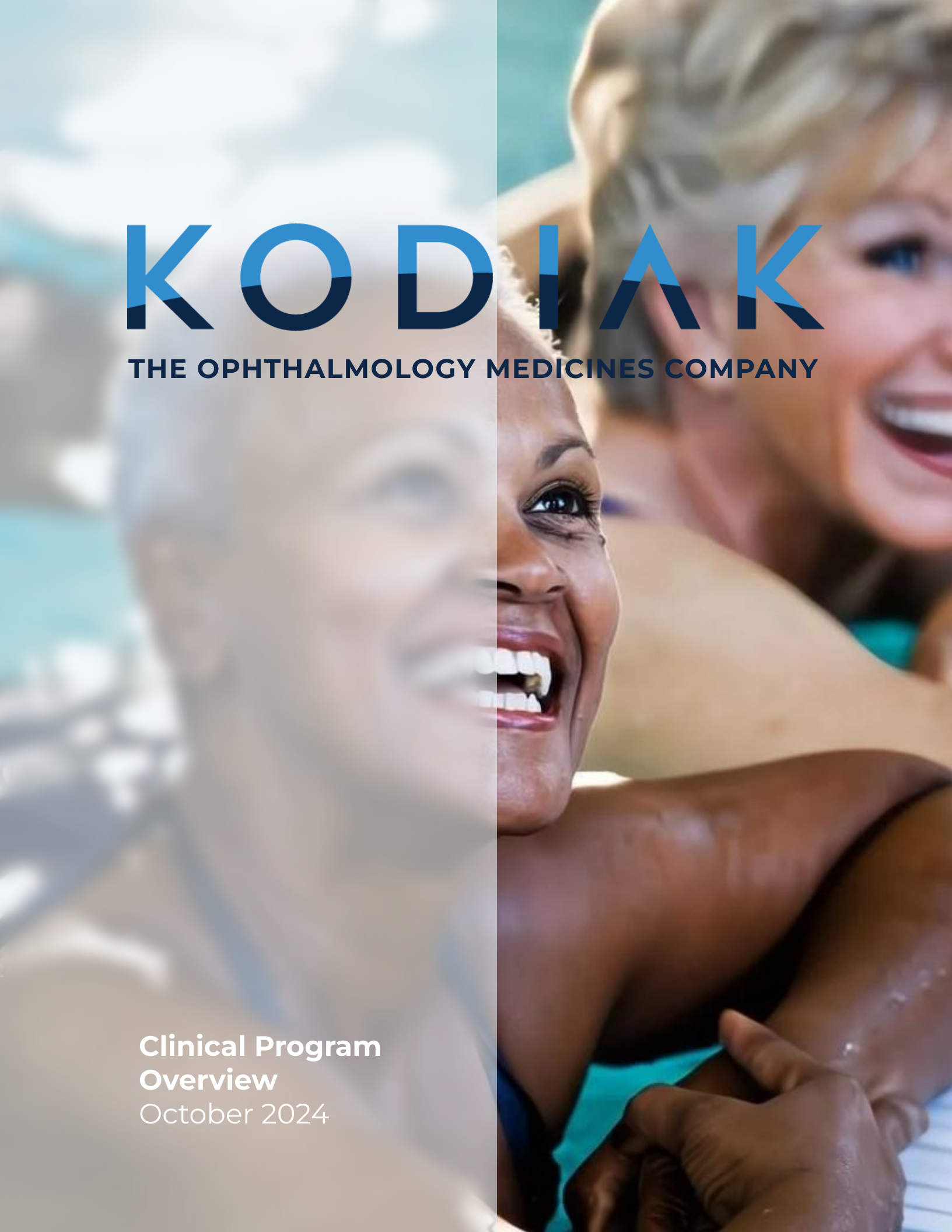


KODIAK

THE OPHTHALMOLOGY MEDICINES COMPANY

Clinical Program Overview

October 2024

Forward-Looking Statements

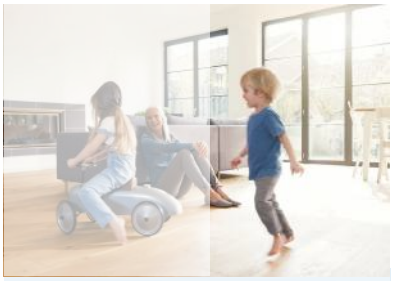
This communication contains “forward-looking statements.” Forward-looking statements are based on our current expectations of future events and are subject to risks and uncertainties that could cause actual results to differ materially and adversely from those in or implied by such forward-looking statements. 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.

THE OPHTHALMOLOGY MEDICINES COMPANY

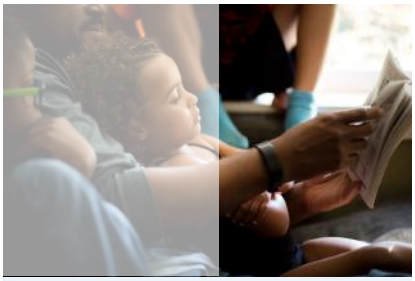
Kodiak Sciences is a biopharmaceutical company committed to researching, developing and commercializing transformative therapeutics to treat a broad spectrum of retinal diseases

Our Mission

To prevent and treat the leading causes of blindness



TRAILBLAZING SCIENCE
Our creative and thoughtful foundation



"GO-TO" MEDICINES
Our challenge to the status quo

SINGULAR FOCUS IN OPHTHALMOLOGY
Our 24/7/365

OUR SCIENCE

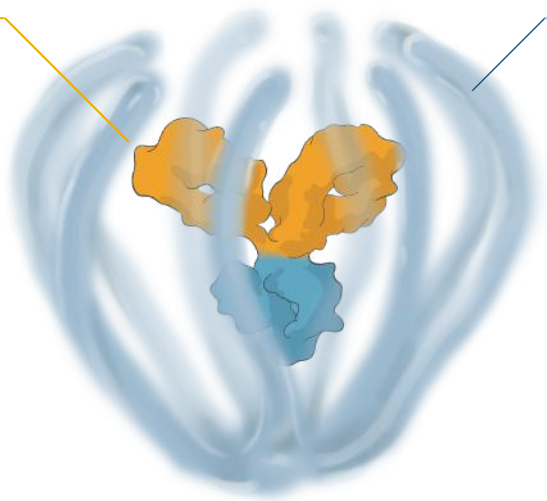
We are focused on bringing new science to the design and development of next generation retinal medicines to prevent and treat the leading causes of blindness

ABCD Platform:
Enabling Multi-Mechanism Therapies Empowered for Durability

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

Antibody or Other Biologic

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

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 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

Drug Cargo

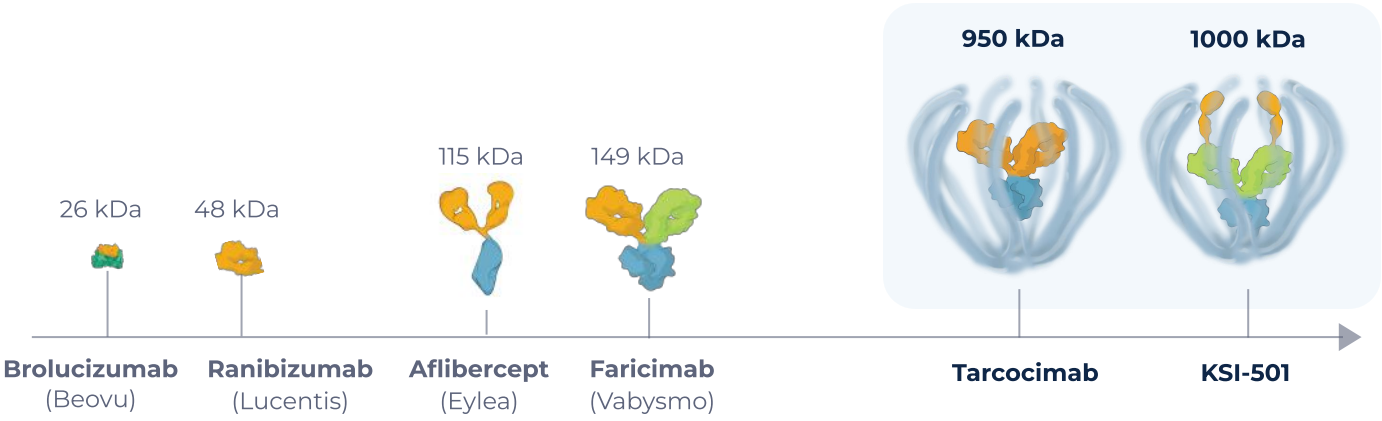
Diverse APIs of varying biophysical properties are embedded in the biopolymer and released over a designed-in time

ABCD Platform:
Supported by Our Science of Durability

Tarcocimab, KSI-501 and the ABCD Platform are supported by our science of durability, which is grounded in **4 key attributes:**

CONJUGATE DESIGN

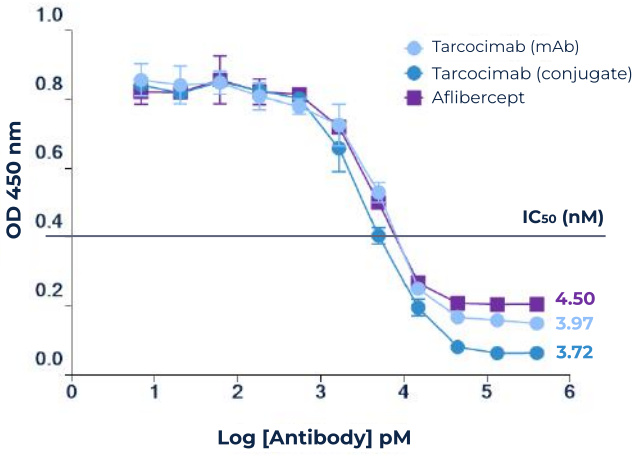
- Ocular half-life of an intravitreal biologic increases proportionally with molecular size
- The ABCD Platform leverages a high molecular weight, phosphorylcholine-based biopolymer that enables an extended ocular residence time compared to today's intravitreal biologics



POTENCY

- Tarcocimab unconjugated protein and conjugated protein demonstrate high binding affinity and potency in pre-clinical assays – similar as aflibercept
- The increased molecular size from conjugation to the biopolymer does not impact binding affinity or potency

High potency in inhibiting VEGF binding to its receptors



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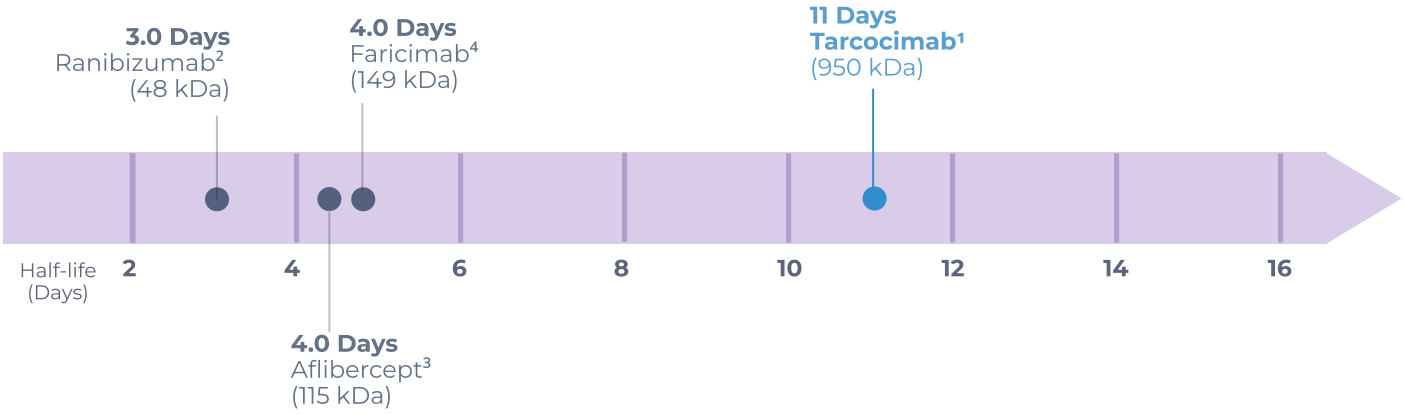
ABCD Platform:
Supported by Our Science of Durability

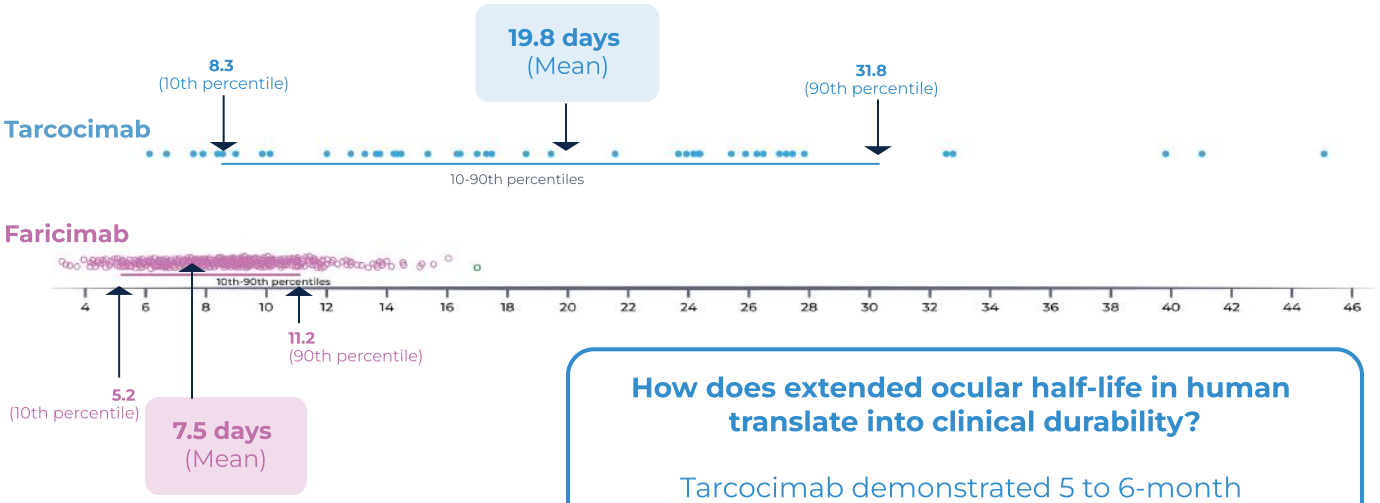
ANIMAL OCULAR HALF-LIFE

- Tarcocimab demonstrates a **~3x – 4x longer ocular half-life** compared to aflibercept or faricimab in rabbit models



HUMAN OCULAR HALF-LIFE

- Tarcocimab has a mean ocular half-life in humans of **20 days, which is 3x longer** than faricimab's mean of 7.5 days[5,6]



How does extended ocular half-life in human translate into clinical durability?

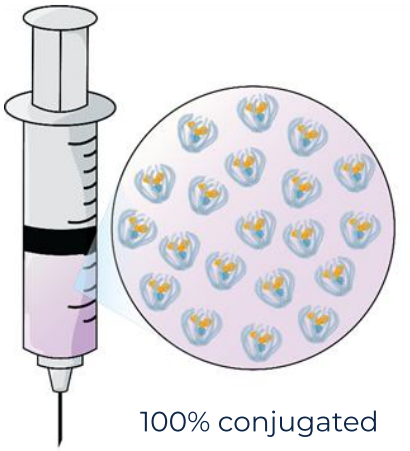
Tarcocimab demonstrated 5 to 6-month predominant durability in pivotal trials across all high-prevalence retinal vascular diseases

1. Kodiak data on file. Ocular half life was determined from a single 50 µL intravitreal injection of 0.725 mg of tarcocimab (conjugate) in rabbits. 2. Gaudreault, et al. Retina 2007, 27: 1260-1266. 3. Park SJ, et al. IOVS 2016, 57: 2612-2617. 4. Pharmacology / Toxicology BLA Review and Evaluation. 5. Ocular half life was determined from aqueous humor concentration of tarcocimab over time in the Phase 1b study in wet AMD, DME and RVO. N=47 patients, all received an intravitreal injection of 5mg tarcocimab clinical formulation on day 1. 6. VABYSMO™ (faricimab solution for injection) Prescribing Information. South San Francisco, USA: Genentech, Inc. PK and ER of faricimab, Report # 1105763

ABCD Platform:
Designed to Deliver Durability *and* Immediacy

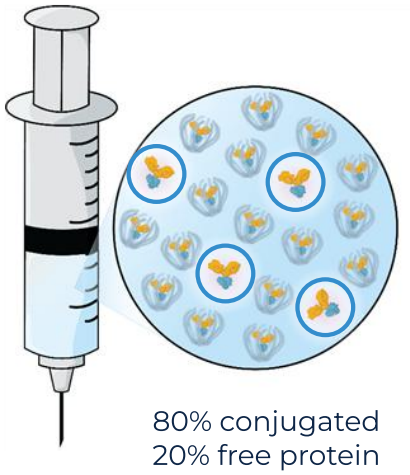
The enhanced formulation of tarcocimab and KSI-501, two ABCD platform-derived medicines, includes a high molar amount of free protein (unconjugated) in addition to the conjugated protein

Original Clinical Formulation for Tarcocimab



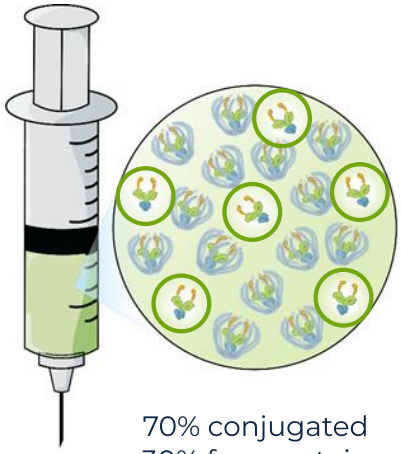
100% conjugated

Enhanced Formulation for Tarcocimab



80% conjugated
20% free protein

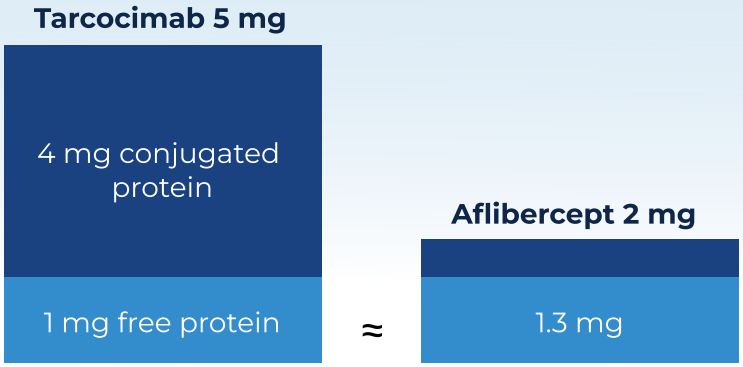
Enhanced Formulation for KSI-501



70% conjugated
30% free protein

The conjugated protein delivers sustained durability and the free protein delivers a strong "pulse" of VEGF inhibition

A closer look at the free protein portion of tarcocimab 5 mg



The 20% of free protein alone in tarcocimab enhanced formulation is equivalent to 1.3 mg of aflibercept

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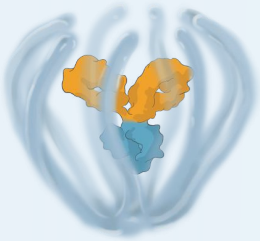
OUR PRODUCT CANDIDATES

A portfolio of three clinical programs intended to address key limitations of today's therapies across a broad spectrum of retinal diseases

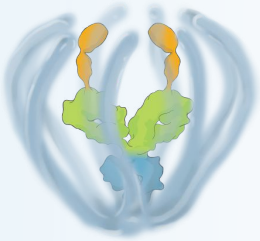
ABCD Platform-derived biologics:
Mainstay biologics for high-prevalence retinal vascular diseases

Unconjugated biologic:
For inflammatory retinal diseases



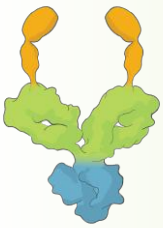
Tarcocimab Tedromer
Anti-VEGF ABCD

- **Objective**: to have a compelling first-line durability profile with uncompromising immediacy
- Longest acting anti-VEGF biologic (6-month predominant) while preserving the flexibility to dose monthly
- An intravitreal biologic that can be used in any patient whether they be in the loading (immediacy) phase or in the maintenance (durability) phase
- **Enhanced 50 mg/mL formulation**



KSI-501
Bispecific Anti-IL-6, VEGF Trap ABCD

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



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

- **Objective**: to address the underlying disease mechanisms of macular edema secondary to inflammation (MESI) 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 ABCD Investigational Medicines (Tarcocimab and KSI-501):

1. Supported by our true science of durability
2. Enhanced formulation is designed to deliver immediacy and durability

TARCOCIMAB TEDROMER

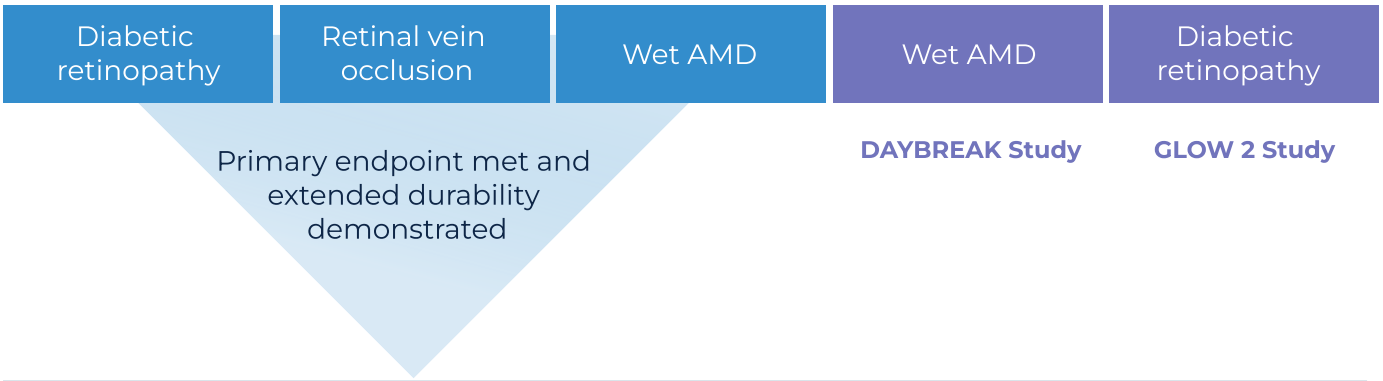
Our objective is for tarcocimab tedromer to have a compelling first-line durability profile with uncompromising immediacy

Three Positive Phase 3 Studies Complete: With Compelling Durability Demonstrated

Five Phase 3 studies across 3 disease indications are planned for inclusion in a Biologic License Application (BLA)

Completed Phase 3 studies using the original clinical formulation

Two Phase 3 studies actively enrolling using the enhanced formulation

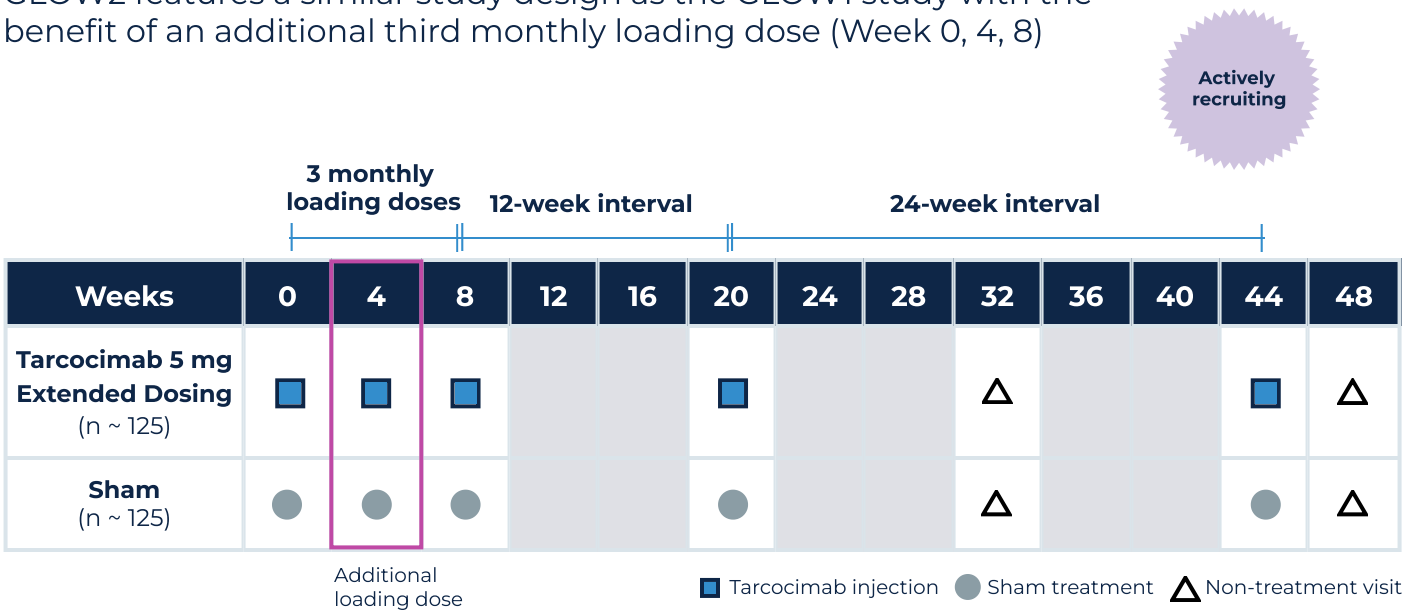


Phase 3 Study	Design	Primary Endpoint	Extended Durability	
Diabetic retinopathy (GLOW1)	• Superiority study • Tarcocimab Q24W after 3 initiating doses vs sham	✓	✓	Signature durability demonstrated with all patients on 6-month dosing
Retinal vein occlusion (BEACON)	• Tarcocimab Q8W after 2 monthly loading 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 (DAYLIGHT)	• Tarcocimab Q4W vs aflibercept Q8W after 3 monthly loading doses	✓	N/A	Monthly dosing of tarcocimab demonstrated favorable safety and non-inferior efficacy at Year 1

BEACON: NCT04592419; GLOW1: NCT05066230; DAYLIGHT: NCT04964089; GLOW2: NCT06270836

New Phase 3 Study: GLOW2 in Diabetic Retinopathy

GLOW2 features a similar study design as the GLOW1 study with the benefit of an additional third monthly loading dose (Week 0, 4, 8)



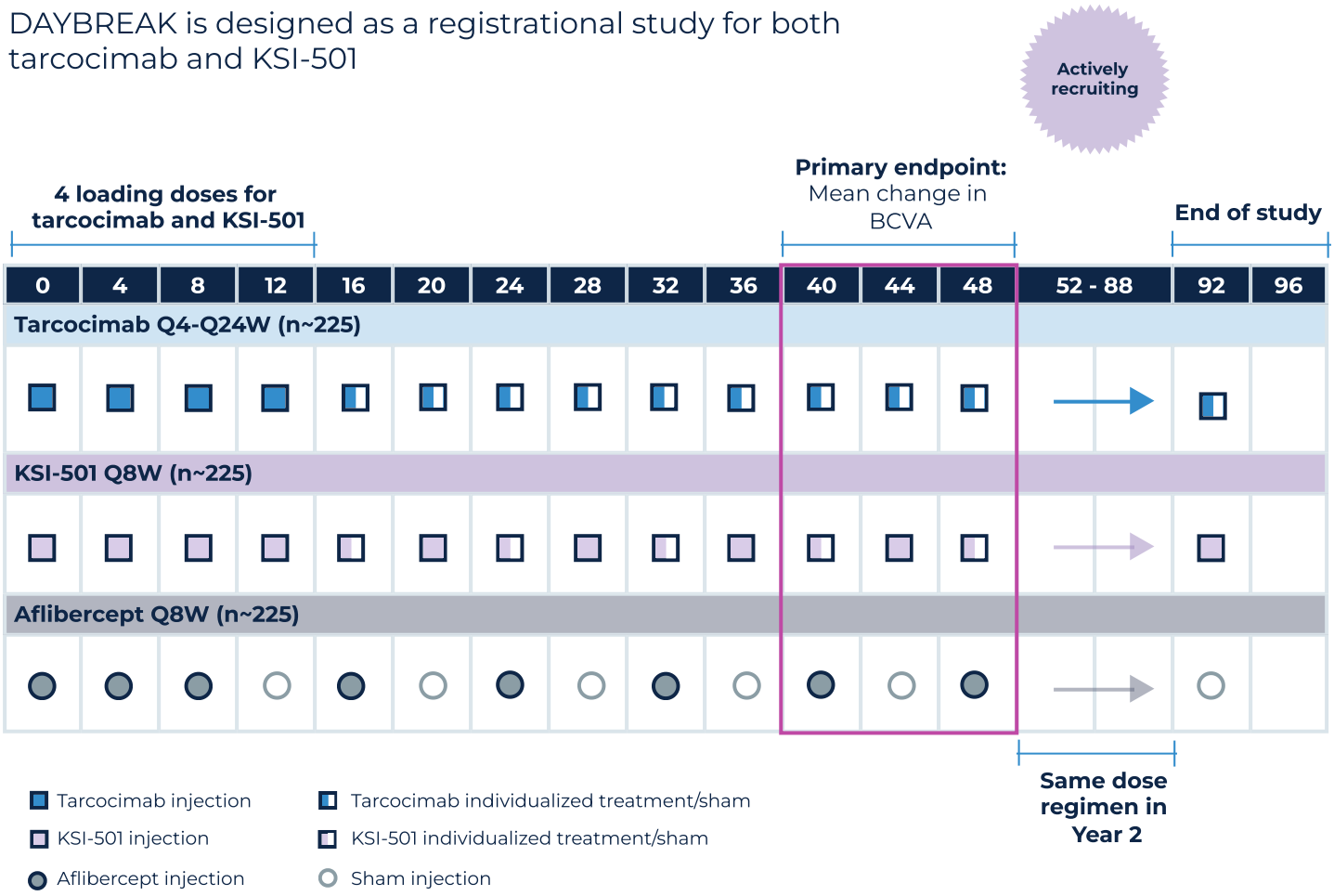
Primary Endpoint	• Proportion of eyes improving ≥2 steps on DRSS from baseline
Key Secondary Endpoints	• Proportion of eyes developing sight-threatening complications* • Proportion of eyes improving ≥3 steps on DRSS from baseline

*Sight-threatening complications are 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; GLOW1 Study: NCT05066230; GLOW2 study: NCT06270836

"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."

New Phase 3 Study: DAYBREAK in Wet AMD

DAYBREAK is designed as a registrational study for both tarcocimab and KSI-501



The study optimizes treatment for each individual patient using objective disease activity criteria that are relevant to how physicians practice in their clinics

Tarcocimab objective	• Assess 6-month durability potential with individualized Q4W-Q24W dosing
KSI-501 objective	• Explore efficacy potential of bispecific IL-6 and VEGF inhibition in fixed Q8W dosing with additional individualized monthly dosing

DAYBREAK: NCT06556368

KODIAK

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

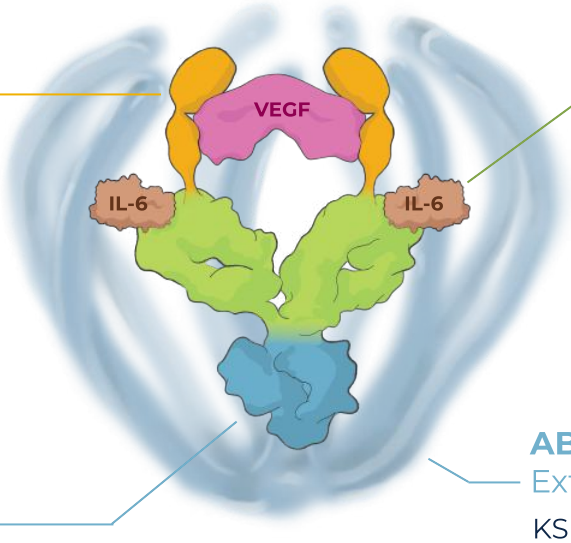
KSI-501 is designed to inhibit VEGF and interleukin-6 (IL-6), a pro-inflammatory cytokine and immune growth factor, combining two powerful mechanisms of action to address retinal vascular disease and the underlying inflammatory cascade

KSI-501 is a First-In-Class Bispecific ABCD
Designed for Highly Efficient Binding to Both IL-6 and VEGF

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

ABCD Platform: Extended durability

KSI-501 leverages our ABCD platform with its signature 6-month predominant durability

The enhanced 50 mg/mL ABCD formulation is designed to maximize durability and efficacy, with conjugated and unconjugated forms

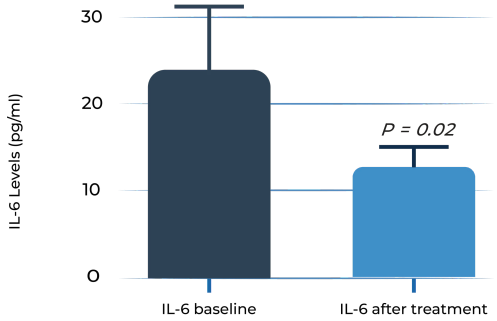
IL-6 and Retinal Vascular Diseases

In addition to VEGF, IL-6 is a pro-inflammatory cytokine and immune growth factor implicated in the pathophysiology of multiple retinal vascular diseases:

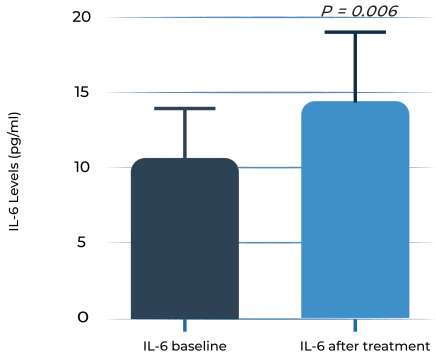
- Vitreous IL-6 levels are significantly elevated in retinal disease patients vs control
- IL-6 stimulates defective angiogenesis independent of VEGF, and is implicated in anti-VEGF treatment resistance
- Increased levels of IL-6 are associated with poor functional outcomes in wet AMD and diabetic macular edema ("DME") patients treated with anti-VEGF monotherapy

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

Patients that respond to anti-VEGF

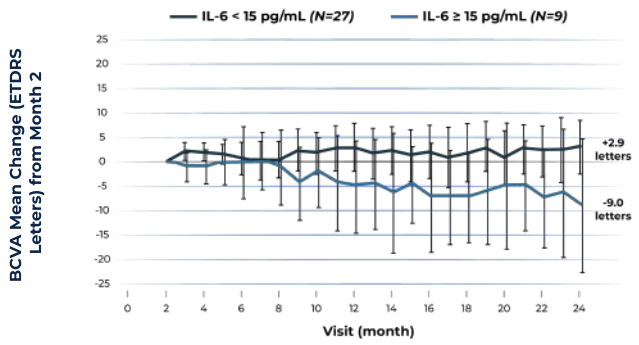


Anti-VEGF treatment resistant patients

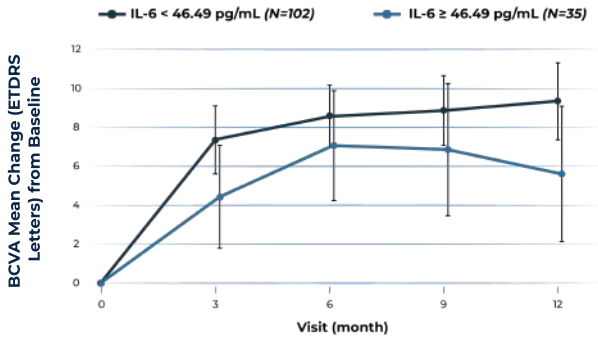


Higher levels of IL-6 in aqueous humor are correlated with poorer BCVA outcomes over time in retinal vascular diseases[2]

BCVA change from month 2 over time in wet AMD patients with high and low aqueous IL-6 levels



BCVA change from baseline over time in DME patients stratified by baseline aqueous IL-6 levels



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).

BCVA, best corrected visual acuity; ETDRS, Early Treatment Diabetic Retinopathy Study

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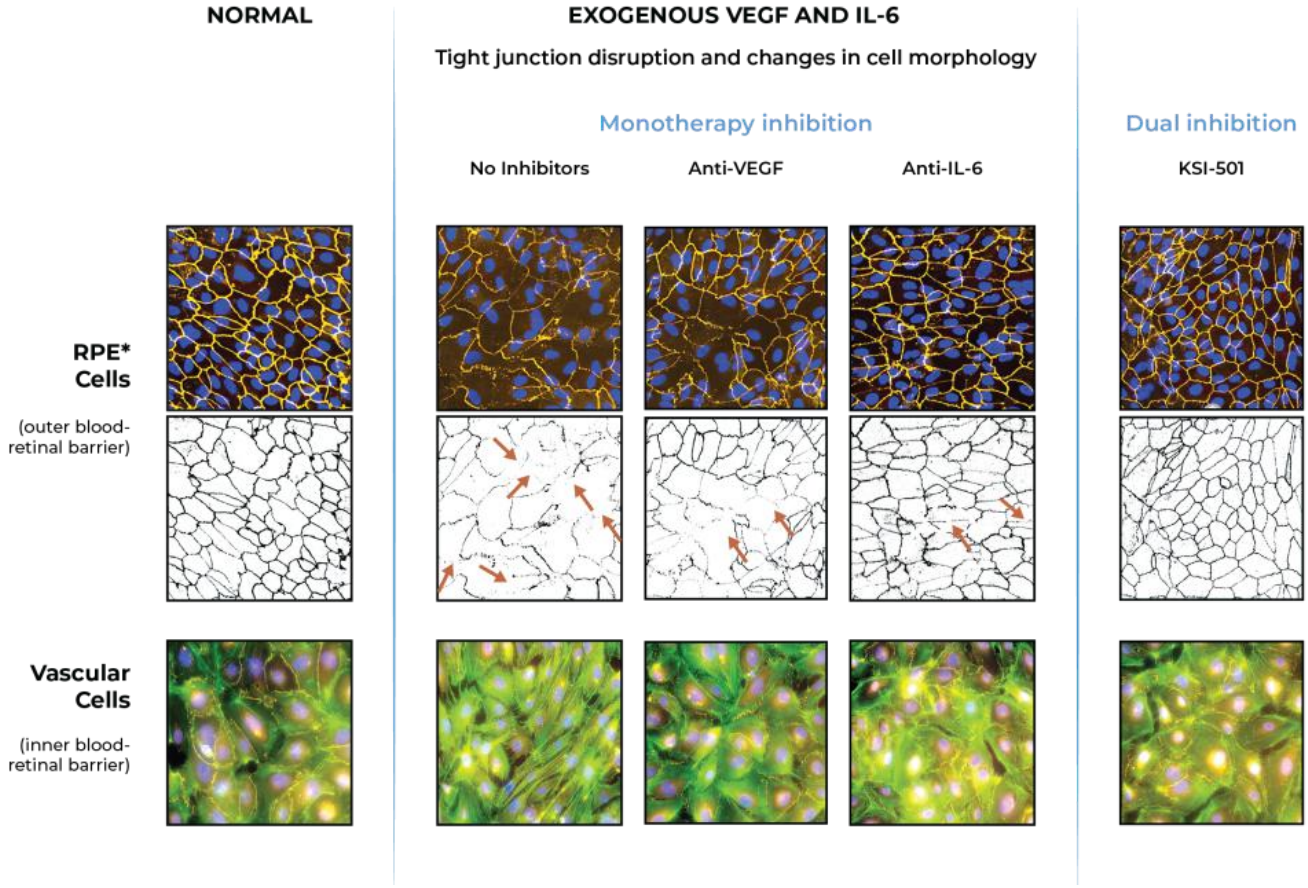
Dual Inhibition of IL-6 and VEGF Show a Synergistic Effect

Dual inhibition of VEGF and IL-6 by KSI-501 confers superior normalization of complex tight junction-mediated barrier biologies compared to either anti-VEGF or anti-IL-6 monotherapy alone demonstrating the synergistic effect of IL-6 and VEGF dual inhibition on retinal vascular disease

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



RPE cells: nuclei in blue, ZO1 (tight junction protein) in yellow. Vascular cells: nuclei in purple, ZO1 (tight junction protein) in yellow, actin in green.
K Williams et al, "Biological Benefits of KSI-501: Novel Bispecific Anti-Inflammatory and Anti-Angiogenic Therapy for the Treatment of both Retinal Vascular and Inflammatory Diseases" Poster 2215 at 2023 ARVO Annual Meeting

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

Clinical Development Plan

KSI-501 is being developed to address the opportunity for improved efficacy with extended durability in high-prevalence retinal vascular diseases

KSI-501 is being investigated in the ongoing Phase 3 DAYBREAK study in wet AMD. The DAYBREAK study is designed to explore the efficacy potential of bispecific IL-6 and VEGF inhibition in fixed Q8W with additional individualized monthly dosing of KSI-501

MULTIPLE DOSE PHASE 1

Phase 1 Study	Diabetic macular edema	
• A multiple ascending dose study. Each patient received 3 monthly doses (Day 1, Week 4 and Week 8) and was followed for 24 weeks total • Evaluated KSI-501 in patients with diabetic macular edema, a disease 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) shown to be safe and well tolerated, and (2) achieved clinically meaningful and sustained visual acuity gains and central subfield thickness ("CST") reduction		✓ Complete

PHASE 3

DAYBREAK Study	Wet AMD	
• Designed to include tarcocimab and KSI-501 investigational groups vs aflibercept to support registration in wet AMD for both investigational medicines • **Tarcocimab objective:** assess 6-month durability potential with individualized Q4W-Q24W dosing • **KSI-501 objective:** explore efficacy potential of bispecific IL-6 and VEGF inhibition in fixed Q8W dosing with additional individualized monthly dosing		Actively recruiting

DAYBREAK: NCT06556368

- 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 ("MESI") 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

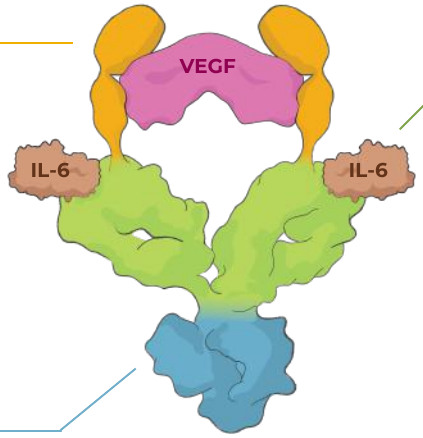
KSI-101 is a First-in-Class Bispecific Protein
For the Powerful Treatment of Macular Edema Secondary to Inflammation

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 PlGF



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

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

Patients with Vision-Threatening Retinal Inflammatory Disease Have Limited Treatment Options Today

Macular edema is the leading cause of vision loss for uveitis patients, a heterogenous group of diseases characterized by intraocular inflammation. Many patients with macular edema have persistent disease activity despite treatment and are at risk for vision loss

In macular edema associated with inflammation there is no standard treatment algorithm and patients are exposed to therapies with limited efficacy and undesirable side effects

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**
• Approximately 30-40% of patients do not respond • Associated with undesirable side effects, such as cataract progression and elevated intraocular pressure or glaucoma	• Used as off-label, steroid sparing therapies • Up to 50% of patients do not have their macular edema resolved • Approximately 35% of patients do not experience improvement in macular edema	• Adalimumab (anti-TNF-alpha) is currently the only FDA-approved non-steroid therapy for non-infectious uveitis • Used as a steroid-sparing therapy • Approximately 55% of patients experienced treatment failure over 85 weeks • Associated with serious systemic side effects	• 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 minimally invasive potent therapies with a better safety profile

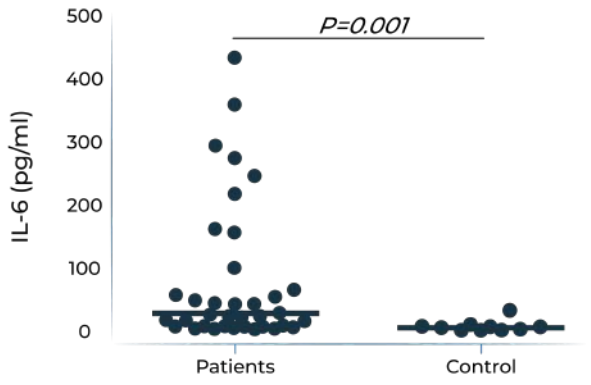
With bispecific IL-6 and VEGF inhibition which confer a synergistic anti-inflammatory and anti-permeability effect, along with the proven safety profile of an intravitreal biologic, KSI-101 can become the first line therapy for all retinal diseases with an inflammatory component

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.

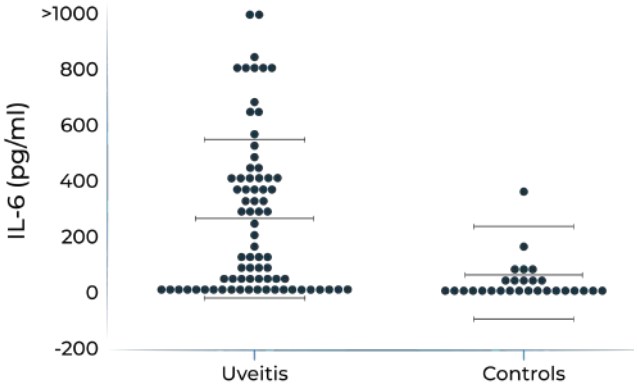
Studies Show that Both IL-6 and VEGF Play a Key Role in Retinal Inflammatory Disease

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

Aqueous humor IL-6 levels are elevated in patients with intermediate uveitis[1]



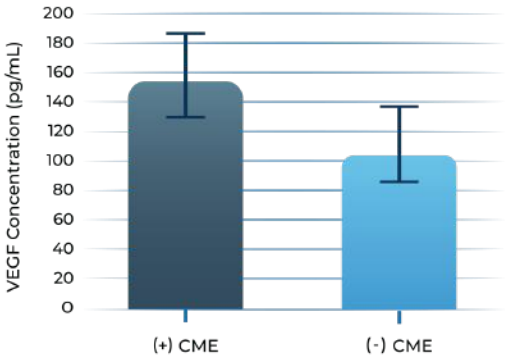
IL-6 levels are elevated in vitreous fluid of patients with active uveitis[2]



1. Valentincic et al. Molecular Vision 2011; 17: 2003-2010
2. de Boer et al. Curr Eye Res. 1992;11 Suppl:181-186

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

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



3. Fine et al. Am J Ophthal. 2001; 132:794-796.
CME: cystoid macular edema

KSI-101 Clinical Development Plan

KSI-101 is being developed to fill the unmet need for a potent, high strength, locally administered biologic in patients with macular edema secondary to inflammation

KSI-101 is to be evaluated in the APEX Phase 1b Study and advance into dual Phase 2b/3 studies in patients with macular edema secondary to inflammation

PHASE 1B

APEX Study	Diabetic macular edema Macular edema secondary to inflammation	Actively recruiting

- Goal is to evaluate safety and tolerability of KSI-101 and to identify two dose levels to progress into pivotal studies
- To evaluate 3 dose levels of KSI-101 in patients with macular edema secondary to inflammation
- To evaluate 3 dose levels of KSI-101 in patients with diabetic macular edema

PHASE 2b/3 (DUAL STUDIES)

PEAK Study **PINNACLE Study**	Macular edema secondary to inflammation	Target enrollment start 1H 2026

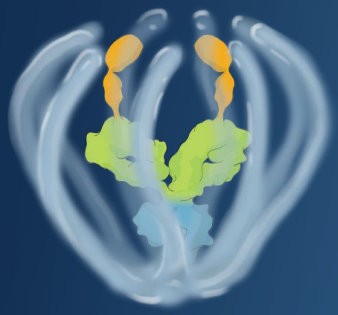
- Objective is to evaluate the efficacy and safety of KSI-101 and to support registration in macular edema secondary to inflammation
- PEAK and PINNACLE are expected to be identically designed studies in patients with macular edema secondary to inflammation

A Pipeline of 3 Clinical Assets Across a Broad Spectrum of Retinal Diseases

Tarcocimab tedromer
Anti-VEGF ABCD

- A "mainstay" intravitreal biologic intended to deliver high efficacy and high durability for retinal vascular diseases
- Consistent 6-month predominant durability
- Supported by our science of durability
- Enhanced 50 mg/mL formulation of conjugated and unconjugated forms is designed to deliver the "pulse and the durability" while improving dose preparation, dose administration and safety
- Three of five Phase 3 studies complete in three indications; two new Phase 3 studies actively enrolling

KSI-501
First-in-Class Bispecific
Anti-IL-6, VEGF Trap ABCD



- Designed to address the opportunity for first-line efficacy with the best durability in high-prevalence retinal vascular diseases by targeting retinal inflammation **and** vascular permeability simultaneously
- 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
- Benefits from the clinical science of immediacy and durability of the ABCD platform
- Phase 3 DAYBREAK study actively enrolling, designed to explore the power of the dual mechanism of action to deliver improved efficacy

KSI-101
First-in-Class Bispecific
Anti-IL-6, VEGF Trap Protein

- Designed to address the underlying disease mechanisms of macular edema secondary to inflammation (MESI) for which no approved intravitreal biologics 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
- Uncorrelated from the ABCD platform
- Differentiation of having dual inhibition mechanism and high strength 100 mg/mL formulation

ABC: Antibody Biopolymer Conjugate