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THE OPHTHALMOLOGY MEDICINES COMPANY

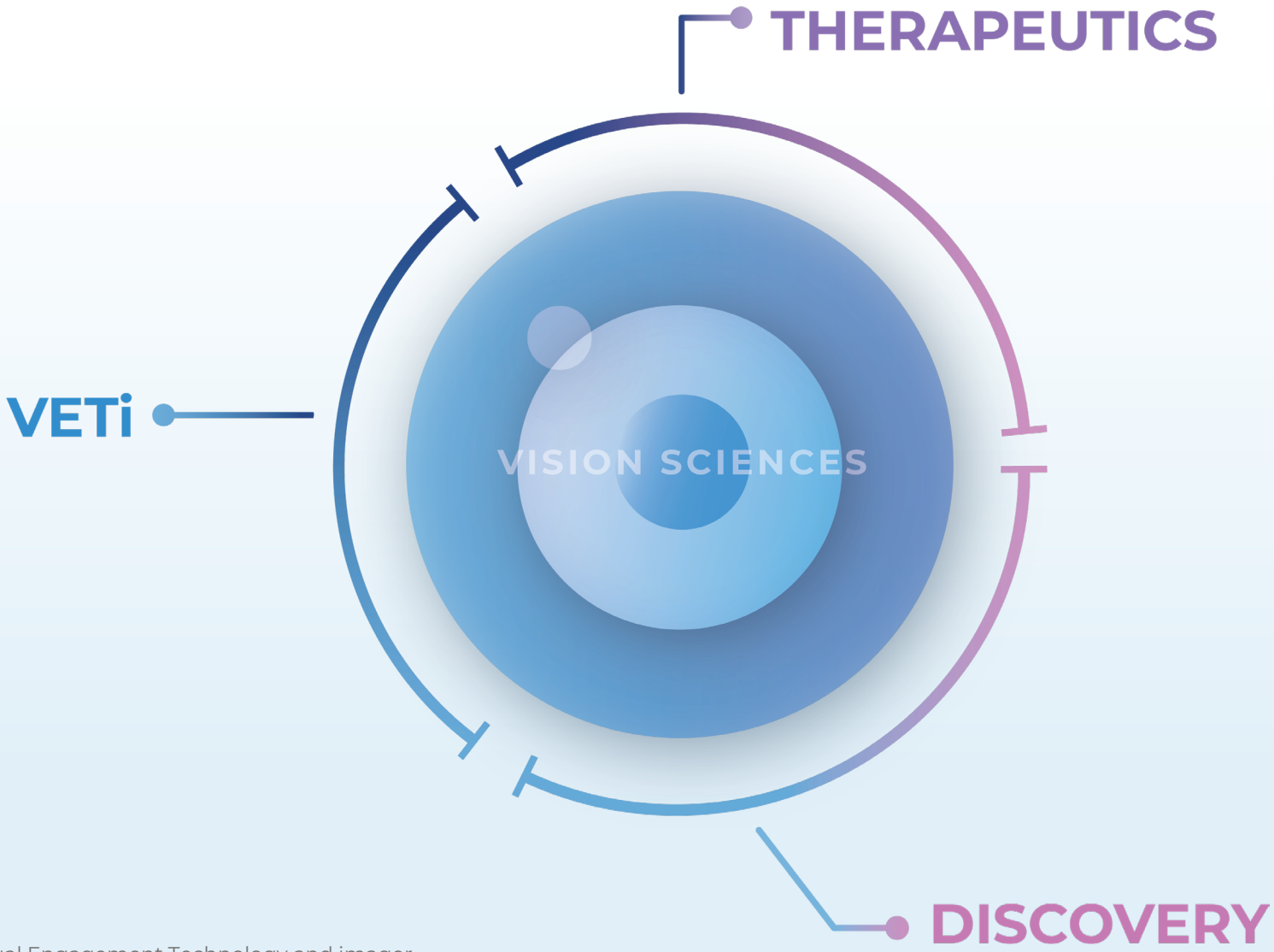
Jefferies Global Healthcare Conference

June 2026

Forward-Looking Statements

This presentation may contain forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 and other federal securities laws, including statements regarding Kodiak's plans, commitments, aspirations and goals related to Kodiak's drug candidates. Actual results may differ materially from those indicated in the forward-looking statements as a result of various important factors which are discussed in the section entitled "Risk Factors" in Kodiak's most recent periodic report filed with the U.S. Securities and Exchange Commission ("SEC") as well as discussions of potential risks, uncertainties, and other important factors in Kodiak's subsequent filings with the SEC. All information in this presentation is as of the date presented, and Kodiak undertakes no duty to update such information unless required by law.

Kodiak is emerging into a fully integrated Vision Sciences company

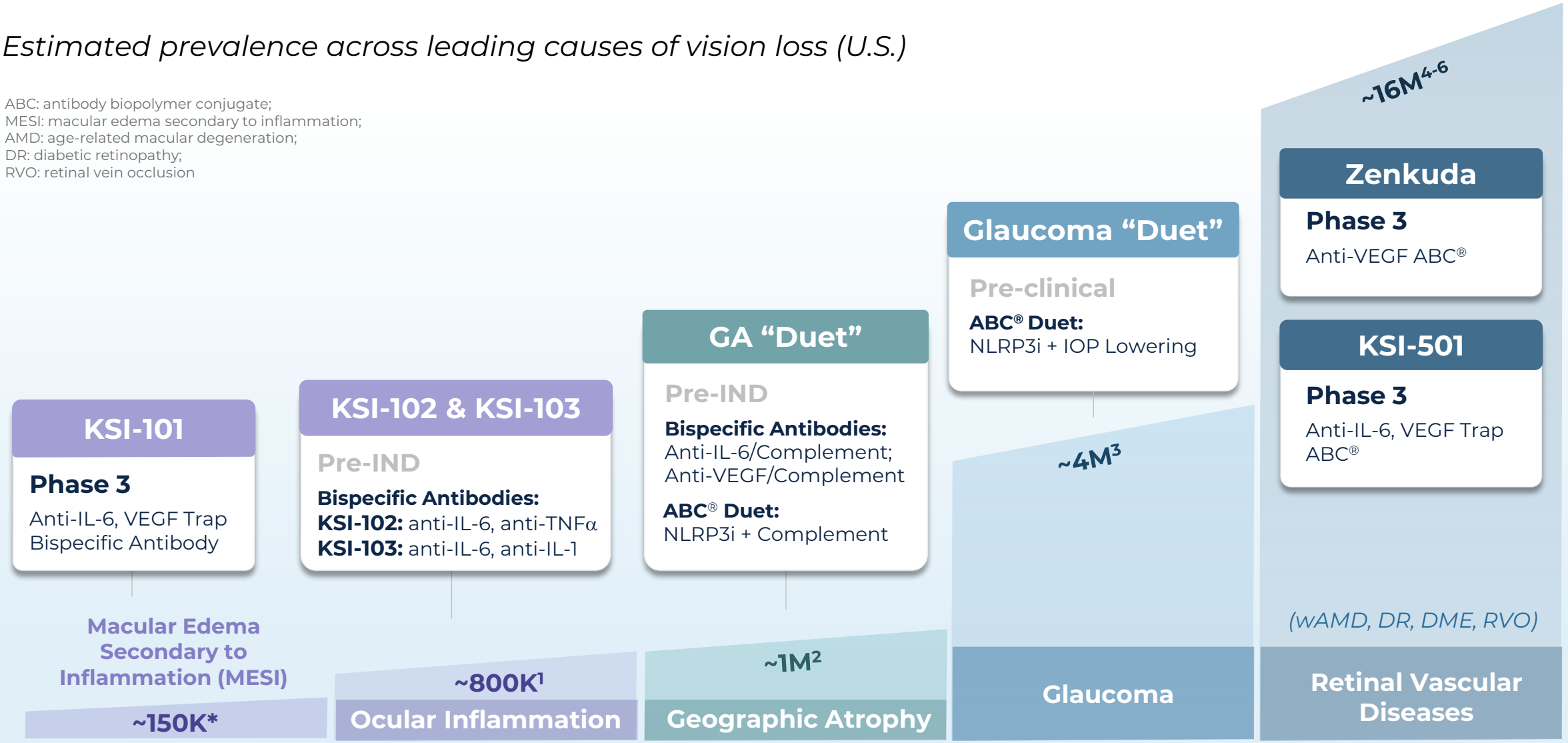


VETi: Visual Engagement Technology and imager

We have both a late-stage clinical portfolio and an emerging pipeline that span multiple retina mechanisms of action, indications and drug types

Estimated prevalence across leading causes of vision loss (U.S.)

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

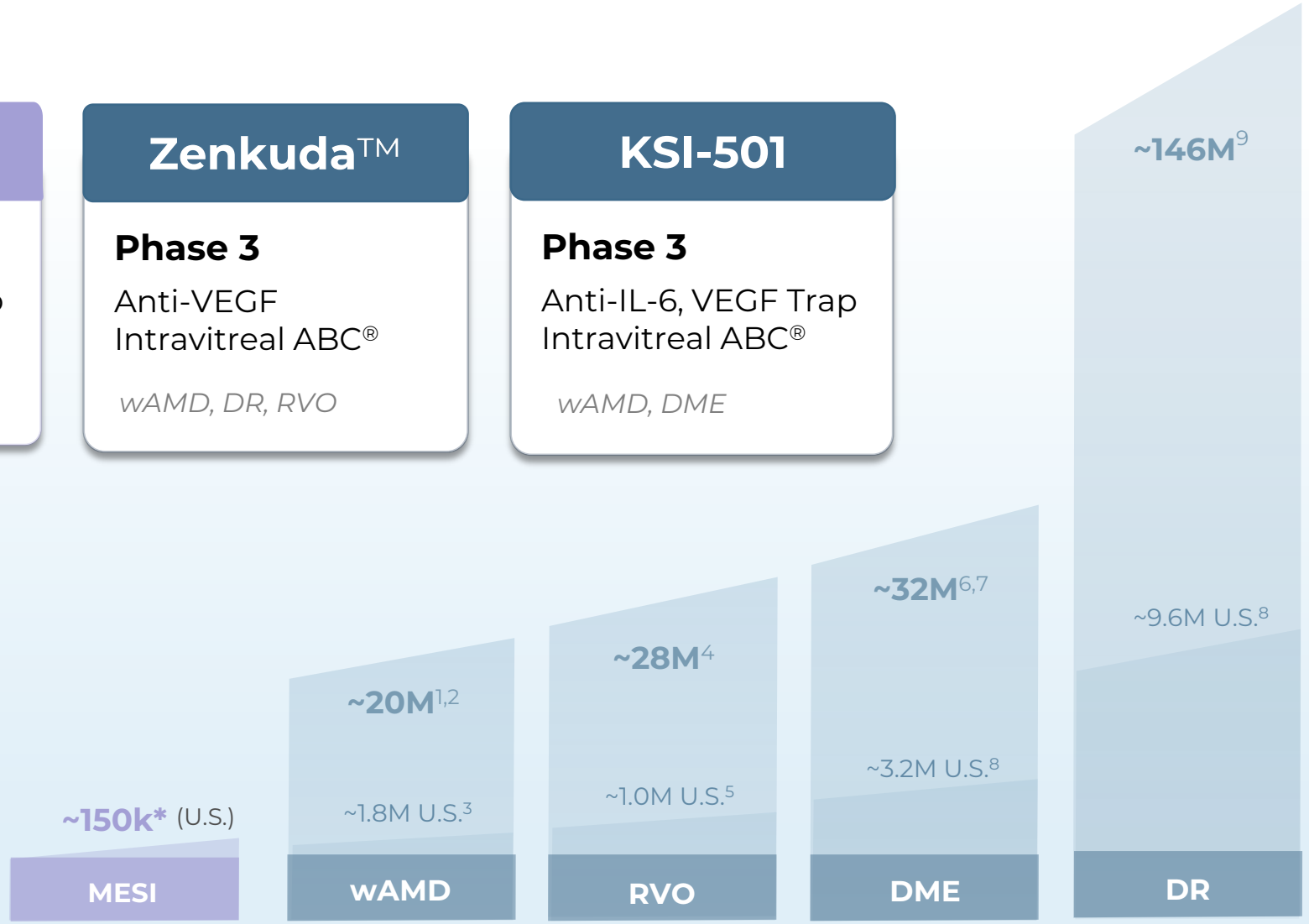


*KSI-101 initial addressable population in MESI. Kodiak Data on File and shared Investor R&D Day July 2025.

1. Nila Kirupaharan et al. *Invest. Ophthalmol. Vis. Sci.* 2024;65(7):6510. 2. Wong WL, et al. *Lancet Glob Health* 2014;2:e106–116. 3. Ehrlich JR et al. *JAMA Ophthalmol.* 2024;142(11):1046–1053. 4. Market Scope. 2024 Retinal Pharmaceuticals Market Report. 5. Roche epidemiology data 2025. 6. Kalva, P et al. *Baylor University Medical Center Proceedings*, 36(3), 335–340

Our three late-stage clinical assets target leading causes of vision loss

KSI-101	Zenkuda™	KSI-501
Phase 3	**Phase 3**	**Phase 3**
Anti-IL-6, VEGF Trap Intravitreal Protein	Anti-VEGF Intravitreal ABC®	Anti-IL-6, VEGF Trap Intravitreal ABC®
MESI	*wAMD, DR, RVO*	*wAMD, DME*



ABC: antibody biopolymer conjugate;
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DME: diabetic macular edema
DR: diabetic retinopathy

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1. Fleckenstein M, et al. *JAMA.* 2024;331(2):147–157. 2. Boopathiraj N, et al. *Mayo Clinic Proceedings: Innovations, Quality & Outcomes*, 2024; 8, 364-374. 3. Market Scope. 2024 Retinal Pharmaceuticals Market Report. 4. Song, P., et al. (2019). *Journal of Global Health.* https://doi.org/10.7189/jogh.09.010427. 5. Kalva, P et al. *Baylor University Medical Center Proceedings*, *36*(3), 335–340. 6. The Diabetes Atlas, Accessed May 2026. 7. James H.B. et al. *Survey of Ophthalmology,* 2022;67(4): 1244-1251. 8. Roche epidemiology data 2025. 9. WHO, 2019, World Report on Vision Executive Summary.

We are gathering speed: all three clinical programs are in Phase 3 with topline data expected in 2026 and Zenkuda is BLA ready

KSI-101

Anti-IL-6, VEGF trap
bispecific protein

Safe and potent control of MESI

- **Positive** Phase 1b results in MESI
- Phase 3 PEAK and PINNACLE studies **actively enrolling**
- PEAK topline data 4Q2026

Zenkuda

Anti-VEGF **ABC® biologic**

Strong immediacy and industry-leading durability

- **4 successful pivotal studies**
- **BLA-ready in DR, RVO, wAMD***
- Phase 3 DAYBREAK study in wAMD with topline data 3Q2026
- BLA filing expected in 2026

KSI-501

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

Differentiated efficacy, strong immediacy and industry-leading durability

- Differentiated efficacy being explored in the Phase 3 DAYBREAK study in wAMD with topline data 3Q2026
- Phase 3 study in DME planned 3Q2026

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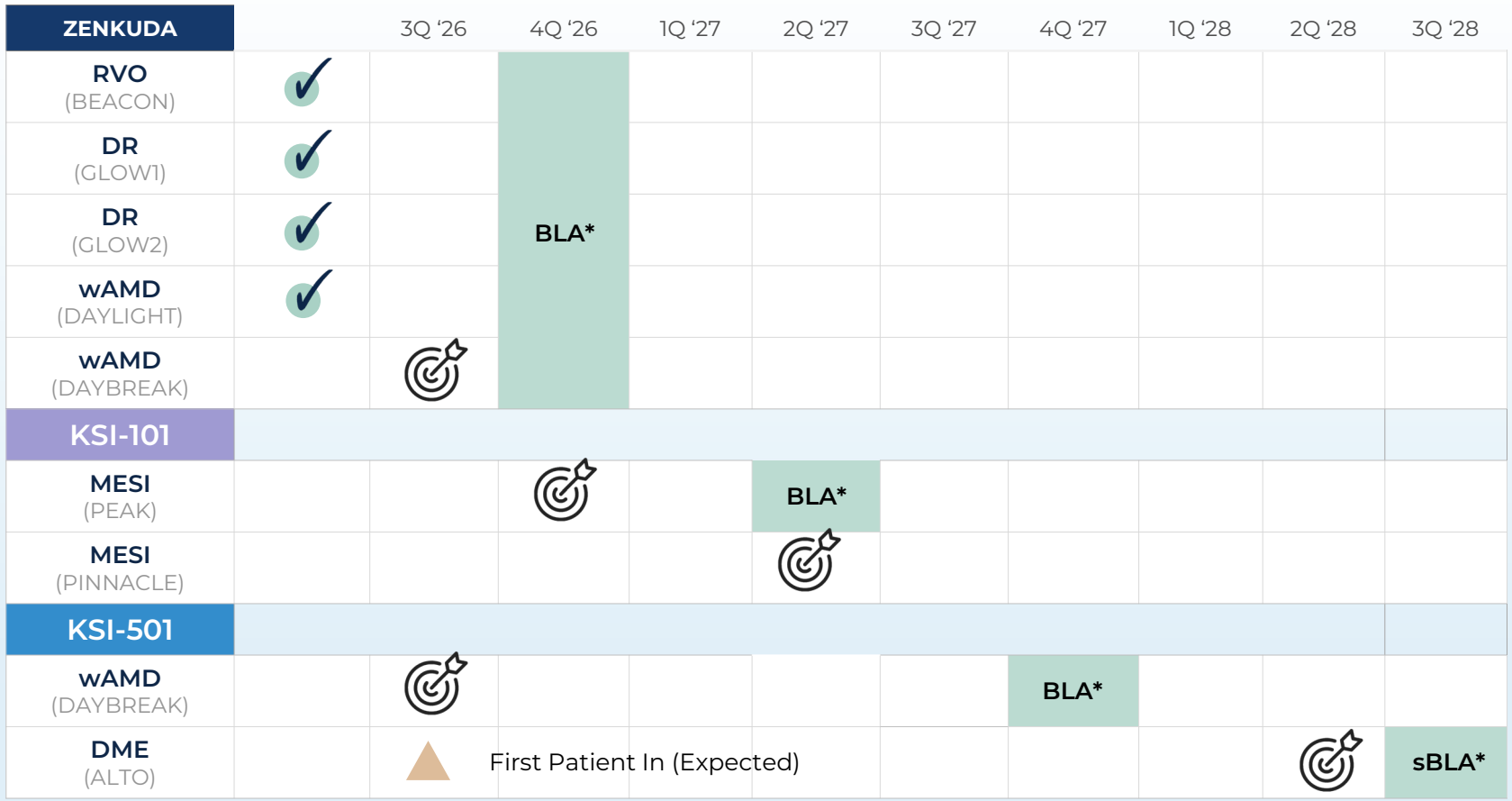
*Based on the positive Phase 3 GLOW1 and GLOW2 studies in diabetic retinopathy, the BEACON study in retinal vein occlusion and the DAYLIGHT study in wet AMD
ABC: antibody biopolymer conjugate; DR: diabetic retinopathy; RVO: retinal vein occlusion; wAMD: wet age-related macular degeneration

Four Phase 3 readouts in 2026, if successful, position Kodiak to file 3 licensing applications (BLA) in 2026 and 2027

- 4 successful pivotal studies completed
- 3 additional topline data readouts expected in 2026
- **3** Licensing applications (BLA) expected in 2026 and 2027



Upcoming catalysts (expected)

- **DAYBREAK** topline data expected 3Q 2026; licensing application in 4Q 2026

- Pivotal analysis 1 (**PEAK**) expected December 2026; potential licensing application in 1H 2027
- Pivotal analysis 2 (**PEAK+PINNACLE**) expected 2Q 2027

- **DAYBREAK** topline data expected 3Q 2026
- Phase 3 **ALTO** study in DME First Patient In expected 3Q 2026

*Timing reflects current expectations; BLA timing pending discussions with regulatory bodies, subject to change
RVO: retinal vein occlusion; DR: diabetic retinopathy; AMD: age-related macular edema; DME: diabetic macular edema; MESI: macular edema secondary to inflammation; BLA: biologics license application; sBLA: supplemental biologics license application; DAYBREAK: NCT06556368; PEAK: NCT06990399; PINNACLE: NCT06996080

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

News Release

Lonza

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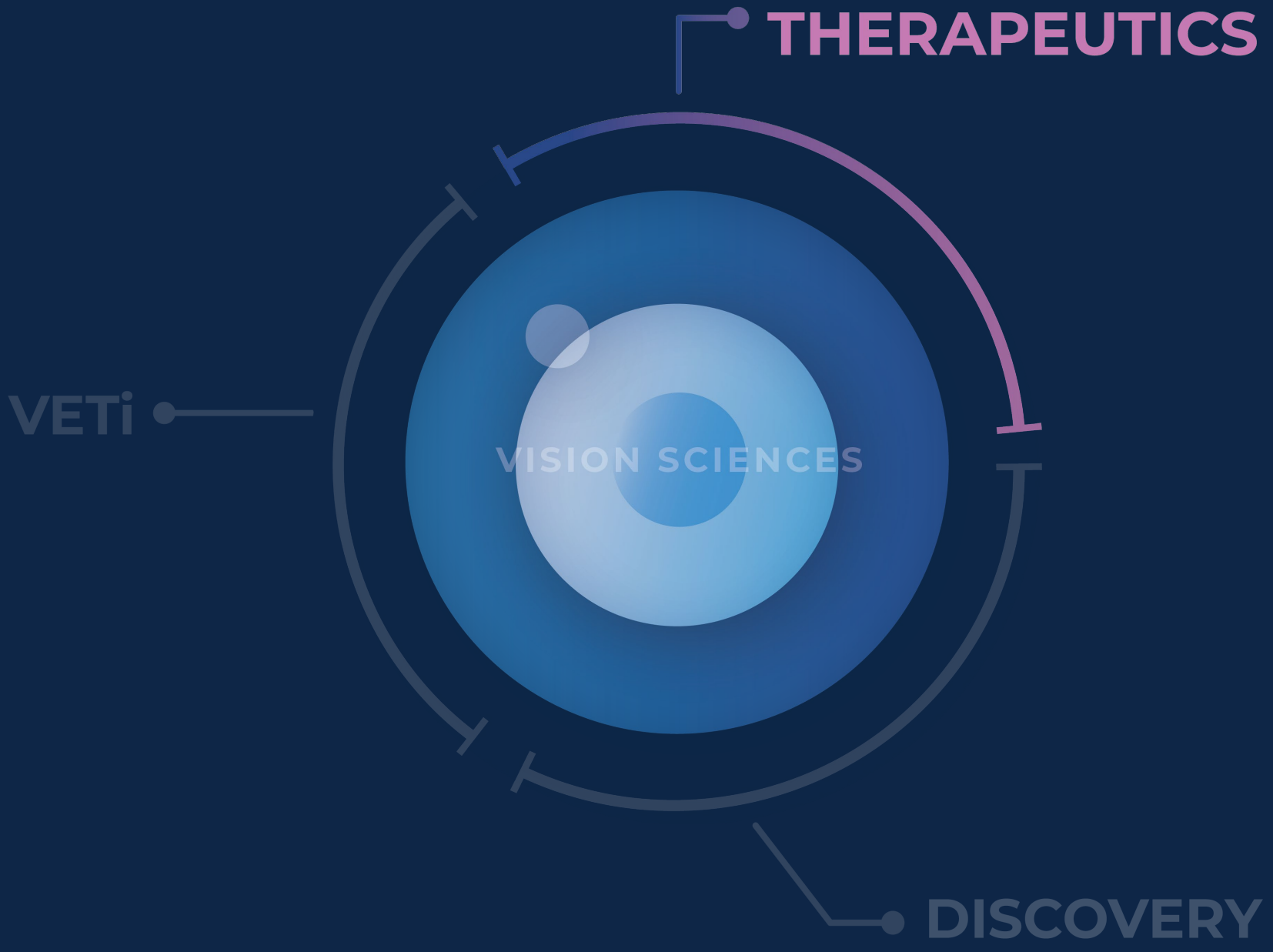
Grand Opening of Kodiak Sciences' Purpose-Built Bioconjugation Facility to Support Potential Commercial Manufacture of KSI-301, an Antibody Biopolymer Conjugate for Retinal Diseases

- Purpose-built bioconjugation facility in Lonza's Ibex® De
  to support the potential commercial launch of Kodiak's
  high-prevalence retinal diseases
- The opening ceremony took place on May 17, 2022 follov
  facility in March 2022

Basel, Switzerland and Palo Alto (CA), USA, 18 May 2022 – Koc
biopharmaceutical company committed to researching,
transformative therapeutics to treat high prevalence retinal dise
the opening of a new, custom-built, bioconjugation facilit
manufacturing complex in Visp (CH).

Ursus, a premium commercial manufacturing 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**



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ZENKUDA + KSI-501

Kodiak's ABC® Platform Science for 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

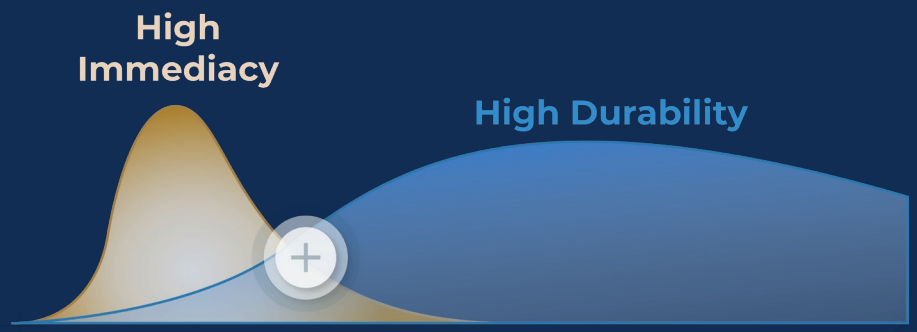
Both unconjugated protein and conjugated protein demonstrate high binding affinity and potency *in vitro*

Extended Ocular Half-Life in Animals

3x the ocular t½ of approved intravitreal biologics when measured in rabbits following an intravitreal injection

Extended Ocular Half-Life in Humans

3x the ocular t½ of faricimab when measured from aqueous humor in patients following an intravitreal injection



Powerful Immediacy via Unconjugated Antibody

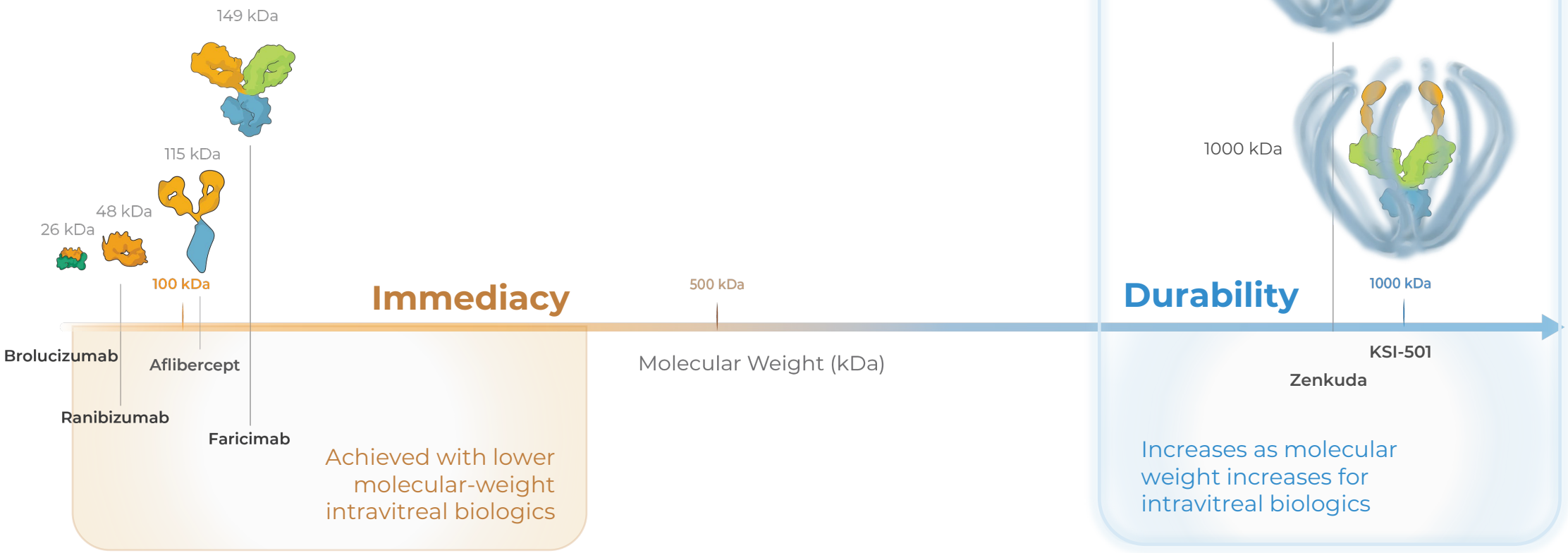
The unconjugated protein delivers a strong "pulse" of anti-VEGF inhibition during the loading phase, or to recapture control of the disease in patients whose disease has reactivated

Extended Clinical Durability via Conjugated Antibody

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

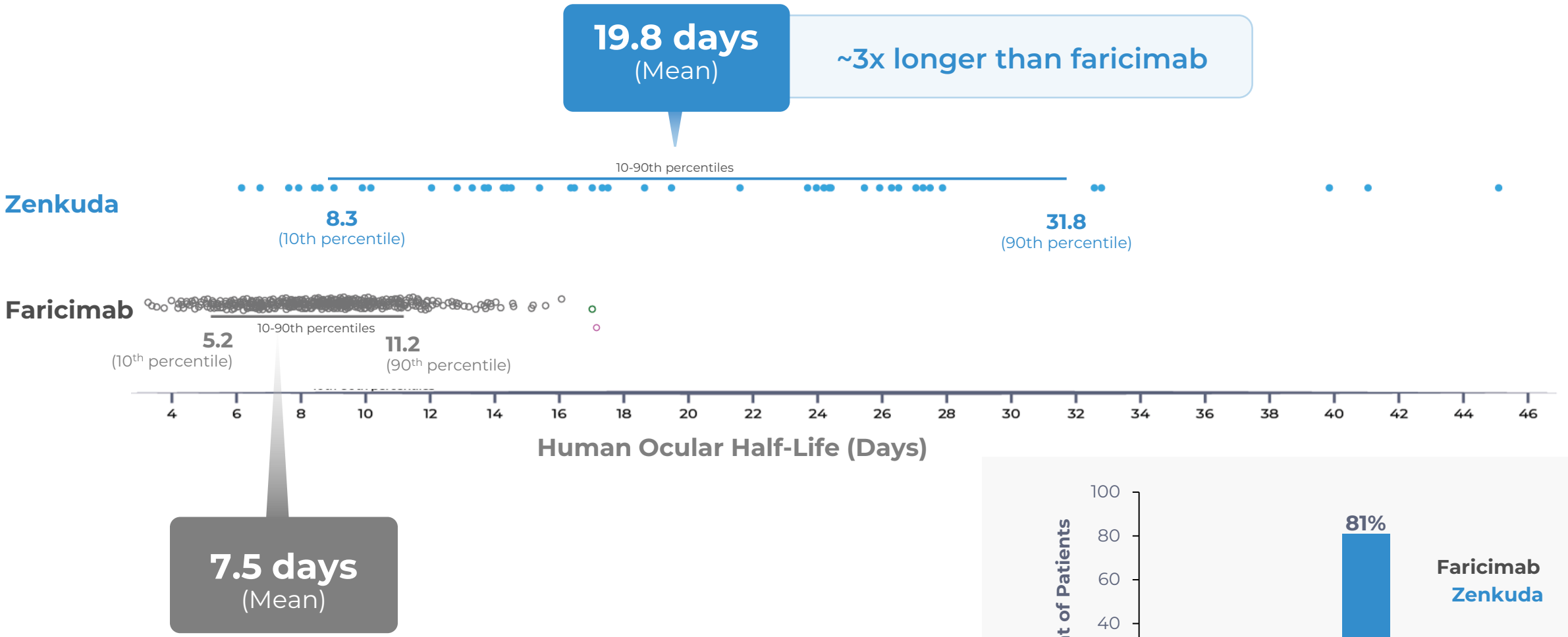
Durability: ABC® Platform-based medicines are designed for longer ocular durability

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



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

Durability: Zenkuda 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 Zenkuda from the Phase 1b study of Zenkuda in patients with wAMD, DME and RVO. Gray dots are faricimab from Genentech, Inc. PK and ER of faricimab, Report # 1105763

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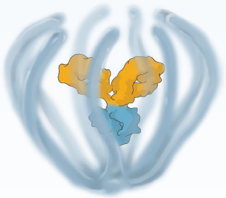
Zenkuda and KSI-501 combine unconjugated and conjugated protein, enabling high immediacy and high durability in a single biologic

Zenkuda – 5 mg

IMMEDIACY
1.0 mg
Unconjugated

+



DURABILITY
4.0 mg
ABC (conjugated)

KSI-501 – 5 mg

IMMEDIACY
1.5 mg
Unconjugated

+

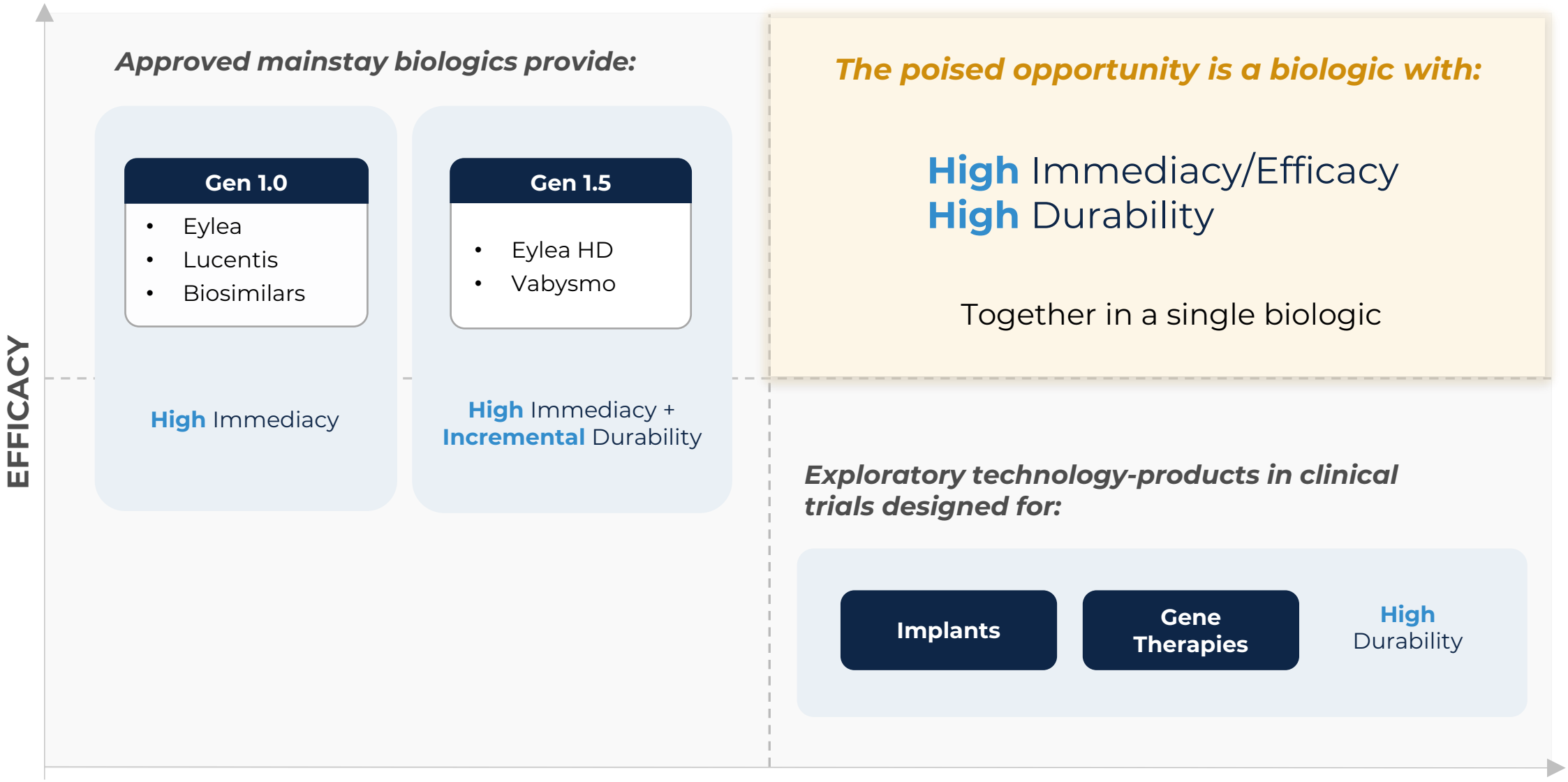
DURABILITY
3.5 mg
ABC (conjugated)

Single biologics engineered for high immediacy and high durability

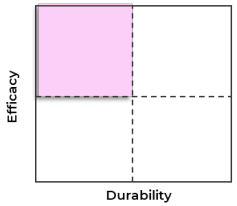
ZENKUDA + KSI-501

Retinal Vascular Diseases and the Anti-VEGF Market

There remains valuable open space in the ~$15B 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



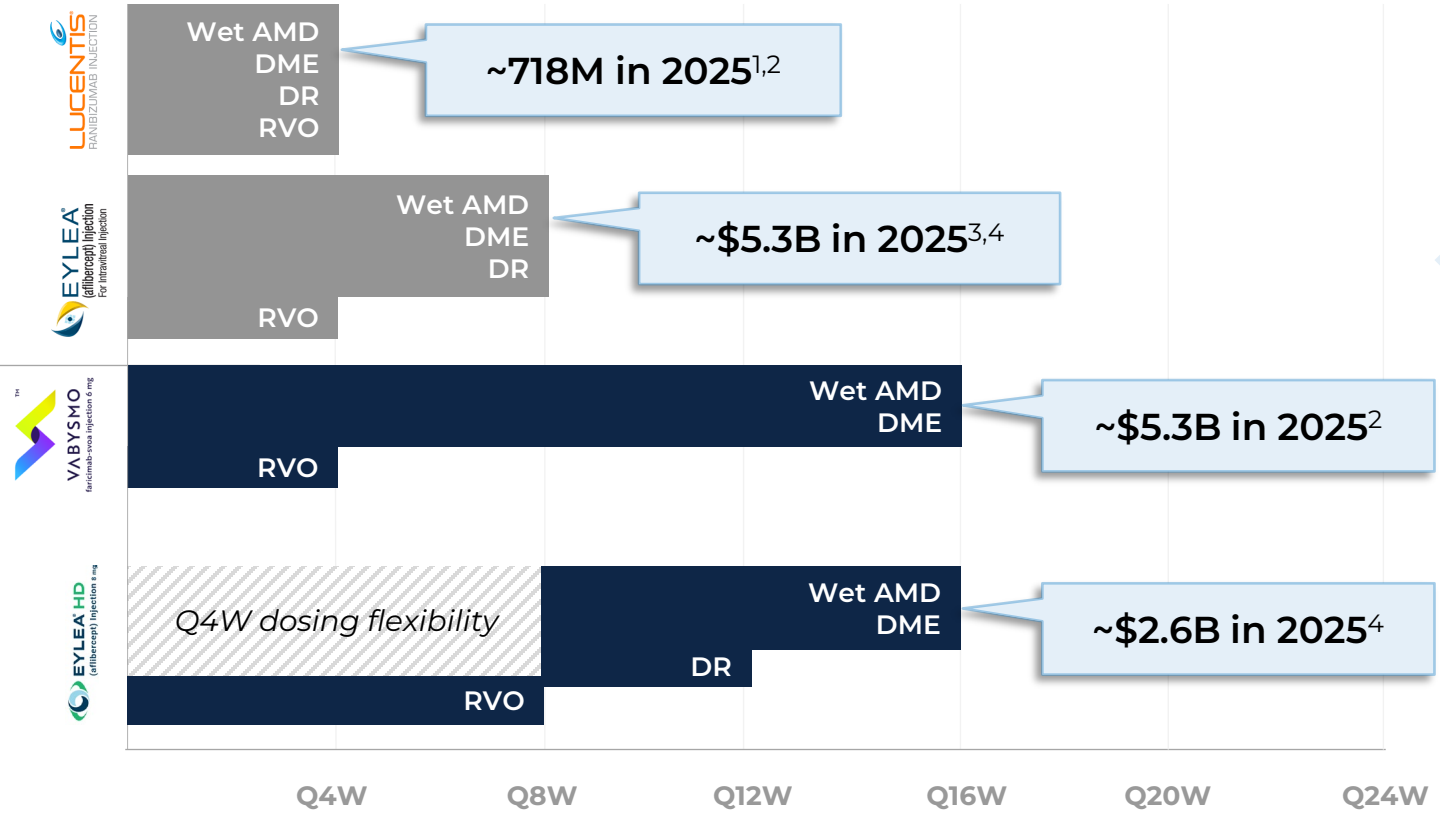
Dosing regimen per label for approved intravitreal biologics and estimated worldwide sales in 2025

Good efficacy, limited durability

Gen 1.0

Gen 1.5

Good 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

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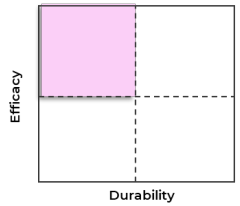
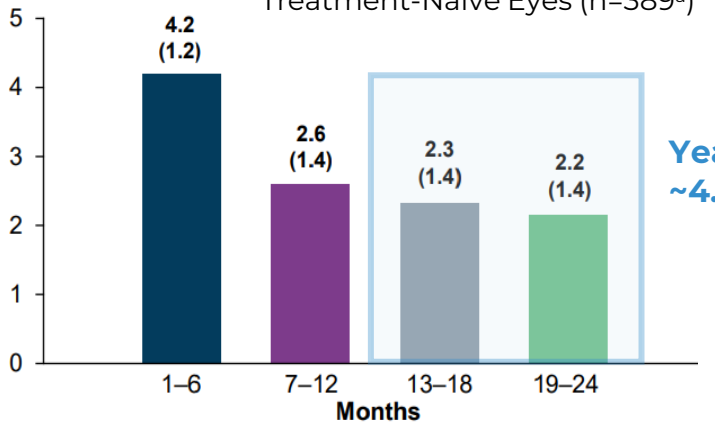
1. Novartis 2025 FY Financial Results. 2. Roche Finance Report 2025. 3. Bayer FY 2025 Investor Call, Pharma sales by product split. March 04, 2026. 4. Regeneron Corporate Presentation, January 2026.

Despite their commercial success, real-world data in wAMD 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 study[1]

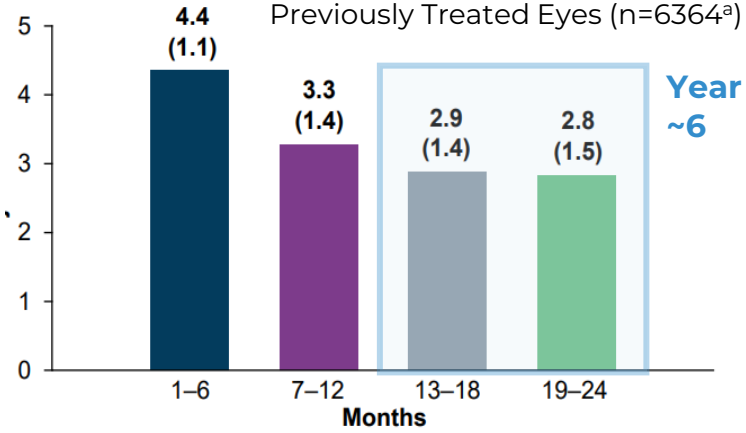
Treatment-Naïve Eyes (n=389[a])



Year 2: ~4.5

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

Previously Treated Eyes (n=6364[a])



Year 2: ~6

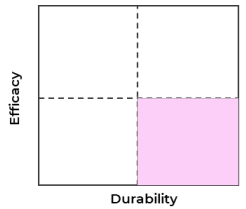
In Year 2, patients received 6.0 injections which is an average dosing **interval of every 8 weeks**

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.

1. Adapted from Durga Borkar, et al. Poster presented at ARVO 2025. Poster #3108.

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

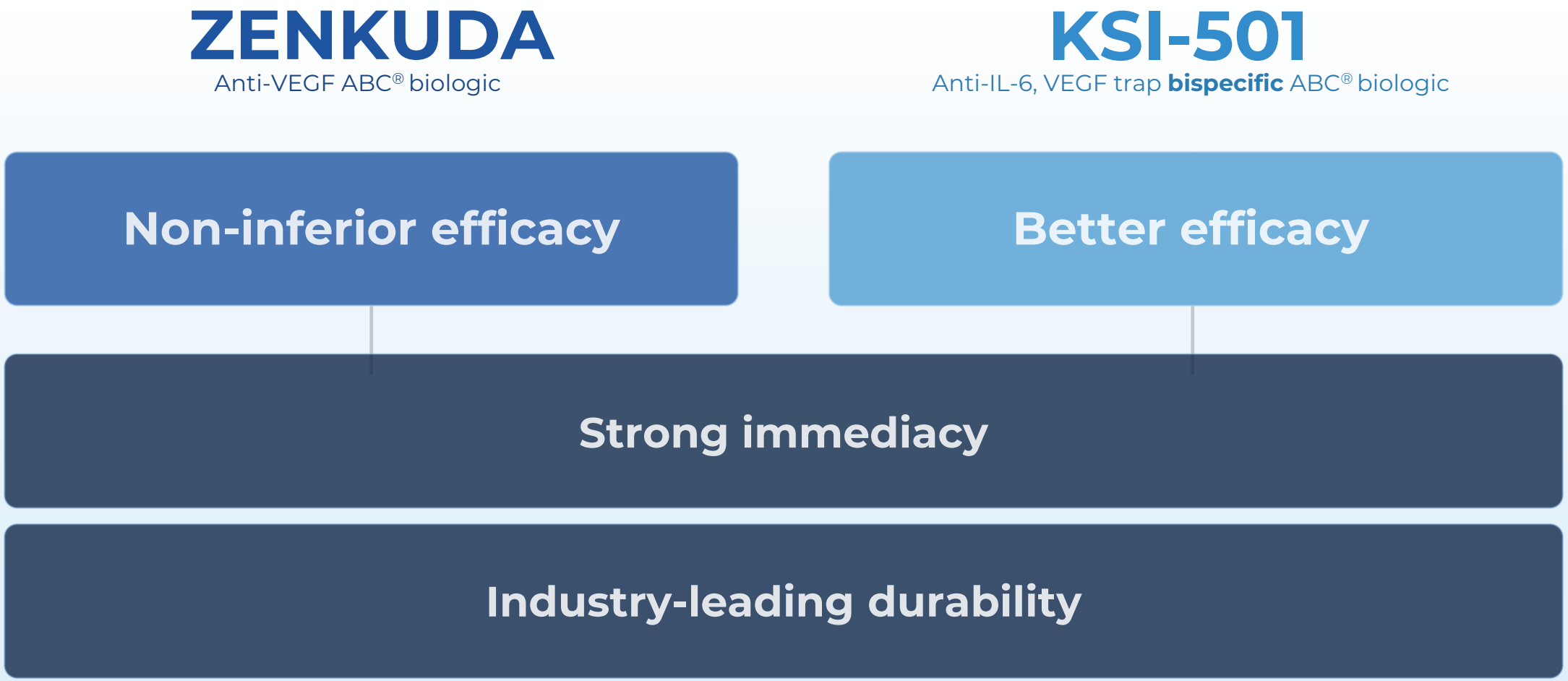
- Engineering for durability
- Onerous monitoring and inflammation control
- Long-term safety unknown

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

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**.

Zenkuda and KSI-501: Differentiated ABC platform mainstay biologics

Target product profiles



Zenkuda and KSI-501's complementary and differentiated product profiles support a broad retinal franchise

Phase 3 Study	Primary Endpoint	6-Month Durability
ZENKUDA		
RVO (BEACON)	✓	✓
DR (GLOW1)	✓	✓
DR (GLOW2)	✓	✓
wAMD (DAYLIGHT)	✓	Not Applicable
wAMD (DAYBREAK)	Non-Inferiority	✓
KSI-501		
wAMD (DAYBREAK)	Non-Inferiority	Not Applicable
DME (ALTO)	Superiority	✓

✓ Successfully completed ✓ Planned

Across 4 pivotal studies, Zenkuda demonstrated:

- Non-inferior efficacy with 6-month predominant durability
- Favorable and well-tolerated safety profile
- In GLOW1 and GLOW2, Zenkuda reduced the risk of developing a Site Threatening Complication by ≥85% with 100% of patients on 6-month dosing at Year 1

Ongoing DAYBREAK study in wAMD explores potential for immediacy and 6-month durability

- Enrollment complete; topline data expected 3Q 2026

Ongoing DAYBREAK study in wAMD explores potential for immediacy and differentiated efficacy

- Enrollment complete; topline data expected 3Q 2026

ALTO study in DME expected to start 3Q2026

Zenkuda in Retinal Vein Occlusion: Well-positioned to capture meaningful share in a large and growing >$3B RVO market

- **RVO is a meaningful and growing market**
  - **~3B** estimated RVO market[1]
    - ~20% of Eylea net sales derived from RVO in 2025[2]
  - **~1M** people in the U.S. with RVO[3]
    - Prevalence of RVO is growing globally, driven by growth in aging population
  - **~750,000** treatable with anti-VEGF

- **RVO is a chronic disease that requires long-term management**
  - Despite acute onset, RVO often requires frequent **long-term** treatment
  - Approved intravitreal biologics **require frequent injections** that can be burdensome for patients and physicians

Zenkuda is designed to address key unmet needs in RVO[4]

Strong efficacy

Zenkuda **doubled** the treatment interval relative to aflibercept at month 6 (Q8W vs. Q4W)

6-month durability

75% of patients achieved 6-month durability at Year 1 in the Phase 3 BEACON study

Fewer injections

Potential to reduce treatment burden

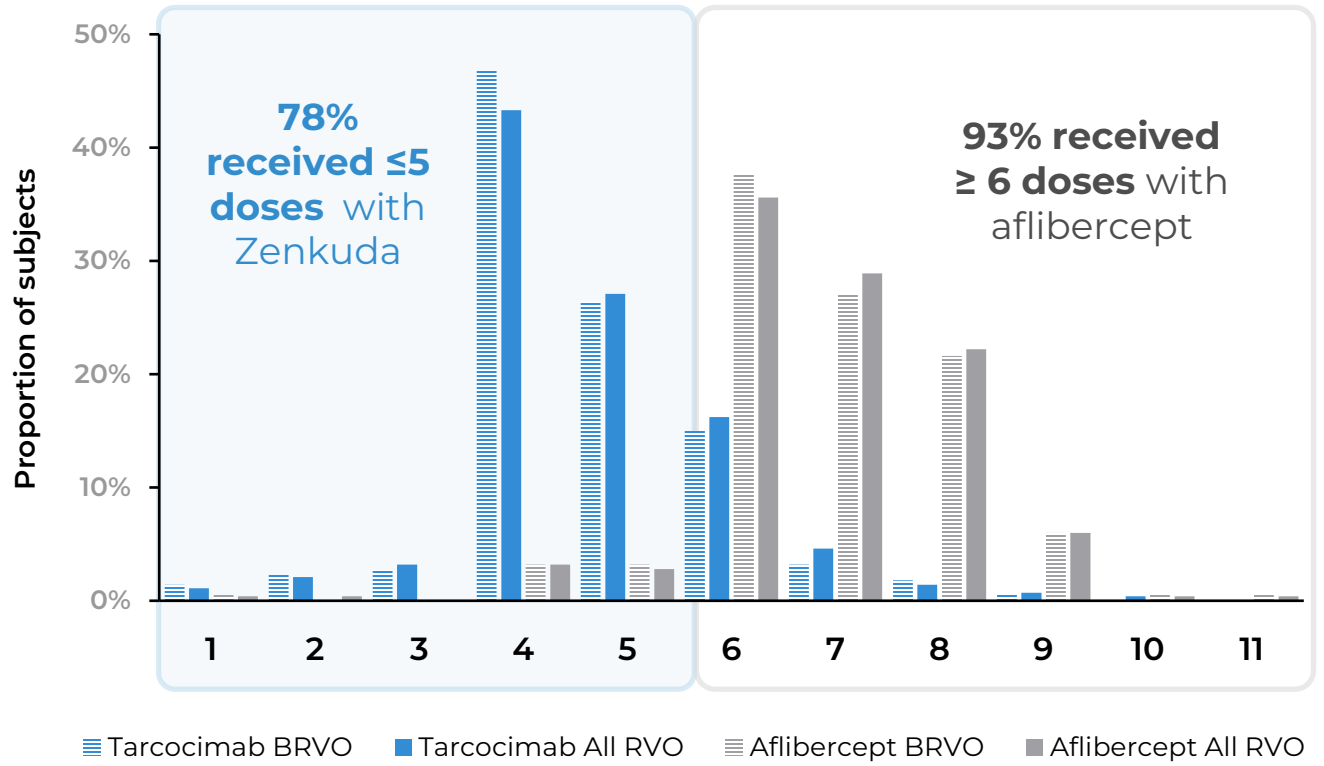
1. RVO market size from imarc: RVO Market Size, Epidemiology, In-Market drug sales, pipeline therapies, and Regional Outlook. 2. Regeneron earnings report. 3. Kalva, P et al. *Baylor University Medical Center Proceedings*, *36*(3), 335–340. 4. Based on data from the Phase 3 BEACON study in RVO.

Zenkuda in Retinal Vein Occlusion: Demonstrated high efficacy and industry-leading durability in the Phase 3 BEACON study

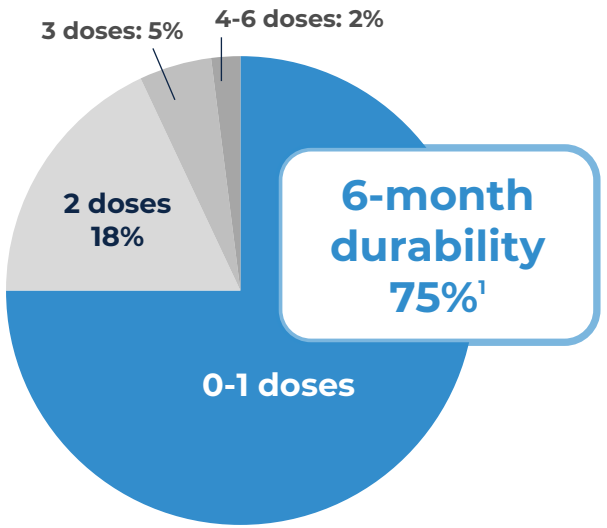
Phase 3
BEACON

- Primary Endpoint ✓
- 6-month Durability ✓

Number of injections through Year 1



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



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.

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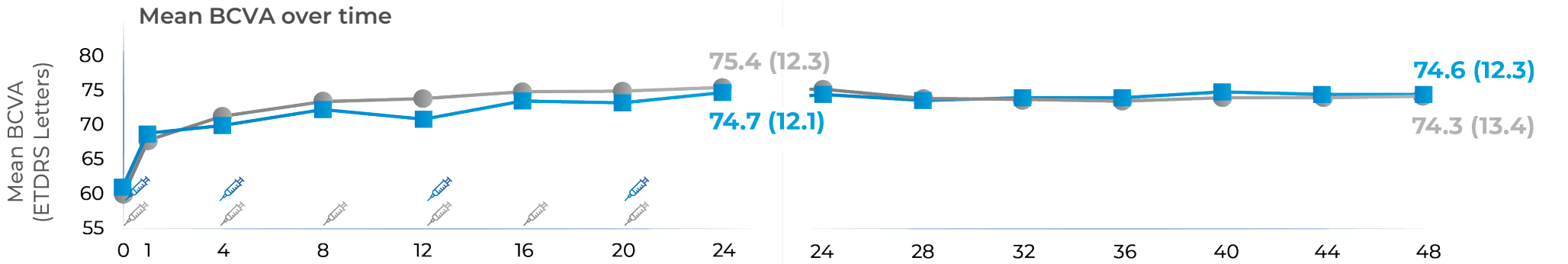
Zenkuda achieved comparable visual and anatomical outcomes in all RVO patients, irrespective of the treatment paradigm used

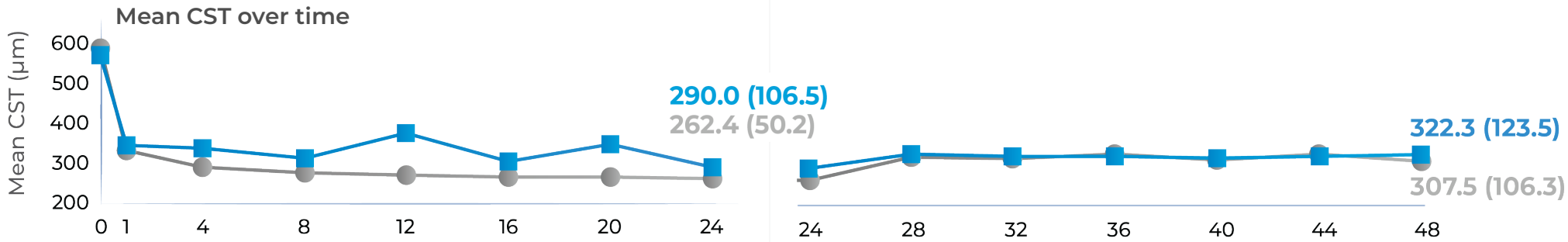
Fixed Dosing (0-24)
Doubling of treatment interval

With two fewer doses (4 vs 6),
similar visual and anatomical gains

Head-to-head Individualized Dosing (24-48)
Zenkuda vs aflibercept

75% achieved 6-month durability,
similar visual and anatomical gains





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

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.

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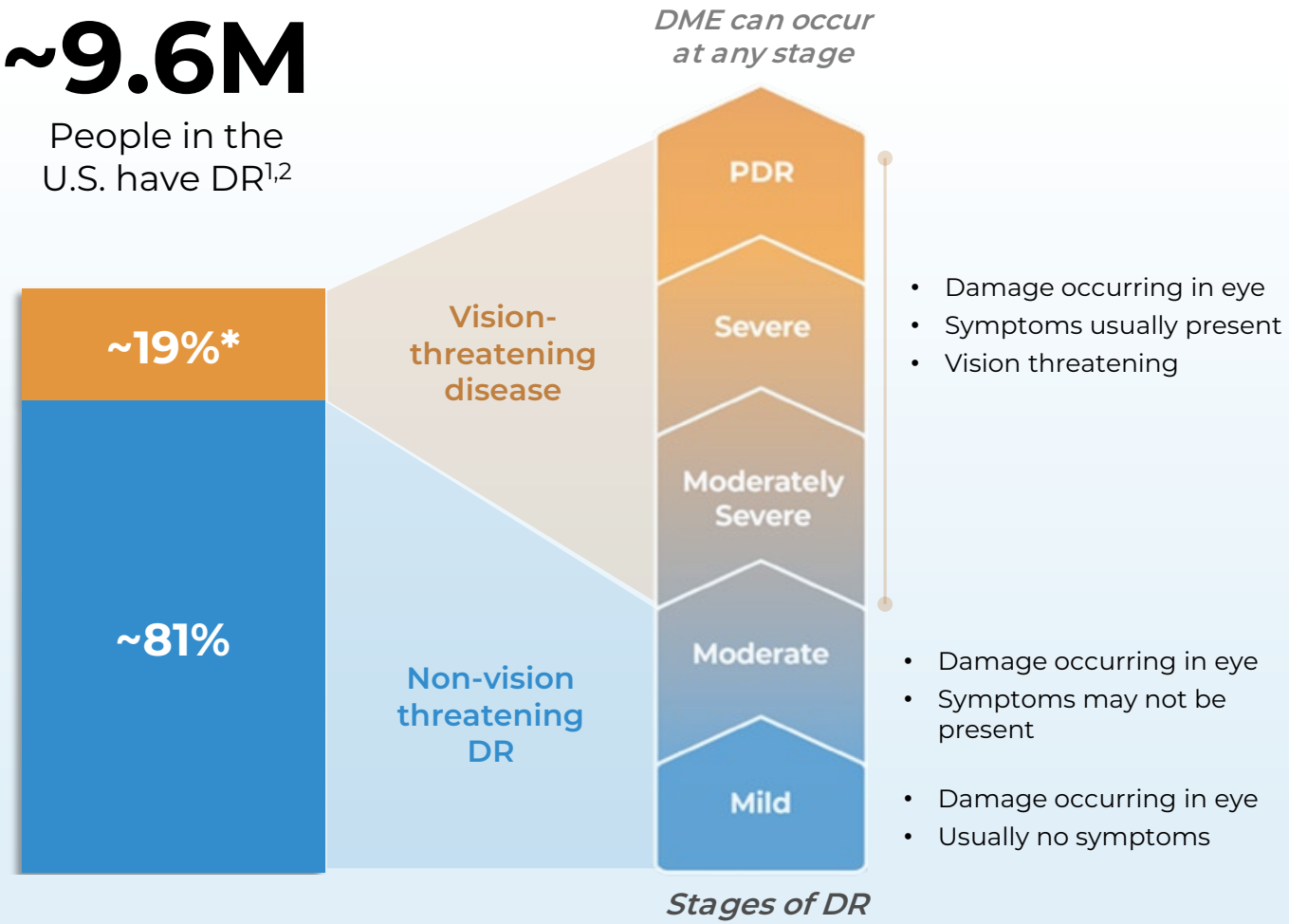
Zenkuda in Diabetic Retinopathy: A large, undertreated disease with meaningful risk progression

~9.6M

People in the U.S. have DR[1,2]



DR can progress quickly into vision-threatening PDR or DME[3]

- DME can occur at any stage, but the risk increases as DR worsens
- **50%** of moderate NPDR and **60%** of severe NPDR eyes developed vision-threatening DME by Year 4

Anti-VEGF therapy reduces the risk of PDR or DME development by ~50%[3,4]

- Compared to laser or no treatment
- Repeated anti-VEGF injections are required to maintain the benefits of therapy

*The CDC defines VTDR as severe NPDR, PDR, and/or DME

1. Roche epidemiology data from 2025. 2. CDC accessed May 13: https://www.cdc.gov/vision-health-data/prevalence-estimates/dr-prevalence.html. 3. Moshfeghi AA, et al. BMC Ophthalmol. 2024 May 31;24(1):229. 4 Maturi RK et al; DRCR Retina Network. JAMA Ophthalmol. 2021 Jul 1;139(7):701-712.

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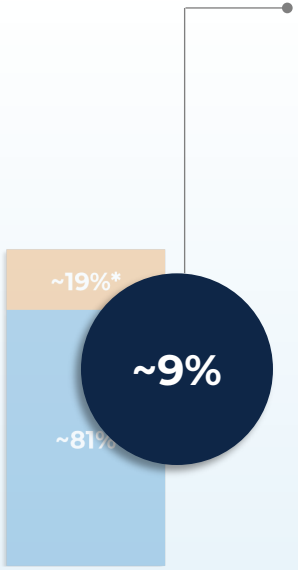
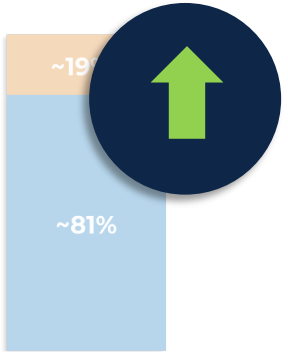
Zenkuda in Diabetic Retinopathy: A durable anti-VEGF therapy could expand treatment in DR



~9% treated with anti-VEGF therapy in 2025[1]

- While higher than previously estimated, **use remains low**
- DR is often **asymptomatic** and approved therapies **require frequent injections**
- These challenges contribute to today's "watch-and-wait" approach

A reduced treatment burden could catalyze the DR market



Zenkuda's 6-month dosing has the potential to expand anti-VEGF adoption

Fewer injections with all patients on 6-month dosing at Year 1*[2]

- Potential to increase patient compliance
- May catalyze more at-risk patients to seek earlier treatment, given risk of disease progression

Strong efficacy[2]

- Superior ≥2-step improvement in DRSS
- Reduced risk of developing sight-threatening complications by ≥85%

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*After initiation phase
1. Roche epidemiology data from 2025. 2. Based on the results from the GLOW1 and GLOW2 Phase 3 studies in DR.

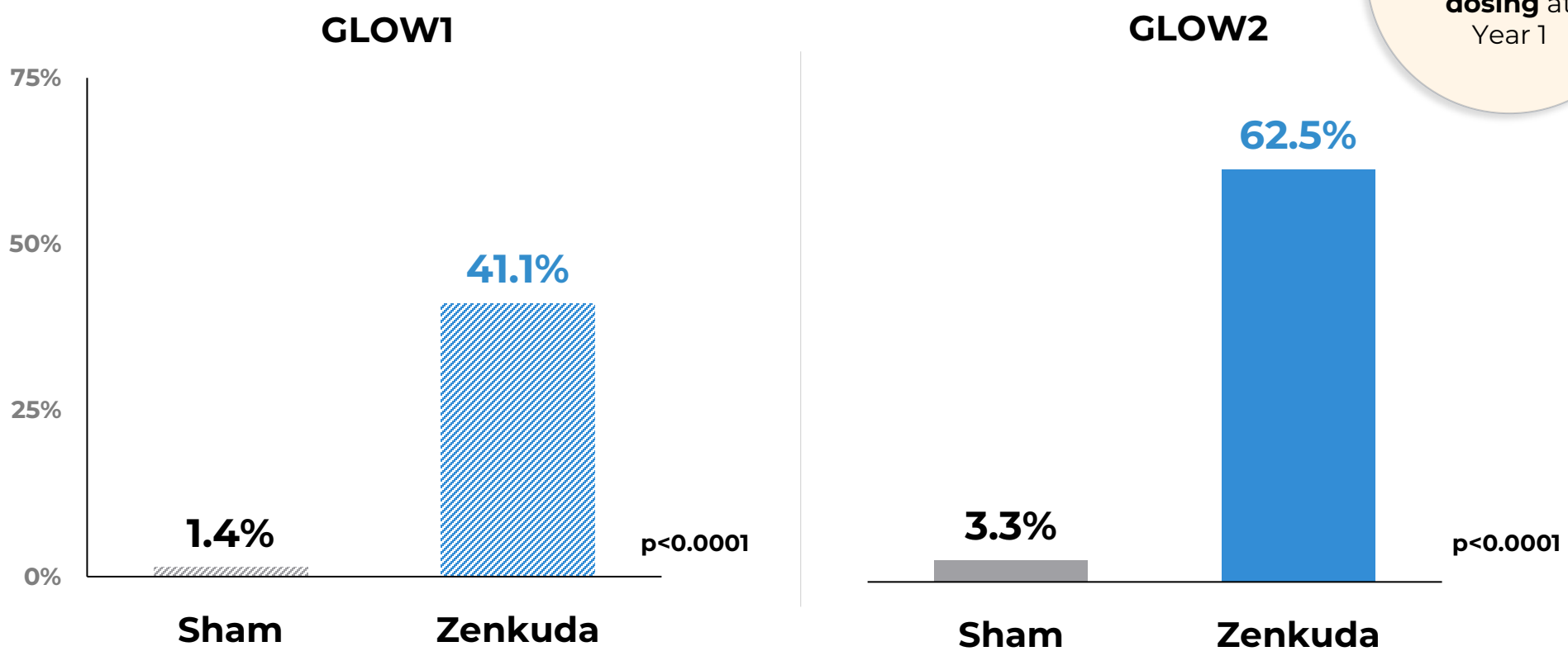
Zenkuda in Diabetic Retinopathy: Established superiority in ≥2-step improvement in DRSS in GLOW1 *and* GLOW2

Phase 3
GLOW1
GLOW2

Superiority ✓

6-month Durability ✓

Proportion of patients with ≥2-Step improvement in Diabetic Retinopathy Severity Score (DRSS) from Baseline to Week 48



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

GLOW1: Sham (n=125); Zenkuda (n=128). **GLOW2:** Sham (n=125); Zenkuda (n=130); Week 48 (LOCF) represents the last available observation while on randomized treatment, within the Week 48 visit window. Note: Weighted percentages are based on weighted average of observed estimates across strata using CMH weights. p-values are based on the difference in response rates

KODIAK

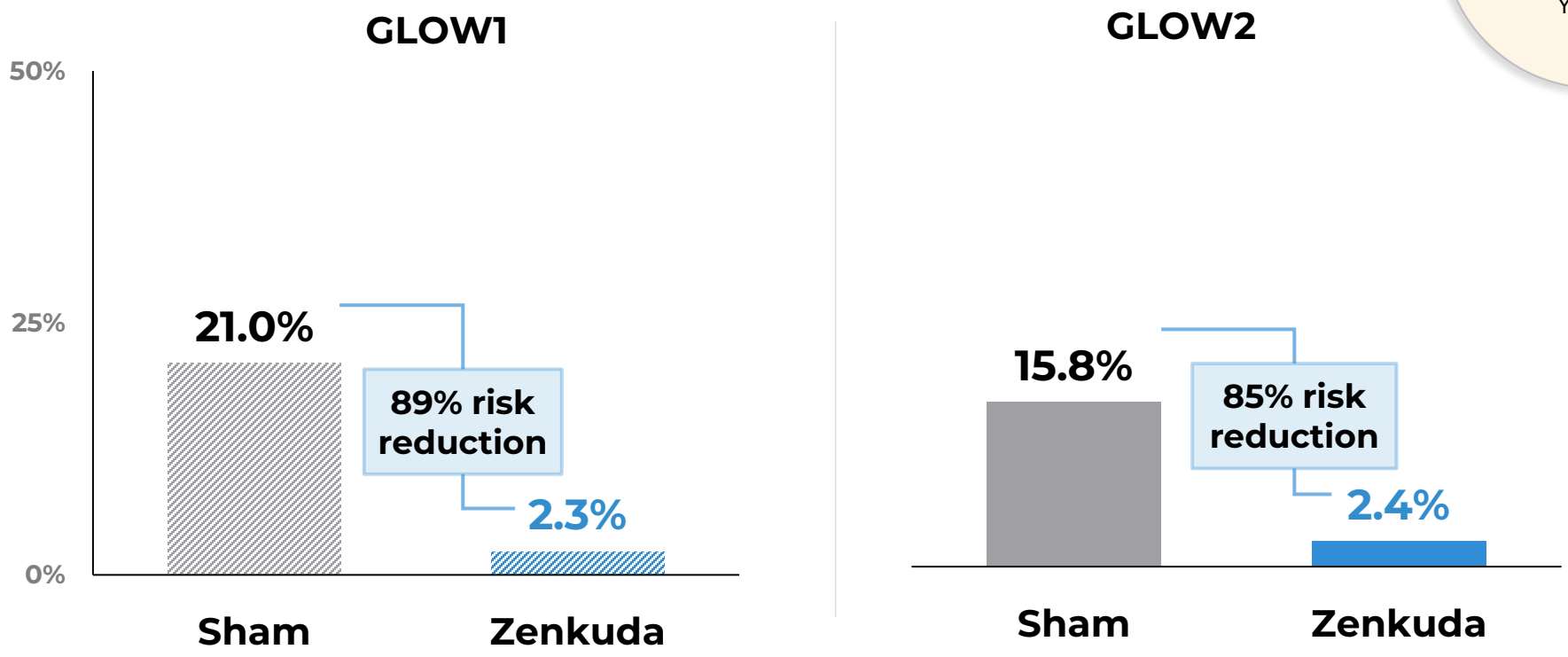
Zenkuda in Diabetic Retinopathy: Reduced the risk of developing sight-threatening complications by ≥85% in GLOW1 *and* GLOW2

Phase 3
GLOW1
GLOW2

Superiority ✓

6-month Durability ✓

Proportion of patients developing sight-threatening complications from Baseline through Week 48



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

GLOW1: Sham (n=125); Zenkuda (n=128). **GLOW2:** Sham (n=125); Zenkuda (n=130). Weighted percentages are based on weighted average of observed estimates across strata using CMH weights. p-values are based on the difference in response rates. Sight threatening complications include: diabetic macular edema, new or worsening proliferative diabetic retinopathy; anterior segment neovascularization; neovascularization of the disc and elsewhere, vitreous hemorrhage and neovascular glaucoma.

KODIAK

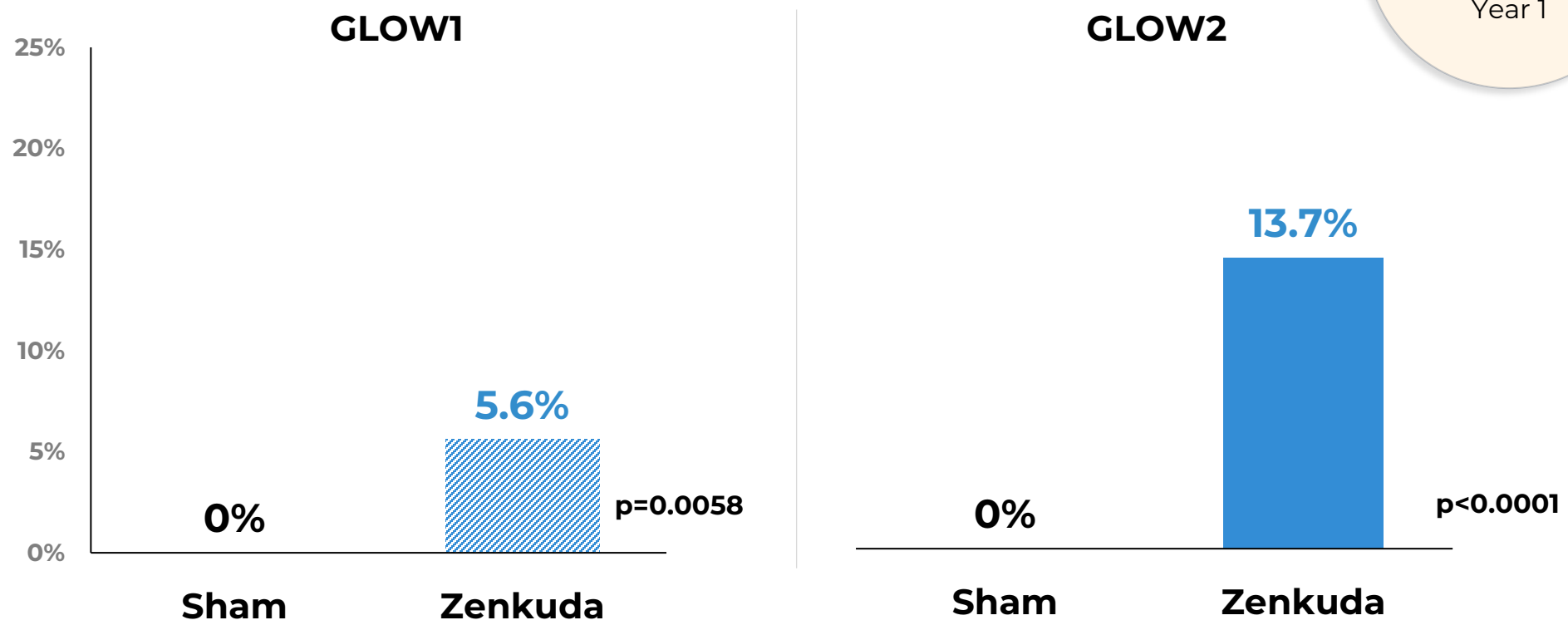
Zenkuda in Diabetic Retinopathy: Zenkuda established superiority in ≥3-step improvement in DR Severity Score (DRSS) in GLOW1 *and* GLOW2

Phase 3
GLOW1
GLOW2

Superiority ✓

6-month Durability ✓

Proportion of patients with ≥3-Step improvement in DRSS from Baseline to Week 48

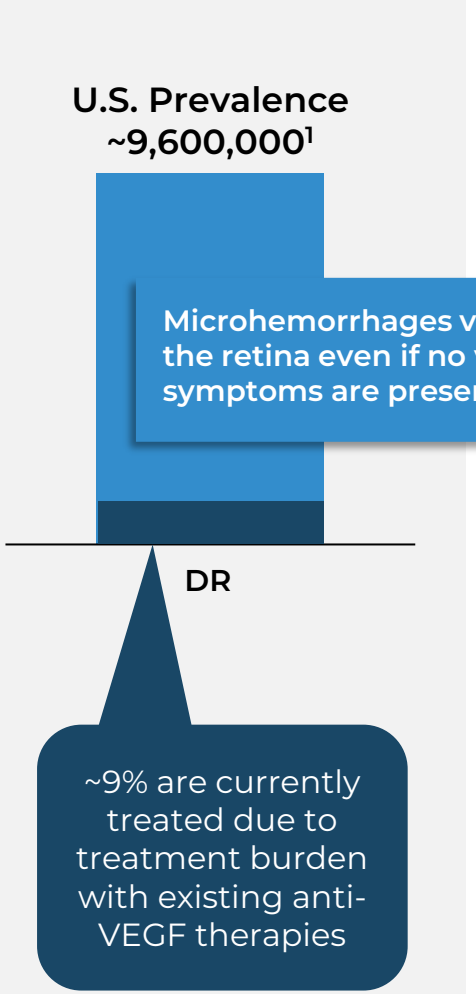


100% of patients on 6-month dosing at Year 1

GLOW1: Sham (n=125); Zenkuda (n=128). **GLOW2:** Sham (n=125); Zenkuda (n=130); Week 48 (LOCF) represents the last available observation while on randomized treatment, within the Week 48 visit window. Weighted percentages are based on weighted average of observed estimates across strata using CMH weights. p-values are based on the difference in response rates

KODIAK

The GLOW studies show that Zenkuda opens the door to earlier treatment with only 2 doses a year – a transformative potential for millions of patients



Baseline

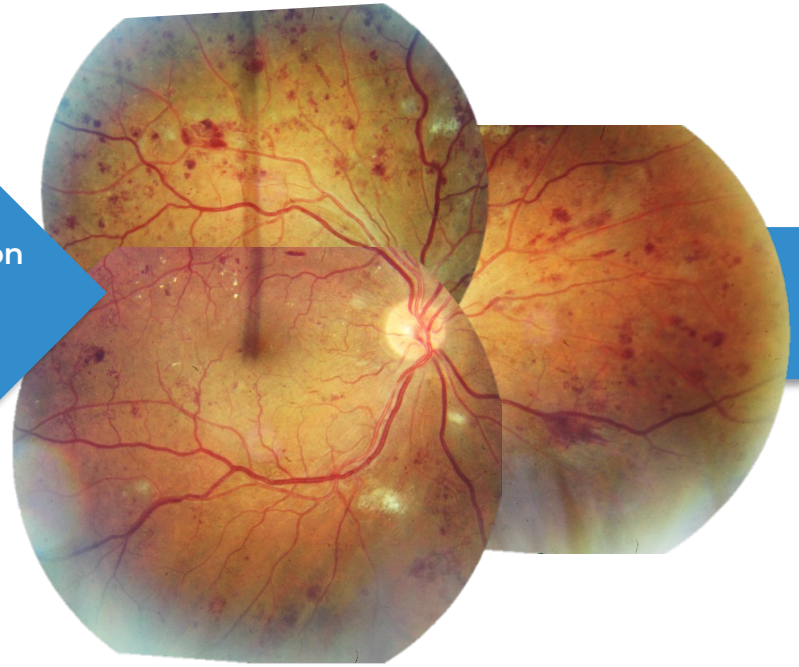


Image from a patient in the Phase 3 GLOW1 study in diabetic retinopathy

Week 48

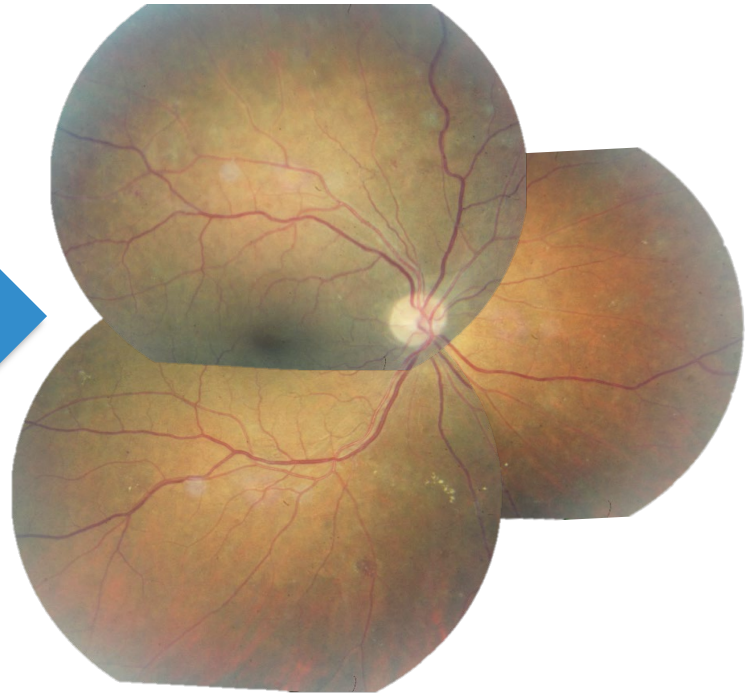


Image from same patient after treatment with Zenkuda

Zenkuda has the potential to improve outcomes for millions of patients with only 2 doses a year*

* after 3 initiating doses

1. Roche epidemiology data from 2025

Zenkuda and KSI-501 in wAMD: The ongoing Phase 3 DAYBREAK study is designed to evaluate immediacy, real-world durability and efficacy

Phase 3
DAYBREAK

Noninferiority

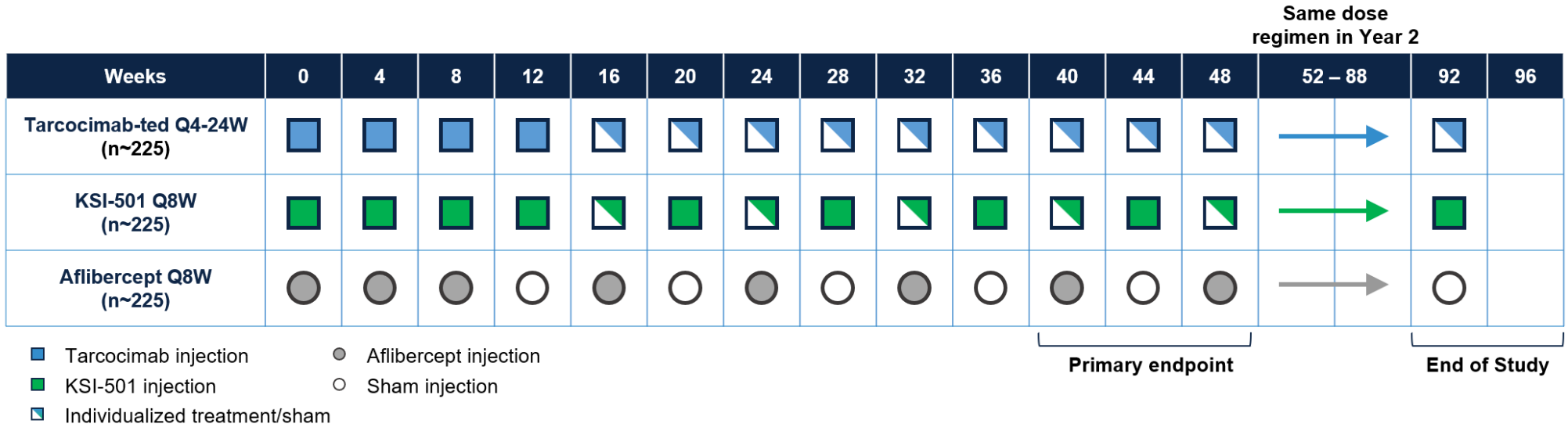
Zenkuda
6-month
durability

KSI-501
efficacy
exploration

Topline data
3Q 2026

Zenkuda objective: Assess **6-month durability** potential in wAMD

KSI-501 objective: Explore **efficacy potential** of bispecific IL-6 and VEGF inhibition in wAMD



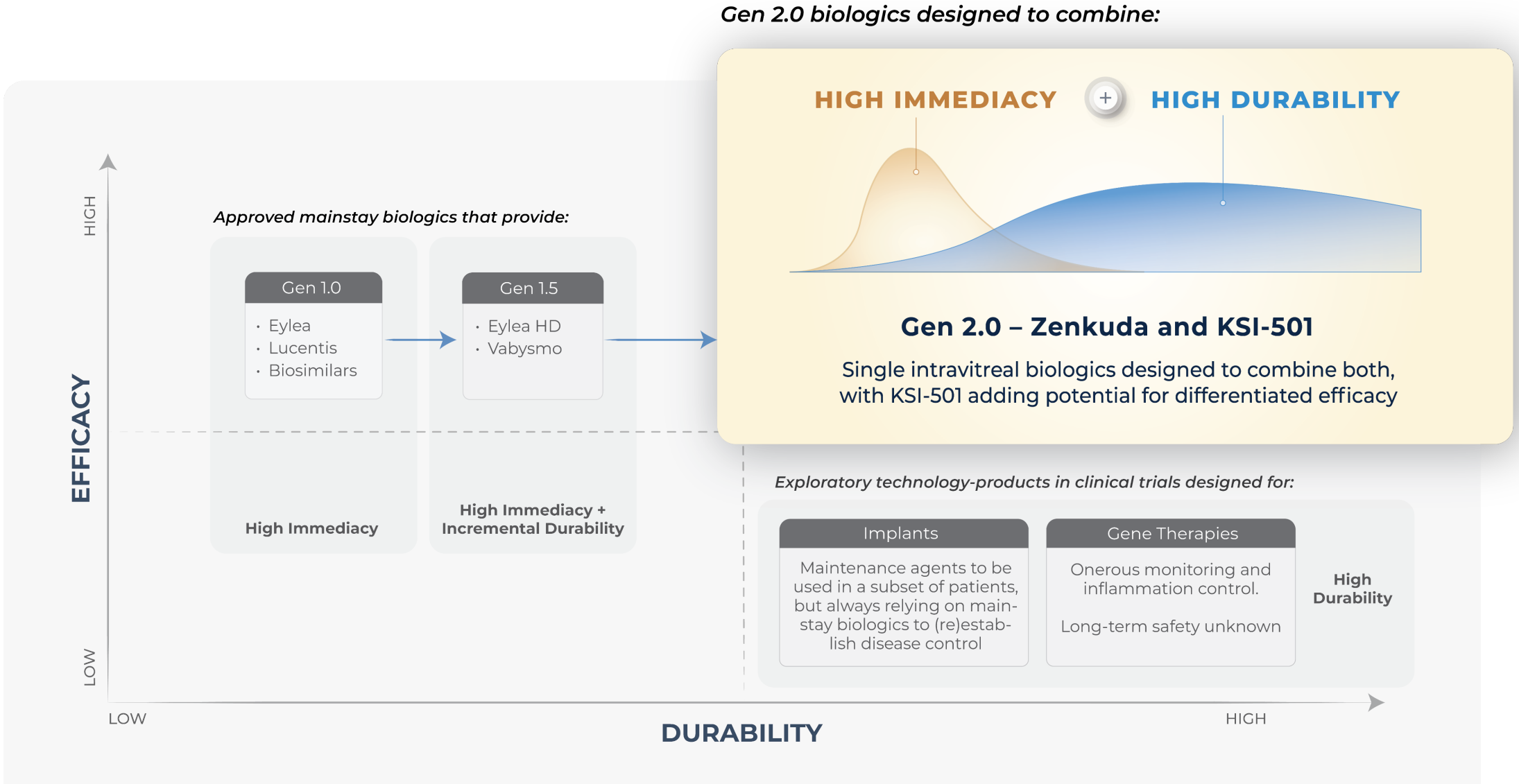
AI-based tool precisely measures fluid in the eye to optimize treatment per patient

- High-need patients: treats until dry, enables monthly dosing and detects disease reactivation earlier
- Long-durability patients: allows patients without active disease to safely go to every 6-month dosing

What are the potential implications and upsides for Zenkuda and KSI-501 in DAYBREAK, if successful?

	ZENKUDA	KSI-501
Strong and immediate disease control (closing the 'immediacy' gap)	✓	✓
Better vision gains vs anti-VEGF mono-therapy (aflibercept Q8W) with good OCT control		✓
Non-inferior vision gains to aflibercept Q8W	✓	
Long-interval dosing with flexible 1-month through 6-month label	✓	Not assessed in DAYBREAK

Revisiting the core unmet need: Kodiak's Gen 2.0 biologics are designed to occupy the high-immediacy, high-durability golden quadrant

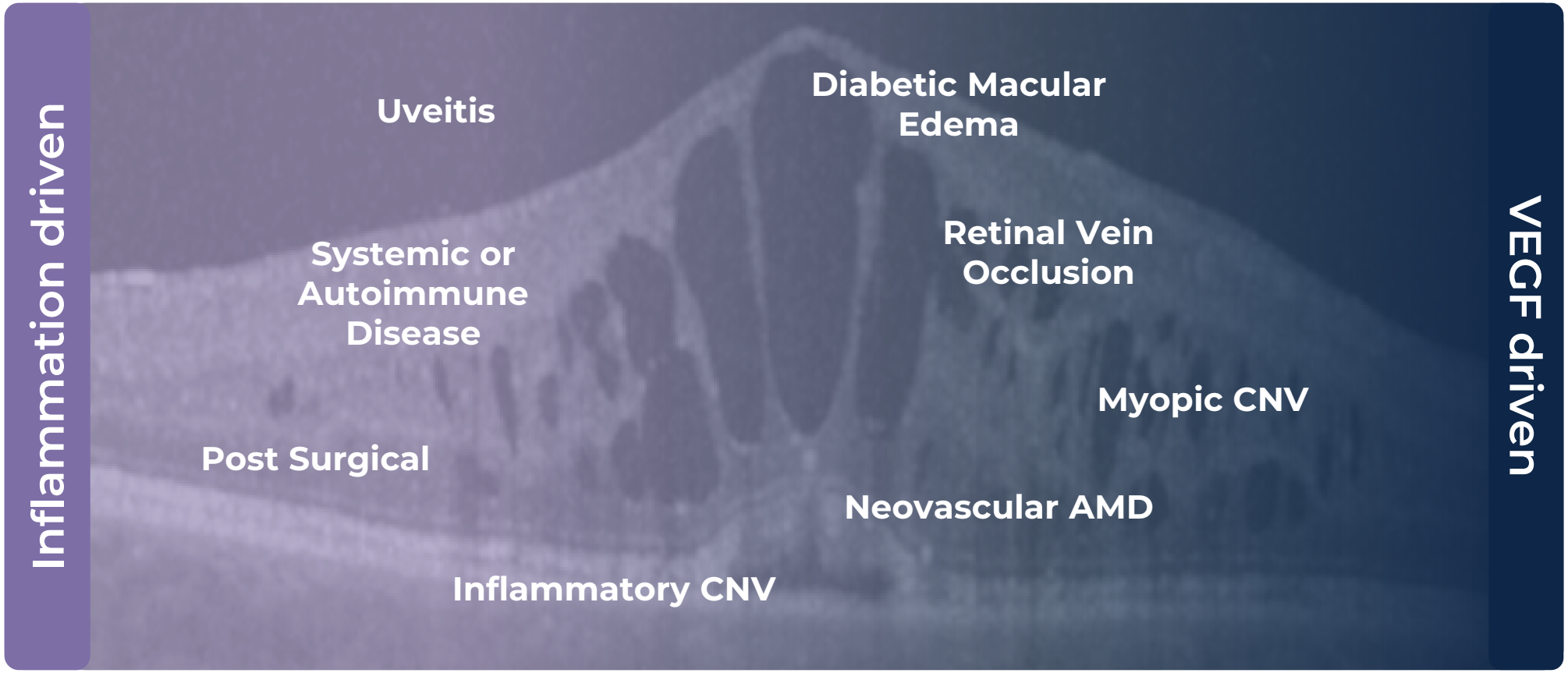


KSI-101

Macular Edema Secondary to Inflammation

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

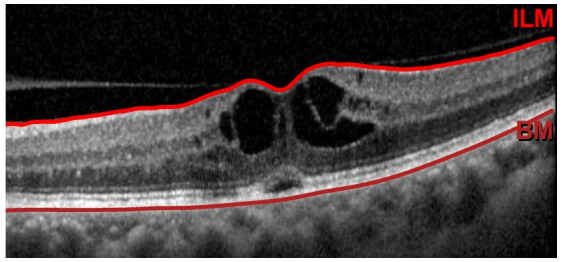


Macular edema secondary to inflammation

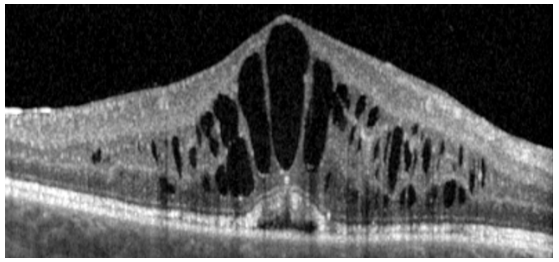
Retinal vascular and degenerative diseases

AMD: age-related macular degeneration; CNV: choroidal neovascularization

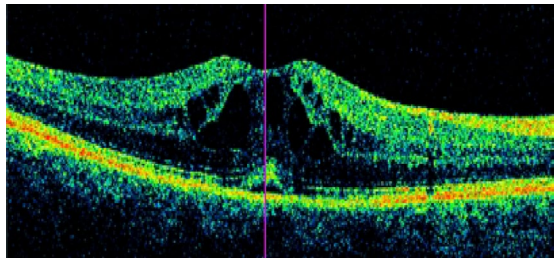
Macular edema and visual impairment are shared clinical features irrespective of etiology or location of inflammation



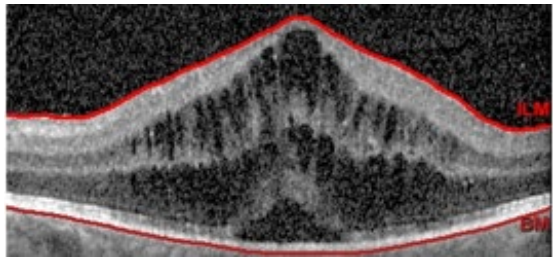
Anterior



Intermediate



Posterior



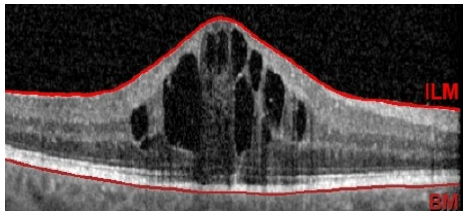
Panuveitis

Location of Inflammation

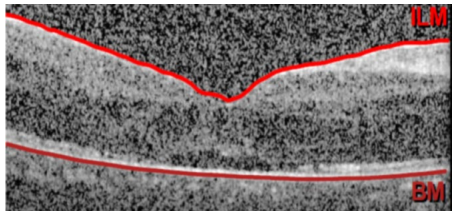
Macular edema is a shared clinical feature irrespective of the anatomical location of inflammation or specific etiology

Specific Etiology

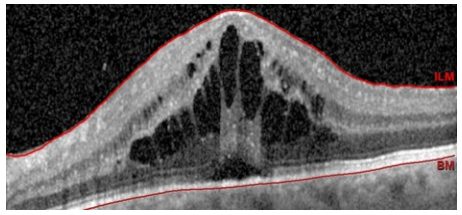
Idiopathic



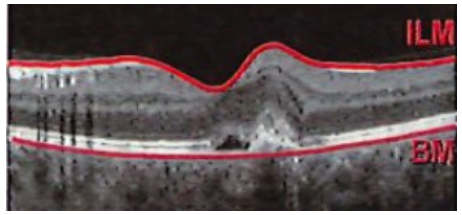
Juvenile Idiopathic Arthritis



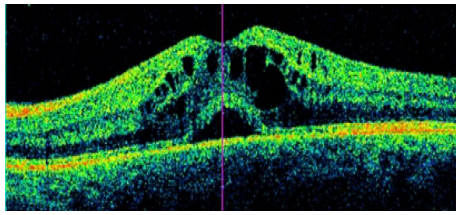
Focal Chorioretinal inflammation



Punctate Inner Choroidopathy



Post-Operative Macular Edema (refractory)

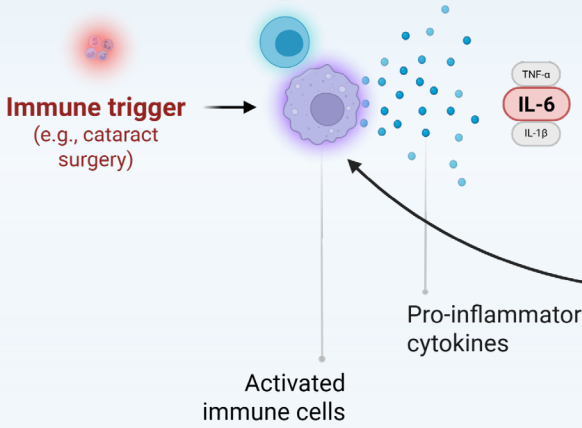


KODIAK

MESI is caused by immune dysregulation, leading to blood-retinal barrier breakdown and release of inflammatory mediators

Immune Trigger

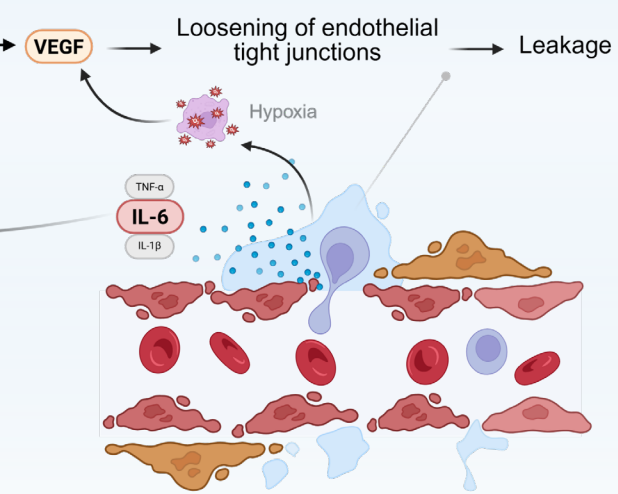
Causes immune cells to release pro-inflammatory cytokines, including IL-6



Leads to: **BRB disrupted**

Blood-Retinal Barrier Breaks Down

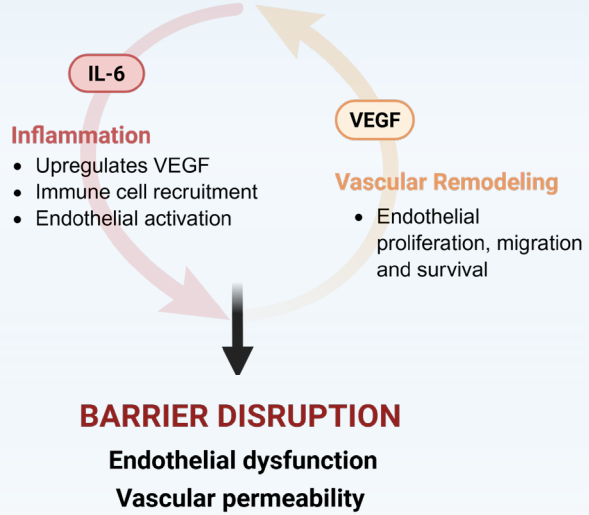
IL-6 upregulates VEGF. IL-6 and VEGF cause tight junction loss, allowing immune cells and blood plasma to leak into the retina



Leads to: **Macular edema, visual impairment and worsening inflammation**

IL-6 and VEGF Amplify Leakage

VEGF promotes vascular leak and neovascularization, while IL-6 sustains inflammation and upregulates VEGF

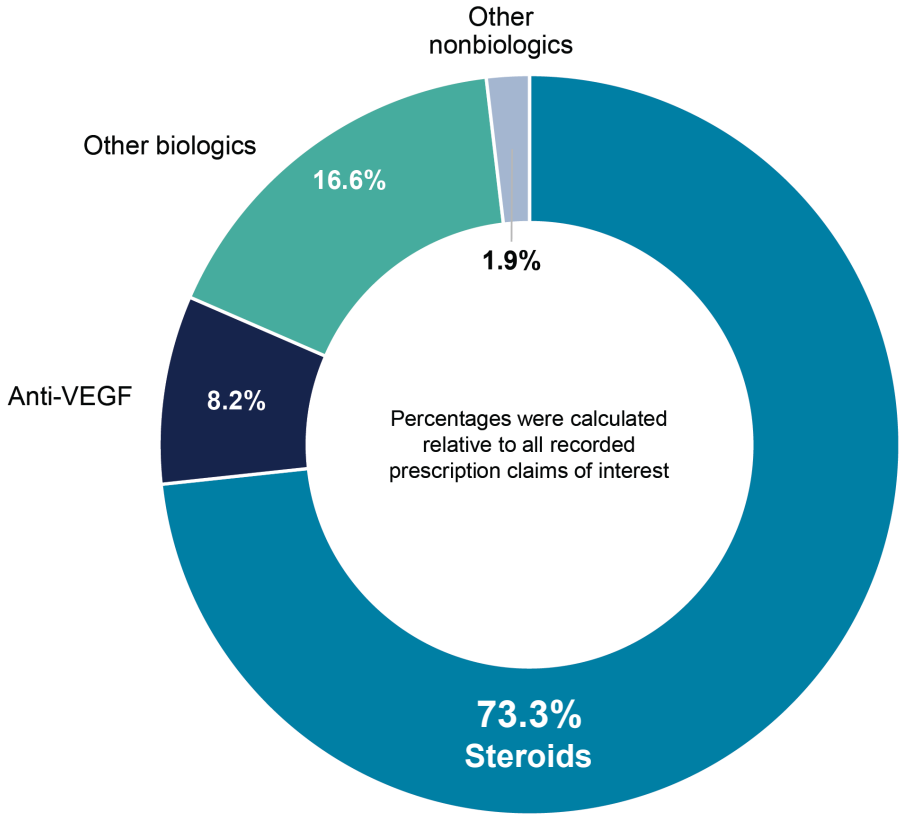


Together, IL-6 *and* VEGF compound the BRB damage

Yang et al. Int. J. Mol. Sci. 2023, 24: 4676. Metcalfe et al. Front. Immunol. 2020, 11: 1424. Shaik et al. Biomolecules. 2020, 10: 1673. Valle et al. Exp Eye Res. 2019, 178: 27. Mesquida et al. Trans Vis Sci Tech. 2019, 8: 32.

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 therapy[1]

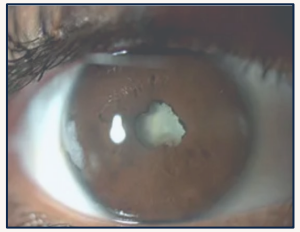


- Steroids were the most common medication class and remain the standard of care[1]
- XIPERE® (suprachoroidal triamcinolone acetonide) is the only approved local ocular treatment in the U.S.
- **Approximately 30-40% of patients do not fully respond to intraocular steroids[2,3]**
- Intraocular steroids are avoided in the pediatric population and **used with caution in adults due to high risk of permanent glaucoma damage and cataract formation**

1. Eichenbaum, D. et al. ARVO 2025 Poster Presentation 643. 2. Teper SJ. Journal of Clinical Medicine. 2021; 10(18):4133. 3. Tomkins-Netzer O et al. Ophthalmology. 2015. 122:2351-2359.

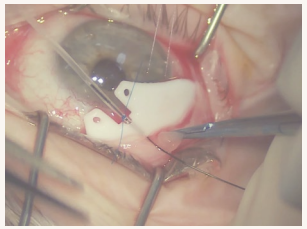
Adverse effects from current MESI treatments can lead to complex, repeated surgeries and irreversible vision loss

Cataract repair is complex in MESI 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® Label

(fluocinolone acetonide intravitreal implant)

- **60% of patients will require chronic IOP lowering medications** to control IOP
- **37% will require filtering procedures** to control IOP
- 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.

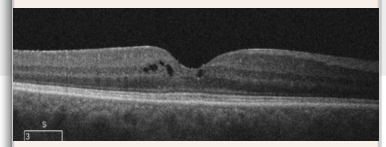
Surgeries in MESI patients are complicated and require complete control of inflammation before, during and after each procedure

KODIAK

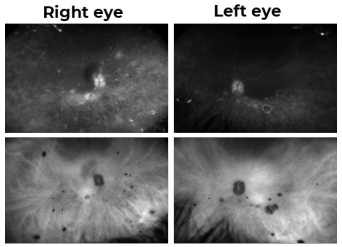
Patient journey case: steroids induced glaucoma with recurrent MESI over the course of approximately 2 years

1

Patient presented in Aug. 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

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

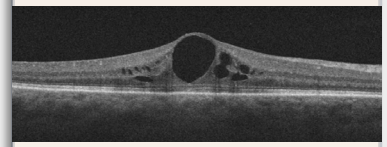
For alternate **immunomodulatory therapy (IMT)**

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**



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

REFERRED

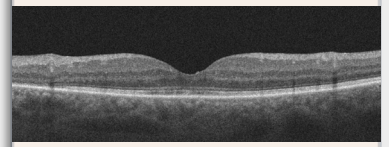
Surgery #2:

Glaucoma tube shunt surgery of the right eye in May 2025

IOP now stable

5

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



Will evaluate further need for IMT next visit

Patient presented to uveitis specialist

Glaucoma specialist

Rheumatologist

This representative case is based on Dr. Quan Nguyen's clinical experience, literature, and reviewed by uveitis consultants.

KODIAK

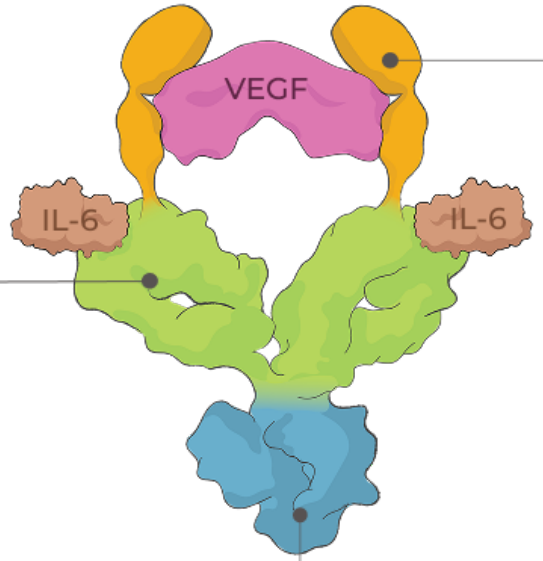
KSI-101 is a first-in-class, high-strength intravitreal biologic designed to target IL-6 mediated inflammation and VEGF-mediated vascular permeability simultaneously

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

Anti-IL-6 Antibody

Potent anti-inflammatory effect

The anti-IL-6 antibody can inhibit up to two IL-6 molecules to block inflammation and normalize the blood retinal barriers



VEGF Trap

Broad VEGF inhibition

The VEGF trap mimics the native receptor and binds multiple targets including VEGF-A, VEGF-B and PIGF

Modified Fc

Immunologically inert antibody

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

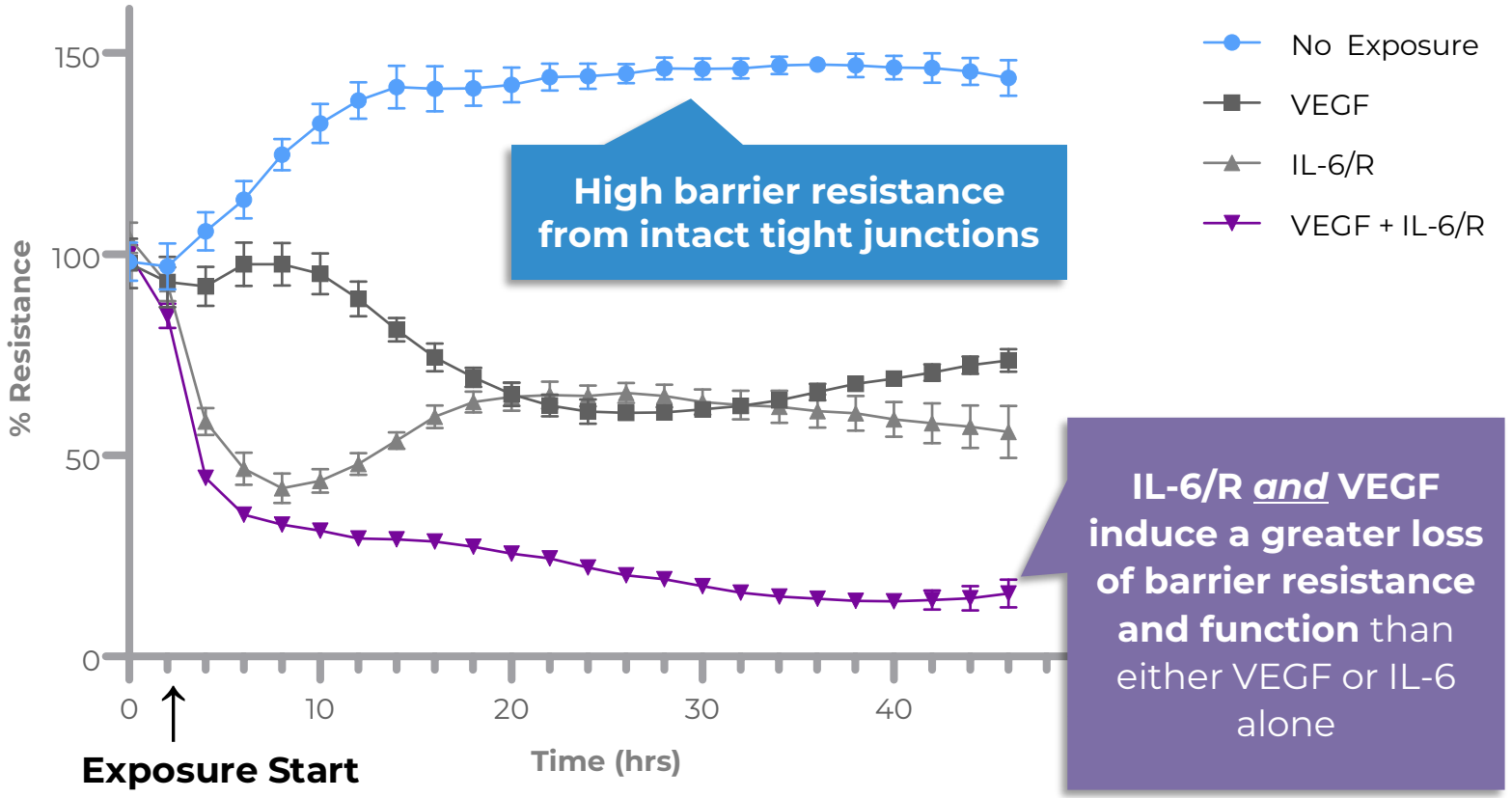
Ocular inflammation damages tight junctions between endothelial cells

- This compromises the integrity of the blood retina barrier, which 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 than either IL-6 or VEGF alone

- As measured by decreased barrier resistance in human umbilical vein endothelial cells (HUVEC)

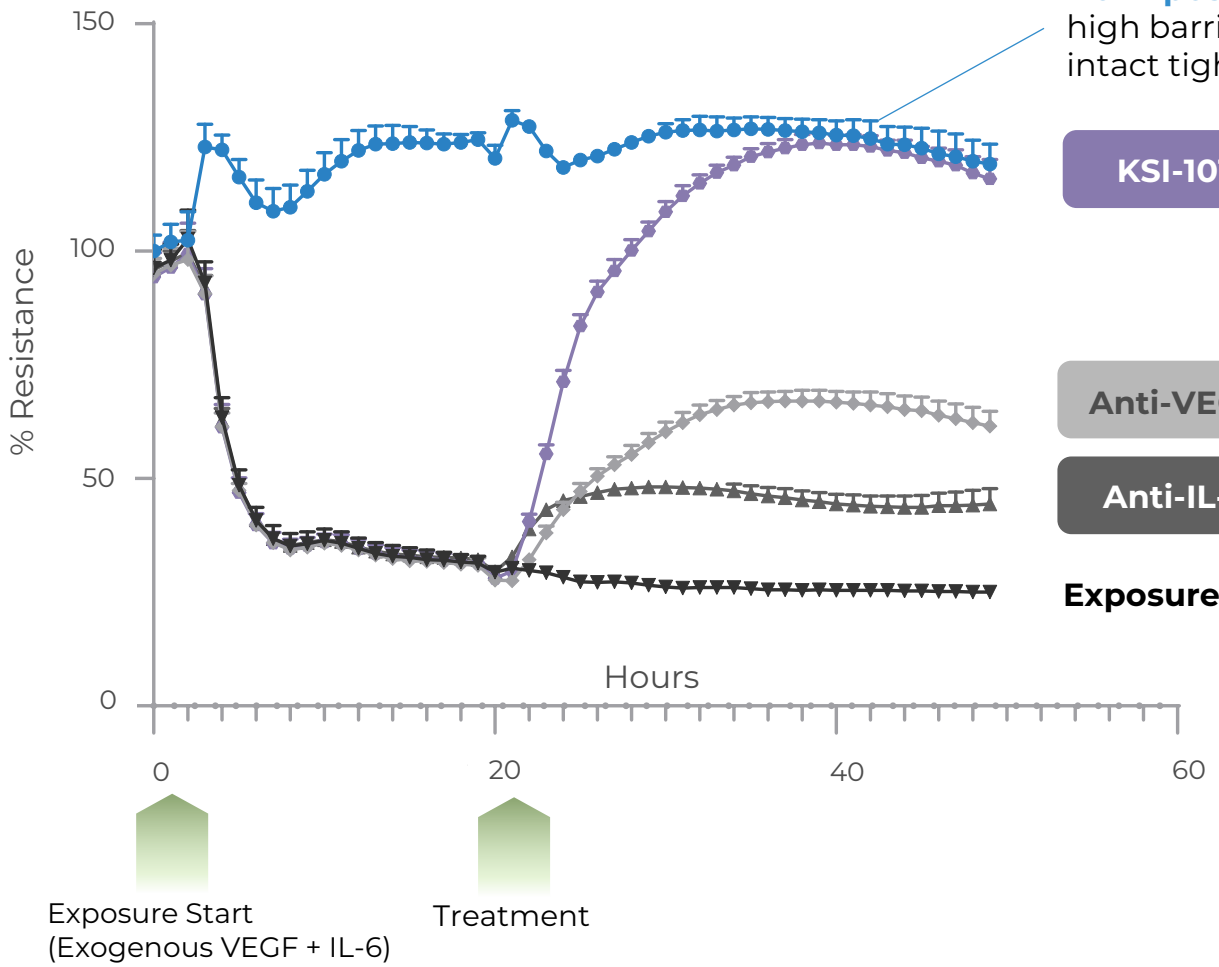
Assessing vascular endothelial barrier integrity using electrical resistance measurements



HUVEC cells were treated as indicated after growth to establish barrier

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



No Exposure to VEGF + IL-6/R: high barrier resistance due to intact tight junctions

KSI-101	+ VEGF + IL-6/R
Anti-VEGF	+ VEGF + IL-6/R
Anti-IL-6	+ VEGF + IL-6/R

Exposure to VEGF + IL-6/R

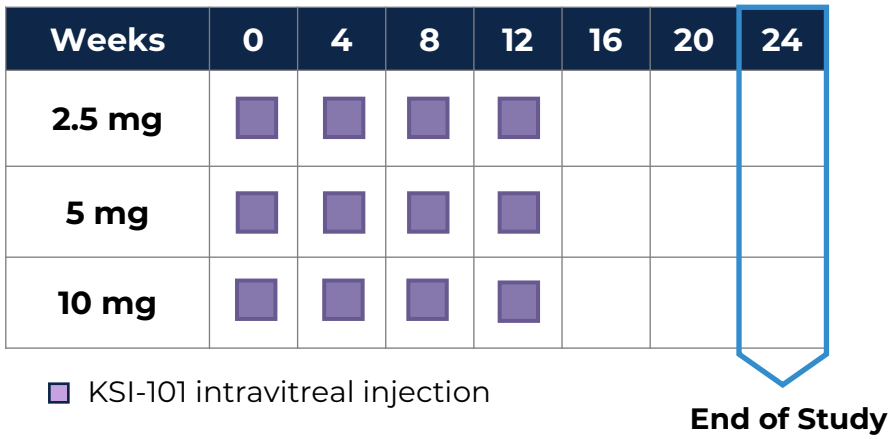
KSI-101 restores barrier integrity to no exposure levels

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

HUVEC cells were treated as indicated after growth to establish barrier

KSI-101 in MESI: Multiple-dose results from APEX Phase 1b study in the U.S. and a clinical cohort of Asian patients from tertiary uveitis centers

Study Design: 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)

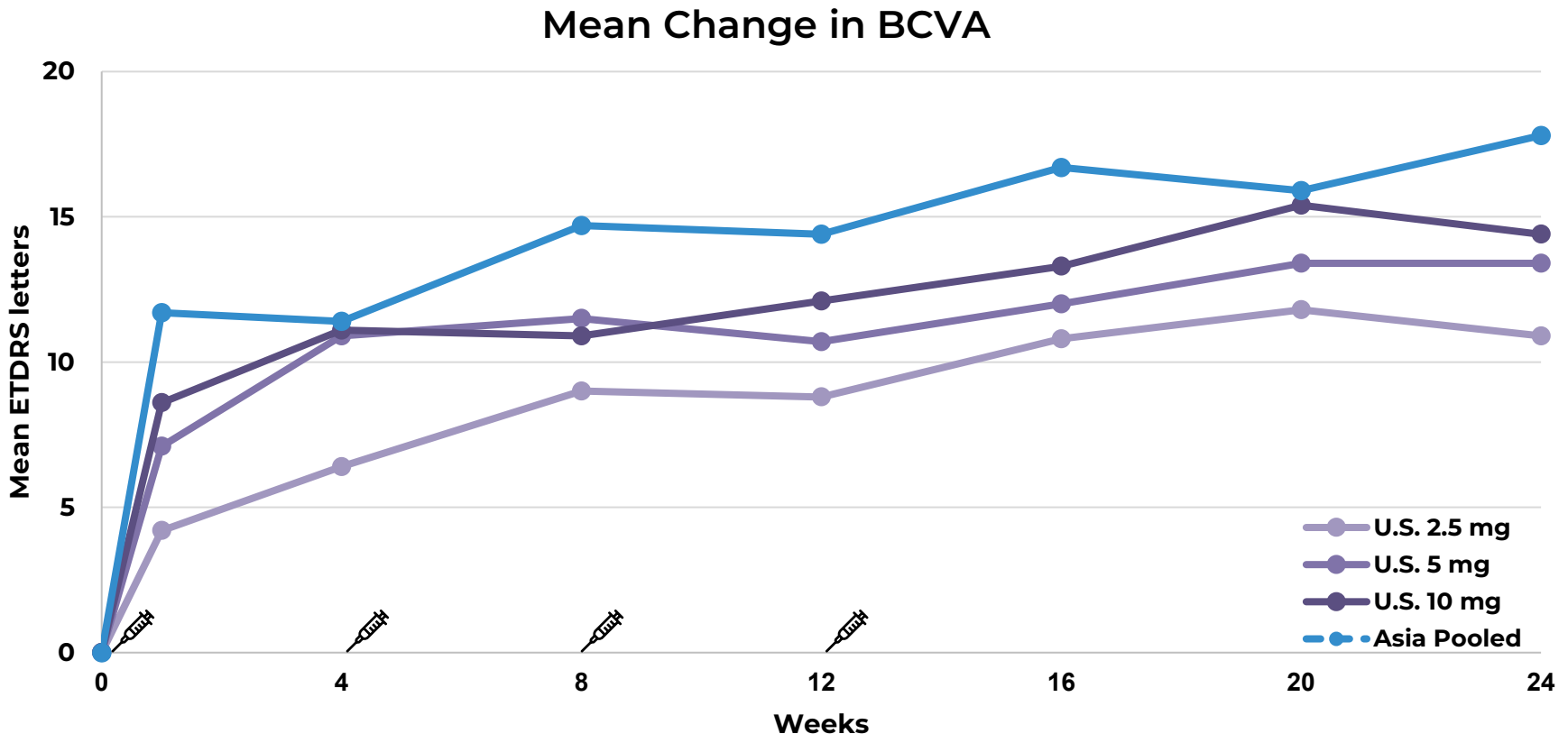
Baseline Characteristics

	APEX U.S. (N=41)			Asia*
	KSI-101 2.5 mg (n=13)	KSI-101 5 mg (n=14)	KSI-101 10 mg (n=14)	KSI-101 pooled (N=13)
Age, years, mean (SD)	74.2 (11.6)	67.4 (8.1)	67.5 (18.8)	49.9 (22.6)
Female, n (%)	8 (61.5)	7 (50.0)	8 (57.1)	11 (84.6)
Race, Asian, n (%)	0	1 (7.1%)	0	13 (100)
Race, White, n (%)	11 (84.6)	11 (78.6)	14 (100)	0
MESI disease duration, months, mean (SD)	12.2 (21.0)	1.7 (1.2)	15.8 (37.2)	2.3 (1.9)
Inflammation anatomical location, n (%)				
Anterior	0	2 (14.3)	0	1 (7.7)
Intermediate	1 (7.7)	0	2 (14.3)	2 (15.4)
Posterior	10 (76.9)	6 (42.9)	10 (71.4)	6 (46.2)
Panuveitis	2 (15.4)	6 (42.9)	2 (14.3)	5 (38.5)
Patients with active inflammation, n (%)	3 (23.1)	11 (78.6)	5 (35.7)	12 (92.3)
Unilateral MESI, n (%)	9 (69.2)	6 (42.9)	5 (35.7)	4 (30.8)
BCVA, ETDRS Letters, mean (SD)	62.7 (7.4)	65.6 (7.9)	62.1 (8.4)	58.5 (14.1)
Snellen equivalent	~20/50	~20/50	~20/63	~20/63
OCT CST, µm, mean (SD) (Site reported)	461.7 (137.7)	487.0 (124.1)	528.6 (157.3)	429.8 (76.3)
Lens Status, pseudophakic, n (%)	9 (69.2)	13 (92.9)	11 (78.6)	7 (53.8)

KODIAK

*Pharmacokinetics, tolerability and clinical activity of KSI-101 was evaluated in a clinical cohort of Asian patients at tertiary uveitis centers in Taiwan

Meaningful vision gains of >10 letters were observed after a single injection with continued improvement through 24 weeks



U.S.	2.5 mg	13	13	13	13	13	13	13	12
	5 mg	13	13	13	13	13	13	13	13
	10 mg	13	13	13	13	13	12	12	12
Asia	Pooled	12	12	12	12	12	11	11	11

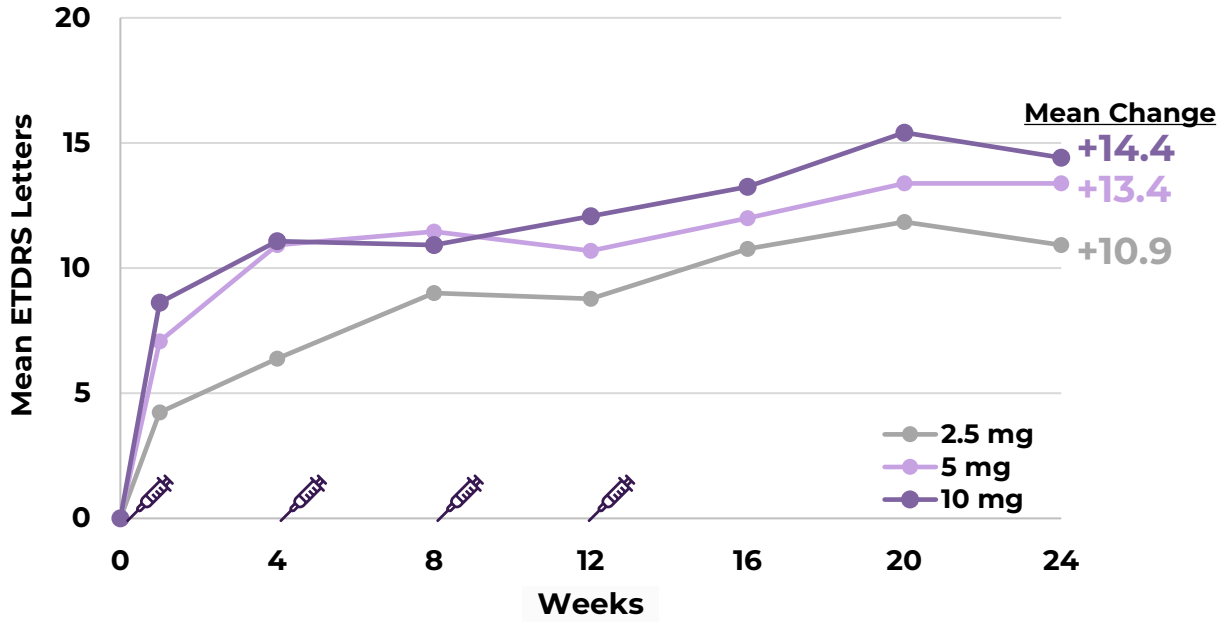
Sample Size

KODIAK

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. US: 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. Asian cohort: excludes one patient with a significant epiretinal membrane at baseline (exclusion criterion). One patient discontinued after Week 12 due to a spinal fracture.

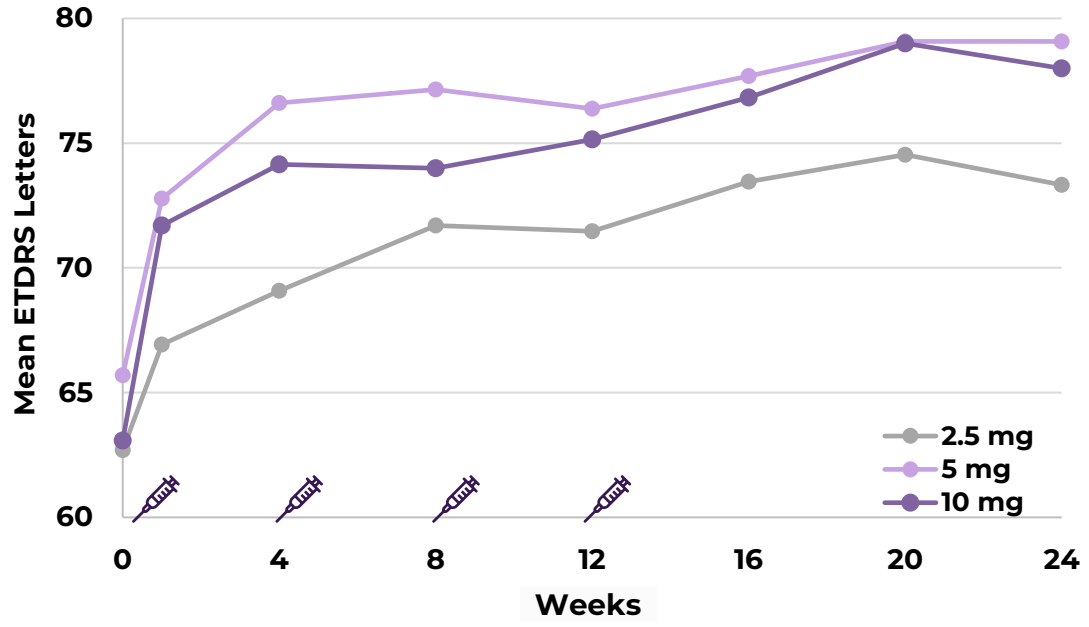
In APEX U.S., the top two dose levels achieve meaningful vision gains of >10 letters by Week 4 and continue to improve over time, achieving a 20/25 Snellen visual acuity by Week 20

Mean Change in BCVA over time



Dose Level	Sample Size							
2.5 mg	13	13	13	13	13	13	13	12
5 mg	13	13	13	13	13	13	13	13
10 mg	13	13	13	13	13	12	12	12

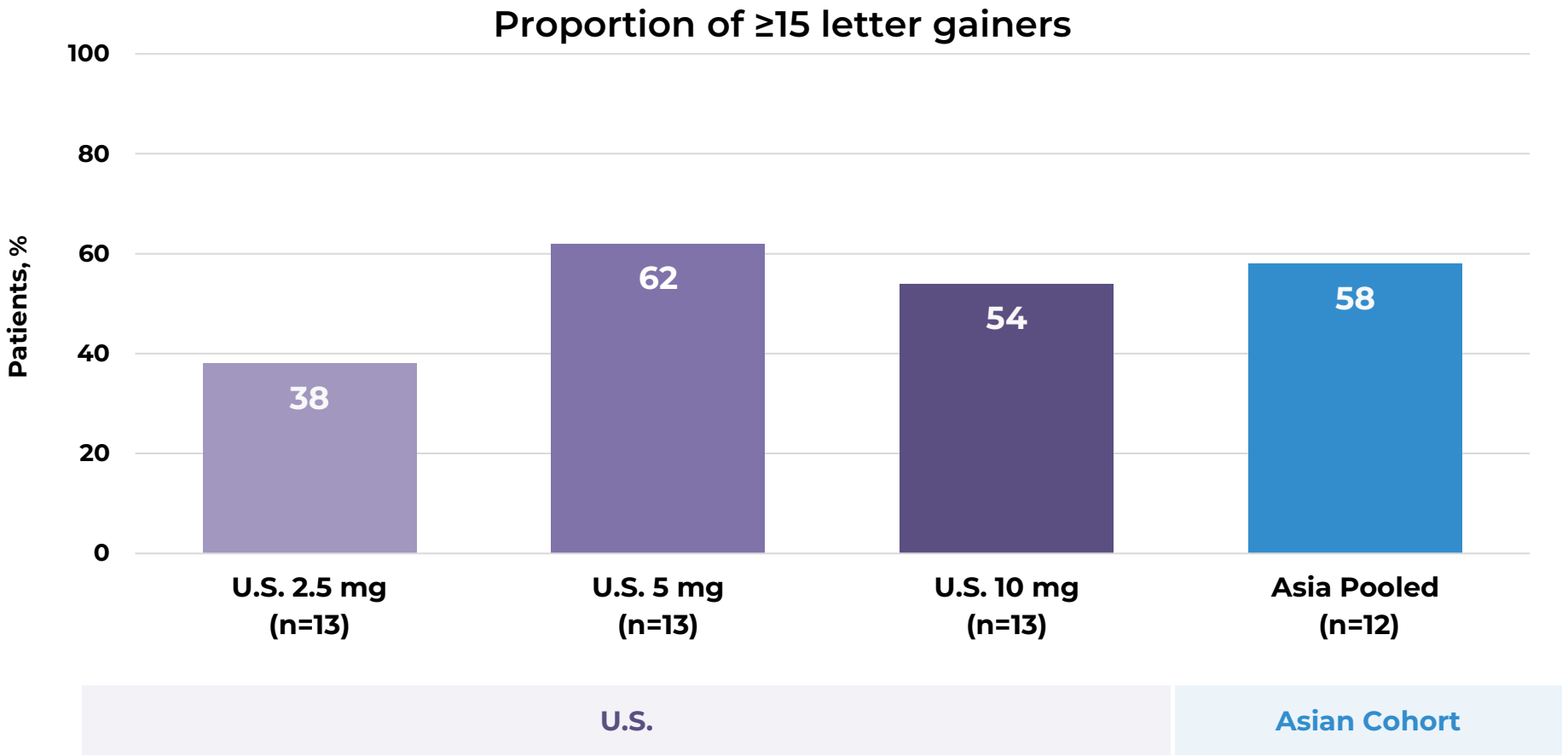
Observed BCVA over time



Dose Level	Sample Size							
2.5 mg	13	13	13	13	13	13	13	12
5 mg	13	13	13	13	13	13	13	13
10 mg	13	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 in the U.S.



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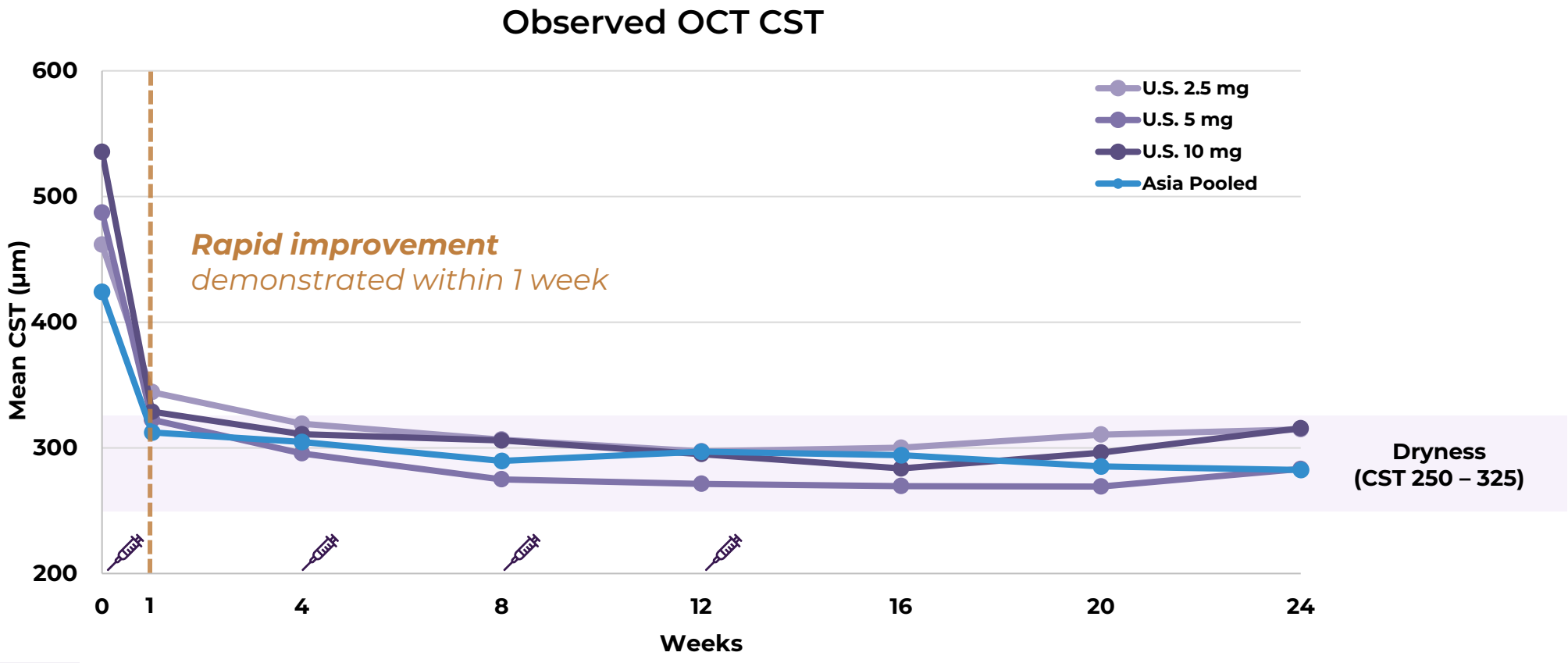
KSI-101 consistently achieved dryness (CST 250-325 µm) after a single injection, with sustained dryness observed during follow-up



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	5 mg	13	13	13	13	13	13	13
	10 mg	13	13	13	13	12	12	12
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Sample Size

KODIAK

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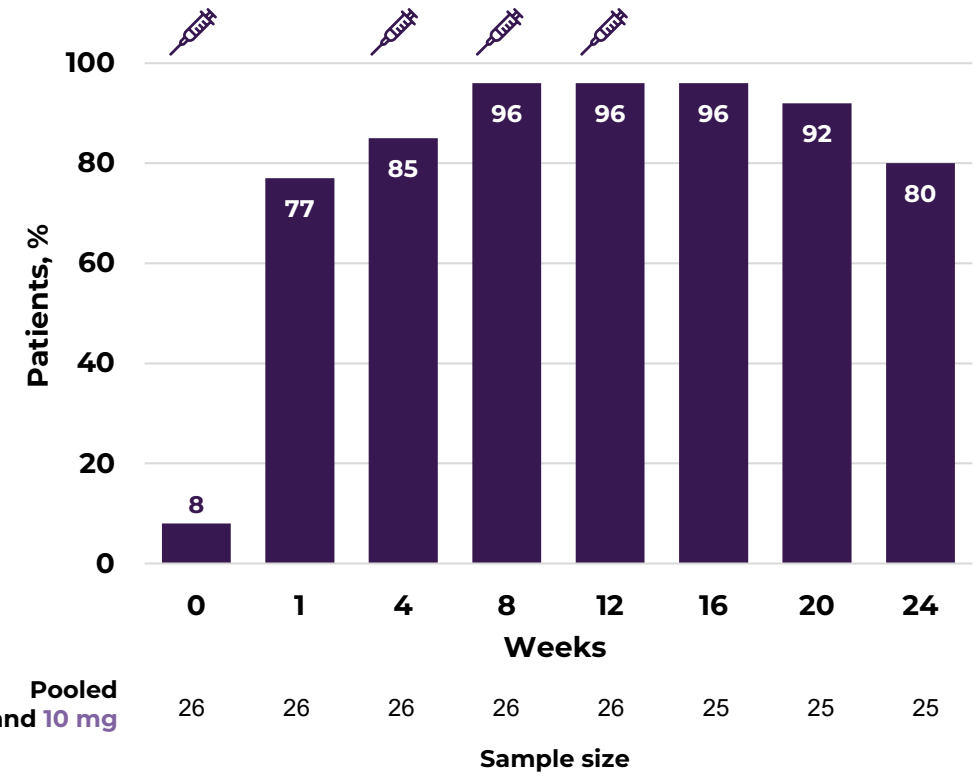
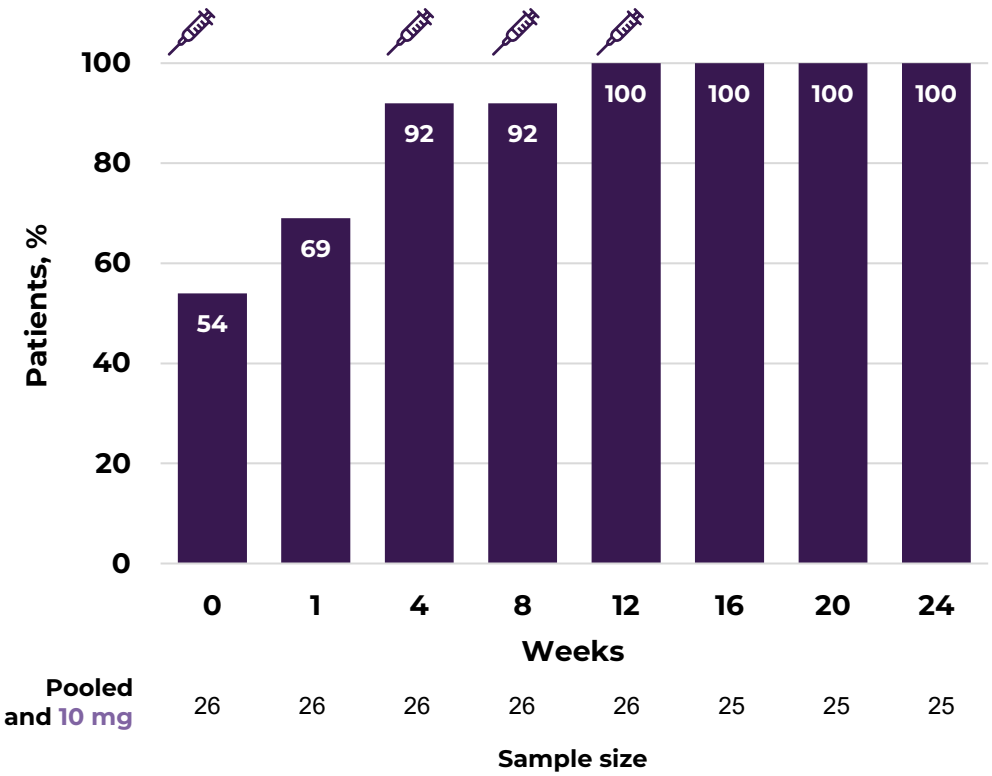
In APEX U.S., ≥90% of patients in the top two dose levels achieved absence of both IRF and SRF. A recurrence in IRF was observed in some patients 8 to 12 weeks after the last study treatment

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



IRF: intraretinal fluid; SRF: subretinal fluid

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.

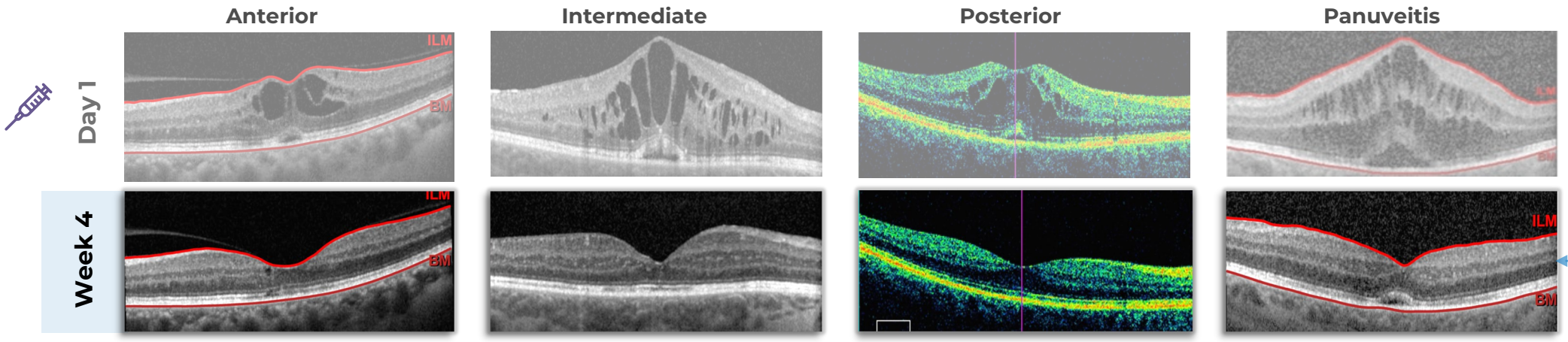
KSI-101 has been well-tolerated

	KSI-101 2.5 mg (n=13)	KSI-101 5 mg (n=14)	KSI-101 10 mg (n=14)	All KSI-101 (N=41)	Asia KSI-101 pooled (N=13)
Summary of AEs in the Study eye, n (%)					
Subjects with ≥1 AEs	2 (15.4)	3 (21.4)	2 (14.3)	7 (17.1)	3 (23.1)
Treatment-related AEs	1 (7.7)[a]	1 (7.1)[b]	0	2 (4.9)	0
Serious AEs	0	0	0	0	0
Treatment-related serious AEs	0	0	0	0	0
Severe AEs	0	0	0	0	0
AEs leading to study discontinuation	0	1 (7.1)[b]	0	1 (2.4)	0
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)	0
Occlusive retinal vasculitis	0	0	0	0	0
Cataract	0	0	0	0	0
Elevated IOP	0	0	0	0	0
Eye pain	1 (7.7)[a]	0	0	1 (2.4)	0
Vitreous hemorrhage	1 (7.7)[a]	0	0	1 (2.4)	0

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.

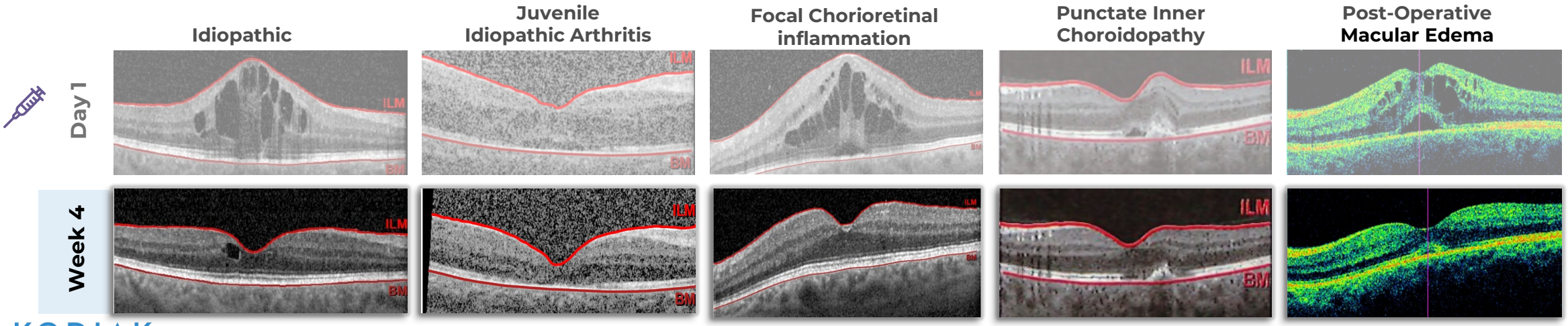
Single-dose KSI-101 demonstrates rapid, meaningful responses in MESI, independent of inflammation location or macular edema etiology

Location of Inflammation

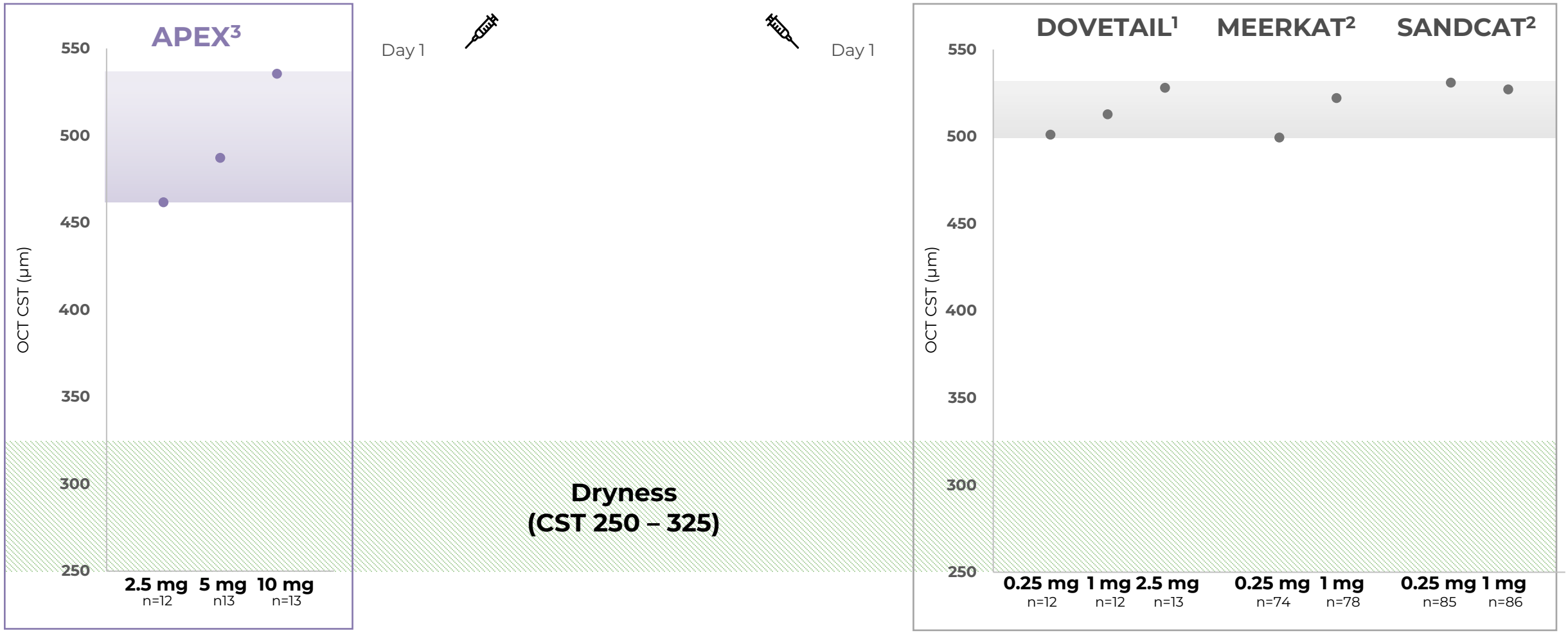


Effect of a single dose of KSI-101

Specific Macular Edema Etiology



Mean Change in OCT CST and Absolute CST



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.

n-values at baseline. 1. Adapted from Sharma S, et al. Phase 1 DOVETAIL Study. Presented at the American Society of Retina Specialists, Seattle, WA, July 2023. Note DOVETAIL had only 3 loading doses. 2. Adapted from Khurana, et al. Phase 3 MEERKAT and SANDCAT. Presented at the American Academy of Ophthalmology Retina Subspecialty Day, Orlando, FL, October 17, 2025. 3. Preliminary results. 3. The APEX study is ongoing. Final results may be different due to data cleaning. Results include patients in the per protocol set that met all 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).

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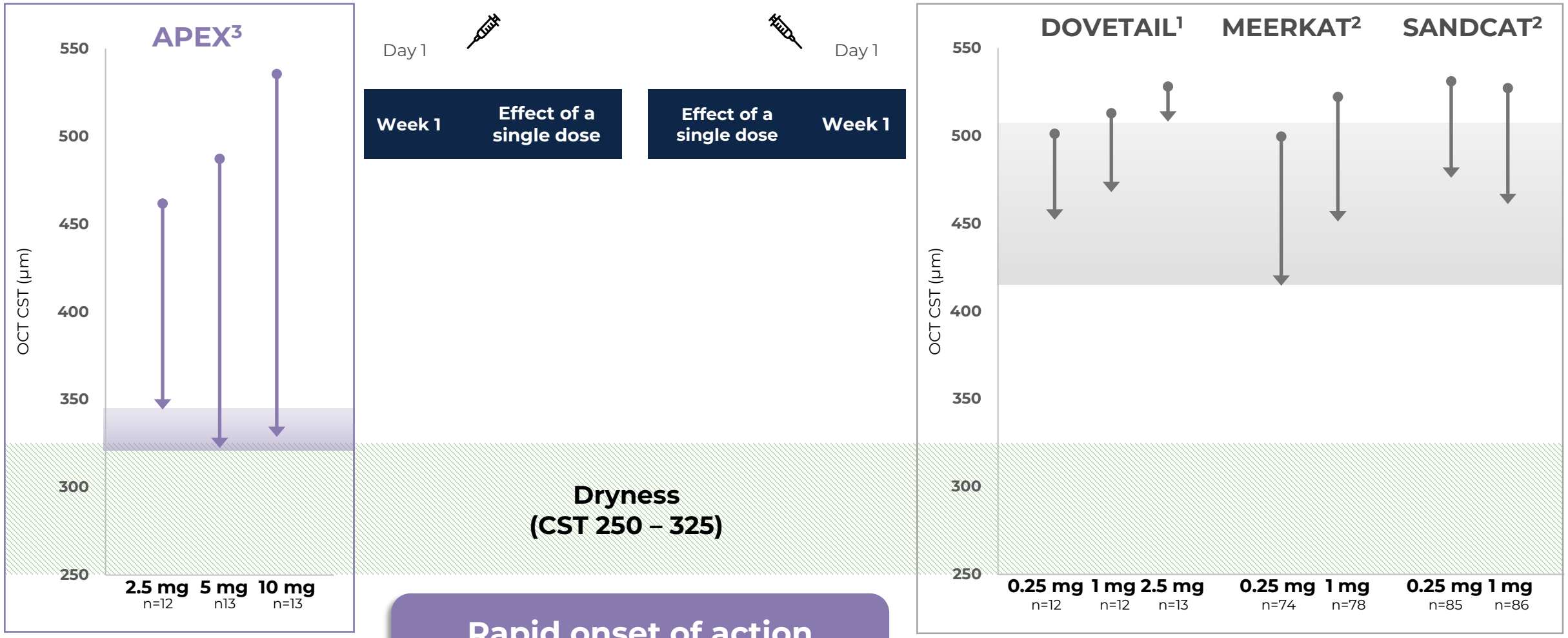

Anti-IL-6, VEGF trap
KSI-101

Rapid onset of action observed with KSI-101

Anti-IL-6
Vamikibart

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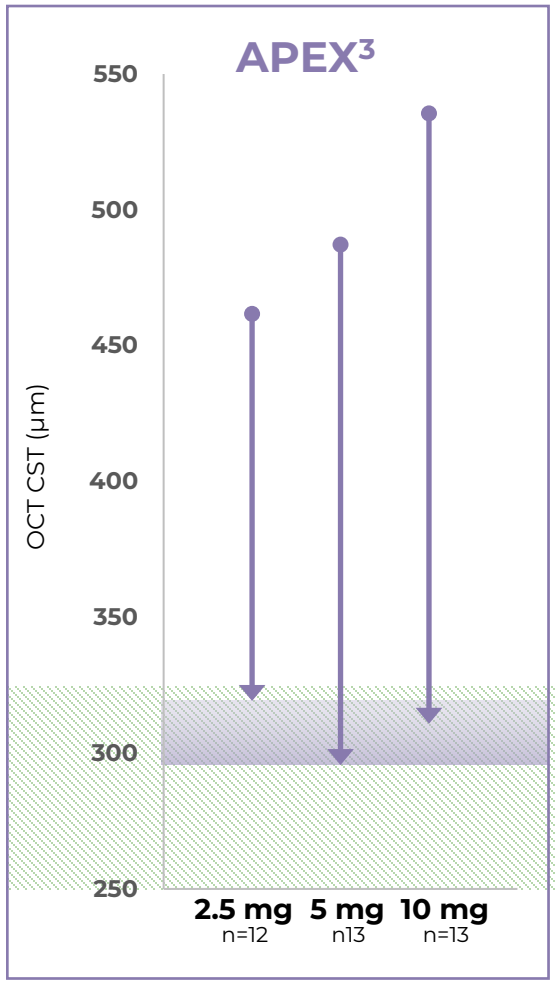
Mean Change in OCT CST and Absolute CST



Anti-IL-6, VEGF trap
KSI-101

Day 1

Week 1

Week 4 | **Effect of a single dose**

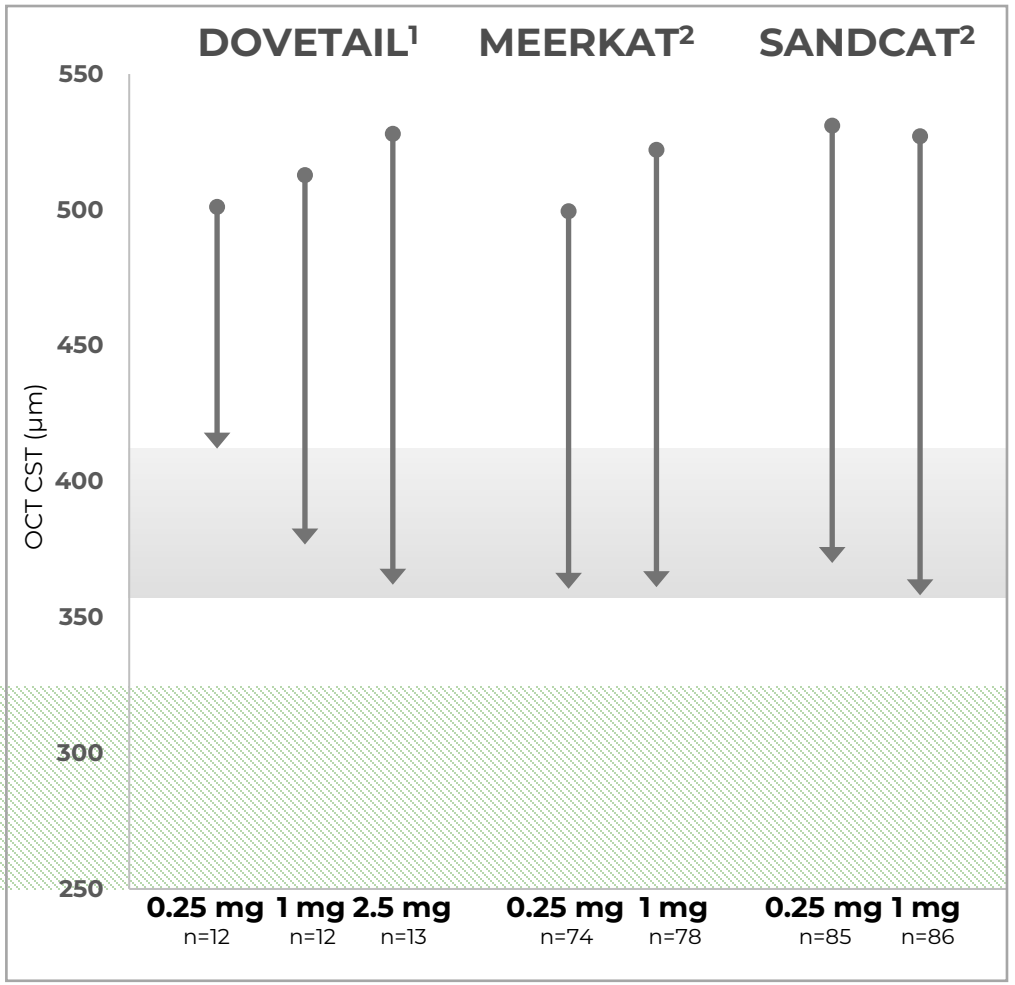
Day 1

Week 1

Effect of a single dose | **Week 4**

Dryness
(CST 250 – 325)

Dryness levels observed with a single dose of KSI-101



Anti-IL-6
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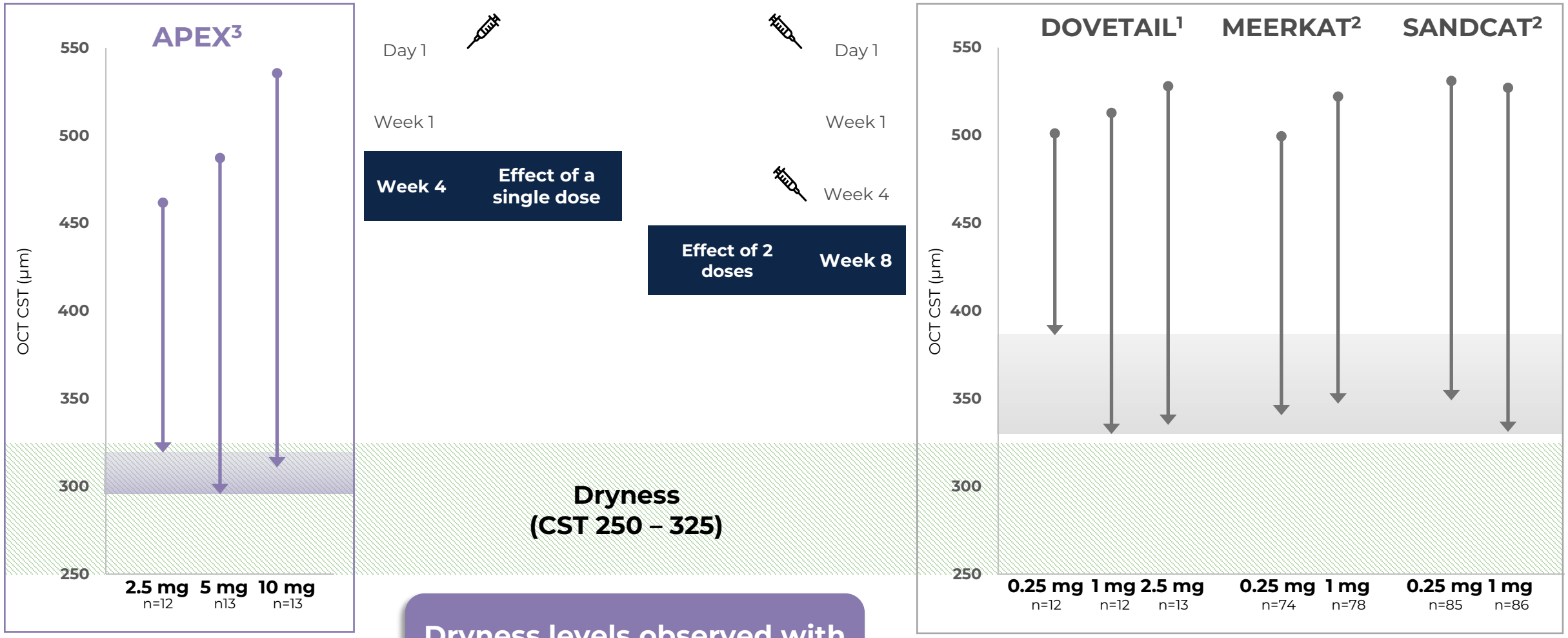

Anti-IL-6, VEGF trap
KSI-101

Dryness levels observed with a single dose of KSI-101

Anti-IL-6
Vamikibart

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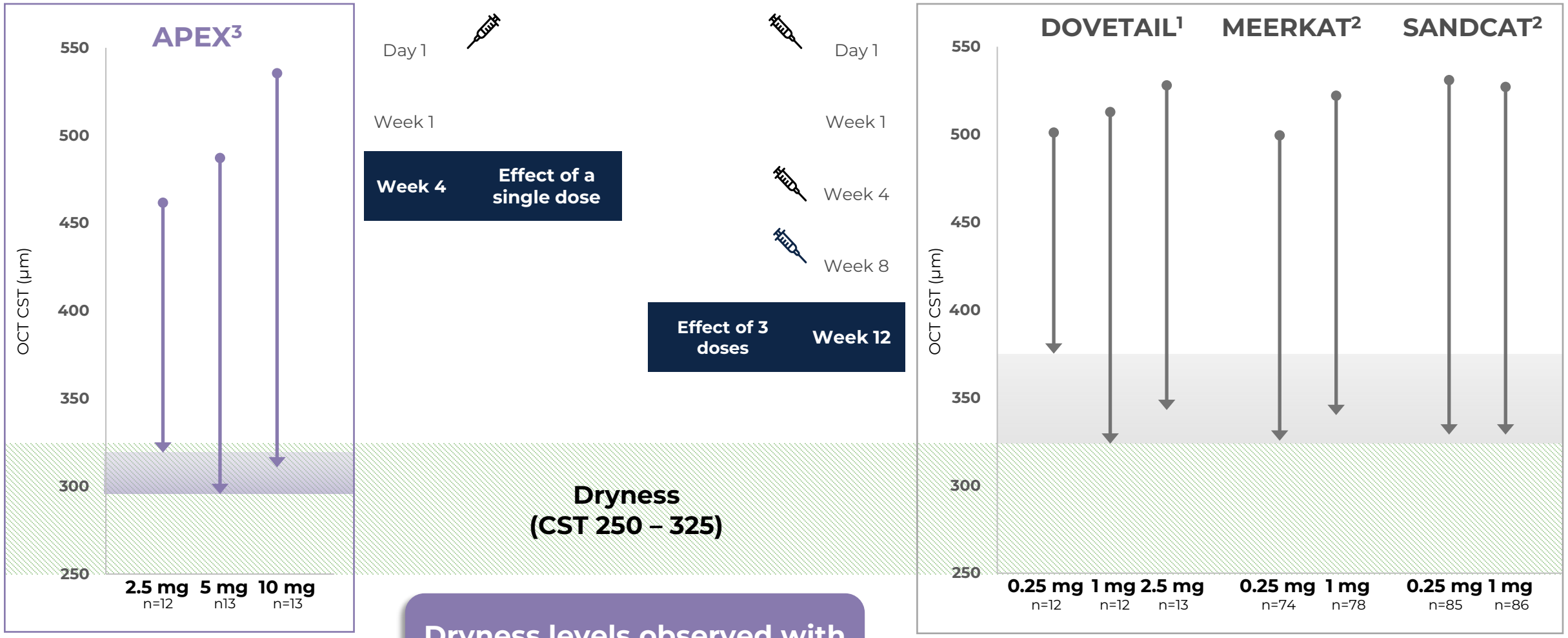

Anti-IL-6, VEGF trap
KSI-101

Dryness levels observed with a single dose of KSI-101

Anti-IL-6
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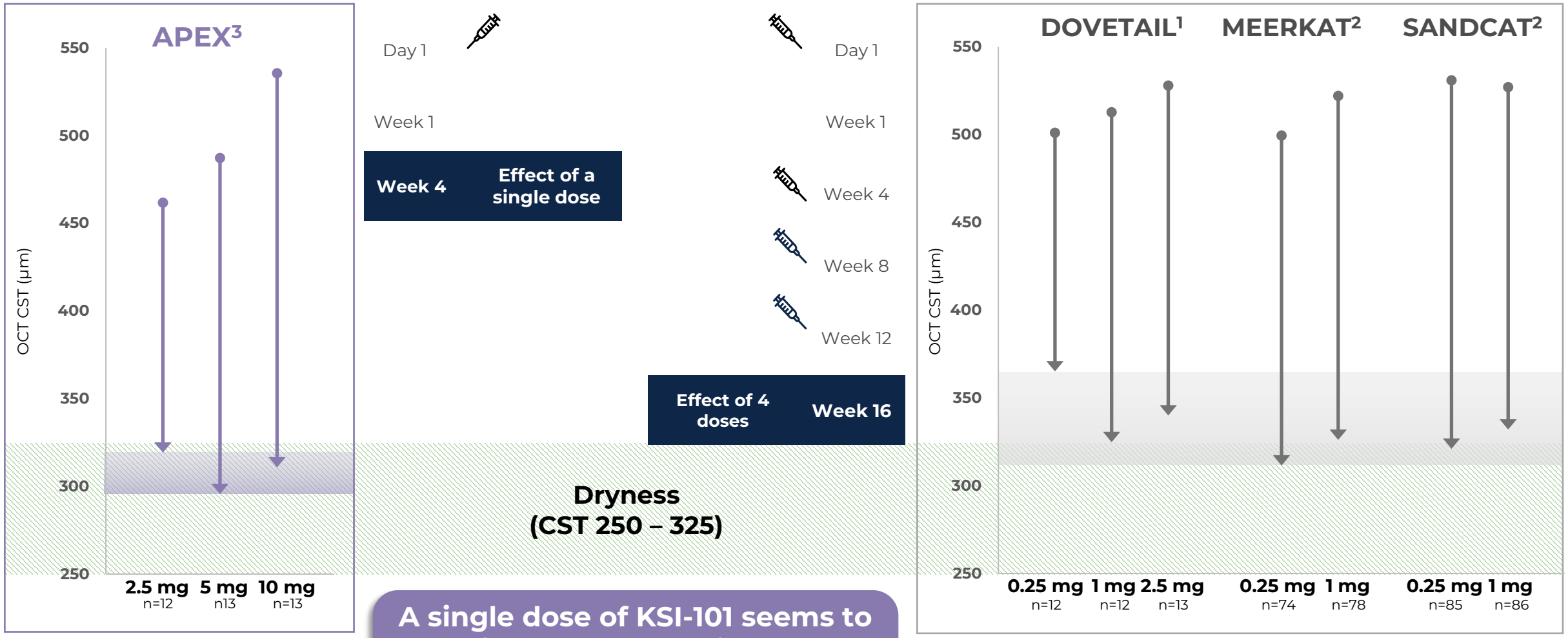

Anti-IL-6, VEGF trap
KSI-101

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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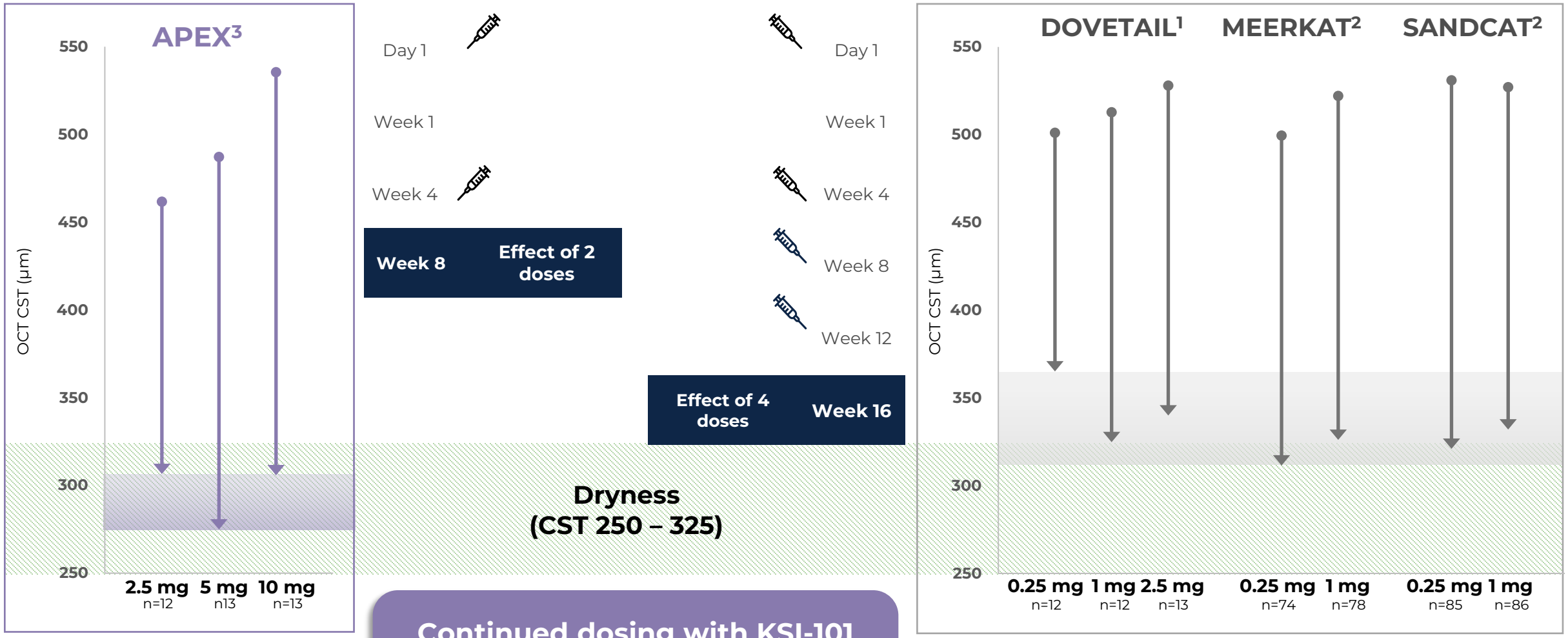

Anti-IL-6, VEGF trap
KSI-101

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

Anti-IL-6
Vamikibart

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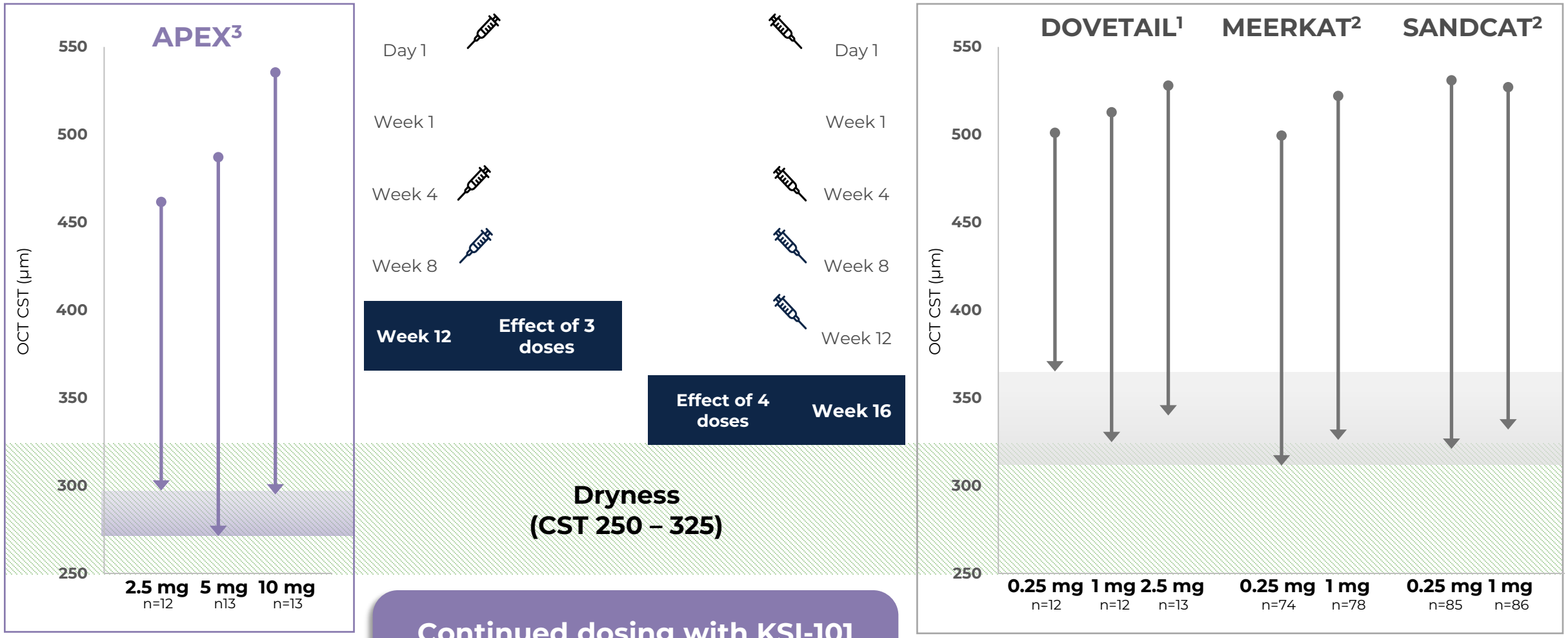

Anti-IL-6, VEGF trap
KSI-101

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

Anti-IL-6
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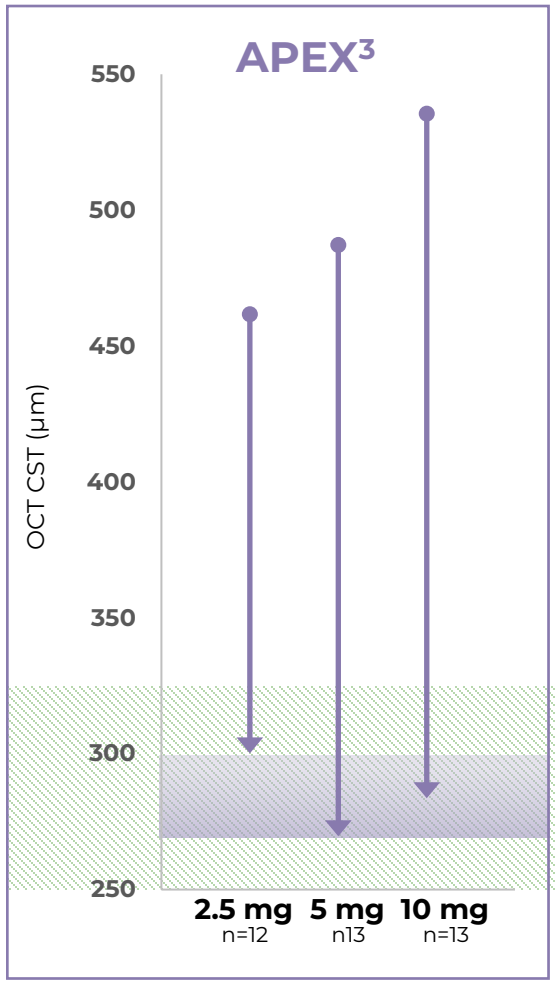
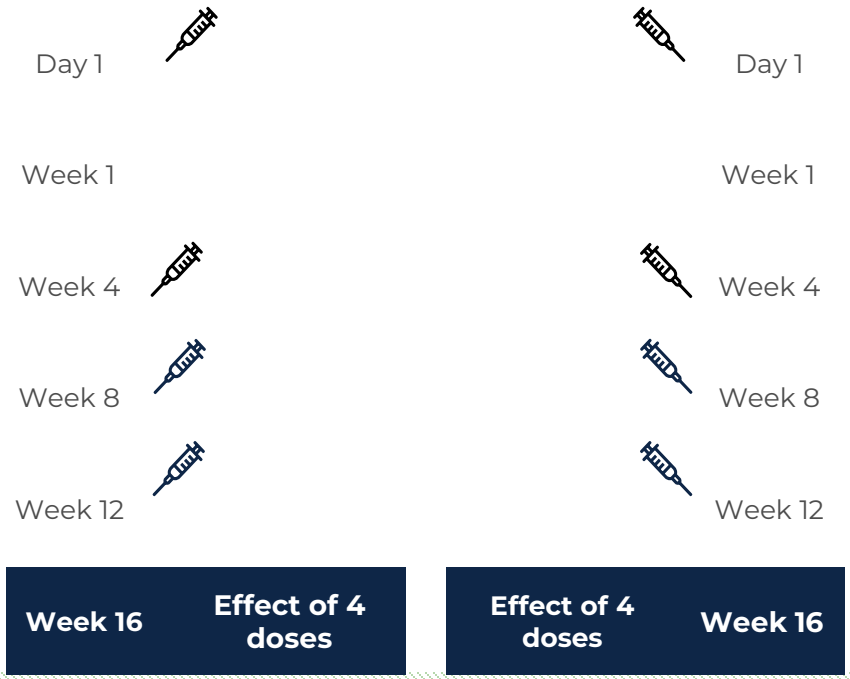
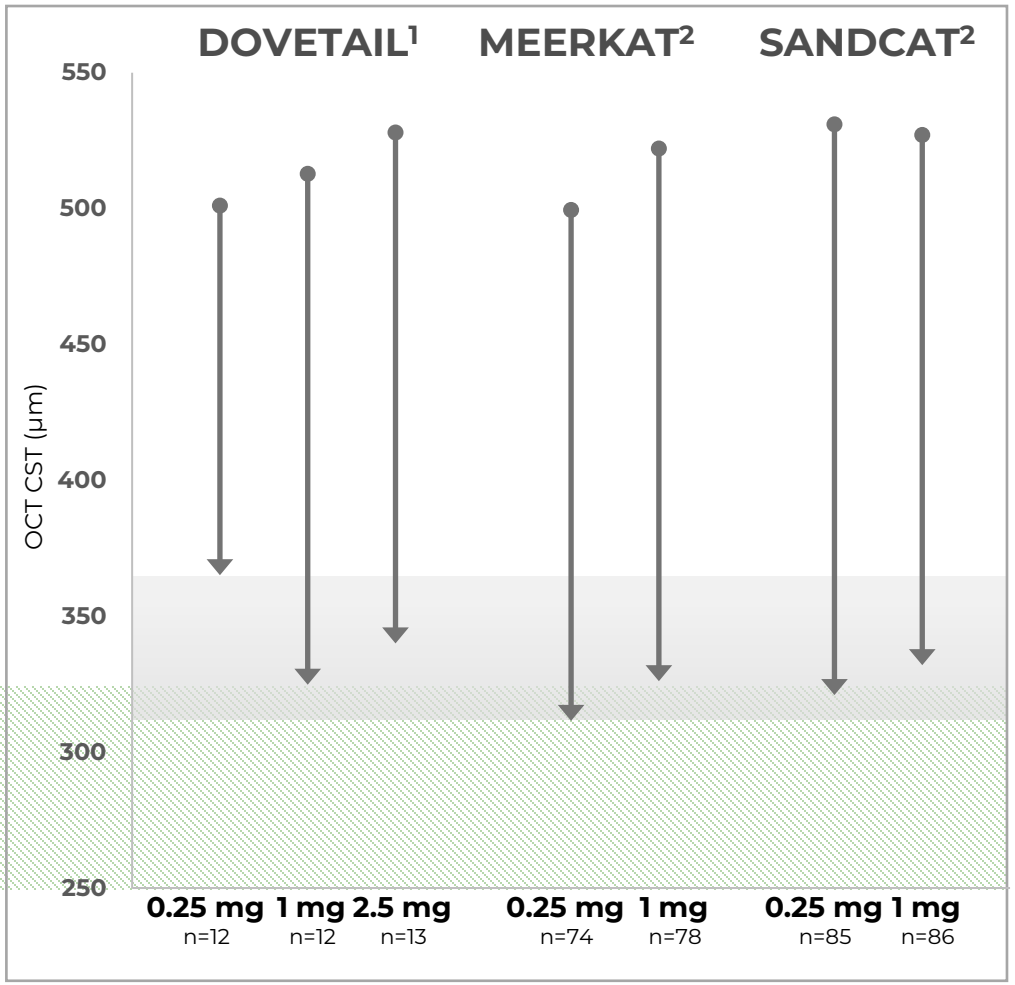


Anti-IL-6, VEGF trap
KSI-101



Dryness (CST 250 – 325)

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



Anti-IL-6
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Actively Enrolling

The Phase 3 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

Individualized treatment criteria (Week 24-44)

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

Primary endpoint

BCVA change from baseline to the average of Week 20 and 24

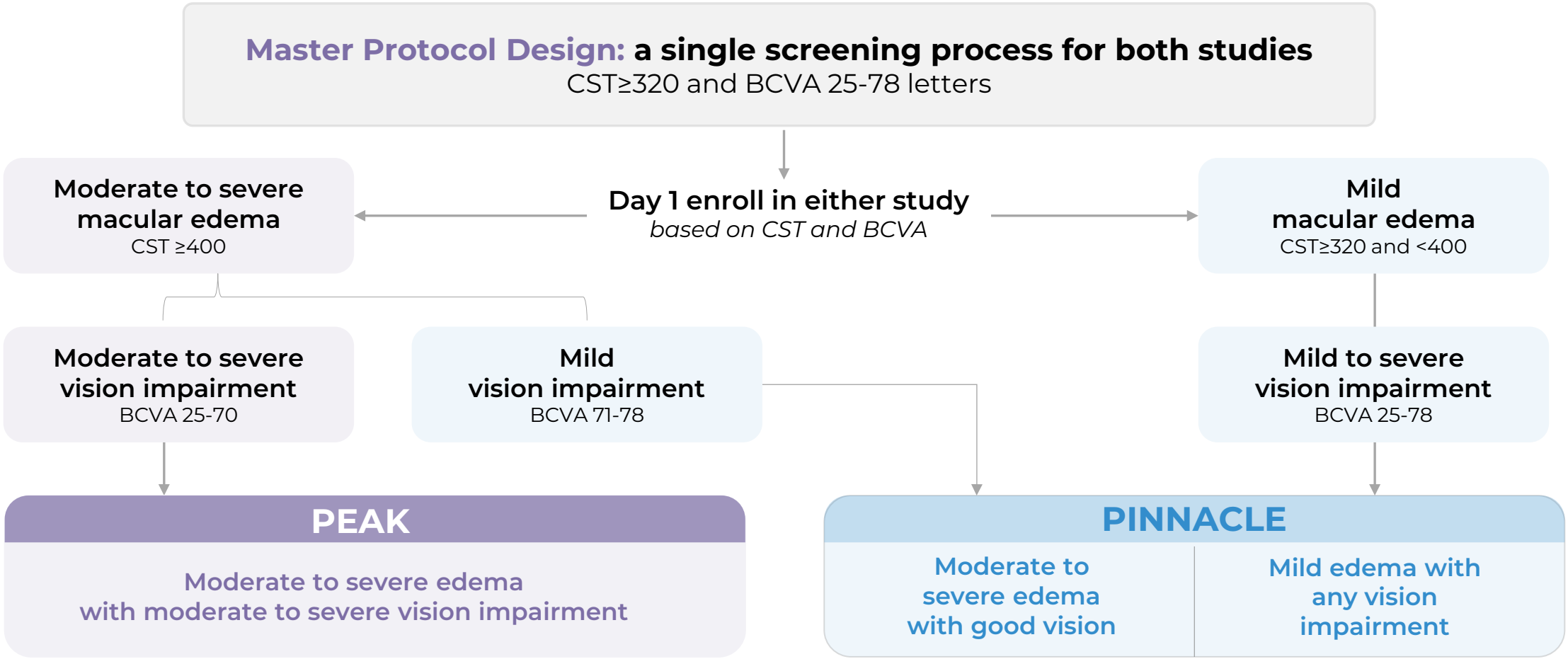
Key secondary endpoint

Proportion of patients in whom BCVA had improved by ≥15 letters from baseline to 24 weeks

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)

PEAK and PINNACLE master protocol design: a single screening process that enrolls distinct and complementary MESI patient populations

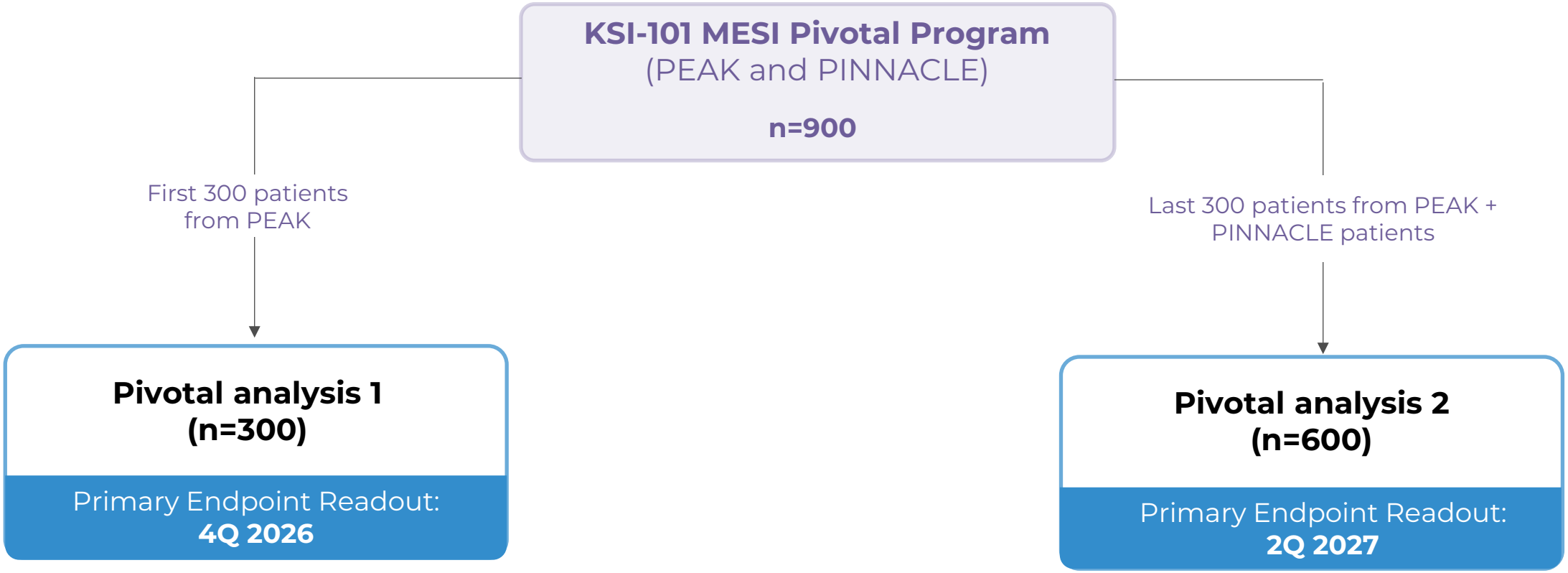


Two **distinct and complementary sub-populations** will be studied
Both studies to **run concurrently** in all study sites
Covers a **wide spectrum** of MESI patients

KODIAK

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

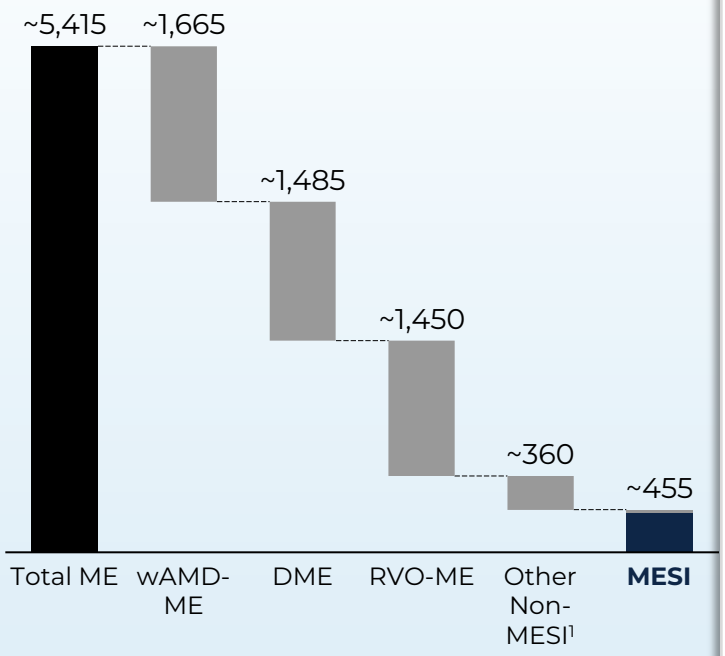
- Patient enrollment faster than expected
- No major changes to expected timelines are anticipated
- Pivotal program aligned with FDA in type C meeting

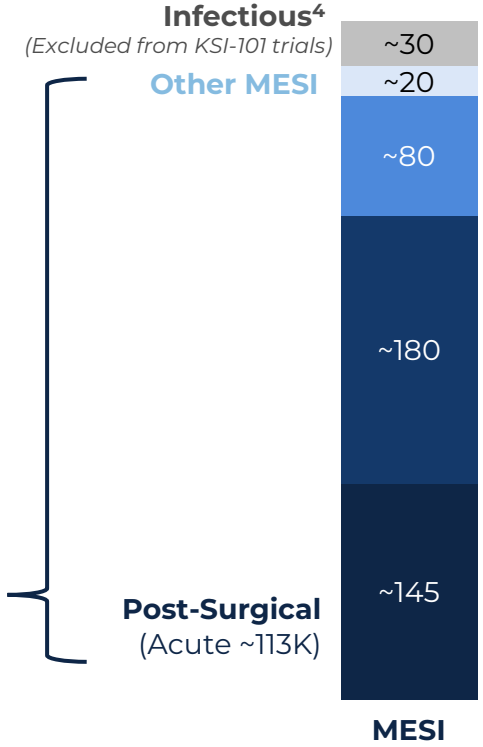


MESI affects ~450,000 US patients (~300,000 trial eligible) often reported by etiology (defined autoimmune, idiopathic, surgical, infectious and other)

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

U.S. MESI EPIDEMIOLOGY
Estimated by two methodologies – method 1 (2025) Thousands of patients





Other MESI
(inflammatory CNV ~6K) *

Defined Autoimmune Related*

Idiopathic
(Etiology unknown)*

Post-Surgical
(Refractory ~32K) *

** = KSI-101 PEAK and PINNACLE TRIAL INCLUSION*

DEFINED AUTOIMMUNE ME[3]

HLA-B27	~20K
Ankylosing spondylitis	~15K
Multiple sclerosis	~10K
Idiopathic Multifocal Choroiditis	~7K
Sarcoidosis	~7K
Serpiginous choroidopathy	~6K
Vogt-Koyanagi-Harada	~4K

OTHER MESI

Scleritis	~10K
Inflammatory CNV	~6K

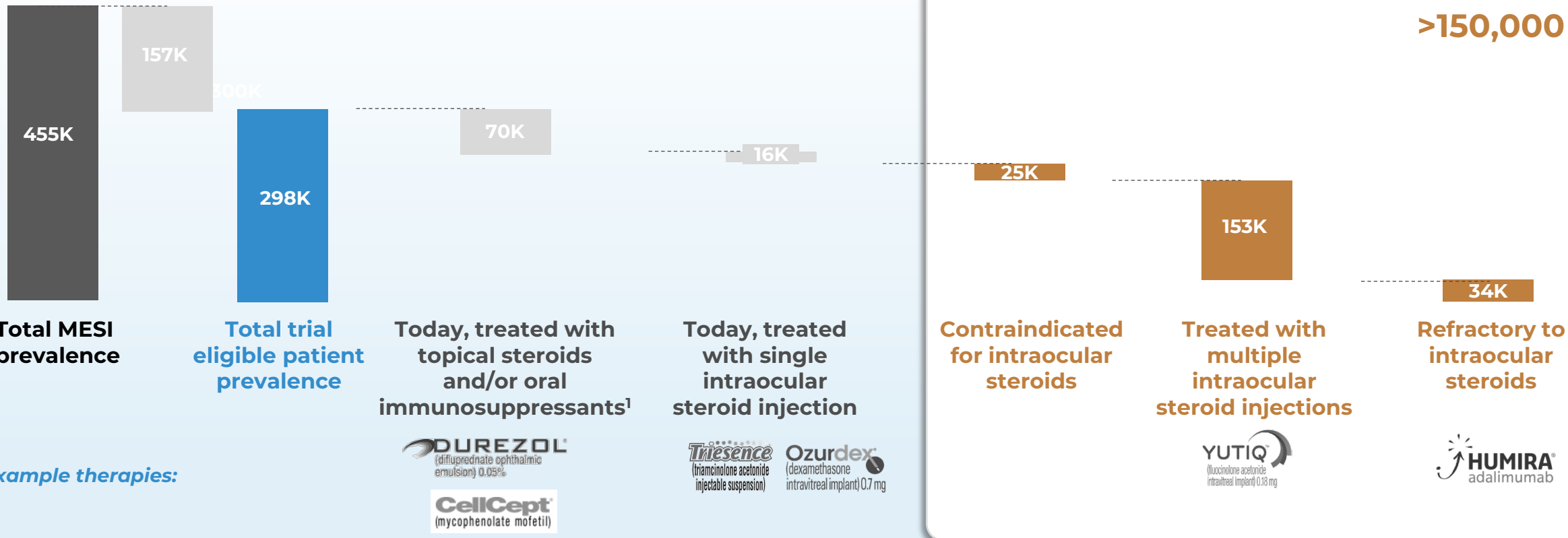
POST-SURGICAL ME[2]

Pars Plana Vitrectomy	~60K
Glaucoma surgery	~50K
Cataract surgery	~30K

[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 retinopexy; [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; 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



KSI-101 initial addressable population is >150,000

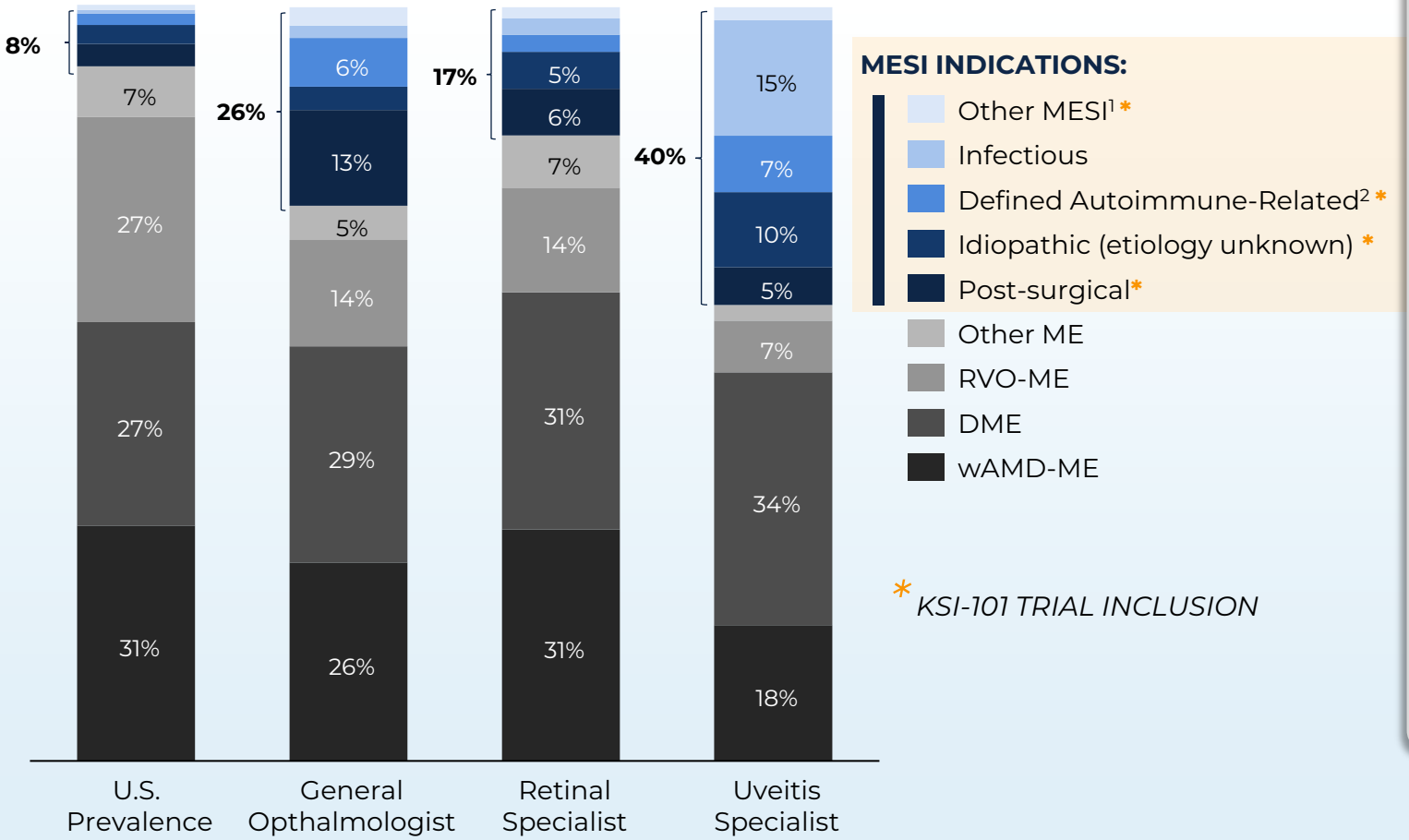
Example therapies:

Note: [1] First line includes eye drops or systemic immunnosuppressants. 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

OPHTHALMOLOGY PRACTICE BREAKDOWN – Method 2
% of ME patients by patient subtype across physician types

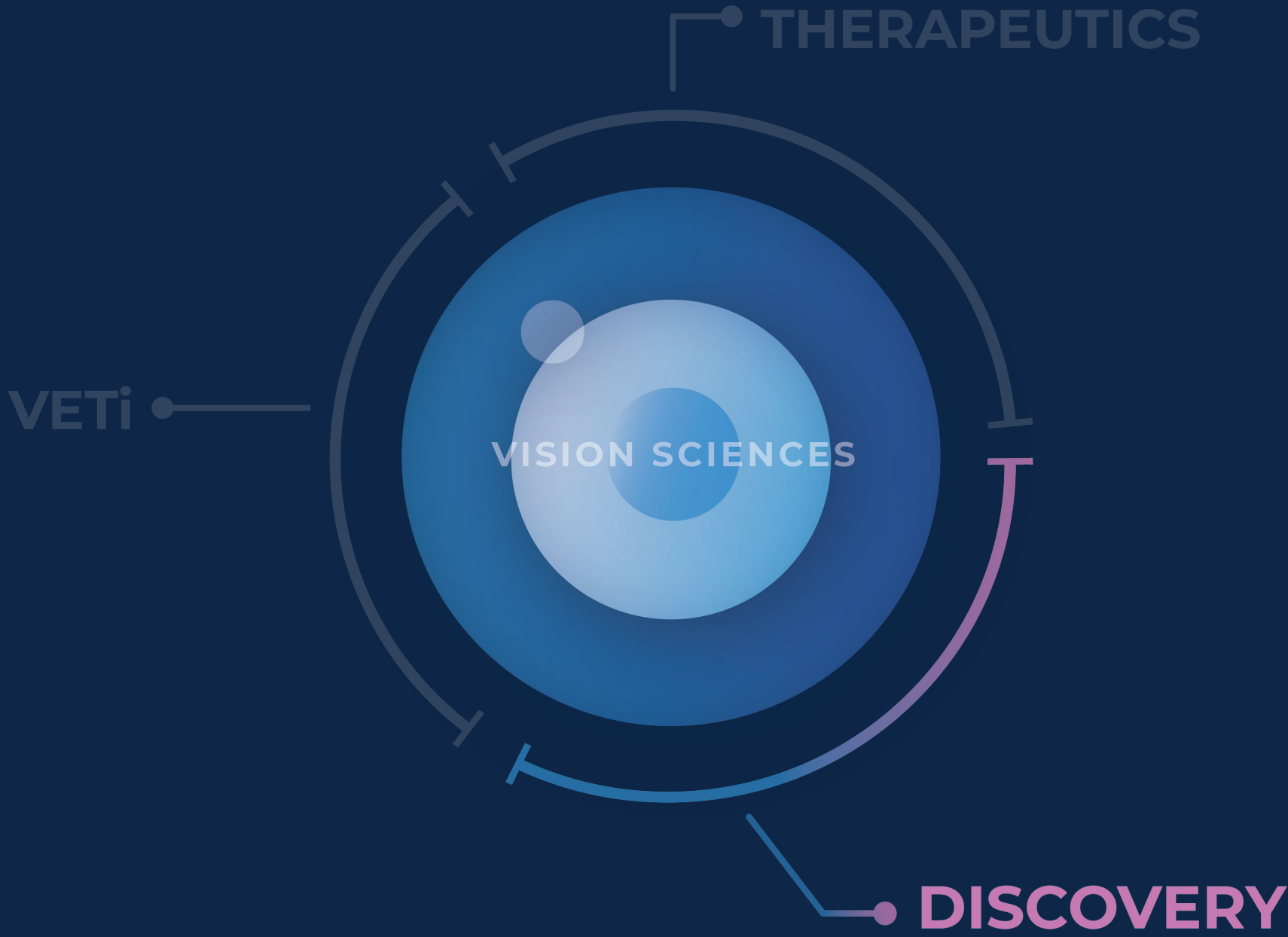


* *KSI-101 TRIAL INCLUSION*

- **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: [1]Other causes can include RP, scleritis, neoplasms, drug-induced, and trauma-induced; [2] Non-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)


THERAPEUTICS
VETi
VISION SCIENCES
DISCOVERY

Discovery: We are broadening our portfolio of investigational medicines to tackle additional high prevalence causes of severe vision loss

Ocular Inflammation

Pre-IND

Bispecific Antibodies:

KSI-102: anti-IL-6, anti-TNFα
KSI-103: anti-IL-6, anti-IL-1

Geographic Atrophy

Pre-clinical

Bispecific Antibodies:
anti-IL-6 , anti-complement;
anti-VEGF, anti-complement

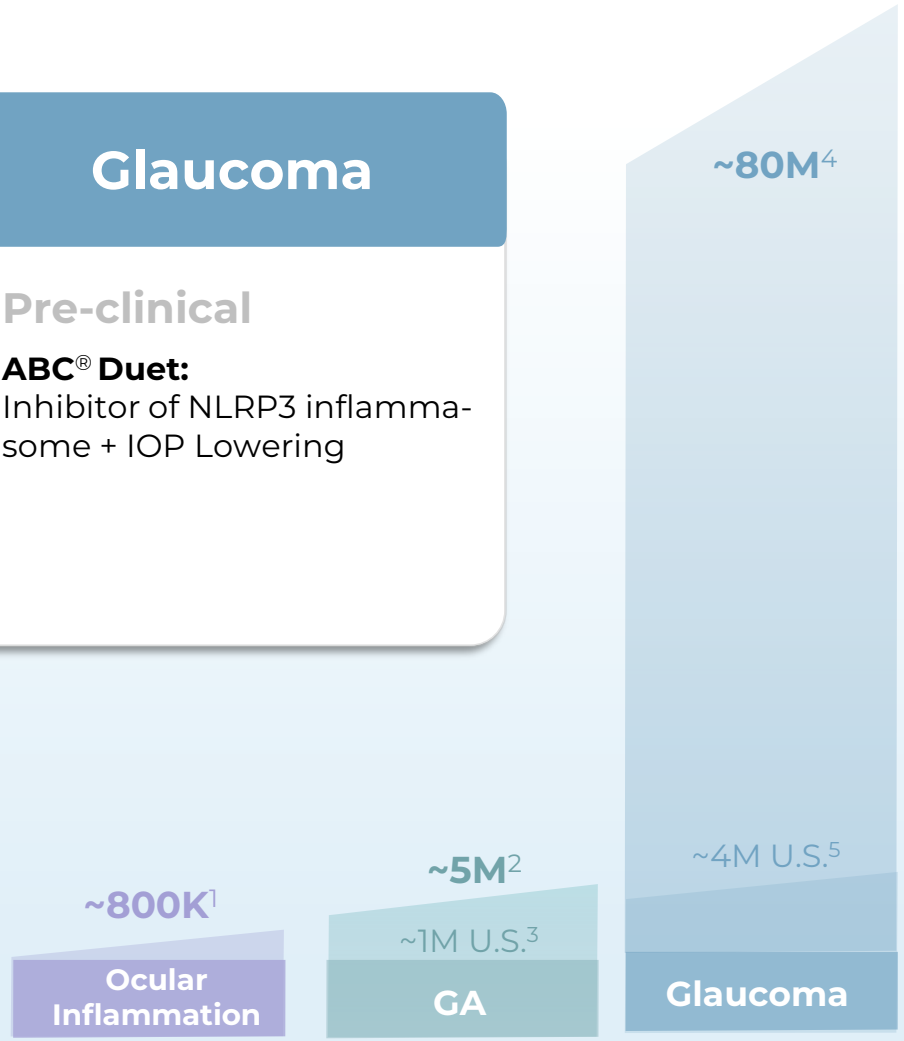
ABC® Duet:
Inhibitor of NLRP3 inflamma-some + Complement

Glaucoma

Pre-clinical

ABC® Duet:
Inhibitor of NLRP3 inflamma-some + IOP Lowering

Estimated global prevalence

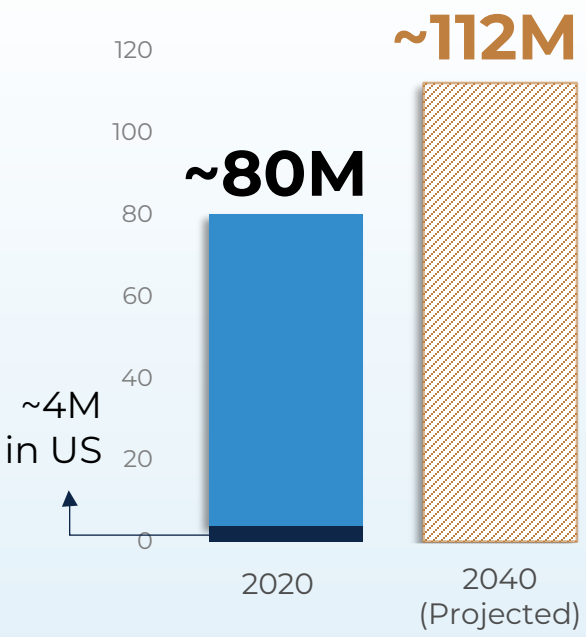


ABC: antibody biopolymer conjugate
GA: geographic atrophy

1. Nila Kirupaharan et al. *Invest. Ophthalmol. Vis. Sci.* 2024;65(7):6510. 2. Rudnicka AR, et al. *Ophthalmology* 2012;119:571–580. 3. Wong WL, et al. *Lancet Glob Health* 2014;2:e106–116. 4. Allison K et al. *Cureus*. 2020 Nov 24;12(11):e11686. 5. Ehrlich JR, et al. *JAMA Ophthalmol.* 2024;142(11):1046–1053.

Glaucoma is the leading cause of irreversible blindness worldwide with no approved neuroprotective treatments

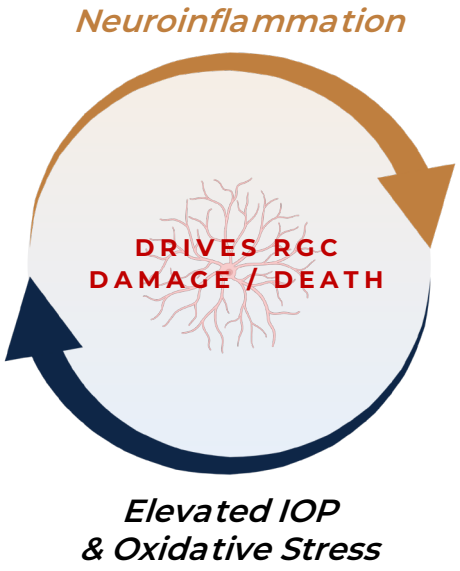
Worldwide prevalence of glaucoma[1]



- U.S. represents largest single market[2]
- Global market expected to grow due to aging population; valued at **~$9B in 2025**[2]

Glaucoma is a progressive optic neuropathy driven by neuroinflammation

- **Current therapies focus on lowering intraocular pressure (IOP)** as it is the only modifiable risk factor
- **The NLRP3 inflammasome complex is a key driver of neuroinflammation** that results in optic neuropathy[3]
- Today's small molecule delivery technologies (eye drops, orally, intravitreally) **are limited in their ability** to reach retinal and optic nerve targets

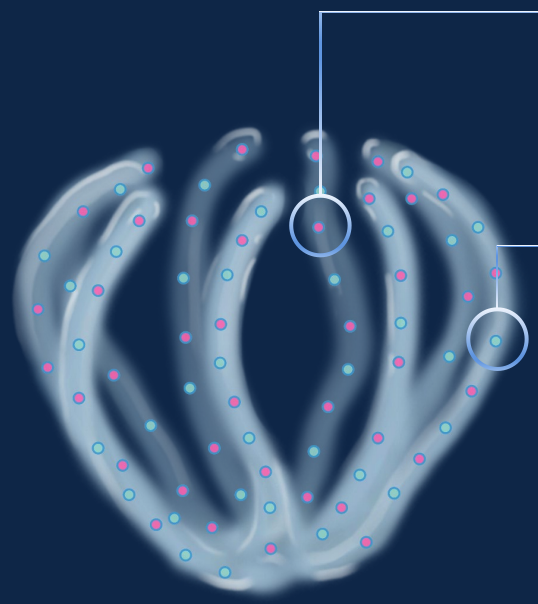


There is an unmet need to develop new therapies that target the underlying neuroinflammation that drives optic neuropathy

KODIAK

1. Allison K et al. *Cureus*. 2020 Nov 24;12(11):e11686. 2. Precision Business Insights. Accessed May 2025.. 3. Coyle S, et al. *Biomolecules*. 2021 Aug 19;11(8):1239.

Glaucoma “duet”: a single molecule designed to target neuroinflammation (that causes optic neuropathy) *and* elevated IOP simultaneously



Glaucoma “Duet”

Biopolymer with two small molecules (“BCD”)

Disease Modifying NLRP3 Inhibitor

Targets the NLRP3 inflammasome complex that drives neuroinflammation causing optic neuropathy

IOP Lowering Small Molecule

Addresses the IOP elevation stressor; NLRP3 inhibitor demonstrates potential to provide *additional* IOP reduction effects

ABCD Platform for Extended Durability

High molecular weight to increase ocular half-life, providing potential for a quarterly dosed intravitreal therapy

Favorable Safety Demonstrated

Zenkuda and the ABCD platform demonstrated a favorable safety profile across multiple pivotal studies (>13,000 intravitreal injections)

Administered via intravitreal injection

Neuroprotective

+

Reduces IOP

+

Durable

+

Safe

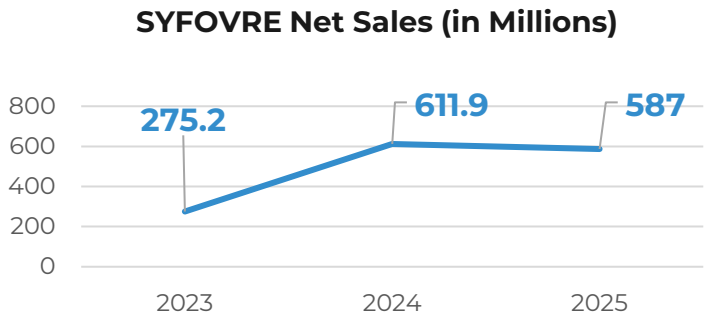
4 key attributes needed in a next generation glaucoma therapy

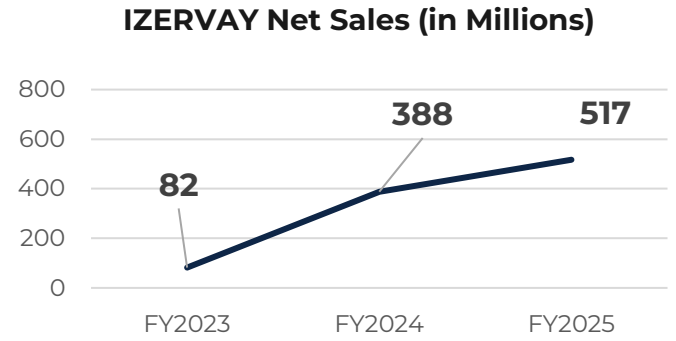
KODIAK

Geographic atrophy is a valuable market with a high unmet need

- Geographic atrophy (GA) is the advanced form of dry age-related macular degeneration, **affecting ~1M people in the U.S.**
- GA is characterized by progressive retinal atrophy that can extend to the macula and fovea, **leading to irreversible vision loss**
- **GA is driven by multiple inflammatory pathways, and current single target therapies have limitations**
  - The C3 complement inhibitor (Syfovre) and C5 complement inhibitor (Izervay) have been validated for GA. Despite their commercial success, monotherapies:
    - Provide modest clinical benefit
    - Require frequent intravitreal injections
    - Have been associated with an increased risk of conversion to choroidal neovascularization

~$1B in net sales for GA monotherapies in 2025



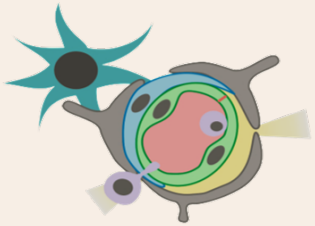


Astellas and Apellis (acquired by Biogen) company reports and SEC filings.

GA is a complex interplay of complement, inflammation, and angiogenic pathways

Smoking
Age
Diabetes

NLRP3 inflammasome, IL-1β, IL-6

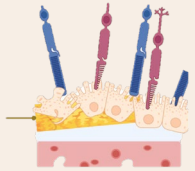


Inflammation

Complement

Human Genetics

VEGF



Hypoxia, lipid metabolism

Advanced AMD/GA

This interplay drives both GA and neovascular AMD, which may co-exist within the same patient or eye—**emphasizing the need for therapeutic strategies that address multiple mechanisms**

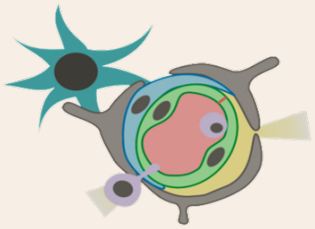
Kodiak's multi-asset pipeline in GA is designed to address the multifactorial nature of the disease

NLRP3 inflammasome, IL-1β, IL-6

Complement

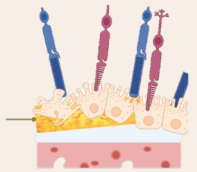
VEGF

Smoking
Age
Diabetes



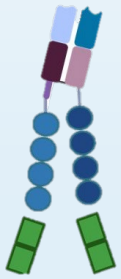
Inflammation

Human Genetics

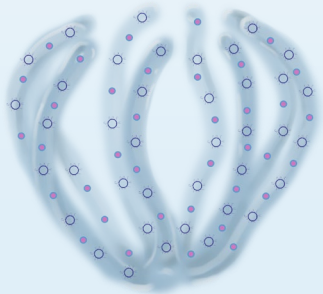


Hypoxia, lipid metabolism

Advanced AMD/GA



IL-6 + complement

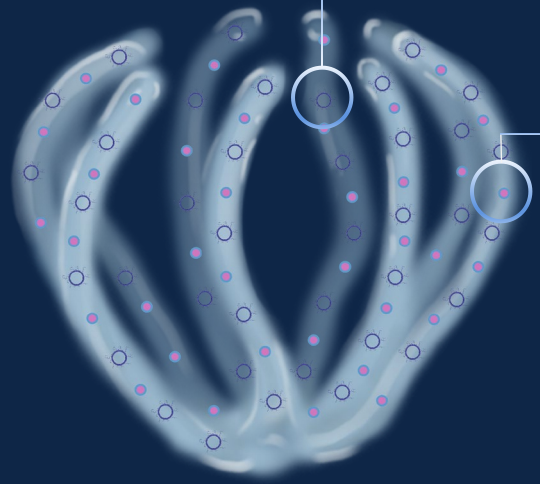


Inhibitors of NLRP3 and Complement

VEGF + complement

KODIAK

GA “duet”: Dual inhibition of validated complement pathways along with the inflammasome pathway (NLRP3)



GA “Duet”

Biopolymer with one macrocycle peptide and one small molecule

Peptide Inhibitor of Complement

Anti-C3 and anti-C5 have been validated for GA, but current therapies do not sufficiently halt disease progression and are limited by frequent injections

NLRP3 Inhibitor Small Molecule

Prevents inflammasome activation that contributes to AMD pathology

ABCD Platform for Extended Durability

High molecular weight to increase ocular half-life, providing potential for a **quarterly dosed intravitreal therapy**

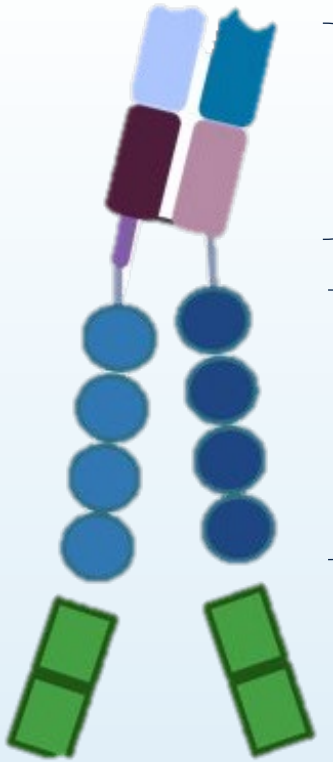
Dual inhibition of inflammatory and complement pathways may better address the multifactorial biology driving GA progression

Administered via intravitreal injection

GA bispecific antibody: Combining validated complement regulation with VEGF or IL-6 inhibition as a therapeutic target for GA

IL-6 + Complement

VEGF + Complement



Anti-IL-6 Fab

Complement Receptor 1

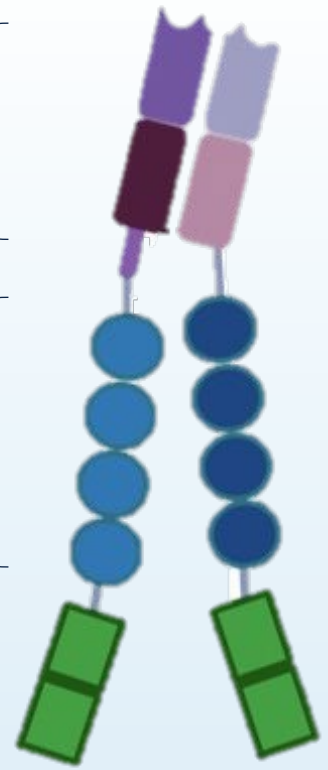
Reduces inflammation-driven GA progression

Prevents **wet-AMD conversion**

Inhibits C3 convertase formation, simultaneously blocking all complement pathways at low concentrations

Anti-VEGF Fab

Complement Receptor 1



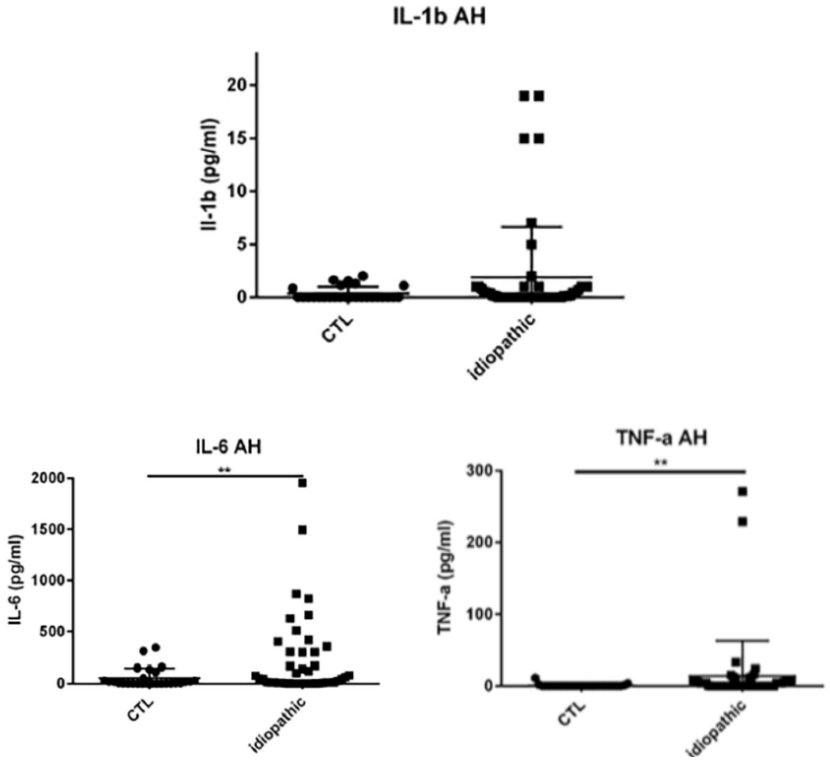
Administered via intravitreal injection

Ocular inflammatory disease: the 4th leading cause of vision loss among working-age adults with no approved biologics

- **~1/3** of patients with ocular inflammation **develop macular edema**, the leading cause of vision loss in this population
- **Steroids** remain the mainstay treatment but can **cause significant and permanent ocular adverse effects**, especially with long-term use or high doses
- Elevated levels of pro-inflammatory cytokines including TNF-α and IL-6 play distinct yet complementary roles in driving inflammation and vascular permeability

Therapies targeting these cytokines individually may not fully address both disease drivers

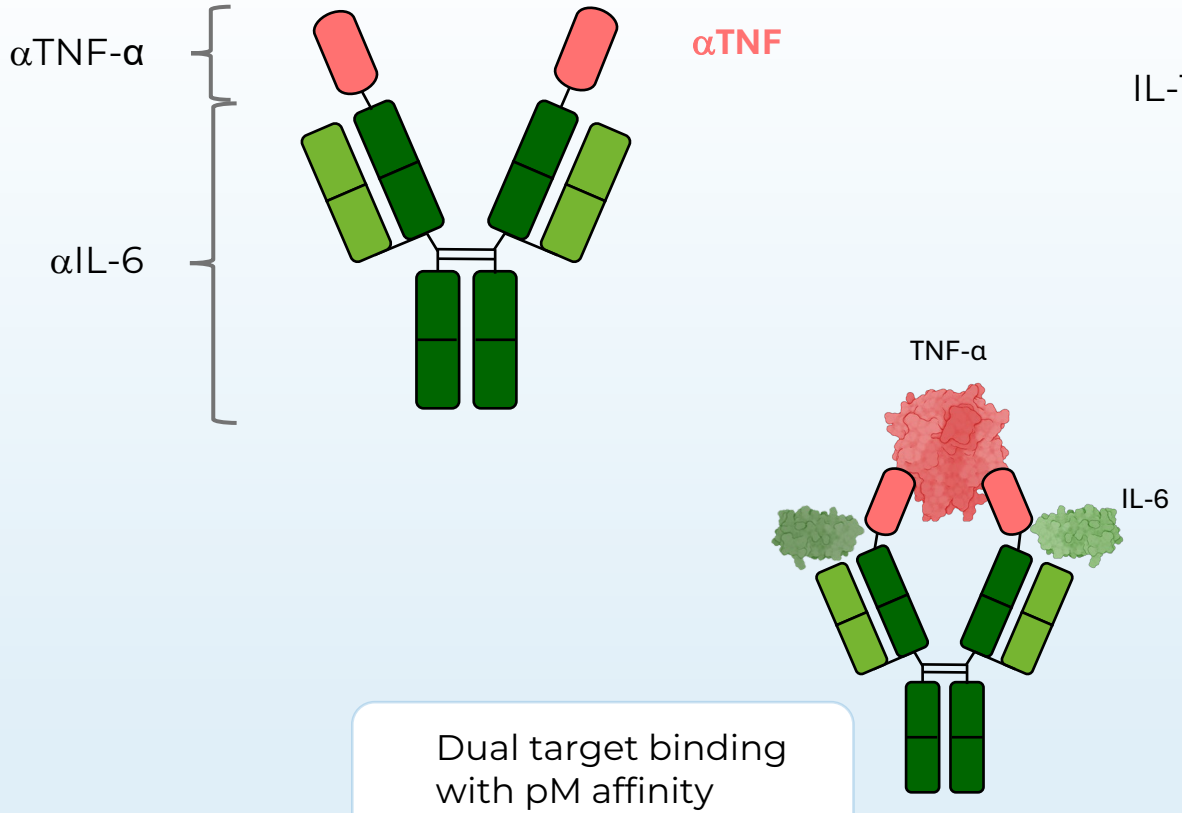
TNF-α, IL-6 and IL-1 levels are upregulated in patients with ocular inflammation



Dot plots comparing immune mediator levels in aqueous humor (AH) of patients with idiopathic uveitis and noninflammatory controls patients (cataract age-related)

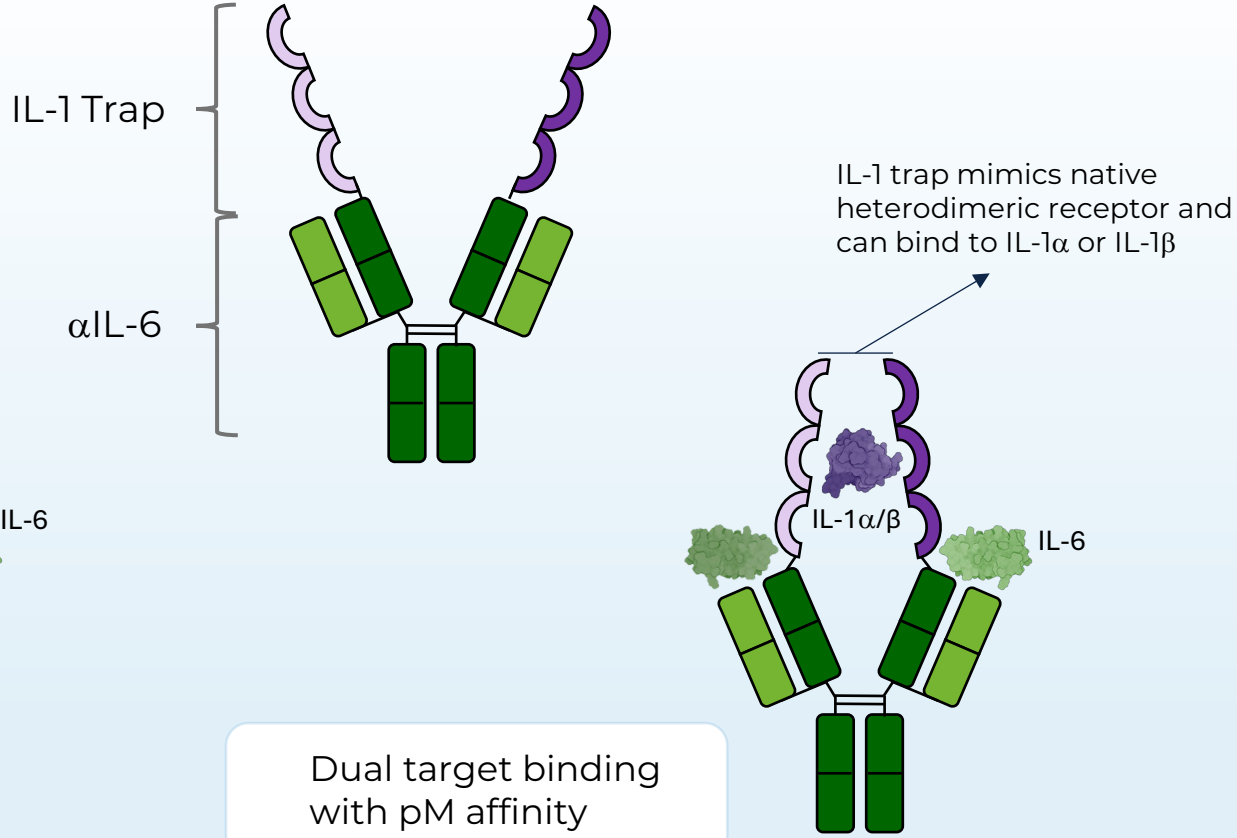
KSI-102 and KSI-103: Engineered to inhibit TNF-α/IL-6 and IL-1/IL-6 through dual cytokine targeting

KSI-102 (αTNF-α / αIL-6 bispecific)



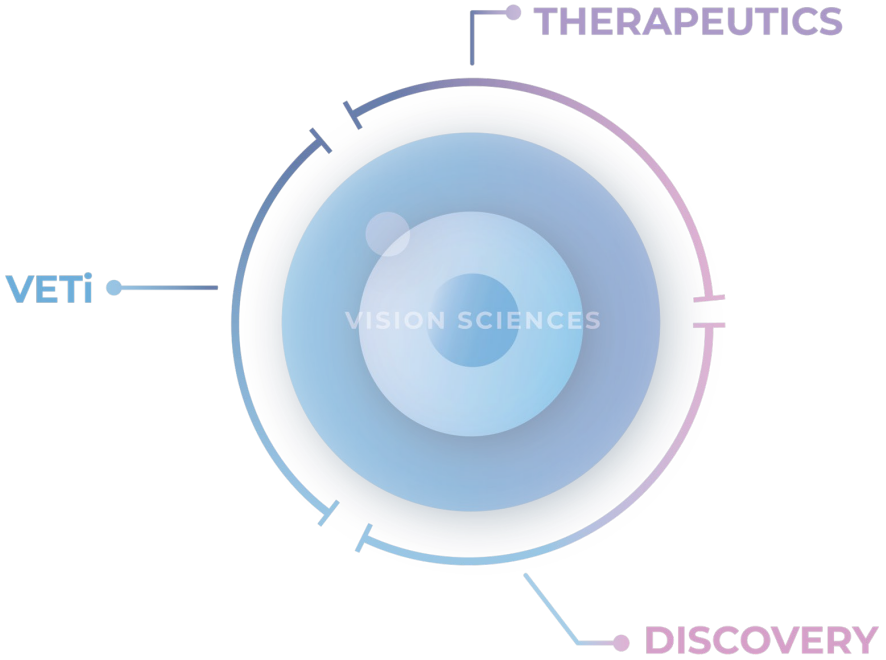
Dual target binding with pM affinity

KSI-103 (IL-1 trap / αIL-6 bispecific)



Dual target binding with pM affinity

Kodiak is gathering speed. Our position at the intersection of retinal biology, therapeutics and optics/AI is a genuine source of differentiation



15+ years experience

Retinal biology, disease and therapeutics development

VETi: Visual Engagement Technology and imager