UNITY BIOTECHNOLOGY

Ophthalmology Day Webcast Presentation

October 15, 2024

NASDAQ: UBX

Special Note Regarding Forward-Looking Statements

This presentation and the accompanying oral commentary contain forward-looking statements including statements related to Unity Biotechnology Inc.'s ("UNITY's") understanding of cellular senescence and the role it plays in retinal diseases and diseases of aging, the potential for UNITY to develop therapeutics to slow, halt, or reverse diseases of aging, including for ophthalmologic and neurologic diseases, UNITY's expectations regarding potential benefits, activity, effectiveness, and safety of UBX1325, the potential for UNITY to successfully commence and complete clinical studies of UBX1325 for DME, AMD, and other ophthalmologic diseases, the expected timing of enrollment and results of the clinical trials in UBX1325, and UNITY's expectations regarding the sufficiency of its cash runway. These statements involve substantial known and unknown risks, uncertainties, and other factors that may cause our actual results, levels of activity, performance, or achievements to be materially different from the information expressed or implied by these forward-looking statements, including risks relating to the uncertainties inherent in the drug development process, the risk that interim results of our clinical studies may not be indicative of future results, and risks relating to UNITY's understanding of senescence biology. We may not actually achieve the plans, intentions, or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make. The forward-looking statements in this presentation represent our views as of the date of this presentation. We anticipate that subsequent events and developments will cause our views to change. However, while we may elect to update these forward-looking statements at some point in the future, we have no current intention of doing so except to the extent required by applicable law. You should, therefore, not rely on these forward-looking statements as representing our views as of any date subsequent to the date of this presentation. For a further description of the risks and uncertainties that could cause actual results to differ from those expressed in these forward-looking statements, as well as risks relating to the business of UNITY in general, see UNITY's most recent Quarterly Report on Form 10-Q for the quarter ended June 30, 2024, filed with the Securities and Exchange Commission on August 6, 2024, as well as other documents that may be filed by UNITY from time to time with the Securities and Exchange Commission. This presentation concerns drug candidates that are under clinical investigation which have not yet been approved for marketing by the U.S. Food and Drug Administration. They are currently limited by Federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated. This presentation does not constitute an offer or invitation for the sale or purchase of securities and has been prepared solely for informational purposes.

Agenda

Welcome and Introduction to UNITY DME Unmet Need and Opportunity for UBX1325	Anirvan Ghosh, PhD, CEO Alicia Tozier, MBA, Chief Strategy Officer
Senolytic Therapeutic Hypothesis	Mike Sapieha, PhD, Chief Scientist
UBX1325 Clinical Development Plan Ph2 BEHOLD Data in DME Patients	Robert Bhisitkul, MD, PhD (UCSF), Senior Clinical Advisor
Ph2B ASPIRE Design and Data Timelines FDA Interaction & Pivotal Study Expectations	Anirvan Ghosh, PhD, CEO
Fireside Chat with Retina Experts Arshad Khanani and Dante Pieramici	Arshad Khanani, MD, MA, FASRS (Sierra Eye Associates) Dante Pieramici, MD (California Retina Consultants) Alicia Tozier, MBA, Chief Strategy Officer

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Executive Leadership Team

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Advisory Team Jeff Heier, MD I David Boyer, MD I Bob Bhisitkul, MD, PhD I Quan Nguyen, MD, MSc I Diana Do, MD

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Developing Transformative Medicines for Retinal Disease based on a Senolytic Mechanism of Action

Focus Area: Ophthalmology

DME (Diabetic Macular Edema),

Diabetic Retinopathy,

Geographic Atrophy

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Investment Highlights: Recent Achievements and UBX1325 Clinical Studies

Senolytic platform to develop transformative therapeutics

Developing a novel therapeutic approach to remodel the retina based on **Senolytic Mechanism of Action**

Potential to be valuable as monotherapy or in combination with anti-VEGF agents to shift the treatment paradigm for progressive vision loss

Lead asset UBX1325 (foselutoclax) has best in class potential for DME

Novel MOA to overcome **limitations of current standard of care, including heavy treatment burden and sub-optimal response**

A single dose of foselutoclax led to **strong visual acuity gains through 48 weeks in Phase 2 BEHOLD study in patients with DME**

Recent achievements and ongoing studies

Phase 2b **ASPIRE** study, evaluating **UBX1325 head-to-head against aflibercept in DME,**

- **Enrollment completed** in Q3 2024
- **Type C Engagement with FDA** provides opportunity for pivotal study largely in line with Ph2 ASPIRE trial
- **24-week data expected in 1Q25** and **36-week data expected in 2Q25**

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UNITY Pipeline

Targeting Cellular Senescence and Aging-Related Biology in Indications with Established Endpoints and Well-Defined Regulatory Pathways to Approval

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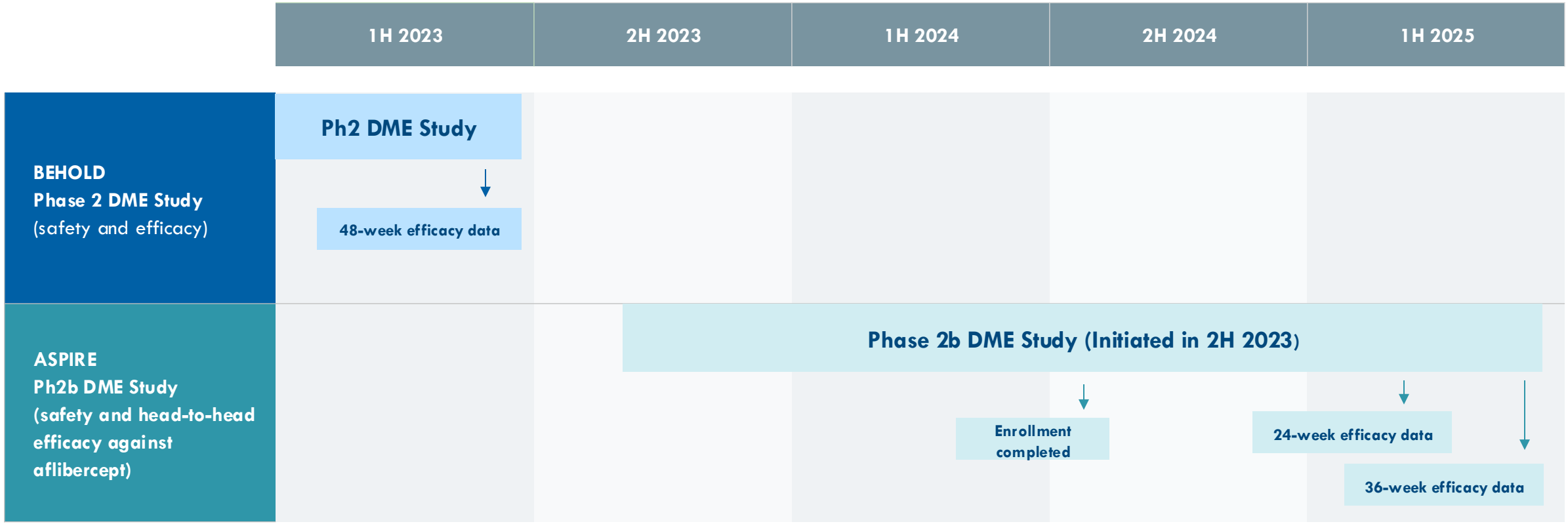
UBX1325 (Foselutoclax) Clinical Program in Diabetic Macular Edema
BEHOLD and ASPIRE Studies



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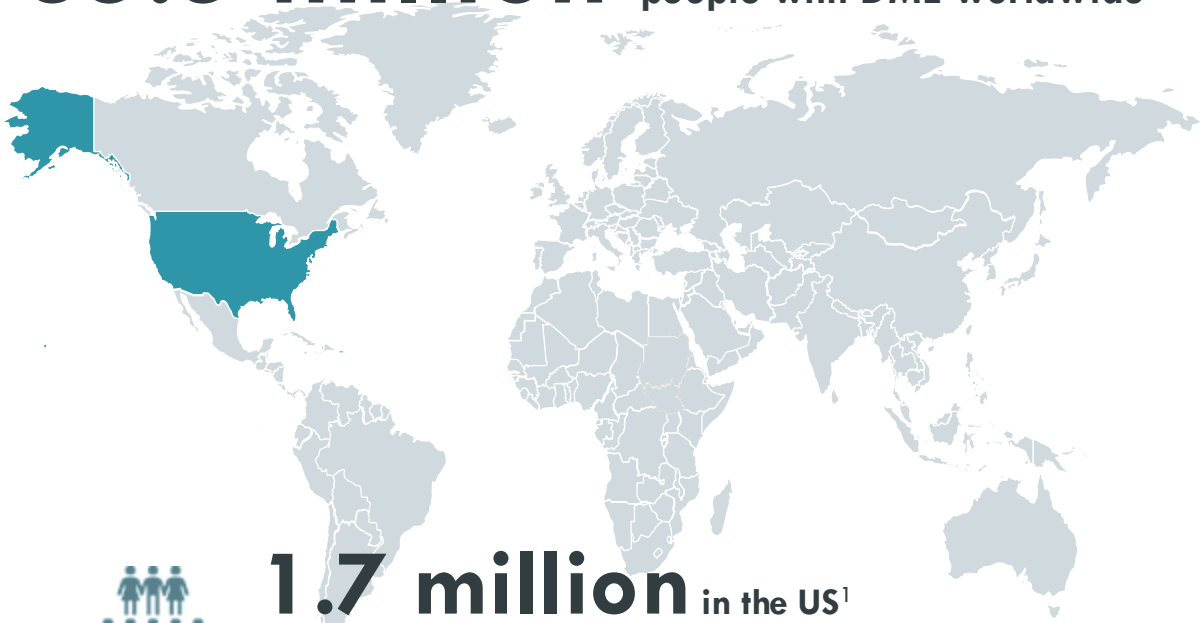
DME Unmet Need and Opportunity for UBX1325

Diabetic Macular Edema (DME) Represents a Large Underserved Market

35.5 million people with DME worldwide[1]



1.7 million in the US[1]

with ~750,000 diagnosed and treated[2]

and **~30% affected in both eyes**[2]

Inadequate Response to Anti-VEGF Standard of Care

- *One half of patients achieve a weak or suboptimal response*[3,8]
- *30% may not respond to anti-VEGF*[4] *at all*
- *Approximately one-third of patients require monthly dosing*[7]

Continued Vision Loss Over Time Despite Treatment

- *Most visual acuity (VA) improvements occur within the first year*[2]
- *Vision gains plateau at 24 months then decline through year 5*[5]
- *28% lose the ability to drive by Year 4*[6]

High Treatment Burden Leading to Discontinuation

- *50%+ discontinue anti-VEGF treatment by 6 months*[2]

DME=Diabetic Macular Edema; AE=Adverse Events; VEGF=Vascular Endothelial Growth Factor; Newest agents=VABYZMO and EYLEA HD

1. Downs P. Global Retinal Pharmaceuticals Market Report. Market Scope; 2023 Aug; 2. Kuo B, et al. Long-term Treatment Patterns for Diabetic Macular Edema – Up to 6 Year Follow-up in the IRIS Registry. Ophthalmology Retina. Articles in Press. 2024 Jun 01; 3. Gonzales V et al. Early and Long-Term Responses to Anti-Vascular Endothelial Growth Factor Therapy in Diabetic Macular Edema. American Journal of Ophthalmology. 2016 Dec. 172:72:79; 4. Sharma D et al. Mechanisms of Acquired Resistance to Anti-VEGF. iovs.arvojournals.org; ISSN: 1552-5783; May 2023; 5. Glassman AR et al, Diabetic Retinopathy Clinical Research Network. Ophthalmol. 2020 Aug; 127 (9): 1201-10; 6. Emami-Naeini, P et al. Ophthalmology Retina. 2024 Apr. Volume 8, Issue 4, 388 – 398. 7. Giust J et al. Treat and Extend Versus Bi-monthly Dosing with Aflibercept for the Treatment of Diabetic Macular Edema, One Year Outcomes (EVADE STUDY). ARVO Abstract. 2018; 8. Sun J et al. Defining "Strong" versus "Weak" Response to Anti-VEGF Treatment for Center-Involved Diabetic Macular EdemaRetina. 2023 April 01; 43(4): 616–623.

UBX1325 is Designed to Address Current DME Unmet Needs

POOR VISUAL OUTCOMES

- One half of DME patients achieve a weak or suboptimal response to anti-VEGF[3,4,8]
- With anti-VEGF agents, vision gains plateau at 24 months & then continue to decline[3,5,8]

+

HIGH TREATMENT BURDEN

- Approximately one third of DME patients still require monthly dosing[7]
- Dosing frequencies are burdensome leading to >50% dropping out after 6 months[3]

+

NEW MECHANISM OF ACTION

- Sub-optimal response to anti-VEGF point to the need for additional mechanisms of action[1,2,3,4]
- Patients with diabetes are 2x more likely to experience systemic AEs[6] with anti-VEGF therapy

=

STRONG UBX1325 OPPORTUNITY

A novel therapeutic approach with the potential to improve long-term visual outcomes for DME patients, via a proven and safe IVT route of administration

1. Global Data. Diabetic Macular Market 2021-2031. 2022 Aug 01; 2. Hahn P, Garg SJ, eds. Membership Preferences and Trends (PAT). American Society of Retina Specialists. 2023; 3. Kuo B, et al. Long-term Treatment Patterns for Diabetic Macular Edema – Up to 6 Year Follow-up in the IRIS Registry. Ophthalmology Retina. Articles in Press. 2024 Jun 01; 4. Sharma D et al. Mechanisms of Acquired Resistance to Anti-VEGF. iovs.arvojournals.org; ISSN: 1552-5783; May 2023; 5. Glassman AR et al. Diabetic Retinopathy Clinical Research Network. Diabetic Macular Edema Protocol T Extension Study. Ophthalmol. 2020 Aug; 127 (9): 1201-10. 6. Zafae S et al. Systemic Adverse Events Among Patients With Diabetes Treated With Intravitreal Anti–Vascular Endothelial Growth Factor Injections. JAMA Ophthalmol. 2023;141(7):658-666. doi:10.1001/jamaophthalmol.2023.2098. 2023 Jun 01. 7. Giust J et al. Treat and Extend Versus Bi-monthly Dosing with Aflibercept for the Treatment of Diabetic Macular Edema, One Year Outcomes (EVADE STUDY). ARVO Annual Meeting Abstract. 2018. 8. Gonzales V et al. Early and Long-Term Responses to Anti-Vascular Endothelial Growth Factor Therapy in Diabetic Macular Edema. American Journal of Ophthalmology. 2016 Dec. 172:72:79;

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Senolytic Therapeutic Hypothesis

UBX1325 Mechanism of Action

Cellular Senescence

a *UNIFYING* mechanism in aging biology

Experimental Cell Research **25**, *585–621 (1961)*

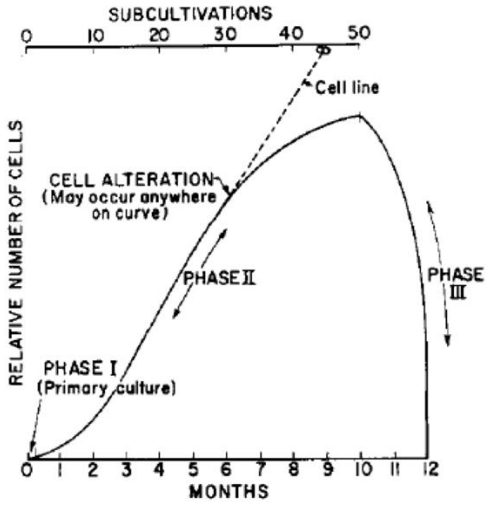
THE SERIAL CULTIVATION OF HUMAN DIPLOID CELL STRAINS[1]

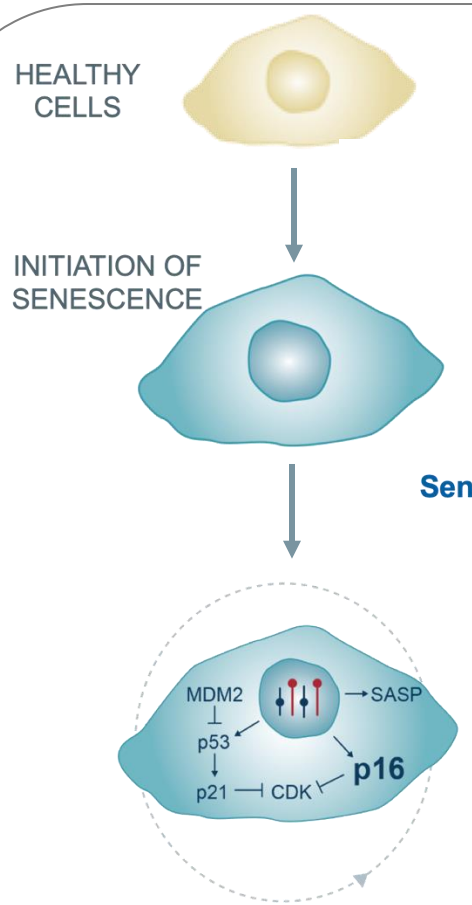
L. HAYFLICK and P. S. MOORHEAD

Wistar Institute of Anatomy and Biology, Philadelphia, Pa., U.S.A.

Received May 15, 1961

Leonard Hayflick (1928-2024)





Cellular Stress:

Aging

Diabetes

Inflammation

Unchecked Proliferation

Senescence-Associated Secretory Phenotype (SASP)

(e.g., IL-1β, IL-1A, IL-6, IL-7 IL-8, MMP1, TNF-α, PAI-1 …)

(e.g., TGFβ1, TIMP-1, MCP-1,…)

(e.g., VEGF-A, FGF-2, EGF, PDGF, PIGF,…)

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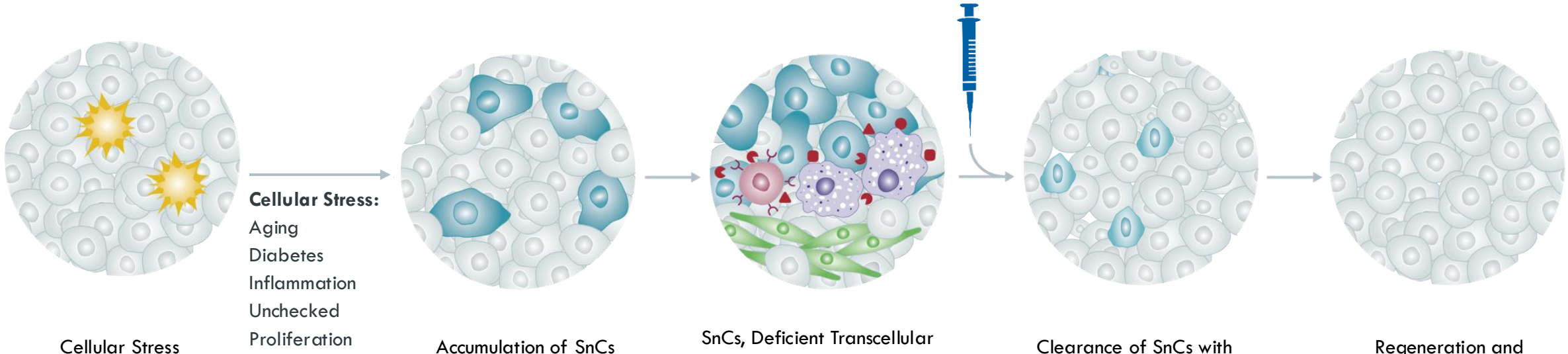
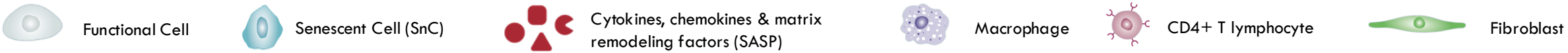
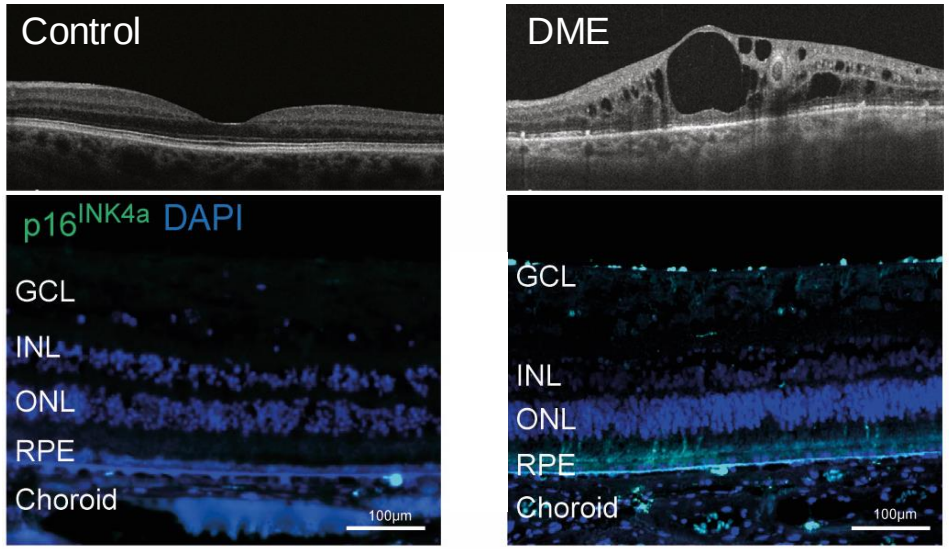
Targeting Senescent Cells to Restore Tissue Health

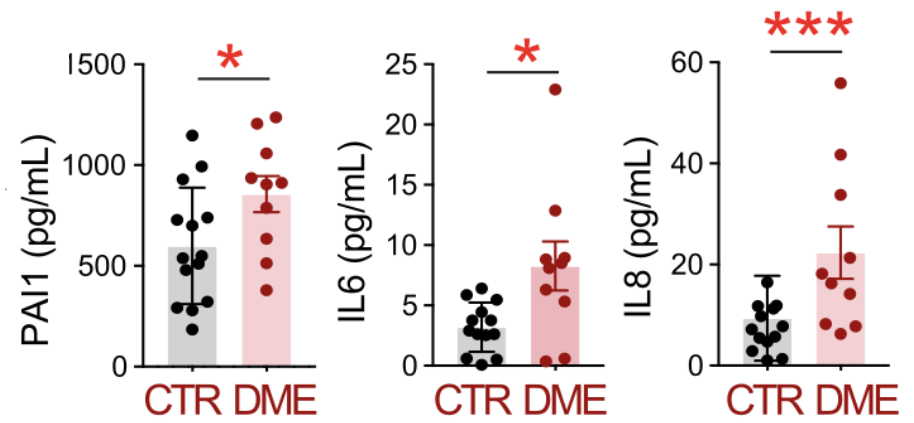
Target senescent cells to neutralize SASP factors and eliminate dysfunctional cells that are the root cause of disease progression

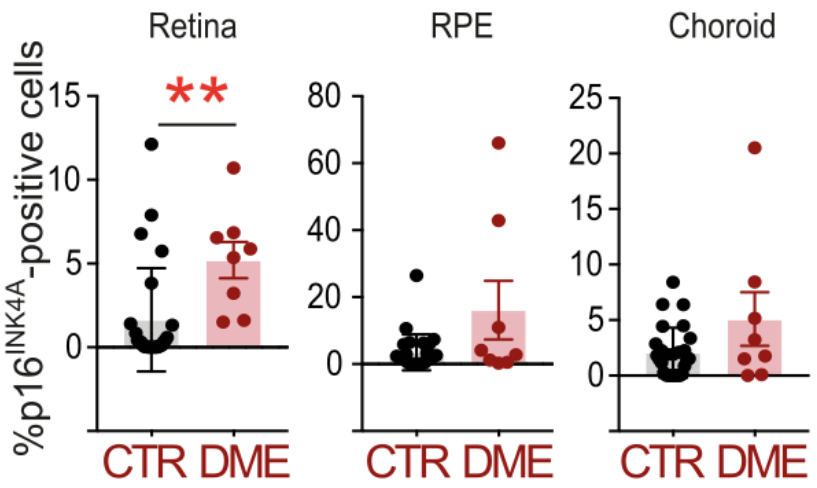




Senescent Cells & SASP Factors Increase in Patients with DME

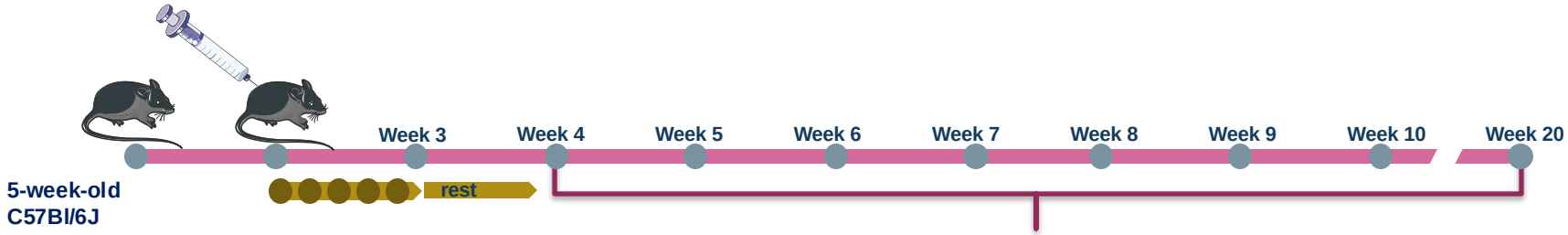


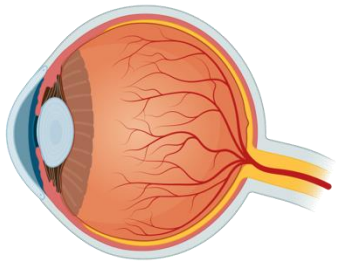




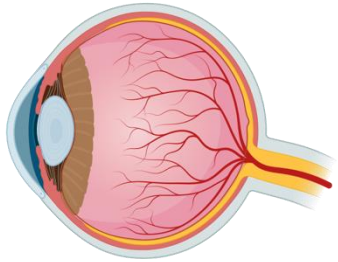
Crespo-Garcia et al *Nature Med*, 2024

Pathways of Cellular Senescence are Triggered in Endothelial Cells of the Diabetic Mouse Retina

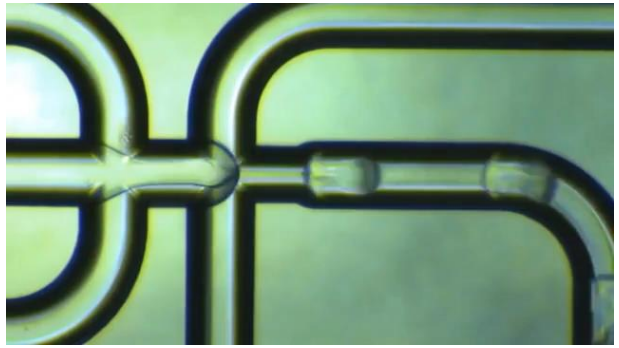




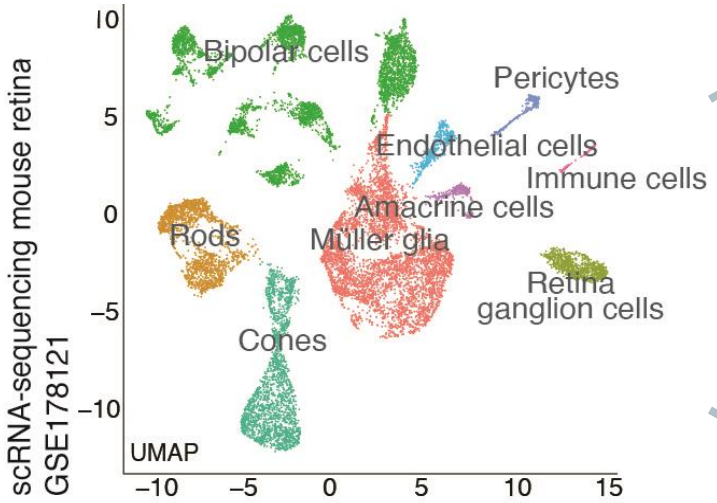
Healthy retina

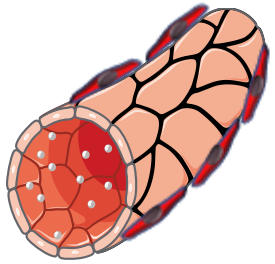


Diabetic retina

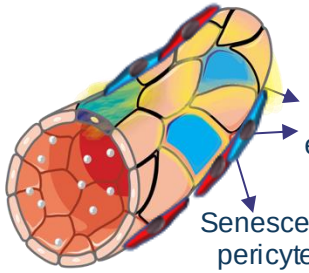


Single cell encapsulation technology



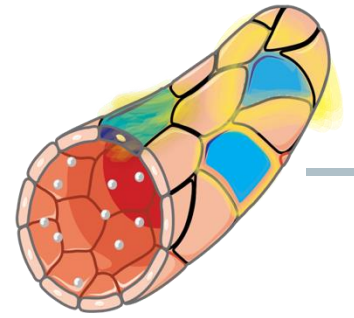


Healthy Retinal Blood Vessel



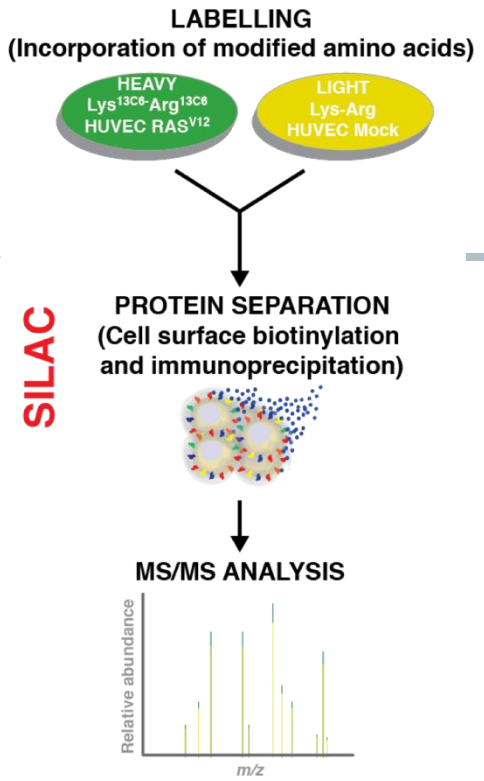
Diabetic Retinal Blood Vessel

Senescent Endothelial Cells (ECs) Produce Inflammatory Factors Through the SASP

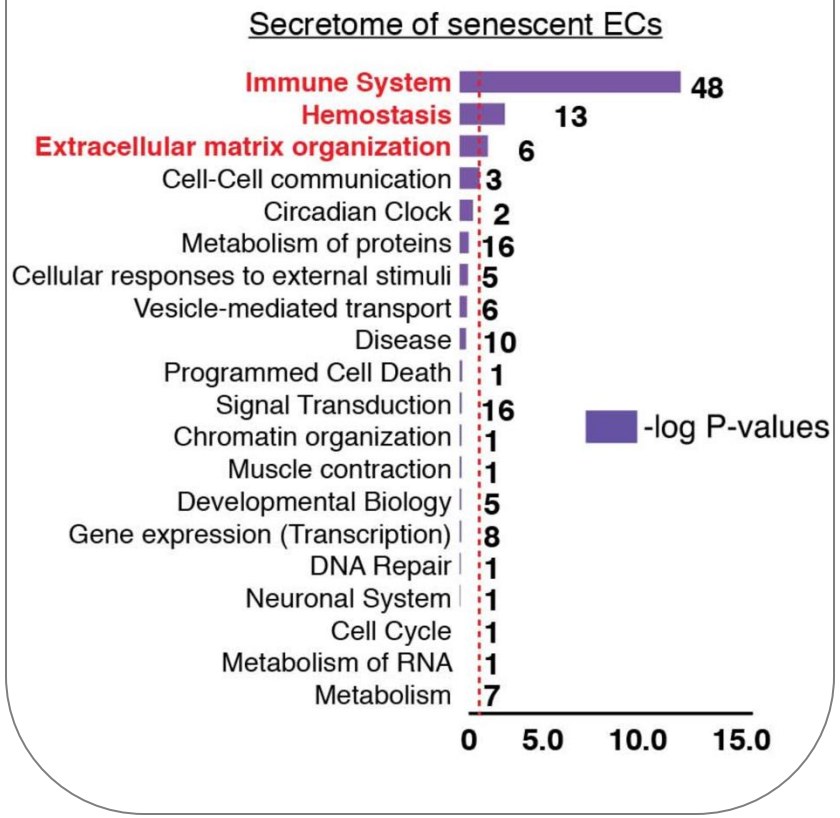


Diabetic Retinal Blood Vessel

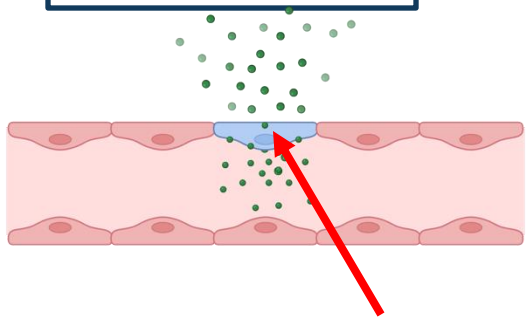
Labelling
(incorporation of modified amino acids)



Secretome of senescent ECs



Inflammatory Cytokines



Senescent EC

Binet et al *Science*, 2020

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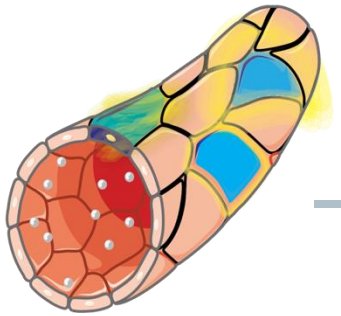
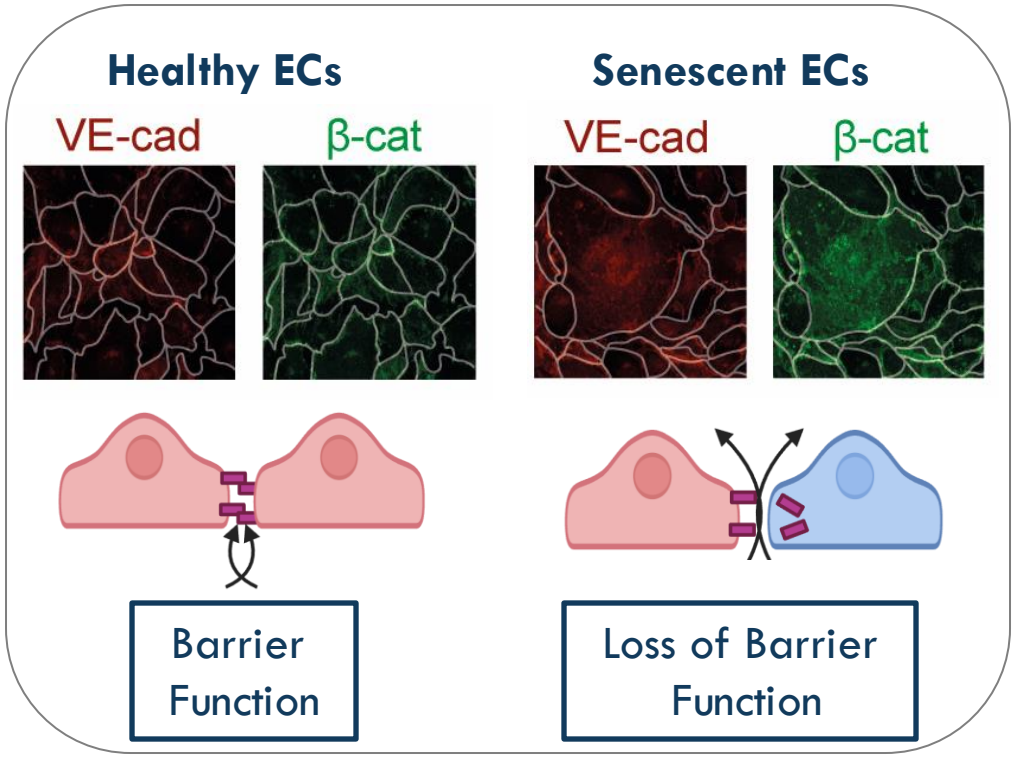
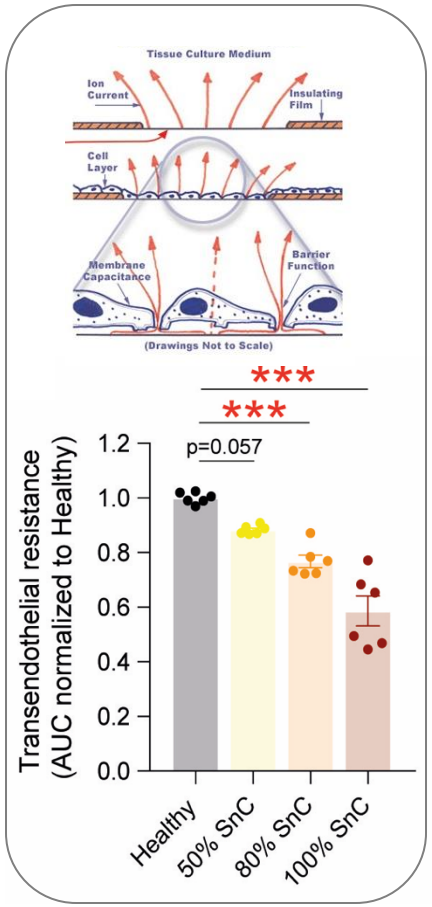
Senescent Endothelial Cells (ECs) Have Compromised Transcellular Junctions

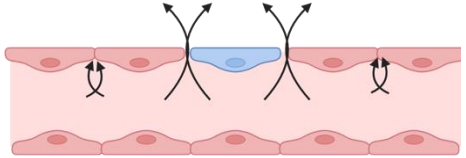


Diabetic Retinal Blood Vessel





Leaky blood vessel



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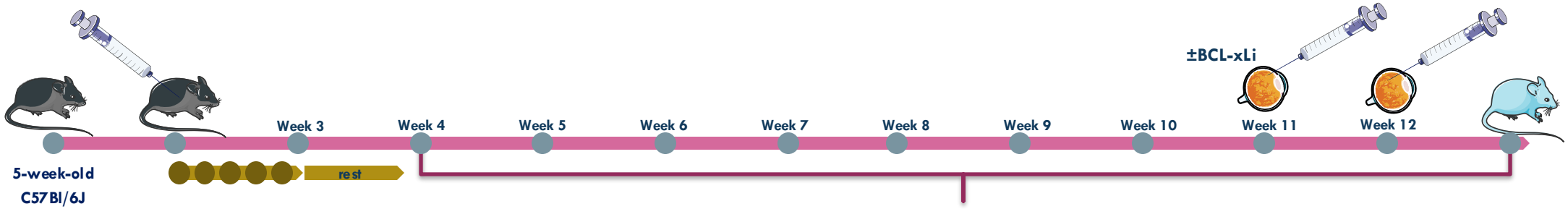
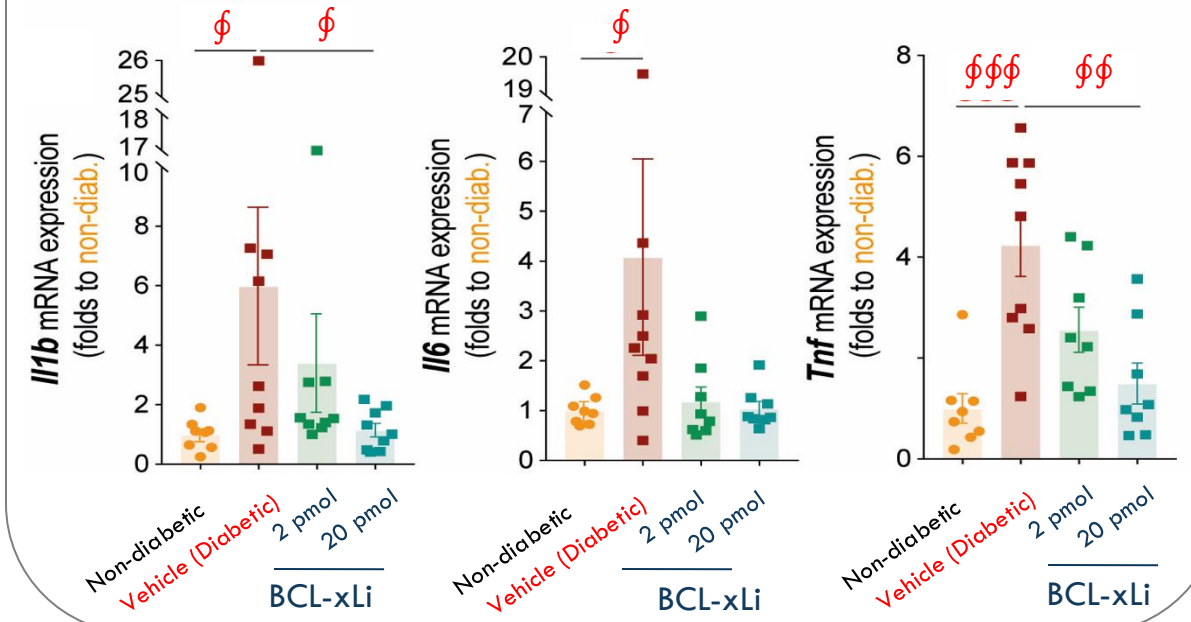
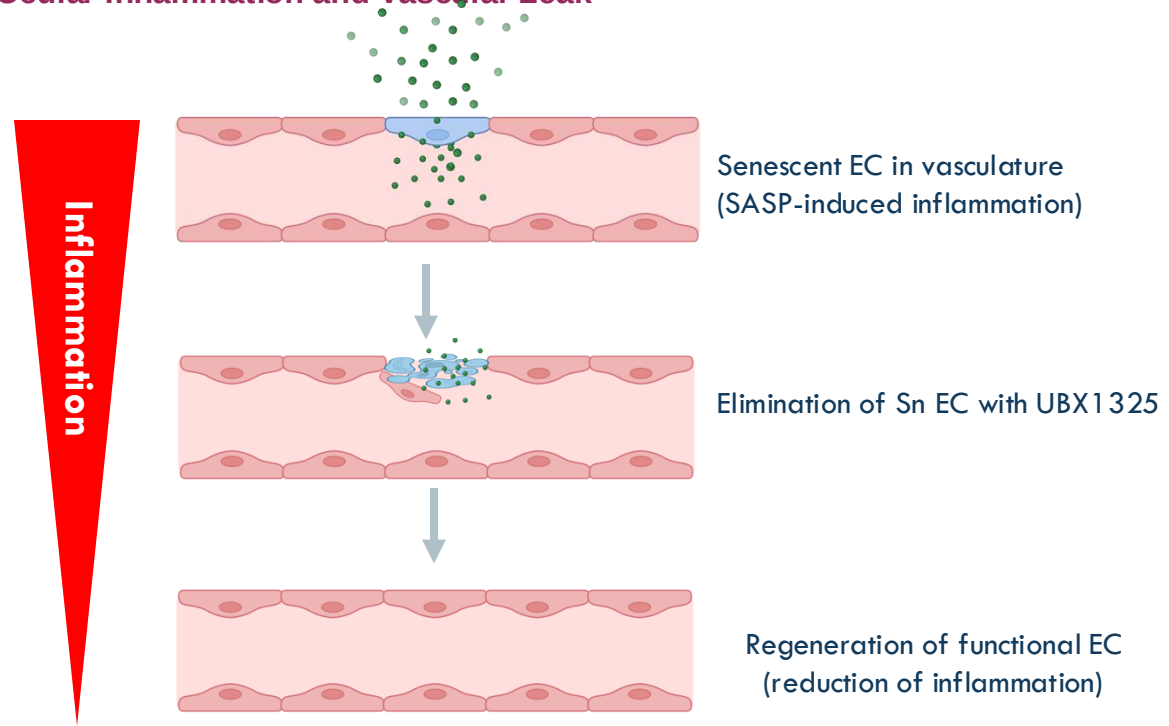
BCL-xL Inhibition Reduces Inflammation in a Mouse Model of Diabetes

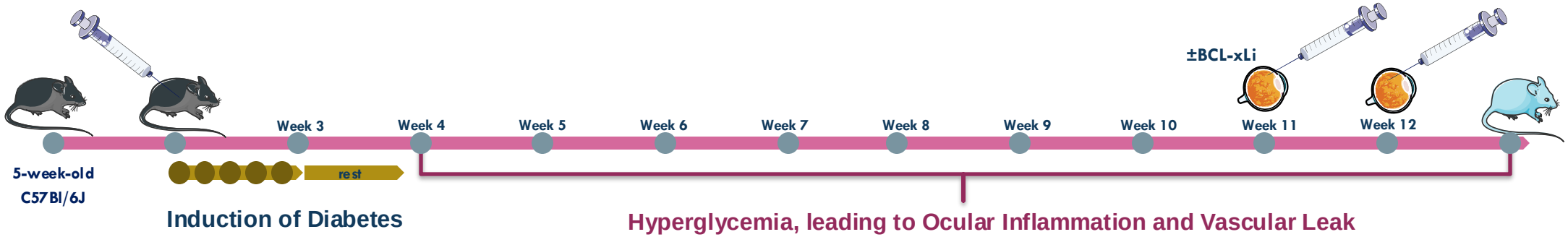


Resolution of Inflammation

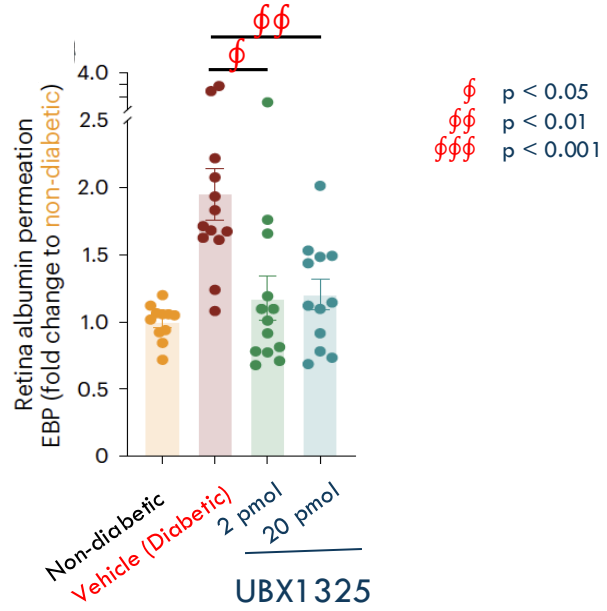


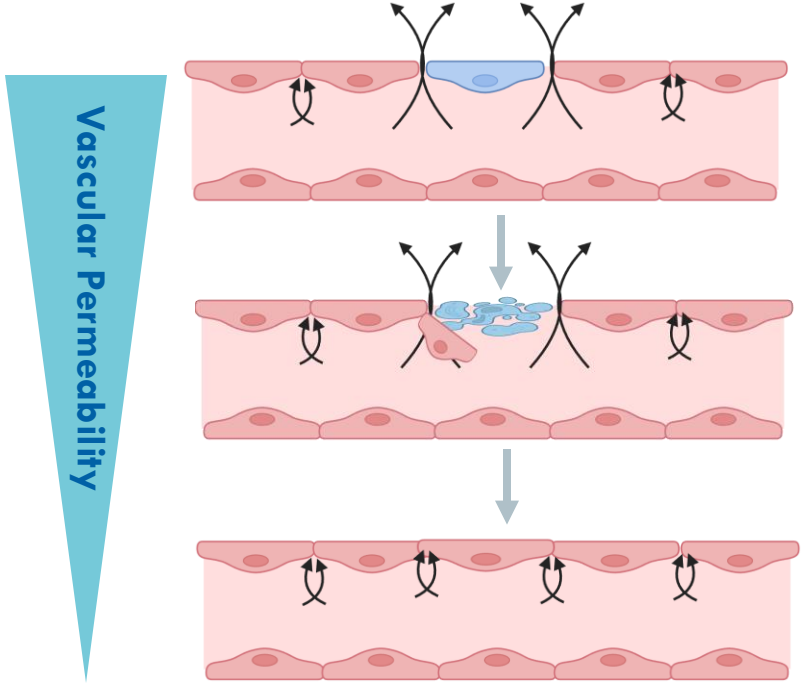


BCL-xL Inhibition Reduces Vascular Leakage in a Mouse Model of Diabetes



Reduction of Vascular Permeability



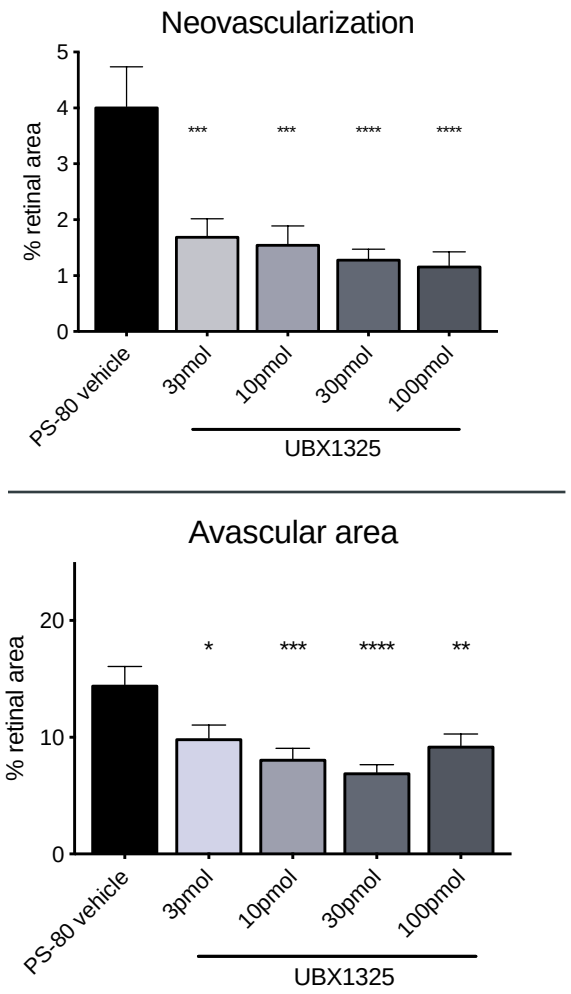


Senescent EC in vasculature
(compromised barrier function)

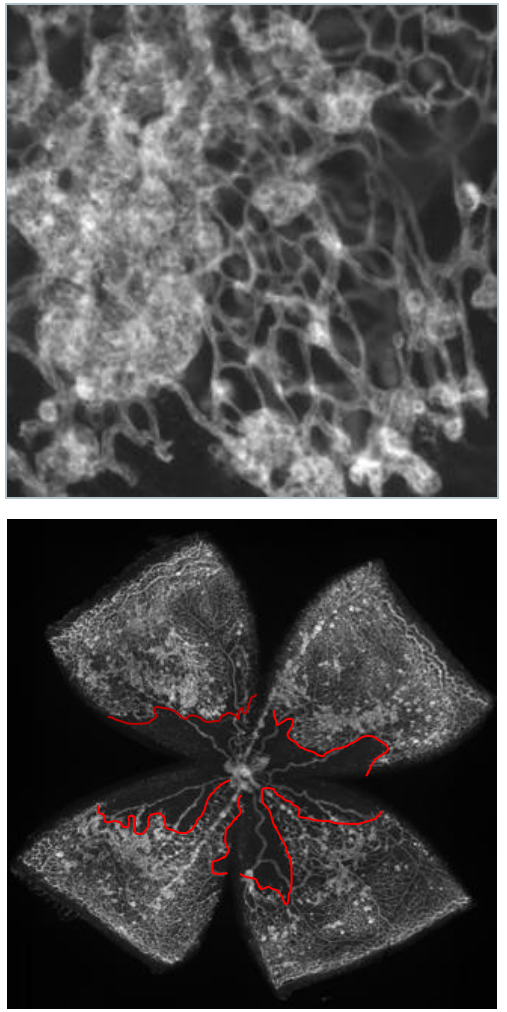
Elimination of Sn EC with UBX1325

Regeneration of functional EC
(restoration of barrier function

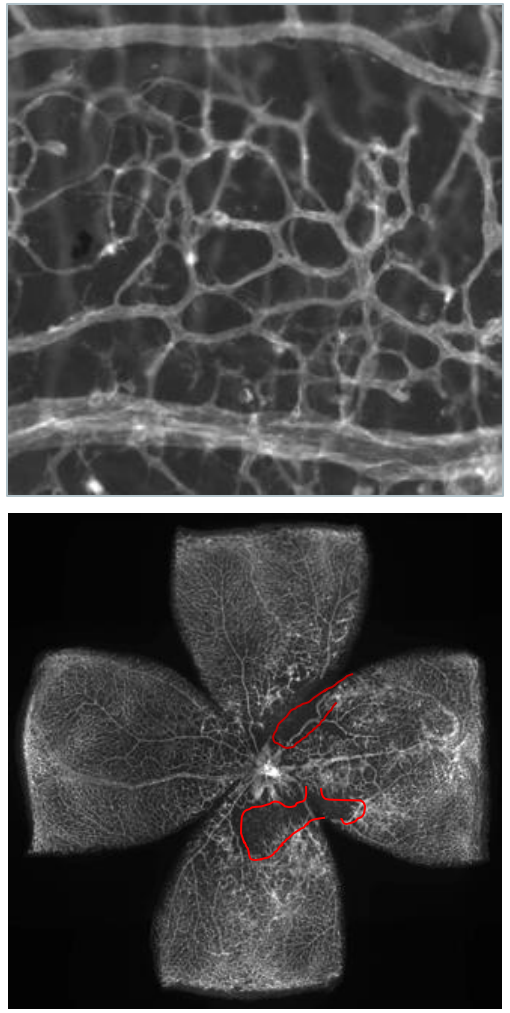
UBX1325 Improves Retinal Vasculature in Mouse Model of Neovascularization



OIR Vasculature



OIR Vasculature following UBX1325



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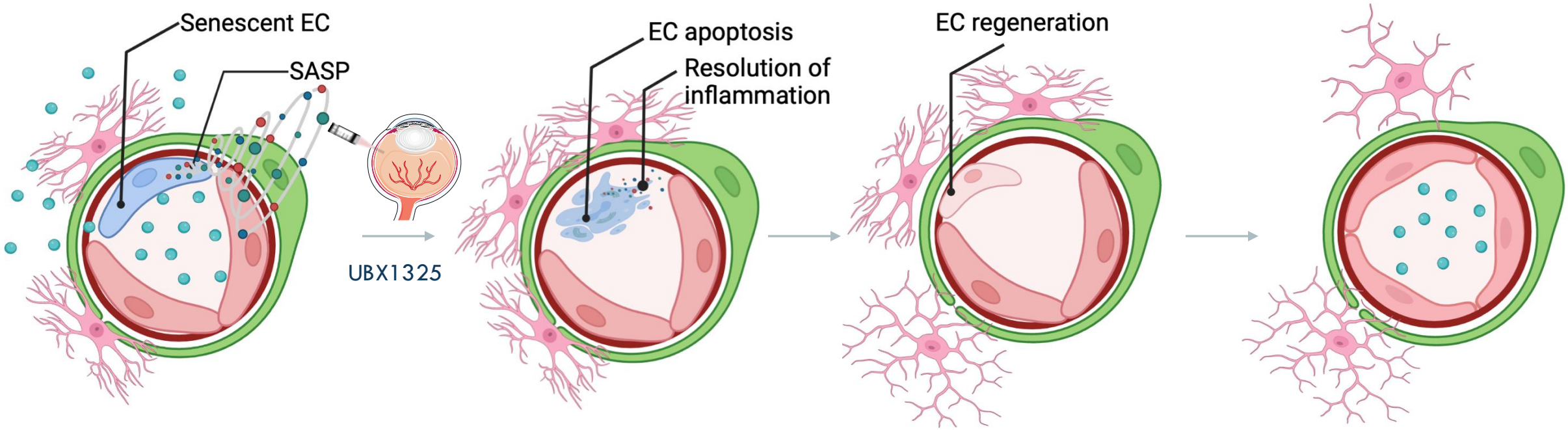
Proposed Mechanism of Action for UBX1325 in Retinal Disease



Diabetic blood vessel

Senescent (Sn) ECs accumulate in diabetic retinas in areas of disease activity

Vessel remodeling

UBX1325 selectively triggers cell death of Sn ECs. UBX1325 reduces retinal inflammation and leakage

Repaired blood vessel

Preclinical data predicts progressive disease modification through vascular remodeling

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UNITY Publication Update:
Mechanism of Action for UBX1325 published in *Nature Medicine*

nature medicine

Article

https://doi.org/10.1038/s41591-024-02802-4

Therapeutic targeting of cellular senescence in diabetic macular edema: preclinical and phase 1 trial results

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Check for updates

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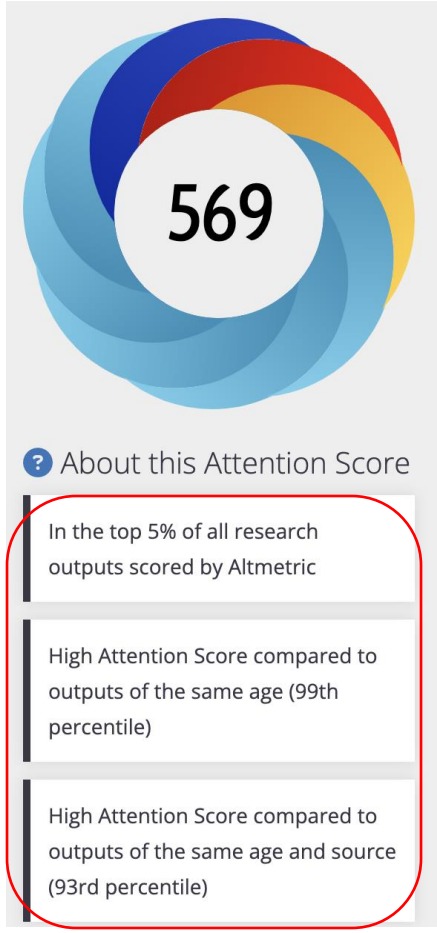

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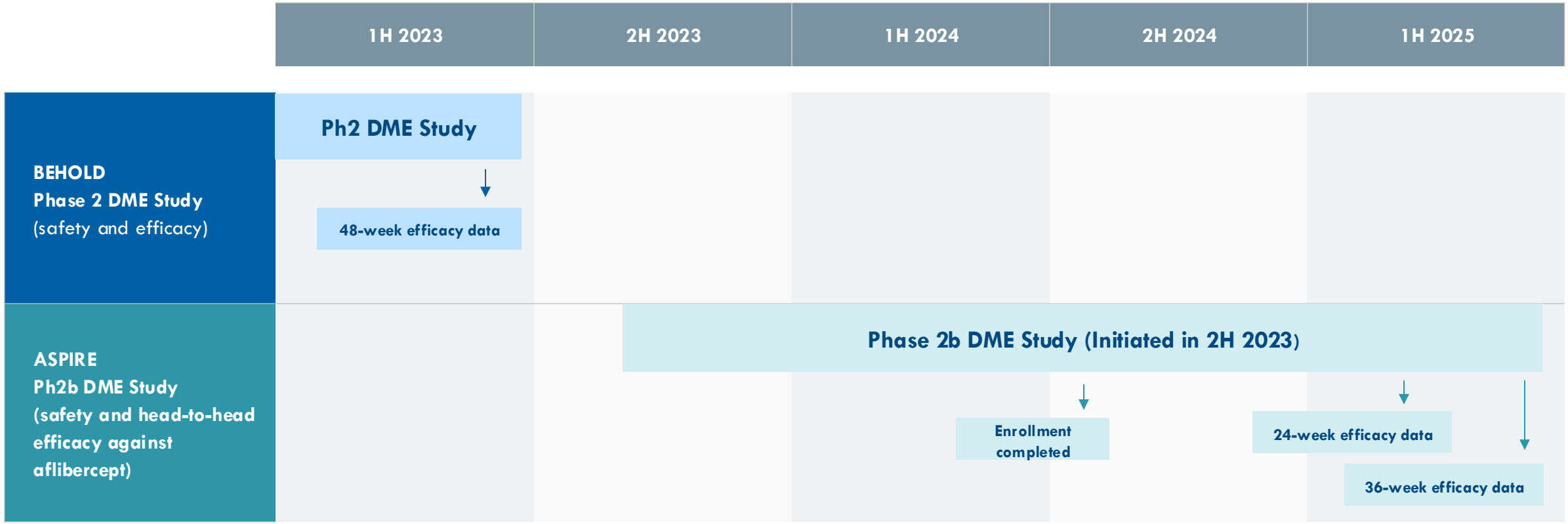
UBX1325 Clinical Development Plan

Ph2 BEHOLD Study in Patients with DME

UBX1325 (Foselutoclax) Clinical Program in Diabetic Macular Edema (DME)
BEHOLD and ASPIRE Studies



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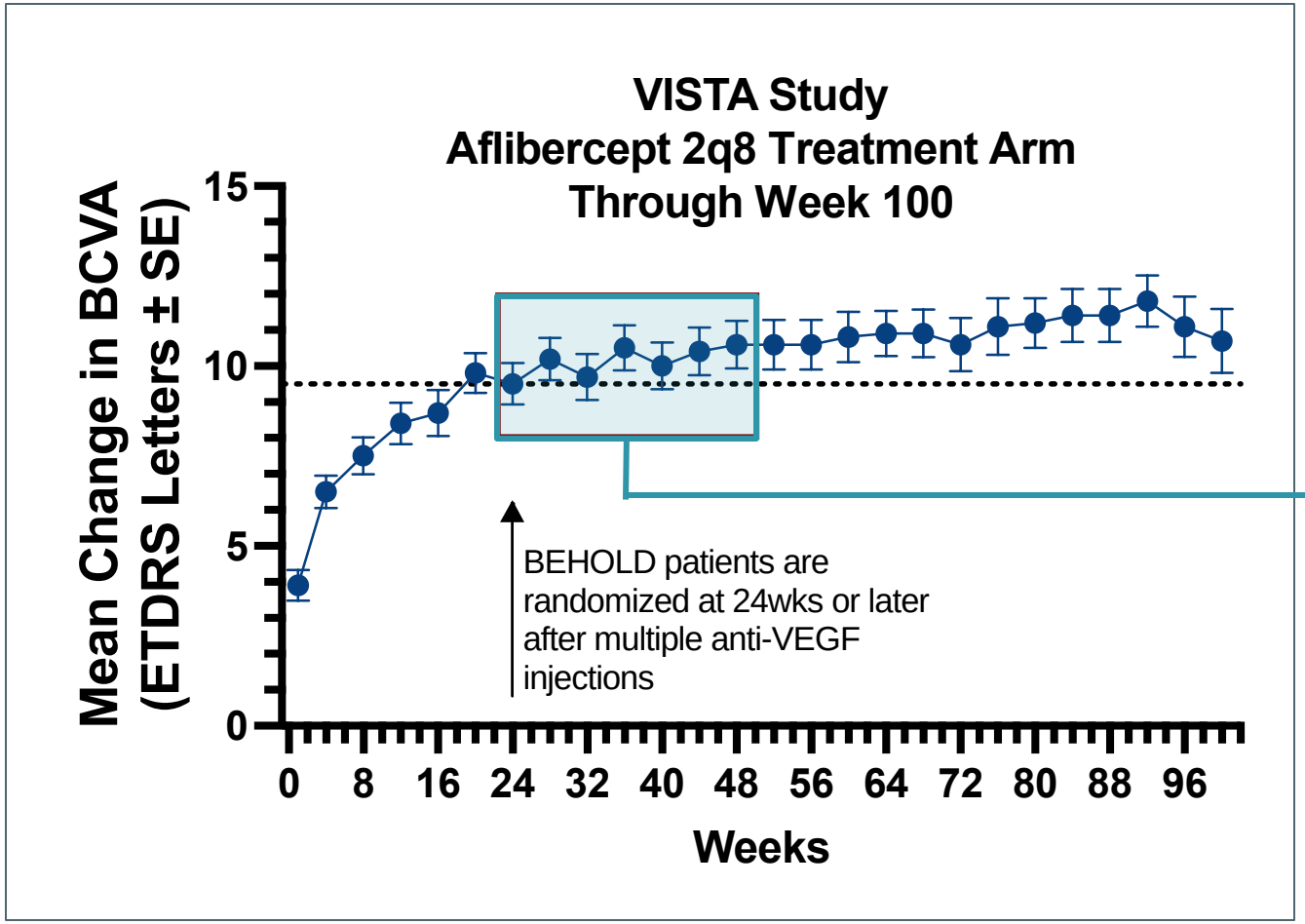
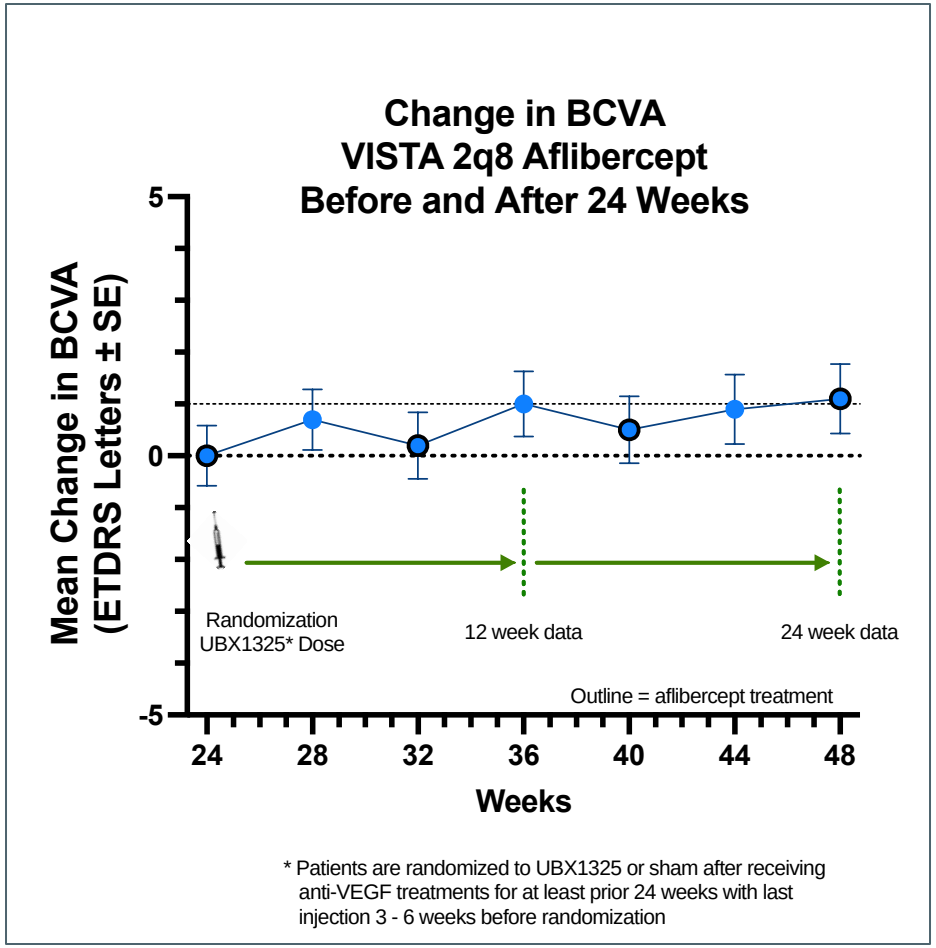
Context for Novel Therapeutic Options for Patients with DME: Patients on anti-VEGF Plateau After 6 months of Treatment and Stop Gaining Vision





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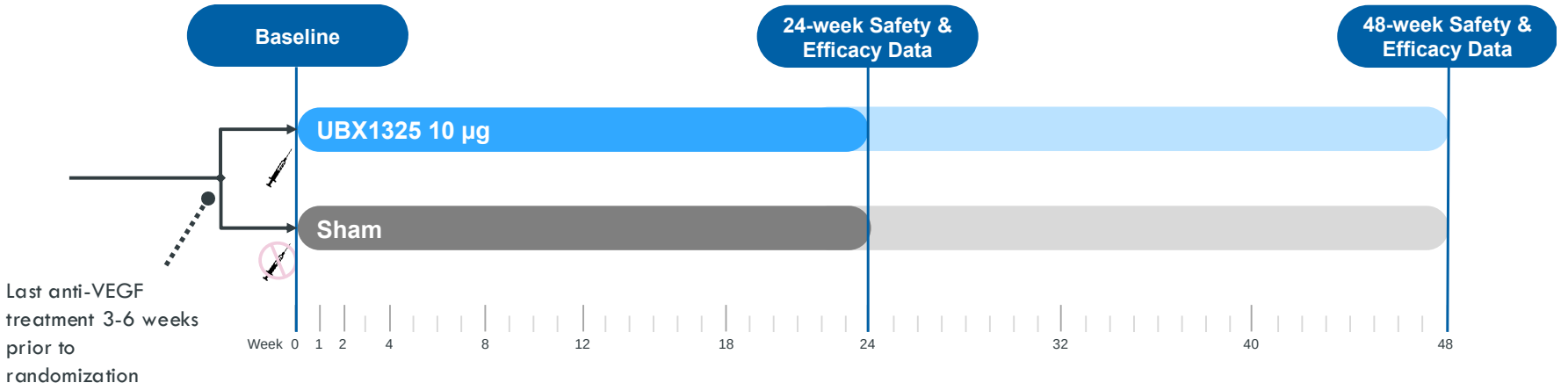
BEHOLD Study Design, Patient Population, and Endpoints

Patient Population: Sub-optimal response to anti-VEGF

- Individuals with **Diabetic Macular Edema**
- **Repeated anti-VEGF** treatments (≥2 injections/6 months) – Actual: 4.1 injections in prior 6 months
- **Residual retinal fluid** (≥300 µm) – Actual: 439.6 µm
- **Visual acuity deficit** (73 ETDRS letters or worse) – Actual: 61.4 ETDRS letters



	Sham	UBX	Total
Full Analysis Set	33	32	65
Completed to 24 Weeks only	4	5	9
Lost to follow-up	1	3	4
Site Closure	1	0	1
Patient withdrawal	1	0	1
Available through 48 Weeks	26	24	50

= UBX1325 injection

= Sham injection

Endpoints

Safety and tolerability

BCVA change from baseline

Durability of response

Sub- and intra-retinal fluid, CST changes

Proportion of UBX1325 patients requiring 2 or more rescue treatments

UBX1325-treated Patients had Marked Reduction in Need for Anti-VEGF Rescue Compared to Sham-treated Patients Through 48 weeks

- Median Time-To-First-Rescue in UBX arm was >48 weeks (at least 30 weeks greater than Sham arm)
- ~50% of UBX-treated patients went without rescue through 48 weeks
- ~80% of sham-treated patients required rescue before 48 weeks

Rescue Criteria (Either)

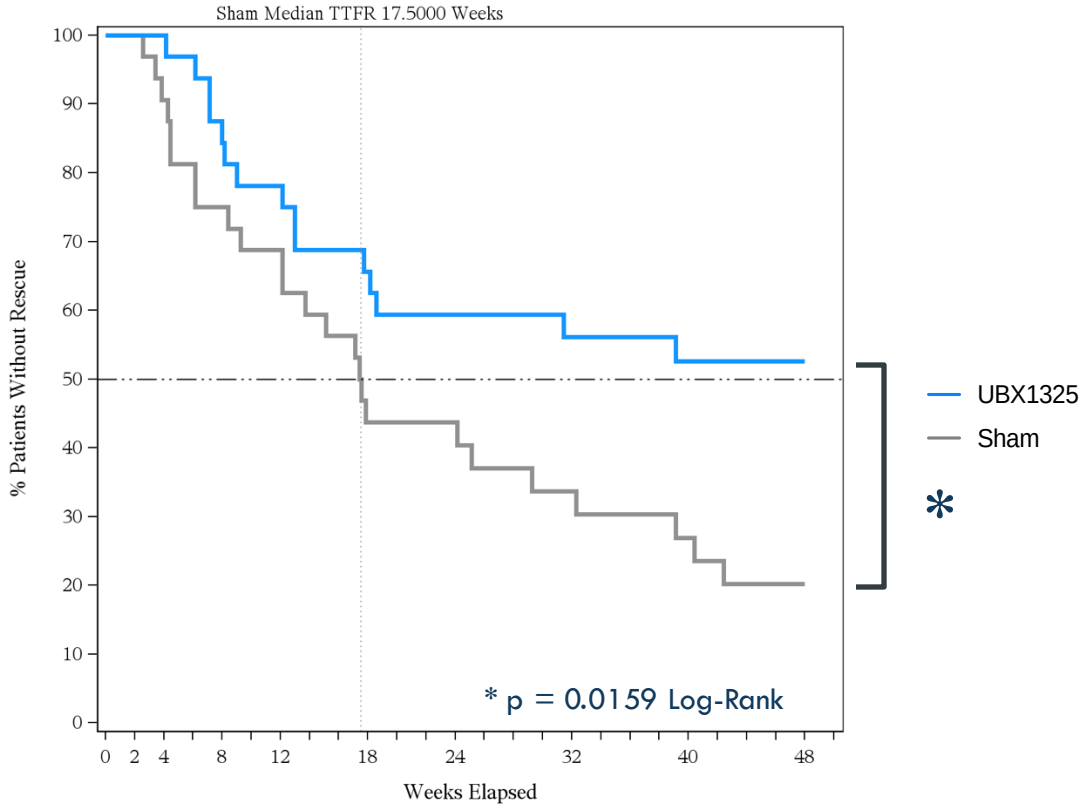
Decrease of 10 ETDRS or more letters from any peak value

Increase in CST of 75 µm or more from baseline

Physician discretion

Efficacy analyses *excluding* and *including* data post anti-VEGF rescue show a treatment benefit of UBX1325

Median Time to First Rescue Sham: 17.5 Weeks



BEHOLD Ph2 Study in Patients with DME

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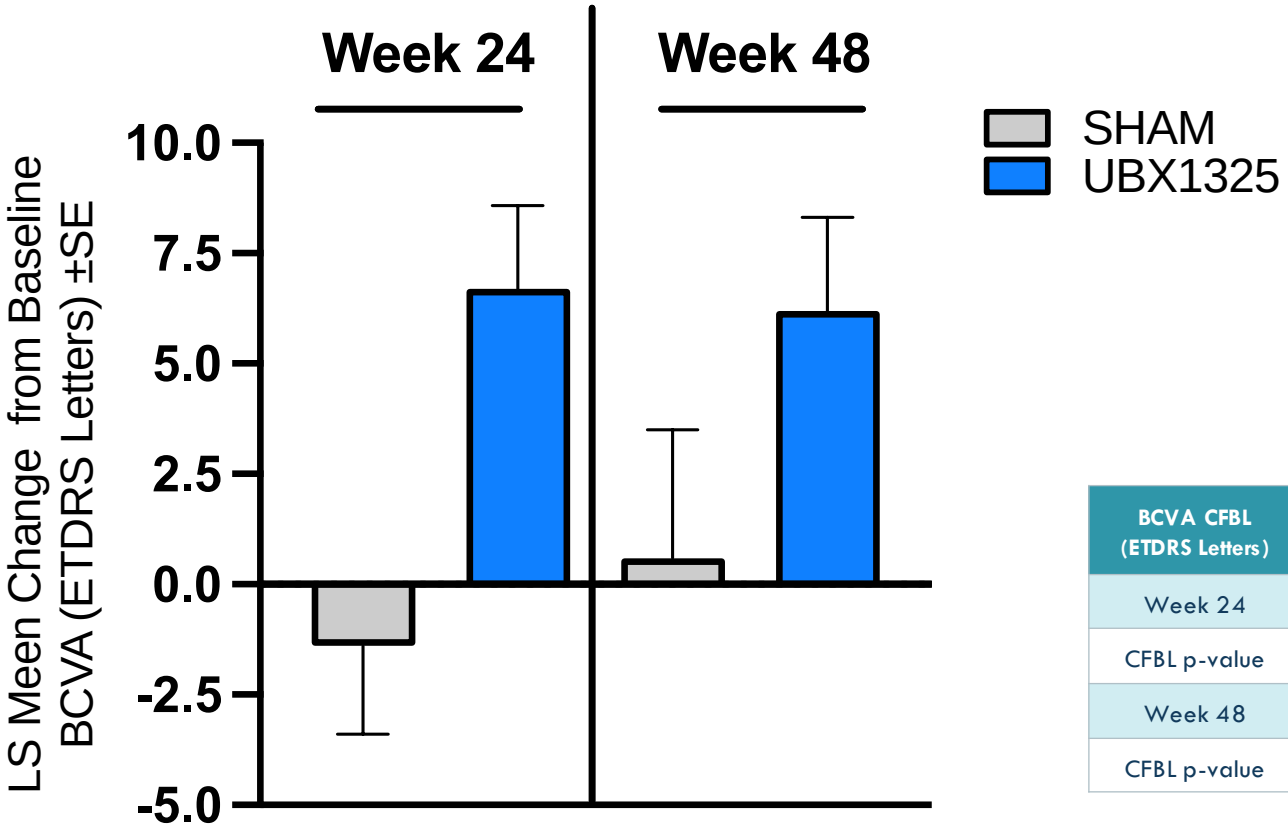
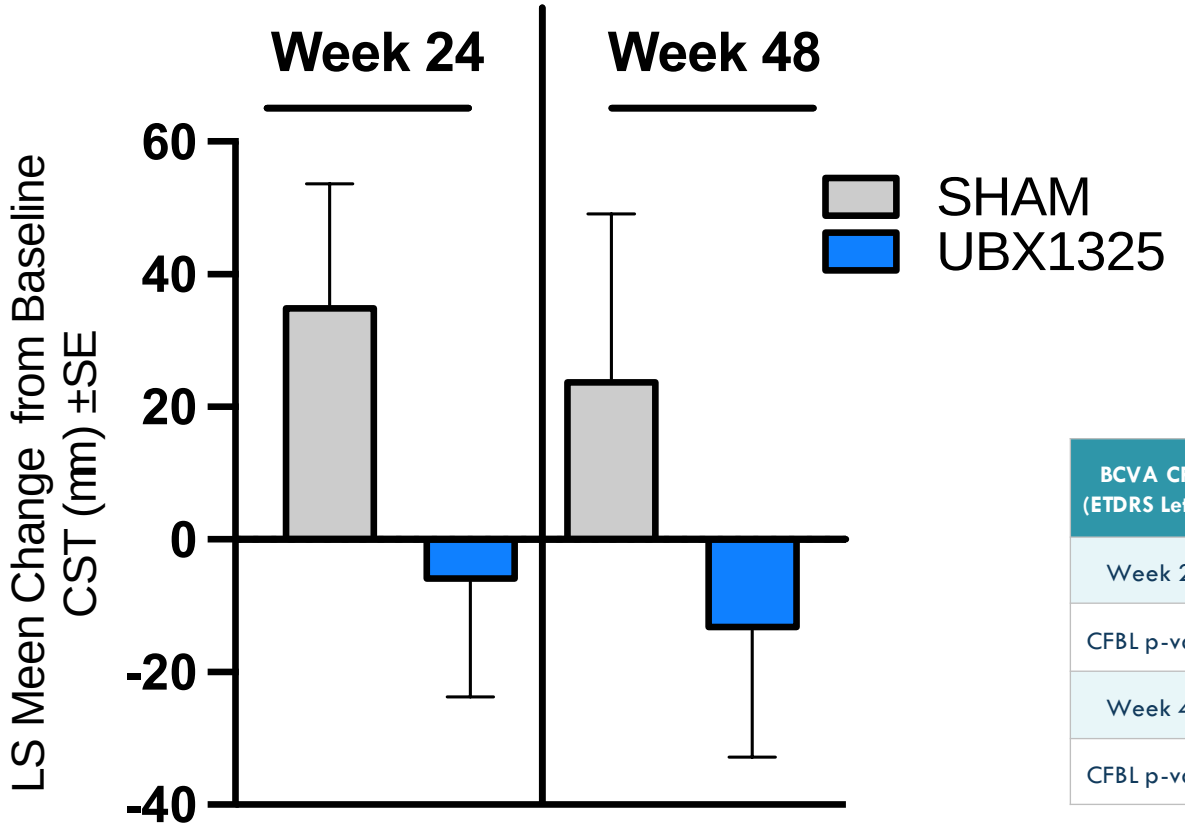
UBX1325-treated Patients had a Significant Improvement in BCVA from Baseline at Weeks 24 and 48



BCVA CFBL (ETDRS Letters)	UBX1325	Sham	Delta	Between Group p-value
Week 24	6.7	-1.4	8.1	0.0031
CFBL p-value	0.0004	NS		
Week 48	6.2	0.6	5.6	0.1198
CFBL p-value	0.0037	NS		

MMRM analysis, excluding post-rescue data

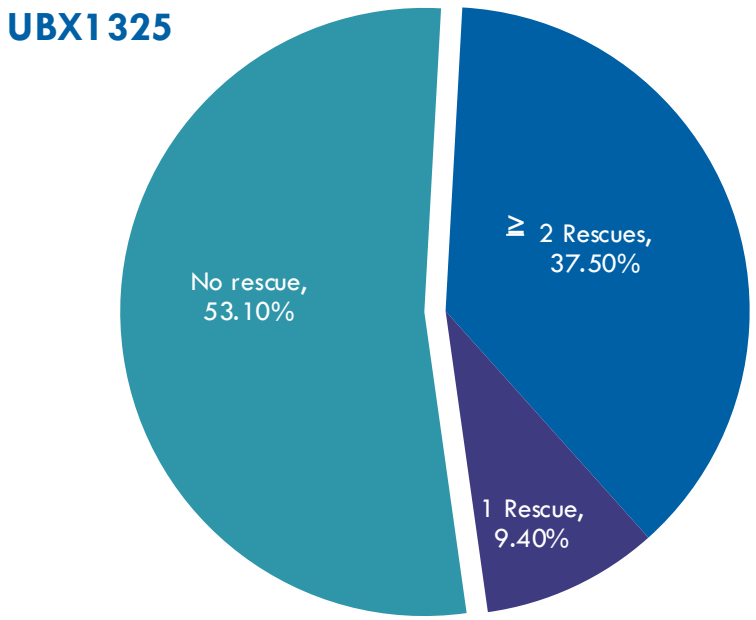
CST Remained Stable or Improved in UBX1325-treated Patients Compared to Worsening in Sham Patients

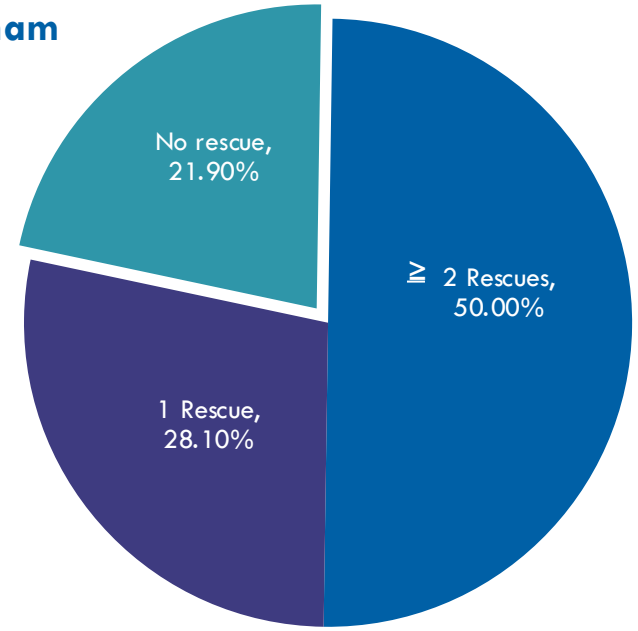


BCVA CFBL (ETDRS Letters)	UBX1325	Sham	Delta	Between Group p-value
Week 24	-6.4	35.4	-41.8	0.0985
CFBL p-value	NS	0.0534		
Week 48	-13.7	24.2	-37.9	NS
CFBL p-value	NS	NS		

MMRM analysis, excluding post-rescue data

53% of UBX1325-Treated Patients Did Not Require Anti-VEGF Rescue Through 48 weeks





UBX vs. Sham

P = 0.0096

Rescue Criteria (EITHER)

Decrease of 10 ETDRS or more letters from any peak value

Increase in CST of 75 µm or more baseline

Physician discretion

BEHOLD Ph2 Study in Patients with DME

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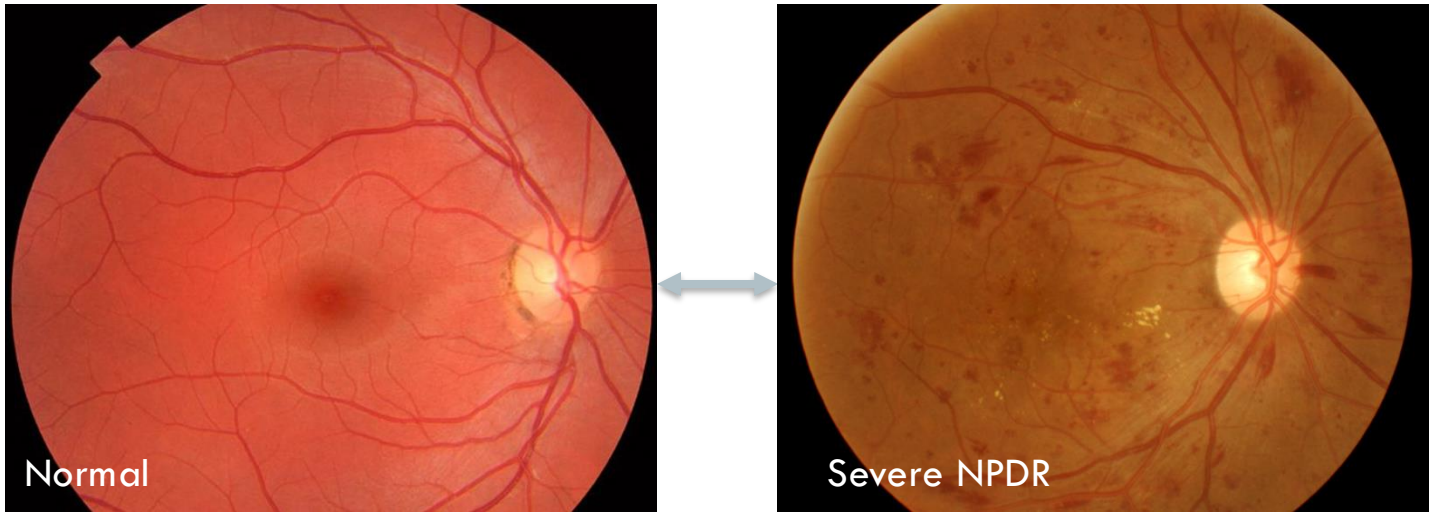
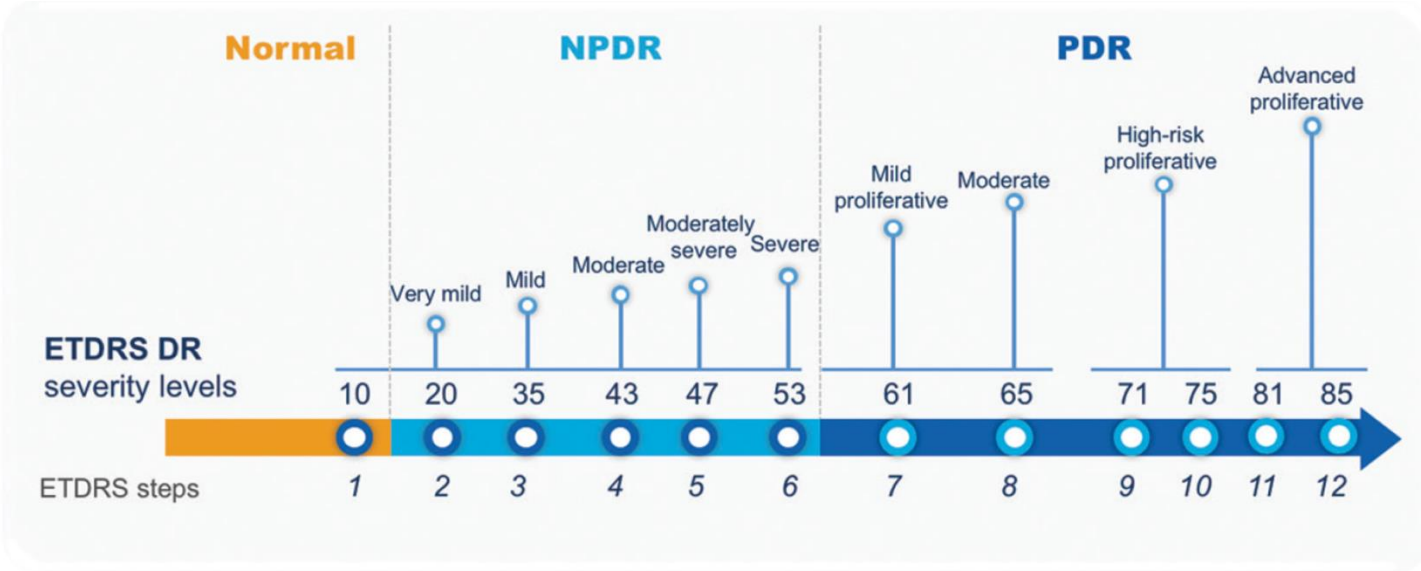
UBX1325 Demonstrated a Favorable Overall Safety and Tolerability Profile with no Instances of Intraocular Inflammation

Parameter	Sham (n=33)	UBX1325 10 µg (n=32)
Subjects with at least one TEAE	31 (93.9)	26 (81.3)
Related TEAE	3 (9.1)	6 (18.8)
Grade >=3 TEAE	4 (12.1)	5 (15.6)
Serious TEAE	3 (9.1)	5 (15.6)
Ocular TEAE for Study Eye	28 (84.8)	23 (71.9)
Treatment-related Ocular TEAE for Study Eye	3 (9.1)*	6 (18.8)*
TEAE leading to death	0	0
Intraocular inflammation, endophthalmitis, retinal artery occlusion, or vasculitis	0	0

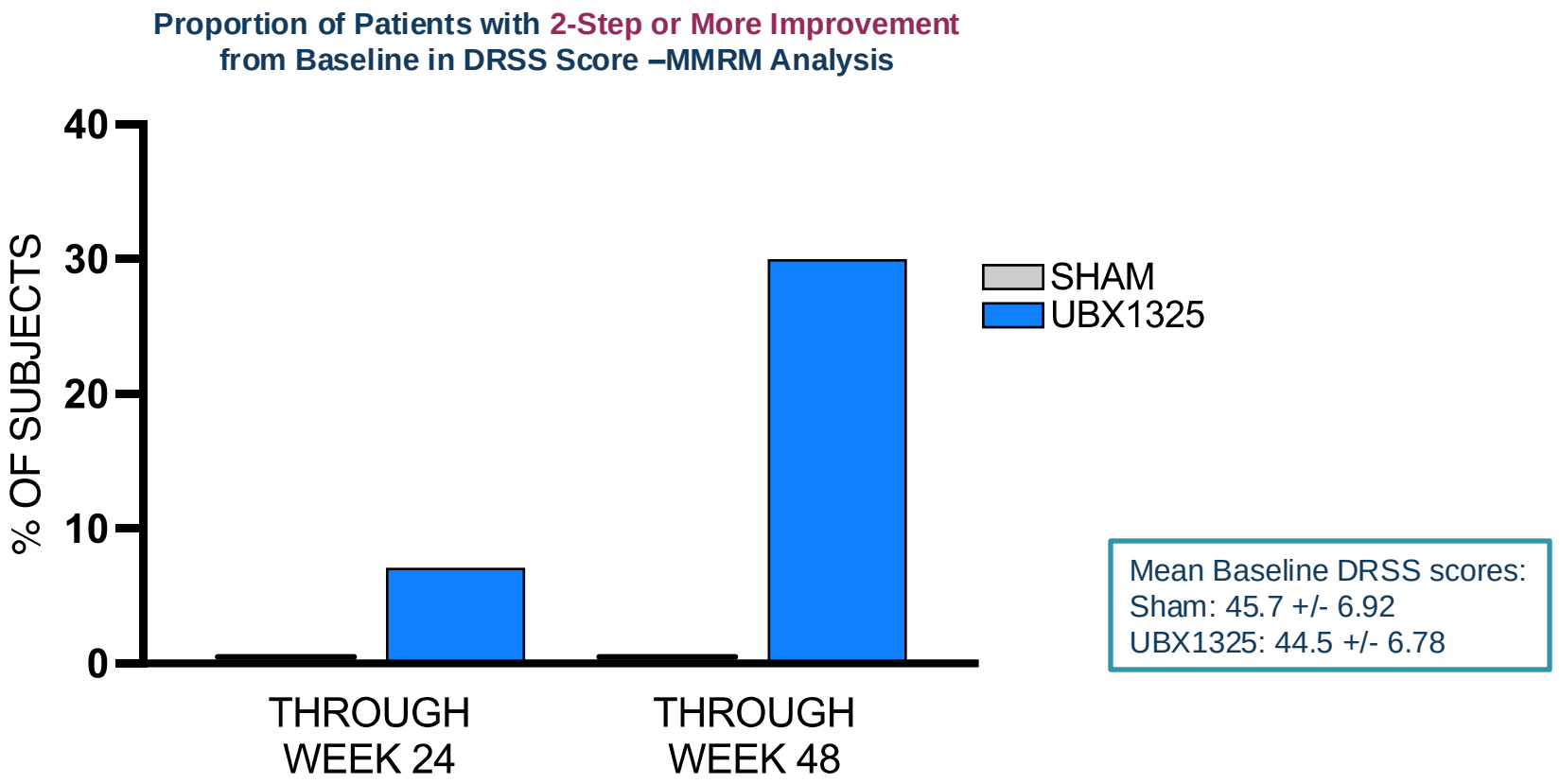
* Most are likely procedural related, all were mild-mod, and self-limited: Sham: 1 conj. hemorrhage, 1 conj. hyperemia, 1 diabetic macular edema. UBX: 5 conj. hemorrhage, 1 ant. chamber pigmentation, 1 eye irritation

Diabetic Retinopathy is Assessed Based on the Diabetic Retinopathy Severity Score (DRSS)





30% of UBX1325-treated Patients Had a 2-step Improvement in Diabetic Retinopathy Severity Score (DRSS) at Week 48 Compared to No Improvement in the Sham Arm



Mean Baseline DRSS scores:
Sham: 45.7 +/- 6.92
UBX1325: 44.5 +/- 6.78

MMRM analysis, excluding post-rescue data

BEHOLD Ph2 Study in Patients with DME

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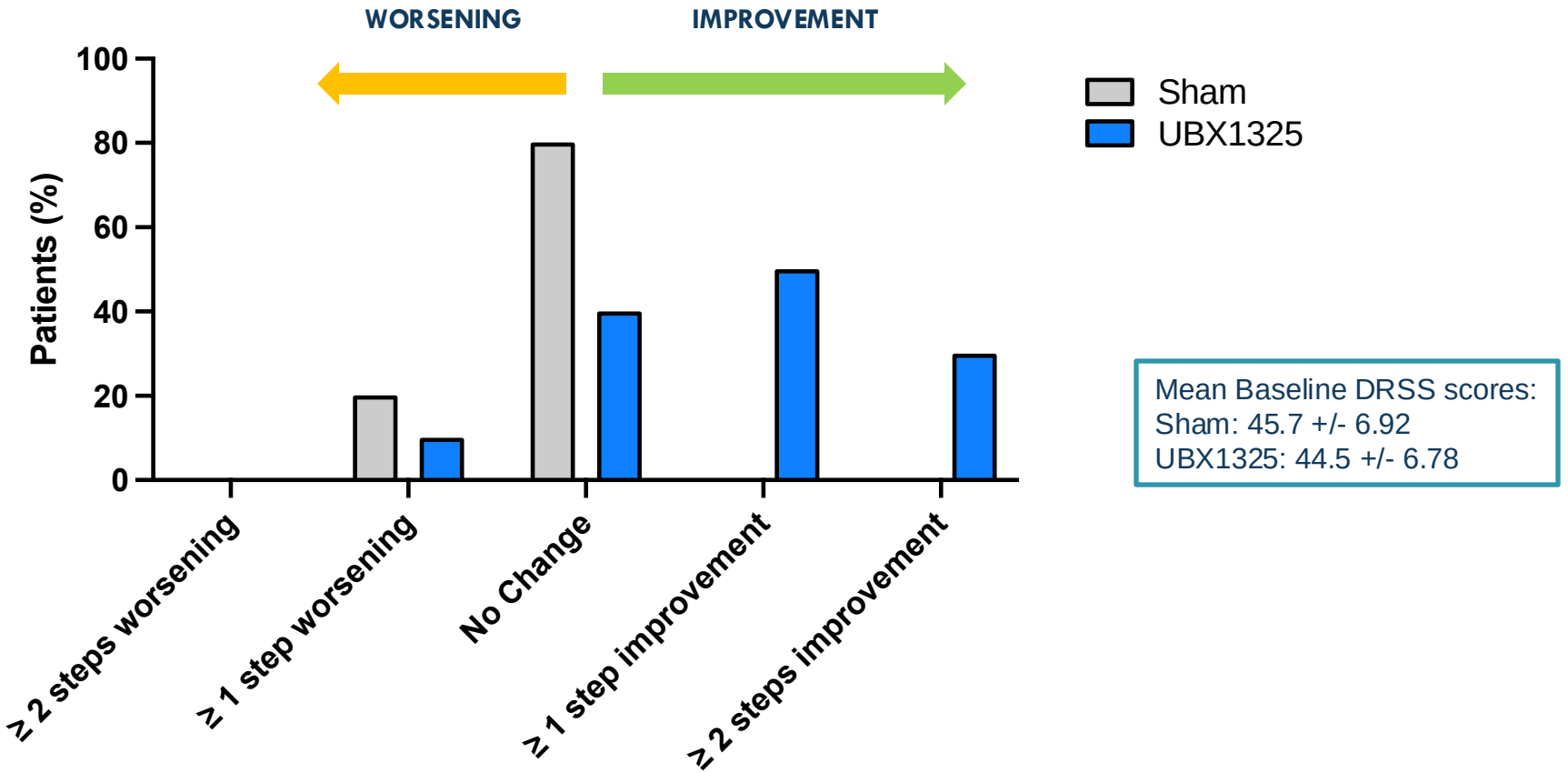
Over 40% of UBX1325-treated Patients Had a DRSS Score Improvement at Week 48 Compared to No Improvement or Worsening in the Sham Arm



Mean Baseline DRSS scores:
Sham: 45.7 +/- 6.92
UBX1325: 44.5 +/- 6.78

MMRM analysis, excluding post-rescue data

BEHOLD Ph2 Study in Patients with DME

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In the BEHOLD Study, UBX1325:

- Improved visual acuity at 48 weeks by **6.2 letters from baseline after a single injection**
- Led to ~50% of patients achieving a **rescue-free interval of at least 48 weeks** and may represent the **potential for disease modification**
- **Maintained retinal structure** throughout the duration of the study without the need for anti-VEGF rescue
- Had a **generally favorable safety and tolerability profile** with no intraocular inflammation or other serious TEAE

UBX1325 may be an important future therapeutic option for patients with diabetic macular edema

UBX1325

Summary of Findings and Concordance of Evidence Supporting a Treatment Effect of UBX1325 in Diabetic Macular Edema

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ASPIRE Ph2b Study

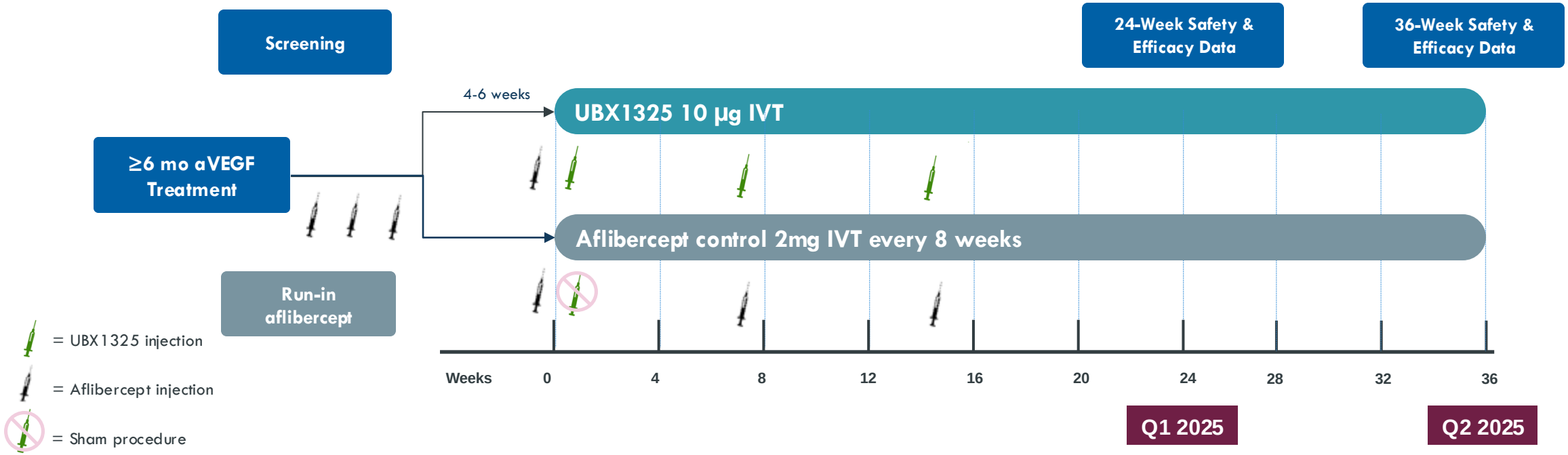
UBX1325 head-to-head against aflibercept in patients with DME

FDA Type C Interaction and Pivotal Study Expectations

CMC Readiness

Pivotal Drug Product

ASPIRE: DME Phase 2b Study Design (Head-to-head against aflibercept)



Patient Population: Participants with NPDR who have active DME despite treatment with ≥3 anti-VEGF injections in preceding 6 months; BCVA 70 – 30 ETDRS letters; CST >325µm

- **Duration: 36 Weeks; Randomization: 1:1**
- **Size: n=50 (25 /arm)**

Endpoints

Primary endpoint: BCVA change from baseline to week 24 (noninferiority)

Secondary endpoints include: BCVA change from BL over time • CST change from BL over time • proportion of patients gaining ≥15, ≥10, ≥5, or ≥0 letters from BL • safety and tolerability • proportion of participants who do not require anti-VEGF rescue

Exploratory endpoints: DRSS change from BL at weeks 24 and 36

FDA Type C Interaction and Implications for Ph3 Design

Type C Engagement

Recent FDA Type C engagement with written feedback received on Ph3 Study expectations

Opportunity for a **pivotal study largely in line with Ph2 ASPIRE trial**

Ph3 Design Implications

Primary Endpoint: Non-Inferiority to Aflibercept as assessed by BCVA with 4.0-letter non-inferiority margin

Comparator: Aflibercept 2mg Q8 weeks

ASPIRE Top Line Results expected in Q1 2025 to inform End of Ph2 meeting and final Phase 3 design

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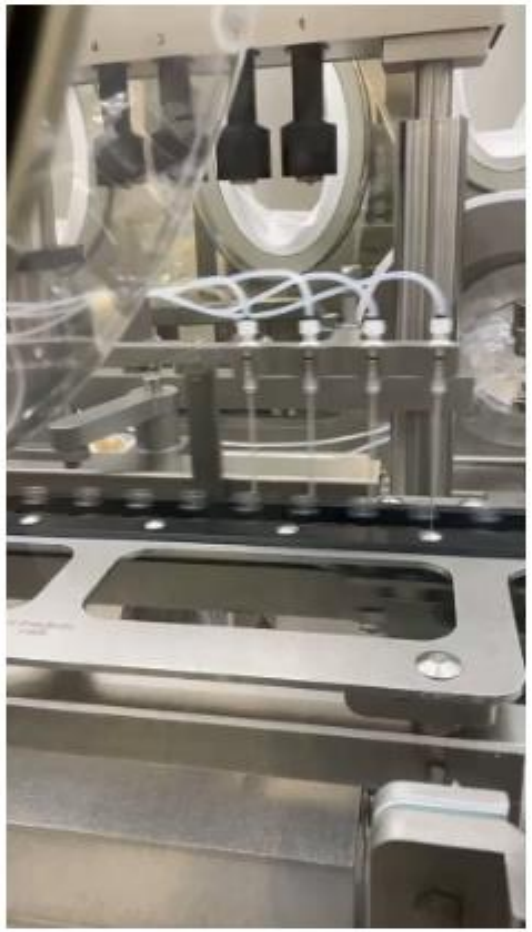
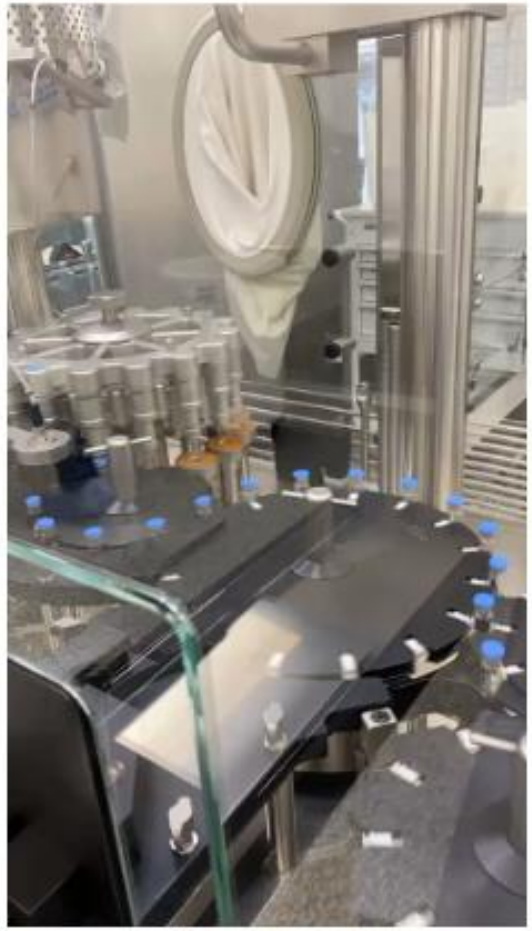
CMC Readiness: Successful Engineering Batch run for Pivotal Drug Product



Filling



Capping

One tray of UBX1325 DP

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Fireside Chat with Retina Experts

Arshad Khanani, MD, MA, FASRS

Dante Pieramici, MD, FASRS

Fireside Chat: Welcome and Introductions

Dante Pieramici, MD, FASRS

Partner California Retina Consultants/Retina Consultants of America

Medical Director Clinical Research California Retina Consultants

President California Retina Research Foundation

Arshad M. Khanani, MD, MA, FASRS

Managing Partner, Director of Clinical Research, and Director of Fellowship at Sierra Eye Associates and Clinical Professor at the University of Nevada, Reno School of Medicine

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Closing Remarks

Anirvan Ghosh, CEO