OPUS GENETICS®

Delivering on the Promise of Gene Therapy for Rare Inherited Retinal Diseases

July 2026

Kendall, *RDH12* patient, with Maya

Disclosures and Forward-Looking Statements

Certain statements contained in this presentation are not statements of historical fact and are forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. Forward-looking statements give current expectations or forecasts of future events or our future financial or operating performance. Such statements include, but are not limited to, statements concerning our data from and future enrollment for our clinical trials, our pipeline of additional indications and anticipated regulatory milestones.

In some cases, you can identify forward-looking statements by the following words: “aim,” “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “ongoing,” “plan,” “potential,” “predict,” “project,” “should,” “will,” “would” or the negative of those terms, and similar expressions that convey uncertainty of future events or outcomes to identify these forward-looking statements.

These forward-looking statements reflect our management’s beliefs and views with respect to future events, are based on estimates and assumptions as of the date of this presentation and are subject to risks and uncertainties, many of which are beyond our control, that could cause our actual results to differ materially from those in these forward-looking statements, including, without limitation: our gene therapy product candidates are based on a novel technology and manufactured by a third party, which may result in delays and difficulties in obtaining regulatory approval; our planned clinical trials may face substantial delays, result in failure, or provide inconclusive or adverse results that may not satisfy U.S. Food and Drug Administration (“FDA”) requirements to further develop our therapeutic products; delays or difficulties associated with patient enrollment in clinical trials may affect our ability to conduct and complete those clinical trials and obtain necessary regulatory approvals; changes in regulatory requirements could result in increased costs or delays in development timelines; we depend heavily on the success of our product pipeline; if we fail to find strategic partners or fail to adequately develop or commercialize our pipeline products, our business will be materially harmed; others may discover, develop, or commercialize products similar to those in our pipeline before or more successfully than we do or develop generic variants of our products even while our product patents remain active, thereby reducing our market share and potential revenue from product sales; we do not currently have any sales or marketing infrastructure in place and we have limited drug research and discovery capabilities; the future commercial success of our products could significantly depend upon several uncertain factors, including third-party reimbursement practices and the existence of competitors with similar products; product liability lawsuits against us or our suppliers or manufacturers could cause us to incur substantial liabilities and could limit commercialization of any product candidate that we may develop; failure to comply with health and safety laws and regulations could lead to material fines; we have not generated significant revenue from sales of any products and expect to incur losses for the foreseeable future; our future viability is difficult to assess due to our short operating history and our future need for substantial additional capital, access to which could be limited by any adverse developments that affect the financial markets; raising additional capital may cause our stockholders to be diluted, among other adverse effects; instability and operational disruptions at government agencies, such as the FDA, may adversely impact our development and commercialization plans by causing delays and requiring the use of additional, unforeseen resources to obtain regulatory approval for trials or products in our pipeline; we operate in a highly regulated industry and face many challenges adapting to sudden changes in legislative reform or the regulatory environment, including due to government shutdowns and disruptions at government agencies, which cause delays, requires the use of additional, unforeseen resources, affects our pipeline stability, and could impair our ability to compete in international markets; we may not receive regulatory approval to market our developed product candidates within or outside of the U.S.; with respect to any of our product candidates that receive marketing approval, we may be subject to substantial penalties if we fail to comply with applicable regulatory requirements; our potential relationships with healthcare providers and third-party payors will be subject to certain healthcare laws and regulations, which could expose us to extensive potential liabilities; we rely on third parties for material aspects of our business, such as conducting our nonclinical and clinical trials and supplying and manufacturing bulk drug substances, which exposes us to certain risks; we may be unsuccessful in entering into or maintaining licensing arrangements or establishing strategic alliances on favorable terms, which could harm our business; inadequate patent protection for our product candidates may result in our competitors developing similar or identical products or technology, which would adversely affect our ability to successfully commercialize; we may be unable to obtain full protection for our intellectual property rights under U.S. or foreign laws; we may become involved in lawsuits for a variety of reasons associated with our intellectual property rights, including alleged infringement suits initiated by third parties; we are dependent on our key personnel, and if we are not successful in attracting and retaining highly qualified personnel, we may not be able to successfully implement our business strategy; as we grow, we may not be able to operate internationally or adequately develop and expand our sales, marketing, distribution, and other corporate functions, which could disrupt our operations; the market price of our common stock is expected to be volatile and if we fail to comply with the continued listing standards of Nasdaq, our common stock may be delisted; and factors out of our control related to our securities, such as securities litigation or actions of activist stockholders, could adversely affect our business and stock price and cause us to incur significant expenses.

We discuss many of these risks in greater detail under Part I, Item 1A “Risk Factors” in our Annual Report on Form 10-K for the year ended December 31, 2025 and in subsequent reports filed with or furnished to the Securities and Exchange Commission. Moreover, we operate in a very competitive and rapidly changing environment. New risks emerge from time to time. It is not possible for our management to predict all risks, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statements we may make. Given these uncertainties, you should not place undue reliance on these forward-looking statements.

Any forward-looking statement in this presentation speaks only as of the date hereof or as of the date specified herein. We undertake no obligation to publicly update any forward-looking statement, whether as a result of new information, future developments or otherwise, except as may be required by applicable laws or regulations.

The Opus Opportunity: Fully-Funded to Advance 5 IRD Clinical Programs

7 Targeted IRD AAV gene therapy assets

Portfolio approach produces multiple data readouts and milestones

Validated science & delivery approach

Follow-on treatments from the first approved IRD gene therapy

1st Mover advantage in multiple indications

Broad IP protection and eligible for Orphan Drug exclusivity

Streamlined timelines & capital efficiency

Cost-effective development: Efficient programs with compelling economics

Rare disease regulatory advantages

Flexibility & potentially streamlined paths to approval

Revenue & partnership streams drive value

Non-dilutive & voucher funding plus partnered strategic financial asset

LCA5 — BEST1 — RDH12 — MERTK — RHO — CNGB1 — NMNAT1

AAV, adeno-associated virus; BEST1, bestrophin 1; CNGB1, cyclic nucleotide-gated channel β1; IP, intellectual property; IRD, inherited retinal disease; LCA5, Leber congenital amaurosis 5; MERTK, MER proto-oncogene tyrosine kinase; NMNAT1, nicotinamide mononucleotide adenylyltransferase; RDH12, retinol dehydrogenase 12; RHO, rhodopsin.

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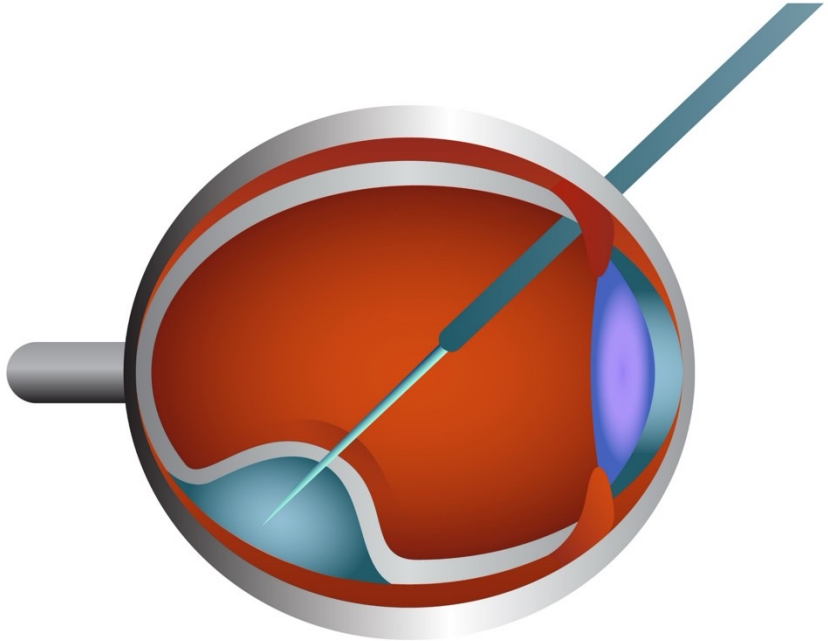
Precision-Targeted, One-Time-Treatment for Rare Diseases

350+ genes known to cause IRDs

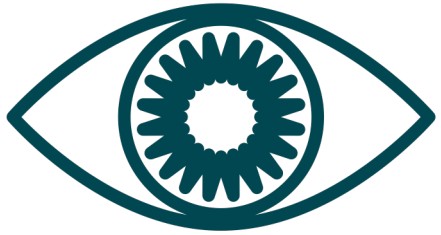
- World-class science from pioneers in gene therapy
- Structure-function biology well-characterized with measurable outcomes amenable to gene augmentation
- Rigorous selection of clinical programs
  - Grounded in natural history studies and patient registries
  - Validation using large animal models
- Single-vector technology for each indication with clear development paths; not discovery-stage
- Faster development path – able to quickly assess efficacy

Proven subretinal delivery with established safety profile and clinical precedent



Structure-Function Dissociation: The Clinical Imperative

Targeting Diseases Where the Structure is Intact



Treat the function to reverse pathology and restore or preserve vision

Retinal structure is relatively preserved even though visual function is already impaired

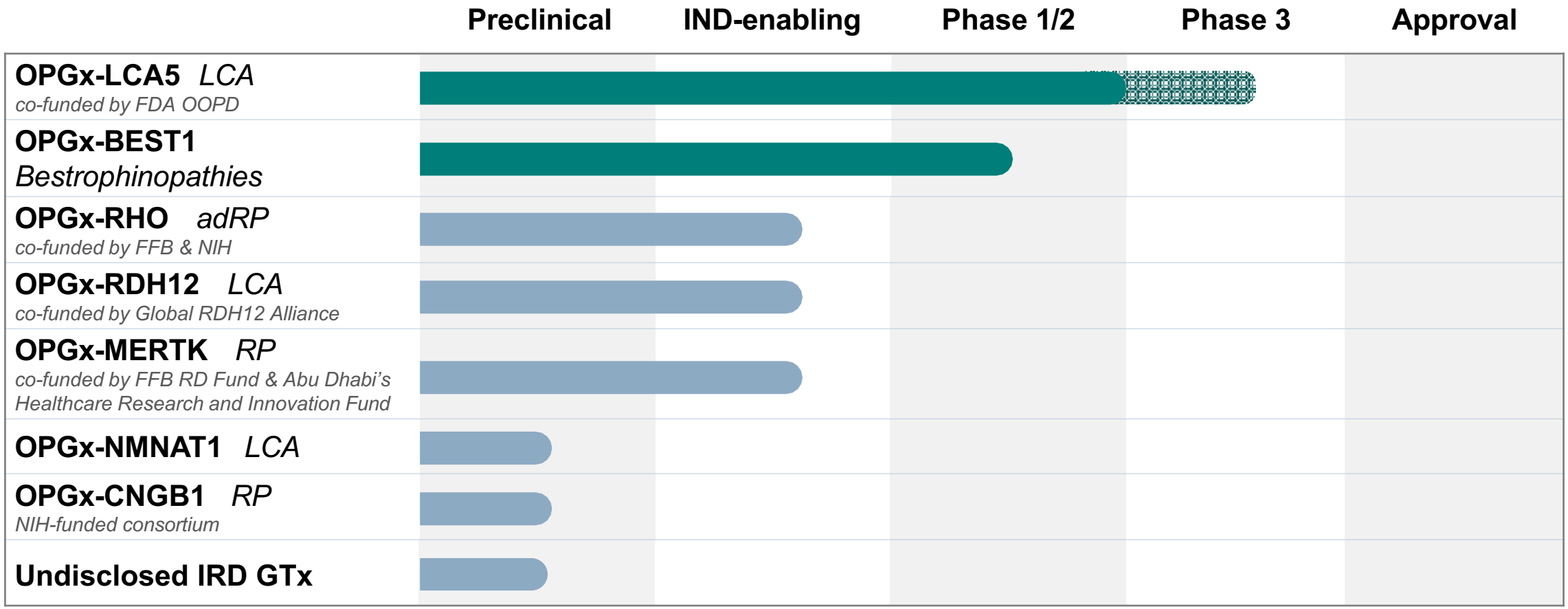
This creates a “therapeutic window” where there are still enough viable cells for AAV gene replacement to restore function

Pick the right patients, and choose meaningful endpoints for our clinical trials

Clinical evidence for curative potential in IRDs

AAV, adeno-associated virus; IRD, inherited retinal disease.

Building a Differentiated Gene Therapy Pipeline



All gene therapy programs have the potential to qualify for a Priority Review Voucher

Opus Genetics owns worldwide rights to all gene therapy programs.

adRP, autosomal dominant retinitis pigmentosa; BEST1, bestrophin 1; CNGB1, cyclic nucleotide-gated channel β1; FDA OOPD, Food and Drug Administration Office of Orphan Products Development; FFB, Foundation Fighting Blindness; LCA5, Leber congenital amaurosis 5; NIH, National Institutes of Health; RD, retinal degeneration; RDH12, retinol dehydrogenase 12; RHO, rhodopsin; RP, retinitis pigmentosa; MERTK, MER proto-oncogene tyrosine kinase; NMNAT1, nicotinamide mononucleotide adenylyltransferase.

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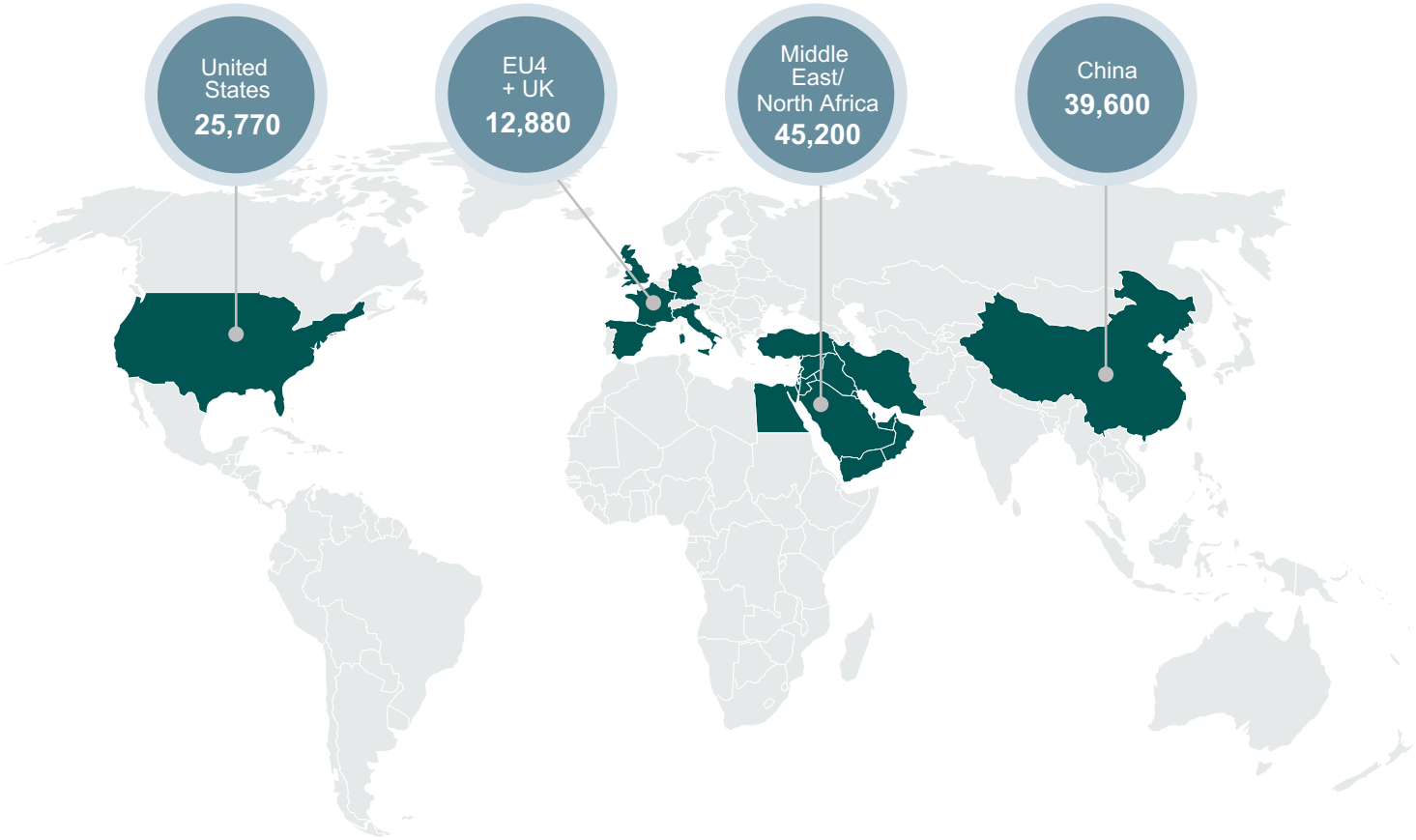
IRD Patient Prevalence Across Select Global Markets Provides Significant Opportunity

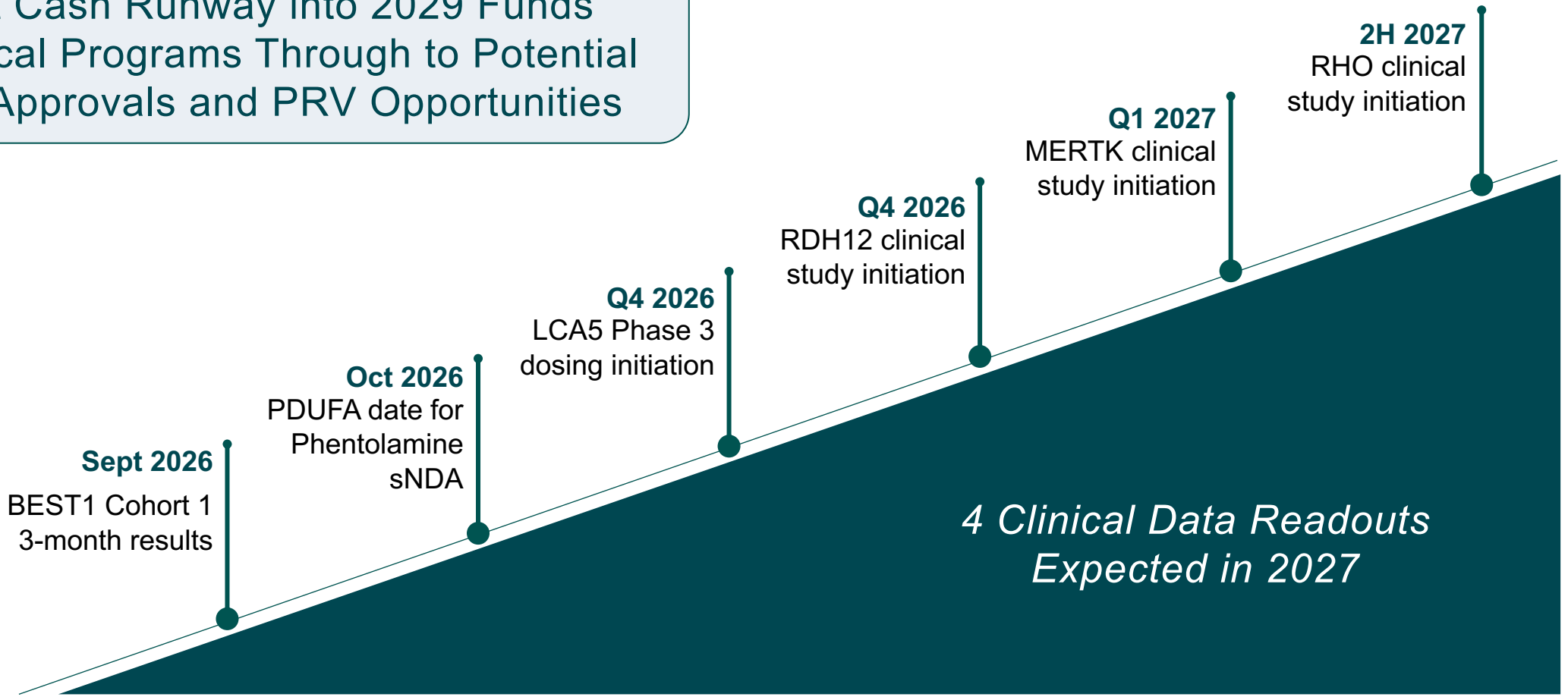


Gene	U.S.	EU4 + UK	Middle East/ North Africa	China	Total Prevalence by Gene
LCA5	~170	~170	~1,400	~1,500	~3,240
BEST1	~8,400	~4,900	~3,600	~4,900	~21,800
RHO	~8,800	~4,600	~2,200	~14,600	~30,200
RDH12	~2,500	~1,000	~17,500	~9,900	~30,900
MERTK	~2,600	~460	~14,300	~4,600	~21,960
NMNAT1	~1,200	~750	~1,100	~2,200	~5,250
CNGB1	~2,100	~1,000	~5,100	~1,900	~10,100
Total Prevalence by Region	**~25,770**	**~12,880**	**~45,200**	**~39,600**	

EU4 = France, Spain, Germany, & Italy. Source: Triangle Insights Group Analysis, February 2026.

Fully-Funded to Support Multiple Clinical Inflection Points

Current Cash Runway into 2029 Funds Five Clinical Programs Through to Potential Product Approvals and PRV Opportunities



Clinical development timelines are based on current estimates and are subject to change; data readouts are targeted for ~9-12 months after study initiation.

Cash balance: ~$90M as of 5/12/26 includes $35M of notes issued related to Oberland Capital financing; Common shares outstanding: 81.4 million as of 05/07/26; Pre-funded warrants:16 million

Phentolamine ophthalmic solution 0.75% is a commercial partnered program; it is FDA-approved for the treatment of pharmacologically-induced mydriasis; a supplemental New Drug Application (sNDA) has been submitted for the treatment of presbyopia. PDUFA, Prescription Drug User Fee Act;

OPGx-BEST1

Juan,
BEST1 patient

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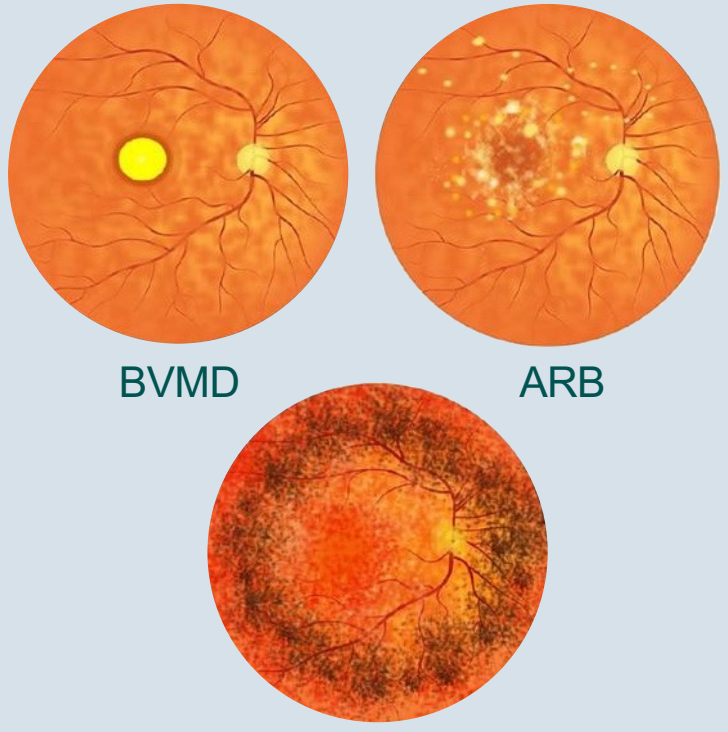
BEST1: Group of IRDs with a Range of Onset and Slow Rate of Progression

Overview & prevalence	• Mutations in *BEST1* have been associated with at least five clinically distinct retinal degenerative diseases, with onset from childhood to adulthood[1] • Accounts for ~3.5% of all IRDs[1] • **Global prevalence*:** ~21,800 patients[2] • **U.S. prevalence:** 8,400 patients (~8,000 BVMD and ~400 ARB)[2]
Clinical features[1,3]	• Serous retinal detachment • BVMD (most common) is characterized by vitelliform (“egg-yolk”) lesion beneath the macula[1] • Macular atrophy • CNV
Symptoms[3,4]	• Loss of central vision • Metamorphopsia (distorted vision) • Scotoma (blind spot) • Photophobia[4]

Most Common BEST1 Phenotypes

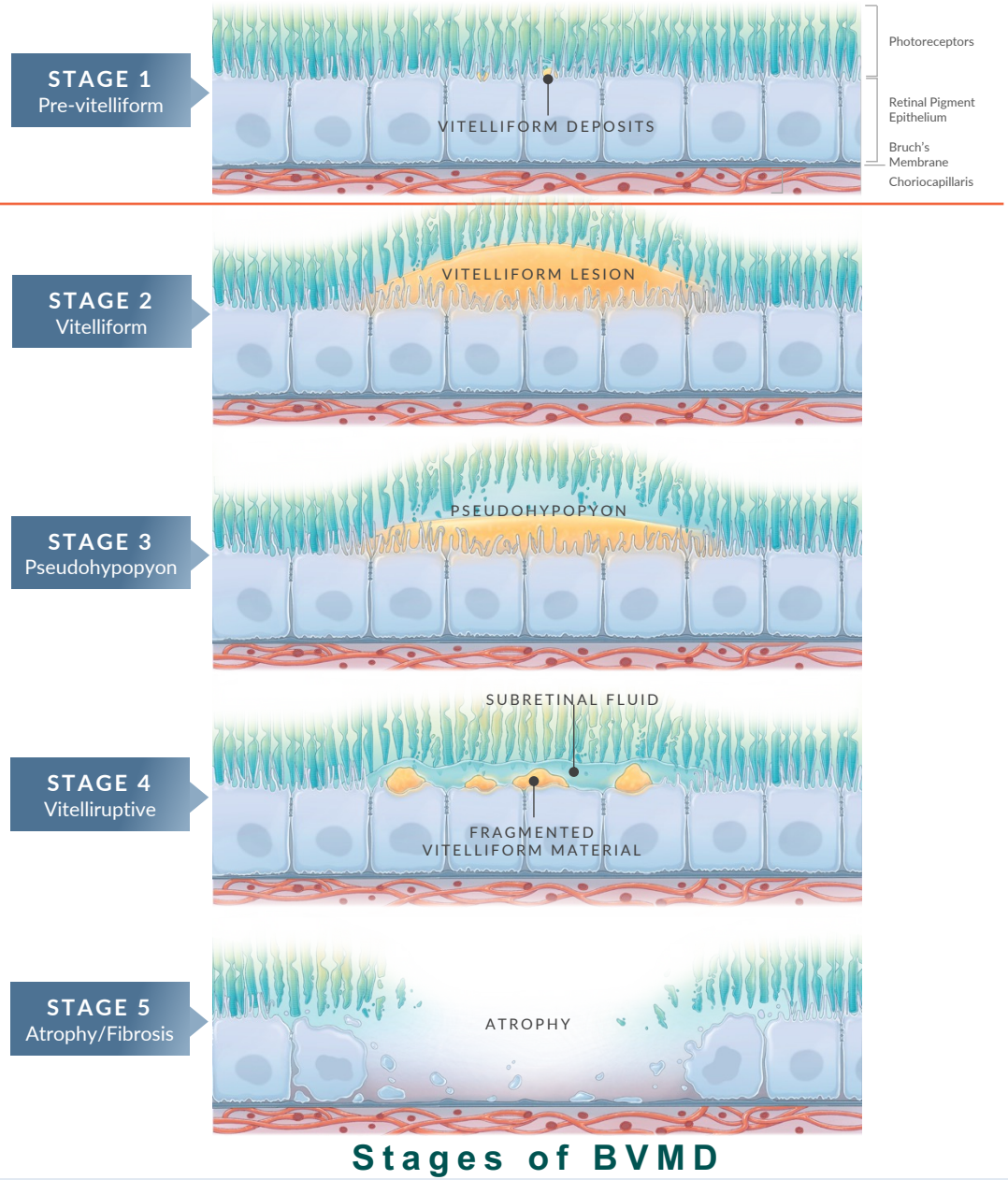


*Global prevalence estimate includes United States, EU4 (France, Spain, Germany, & Italy), UK, Middle East/North Africa, and China.
ADVIRC, autosomal dominant vitreoretinochoroidopathy; ARB, autosomal recessive bestrophinopathy; BEST 1, bestrophin 1; BVMD, best vitelliform macular dystrophy; CNV, choroidal neovascularization; IRD, inherited retinal disease.

1. Amato A, et al. *Saudi J Ophthalmol.* 2023;37(4):287-295. 2. Triangle Insights Group Analysis, February 2026. 3. Johnson AA, et al. *Prog Retin Eye Res.* 2017;58:45-69. 4. Tripathy K, et al. StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024.

BEST1 Disease Biology

- ***BEST1* gene encodes for bestrophin-1**, a homopentameric (i.e. 5 identical monomers) Ca^{2+}-activated chloride channel required for RPE maintenance and retinal physiology
- ***BEST1* mutations disrupt cellular ion and fluid homeostasis** resulting in electrophysiological abnormalities, RPE dysfunction, and retinal degeneration via:
  - **Fluid accumulation** in subretinal space (detachment risk)
  - Defective **clearance of toxic waste** products (eg, lipid deposits)
  - **Impaired RPE-photoreceptor interactions** (eg, defective phagocytosis)



Stages of BVMD

BEST1, bestrophin 1; RPE, retinal pigment epithelium
Guziewicz KE, et al. *Prog Retin Eye Res.* 2017;58:70-88.

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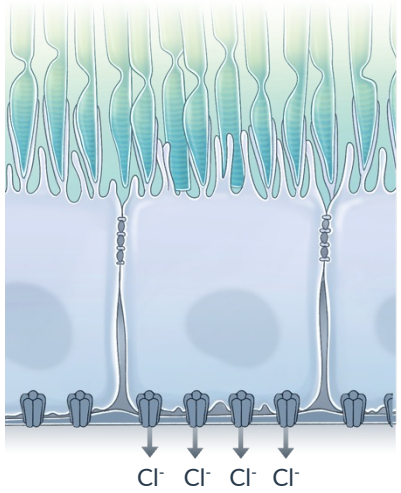
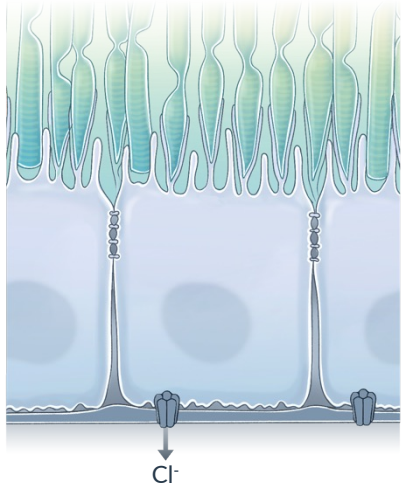
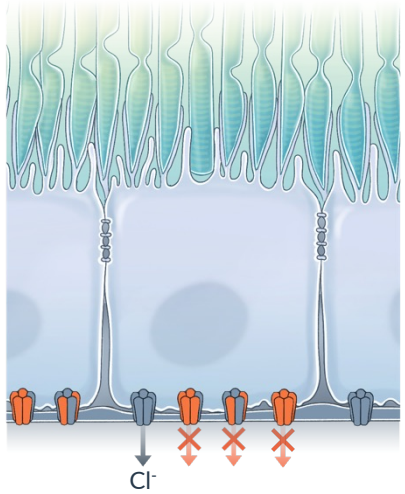
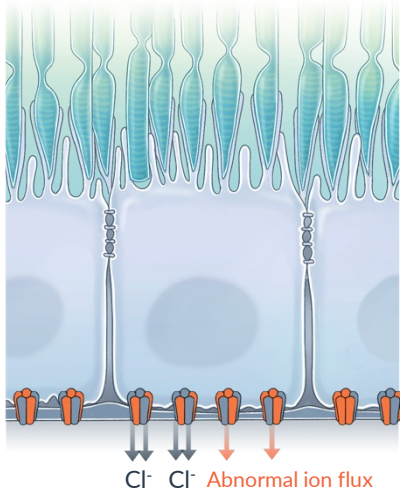
Pathogenic Mechanisms of *BEST1* Variants

NORMAL	RECESSIVE	DOMINANT NEGATIVE	
	LOSS-OF-FUNCTION	LOSS-OF-FUNCTION	GAIN-OF-FUNCTION
			
Normal *BEST1*	↓Reduced protein expression, trafficking, or channel activity E.g., *BEST1* variants in ARB	Mutant protein inhibits normal protein function E.g., *BEST1* variants in BVMD	Mutant protein increases protein activity or introduces new function E.g., *BEST1* variants in ADVIRC (<2% of population, very rare)

Normal monomer

Mutant monomer

BEST1, bestrophin 1 gene; ARB, autosomal recessive bestrophinopathy; BVMD, Best vitelliform macular dystrophy; ADVIRC, autosomal dominant vitreoretinochoroidopathy.
Haldrup SB, et al. *Int J Mol Sci.* 2025;26(19).

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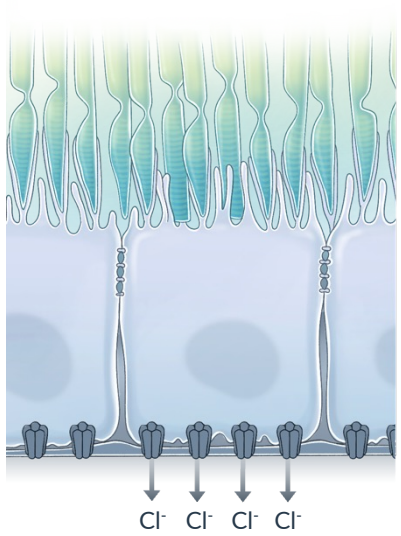
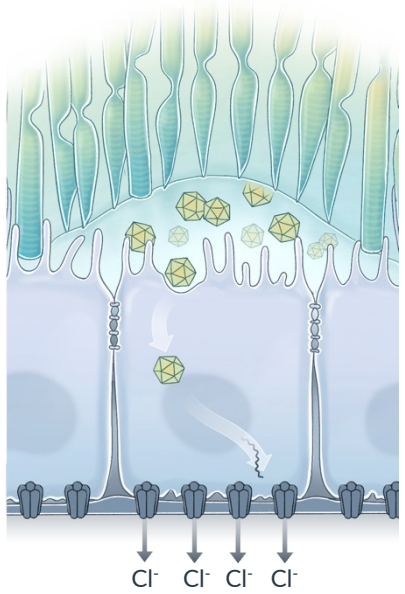
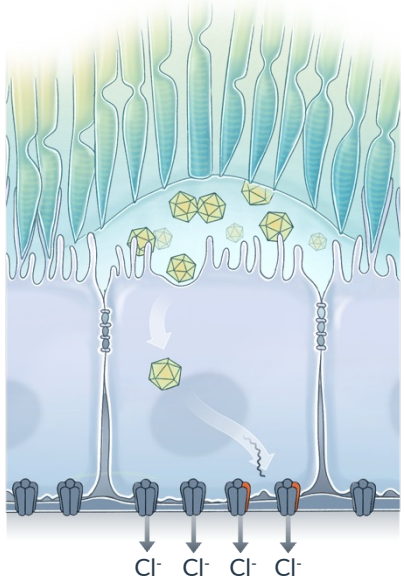
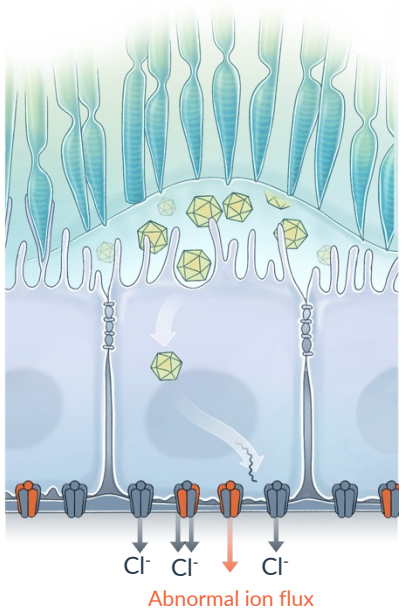
Most *BEST1* Variants May be Amenable to Gene Augmentation

NORMAL	RECESSIVE	DOMINANT NEGATIVE	
	LOSS-OF-FUNCTION	LOSS-OF-FUNCTION	GAIN-OF-FUNCTION
			
Normal *BEST1*	Introduction of functional *BEST1* restores normal function E.g., ARB	Increasing functional *BEST1* expression *may* overcome mutant protein's dominant-negative effect E.g., BVMD	Increasing functional *BEST1* does **not** overcome mutant protein function; **may require gene editing** E.g., ADVIRC

Normal monomer

Mutant monomer

BEST1, bestrophin 1 gene; ARB, autosomal recessive bestrophinopathy; BVMD, Best vitelliform macular dystrophy; ADVIRC, autosomal dominant vitreoretinochoroidopathy.
Haldrup SB, et al. *Int J Mol Sci.* 2025;26(19).

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Paradigm for Guiding Treatment Decisions

Clinical Diagnosis

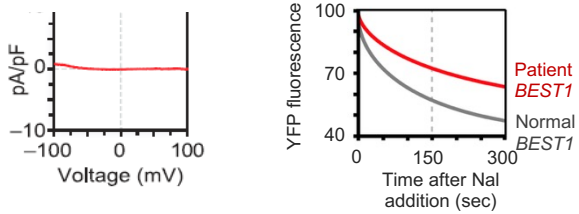
Genetic Testing to Identify *BEST1* Variant

‘Disease-in-a-dish’ Model

In vitro engineering of cell line with patient-specific *BEST1* mutation

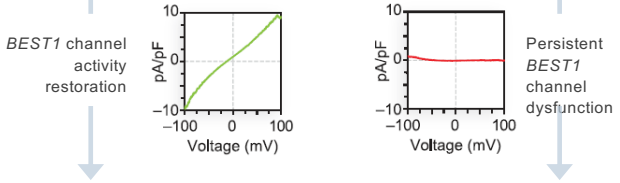
Functional Validation

Measure *BEST1* channel function via two ass



Treatment-Response Assessment*

Gene Augmentation



Clinician Recommendation

Potential responder	Potential non-responder
Eligible for gene augmentation	Not eligible for gene augmentation

*Treatment-response assessment conducted on applicable mutations.
BEST1, bestrophin 1 gene; pA/pF, picoamperes per picofarad; YFP, yellow fluorescent protein; NaI, sodium iodide.
Sinha D, et al. *Am J Hum Genet.* 2020;107(2):278-292.

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OPGx-BEST1 Gene Therapy is Designed to Target RPE and Restore Bestrophin Function

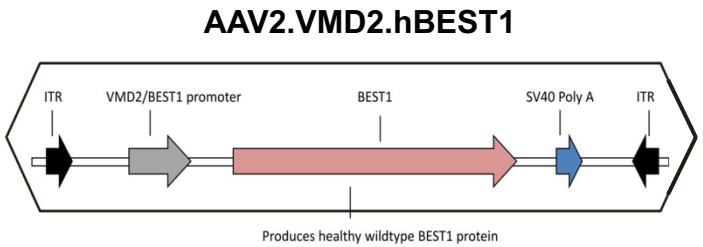
- Designed to restore retinal ion homeostasis in bestrophinopathies, ameliorating retinal structural and functional deficits
- *BEST1* is targeted using the AAV2 capsid employed in LUXTURNA® and an RPE-specific promoter
- Most bestrophinopathies exhibit a slow rate of decline and central photoreceptors usually remain viable for decades, providing a wide therapeutic window
- BIRD-1: Phase 1/2 Study of OPGx-BEST1 Subretinal Gene Therapy in adult (≥18 years old) participants with BVMD or ARB
  - Cohort 1: low dose - 1.5E9 vg/eye
  - Cohort 2: high dose - 4.5E9 vg/eye

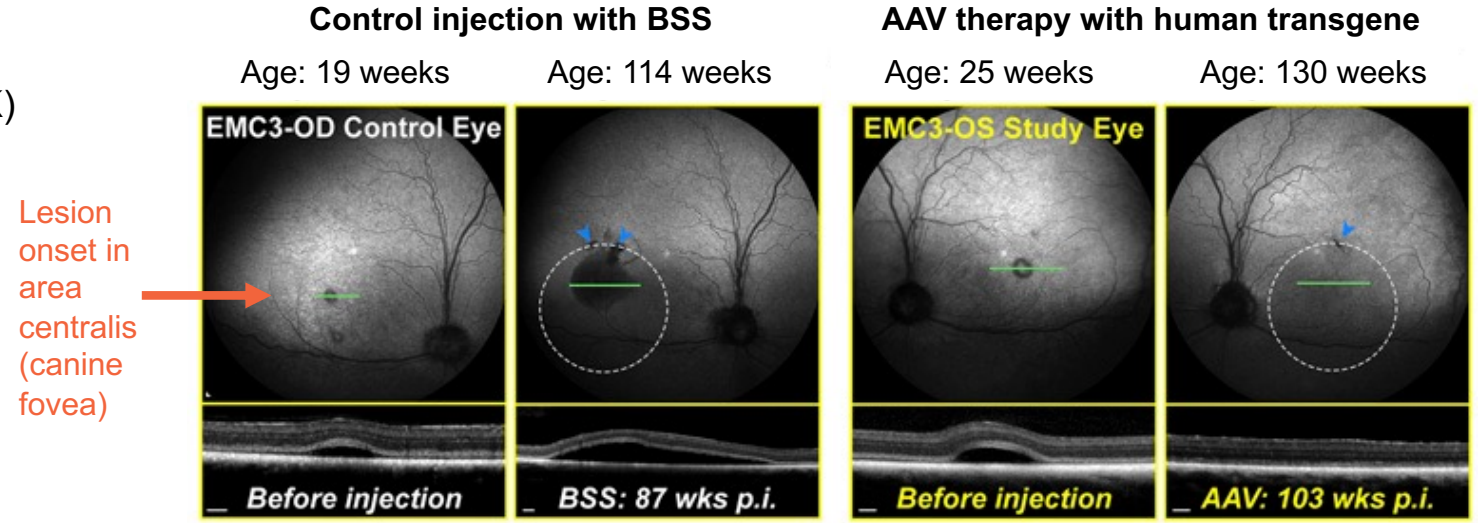
OPGx-BEST1 Gene Therapy	
Vector	AAV2
Delivery	Single subretinal injection

AAV2, adeno-associated virus serotype 2; RPE, retinal pigment epithelium.

Proof of Concept of OPGx-BEST1 AAV2 in a Canine Model of ARB

- Robust restoration of RPE-photoceptor interface demonstrated in canine models of ARB using an AAV2.VMD2.hBEST1 construct
- Treated cBEST1 models exhibit reversal of lesions and retinal microdetachments, which are hallmarks BEST1 disease
- De-risked AAV2 capsid, with AAV2.VMD2 clinical precedent (MERTK) with no known safety issues
- Safety/efficacy studies in cBEST1: Regression of lesions and dose-dependent ERG improvement with favorable safety profile supporting clinical dosing
  - n=9 dogs at 16-108 weeks, low dose of 1.4E9 vg/eye and high dose of 4.5E9 vg/eye





BSS, balanced salt solution; ERG, electroretinogram; MERTK, MER proto-oncogene, tyrosine kinase; RPE, retinal pigment epithelium. Guziewicz, et al. *PNAS.* 2018;115:E2839–E2848.

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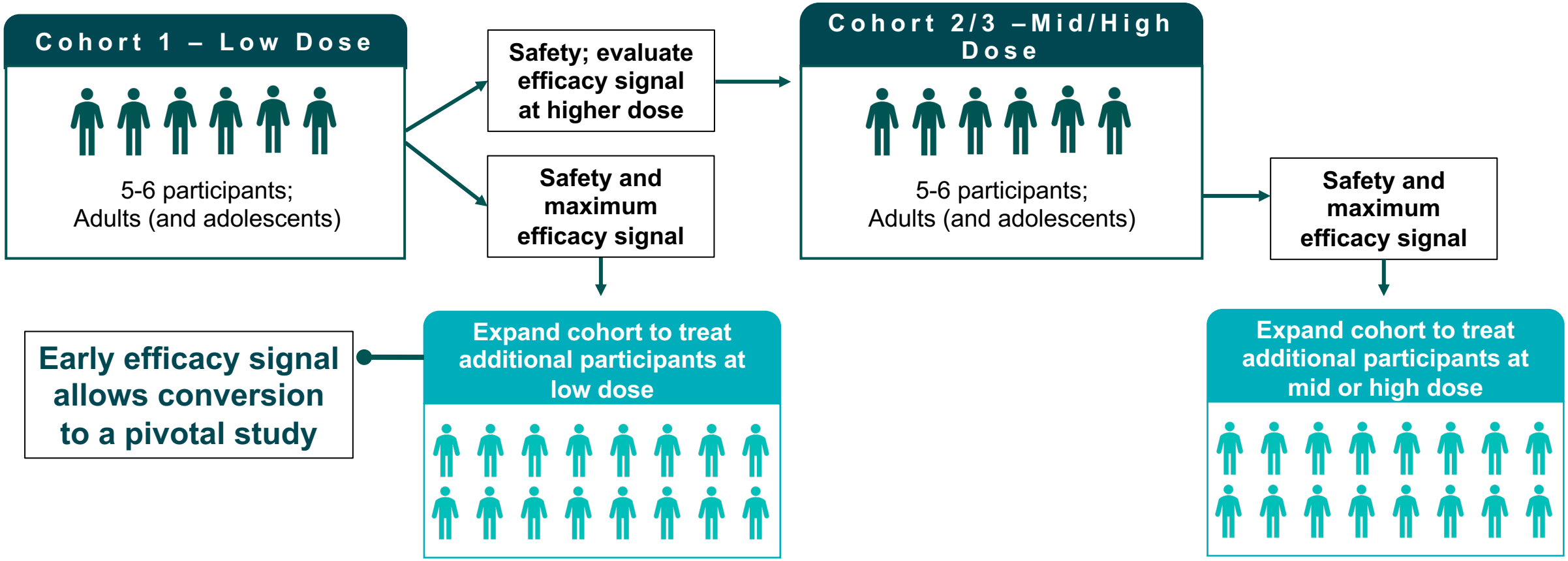
Clinical Studies Follow a Dose Exploration, Data-Driven Approach



Representative study design.

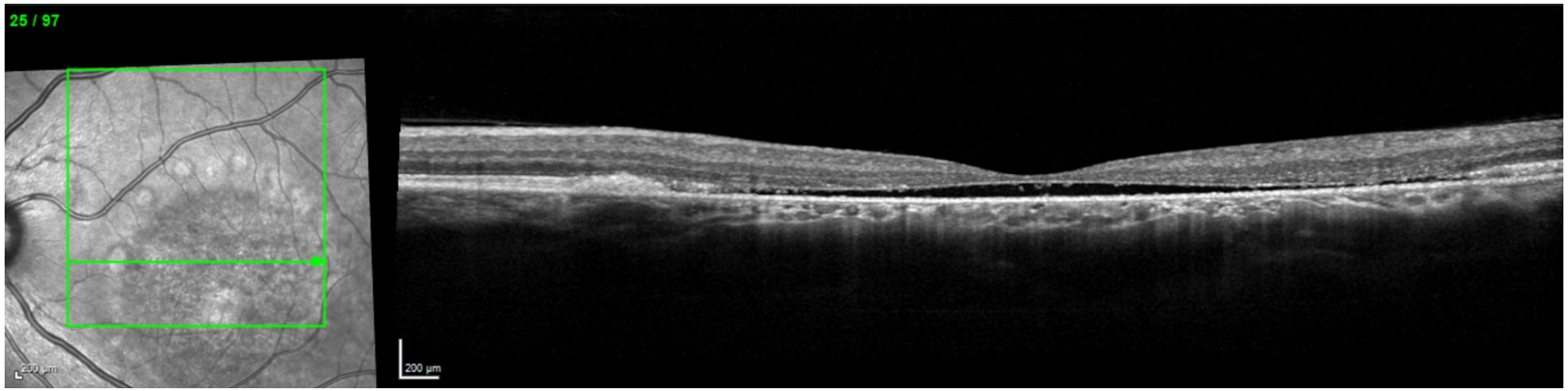
Baseline Participant Demographics

Participant #	101-101	101-104	102-101	102-102	101-106
Age	63	59	50	45	31
Sex	Female	Female	Male	Male	Male
BEST phenotype/mutation	ARB	ARB	BVMD	BVMD	BVMD
Study (treated) eye*	Left (OS)	Right (OD)	Left (OS)	Left (OS)	Left (OS)
Baseline VA (study/treated eye)	1.68	0.70	0.72	0.49	0.77
Baseline VA (fellow eye)	0.84	0.41	0.34	0.27	0.58

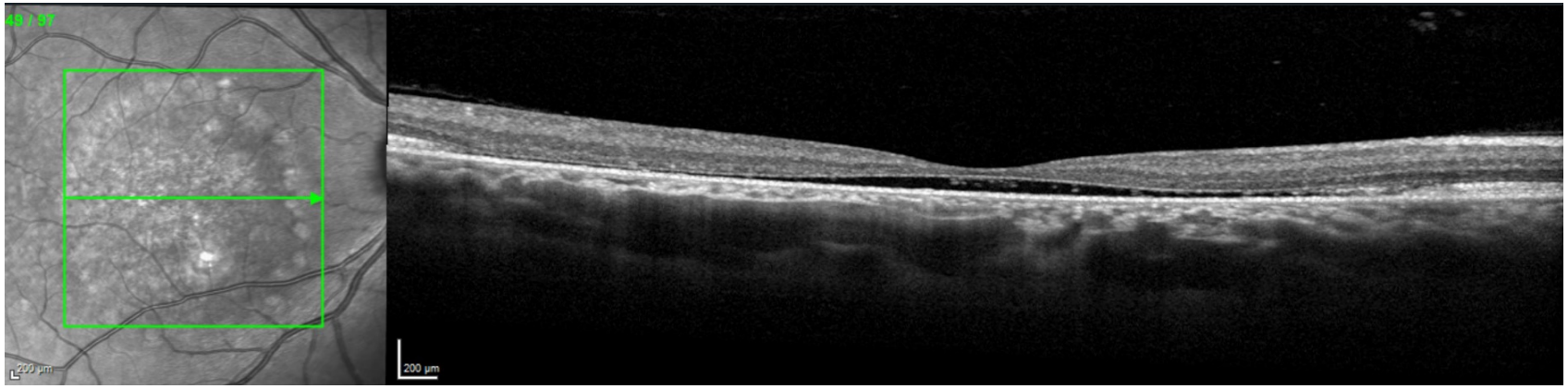
*Worse eye deemed study (treated) eye.
ARB, autosomal recessive bestrinopathy; BVMD, best vitelliform macular dystrophy; OD, right eye; OS, left eye; VA, visual acuity.

Baseline OCT Example: 101-106 (BVMD)

Study Eye (Treated)

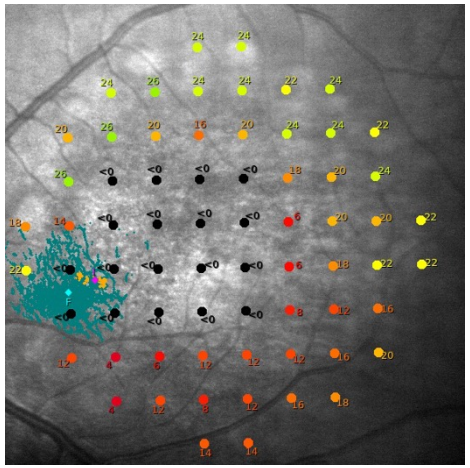
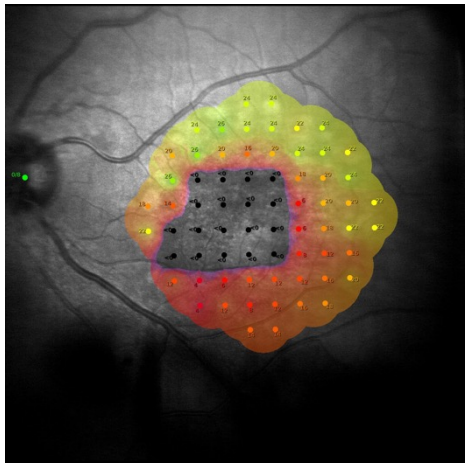
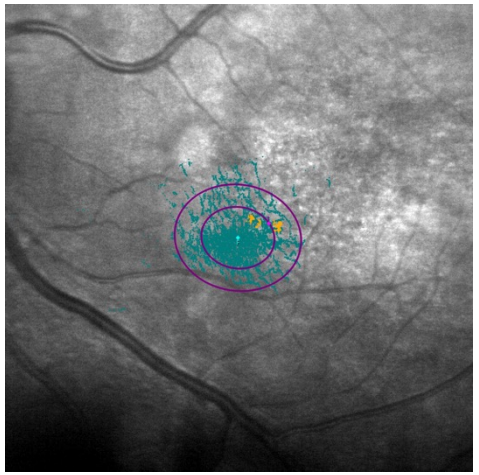
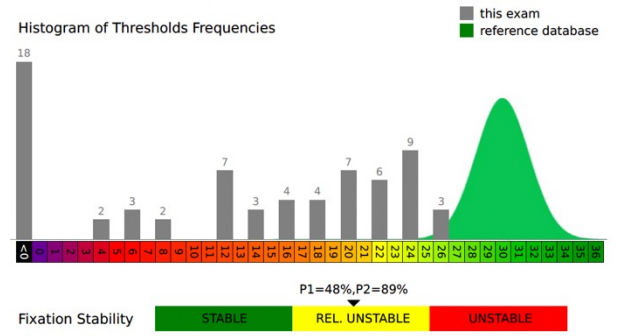
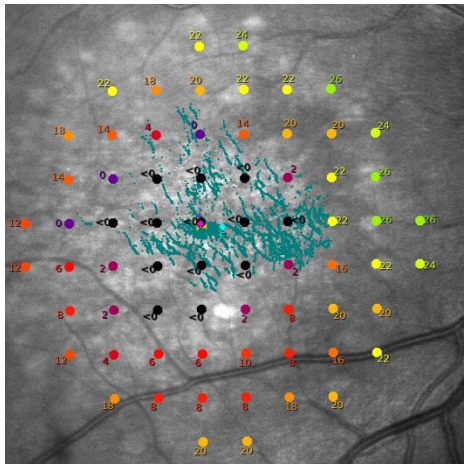
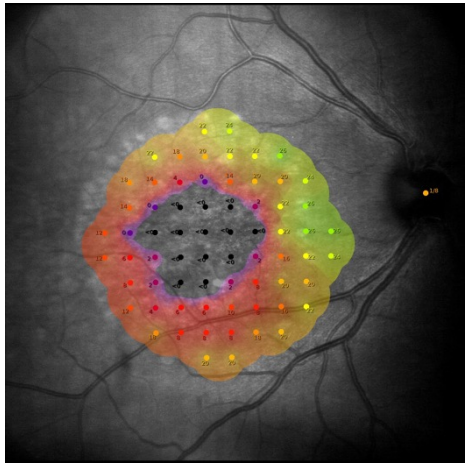
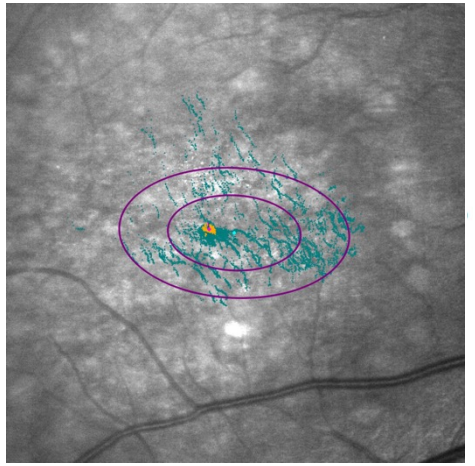
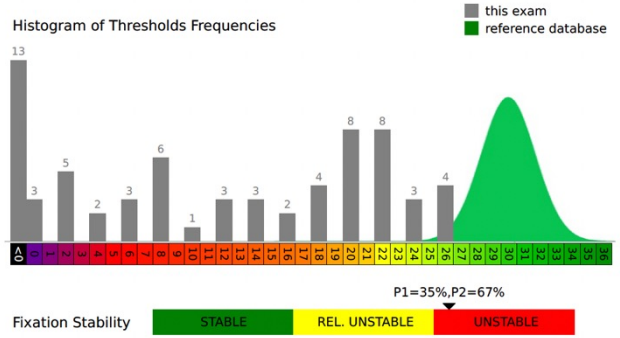


Fellow Eye (Untreated)



BVMD, best vitelliform macular dystrophy; OCT, optical coherence tomography.

Baseline Microperimetry Example: 101-106 (BVMD)

		Sensitivity Map	Fixation Plot	Average Threshold
Study Eye (Treated)				
Fellow Eye (Untreated)				

BVMD, best vitelliform macular dystrophy.

OPGx-BEST1 was Well Tolerated in Sentinel Participant (101-101) at 3 Months

Demographics

Age	63
Sex	Female
Diagnosis year	2015
BEST phenotype	ARB
Study (treated) eye	OS
VA at Baseline (OS)	LogMAR 1.66 (CF)
CST at Baseline (OS)	Atrophic macula
Follow-up duration	3 months (to date)

No ocular inflammation, treatment-related adverse events, or dose-limiting toxicities

Day 14

- Few pigmented cells in vitreous
- No AC inflammation
- Steroid taper initiated

Postop Month #1

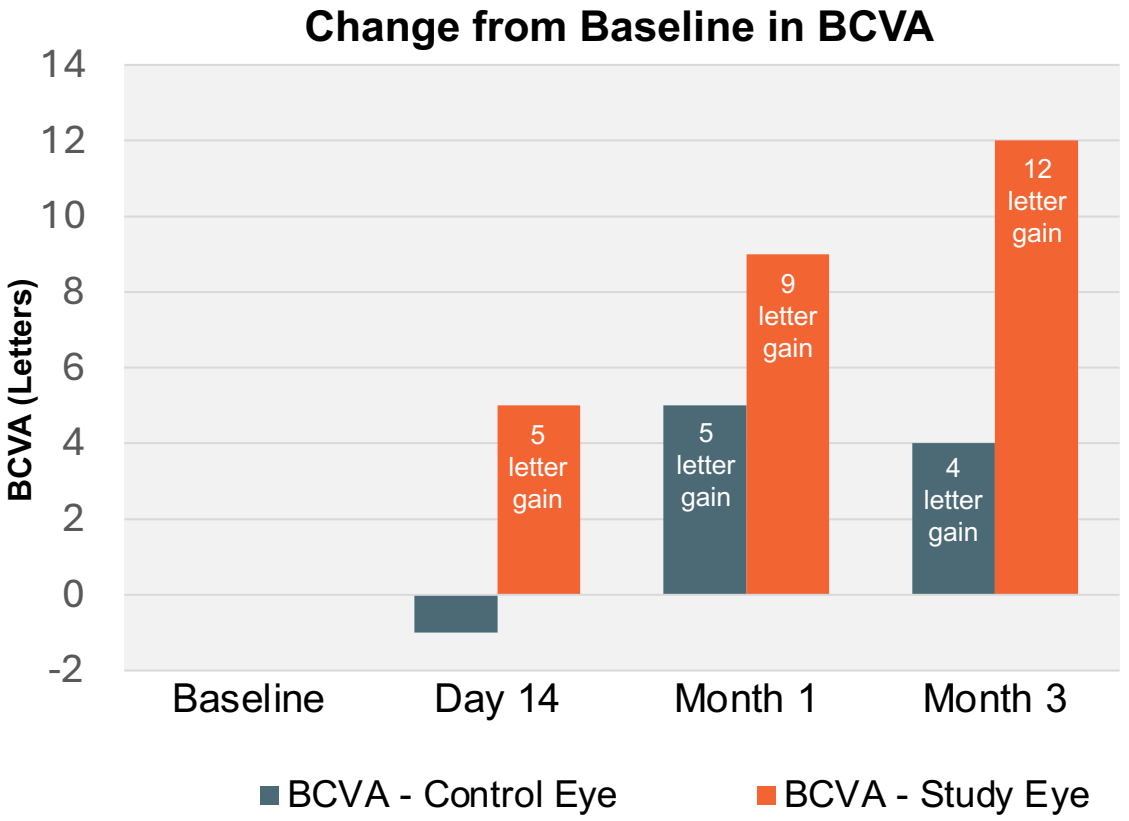
- No ocular inflammation
- No ocular AEs
- No treatment-related AEs or DLT

Postop Month #3

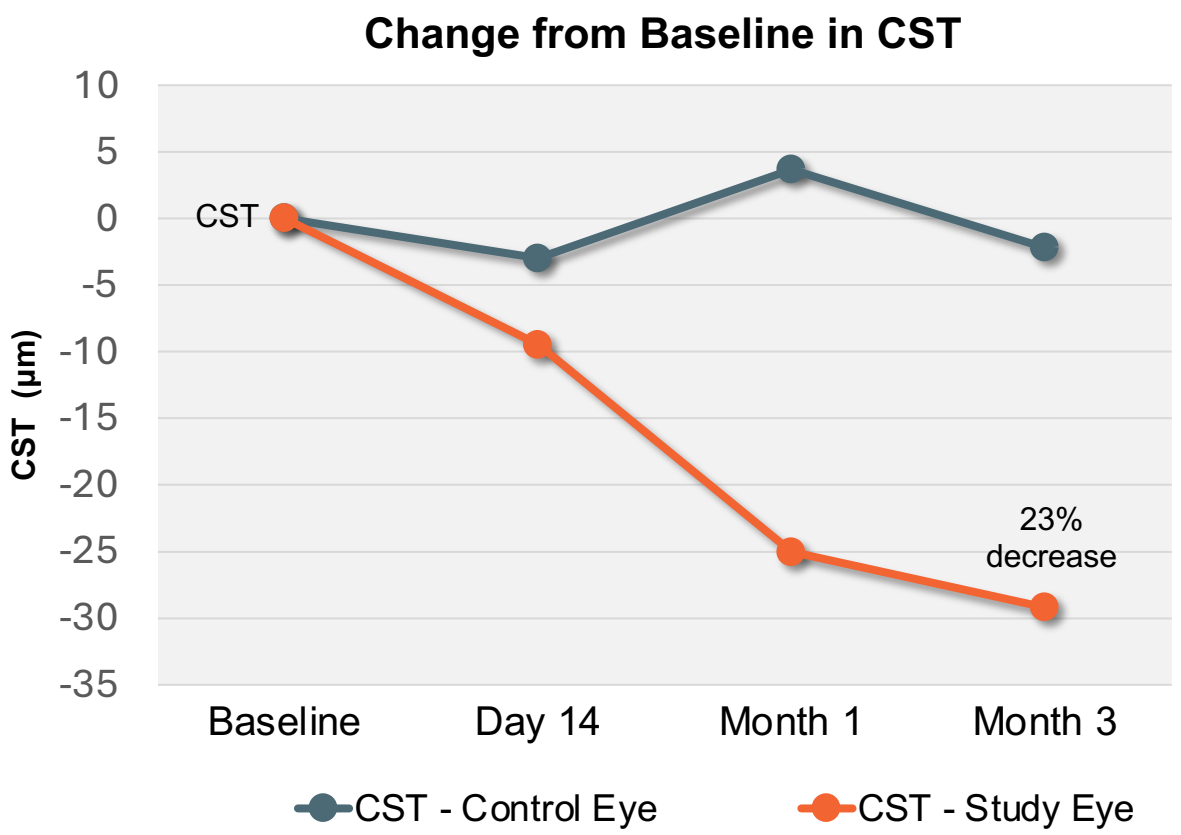
- No ocular inflammation
- No ocular adverse events
- Steroid taper complete

Worse eye deemed study (treated) eye. ARB, autosomal recessive bestrinopathy; BL, baseline; CF, counting fingers; OCT, optical coherence tomography; OS, left eye; VA, visual acuity. AC, anterior chamber; AE, adverse event; DLT, drug limiting toxicities.

BCVA and CST in the Treated Eye Improved Over 3 Months (101-101)



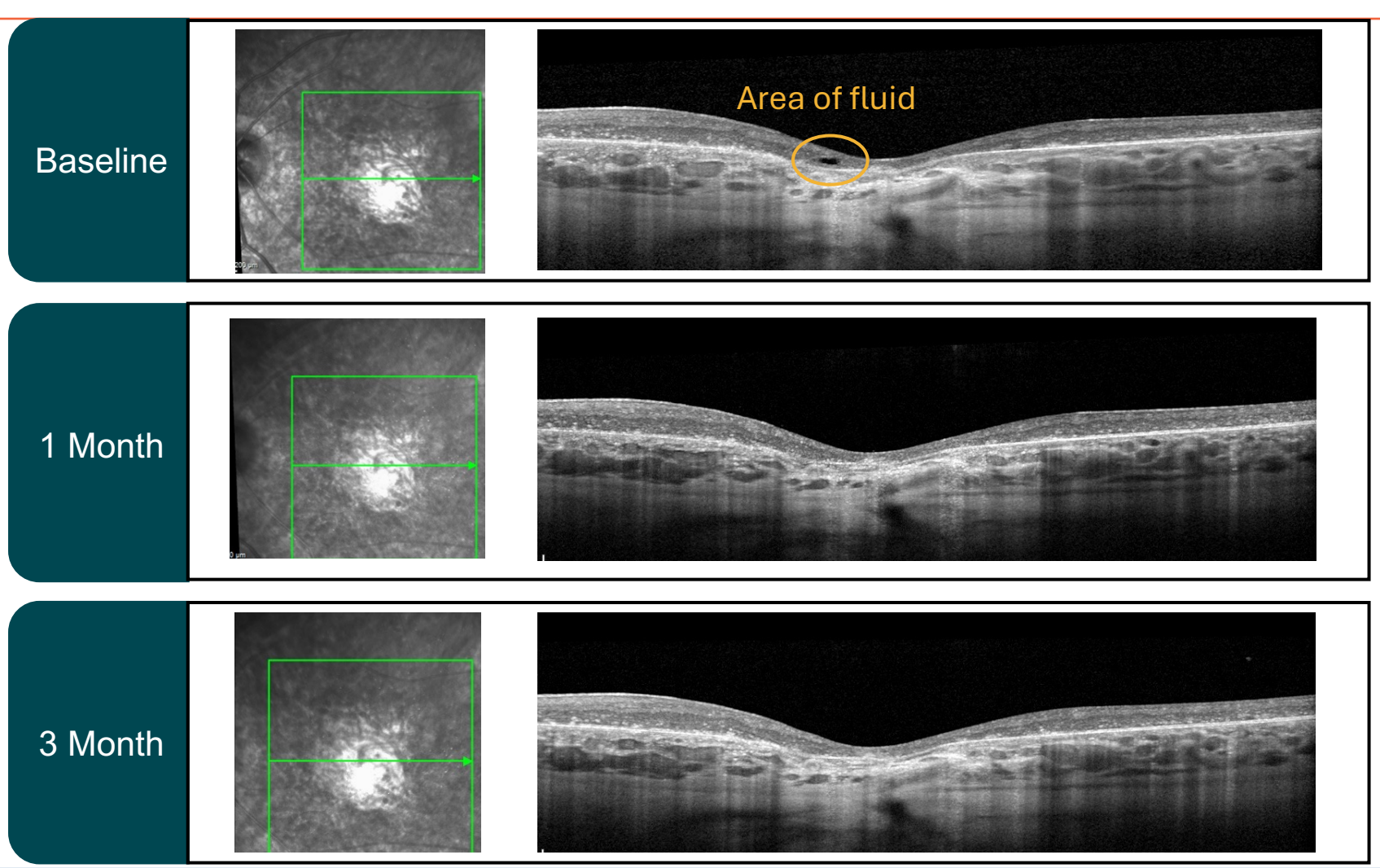
Early signal of functional improvement (12 letter gain) observed in the study eye; **Participant commented that their vision was no longer "darkening"**



Structural improvement (23% decrease) observed in the study eye

BCVA: equivalent letters. BCVA, best-corrected visual acuity; CST, central subfield thickness.

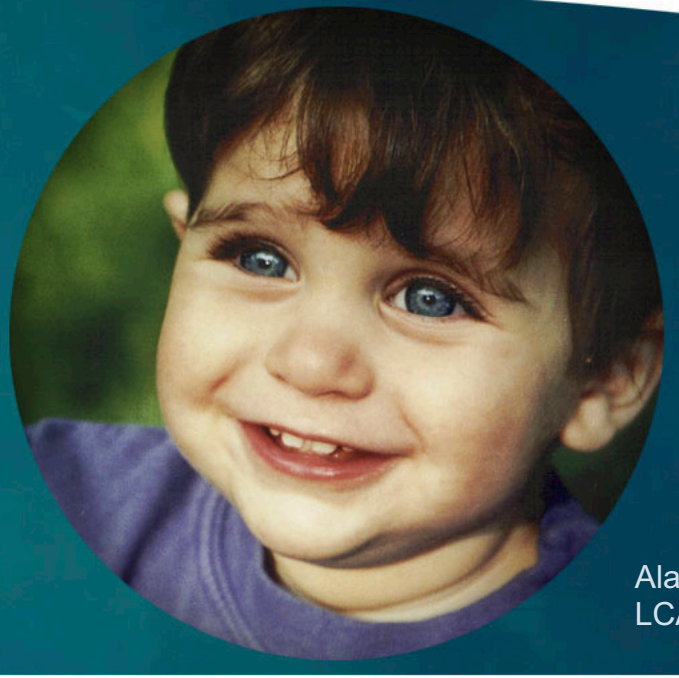
Treated Area Improved Over 3 Months (101-101)



Reduction of intraretinal fluid as early as 1 month in areas with less atrophy

OCT, optical coherence tomography; OS, left eye.

OPGx-LCA5



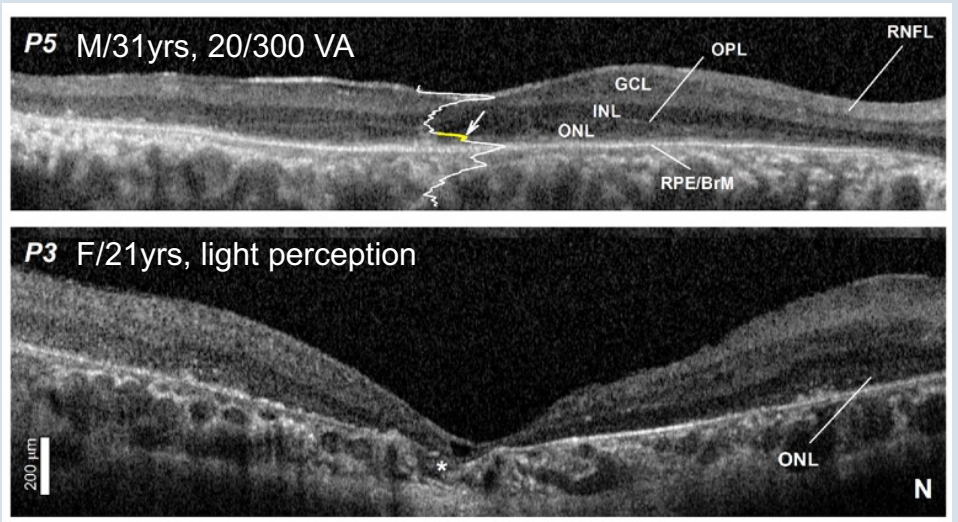
Alan,
LCA5 patient

OPUS GENETICS

LCA5: Early-onset, Severe IRD with Early Vision Loss

Overview & prevalence	• Very early onset severe disease typically presenting in infancy or early childhood[1] • *LCA5* represents ~2% of all LCA cases[1] • **Global prevalence*:** ~3,240 patients[2] • **U.S. prevalence:** ~170 patients[2]
Clinical features[1,3]	• Pigmentary retinopathy • Macular atrophy • Photoreceptor structure often preserved
Symptoms[1,3]	• Vision loss (typically starting in infancy); often limited to hand motions or light perception • Nystagmus • Hyperopia • Severe and early photoreceptor loss results in severely abnormal or non-detectable visual fields

LCA5 patients exhibit preserved photoreceptors in the central retina in adulthood despite disease severity and early onset



*Global prevalence estimate includes United States, EU4 (France, Spain, Germany, & Italy), UK, Middle East/North Africa, and China.
LCA, Leber congenital amaurosis; VA, visual acuity.

1. Boldt K, et al. *J Clin Invest.* 2011;121(6):2169-2180. **2.** Triangle Insights Group Analysis, February 2026. **3.** Uyhazi KE, et al. *Invest Ophthalmol Vis Sci.* 2020;61:30.

OPUS GENETICS

OPGx-LCA5 Gene Therapy is Designed to Restore a Key Protein of the Visual Cycle

- Lebercilin is a ciliary protein critical for the function of photoreceptor inner and outer segments[1]
- In *LCA5* patients, photoreceptor function is severely impaired due to a lack of functioning lebercilin[1]
  - However, photoreceptors can survive through the third decade of life, suggestive of a broad window for therapeutic intervention[2]
- **OPGx-LCA5 is designed to address mutations in the *LCA5* gene, which encodes for the lebercilin protein**
  - Clinically derisked AAV8 vector delivers a functional *LCA5* gene directly to photoreceptor cells, using same promoter technology as LUXTURNA®

OPGx-LCA5 Gene Therapy	
Vector	AAV8
Delivery	Single subretinal injection

AAV8, adeno-associated virus serotype 8; LCA5, Leber congenital amaurosis 5.

1. Uyhazi KE, et al. *Invest Ophthalmol Vis Sci.* 2020;61:30. **2.** Song JY, et al. *Mol Ther.* 2018;26:1581-1593.

OPUS GENETICS

OPGx-LCA5 Phase 1/2 Trial Participant Demographics

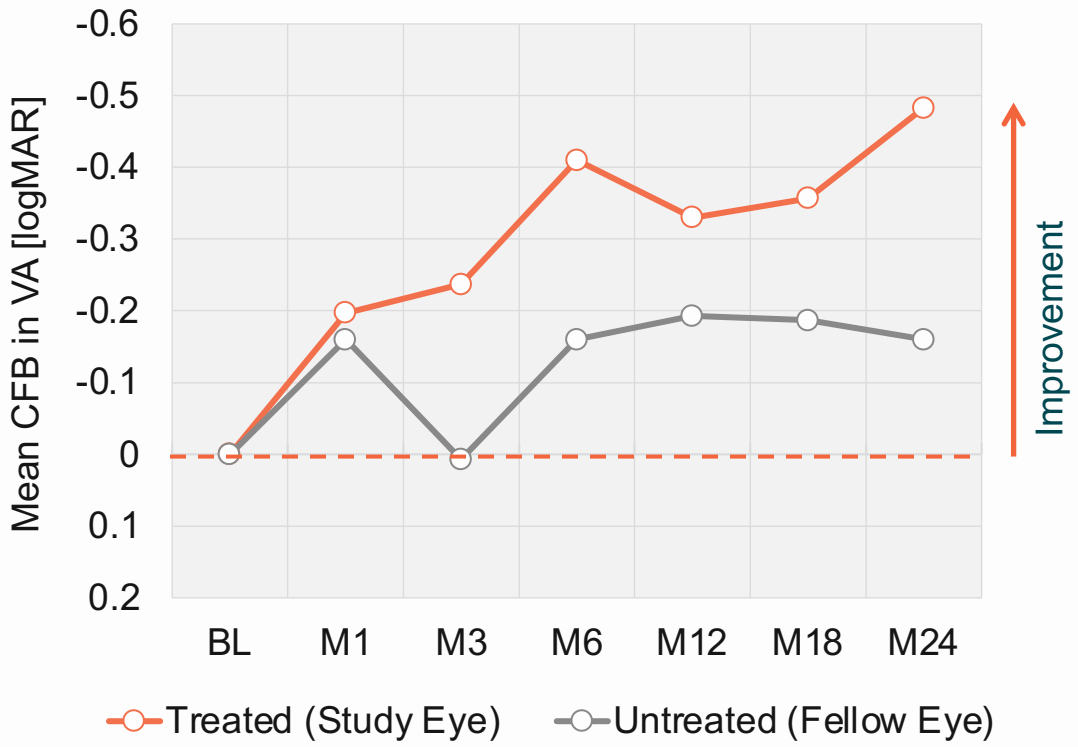
	Adult Participants			Pediatric Participants		
Participant #	**01-01**	**01-03**	**01-04**	**01-05**	**01-06**	**01-07**
Age	34	26	19	17	16	17
Gender	Female	Male	Female	Female	Male	Female
Study eye treated	Left (OS)	Left (OS)	Right (OD)	Right (OD)	Right (OD)	Right (OD)
Baseline visual acuity logMAR	1.38	2.90	0.96	2.2	0.96	2.3
Follow-up duration	18 mo.	18 mo.	18 mo.	3 mo.	3 mo.	3 mo.

The participant's eye with the worst vision was treated in all cases.

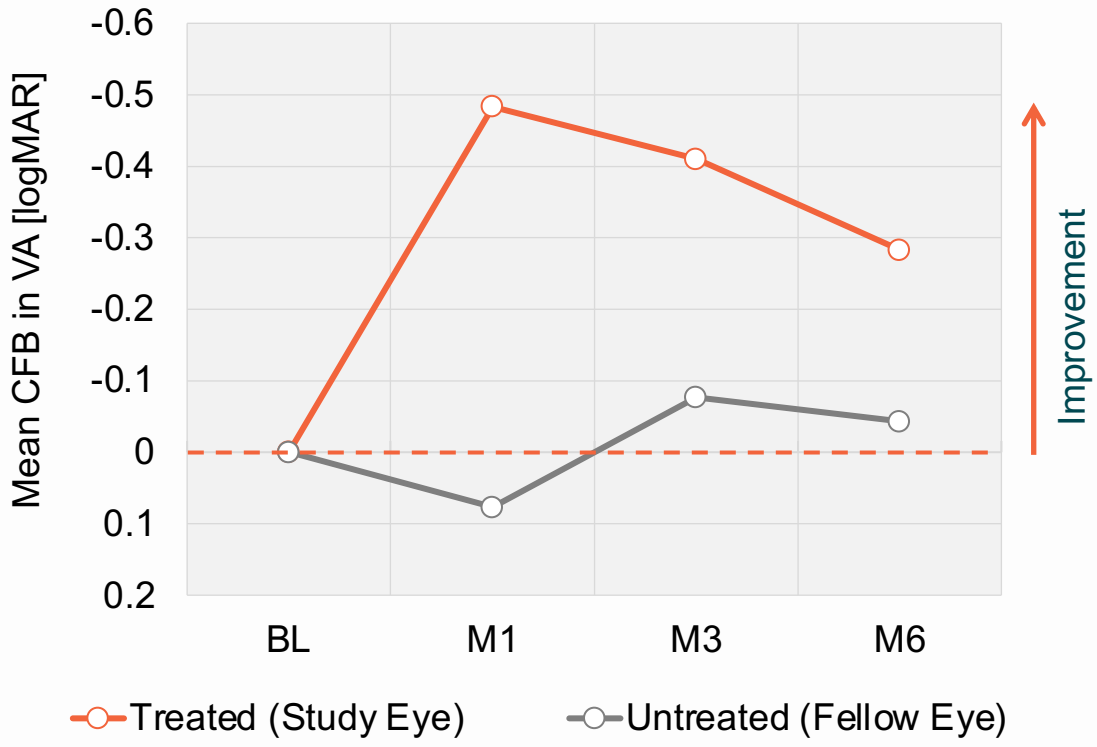
Deemed study eye: OD, right eye; OS, left eye.

Visual Acuity Maintained in Adults Over 24 Months and Improved Over 6 Months in Pediatric Cohort

Mean Change from Baseline in VA Adult Cohort (N=3)



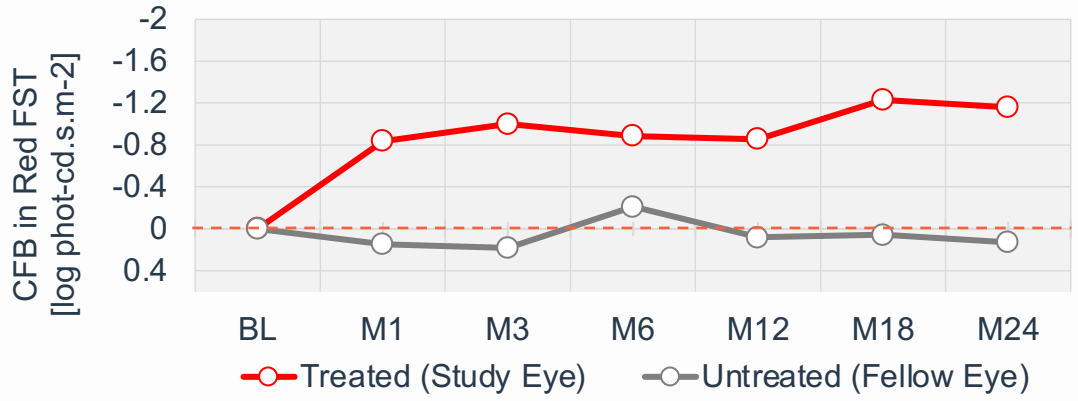
Mean Change from Baseline in VA Pediatric Cohort (N=3)

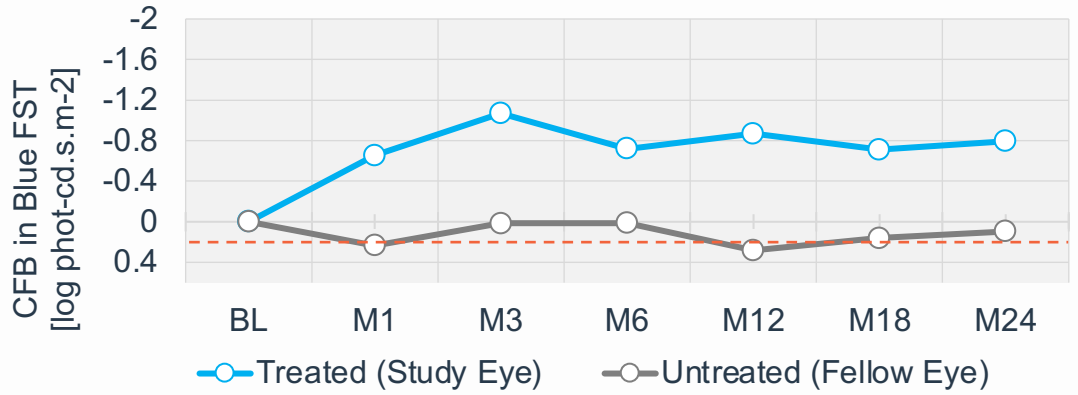


BL, baseline; CFB, change from baseline; M, month; VA, visual acuity.

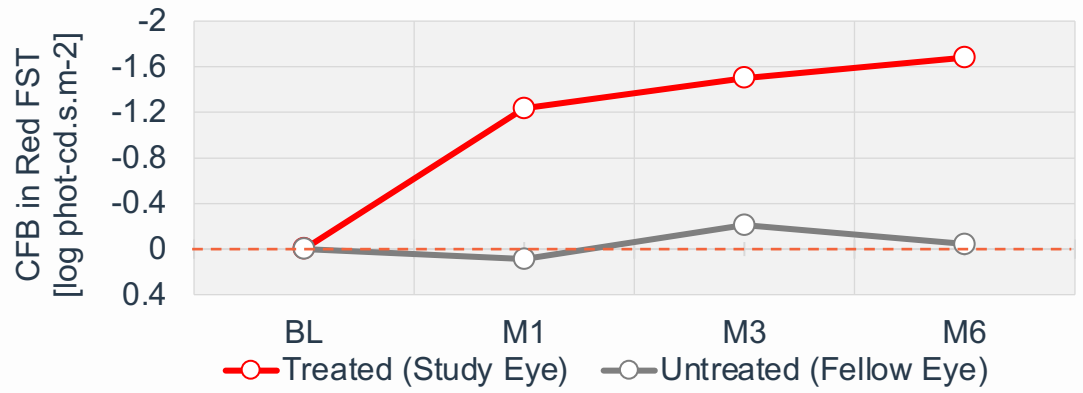
Full-Field Stimulus Test Demonstrated Durable Vision Improvement

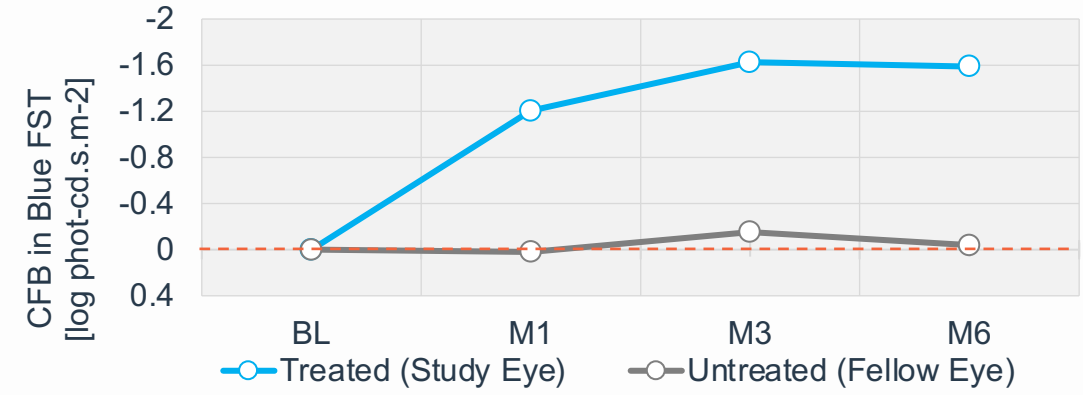
Mean CFB in Red and Blue FST Adult Cohort (N=3)





Mean CFB in Red and Blue FST Pediatric Cohort (N=3)

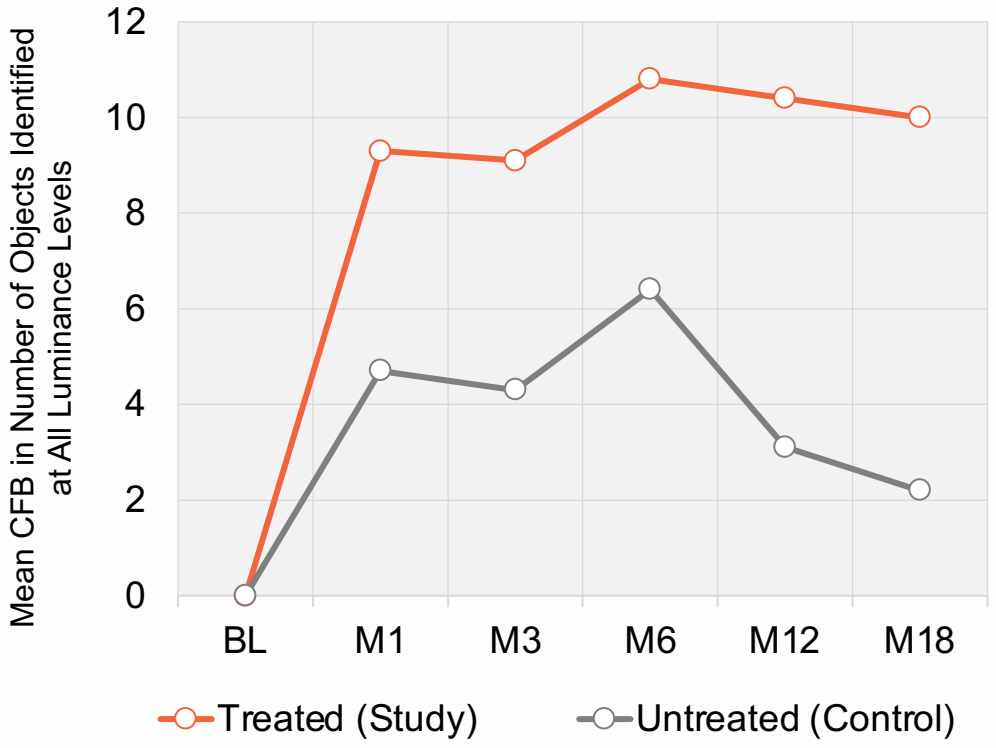




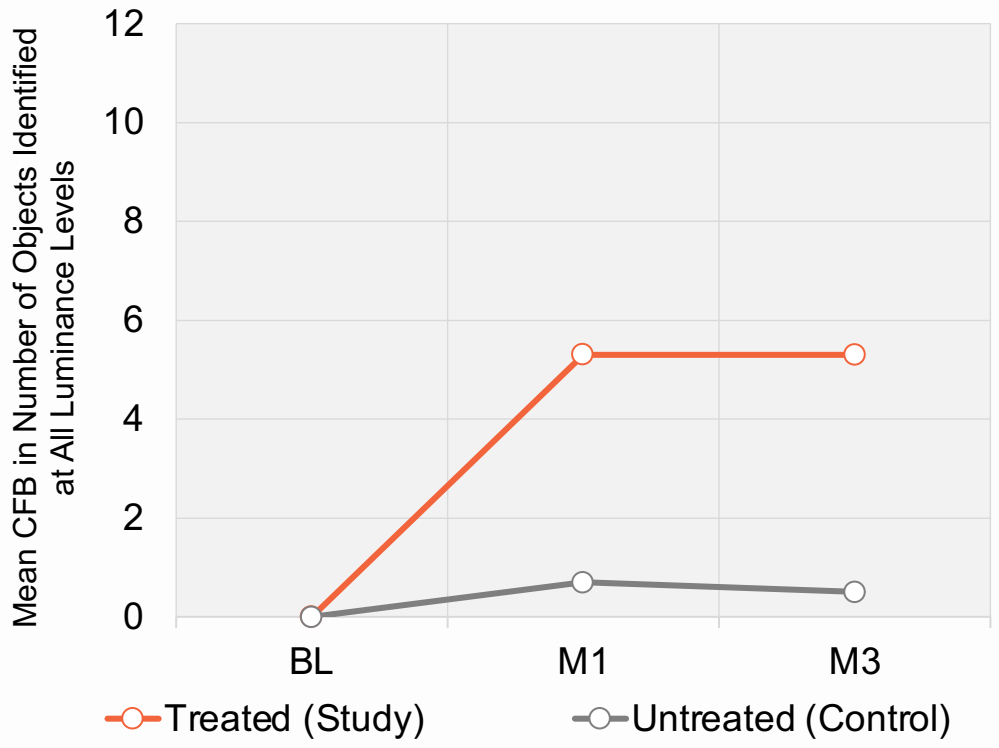
BL, baseline; CFB, change from baseline; FST, Full-field stimulus test; M, month.

Treated Eyes Identified More Objects on the Virtual Reality Mobility Test

Mean Change from Baseline in MLoMT Adult Cohort (N=3)

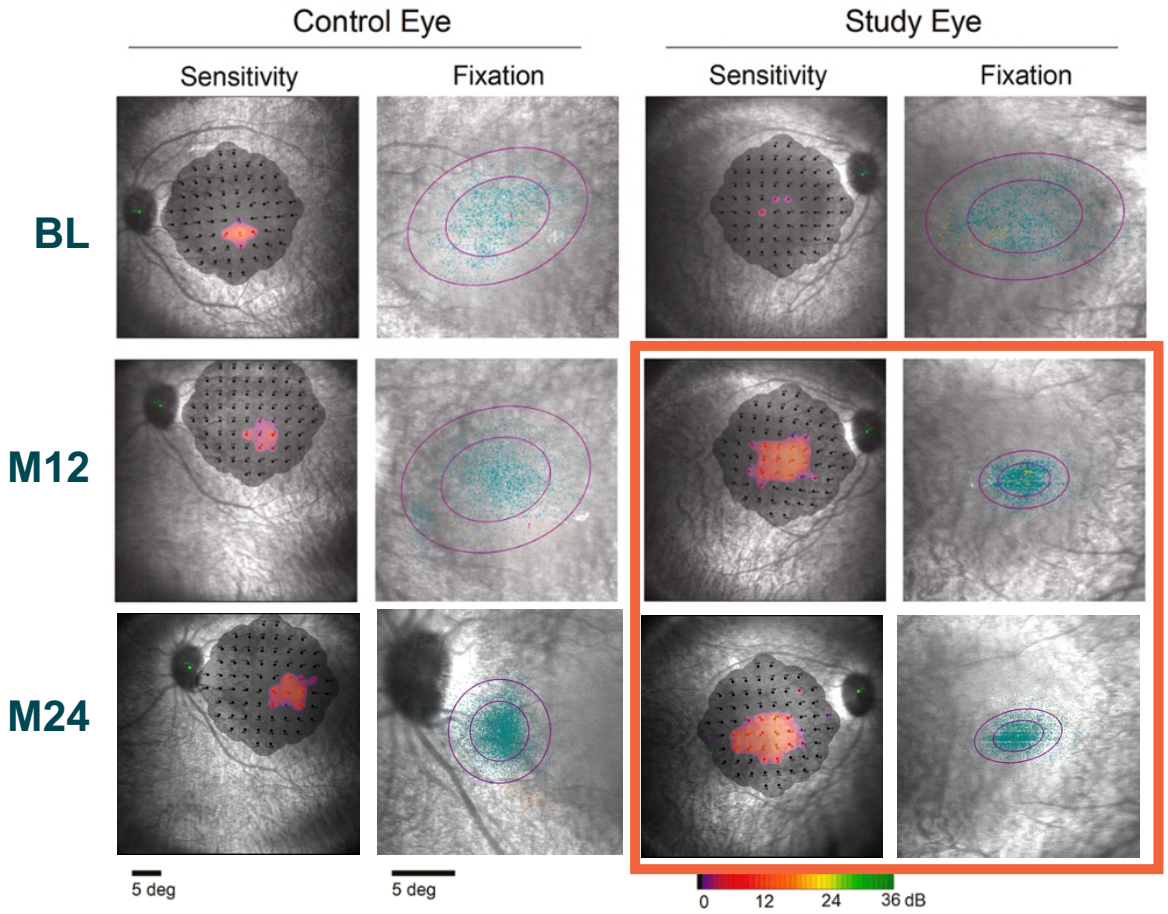


Mean Change from Baseline in MLoMT Pediatric Cohort (N=3)

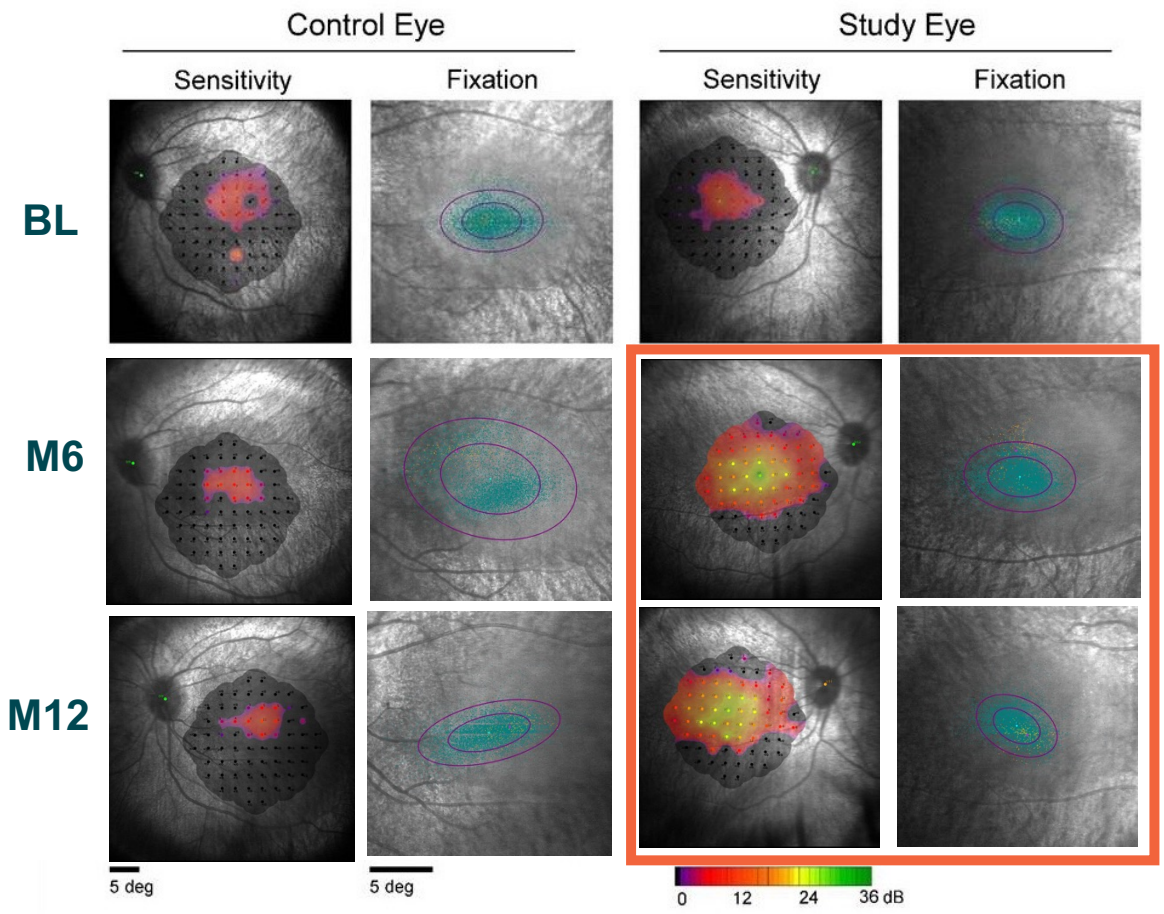


BL, baseline; CFB, change from baseline; M, month; MLoMT, Multi-Luminance orientation and Mobility Test; VA, visual acuity; VR, virtual reality.

Microperimetry Data Provides Evidence of Increased Sensitivity and Movement of Fixation Toward the Fovea



Adult Participant 01-04



Pediatric Participant 01-06

Microperimetry is a detailed, eye-tracking-assisted visual field test that creates a retinal sensitivity map of the macula by testing a patient's response to light at specific points.
BL, baseline; M, month.

OPGx-LCA5 Program Positioned for Rapid Advancement

FDA Alignment in Type B RDEP Meeting on Phase 3 Trial Design:

- Expected to enroll 8 participants able to complete microperimetry testing with both eyes treated.
- Six-month run-in period; participants serve as their own natural history control prior to treatment.
- Primary efficacy endpoint is a mean improvement of at least 7 decibels (dB) in retinal sensitivity across the central 16 test loci, a clinically meaningful measure of visual function.
- FDA indicated that Opus may submit a Biologics License Application (BLA) based on compelling efficacy at the 6-month primary endpoint, with 12-month durability data submitted during the BLA review process.

Phase 3 enrollment ongoing: 7 of the 8 planned participants have been enrolled and are currently completing the run-in period. Expect to initiate dosing in Q4 2026.

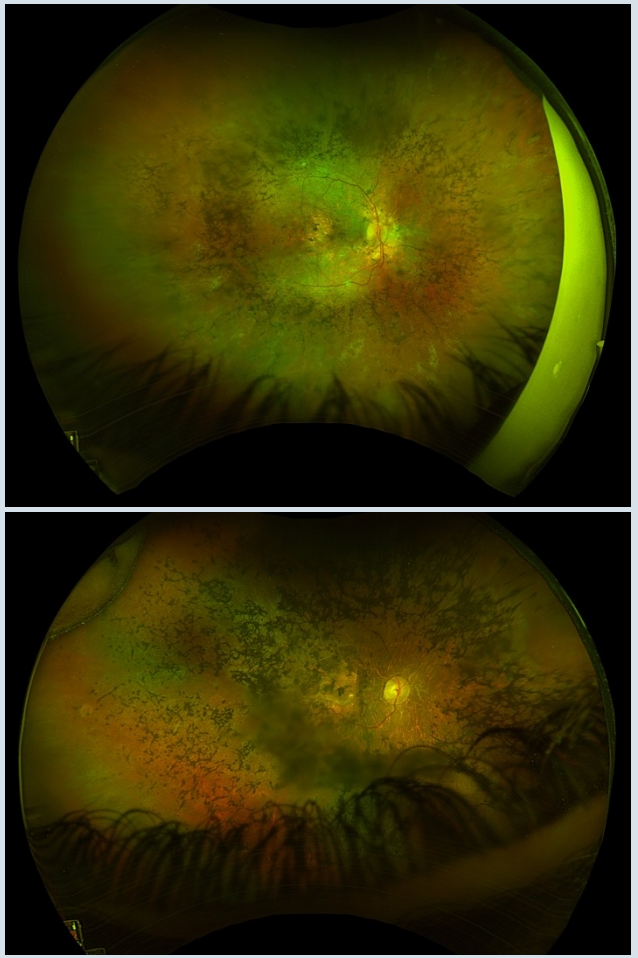
FDA Rare Pediatric Disease, Orphan Drug, and Regenerative Medicine Advanced Therapy (RMAT) designations and Rare Disease Evidence Principles (RDEP) program acceptance; potential eligibility for Priority Review Voucher (PRV) upon BLA approval

OPUS GENETICS®

OPGx-RDH12
OPGx-MERTK
OPGx-RHO

RDH12: An Early-Onset Inherited Retinal Degeneration

Overview & prevalence	• Mutations in *RDH12* cause a severe form of LCA, which leads to early, rapid vision loss in infancy/childhood, often resulting in legal blindness before the third decade of life[1] • Most frequent phenotype of *RDH12* is autosomal recessive/LCA[2] • Accounts for ~3.5-10.5% of all LCA cases[1] • **Global prevalence*:** ~30,900 patients[3] • **Middle East / North Africa prevalence:** ~17,500 patients[3] • **U.S. prevalence:** ~2,500 patients[3]
Clinical features	• Dense intraretinal pigmentation and macular atrophy with yellowish discoloration and pigmentation[1,4] • Gold foil-like reflectance and watercolor appearance[1] • Early peripheral RPE atrophy with pigmented deposits, including bone spicules[1] • Peripapillary sparing[1]
Symptoms[1,4]	• Loss of peripheral and central vision • Photophobia • Nystagmus • Night blindness



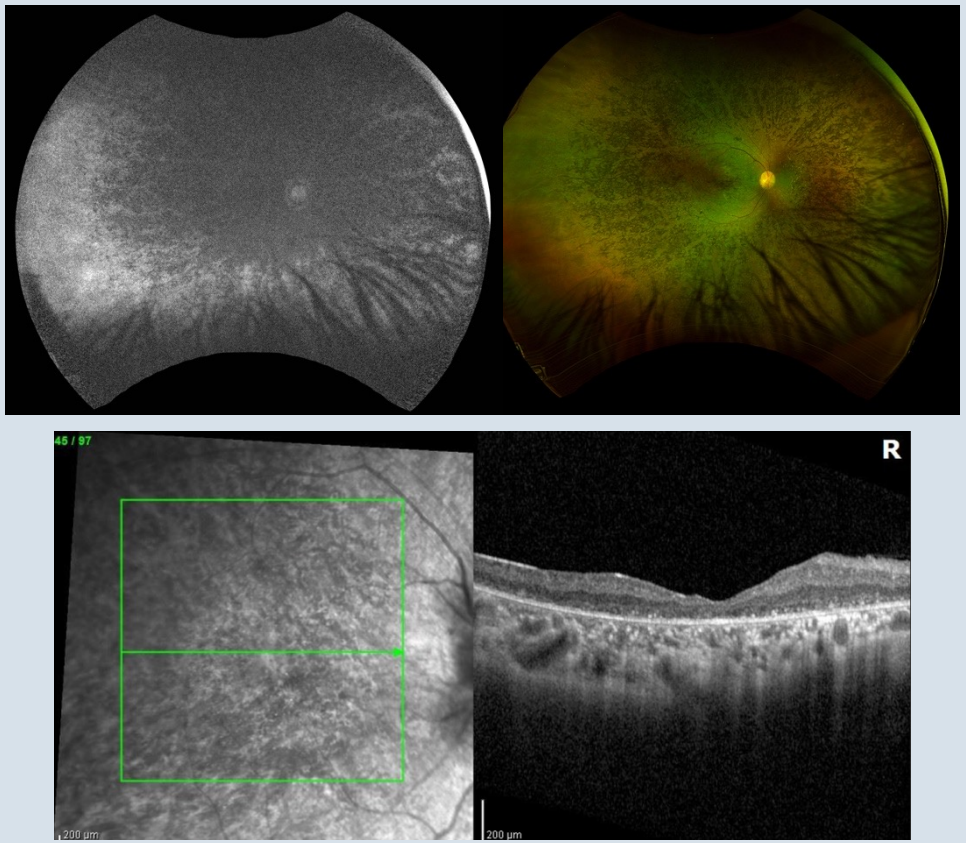
Images courtesy of Kenneth Fan, MD

*Global prevalence estimate includes United States, EU4 (France, Spain, Germany, & Italy), UK, Middle East/North Africa, and China.
LCA, Leber congenital amaurosis; RDH12, retinol dehydrogenase 12; RPE, retinal pigment epithelium.

1. Varela MD, et al. *Ophthalmic Genet.* 2022;43:301-306. **2.** Aleman TS, et al. *Invest Ophthalmol Vis Sci.* 2018;59:5225-5236. **3.** Triangle Insights Analysis 2026. Opus Genetics, Inc. **4.** Kumaran N. *Br J Ophthalmol.* 2017;101:1147-1154.

MERTK: Early-onset Inherited Retinal Disease with Rapid Vision Loss

Overview & prevalence	• Severe form of autosomal recessive RP, which has an early-onset (childhood or adolescence) and results in rapid vision loss[1] • Accounts for ~2% of all autosomal recessive RP cases[1] • **Global prevalence*:** ~21,960 patients[2] • **Middle East / North Africa prevalence:** ~14,300 patients[2] • **U.S. prevalence:** ~2,600 patients[2]
Clinical features[1]	• Early macular atrophy • Optic disc pallor (often alongside retinal vessel attenuation) • Pigmentary retinopathy • Abnormal autofluorescence of macula or bullseye maculopathy (ring-like macular lesion) • Subretinal deposits
Symptoms[1]	• Peripheral vision loss, followed by central vision • Night blindness • Photophobia • Myopia • Abnormal color vision



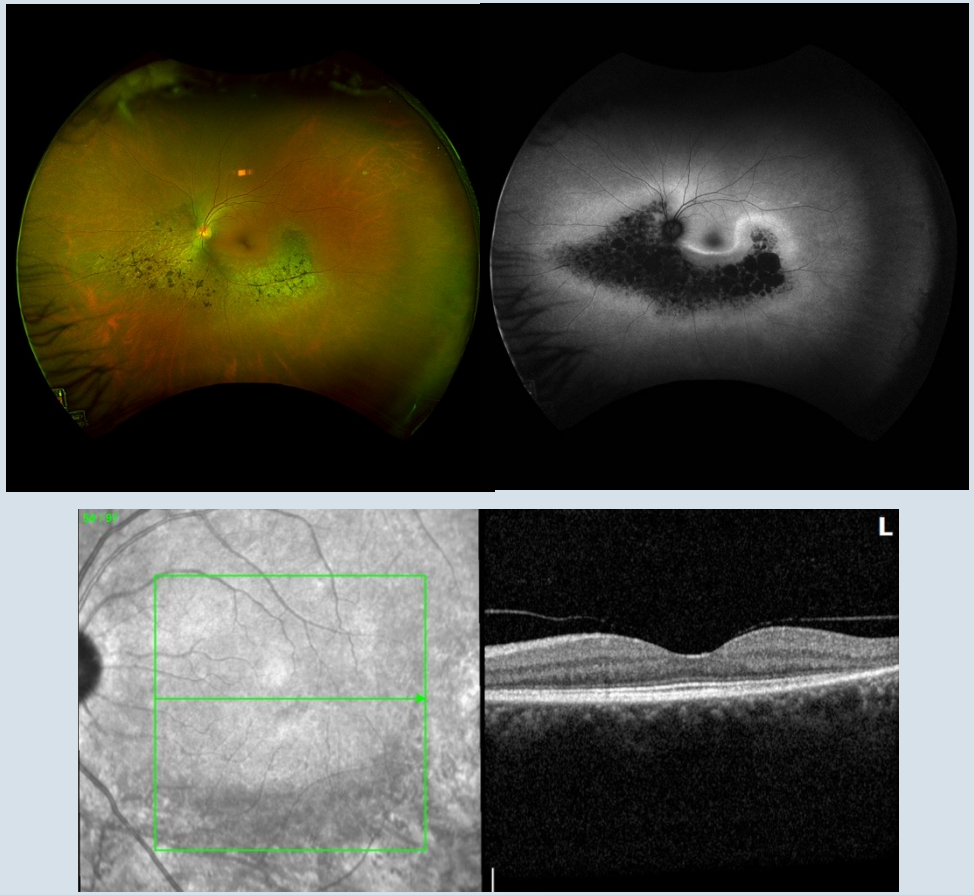
Images courtesy of Kenneth Fan, MD

*Global prevalence estimate includes United States, EU4 (France, Spain, Germany, & Italy), UK, Middle East/North Africa, and China.
MERTK, MER proto-oncogene tyrosine kinase; RP, retinitis pigmentosa.

1. Audo I, et al. *Hum Mutat.* 2018 Jul;39:887-913. **2.** Triangle Insights Analysis 2026. Opus Genetics, Inc.

RHO: Highly Variable, Often Slowly Progressive Rod-Cone Dystrophy

Overview & prevalence	• Autosomal dominant RP is the most common autosomal dominant retinal disease[1] • More than 290 identified *RHO* mutations[1] • Variants in *RHO* are the most frequent cause of adRP (~20-30% of cases) [1] • **Global prevalence*:** ~30,200 patients[2] • **U.S. prevalence:** ~8,800 patients[2]
Clinical features[1,3]	• Bone spicule-like pigment • Mottled/granular appearance • Atrophy • Vessel attenuation • Hypoautofluorescent spots along with a hyperautofluorescent macular ring
Symptoms[1,3]	• Early-onset night blindness • Progressive peripheral visual field loss • Decline of central vision in later stages



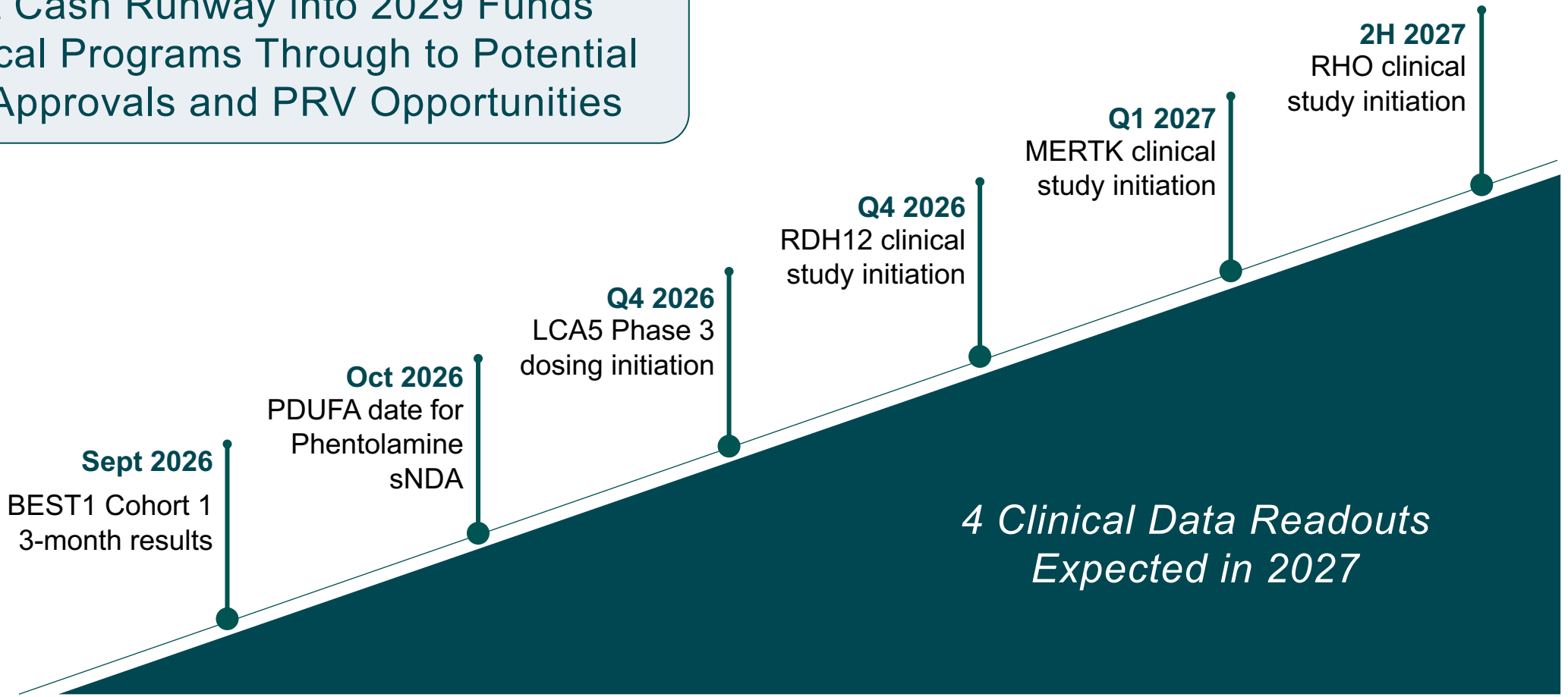
Images courtesy of Kenneth Fan, MD

*Global prevalence estimate includes United States, EU4 (France, Spain, Germany, & Italy), UK, Middle East/North Africa, and China.
adRP, autosomal dominant retinitis pigmentosa; RHO, rhodopsin; RP, retinitis pigmentosa;

1. Amaral RAS, et al. Exp. Biol. Med. 251:10893. doi: 10.3389/ebm.2026.10893. **2.** Triangle Insights Analysis 2026. Opus Genetics, Inc. 3. Varela MD, et al. *Invest Ophthalmol Vis Sci.* 2025;66:69.

Fully-Funded to Support Multiple Clinical Inflection Points

Current Cash Runway into 2029 Funds Five Clinical Programs Through to Potential Product Approvals and PRV Opportunities



Clinical development timelines are based on current estimates and are subject to change; data readouts are targeted for ~9-12 months after study initiation.

Cash balance: ~$90M as of 5/12/26 includes $35M of notes issued related to Oberland Capital financing; Common shares outstanding: 81.4 million as of 05/07/26; Pre-funded warrants:16 million

Phentolamine ophthalmic solution 0.75% is a commercial partnered program; it is FDA-approved for the treatment of pharmacologically-induced mydriasis; a supplemental New Drug Application (sNDA) has been submitted for the treatment of presbyopia. PDUFA, Prescription Drug User Fee Act;

Thank you

OPUS GENETICS®