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THE OPHTHALMOLOGY MEDICINES COMPANY

Jefferies Global Healthcare Conference

June 2024

SPECIAL NOTE REGARDING

FORWARD-LOOKING STATEMENTS

These slides contain “forward-looking statements.” Forward-looking statements are based on our current expectations of future events and are subject to risks and uncertainties that could cause actual results to differ materially and adversely from those in or implied by such forward-looking statements. For a discussion of risks and uncertainties and other important factors, any of which could cause our actual results to differ from those contained in the forward-looking statements, see the section entitled “Risk Factors” in our most recent Form 10-K, as well as discussions of potential risks, uncertainties, and other important factors in our subsequent filings with the SEC. These forward-looking statements speak only as of the date hereof and Kodiak undertakes no obligation to update forward-looking statements, and readers are cautioned not to place undue reliance on such forward-looking statements. Kodiak®, Kodiak Sciences®, ABC™, ABC Platform™, and the Kodiak logo are registered trademarks or trademarks of Kodiak Sciences Inc. in various global jurisdictions.

THE OPHTHALMOLOGY MEDICINES COMPANY

OUR MISSION

1 TRAILBLAZING SCIENCE

Our creative and thoughtful foundation

2 "GO-TO" MEDICINES

Our challenge to the status quo

3 SINGULAR FOCUS IN OPHTHALMOLOGY

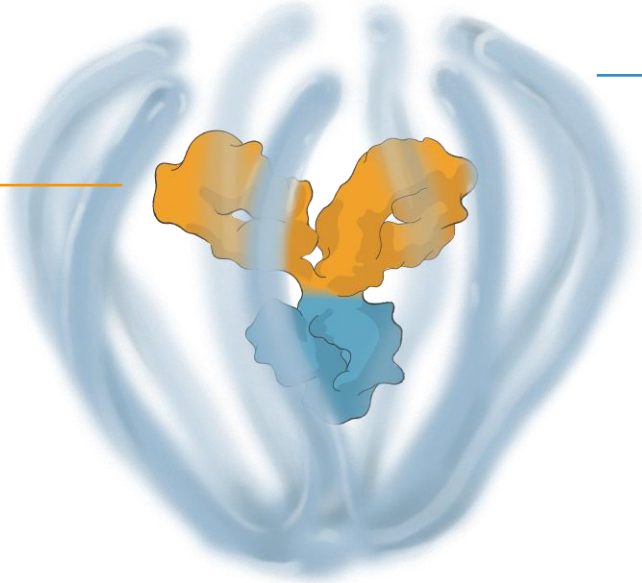
Our 24 / 7 / 365

ABC Platform:
Enabling multi-mechanism therapies empowered for durability

- Our Antibody Biopolymer Conjugate ("ABC") Platform combines the best durability with the right efficacy and is the foundation of tarcocimab tedromer and KSI-501, two "ABC" investigational medicines in late-phase clinical development

Antibody

Engineered to exhibit high binding affinity and specificity. Any protein therapeutic including monospecific, bispecific and trispecific antibodies or proteins can be conjugated to the biopolymer via a stable, site-specific linkage



Biopolymer

Engineered to make medicines last longer and extend their therapeutic benefit. It is also powered to combine multiple modalities

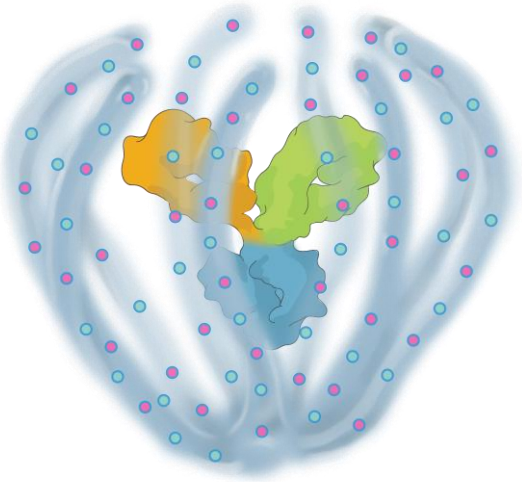
The biopolymer is optically clear and made of phosphorylcholine, the primary hydrophilic component of human cell membranes

Antibody Biopolymer Conjugate (“ABC”)

ABC Platform Evolution:
Powering multi-specific, high “DAR” medicines

- We have expanded our early research pipeline of duet and triplet inhibitors that embed small molecules and other active pharmaceutical ingredients ("API"), such as oligonucleotides and peptides, in the biopolymer backbone to enable targeted, high drug-antibody ratio ("DAR") medicines

The diverse API's are embedded in the biopolymer backbone and designed to be released over time to achieve targeted, multi-specific and tailored modulation of biological pathways



The unique combination of high “DAR” and tailored therapeutic benefit offers potential for broad application to multifactorial ophthalmic and systemic diseases

Our product candidates: a portfolio of three clinical programs to address key limitations of today's therapies across a broad spectrum of retinal diseases

"ABC" Platform-derived biologics:
The best durability with the right efficacy for high-prevalence retinal vascular diseases

Tarcocimab
Anti-VEGF ABC

- **Objective:** to have a compelling first-line durability profile without compromising immediacy
- Longest-acting anti-VEGF biologic (6-month predominant) while preserving the flexibility to dose monthly
- **Enhanced 50 mg/mL formulation**

KSI-501
Bispecific Anti-IL-6, VEGF Trap "ABC"

- **Objective:** to address the opportunity for first-line efficacy with the best durability
- First-in-class bispecific "ABC" designed to address retinal inflammation and vascular permeability simultaneously
- Reflects 10 years of learnings of the "ABC" platform to maximize each patient's efficacy and durability potential
- **Enhanced 50 mg/mL formulation**

Unconjugated biologic:
For inflammatory retinal diseases

KSI-101
Bispecific Anti-IL-6, VEGF Trap Protein

- **Objective:** to address the underlying disease mechanisms of macular edema secondary to inflammation for which no approved intravitreal biologic therapies exist today
- First-in-class bispecific protein designed to address retinal inflammation and vascular permeability simultaneously
- **100 mg/mL formulation provides high strength and potency**

Science Updates for our "ABC" Platform Biologics (Tarcocimab and KSI-501)

1. Supported by a true science of durability (conjugate design, animal ocular t½ data and human ocular t½ data) in contrast to current anti-VEGFs
2. Enhanced formulation of conjugated and unconjugated forms balances towards durability without compromising immediacy

VEGF: vascular endothelial growth factor; IL-6: interleukin 6; mAb: monoclonal antibody

TARCOCIMAB TEDROMER

Our objective is for tarcocimab to have a compelling first-line durability profile without compromising immediacy for patients with high-prevalence retinal vascular diseases

- Five Phase 3 studies are planned for inclusion in a Biologic License Application (BLA)
- Three are complete with compelling durability demonstrated and two are in process

Completed Phase 3 studies:
Primary endpoint met and extended durability demonstrated

Wet AMD	Retinal vein occlusion	Diabetic retinopathy
DAYLIGHT Study	BEACON Study	GLOW1 Study

Two new Phase 3 studies in process:
Using the enhanced formulation of tarcocimab

Diabetic retinopathy	Wet AMD
GLOW2 Study Actively enrolling	DAYBREAK Study Enrollment start mid-2024

Planned BLA package

- AMD: age-related macular degeneration; BEACON: NCT04592419; GLOW1: NCT05066230; DAYLIGHT: NCT04964089; GLOW2: NCT06270836

Three successful Phase 3 studies in diabetic retinopathy (DR), retinal vein occlusion (RVO) and wet AMD with compelling durability demonstrated

	Study design	Primary endpoint	Extended durability	
Diabetic retinopathy Phase 3 GLOW1 Study	• Superiority study • tarcocimab **Q24W** after **3** initiating doses vs sham	✓	✓	Signature durability demonstrated with all patients on 6-month dosing
Retinal vein occlusion Phase 3 BEACON Study	• Tarcocimab **Q8W** after **2** monthly loading doses vs aflibercept Q4W	✓	✓	Doubled treatment interval at primary endpoint (month 6) and ~50% of patients on 6-month dosing at Year 1
Wet AMD Phase 3 DAYLIGHT Study	• Tarcocimab **Q4W** vs aflibercept Q8W after 3 monthly loading doses	✓	Not Applicable	Monthly dosing of tarcocimab demonstrated favorable safety and non-inferior efficacy at Year 1

- In addition to these studies, tarcocimab was also studied in the Phase 2b/3 DAZZLE study in wet AMD and in the Phase 3 GLEAM and GLIMMER studies in DME. These studies did not meet primary endpoint but did demonstrate strong 5 and 6-month durability in the majority of patients.

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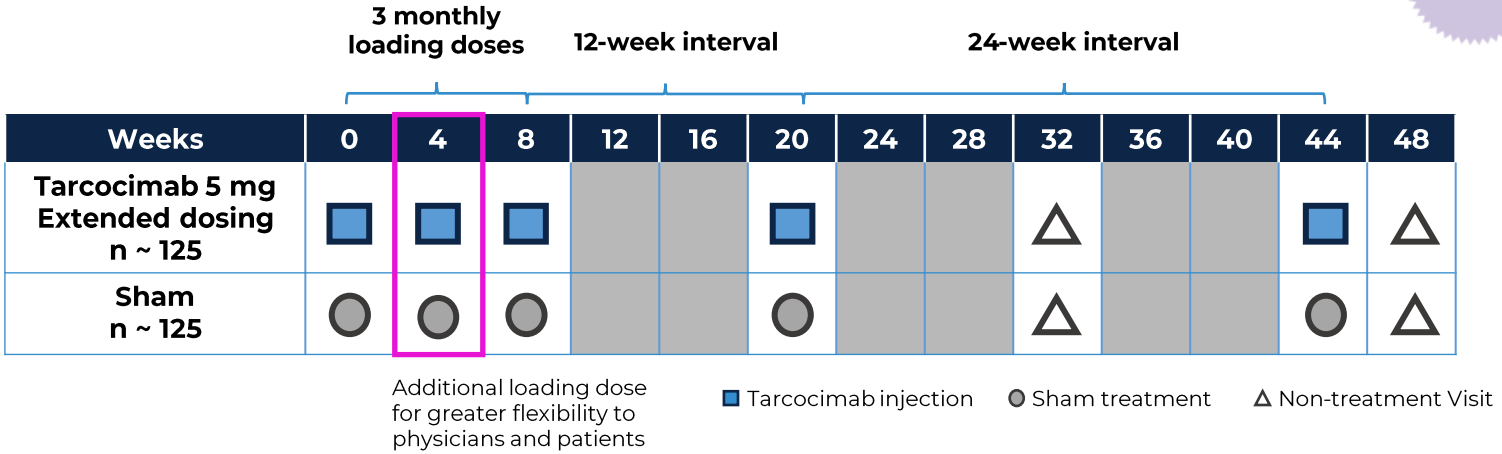
DME: diabetic macular edema. AMD: age-related macular degeneration.
Q4W: every 4-week dosing; Q8W: every 8-week dosing; Q24W: every 24-week dosing

New Phase 3 study: GLOW2 in Diabetic Retinopathy

- GLOW2 features a similar study design as the successful GLOW1 study, with the benefit of an additional 3rd monthly loading dose (Week 0, 4, 8) for greater flexibility to physicians and patients

Actively recruiting



Primary endpoint	• Proportion of eyes improving ≥2 steps on DRSS from baseline at Week 48
Key secondary endpoints	• Proportion of eyes developing sight-threatening complications* • Proportion of eyes improving ≥3 steps on DRSS from baseline

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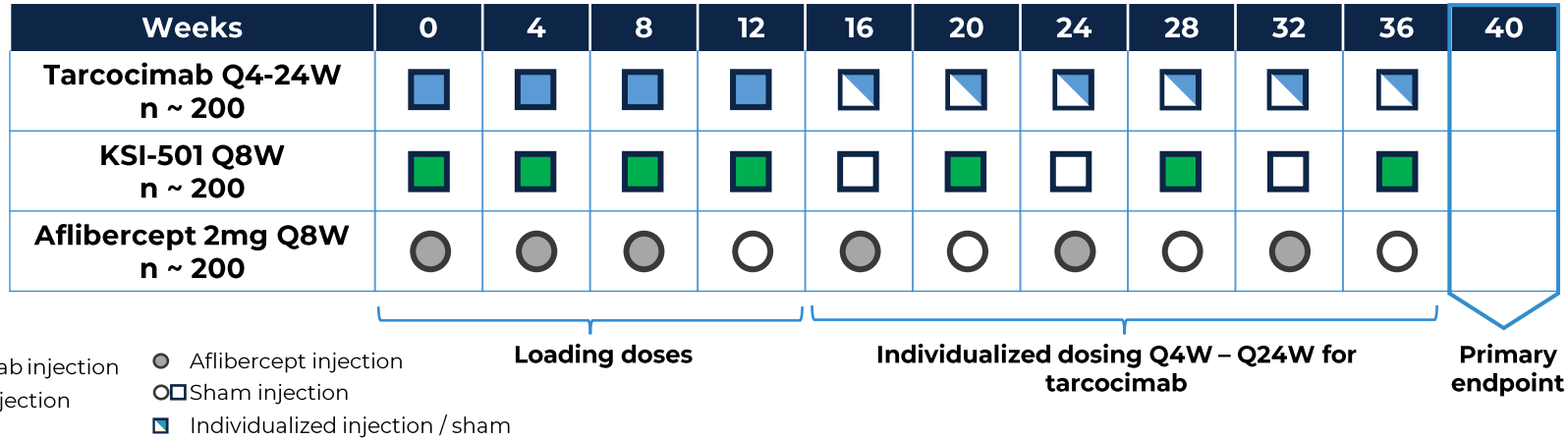
*Sight-threatening complications were defined as: proliferative diabetic retinopathy (PDR), Vitreous hemorrhage or tractional retinal detachment due to PDR, diabetic macular edema, and anterior segment neovascularization. DRSS: diabetic retinopathy severity score; GLOW 1 Study NCT05066230.

New Phase 3 study: DAYBREAK in Wet AMD

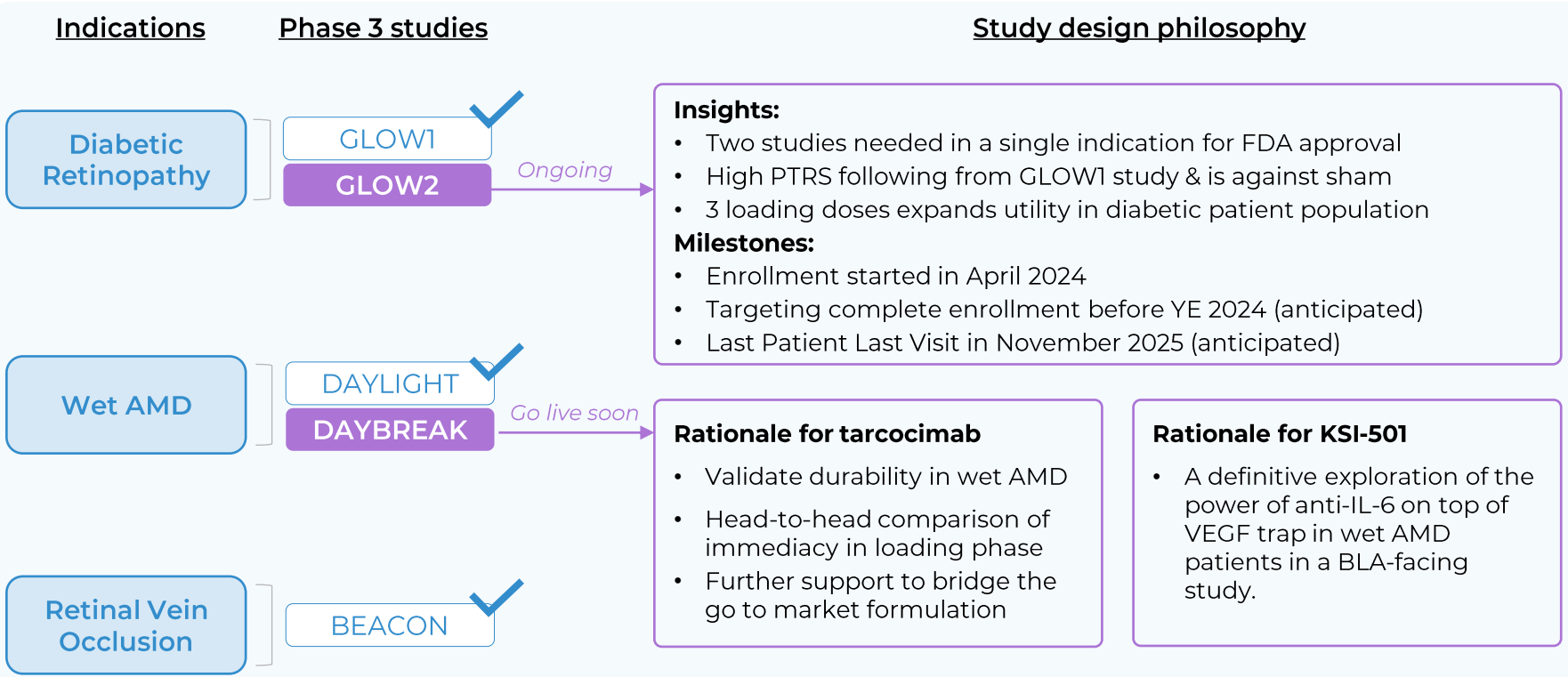
- DAYBREAK is designed to include tarcocimab and KSI-501 investigational groups vs aflibercept
- The objective is to evaluate the efficacy, safety and durability of tarcocimab and KSI-501 and to support registration in wet AMD for both investigational medicines



Primary endpoint	• Mean change in BCVA (ETDRS letters) from baseline to Week 40
Key secondary endpoints	• Proportion of participants who gain or lose ≥ 5, ≥ 10, ≥ 15 ETDRS letters from baseline to Week 40 and over time • Mean change in OCT CST from baseline to Week 40 and over time • Proportion of tarcocimab participants who are treated on a Q4W, Q8W, Q12W, Q16W, Q20W and Q24W dosing interval

BCVA: best corrected visual acuity; ETDRS: early treatment diabetic retinopathy study; OCT: optical coherence tomography; CST: central subfield thickness;

GLOW2 and DAYBREAK study: philosophy and rationale



Product vision for tarcocimab

Illustrative

"My experience with tarcocimab-treated patients in your trial is you have the durability but you didn't dry as well in the loading phase. But with a formulation of conjugated and unconjugated antibody, then you have fixed that, and you have a drug that primes itself and then takes patients longer. Together with monthly reimbursement where needed, I don't know why you wouldn't be a contender for first-line after step therapy from Avastin."

KSI-501

Designed to address the opportunity for improved efficacy with extended durability in high-prevalence retinal vascular diseases by targeting retinal inflammation and vascular permeability simultaneously

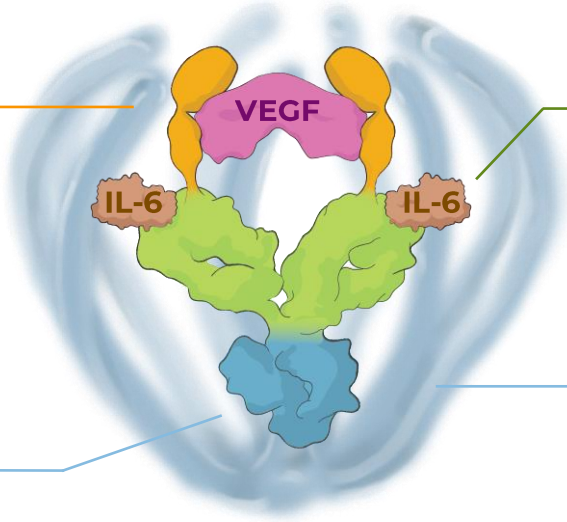
- Inflammation has been shown to play a significant role in high-prevalence retinal vascular diseases. However, no treatments exist that concurrently address vascular permeability and inflammation
- KSI-501 is designed to inhibit VEGF and interleukin-6 (IL-6), a pro-inflammatory cytokine and immune growth factor, combining two powerful mechanisms of action to address retinal vascular disease and the underlying inflammatory cascade

KSI-501 is a first-in-class bispecific designed for highly efficient binding to both IL-6 and VEGF, built on Kodiak's "ABC" platform

- The anti-permeability effect of VEGF inhibition is the primary effector, with the anti-inflammatory effect of IL-6 inhibition offering the potential for additional clinical benefits

VEGF Trap:
Broad VEGF inhibition

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



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

Modified Fc

Immunologically inert antibody

"ABC" Platform:
Extended durability

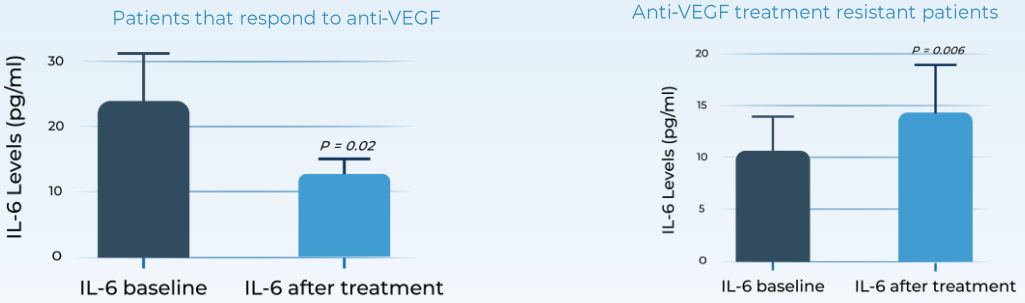
KSI-501 leverages our "ABC" platform with its signature durability of 5- to 6-months

50 mg/mL formulation of conjugated and unconjugated forms reflects 10 years of learnings of the "ABC" platform to maximize each patient's efficacy and durability potential

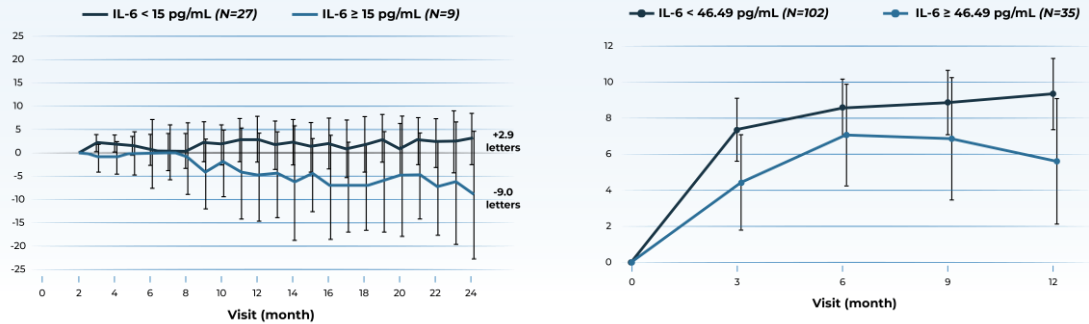
In addition to VEGF, IL-6 driven inflammation is implicated in retinal vascular disease

- IL-6 is a pro-inflammatory cytokine and immune growth factor implicated in the pathophysiology of multiple retinal diseases
  - Vitreous IL-6 levels are significantly elevated in retinal disease patients vs controls
  - IL-6 stimulates defective angiogenesis independent of VEGF and is implicated in anti-VEGF treatment resistance
  - Increased levels of IL-6 are associated with poor functional outcomes in wet AMD and diabetic macular edema patients treated with anti-VEGF monotherapy

Aqueous humor IL-6 levels significantly correlate with anti-VEGF treatment response in wAMD[1]



Higher levels of IL-6 in aqueous humor are correlated with poorer BCVA outcomes over time in retinal vascular diseases[2]



1. Adapted from Chalam et al. (2014). Journal of Ophthalmology, Article ID 502174. Mean with SEM plotted.
2. Sepah, Y.J., Do, D.V., Mesquida, M. et al. Aqueous humour interleukin-6 and vision outcomes with anti-vascular endothelial growth factor therapy. Eye (2024).

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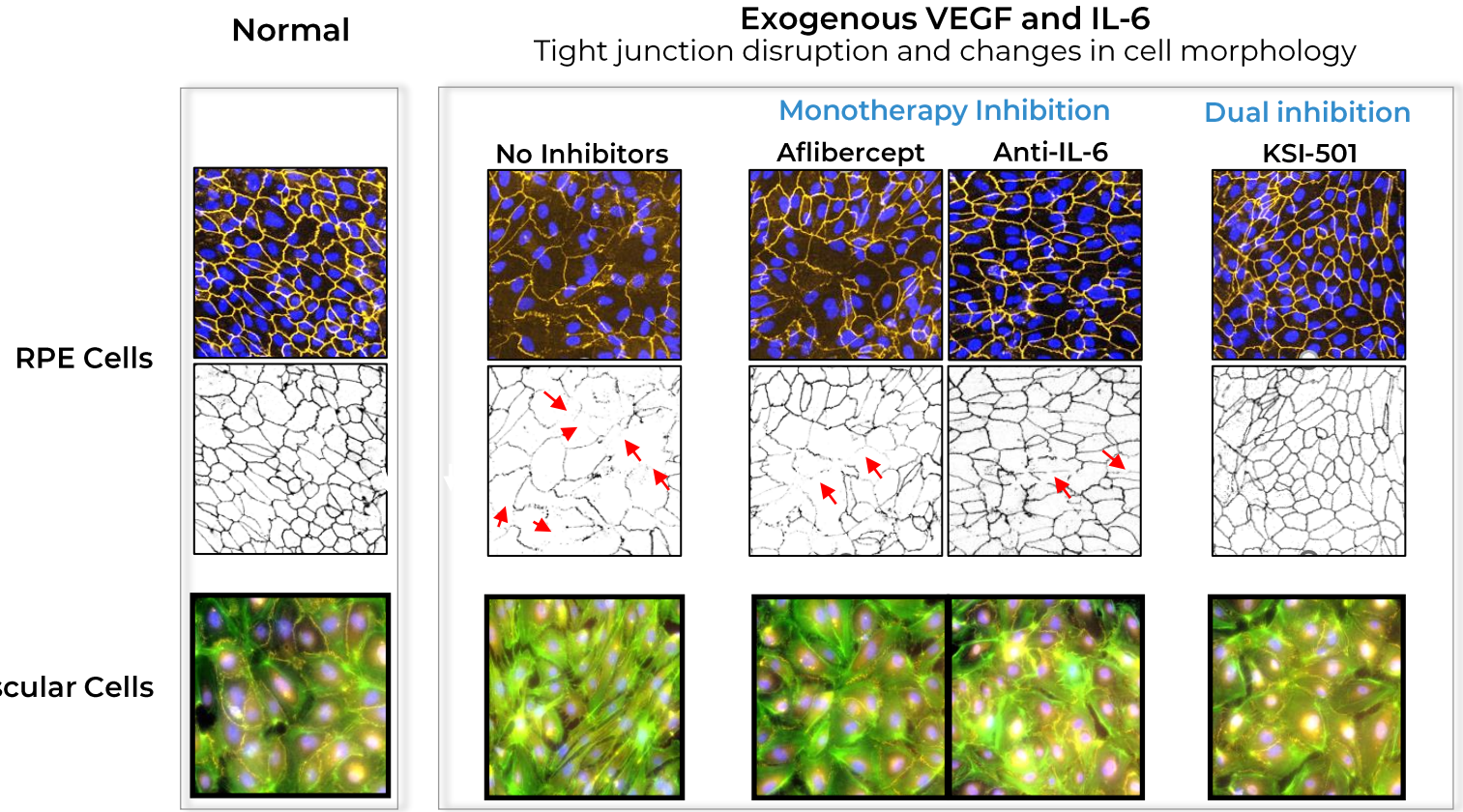
Dual inhibition of VEGF and IL-6 by KSI-501 confers superior normalization of barrier biology vs anti-VEGF or anti-IL-6 monotherapy in preclinical studies



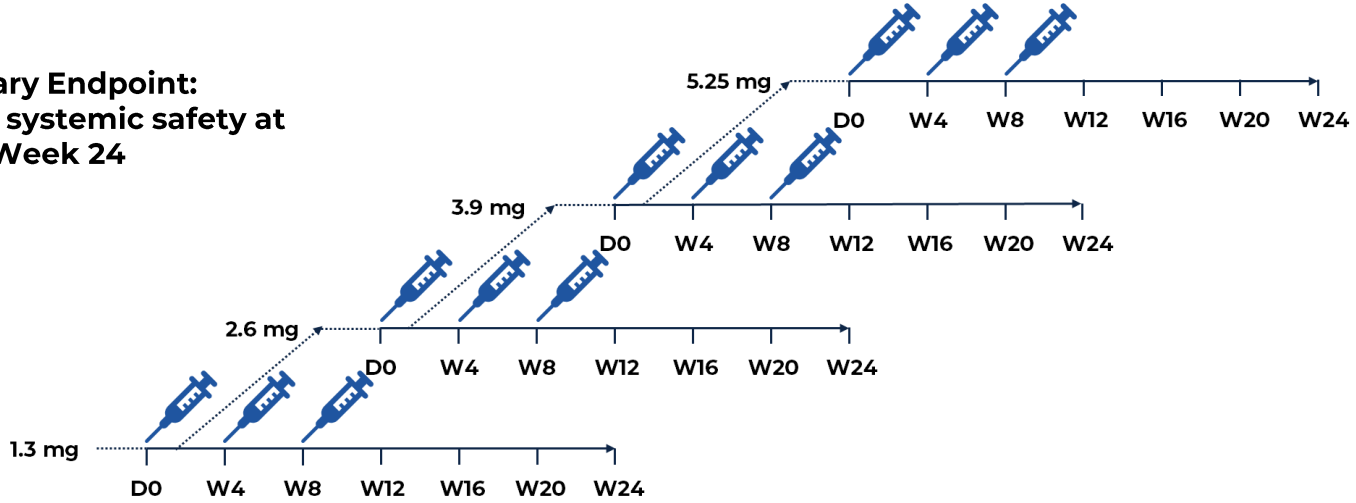
In additional studies, KSI-501 has been shown to inhibit endothelial cell proliferation and tube formation to a greater extent than anti-VEGF or anti-IL-6 monotherapy

With synergistic effect on the blood retinal barrier, KSI-501 holds potential to be a new disease-modifying therapy

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.

Phase 1 study of KSI-501 was a multiple ascending dose study in patients with diabetic macular edema

Primary Endpoint:
Ocular and systemic safety at Week 24



Study Design

- Multiple ascending dose design
- Conducted at 5 sites in the US
- 3-5 subjects planned to be enrolled for each dosing group, with option for expansion of each group if indicated
- Each subject received 3 monthly doses and was followed for 24 weeks total

Key Inclusion / Exclusion Criteria

- Adults ≥21 years of age
- Diabetes mellitus type 1 and 2 (HbA1c ≤12%)
- Vision loss due to DME
- BCVA between 25 and 70 ETDRS letters (20/40 – 20/320 Snellen)
- DME (CST ≥320 microns)
- Treatment naïve and previously treated with an 8-week washout period

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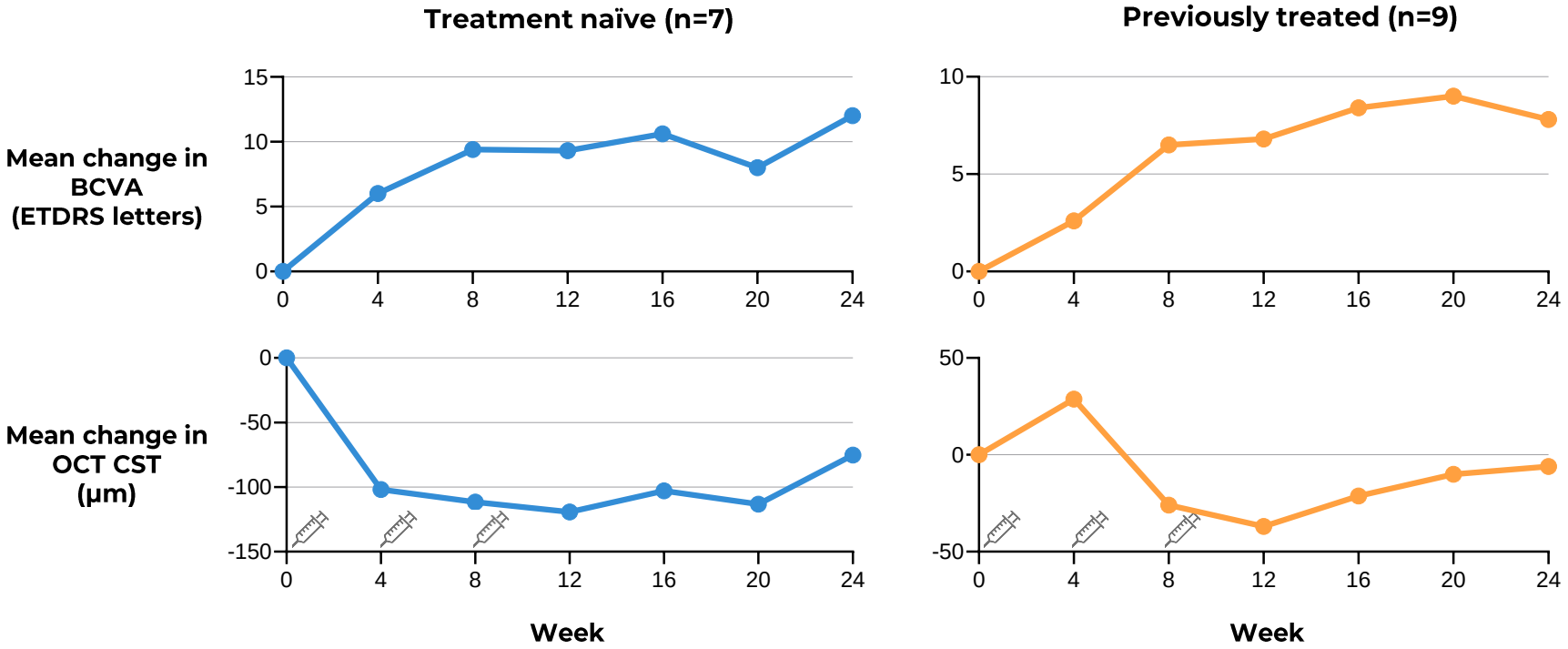
DME: Diabetic Macular Edema; HbA1c: Hemoglobin A1c; BCVA: best-corrected visual acuity; ETDRS: Early Treatment Diabetic Retinopathy Study; CST: Central Subfield Thickness; D: Day; W: Week

KSI-501 demonstrated robust and meaningful visual acuity gains that were sustained over 16 weeks in both treatment naïve and pre-treated patients



- Corresponding anatomical improvement was observed in both treatment naïve and pre-treated patients, with meaningful and sustained improvement in treatment-naïve patients
- Treatment naïve patients are planned to be the target population of Phase 3 studies

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n = Number of participants treated;
BCVA: best-corrected visual acuity; ETDRS: early treatment diabetic retinopathy study; OCT: optical coherence tomography; CST: central subfield thickness

KSI-501 development plan: advancing from Phase 1 study into staggered Phase 3 registrational studies to accelerate time to pivotal data inflection points

Phase 1 study	Diabetic macular edema	
• A multiple ascending dose study of KSI-501 in patients with DME, both treatment naïve and pre-treated patients • DME is known to have high levels of cytokine-mediated microvascular inflammation in addition to VEGF-mediated vascular permeability • **Results:** repeated monthly dosing of KSI-501 was (1) safe and well tolerated, and (2) achieved clinically meaningful and sustained visual acuity gains and CST reduction		✓ Complete

Phase 3 DAYBREAK study	Wet AMD	
• A 3-arm study with investigational arms of tarcocimab dosed Q4W-Q24W after 4 monthly loading doses, KSI-501 dosed Q8W after 4 monthly loading doses and active comparator arm aflibercept 2mg dosed per label • The objective is to evaluate the efficacy, safety and durability of tarcocimab and KSI-501 and to support registration in wet AMD for both investigational medicines • Uses formulation of 50 mg/mL of conjugated and unconjugated forms for both tarcocimab and KSI-501, which balances towards durability without compromising immediacy		Enrollment start mid-2024

- Planning for further Phase 3 studies is underway and pending regulatory alignment

KSI-101

A potent 100 mg/mL high strength bispecific protein being developed for the treatment of macular edema secondary to inflammation for which no approved intravitreal biologic therapies exist today

- In patients with intraocular inflammation, significant vision loss is most commonly a consequence of macular edema
- Studies show that inflammation and vascular permeability have a synergistic effect on driving disease progression and vision loss due to macular edema, but there are no approved therapies that target both drivers of disease

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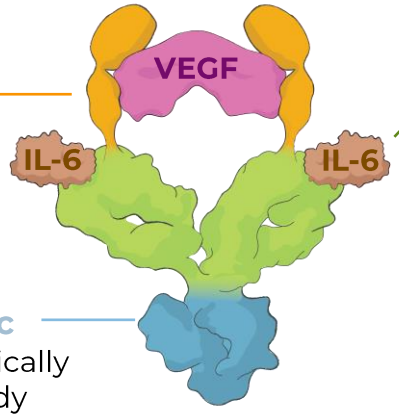
- AMD: age-related macular degeneration; BEACON: NCT04592419; GLOW1: NCT05066230; DAYLIGHT: NCT04964089; GLOW2: NCT06270836

KSI-101 is a first-in-class bispecific for the powerful treatment of macular edema secondary to inflammation

- KSI-101 is a bispecific protein designed to directly target both IL-6 mediated inflammation and edema, and VEGF-mediated vascular permeability
- The anti-inflammatory effect of IL-6 inhibition is the primary effector, with the anti-permeability effect of VEGF inhibition having an additive and synergistic effect

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

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

Currently there are no available intravitreal biologic therapies addressing the spectrum of inflammatory conditions of the retina. Our goal is for KSI-101 to target both underlying disease mechanisms concurrently to prevent vision loss for patients who have macular edema and inflammation

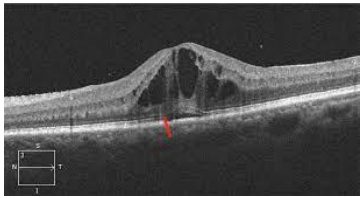
Macular edema is the leading cause of vision loss among patients with intraocular inflammation and IL-6 mediated pro-inflammatory signaling is a key disease driver

- Macular edema is the leading cause of vision loss among patients with intraocular inflammation
- Signaling mediated by pro-inflammatory cytokines including IL-6 is a key disease driver of macular edema secondary to inflammation (MESI)
  - Leads to the disruption of the inner and/or outer blood-retina barrier and accumulation of fluid
- Currently there are no approved, targeted therapies
  - Existing treatment is limited in efficacy and has undesirable side effects
  - There is only one other biologic in late-stage clinical development

Intraocular inflammation is the 4th leading cause of vision loss in the developed world

- **Up to 50% of patients experience reduced vision**
- **10-15% of patients become blind**

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



1/3 of patients with intraocular inflammation (~110,000 patients in the U.S.) develop macular edema

Current treatment algorithm for macular edema secondary to inflammation: high unmet need for safer therapies that target the underlying mechanisms of disease

First line (Mainstay of treatment)	Second line	Second or third line	Third or fourth line or adjunct
Local or systemic corticosteroids	**Immunomodulators**	**Biologic**	**Anti-VEGF agents**
• Associated with **elevated intraocular pressure/glaucoma** that often require therapy and even surgery as well as cataract progression • **30–40% of patients do not respond**	• Off-label use • Used as steroid-sparing agents • Up to 50% of patients do not have macular edema resolved • ~35% of patients do not experience improvement in macular edema	• Adalimumab (anti-TNFα) is currently the only FDA-approved non-steroid therapy for NIU • Used as a steroid-sparing therapy • **~55% of patients experienced treatment failure** over 85 weeks • Associated with **serious side effects** (e.g., infections, malignancies)	• Used for patients with persistent macular edema associated with inflammation that fail conventional therapies • **However, the underlying inflammatory component of the pathophysiological process is not addressed by inhibiting VEGF alone**

There is an unmet need for potent therapies with a better safety profile. With bispecific IL-6 and VEGF inhibition which confer a synergistic anti-inflammatory and anti-permeability effect, along with the proven safety profile of an intravitreal biologic, KSI-101 can become the first line therapy for all retinal diseases with an inflammatory component

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NIU: non-infectious uveitis

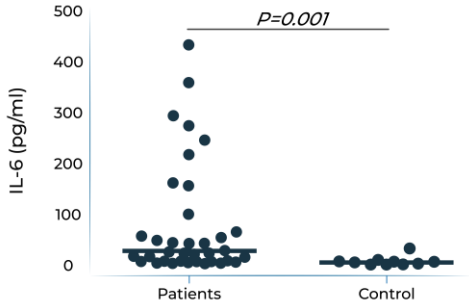
Sources: 1. Tomkins-Netzer O et al. Ophthalmology. 2015. 122:2351-2359. 2. Jaffe et al. N Engl J Med. 2016. 375:932-43. 3. Rosenbaum et al. Sem Arthrit. 2019. 49: 438-445.

Studies show that both IL-6 and VEGF play a key role in retinal inflammatory disease

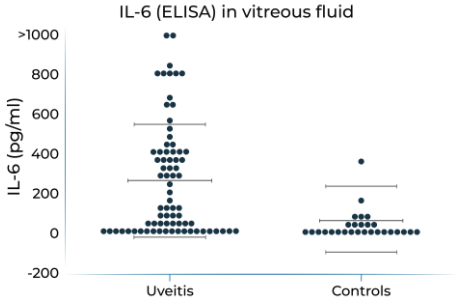
- IL-6 levels are elevated in ocular compartments and in serum in patients with non-infectious uveitis, and further elevated in uveitis patients with macular edema

- Additionally, persistent inflammation triggers VEGF upregulation. VEGF levels are found to be elevated in aqueous humor of eyes with uveitis and uveitic macular edema, which can lead to angiogenesis, vascular leakage and blood-retinal barrier dysfunction

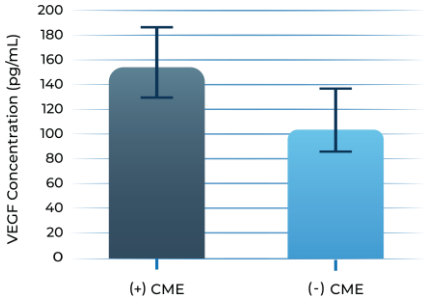
Aqueous Humor IL-6 levels were elevated in patients with intermediate uveitis[1]



IL-6 levels are elevated in vitreous fluid of patients with active uveitis[2]



VEGF levels are elevated in aqueous humor of uveitis patients with macular edema vs without macular edema[3]



1. Adapted from Valentincic et al. Molecular Vision 2011; 17: 2003-2010. 2. Adapted from de Boer et al. Curr Eye Res. 1992;11 Suppl:181-186.
3. Adapted from Fine et al. Am J Ophthal. 2001; 132: 794-796

KSI-101 development plan: Phase 1b APEX study to activate mid-2024; FDA alignment on pivotal program: paired studies with 16-week primary endpoint

PHASE 1B

APEX study	Diabetic macular edema Macular edema secondary to inflammation (MESI)	Target activation mid-2024

- To evaluate 3 dose levels of KSI-101 in DME patients with 3-5 patients per dose level
- To evaluate 3 dose levels of KSI-101 in patients with macular edema secondary to inflammation (MESI) with 5-10 patients per dose level
- Goal is to evaluate safety and tolerability of KSI-101 and to identify 2 dose levels to progress into pivotal studies

PHASE 2B/3 (DUAL STUDIES)

PEAK study PINNACLE study	Macular edema secondary to inflammation (MESI)	Target enrollment start 2H 2024

- Objective is to evaluate the efficacy and safety of KSI-101 and to support registration in macular edema secondary to inflammation
- PEAK and PINNACLE are expected to be identically designed studies with 3 arms: a low dose and a high dose investigational arm of KSI-101 with 4 monthly doses followed by PRN (as needed) dosing and a sham arm
- Primary endpoint will be vision outcomes at week 16 with study completion at week 48

Phase 1b APEX study: multiple ascending dose study of KSI-101 in patients with DME (Part 2) and macular edema secondary to inflammation (Part 3)

Part 2: Subjects with DME (n ~ 9-15)

Weeks	0	4	8	12	16	20	24
5 mg	■	■	■				
7.5 mg	■	■	■				
10 mg	■	■	■				

Part 3: Subjects with macular edema secondary to inflammation (MESI) (n ~ 15-30)

Weeks	0	4	8	12	16	20	24
5 mg	■	■	■	■			
7.5 mg	■	■	■	■			
10 mg	■	■	■	■			

■ KSI-101 injection

End of Study

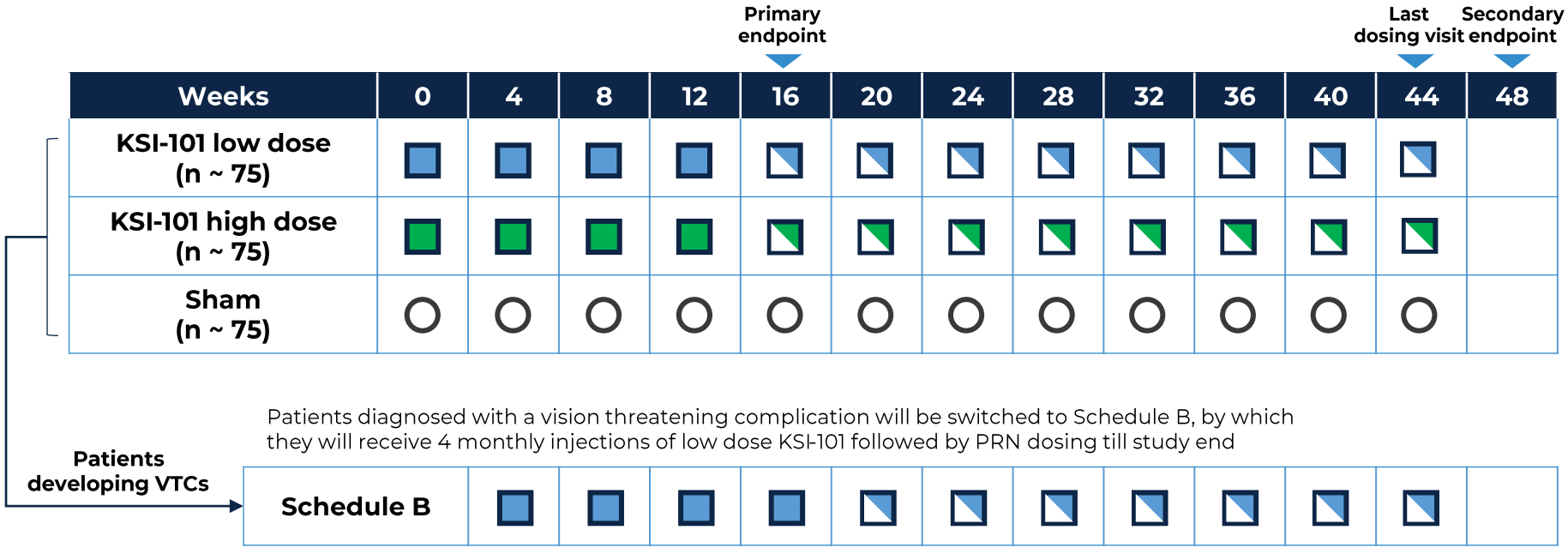
Key Inclusion / Exclusion Criteria

- Adults ≥21 years of age
- Diabetes mellitus type 1 and 2 (HbA1c ≤12%)
- DME (CST ≥325 microns)

- Adults ≥21 years of age
- Macular edema secondary to inflammation, past or concurrent
- CST ≥325 microns
- Diagnosis of active or inactive non-infectious intraocular inflammation, acute or chronic

A low and high dose of KSI-101 will be selected to progress into dual Phase 2b/3 pivotal studies (PEAK and PINNACLE) in MESI later in 2024

Phase 2b/3 Studies PEAK and PINNACLE planned based on regulatory input: identically designed 48-week studies with primary endpoint at Week 16



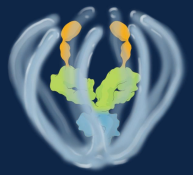
Primary endpoint: Proportion of eyes improving ≥ 15 ETDRS letters in BCVA from baseline to Week 16
Key secondary endpoints: OCT CST, safety and tolerability

PRN: *pro re nata* (as needed); BCVA: best corrected visual acuity; ETDRS: early treatment diabetic retinopathy study; VTC: vision threatening complications

Tarcocimab tedromer

- Longest-acting anti-VEGF biologic (6-month predominant) while preserving the flexibility to dose monthly in high-prevalence retinal vascular diseases
- Supported by our science of durability
- Enhanced 50 mg/mL formulation of conjugated and unconjugated forms balance towards durability without compromising immediacy
- Three of five Phase 3 studies complete in three indications

KSI-501



- Designed to address the opportunity for improved efficacy with extended durability in high-prevalence retinal vascular diseases by targeting retinal inflammation and vascular permeability
- Supported by our science of durability
- Enhanced 50 mg/mL formulation of conjugated and unconjugated forms reflect 10 years of learnings from the “ABC” platform to maximize each patient’s efficacy and durability potential
- The anti-permeability effect of VEGF inhibition is the primary effector, with the anti-inflammatory effect of IL-6 inhibition offering the potential for additional clinical benefits

KSI-101

- A potent 100 mg/mL high strength bispecific protein being developed for the treatment of macular edema secondary to inflammation for which no approved intravitreal biologic therapies exist today
- The anti-inflammatory effect of IL-6 inhibition is the primary effector, with the anti-permeability effect of VEGF inhibition having an additive and synergistic effect

KODIAK SCIENCES

- *$246 million in cash and cash equivalents as of end of 1Q24*
- *Advancing 3 clinical programs into Phase 3 studies in 2024*
- *Planning to achieve meaningful inflection points within current cash runway*