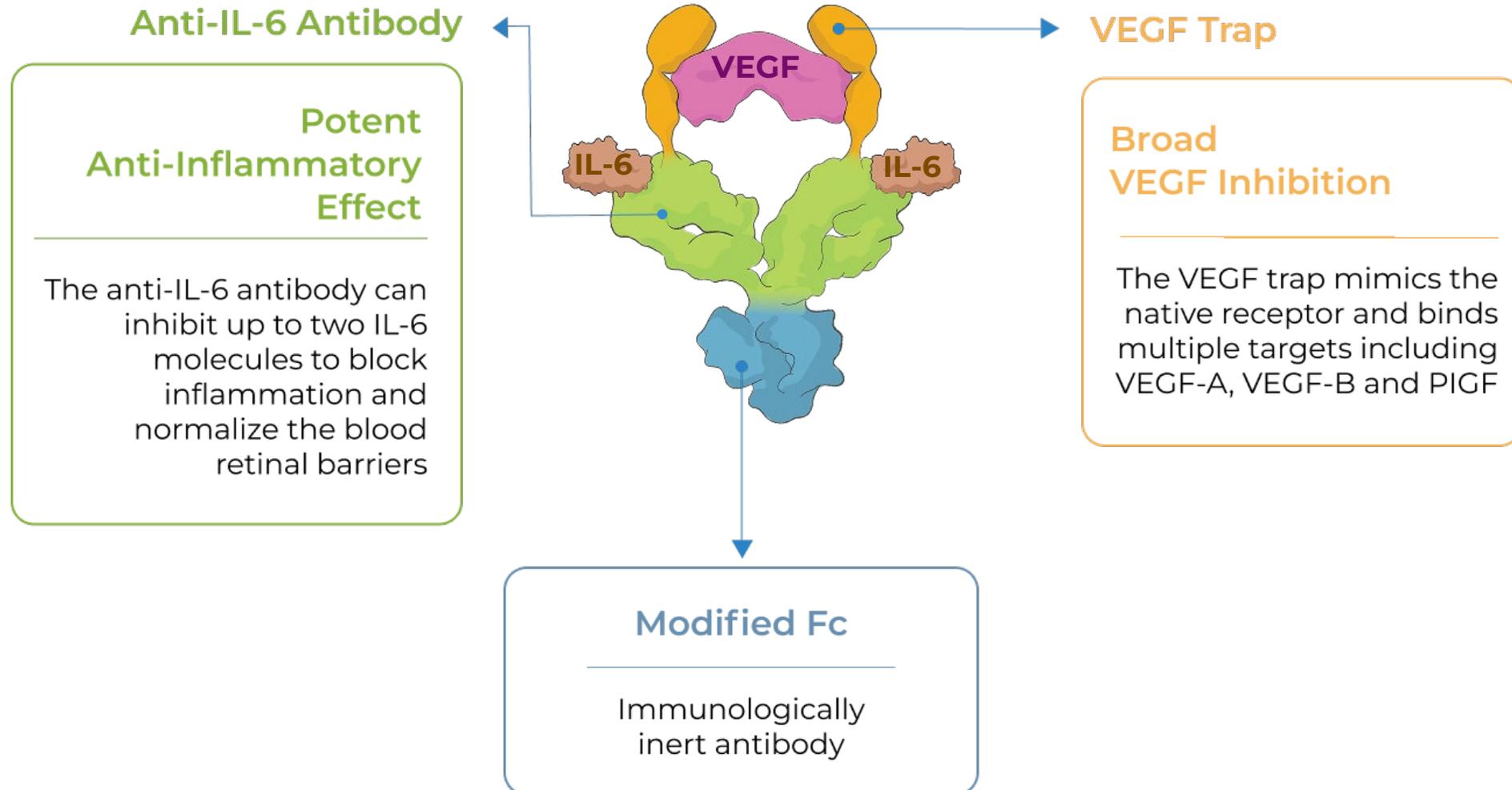
A grayscale microscopic image of retinal tissue, showing a dense field of cells with distinct nuclei and cytoplasmic structures, likely representing the macula.

Macular Edema Secondary
to Inflammation

**How can this
unmet need be
addressed?**

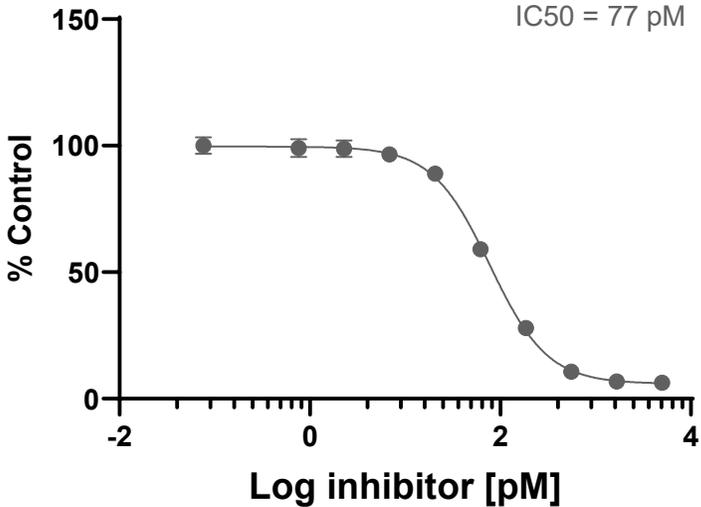
KSI-101 is a first-in-class, high-strength intravitreal biologic designed to target IL-6 mediated inflammation and VEGF-mediated vascular permeability simultaneously

KSI-101: high formulation strength (100 mg/mL)

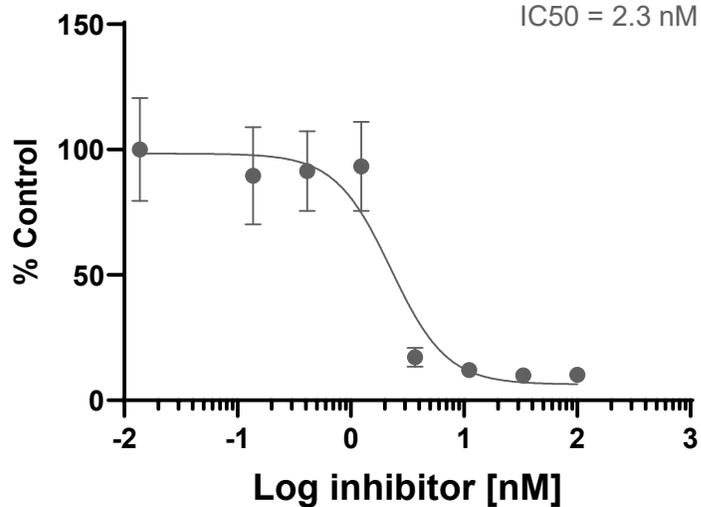


KSI-101 is a potent dual inhibitor of VEGF signaling and IL-6 classic (cis) and soluble (trans) receptor-mediated pathways

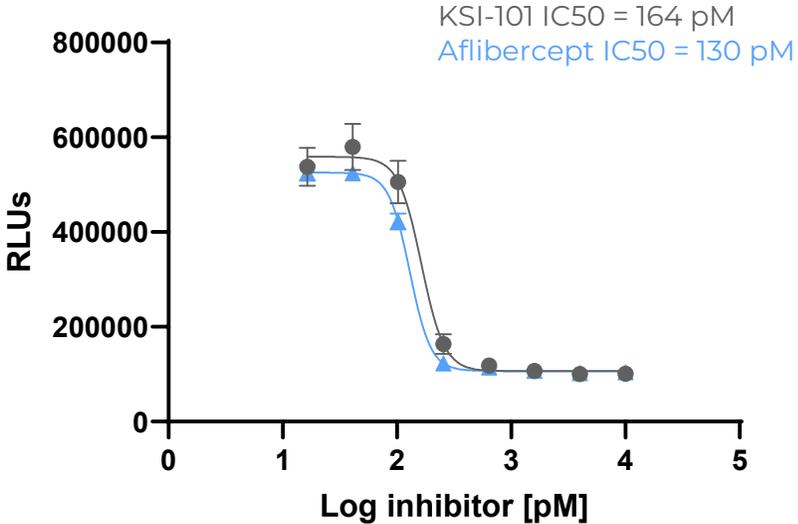
KSI-101 inhibits IL-6 classic signaling



KSI-101 inhibits soluble IL-6 receptor-mediated signaling

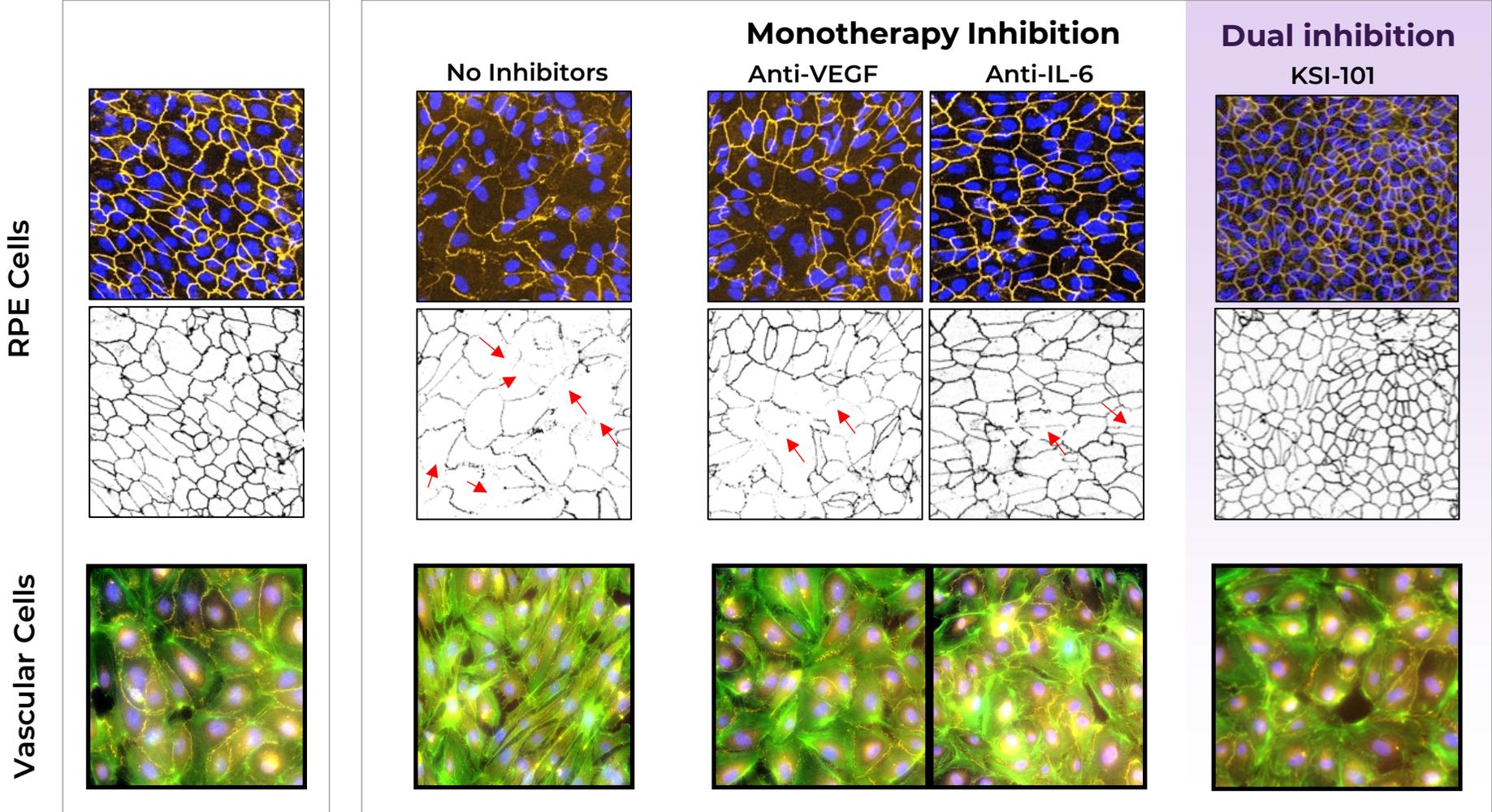


KSI-101 inhibits VEGF signaling



Bispecific KSI-101 improves barrier tight junctions greater than anti-IL-6 or anti-VEGF monotherapies alone

Exogenous VEGF and IL-6
Tight junction disruption and changes in cell morphology



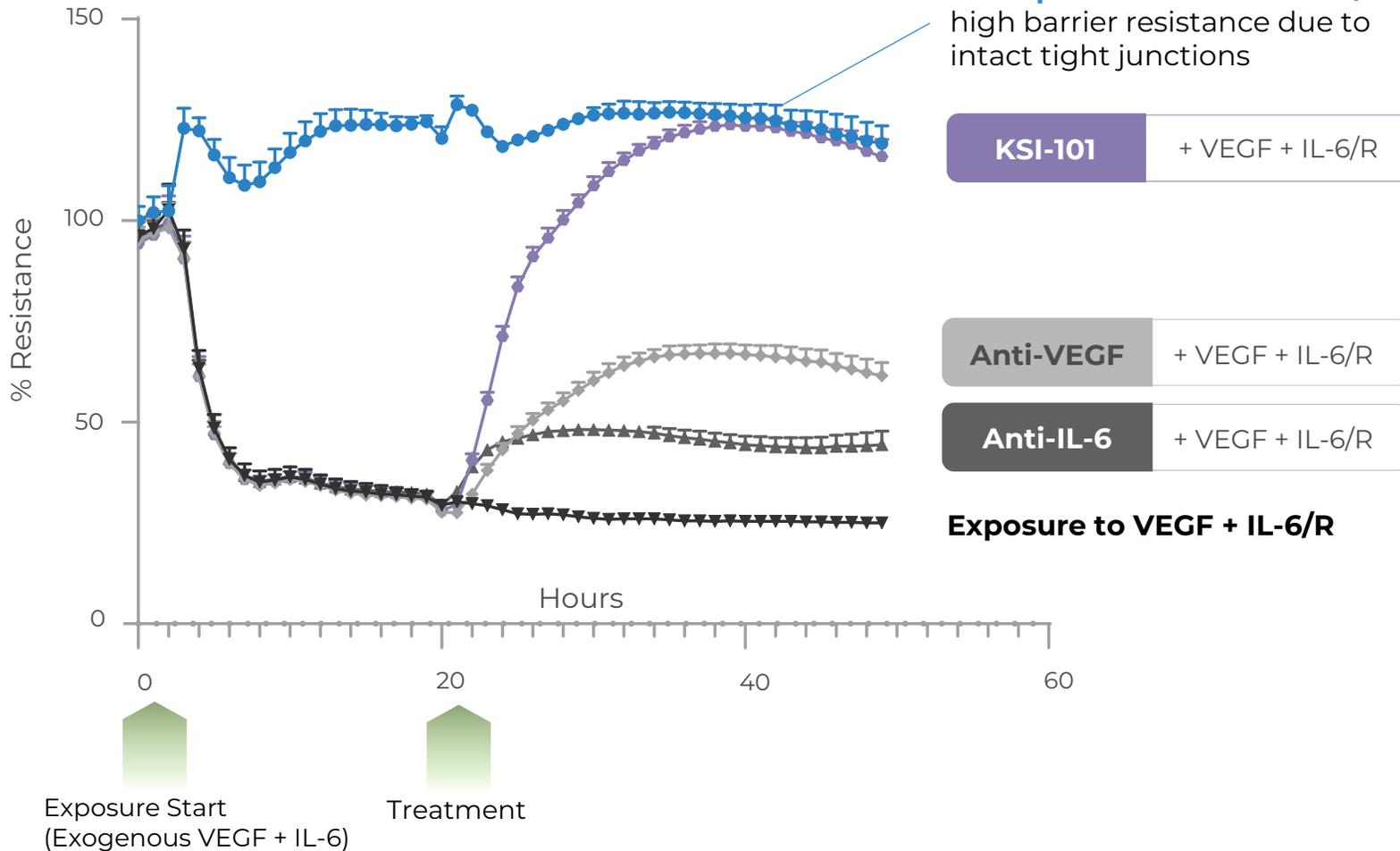
Dual inhibition by KSI-101 confers superior normalization of complex tight junction-mediated barrier biology compared to either anti-VEGF or anti-IL-6 monotherapy alone

Demonstrates the synergistic effect of IL-6 and VEGF dual inhibition

RPE cells: nuclei in blue, ZO1 (tight junction protein) in yellow. Red arrows indicate gaps in ZO-1 tight junction protein. Vascular cells: nuclei in purple, ZO1 (tight junction protein) in yellow, actin in green.

Bispecific KSI-101 restores barrier resistance from pre-existing insult greater than anti-IL-6 or anti-VEGF monotherapies alone

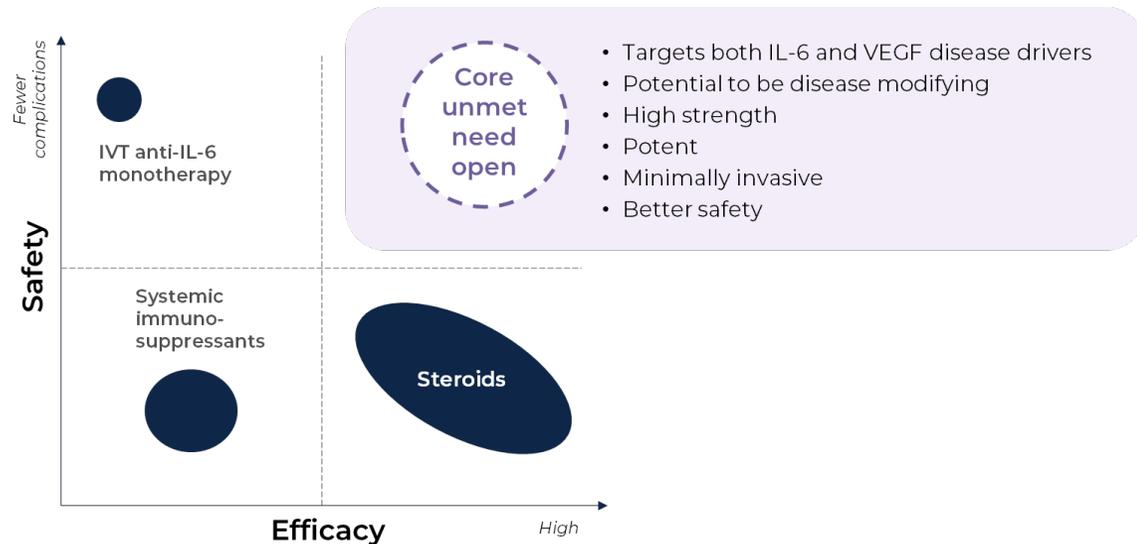
Preclinical model of endothelial cells simulating the blood-retinal barrier



KSI-101 restores barrier integrity to no exposure levels

Anti-VEGF and anti-IL-6 monotherapy only partially restore barrier function

KSI-101 is poised to fulfill the core unmet need in MESI based on its potential to be a disease modifying, high-strength, locally administered and safe biologic



KSI-101 is designed to address the core unmet need

1. **Dual anti-IL-6 and anti-VEGF inhibition**
2. **Potential for disease modifying effect** based on its synergistic inhibition of IL-6 and VEGF, as demonstrated in preclinical models and clinical cases
3. **High strength formulation** (100 mg/mL) and **high potency** provide the fire-power needed to treat “angry” inflammation and macular edema
4. **Local (intravitreal) administration**
5. **Safety profile in line with intravitreally administered biologics** (i.e., Eylea, Lucentis)

A unified treatment irrespective of presumed etiology

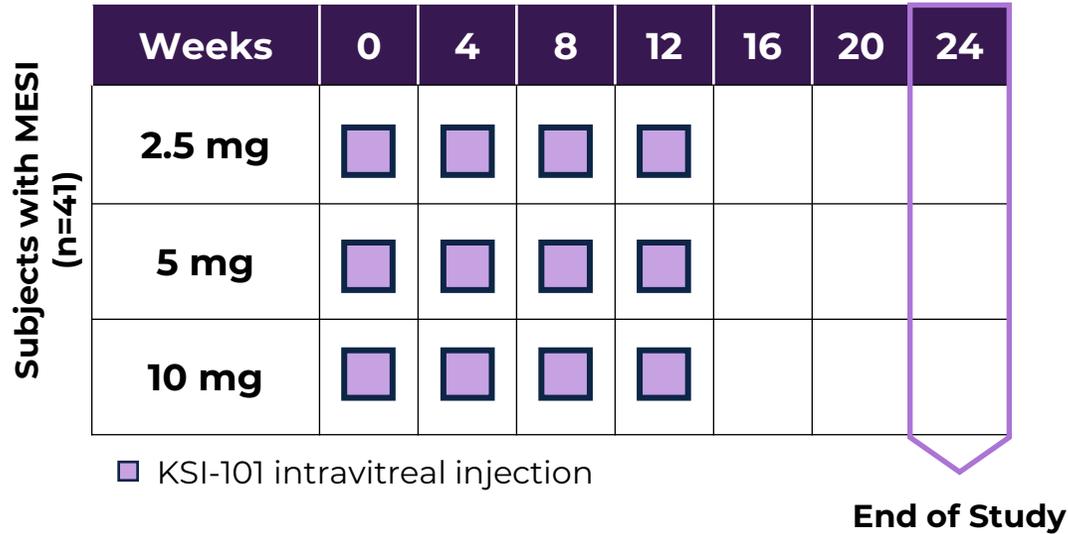
A vertical panel on the left side of the slide features a grayscale, high-magnification microscopic image of cells. The cells are densely packed and exhibit various morphologies, including some with prominent nuclei and others that are more rounded or elongated. The overall appearance is that of a tissue section or a cell culture preparation.

Phase 1b APEX KSI-101 in MESI

End-of-Study Results

Phase 1b APEX study: multiple dose study of KSI-101 in patients with MESI

Study Design: Ongoing, Open-label Phase 1b in MESI



Key inclusion criteria

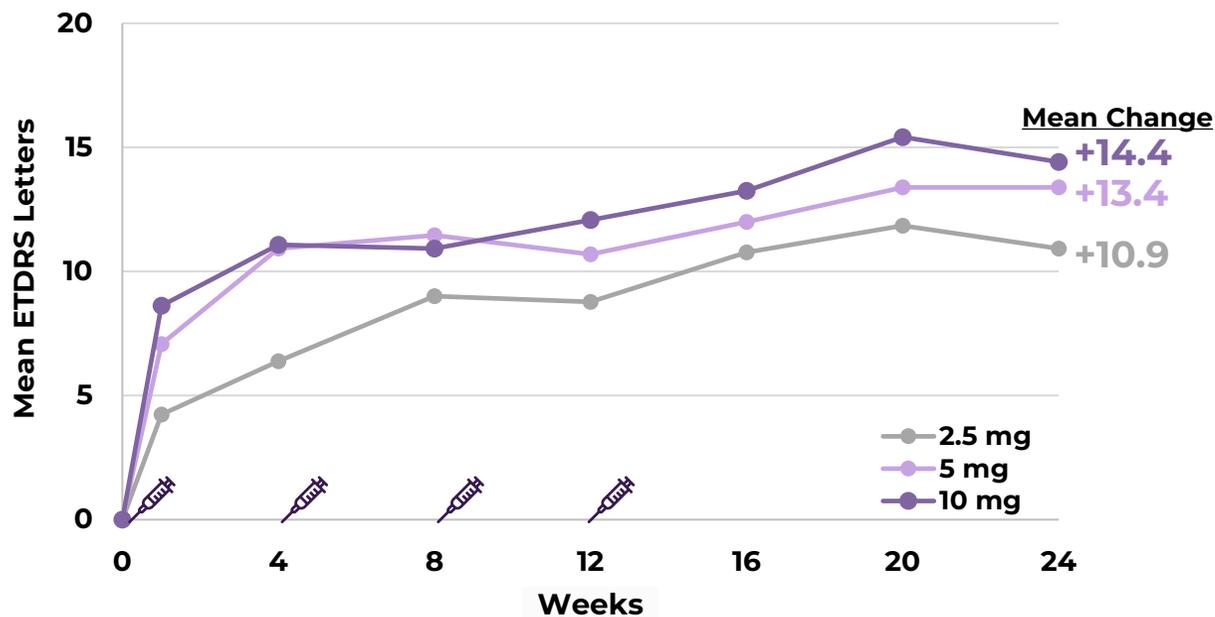
- Macular edema secondary to inflammation (MESI)
- Diagnosis of active or inactive non-infectious intraocular inflammation, acute or chronic
- Active leakage as evidenced by fluorescein angiogram
- OCT CST of ≥ 320 microns
- BCVA score ≤ 75 and ≥ 25 (20/32 to 20/320 Snellen equivalent)

Baseline Characteristics

	KSI-101 2.5 mg (n=13)	KSI-101 5 mg (n=14)	KSI-101 10 mg (n=14)	All KSI-101 (N=41)
Age, years, mean (SD)	74.2 (11.6)	67.4 (8.1)	67.5 (18.8)	69.6 (13.7)
Female, n (%)	8 (61.5)	7 (50.0)	8 (57.1)	23 (56.1)
Race, White, n (%)	11 (84.6)	11 (78.6)	14 (100)	36 (87.8)
MESI disease duration, months, mean (SD)	12.2 (20.1)	1.7 (1.2)	15.8 (37.2)	11.1 (26.5)
Inflammation anatomical location, n (%)				
Anterior	0	2 (14.3)	0	2 (4.9)
Intermediate	1 (7.7)	0	2 (14.3)	3 (7.3)
Posterior	10 (76.9)	6 (42.9)	10 (71.4)	26 (63.4)
Panuveitis	2 (15.4)	6 (42.9)	2 (14.3)	10 (24.4)
Patients with active inflammation, n (%)	3 (23.1)	10 (71.4)	5 (35.7)	18 (43.9)
Unilateral MESI, n (%)	9 (69.2)	6 (42.9)	5 (35.7)	20 (48.8)
BCVA, ETDRS Letters, mean (SD)	62.7 (7.4)	65.5 (7.8)	62.1 (8.4)	63.5 (7.8)
Snellen equivalent	~20/50	~20/50	~20/63	~20/50
OCT CST, μm, mean (SD)	461.7 (137.7)	487.0 (124.1)	528.6 (157.3)	493.2 (139.7)
Lens Status, pseudophakic, n (%)	9 (69.2)	13 (92.9)	11 (78.6)	33 (80.5)

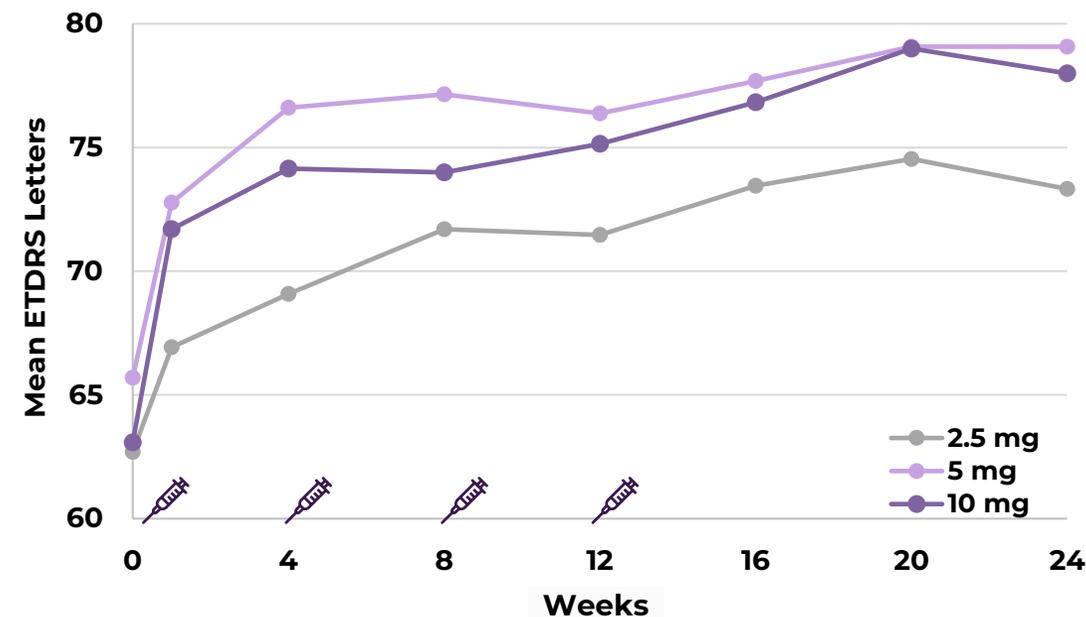
The top two dose levels achieve meaningful vision gains of >10 letters by Week 4 and continue to improve over time, achieving a 20/25 Snellen visual acuity by Week 20

Mean Change in BCVA over time



Dose Level	0	4	8	12	16	20	24
2.5 mg	13	13	13	13	13	13	12
5 mg	13	13	13	13	13	13	13
10 mg	13	13	13	13	12	12	12

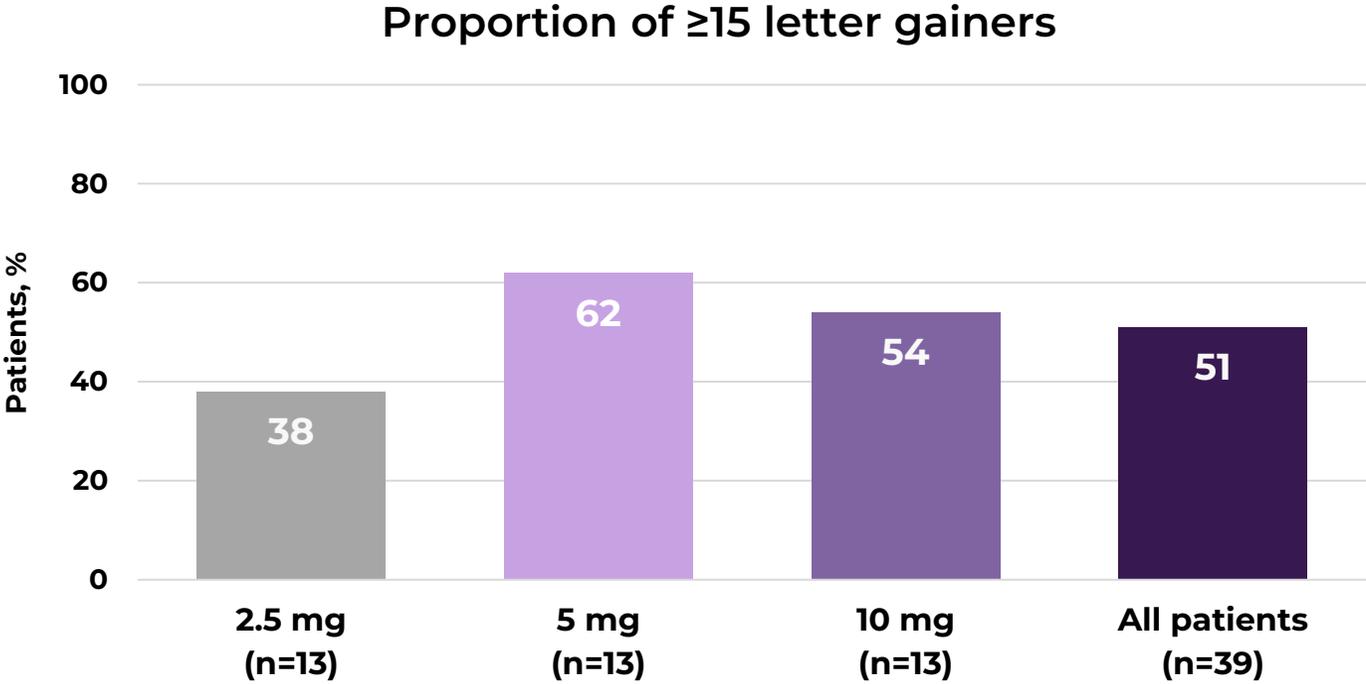
Observed BCVA over time



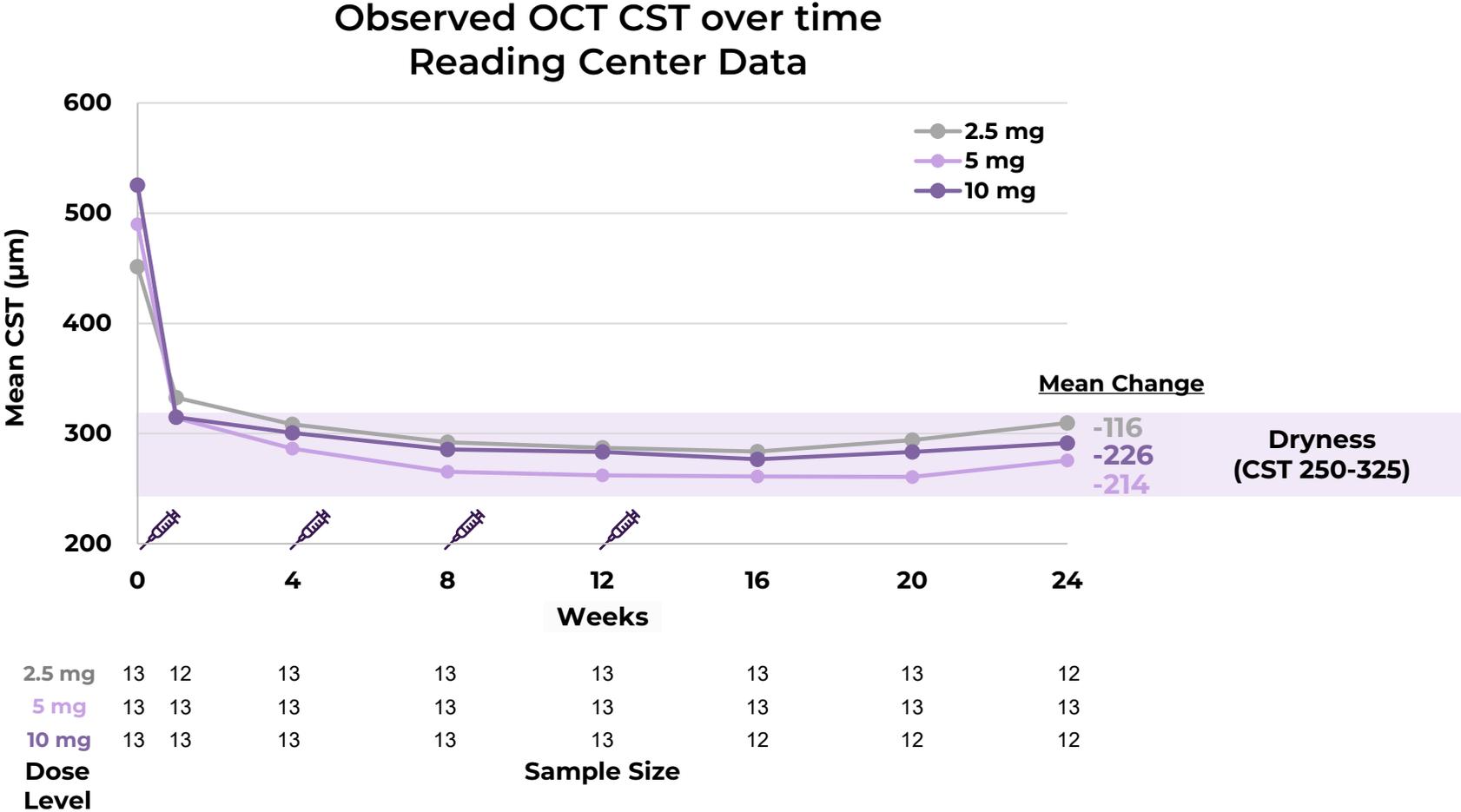
Dose Level	0	4	8	12	16	20	24
2.5 mg	13	13	13	13	13	13	12
5 mg	13	13	13	13	13	13	13
10 mg	13	13	13	13	12	12	12

Final results of the APEX study in MESI. Includes patients in the per protocol set that completed the Week 4 visit and met all the eligibility criteria. Excludes one patient in the 5 mg dose that discontinued treatment before Week 4, and one patient in the 10 mg dose with a significant epiretinal membrane at baseline (exclusion criterion). One patient in the 10 mg dose level discontinued after Week 12 due to recurrent uveitis flare-up

More than half of patients achieved a ≥ 15 letter gain, with additional benefit observed in the top dose levels



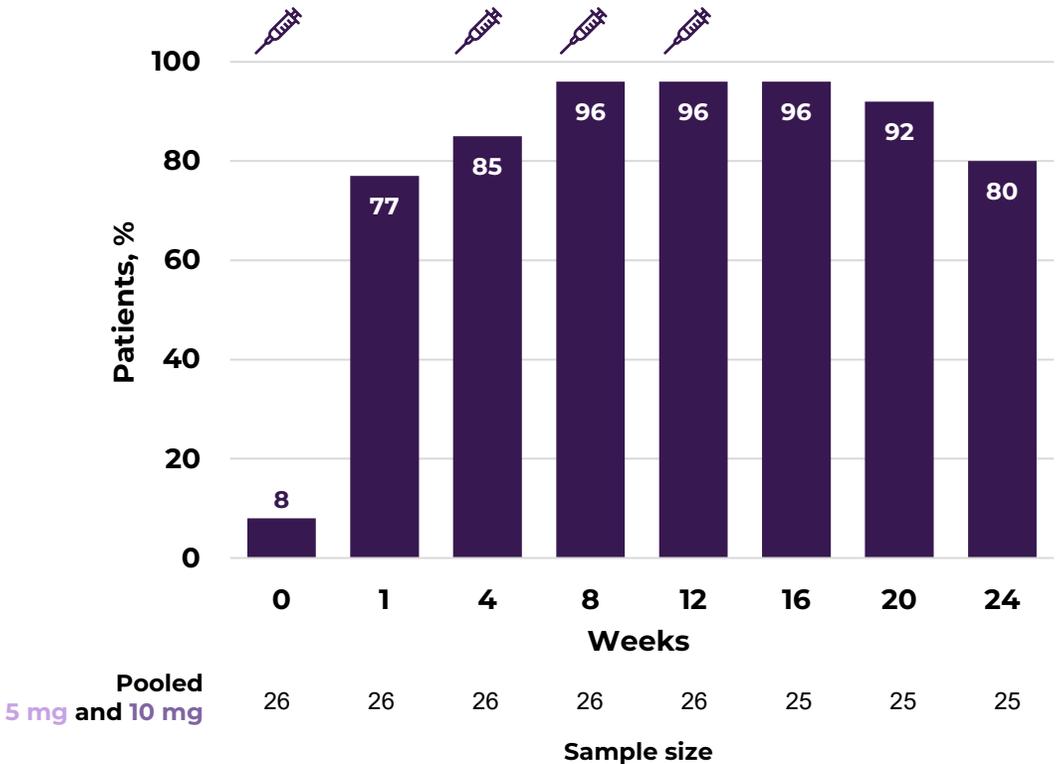
Meaningful anatomical improvements are rapidly achieved, with OCT CST levels <325 μm observed in the top two dose levels as early as Week 1, further deepening over time



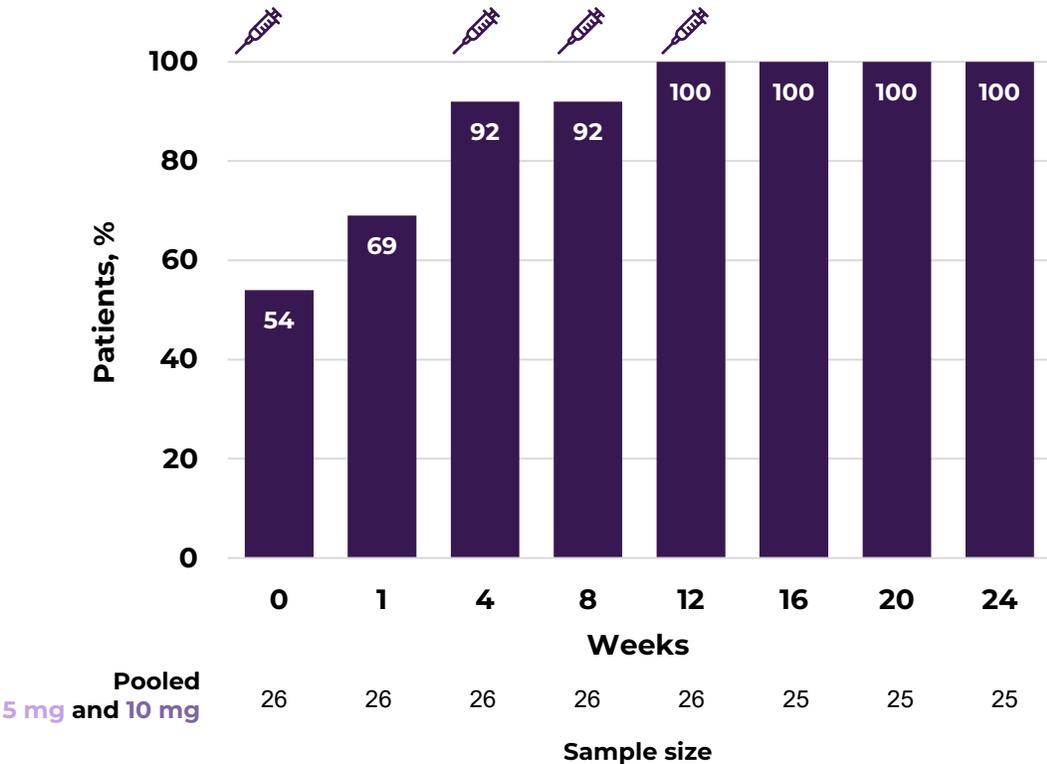
Final results of the APEX study in MESI. Includes patients in the per protocol set that completed the Week 4 visit and met all the eligibility criteria. Excludes one patient in the 5 mg dose that discontinued treatment before Week 4, and one patient in the 10 mg dose with a significant epiretinal membrane at baseline (exclusion criterion). One patient in the 10 mg dose level discontinued after Week 12 due to recurrent uveitis flare-up.

≥90% of patients in the top two dose levels achieved absence of both intraretinal and subretinal fluid. A recurrence in IRF was observed in some patients 8 to 12 weeks after the last study treatment

Proportion of patients in the 5 and 10 mg (pooled) dose level achieving absence of IRF



Proportion of patients in the 5 and 10 mg (pooled) dose level achieving absence of SRF



Final results of the APEX study in MESI. Includes patients in the per protocol set that completed the Week 4 visit and met all the eligibility criteria. Excludes one patient in the 5 mg dose that discontinued treatment before Week 4, and one patient in the 10 mg dose with a significant epiretinal membrane at baseline (exclusion criterion). One patient in the 10 mg dose level discontinued after Week 12 due to recurrent uveitis flare-up.

KSI-101 has been well-tolerated

	KSI-101 2.5 mg (n=13)	KSI-101 5 mg (n=14)	KSI-101 10 mg (n=14)	All KSI-101 (N=41)
Summary of AEs in the Study eye, n (%)				
Subjects with ≥1 AEs	2 (15.4)	3 (21.4)	2 (14.3)	7 (17.1)
Treatment-related AEs	1 (7.7) ^a	1 (7.1) ^b	0	2 (4.9)
Serious AEs	0	0	0	0
Treatment-related serious AEs	0	0	0	0
Severe AEs	0	0	0	0
AEs leading to study discontinuation	0	1 (7.1) ^b	0	1 (2.4)
Selected AEs in the Study Eye, n (%)				
Intraocular inflammation (recurrent uveitis flare-up)	1 (7.7) ^a	1 (7.1) ^b	0	2 (4.9)
Occlusive retinal vasculitis	0	0	0	0
Cataract	0	0	0	0
Elevated IOP	0	0	0	0
Eye Pain	1 (7.7) ^a	0	0	1 (2.4)
Vitreous hemorrhage	1 (7.7) ^a	0	0	1 (2.4)

Final results from the APEX Study in MESI.

AE, Adverse event; IOP, intraocular pressure. Events are investigator reported. Adverse events are treatment-emergent events with start date ≥first study drug date and ≤last study drug date + 28 days.

^a Same patient. Vitreous hemorrhage secondary to aqueous humor sampling at the Day 1 visit (pre-dose). The patient had 3+ AC cells and flare and 2+ vitreous haze **prior** to the Day 1 KSI-101 dose. The patient safely received all 4 doses of KSI-101 and is +26 letters in BCVA at their last visit and no intraocular inflammation.

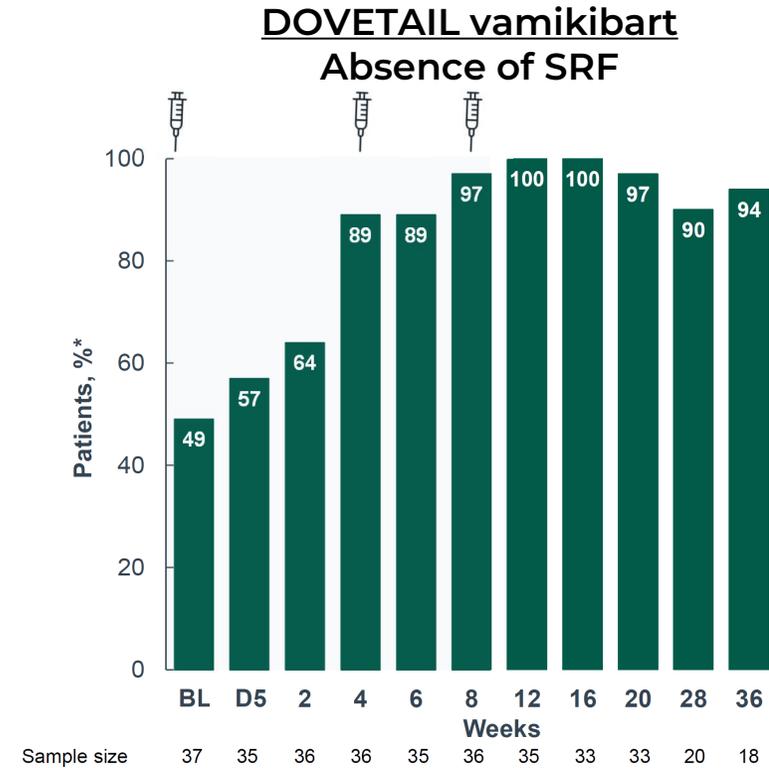
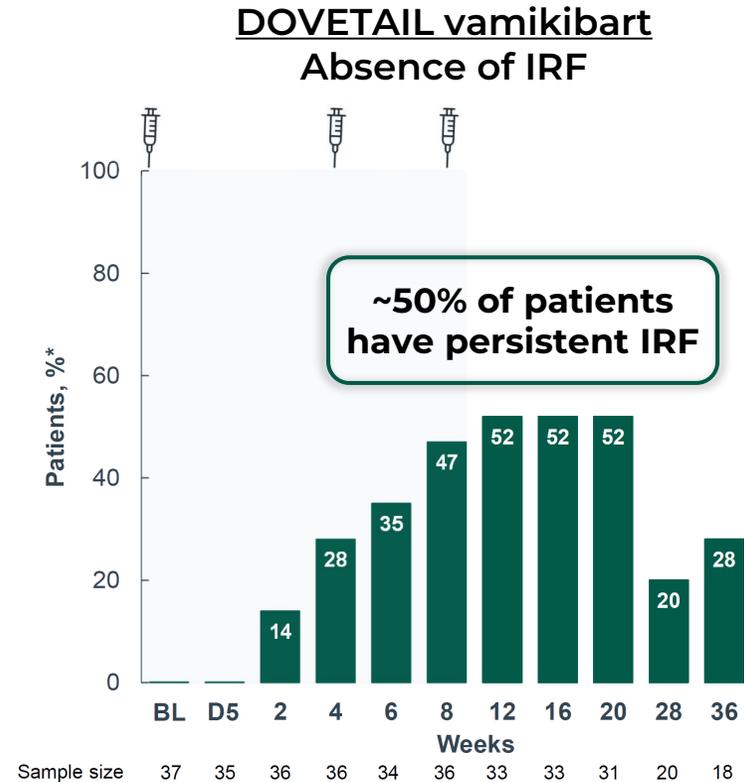
^b Same patient. Uveitis flare-up consistent with underlying disease.

A large, light-colored background image on the left side of the slide, showing a dense field of microscopic cells, likely retinal cells, with various shapes and some darker spots, suggesting a biological or medical context.

Macular Edema Secondary
to Inflammation

**How does KSI-101
fit into the emerging
treatment landscape?**

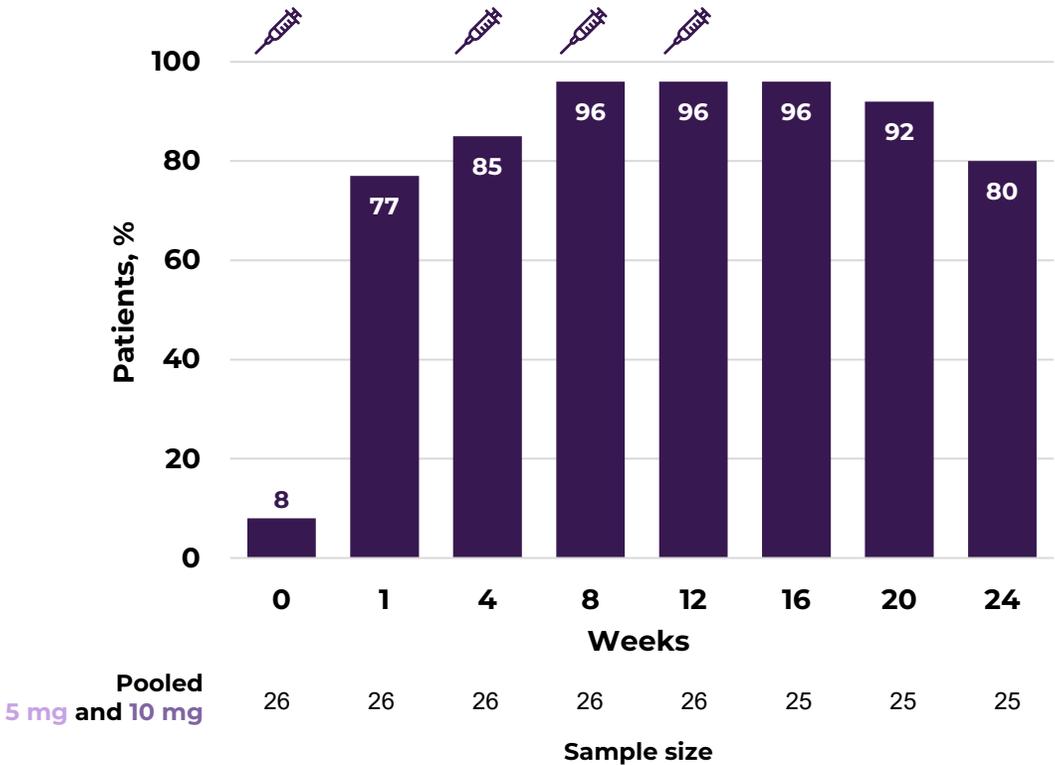
DOVETAIL **vamikibart** – while intravitreal IL-6 monotherapy is helpful, 50% of patients have persistent Intra Retinal Fluid



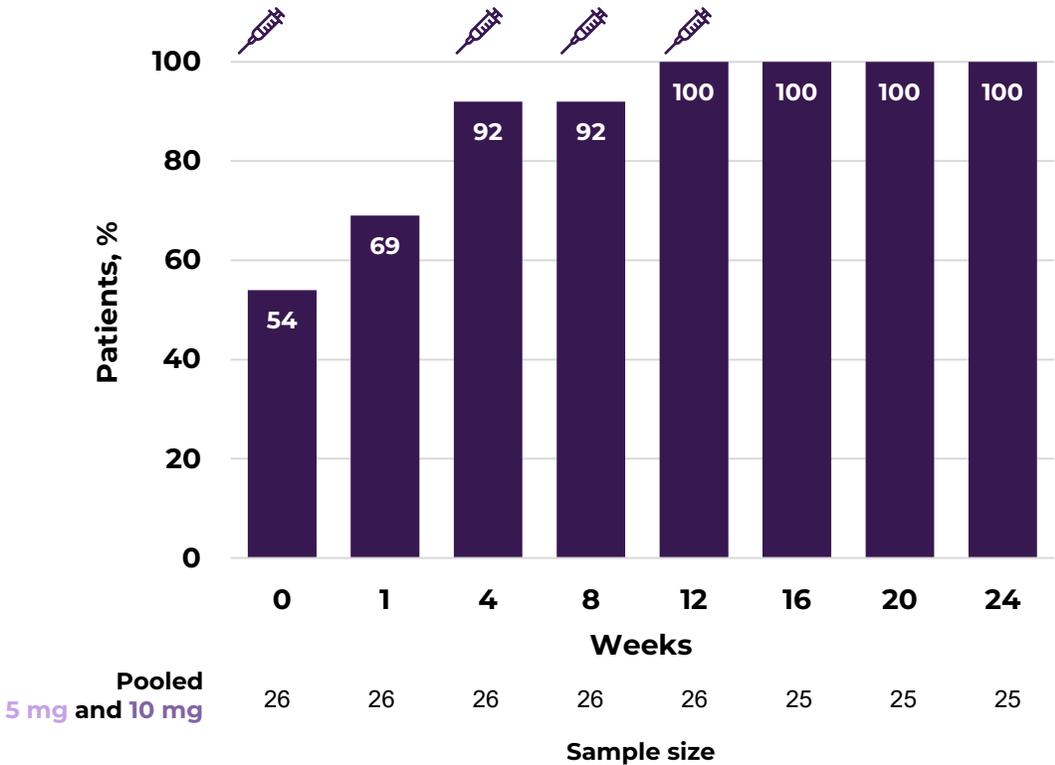
Persistent intraretinal fluid (IRF) is known to cause permanent negative effects on visual function

KSI-101 seems to provide faster and better disease control, with $\geq 90\%$ of patients in the top two dose levels achieving absence of both intraretinal and subretinal fluid

Proportion of patients in the 5 and 10 mg (pooled) dose level achieving absence of IRF

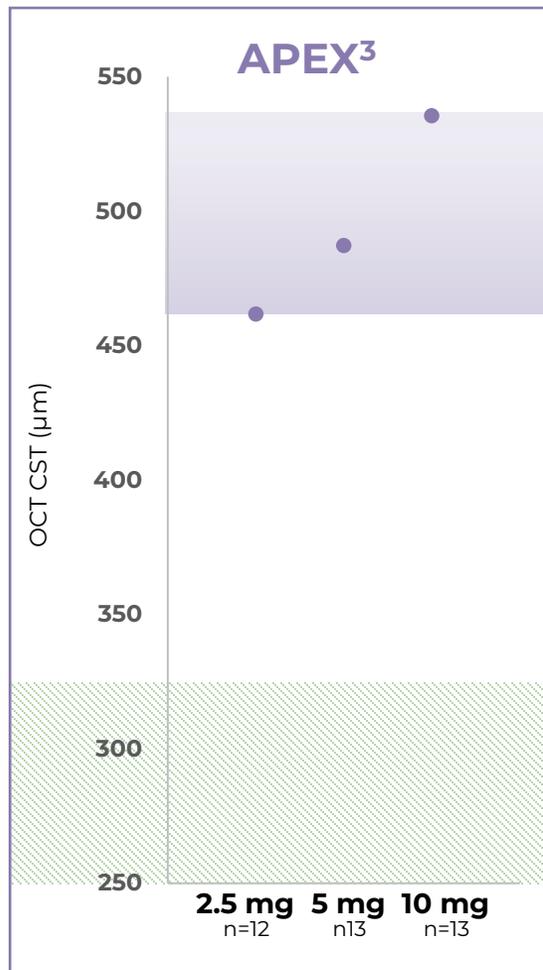


Proportion of patients in the 5 and 10 mg (pooled) dose level achieving absence of SRF



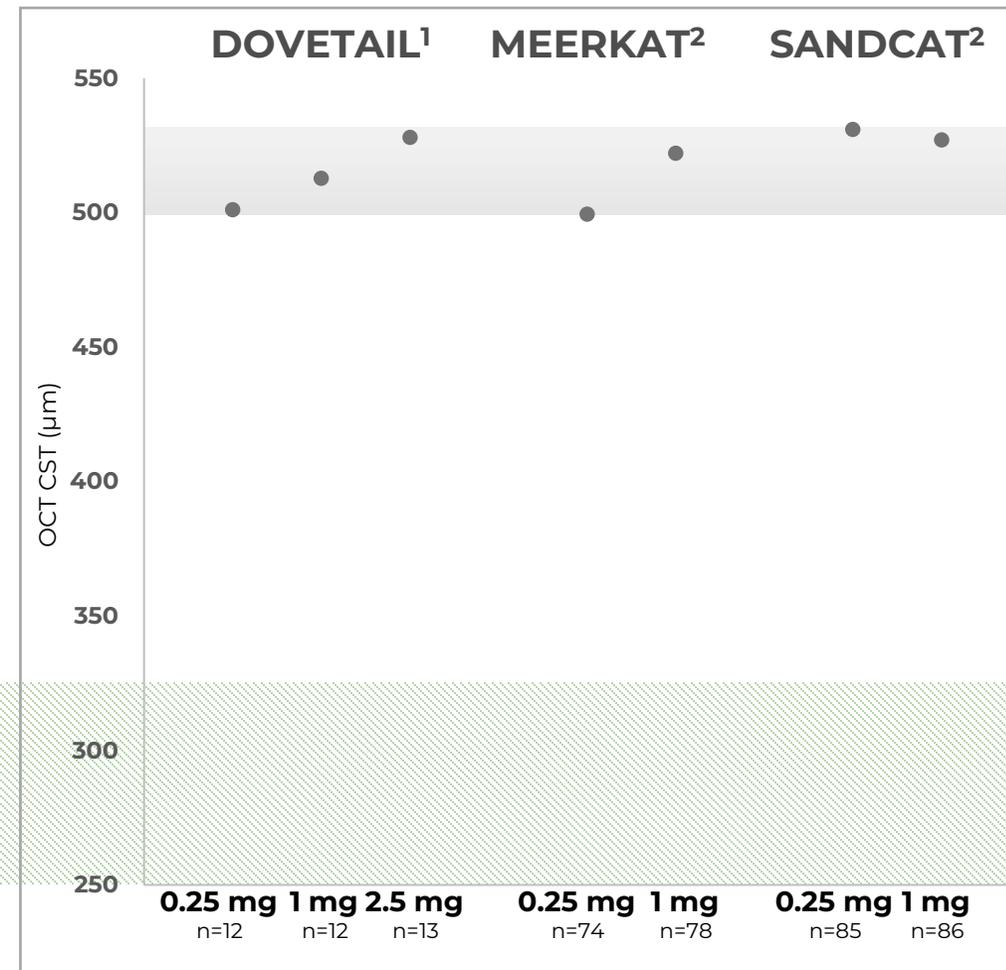
Final results of the APEX study in MESI. Includes patients in the per protocol set that completed the Week 4 visit and met all the eligibility criteria. Excludes one patient in the 5 mg dose that discontinued treatment before Week 4, and one patient in the 10 mg dose with a significant epiretinal membrane at baseline (exclusion criterion). One patient in the 10 mg dose level discontinued after Week 12 due to recurrent uveitis flare-up.

Mean Change in OCT CST and Absolute CST



Day 1 

 Day 1



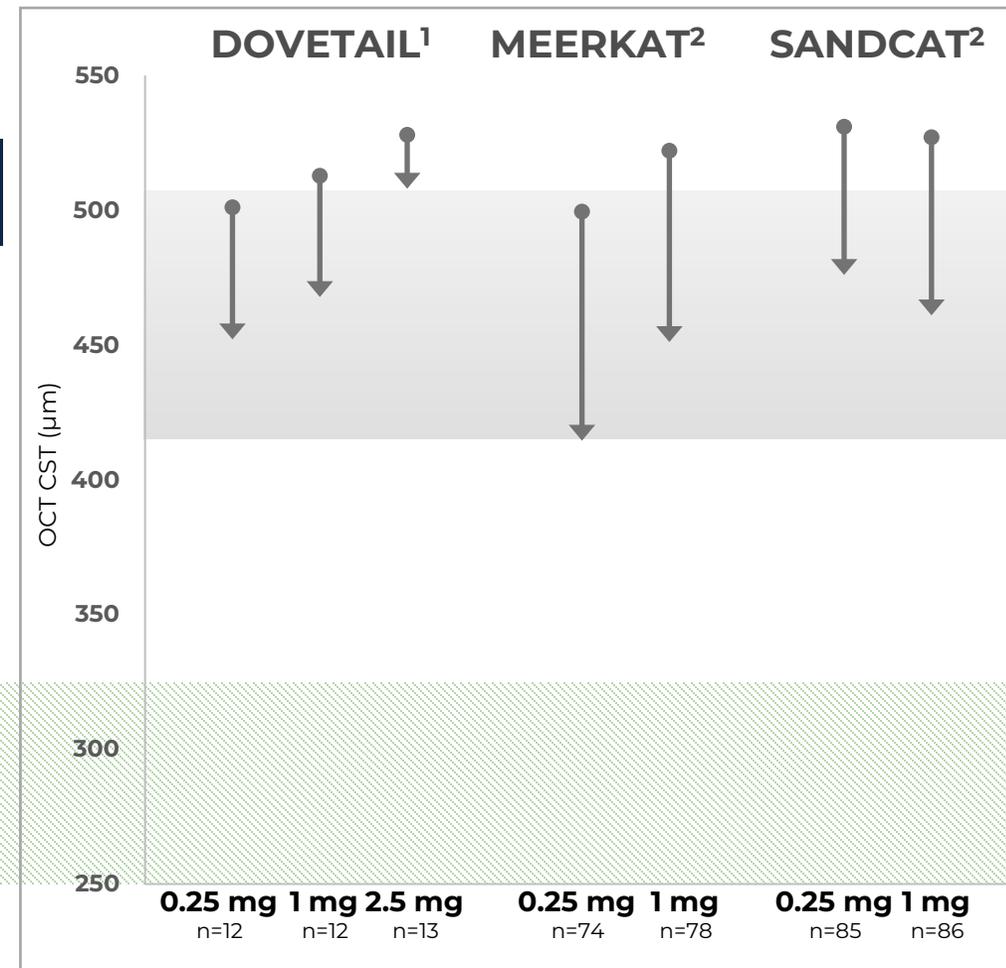
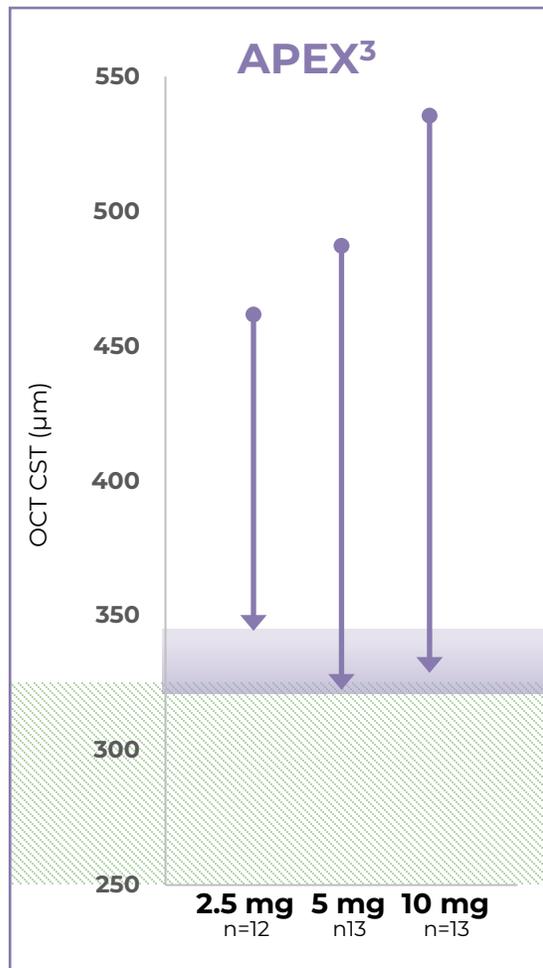
**Dryness
(CST 250 – 325)**

Anti-IL-6, VEGF trap
KSI-101

Anti-IL-6
Vamikibart

For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

Mean Change in OCT CST and Absolute CST



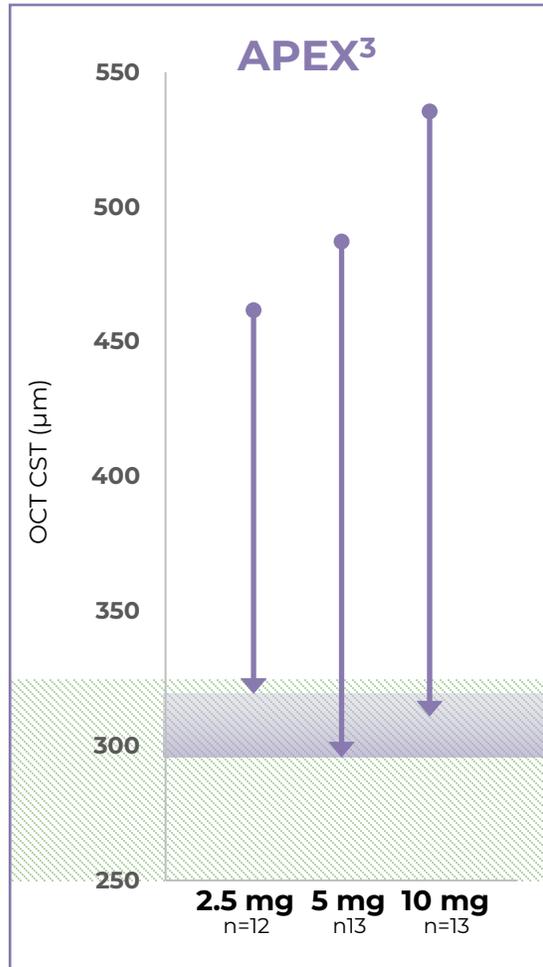
Anti-IL-6, VEGF trap
KSI-101

**Rapid onset of action
observed with KSI-101**

Anti-IL-6
Vamikibart

For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

Mean Change in OCT CST and Absolute CST

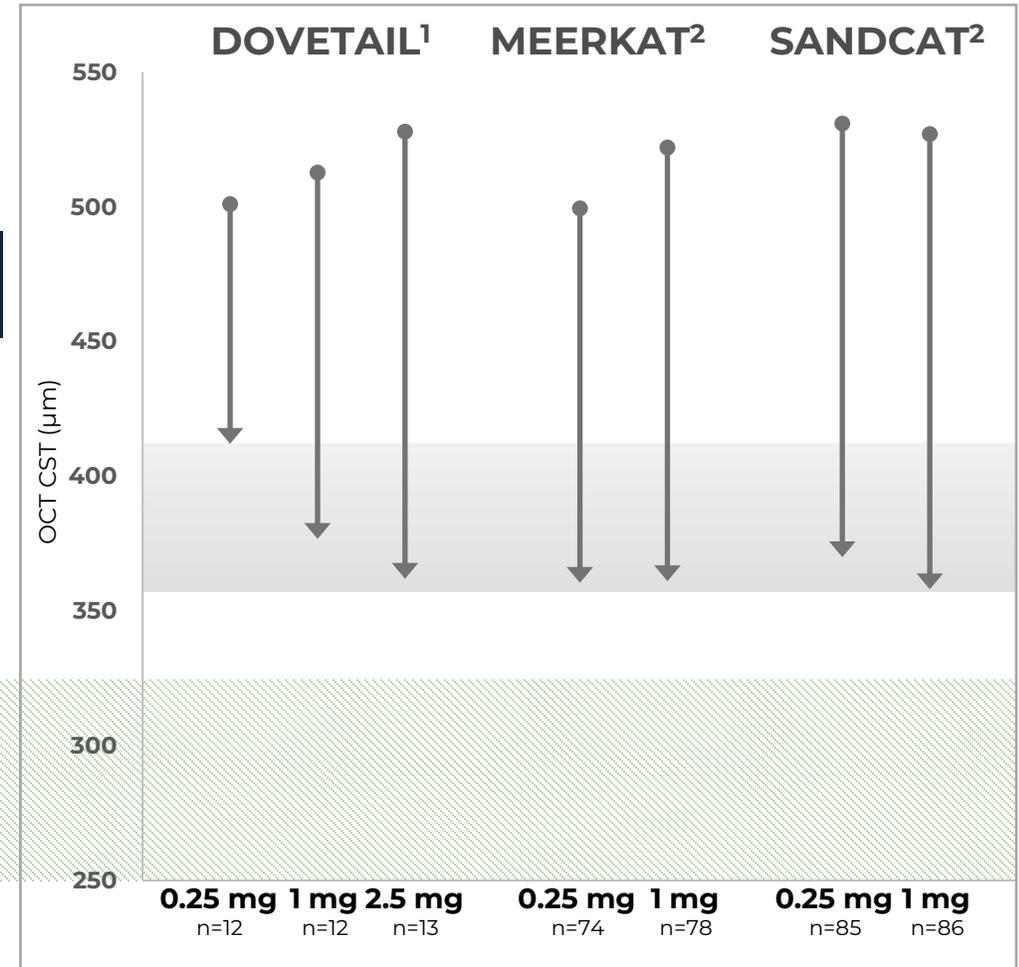


Anti-IL-6, VEGF trap
KSI-101



Dryness (CST 250 – 325)

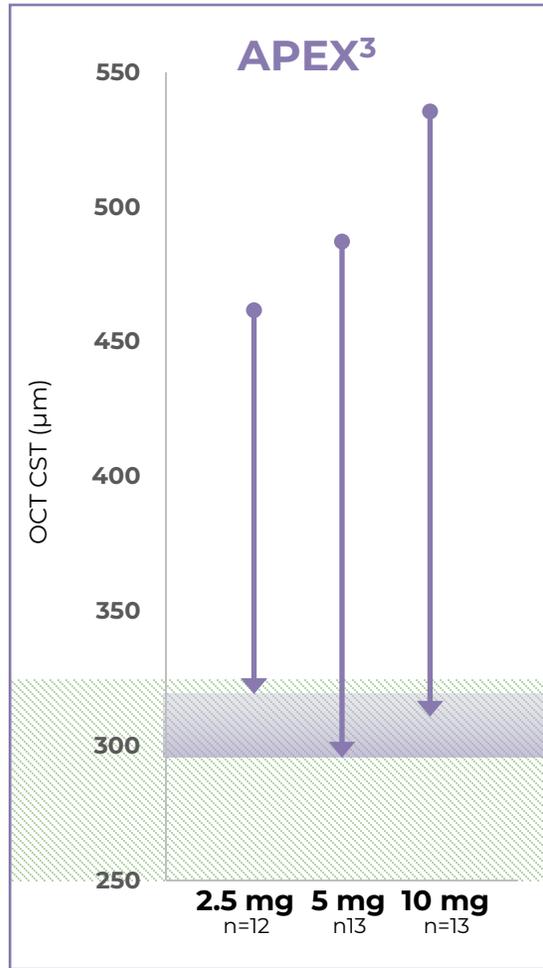
Dryness levels observed with a single dose of KSI-101



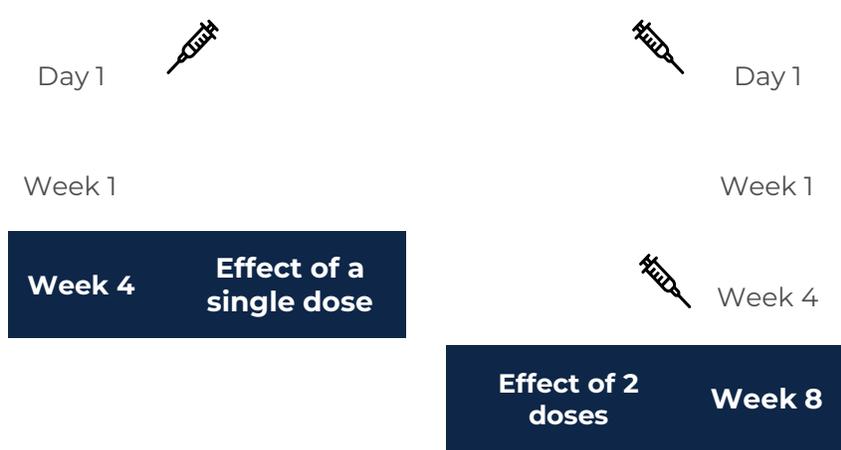
Anti-IL-6
Vamikibart

For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

Mean Change in OCT CST and Absolute CST

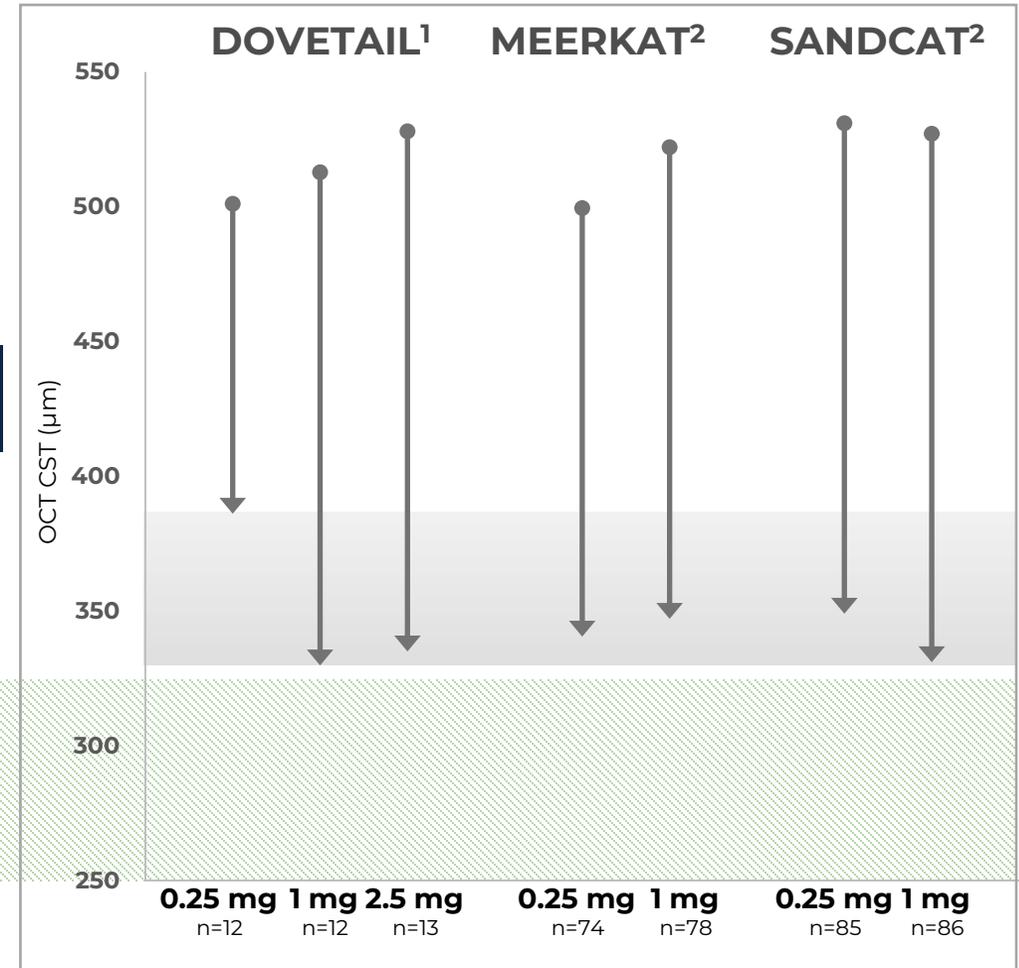


Anti-IL-6, VEGF trap
KSI-101



Dryness
(CST 250 – 325)

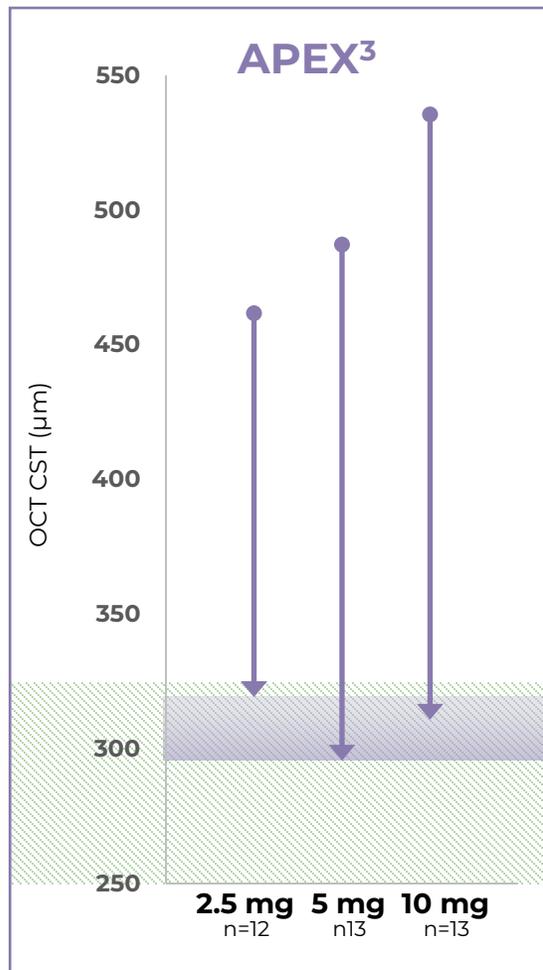
Dryness levels observed with
a single dose of KSI-101



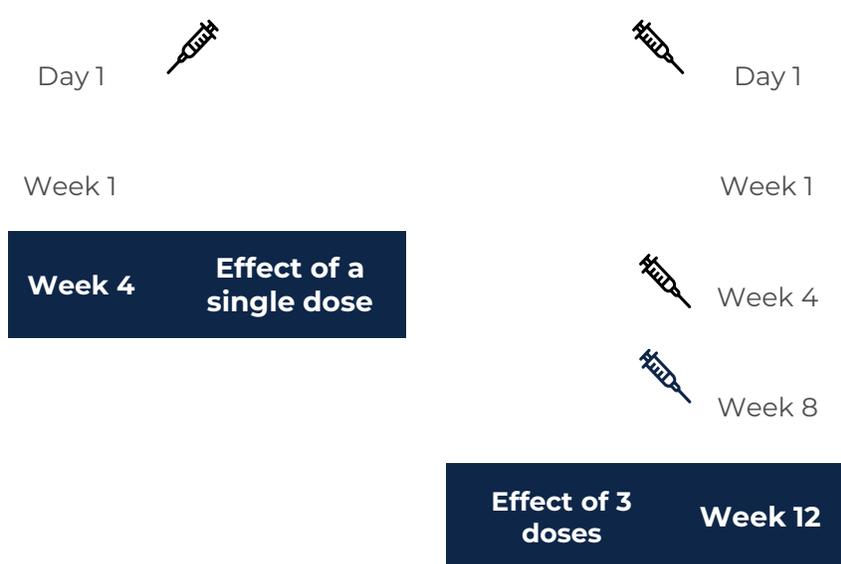
Anti-IL-6
Vamikibart

For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

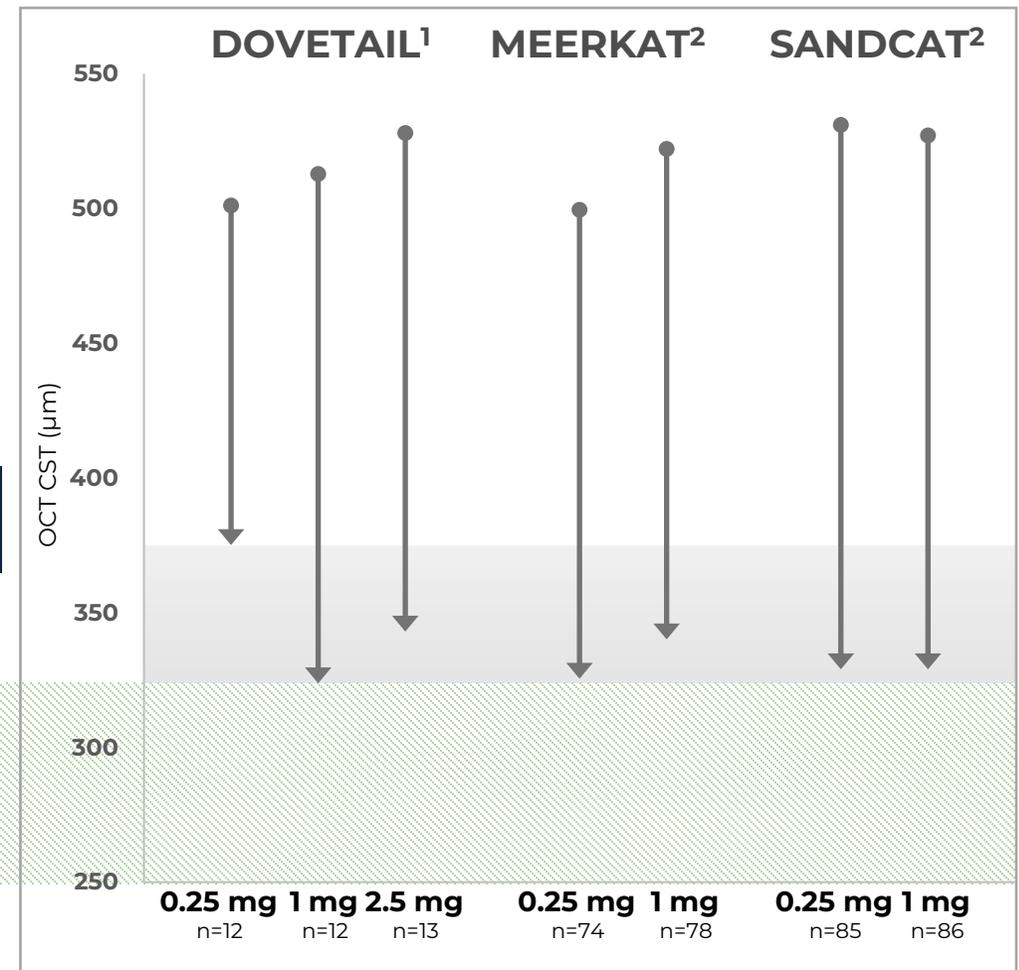
Mean Change in OCT CST and Absolute CST



Anti-IL-6, VEGF trap
KSI-101



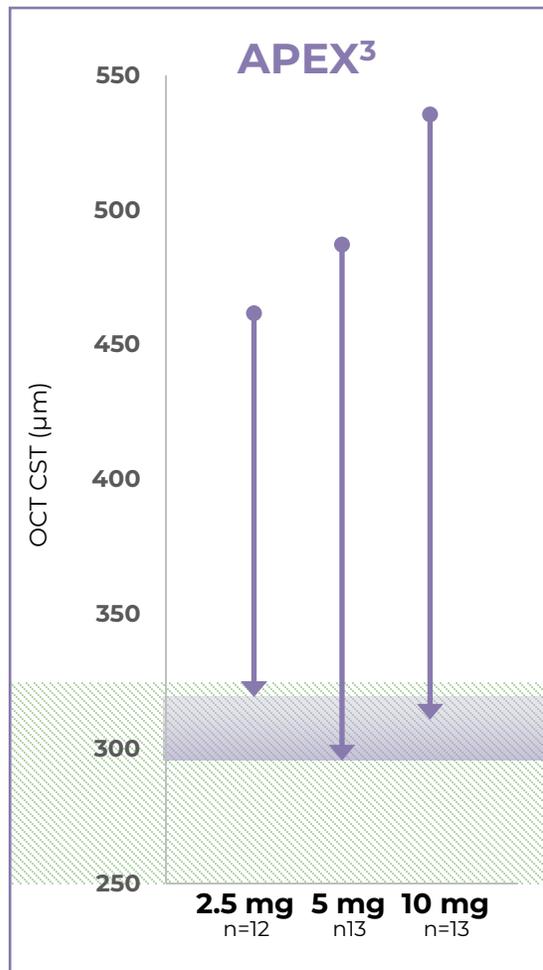
Dryness levels observed with a single dose of KSI-101



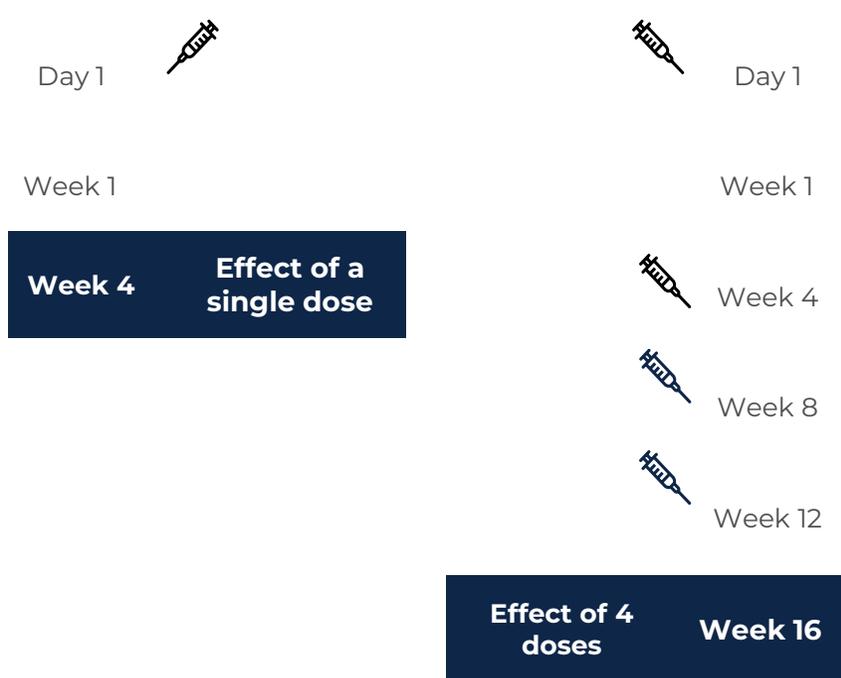
Anti-IL-6
Vamikibart

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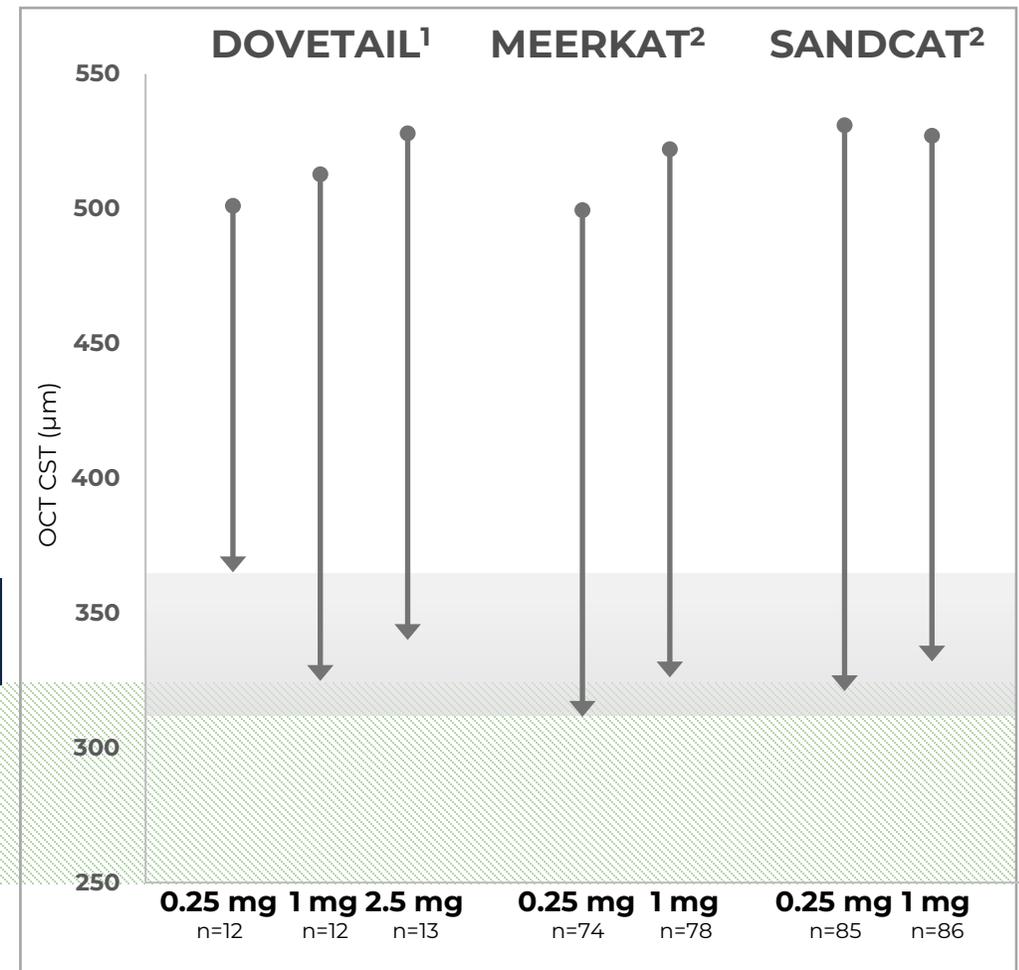
Mean Change in OCT CST and Absolute CST



Anti-IL-6, VEGF trap
KSI-101



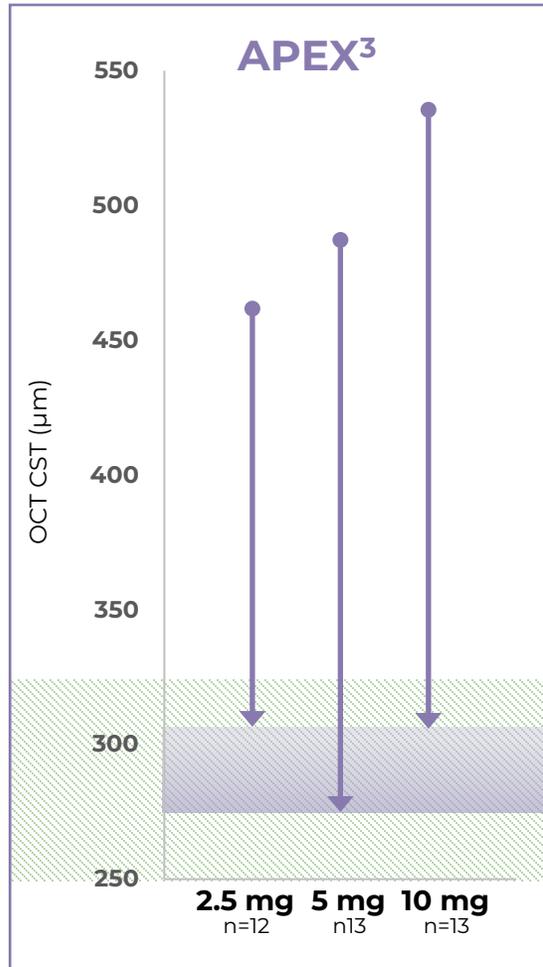
A single dose of KSI-101 seems to provide a deeper drying effect than 4 doses of anti-IL-6 monotherapy



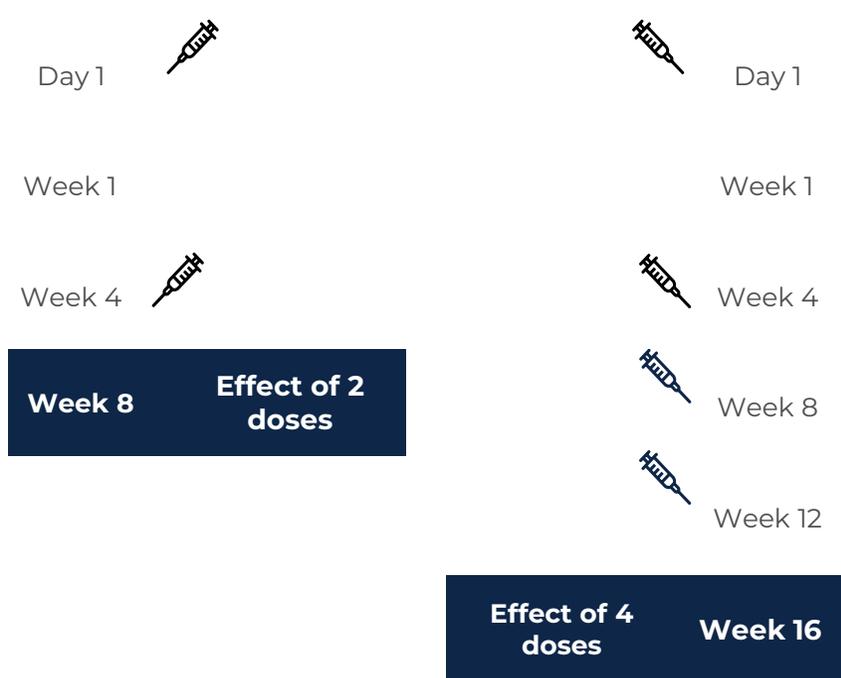
Anti-IL-6
Vamikibart

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Mean Change in OCT CST and Absolute CST

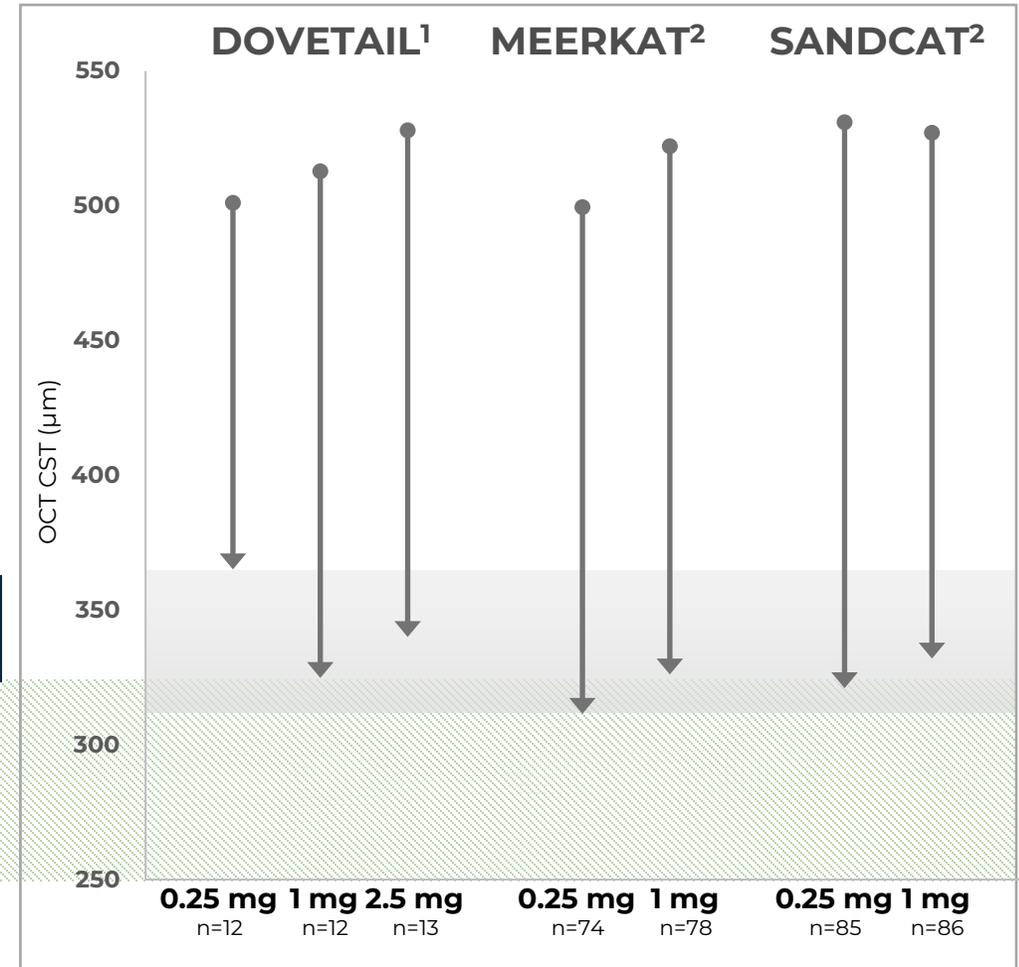


Anti-IL-6, VEGF trap
KSI-101



Dryness
(CST 250 – 325)

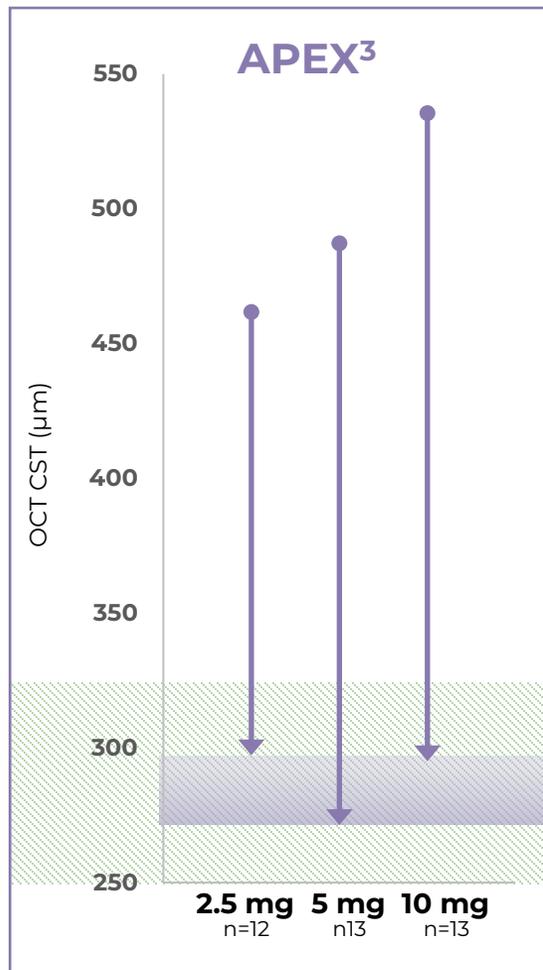
Continued dosing with KSI-101 provides further deepening into the dryness corridor



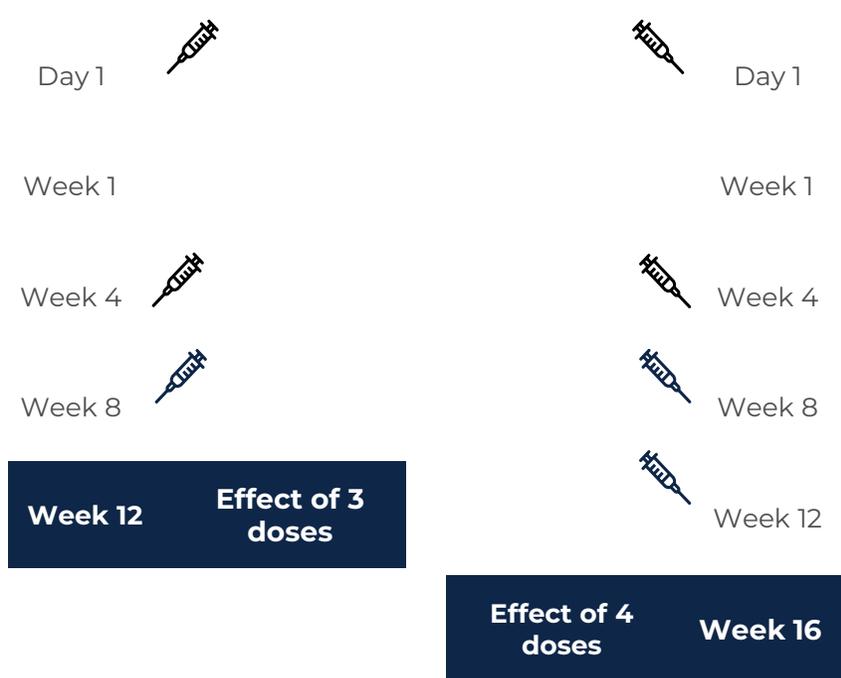
Anti-IL-6
Vamikibart

For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

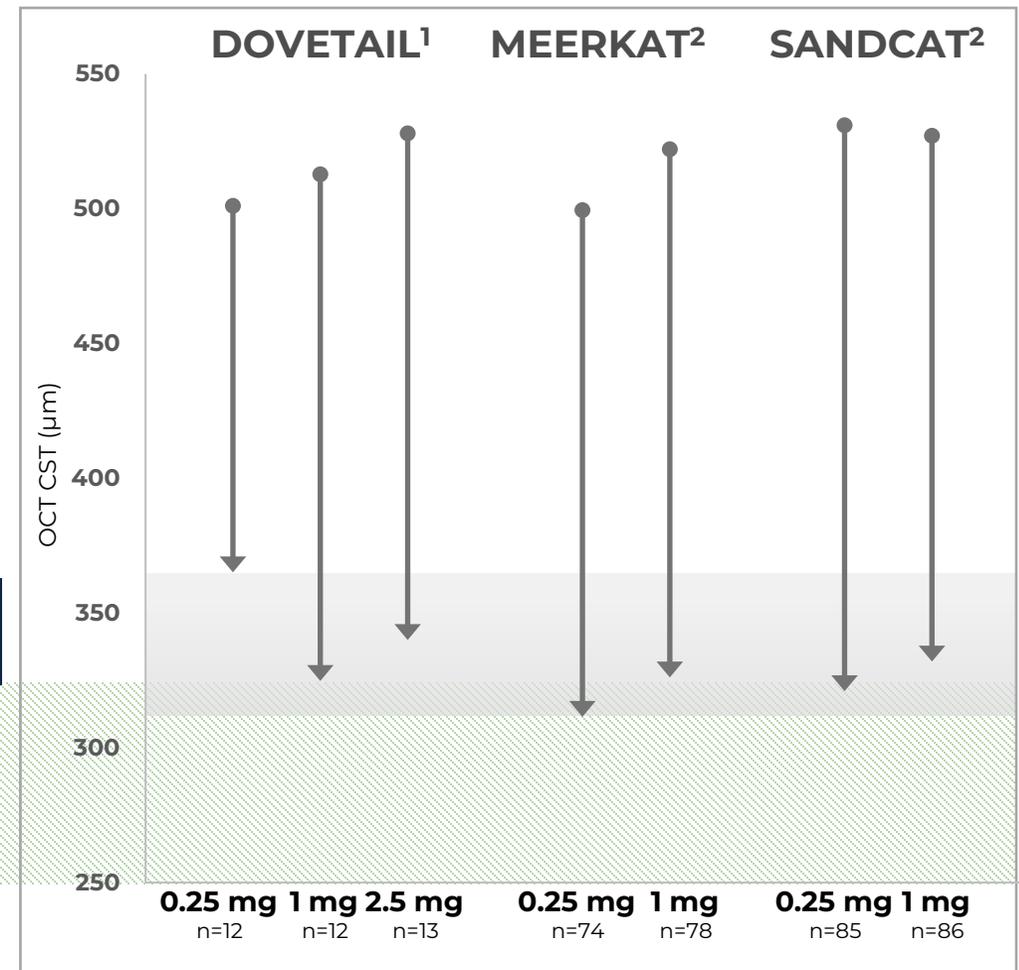
Mean Change in OCT CST and Absolute CST



Anti-IL-6, VEGF trap
KSI-101



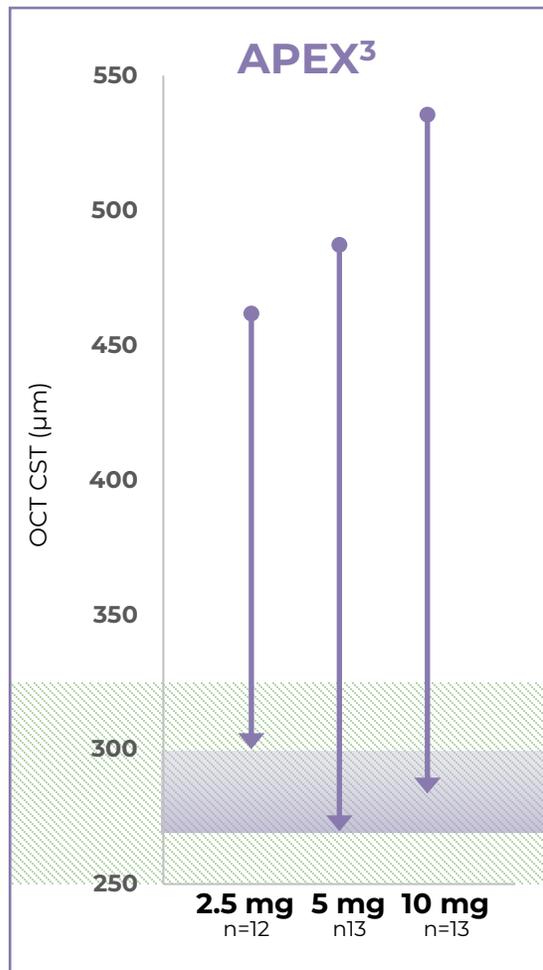
Continued dosing with KSI-101 provides further deepening into the dryness corridor



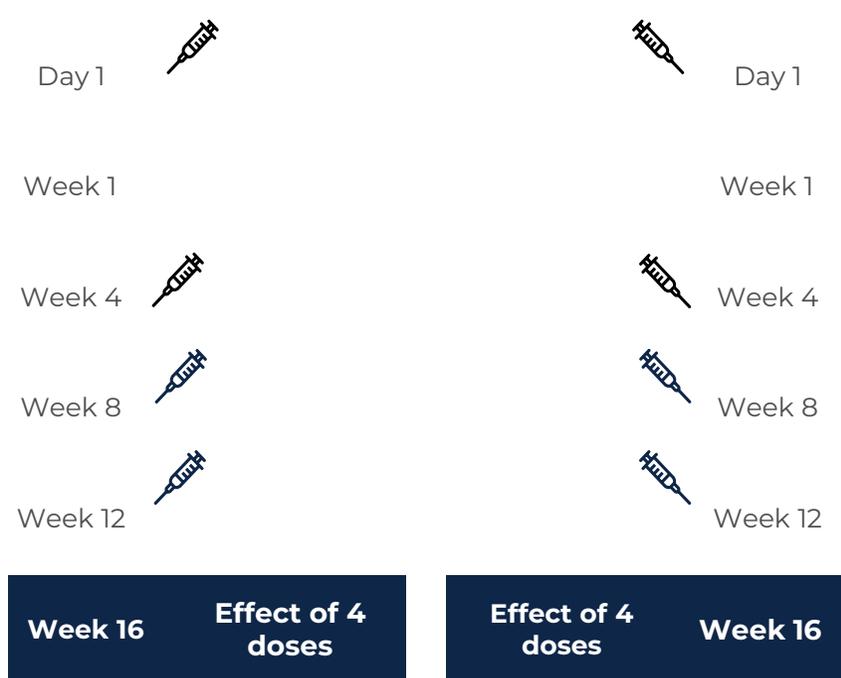
Anti-IL-6
Vamikibart

For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

Mean Change in OCT CST and Absolute CST



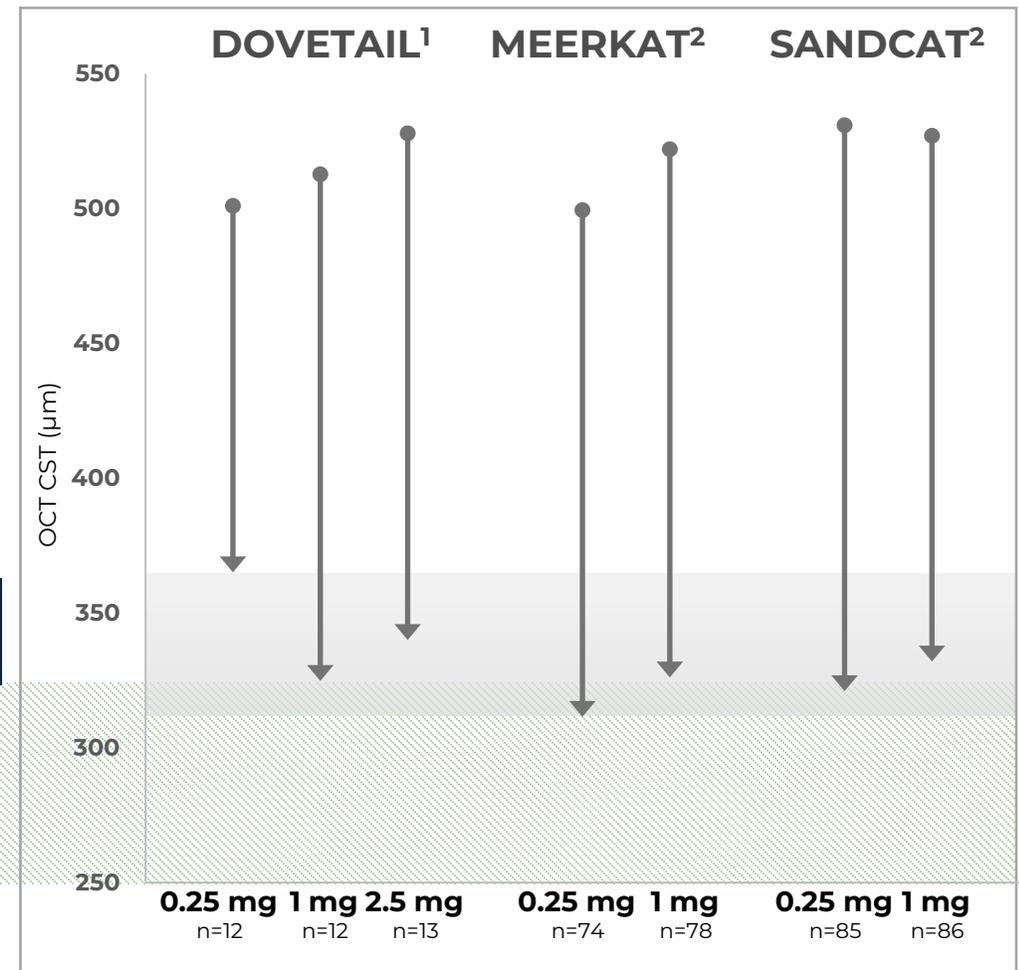
Anti-IL-6, VEGF trap
KSI-101



Week 16 Effect of 4 doses Week 16 Effect of 4 doses

**Dryness
(CST 250 – 325)**

Dual inhibition of IL-6 and VEGF seems to provide a synergistic drying effect



Anti-IL-6
Vamikibart

For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

A vertical strip on the left side of the slide shows a microscopic view of cells, likely retinal cells, with a light blue and white color scheme. The cells are arranged in a somewhat regular pattern, with some showing distinct nuclei and cytoplasm.

Macular Edema Secondary
to Inflammation

**PEAK and PINNACLE
KSI-101 pivotal
program**

Phase 3 pivotal program in MESI – PEAK and PINNACLE Study Design

	Fixed monthly dosing						Individualized dosing						
Weeks	D1	4	8	12	16	20	24	28	32	36	40	44	48
KSI-101 5 mg													
KSI-101 10 mg													
Sham													

Primary endpoint

- KSI-101 5 mg injection
- KSI-101 10 mg injection
- Sham injection
- Individualized treatment (PRN)
- Sham PRN

Key inclusion criteria

- Macular edema secondary to inflammation (MESI)
- Diagnosis of active or inactive non-infectious intraocular inflammation, acute or chronic.
- Active leakage as evidenced by fluorescein angiogram
- OCT CST of ≥ 320 microns
- BCVA score ≤ 78 and ≥ 25 (~20/25 to 20/320 Snellen)

Individualized treatment criteria (Week 24-44)

- Increase in OCT CST ≥ 50 μm compared to the lowest previous measurement, or
- OCT CST > 320 μm

PEAK and PINNACLE are actively enrolling

Phase 3 pivotal program in MESI – PEAK and PINNACLE Study Design

Weeks	Fixed monthly dosing						Individualized dosing						
	D1	4	8	12	16	20	24	28	32	36	40	44	48
KSI-101 5 mg	■	■	■	■	■	■	◐	◐	◐	◐	◐	◐	
KSI-101 10 mg	■	■	■	■	■	■	◐	◐	◐	◐	◐	◐	
Sham	●	●	●	●	●	●	◐	◐	◐	◐	◐	◐	

- KSI-101 5 mg injection
- KSI-101 10 mg injection
- Sham injection
- ◐ Individualized treatment (PRN)
- Sham PRN

Primary endpoint

Rescue Treatment allowed from Week 4 to Week 44, when warranted

Rescue criteria

- BCVA decrease ≥ 15 letters *and* CST worsening by ≥ 100 μm from Day 1, due to MESI.
- Worsening of inflammation by ≥ 2 grade levels in anterior chamber cells and/or vitreous haze; or progression to grade 4.
- The intraocular inflammation complications in the Study Eye did not improve and require rescue treatment to prevent irreversible loss of vision per Investigator's judgment.

PEAK and PINNACLE are actively enrolling

Phase 3 pivotal program in MESI – PEAK and PINNACLE Study Design

	Fixed monthly dosing						Individualized dosing						
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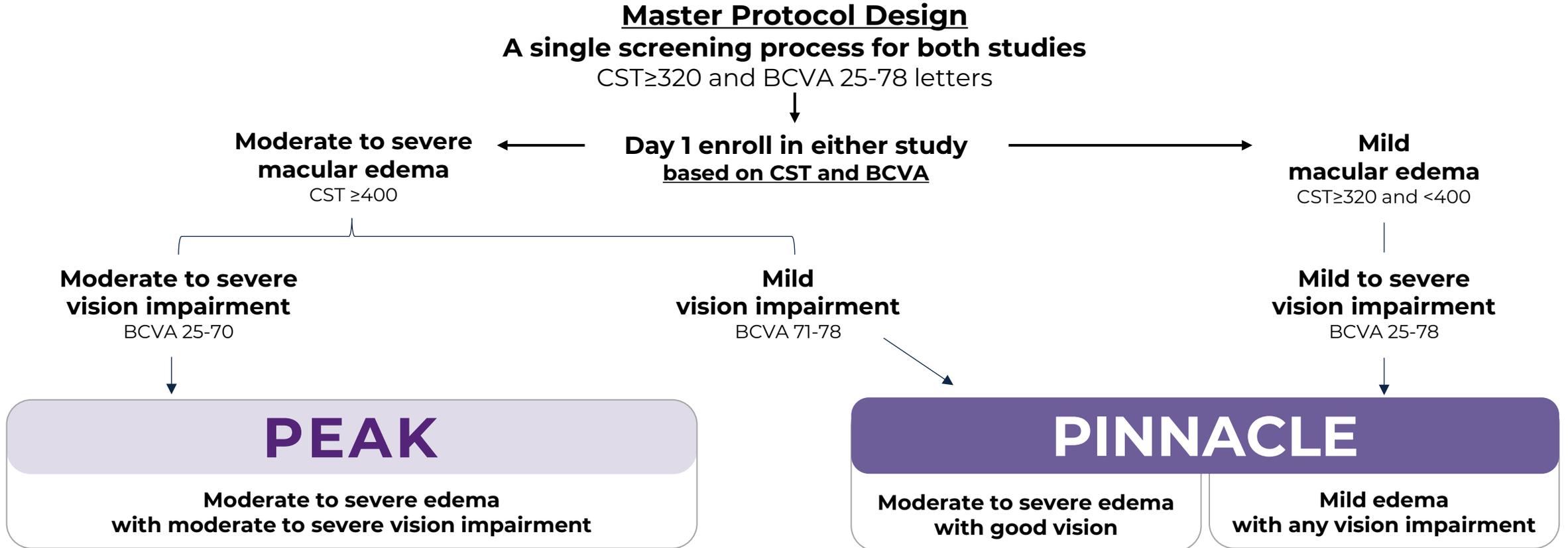
- KSI-101 5 mg injection
- KSI-101 10 mg injection
- Sham injection
- Individualized treatment (PRN)
- Sham PRN

Primary endpoint

	PEAK	PINNACLE
Primary endpoint	BCVA change from baseline to the average of Week 20 and 24	
Key secondary endpoint	Proportion of patients in whom BCVA had improved by ≥15 letters from baseline to 24 weeks	

PEAK and PINNACLE are actively enrolling

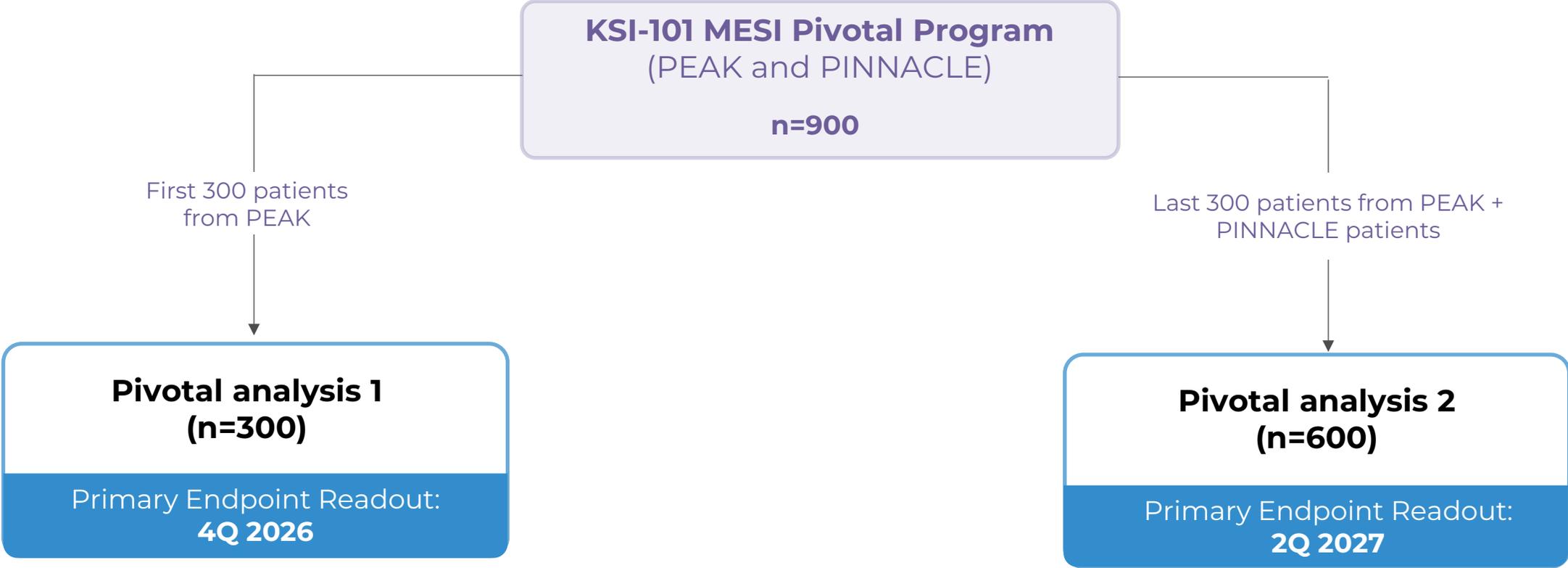
PEAK and PINNACLE – Key question: are these identical studies?



Based on the MESI patient population studied in APEX, two distinct and complementary sub-populations will be studied in PEAK and PINNACLE, allowing both studies to run concurrently in all study sites and covering a wide spectrum of MESI patients

We have increased the size of the KSI-101 MESI pivotal program

- Patient enrollment faster than expected
- No major changes to expected timelines are anticipated
- Pivotal program aligned with FDA in type C meeting





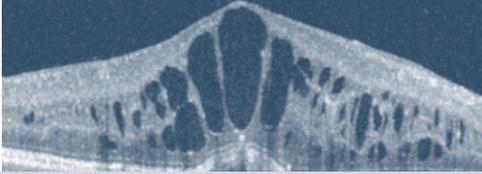
KSI-101 Commercial Opportunity

Problem Statement:

Ocular inflammation is the 4th leading cause of blindness among the working-age population in the developed world

MESI, a serious complication of ocular inflammation, is the primary contributor to vision loss and blindness in this group

A serious vision threatening disease



M

A **chronic** retinal condition that occurs when inflammation (past or present) disrupts the **blood-retinal barrier** triggering **edema**, and leading to **serious risk of vision loss**

MESI can be triggered by a variety of underlying **systemic and local autoimmune conditions** and represents a **prevalence of 450,000** patients in the US of which **~300,000 are trial-eligible**

>150,000 initial KSI-101 addressable MESI patients in the US



E

The **initial KSI-101 addressable population** is the **>150,000 MESI patients in the US** who are **contraindicated** for intraocular steroid injections, at risk of glaucoma or cataract development due to **chronic steroid treatment** or are **refractory from chronic steroid treatment**

Over time, avoiding the consequences of longer-term steroid use together with SOC matched efficacy can support a **first-line therapeutic of choice** profile for KSI-101

Differentiated potent high strength dual MOA



S

KSI-101 is in development for the treatment of MESI:

- Locally injected
- Potent
- High-dose
- First-in-class anti-IL6 and VEGF trap
- Bispecific protein

Potential for a simplified and safer MESI patient journey



I

Historically, physicians segment and treat patients based on presumed etiology, resulting a lengthy **trial and error patient journey**

KSI-101 offers the promise of **simplifying the MESI diagnostic and patient journey**, upgrading the **ophthalmologist** as the primary ocular caregiver, and improving patient outcome across etiologies

Excitement from physician interviews



Interviewed physicians are **impressed by the KSI-101 safety and efficacy product profile**,

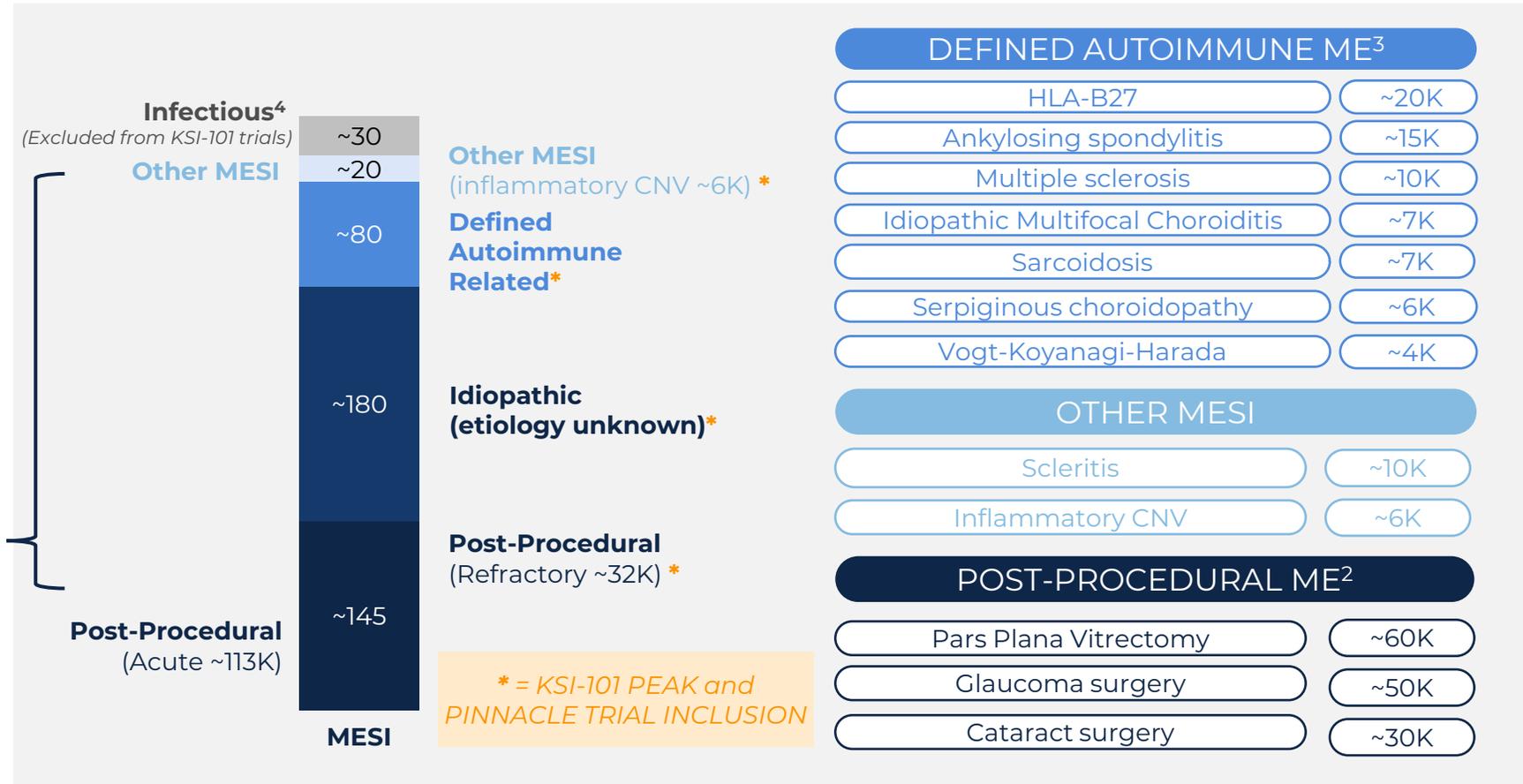
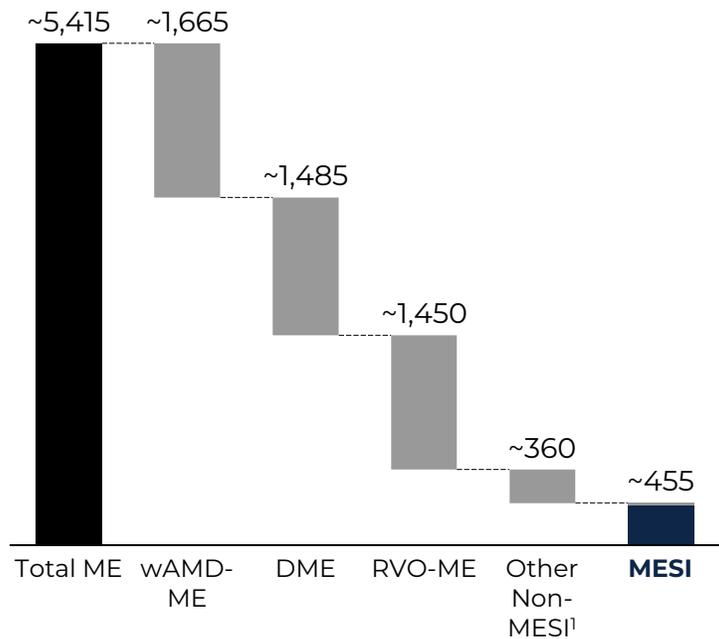
noting that it **lacks the serious negative consequences** of chronic steroid use, including glaucoma and cataract,

but has strong efficacy across etiologies **on par with current intraocular steroids**

MESI affects ~450,000 US patients (~300,000 trial eligible), estimated by two methodologies and often reported by etiology (defined autoimmune, idiopathic, procedural, infectious and other)

U.S. MESI EPIDEMIOLOGY – METHOD 1

(2025) Thousands of patients



~455K out of 5.4M ME patients in the U.S. may be classified under the “MESI” definition, ~298K meet KSI-101 trail inclusion

Note: ¹ Other Non-MESI includes epiretinal membrane ME and RP-associated ME; ² Non-exhaustive list, other types include laser photocoagulation, DMEK, DSEAK-ME, IOL replacement, scleral buckling, pneumatic retinopathy; ³ Non-exhaustive list, others include birdshot choroidopathy, multiple evanescent white dot syndrome, punctate inner choroidopathy, and more; ⁴ Non-exhaustive list, others include bartonella sp. Tuberculosis, endophthalmitis; DME = Diabetic Macular Edema; ERM = Epiretinal Membrane; HLA = Human Leukocyte Antigen, ME = Macular Edema; MESI = Macular Edema Secondary to Inflammation; RP = Retinitis Pigmentosa; RVO = Retinal Vein Occlusion; UME = Uveitic Macular Edema; wAMD = Wet Age-Related Degeneration

KSI-101 initial addressable population is greater than 150,000 MESI patients in the US

KSI-101 ADDRESSABLE POPULATION

of Patients



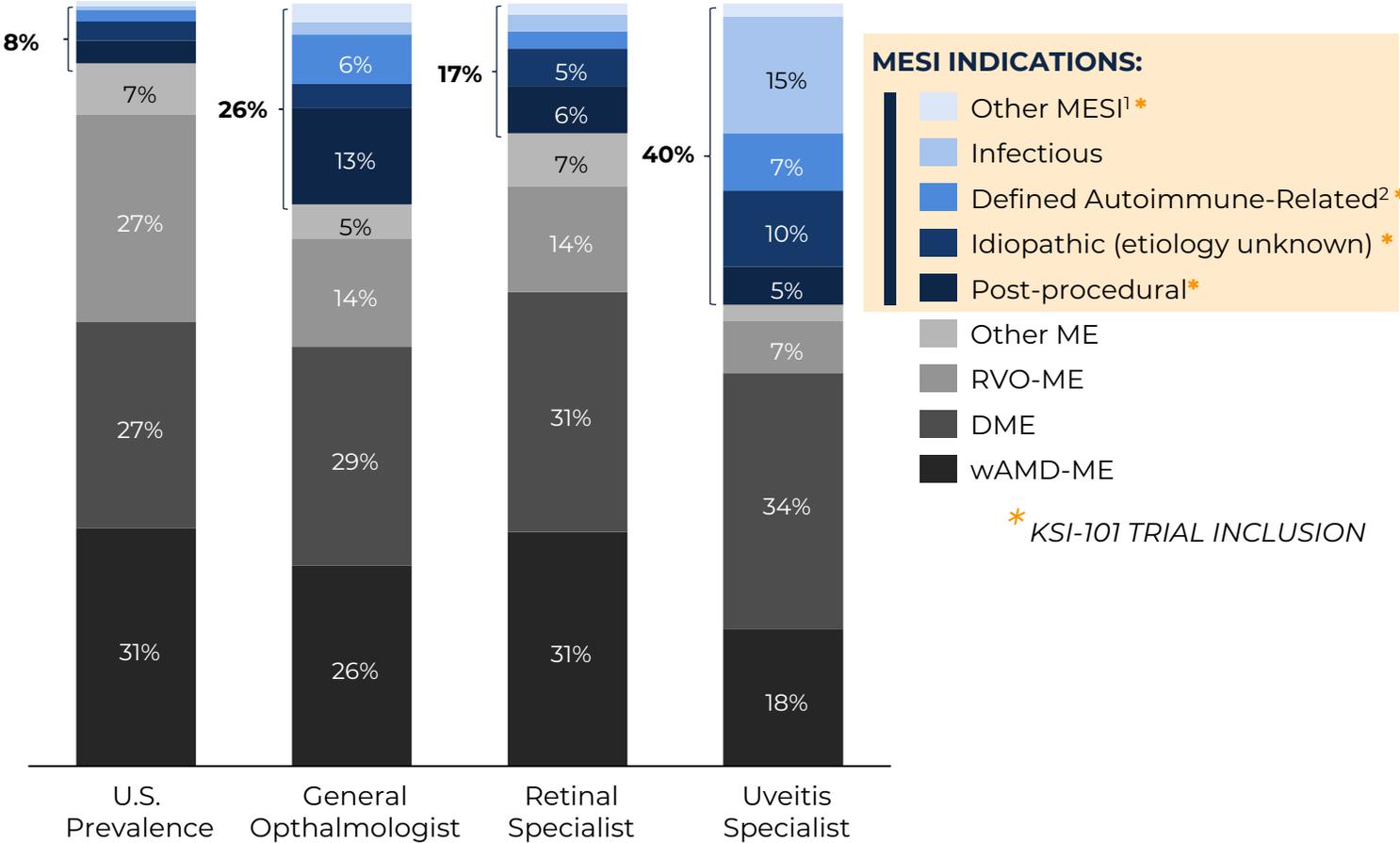
Note: ¹ First line includes eye drops or systemic immunosuppressants. Eyedrops prescribed by physicians includes steroidal and non-steroidal anti-inflammatory drops; IO = Intraocular; ME = Macular Edema; MESI = Macular Edema Secondary to Inflammation;
 Source: Bellocq et al., *BMJ* (2014); Birnbaum et al., *JAMA Ophthalmol.* (2011); Erden et al., *Ocular Immunol. Inflamm.* (2019); Kao et al., *A.A. Ophthalmol.* (2022); Expert Interviews (JUN 2025); MUST Research Group, *Ophthalmol.* (2019); Schallhorn et al., *Am. J. Ophthalmol.* (2018)

Most MESI patients are in the care of General Ophthalmologists and Retinal Specialists; complex cases are referred out to Uveitis Specialists

17% of retina specialists' current case load is MESI today

OPHTHALMOLOGY PRACTICE BREAKDOWN – METHOD 2

% of ME patients by patient subtype across physician types



- **General ophthalmologists** act as the referral gatekeepers, being the most common physician subtype (~18K in the U.S.)
- **Retinal specialists** (~3K in the US) tend to treat a diverse set of macular edema patients (~17% of their current case load is MESI today)
- **Uveitis specialists** are rare (~200 in the U.S.) but are the most familiar with MESI subtypes as it makes up ~40% of their practice

Notes: ¹Other causes can include RP, scleritis, neoplasms, drug-induced, and trauma-induced; ²Non-exhaustive drivers include sarcoidosis, systemic lupus erythematosus, scleroderma, rheumatoid arthritis, Behcet's disease, Vogt-Koyanagi-Harada disease, and others; trauma-induced; DME = Diabetic Macular Edema, ERM = Epiretinal Membrane, ME = Macular Edema, MESI = Macular Edema Secondary to Inflammation, RP = Retinitis Pigmentosa, RVO = Retinal Vein Occlusion, wAMD = Wet Age-Related Degeneration
 Source: Expert Interviews (JUN 2025); Review of Optometry (2024); Ho and Avery, *Retina Today* (2025); Tsui et al., *J Acad Ophthal* (2022)

We are a precommercial, retina-focused biotech on the move

Wholly Owned

KSI-101

- Robust end-of-study results from Phase 1b APEX
- MOA validated by scientific community
- Phase 3 PEAK and PINNACLE topline data expected in 4Q 2026 and 2Q 2027
- Commercial opportunity of 150,000+ initial addressable patients with headroom

Tarcocimab & KSI-501

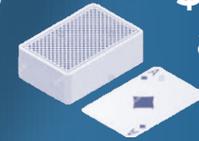
- Science-based “heavyweights”
- Tarcocimab: targeting BLA mid-2026 in wet AMD, RVO and diabetic retinopathy
- KSI-501: bispecific ABC[®] may be even better!

Pipeline, Digital Health, Manufacturing

- KSI-102, KSI-103: bispecifics for inflammation
- Duets for glaucoma and geographic atrophy
- VETi: AI headsets for commercial leadership
- URSUS: commercial manufacturing

A potent reason to believe in Kodiak

2 options in the \$15+ billion anti-VEGF market



Accelerating our technology and pipeline leadership