



Lin BioScience

Bringing Hope to Incurable Diseases

2026/07

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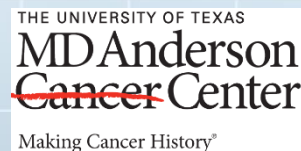
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Novel Treatment for Unmet Medical Need

Lin BioScience, Inc. (TW TPEX: 6696) is a drug development company established in 2016 focusing on advancing novel therapies and first-in-class treatments for unmet medical needs in various therapeutic areas such as ophthalmology, oncology, and metabolic diseases. The Company's pipeline consists of RBP4 IP portfolio, CDC7 IP portfolio and 4 distinct small molecule drug candidates. LBS-008, targeted to treat Stargardt disease and Geographic Atrophy ("GA"), and LBS-009, targeted to treat NASH, derived from the RBP4 IP portfolio, are developed by Belite Bio, a subsidiary company of Lin BioScience. LBS-007, developed from the CDC7 platform technology and targeted to treat various cancers, and LBS-002, targeted to treat glioblastomas and metastatic brain tumors, are developed by Lin BioScience.

LBS-007 is a non-ATP competitive CDC7 inhibitor for the treatment of a broad array of cancers, especially for refractory/relapsed and late-stage cancers such as AML, ALL, ovarian cancer, pancreatic cancer, etc., which has entered phase 1 in 2022. LBS-007 has been granted orphan drug designation (ODD) in the U.S. for the treatment of AML and ALL. LBS-007 has also obtained Fast Track Designation from the US FDA in 2024 for the treatment of AML.

LBS-008 is the first drug candidate intended to treat GA within the current drug development projects of the NIH Blueprint Program ("BPN"), whose mission is to foster small-molecule neurotherapeutic development. The mechanism of action utilized by LBS-008 has been recognized and recommended as a priority for clinical development in both STGD1 and GA in a systematic review published by the U.K. the NIHR, in 2018. LBS-008 phase 3 for Stargardt disease has been completed, with results announced in Q4 2025, followed by the completion of its Rolling Submission of NDA to US FDA in Q2 2026. Additionally, a Phase 1b/2/3 trial for Stargardt disease was initiated in 2024 and has completed its enrollment in Q1 2026. For GA, LBS-008 has initiated its phase 3 trial in 2023 and has completed its enrollment in Q3 2025. LBS-008 has been granted Fast Track Designation, Rare Pediatric Disease designation and Breakthrough Therapy Designation in the U.S., Orphan Drug Designation/Status in the U.S. Europe, Japan, and Switzerland, and Sakigake Designation in Japan for the treatment of STGD1.



Pipeline

Developed by
Lin BioScience

Oncology Programs

LBS
007

Acute Leukemia (FDA ODD)

Multiple Solid Tumors

FDA ODD (AML, ALL), FDA fast track (AML)

**Sponsored by Taiwan Industrial Development
Bureau's Innovation Platform Program**

LBS
002

Glioblastoma /
Brain Metastasis

Developed by
Belite Bio™
Subsidiary company of Lin BioScience

RBP4 IP Portfolio

LBS
008

Geographic Atrophy

Stargardt Disease
(juvenile macular degeneration)

Sponsored by NIH BPN

US/EU/JP/CH ODD; US FTD, RPD, BTD; JP Sakigake Designation

LBS
009

Non Alcoholic Fatty Liver Disease
(NASH) / Type 2 Diabetes

Discovery

Pre-
Clinical

Phase 1

Phase 2

Phase 3

MARKET

Chairman

**Tom Lin, MMED, PhD, MBA
(Chairman)**



- **10+ years of executive management role in biotech, incl. 4 IPO (Lin BioScience & Belite)**

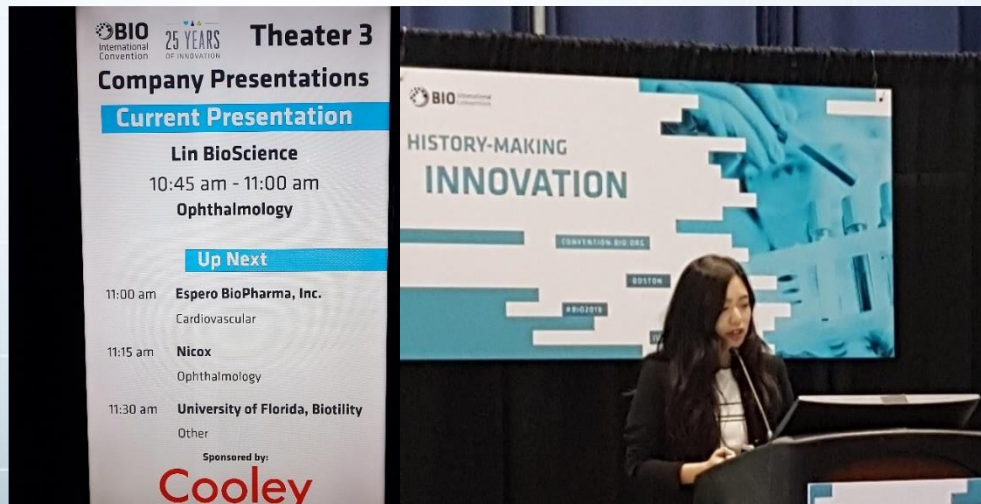
- **Multidisciplinary Specialization Clinical Training & Research in Neuroscience, Cardiovascular & Renal Medicine, Oncology, Immunology & Immunotherapy**

- **PhD in Medicine - University of Sydney**; Specialization: Neurology & Immunology
 - Treatment Strategies for Autoimmune Neuropathies
- **Specialist Certificate in Clinical Neuroscience - University of Melbourne**; Specialization: Neurology
 - Neurological Disorders, Neuroimaging & Diagnostics
 - Clinical Research & Design
- **Master of Medicine - University of Sydney**; Specialization: Multidisciplinary Medicine and Surgery
 - Medicine: Cardiovascular & Renal Medicine, Neonatal Medicine
 - Surgery: Vascular & Endovascular Surgery, Transplant Surgery
- **Cancer Therapeutics & Research Certificate - Harvard Medical School**
- **Master of Business Administration - Columbia University, London Business School, HK University**
- **Extensive Drug development from preclinical to global phase 3 trials**
 - Phase 3 - RBP4 Inhibitor for the Treatment of Atrophic Macular Degeneration & Stargardt Disease
 - Phase 2 - Oubain Antagonist in the Treatment of Essential Hypertension
 - Phase 2 - SERCA2a Inhibitor in the Treatment of Acute Heart Failure
 - Phase 2 - Pan-HER Inhibitor in the Treatment of HER2+ Breast Cancer and Gastric Cancer
 - Phase 3 - Anti-Glycan Active Immunotherapy in the Treatment of Metastatic Breast Cancer
 - Phase 3 - Anti- $\alpha 4$ integrin Antibody in the Treatment of Resistant-Refractory Multiple Sclerosis
 - Phase 2 - mTOR Immunosuppressant in the Treatment of Autoimmune Peripheral Neuropathies
 - Co-invented and applied 64 patents

Management Team

Irene Wang, PhD, MBA (President & CSO)

- PhD in Biochemical Sciences, National Taiwan University, Trained at Scripps Research (TSRI), EMBA from University of California San Diego
- Co-invented and applied 125 patents and published 6 papers
- Extensive Drug development from preclinical to global phase 3 trials and **3 IPOs (including Lin BioScience and Belite Bio)**



“I’ve loved chemistry since I was little. I was dedicated to studying chemistry and scientific research since middle school. And now, I’m working on drug development, doing significant things to improve the lives of human beings.”



Irene Wang, PhD, MBA
President
LIN BIOSCIENCE

Management Team

**Serena Chen
(CFO)**



- Certified Public Accountant & master in accounting from National Taipei University.
- Finance manager in a Taiwan biotech company and as assistant manager of audit department in Deloitte Taiwan
- Vast experience in auditing of **listed companies and initial public offering (including Lin BioScience and Belite Bio)**

**Tzung-Ju Wu, PhD
(Director, R&D)**



- Ph.D. in Cellular and Molecular Pharmacology from Rutgers University
- 10-years of global Pharma/Biotech R&D experience in Sanofi Genzyme, Taiwan Liposome Company and Insilico Medicine
- Experience in leading R&D teams to conduct innovative research and support drug discovery and development in multi-disease area



**LBS-007
FOR ACUTE LEUKEMIA &
SOLID TUMOR**



LBS
007

Therapeutic Options For Cancer

Unmet Medical Needs for Cancer Treatment - Next Generation Therapies

**Genetic
Therapy**

Immunotherapy

Targeted Therapy

Chemotherapy

LBS-007

LBS-007:

An Innovation to Transform the Cancer Treatment Landscape

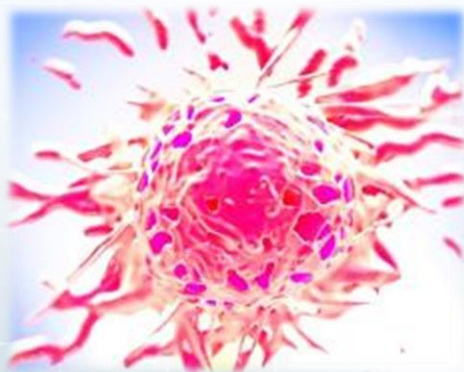
- Cancer treatments are like a pyramid—higher levels have fewer side effects but are more costly and less applicable.
- Chemotherapies form the basis of all cancer treatments. Targeted therapies or immunotherapies are complemented by chemotherapies. Even late-stage cancers and cancers with limited treatment options are often managed by chemotherapies.
- The substantial side effects of chemotherapies, with their technology unchanged over 60 years, remain a significant unmet medical need.

LBS
007

Non-ATP CDC7 Inhibitor

For Treatment of Broad Variety of Cancer types

- DISCOVERY
- PRE-CLINICAL
- ☒ PHASE I/II
- PHASE II/III
- MARKET



Novel Anti-Cancer Target Therapy



Orphan Drug Designation

For ALL: #DRU-2017-6250

For AML: #DRU-2024-10100

Investigational New Drug

#120774 became active on 05Oct2024

Fast Track Designation

For AML, granted on 26Nov2024

MARKET

\$5B

Expected 2026 market
size of AML & ALL

\$55B

Expected 2023 market
size of pancreatic, lung,
ovarian cancers

1.7 in 100k⁽¹⁾

4.1 in 100K⁽²⁾

(1) ALL incidence (2016)

(2) AML incidence (2020)

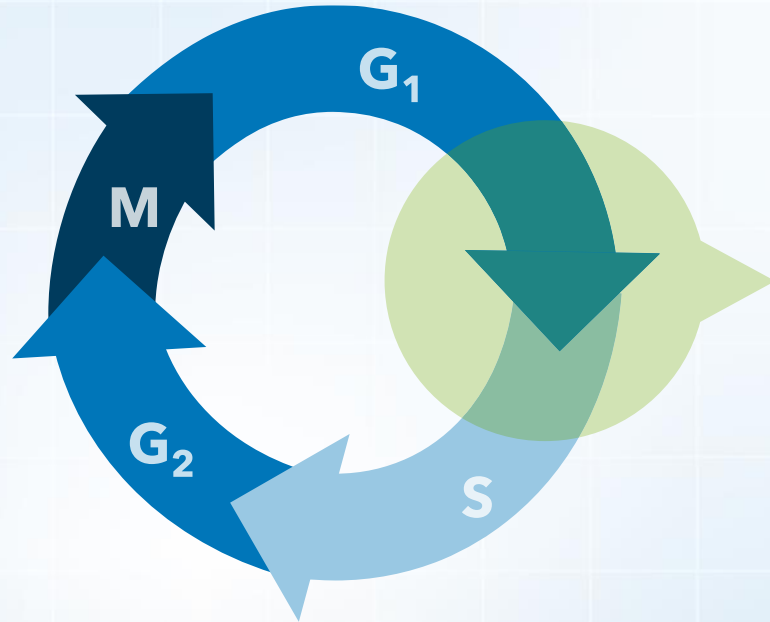
\$6B

Estimated global market

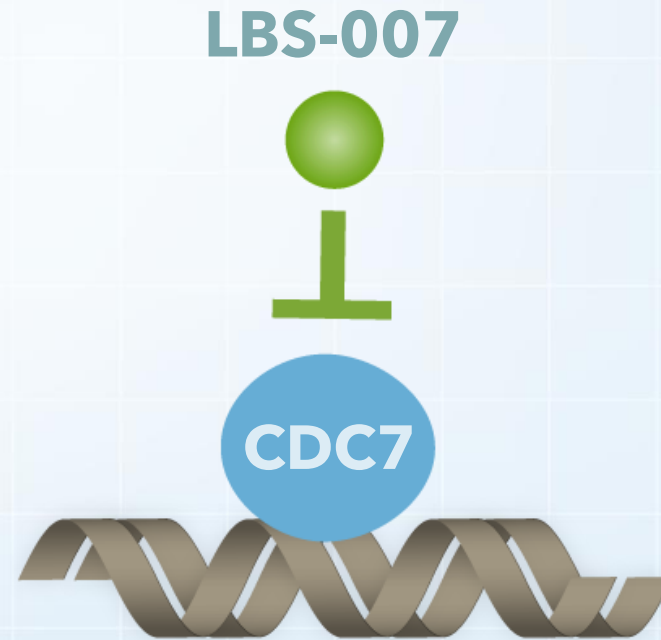
Reference: Globaldata, Marketwatch, NIH National Cancer Institute

LBS
007

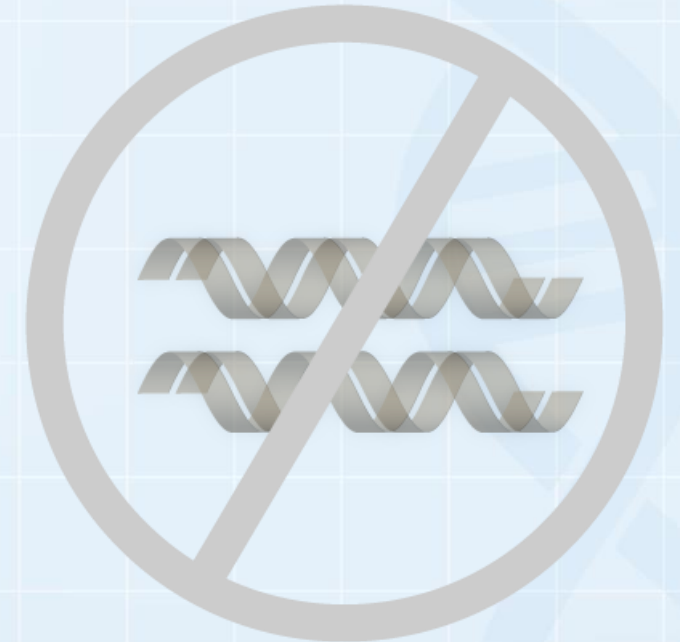
Inhibits CDC7 in Cell Cycle Regulation



- 1 TARGETS**
S Phase Progression



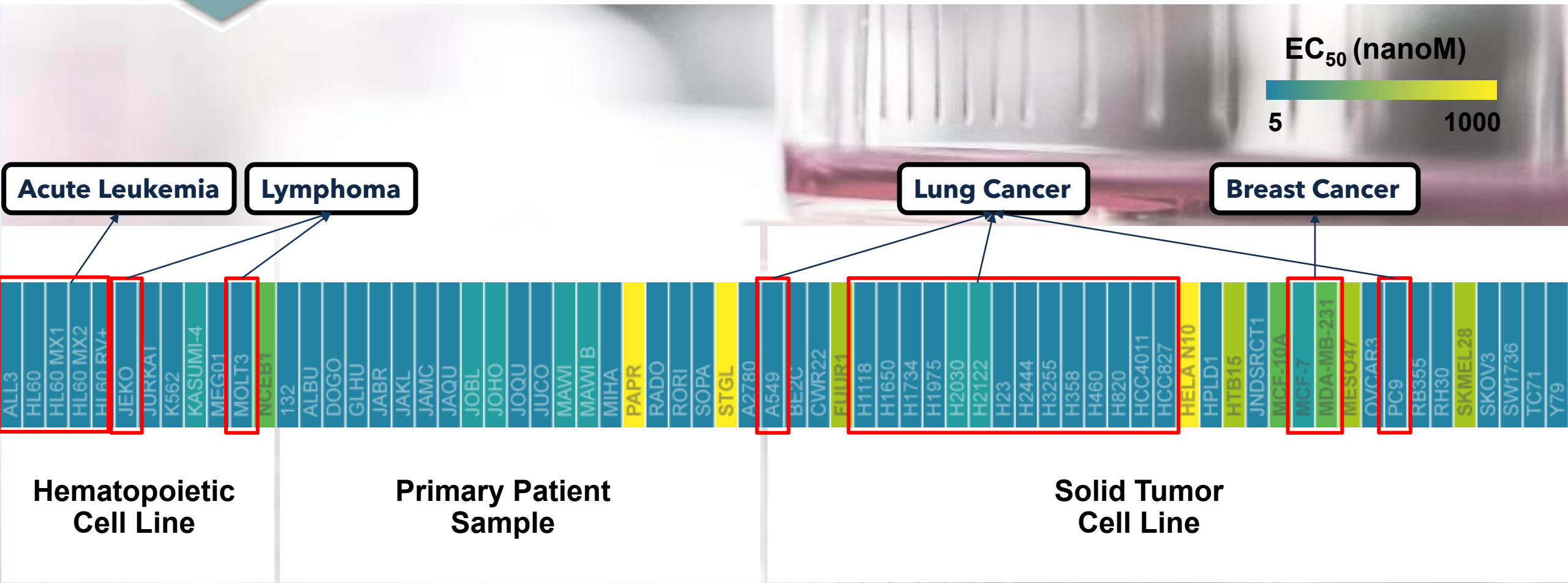
- 2 INHIBITS**
CDC7's role in
DNA Replication



- 3 PREVENTS**
Cell Division



Potently Inhibits Multiple Cancer Cell Lines & Primary Patient Samples of Blood Cancers

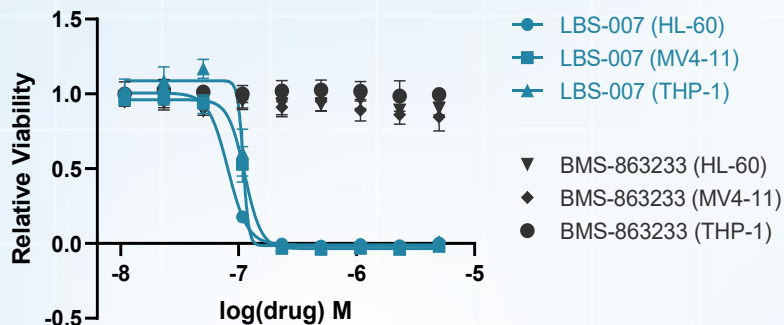




Superior Efficacy at Nanomolar Potency

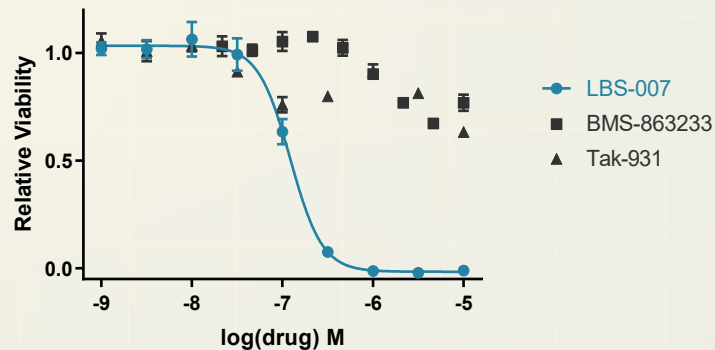
Approx. 150 nanomolar of LBS-007 can achieve therapeutic effect on cancer cells

AML



EC ₅₀ (nM)	HL-60	MV4-11	THP-1
LBS-007	83.8	71.5	108
BMS-863233	> 10,000	5,400	> 10,000

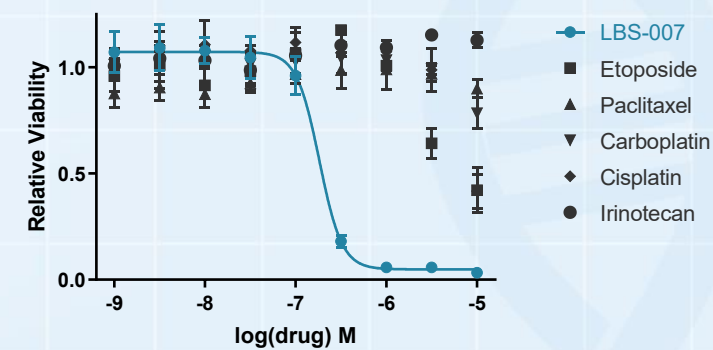
Pancreatic cancer



EC ₅₀ (nM)	Panc-1
LBS-007	127
BMS-863233	1,100
Tak-931	> 10,000

8.6x efficacy

Lung cancer



EC ₅₀ (nM)	H146
LBS-007	183
Cisplatin	2,800
Etoposide	> 10,000
Irinotecan	> 10,000
Paclitaxel	> 10,000
Carboplatin	> 10,000

15x efficacy

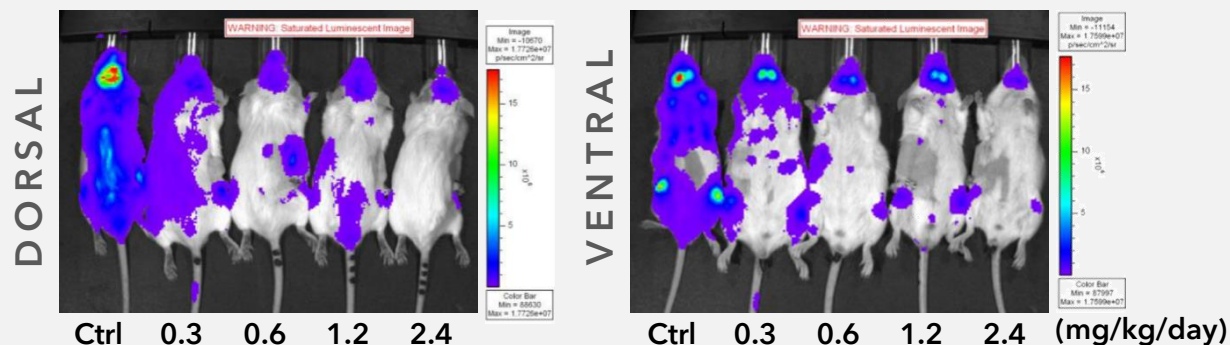
>50x efficacy

LBS
007

In Vivo Efficacy Demonstrated in Animal Models

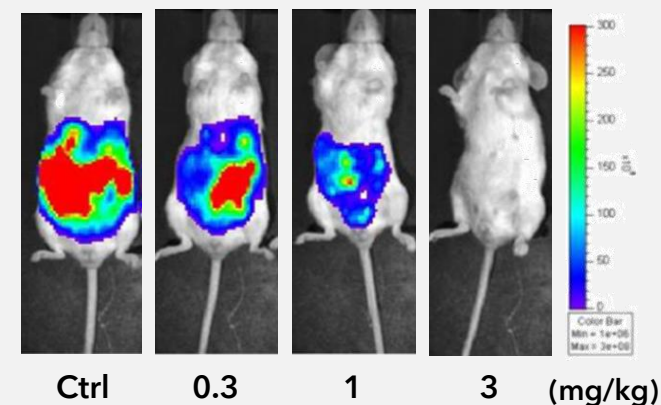
Potent tumor reduction in ALL and solid tumor mouse models

Acute Lymphoblastic Leukemia (ALL)



- ✓ In vivo dose responsive efficacy
- ✓ 95% tumor removal at 2.4 mg/kg/day
- ✓ No significant organ dysfunction or toxicity at therapeutic dose

Ovarian Cancer



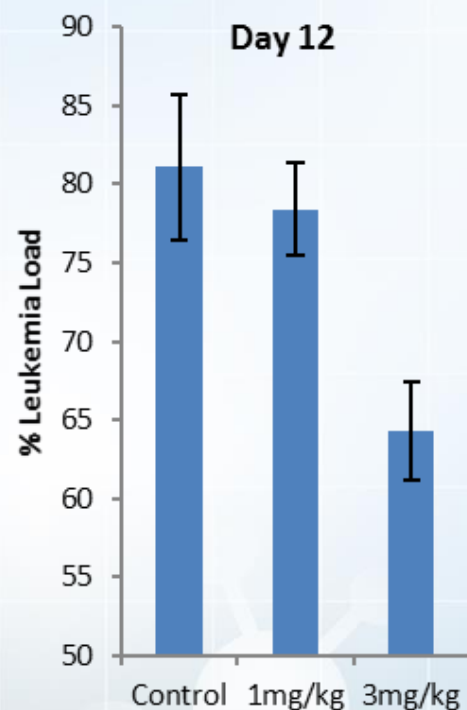
- ✓ In vivo dose responsive efficacy
- ✓ Inhibits ovarian cancer growth in mice
- ✓ Significant improvement in long-term survival

LBS
007

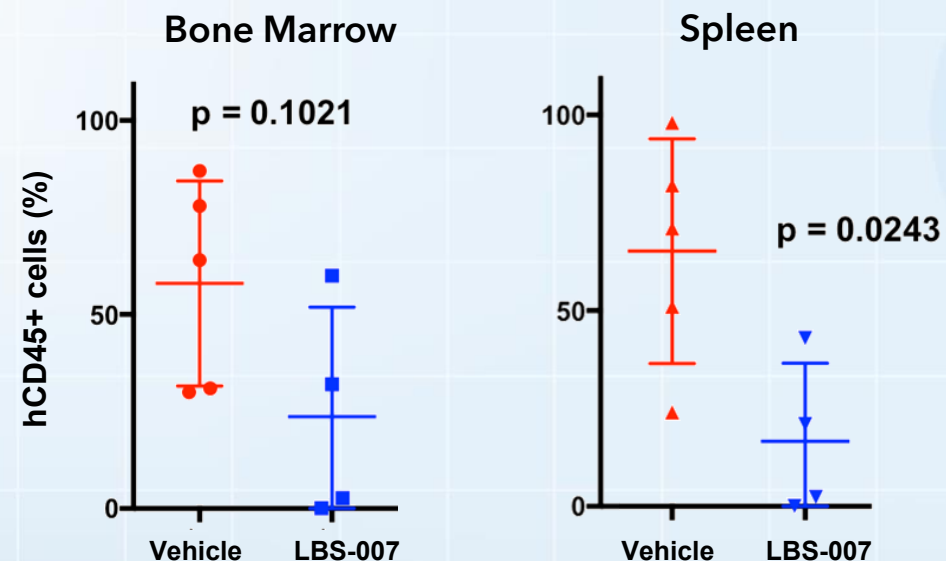
In Vivo Efficacy Demonstrated in Animal Models

Potent tumor reduction in AML mouse models

Aggressive AML mouse model (MLL-AF9)



AML patient-derived xenograft in mice



- ✓ **Disease burden reduced** in aggressive AML mouse model at 3 mg/kg
- ✓ Inhibits human AML growth in mice

LBS
007

Clinical Development Summary



	LBS-007-CT01
Phase	1/2 (Phase 2 dose expansion after determining optimal dose in Phase 1)
Enrollment	Estimated to enroll 90 patients
Sites	Australia, Taiwan, US, China
Masking	Open Label
Treatment duration	7 consecutive days for one 28-day cycle
Primary measures	Safety, tolerability, optimal dose, and PK profile of LBS-007
Other measures	Efficacy of LBS-007
Key Inclusion Criteria	Aged ≥ 18 , with confirmed relapsed or resistant AML or ALL, ineligible for standard therapies with an ECOG of 0 to 2.



MAJOR MILESTONES

Opening a New Era in Cancer Treatment

- ✓ **2023/02/10** - We announced that our new drug, LBS-007, has been approved by the Central Adelaide Local Health Network Human Research Ethics Committee (CALHN HREC) to conduct Phase I/II clinical trials for acute leukemia (including AML and ALL) in Australia. The company aims to address unmet medical needs in the cancer treatment market.
- ✓ **2023/08/11** - We announced that our new drug, LBS-007, has been approved by Taiwan's Food and Drug Administration (TFDA) to conduct Phase I/II clinical trials for acute leukemia (including AML and ALL) in Taiwan.
- ✓ **2024/10/05** - We announced that our new drug, LBS-007, for the treatment of acute leukemia has passed the US FDA IND review for human clinical trials. Efforts to initiate Phase I/II clinical trials in the US are underway.
- ✓ **2024/11/26** - We received Fast Track Designation from the U.S. FDA for our new drug, LBS-007, aimed at treating AML.
- ✓ **2025/07/22** - First U.S. clinical Site Initiation Visit (SIV) completed.
- ✓ **2025/09/29** - We announced that our new drug, LBS-007, has been approved by China's National Medical Products Administration (NMPA) to conduct Phase I/II clinical trials for acute leukemia (including AML and ALL) in China.
- ✓ Following the completion of Phase I clinical trials, the safety and efficacy of LBS-007 will be confirmed. We plan to simultaneously initiate clinical trials targeting other hard-to-treat solid tumors, including pancreatic cancer, small-cell lung cancer, and ovarian cancer.

**TINLAREBANT (LBS-008)
FOR GEOGRAPHIC ATROPHY
& STARGARDT DISEASE**



BELITE BIO PIPELINE OVERVIEW



PRECLINICAL

PHASE 1

PHASE 2

PHASE 3

NDA

Tinlarebant

○ Stargardt Disease (STGD1)

- Ph2, 2-year treatment, completed (**13 subjects**)
- Ph3, 2-year treatment, global trial ("DRAGON" Study) completed (**104 subjects**)
- Ph2/3, 2-year treatment, global trial ("DRAGON II" Study) is ongoing (completed enrollment, **73 subjects**)
- US FDA rolling submission of NDA completed

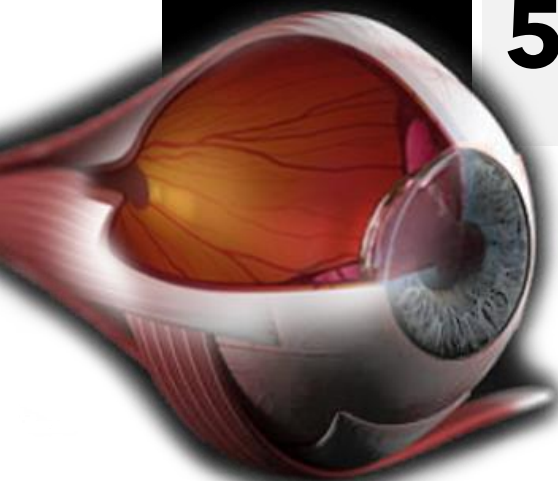
○ Geographic Atrophy (GA)

- A Ph3, 2-year treatment, global trial ("PHOENIX" Study) is ongoing (completed enrollment, **530 subjects**)

- **Tinlarebant** is a **novel, once daily oral tablet** designed to bind to serum **retinol binding protein 4 (RBP4)** as a means to specifically reduce retinol delivery to the eye. This approach is intended to **slow or halt the formation of the toxic retinol-derived by-products** that are generated in the visual cycle and are **implicated in progression of STGD1 and GA**.
- Belite Bio believes that **intervention directed at a root cause of retinal pathology**, namely bisretinoid-mediated toxicity but not complement activation, will be the best approach to potentially slow disease progression in STGD1 & GA.
- **Unmet Market Opportunity:** No FDA approved treatments for STGD1 and orally administered treatments for GA
- **Breakthrough Therapy, Fast Track, and Rare Pediatric Disease Designation** in US and **Orphan Drug** designation/status in US / EU / JP/CH, **Pioneer Drug** designation in JP, for STGD1
- **14 active patent families**; composition of matter patent until at least **2040** without patent term extension

Tinlarebant (LBS-008)

- DISCOVERY
- PRE-CLINICAL
- PHASE I
- PHASE II
- **PHASE III**
- MARKET



MARKET OPPORTUNITY

STARGARDT

1 in 8,800^(1,2)

The most common
inherited retinal dystrophy

Patient population with
Stargardt Disease:

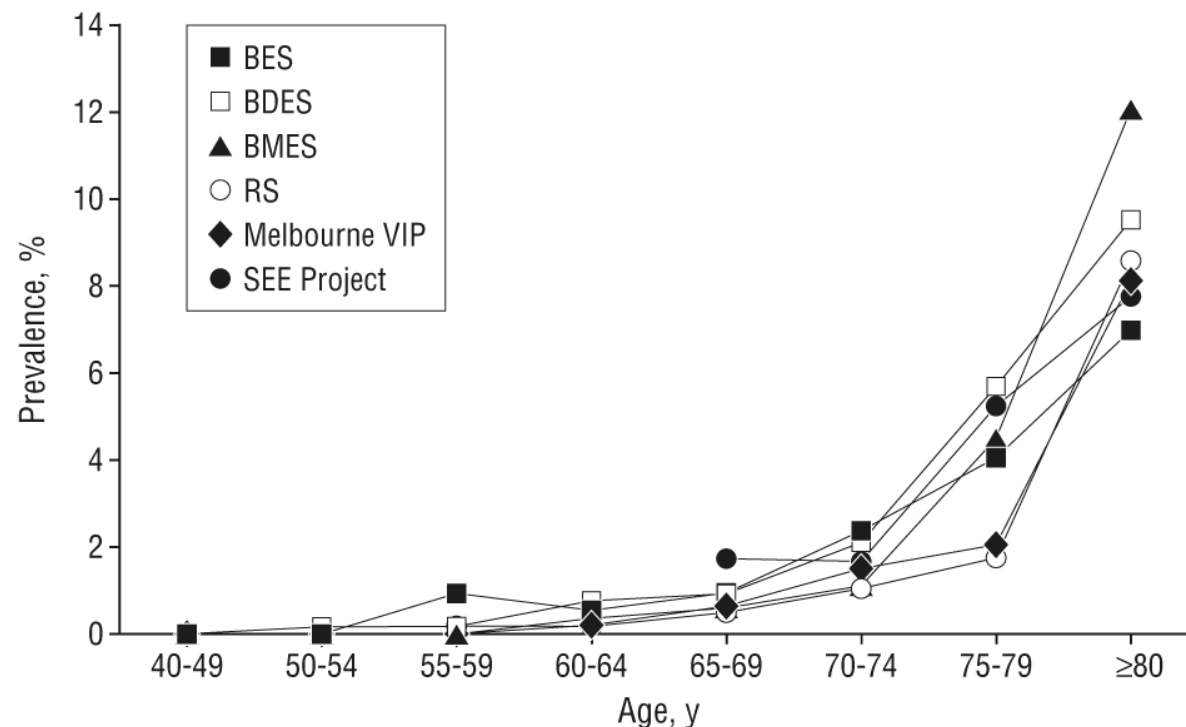
53k⁽²⁾
US

109k⁽²⁾
China

Columbia University + NIH Blueprint

"a promising first-in-class oral medication
intended to slow or halt the progression of
dry AMD"

GEOGRAPHIC ATROPHY



An estimated 973,000 people in the United States⁽³⁾ and approximately 5 million globally⁽⁴⁾ have GA in at least one eye.

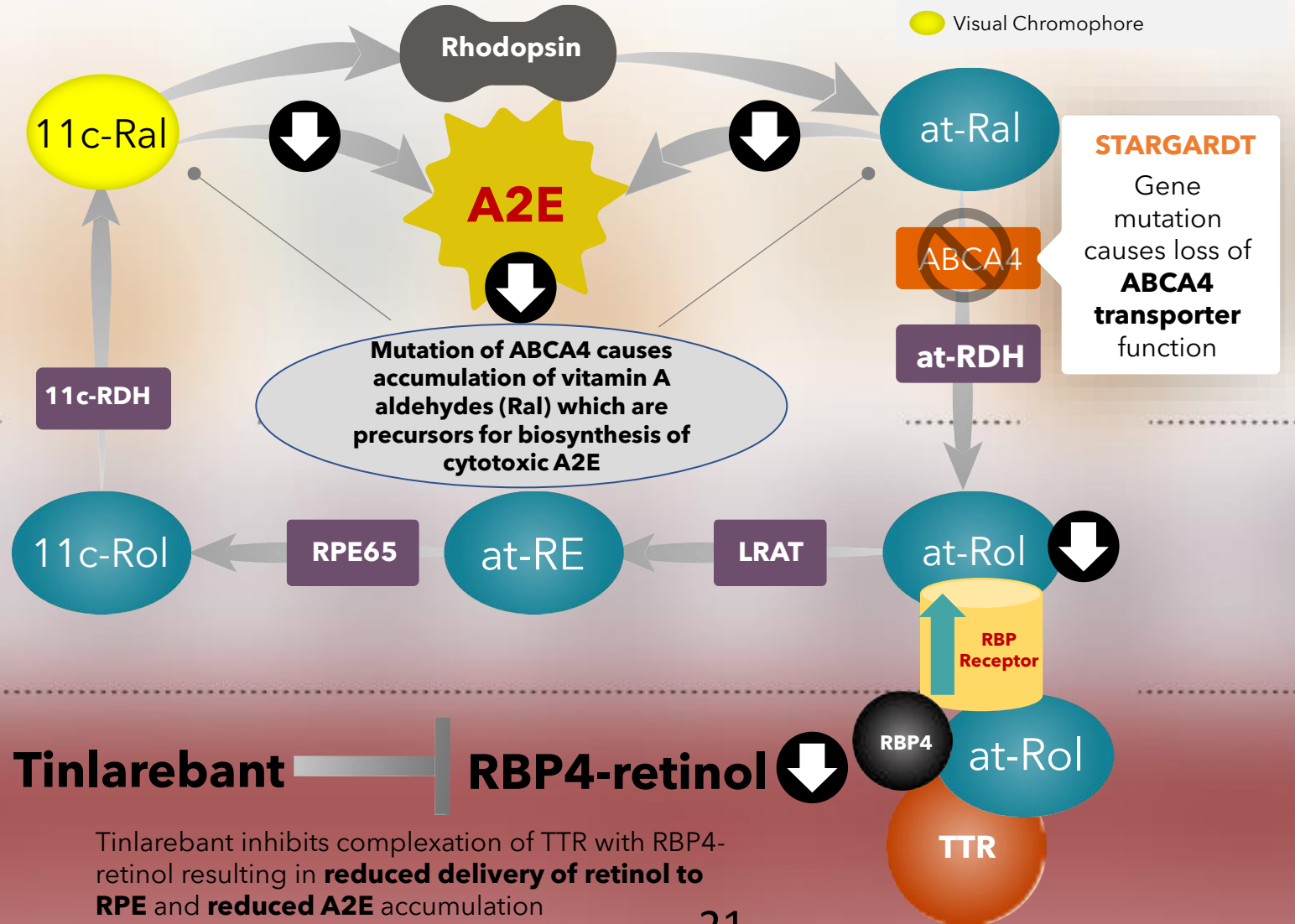


MECHANISM OF TINLAREBANT ACTION

PHOTORECEPTORS (PR)

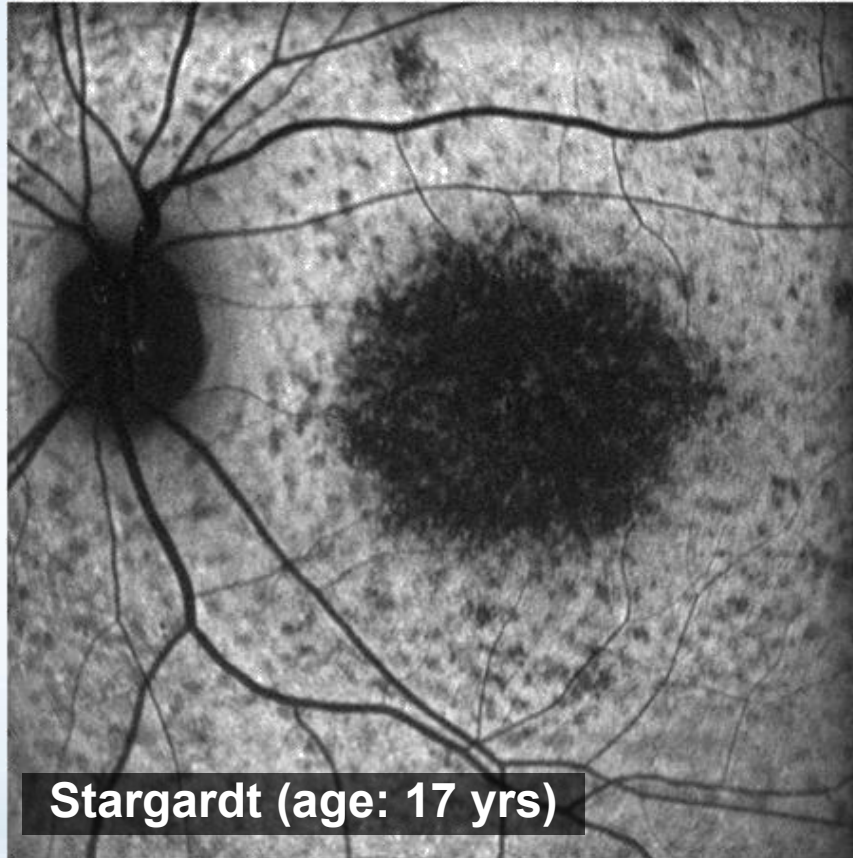
RETINAL PIGMENT EPITHELIUM (RPE)

BLOODSTREAM

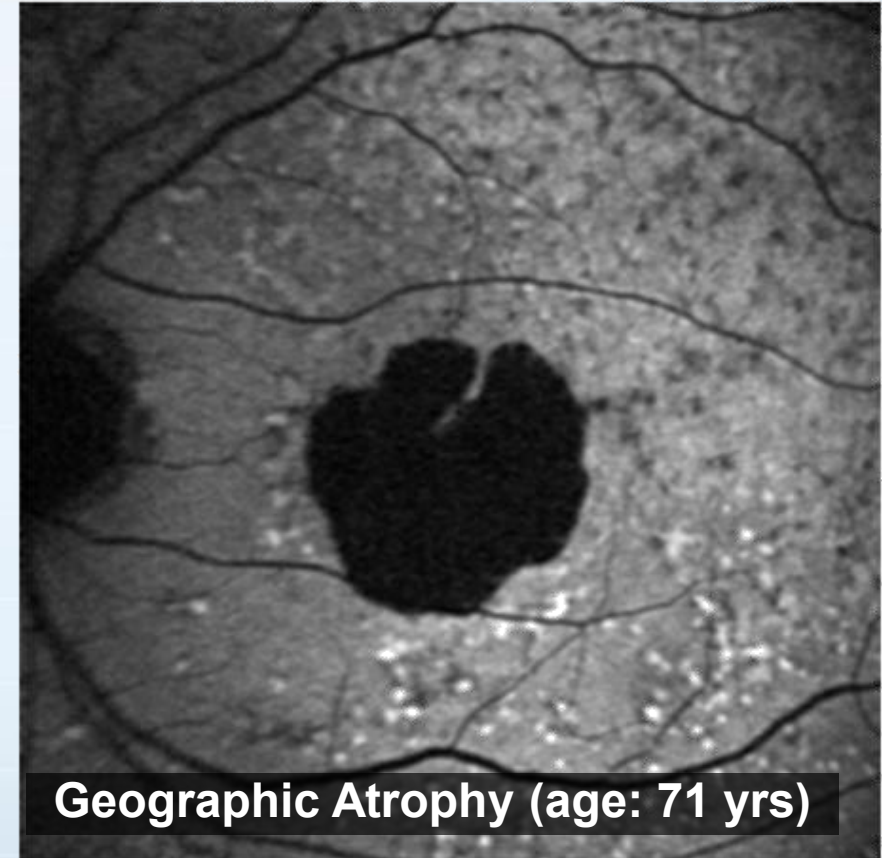


SIMILAR PATHOPHYSIOLOGY IN STGD1 & GA

- **STGD1 and GA show a very similar phenotype**, where loss of RPE and photoreceptors in the center of the retina, the macula, leads to loss of vision
- Fundus autofluorescence imaging, visualizing bisretinoid content in the RPE, **shows loss of signal in degenerated areas** and **increase in signal peripheral to lesions**
- **Slowing or halting the spread of retinal lesions** is the intended effect of **Tinlarebant treatment**



Stargardt (age: 17 yrs)



Geographic Atrophy (age: 71 yrs)



DRAGON CLINICAL TRIAL



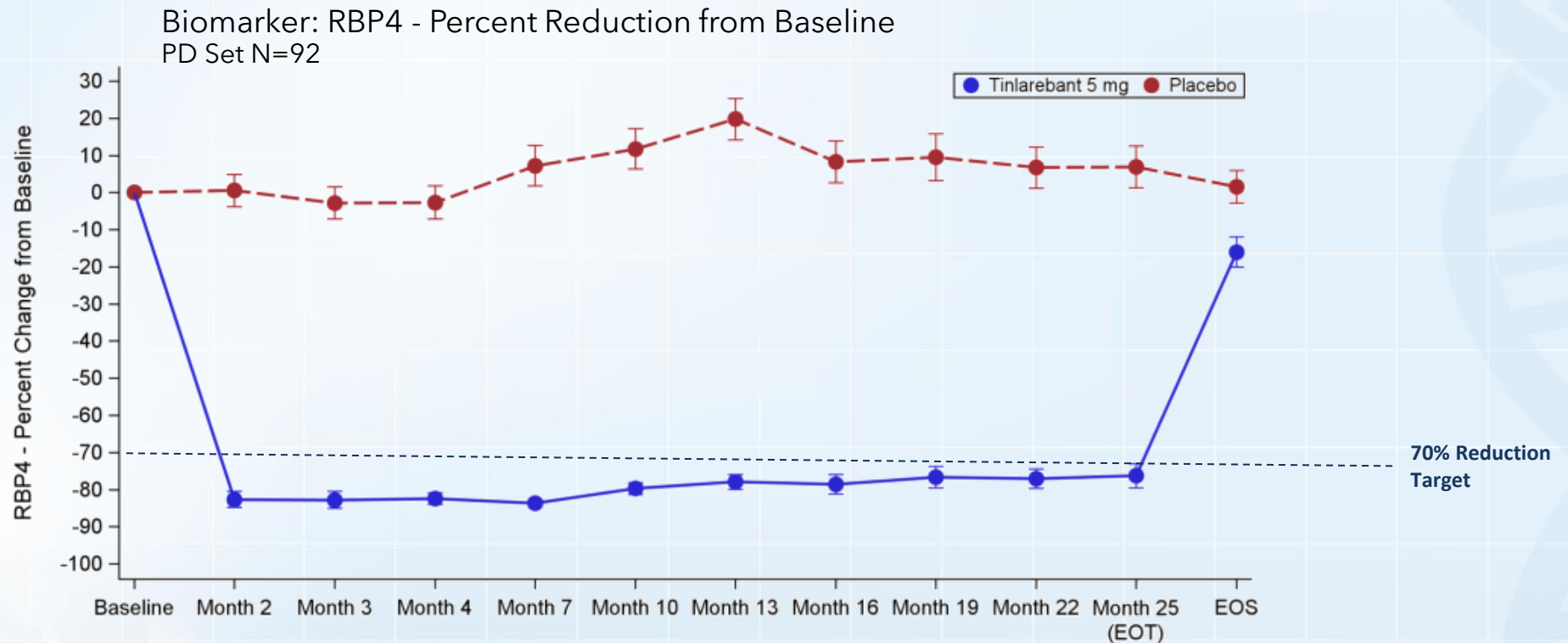
DRAGON & DRAGON II CLINICAL TRIAL DESIGN IN STGD1

Reduction in Lesion Growth Rate (DDAF) as Measured by Retinal Imaging is an FDA Accepted Primary Endpoint in STGD1 and GA

	STGD1 “DRAGON” Phase 3 ⁽¹⁾	STGD1 “DRAGON II” Phase 1b/2/3
Enrollment	104 subjects (have DDAF)	60 subjects (have DDAF)
Sites	Global	Japan, US, UK
Randomization	2:1 ratio (Tinarebant : Placebo)	1:1 ratio (Tinarebant : Placebo)
Masking	Double Blind	
Treatment duration	2 years	
Primary measures	Efficacy as measured through DDAF lesion growth rate, safety & tolerability	
Other measures	QDAF, BCVA, SD-OCT, microperimetry	
Interim analysis	Yes	
Key inclusion criteria	12-20 years old, diagnosed STGD1 with at least 1 mutation identified in the ABCA4 gene, atrophic lesion size within 3 disc areas (7.62 mm ²), a BCVA of 20/200 or better	

⁽¹⁾FDA may require another clinical trial depending on the data from the ongoing Phase 3 study.

TINLAREBANT TREATMENT LED TO 80% REDUCTION IN RPB4, WELL ABOVE GOAL OF 70%*

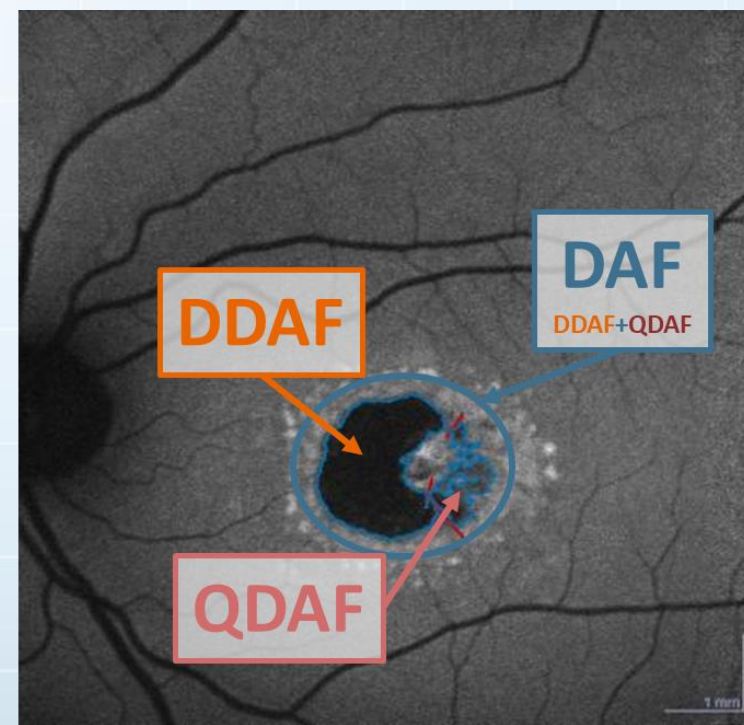
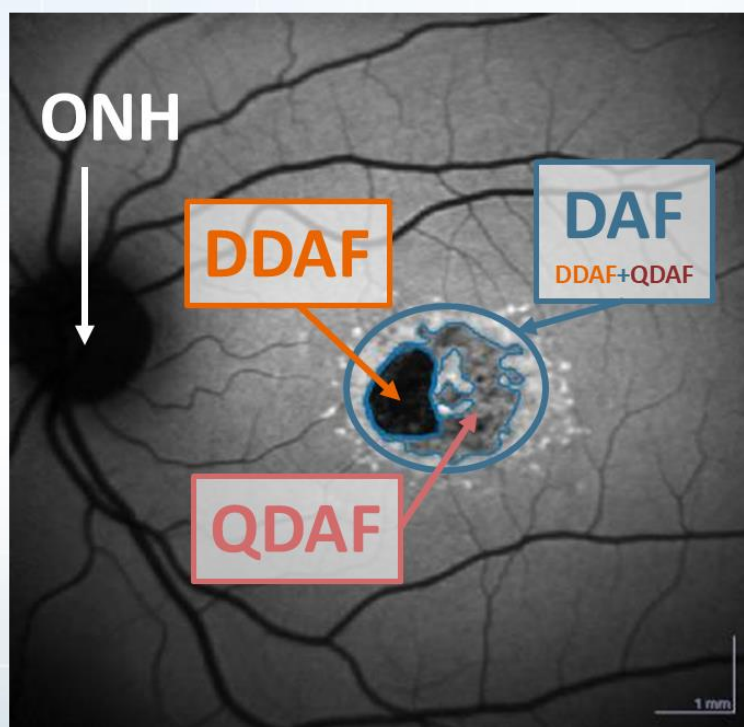


* In a prior study of a surrogate RBP4 antagonist (fenretinide) in patients with Geographic Atrophy, an RBP4 reduction of $\geq 70\%$ was associated with a statistically significant slowing of lesion growth [Mata et al., Retina. 2013; 33(3): 498-507.]

Daily dosing of 5 mg/day Tinlarebant led to a sustained 80% reduction of RPB4 and RPB4 levels returned to 84 % of the baseline value at the End of Study (EOS)

DDAF REPRESENTS WELL-DEMARCATED AREAS OF COMPLETE RPE LOSS & GROWS PREDICTABLY, MAKING IT AN APPROVABLE PRIMARY ENDPOINT

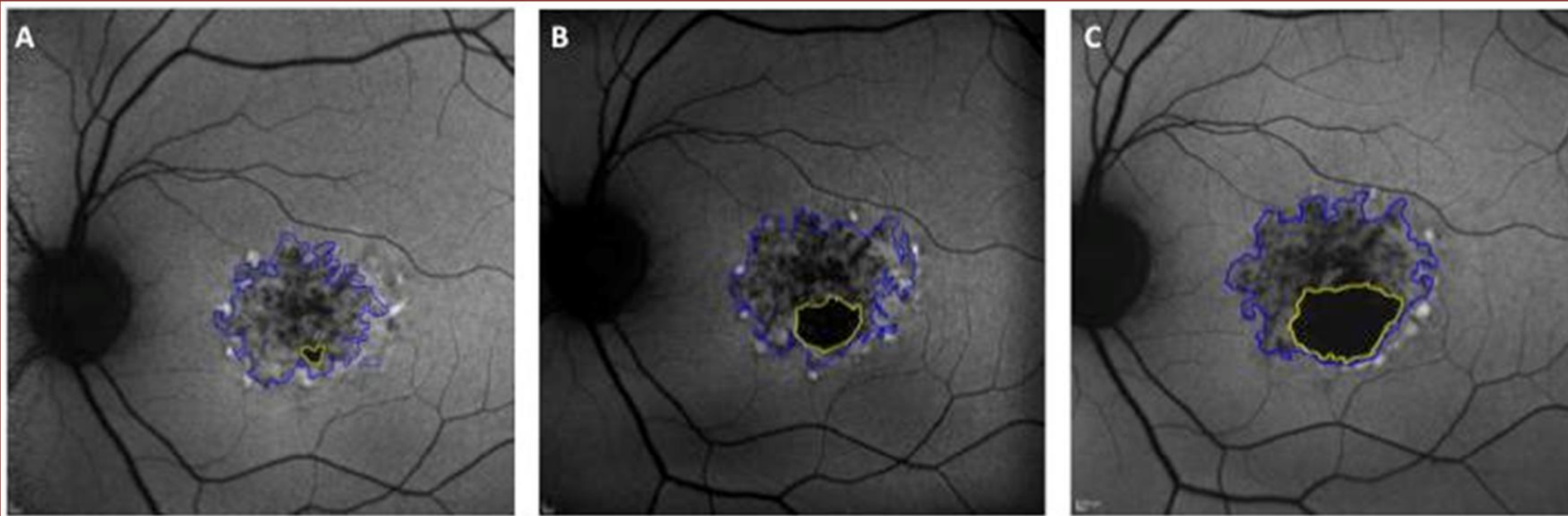
- **DDAF** (definitely decreased autofluorescence): level of darkness close to 100% (at least 90%) in reference to the ONH
- **QDAF** (questionably decreased autofluorescence): between 50% and 90% darkness
- **DAF** (decreased autofluorescence): the sum of DDAF and QDAF



DDAF PROGRESSION RATE IN STARGARDT

Natural History of the Progression of Atrophy Secondary to Stargardt Disease (ProgStar) study

Overall DDAF growth rate in the ProgStar cohort over 24 months: $0.74 \text{ mm}^2/\text{year}$
(confidence interval: $0.64 - 0.85 \text{ mm}^2/\text{year}$)

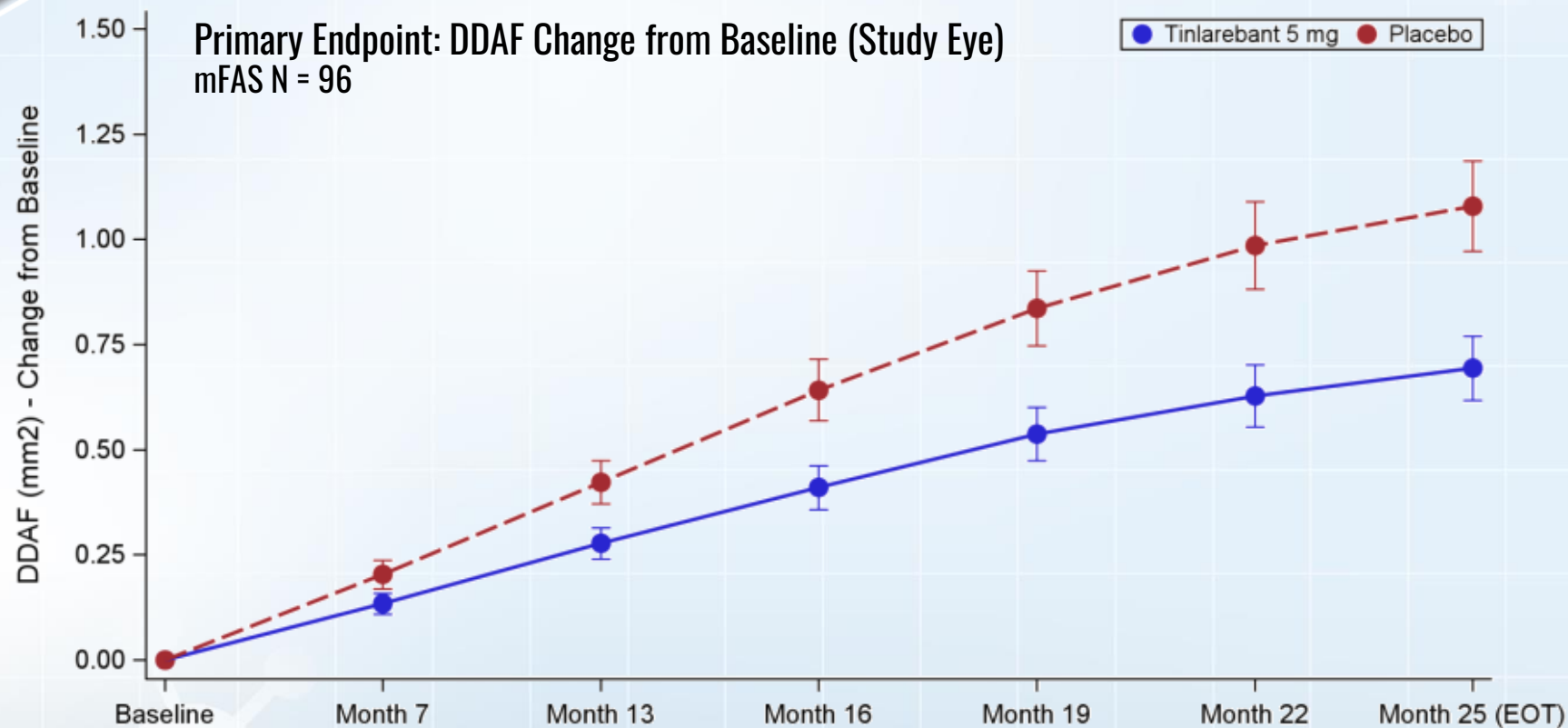




PRIMARY ENDPOINT: DDAF IN THE STUDY EYE (CHANGE FROM BASELINE)

- Annualized rate of lesion growth in the aggregate area of atrophy (DDAF) from baseline as assessed by fundus autofluorescence imaging at Month 25.
- Data is shown for the modified full analysis set (mFAS) which consists of all subjects who were randomly assigned to receive study drug and have received at least one dose of study medication. In addition, the mFAS subjects must have a defined DDAF lesion meeting the eligibility criteria at baseline and have at least one post baseline assessment.
- Data analysis used a Mixed Model for Repeated Measures (MMRM) measuring change from baseline in DDAF in the study eye and including terms for treatment, visit, treatment*visit interaction, baseline focality of lesions, and baseline DDAF lesion size.
- The Statistical Analysis Plan (SAP) specified an unstructured covariance matrix for the MMRM. The CRO also performed a post-hoc analysis using a first-order autoregressive covariance matrix to account for the longitudinal nature of the data while maintaining model stability in a relatively small sample such as in the DRAGON trial.

PRIMARY ENDPOINT SHOWED A STATISTICALLY SIGNIFICANT & CLINICALLY MEANINGFUL OUTCOME

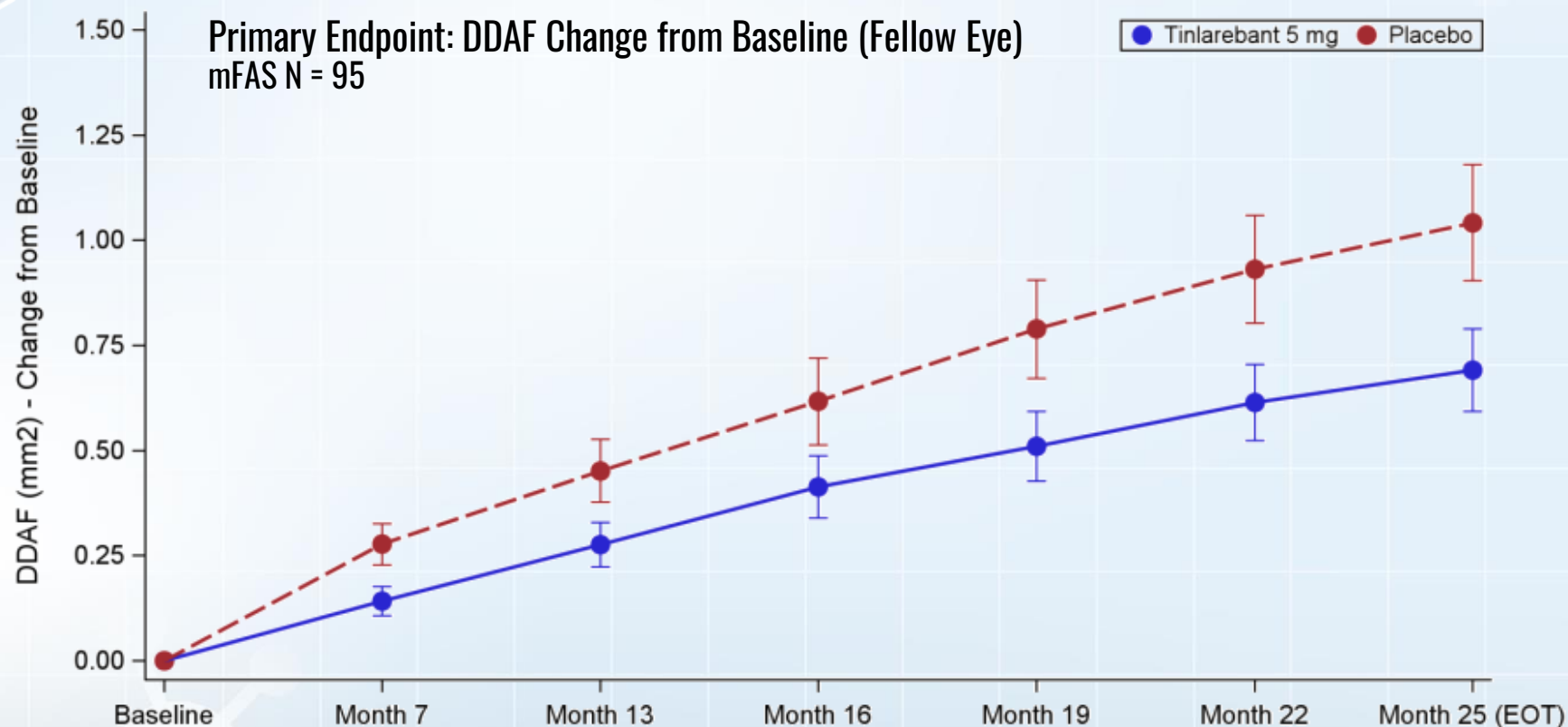


Applying an unstructured covariance matrix, the **treatment effect size was 35.7%** compared to placebo and yielded a **p-value of P = 0.0033**

With a first-order autoregressive covariance matrix, the **treatment effect size remained consistent (35.4%)** with **P < 0.0001**

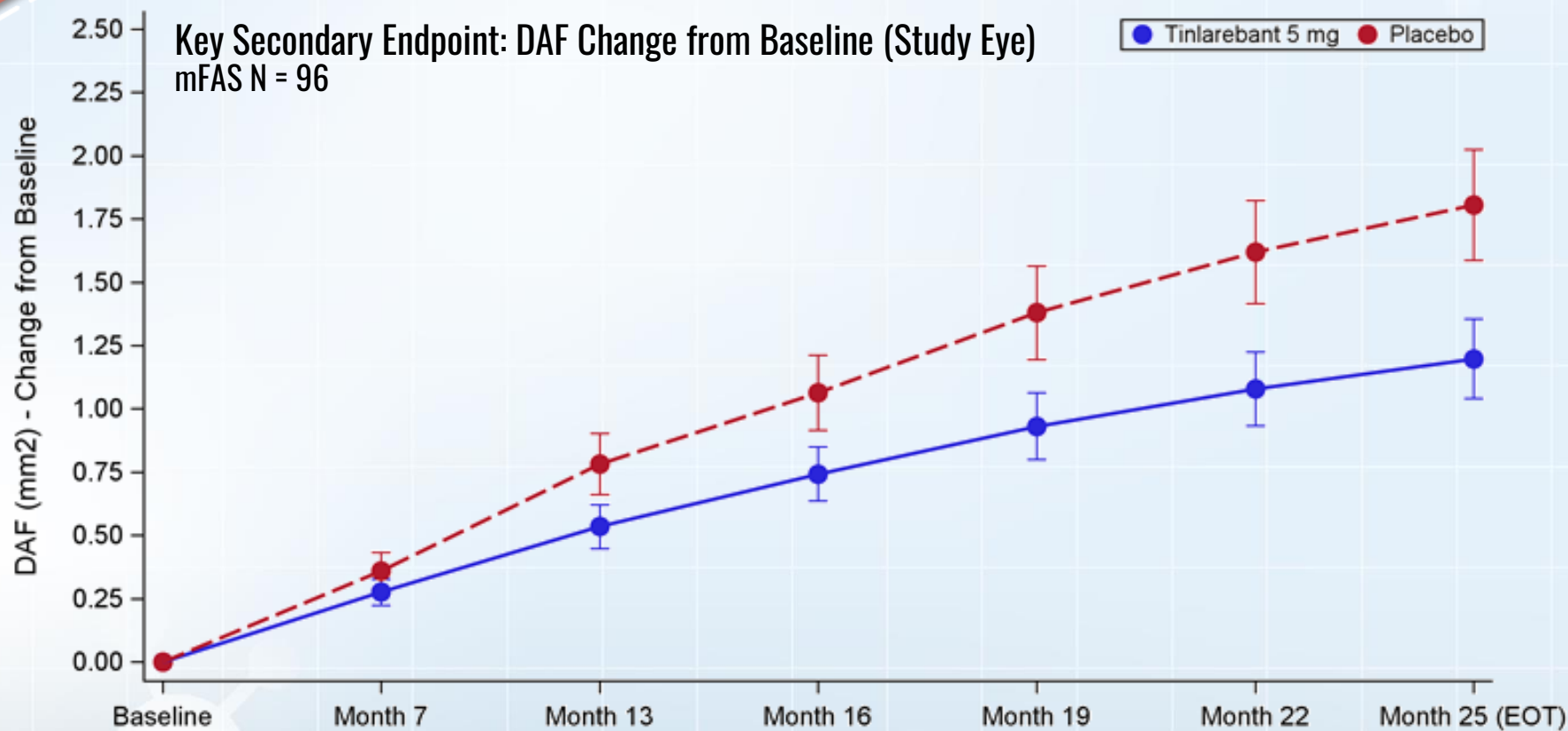
DDAF lesion growth was **slowed to 0.38 mm²/year vs. 0.59 mm²/year for placebo and 0.74 mm²/year observed in ProgStar**

A STATISTICALLY SIGNIFICANT TREATMENT EFFECT WAS ALSO OBSERVED IN THE FELLOW EYE FOR THE PRIMARY ENDPOINT



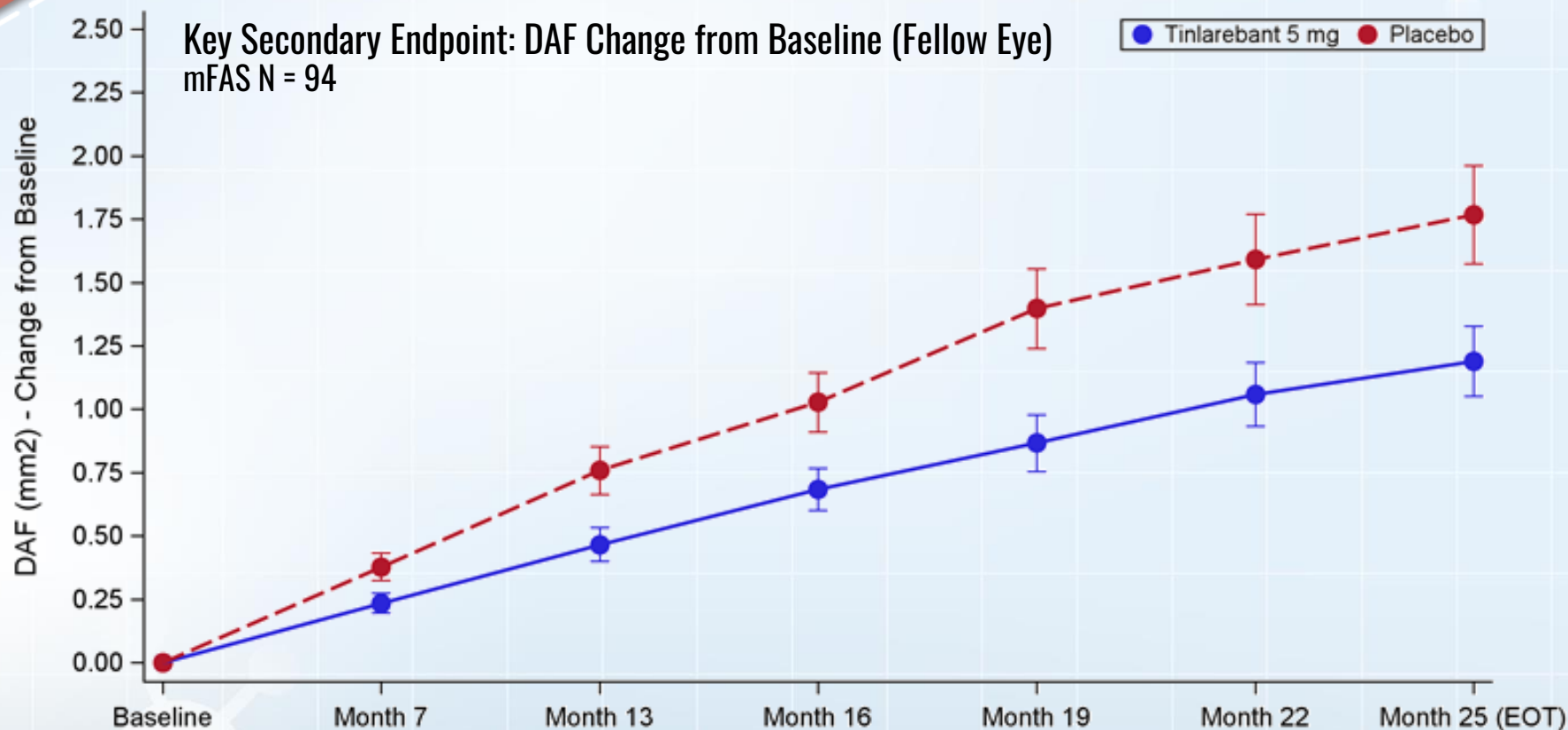
Tinlarebant slowed DDAF lesion growth in the fellow eye by 33.6% compared to placebo (P = 0.041)

TINLAREBANT SLOWED DAF LESION GROWTH, THE KEY SECONDARY ENDPOINT, IN THE STUDY EYE BY 33.7%



**Tinlarebant slowed DAF lesion growth by 33.7%
compared to placebo (P = 0.027)**

TINLAREBANT SLOWED DAF LESION GROWTH, THE KEY SECONDARY ENDPOINT, ALSO IN THE FELLOW EYE BY 32.7%



Tinlarebant slowed DAF lesion growth in the fellow eye by 32.7% compared to placebo (P = 0.017)

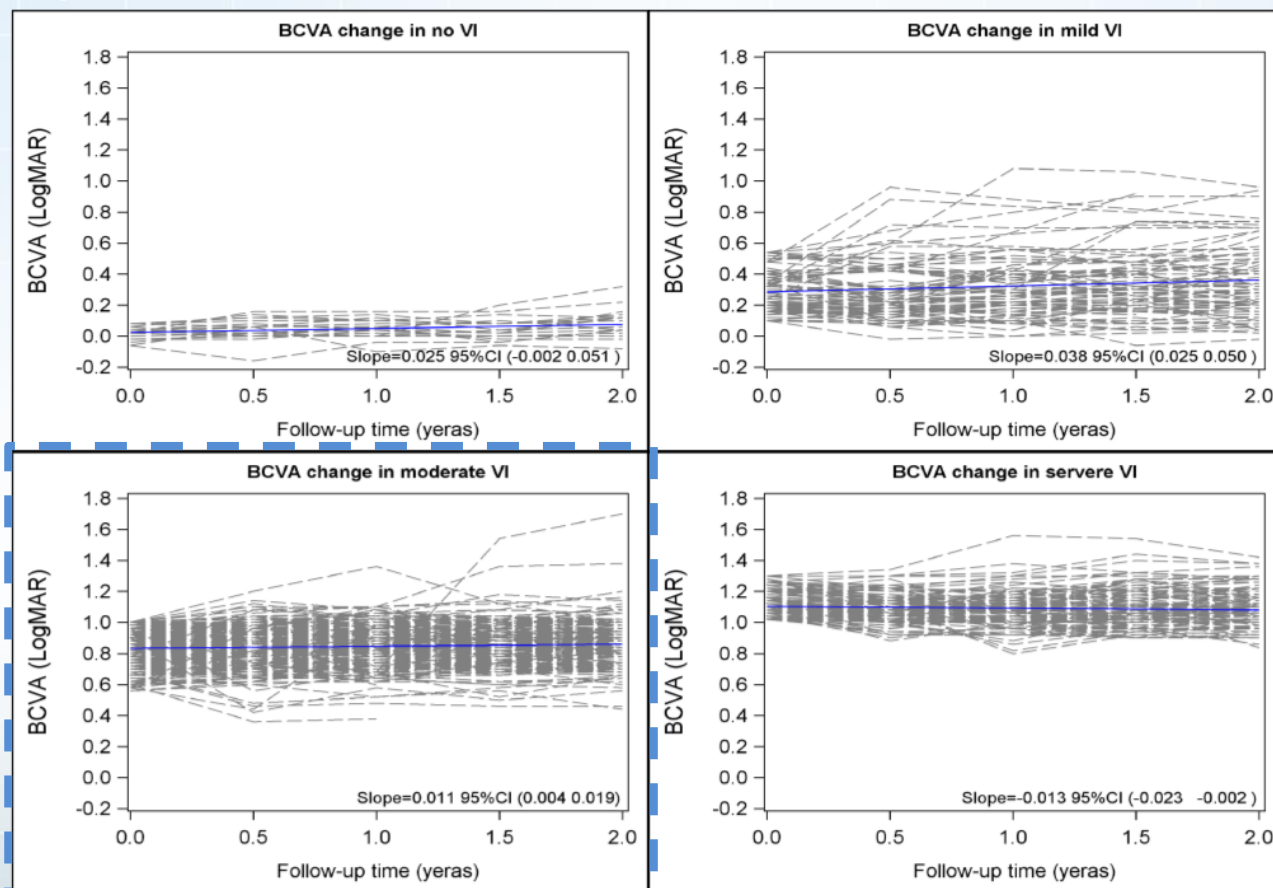
AS EXPECTED, BCVA IN STUDY EYE DID NOT SHOW ANY SIGNIFICANT CHANGE

	Tinlarebant	Placebo
BCVA at Baseline	39.9	39.4
BCVA at EOS	39.7	40.0

- The overall change of visual acuity was minimal over the period of 24 months in both treatment groups
- Test–retest variability for ETDRS change scores in Stargardt disease are known to yield a repeatability coefficient ≈ 8 letters ⁽¹⁾
- Such minor changes in average visual acuity over two years are in line with the natural history of Stargardt disease and were observed in the ProgStar Study

(1) Parker MA, Choi D, Erker LR, Pennesi ME, Yang P, Chegarnov EN, Steinkamp PN, Schlechter CL, Dhaenens CM, Mohand-Said S, Audo I, Sahel J, Weleber RG, Wilson DJ. Test-Retest Variability of Functional and Structural Parameters in Patients with Stargardt Disease Participating in the SAR422459 Gene Therapy Trial. Transl Vis Sci Technol. 2016 Oct 1;5(5):10.

PROGSTAR: VISUAL ACUITY CHANGE OVER 24 MONTHS PROSPECTIVE COHORT (N=434)



- Overall rate of BCVA loss was 0.55 letters/year over two years
- BCVA of eyes with baseline BCVA between 20/70 and 20/200 declined at a rate of 0.6 letters/year



SAFETY RESULTS



TINLAREBANT DEMONSTRATED A WELL TOLERATED SAFETY PROFILE

SAFETY SET N = 104

Subjects Who Experienced at Least One Non-Ocular Treatment-Emergent Adverse Events (TEAE), N / (%)

Category	Tinlarebant 5mg (N=69)	Placebo (N=35)
TEAE	59 (85.5%)	27 (77.1%)
Severe TEAE	2 (2.9%)	1 (2.9%)
Serious TEAE	2 (2.9%)	4 (11.4%)
Study Drug-Related TEAE	14 (20.3%)	4 (11.4%)
Study Drug-Related Serious TEAE	0 (0.0%)	0 (0.0%)
TEAE Leading to Study Drug Discontinuation	0 (0.0%)	0 (0.0%)
TEAE Leading to Study Discontinuation	0 (0.0%)	0 (0.0%)
TEAE Leading to Death	0 (0.0%)	0 (0.0%)

- Total of 6 serious adverse events (SAEs) reported in the study – all events were non-ocular, with 4 assessed as unrelated and 2 assessed as unlikely related to the study treatment
- Most reported Non-Ocular adverse events (AEs): Nasopharyngitis (all cases were assessed as unrelated/unlikely related to treatment), Headache, and Acne – most events were mild and resolved during the study period



THE MAJORITY OF OCULAR ADVERSE EVENTS WAS MILD

SAFETY SET N = 104

Subjects Who Experience at Least One Ocular TEAE, N / (%)

Category	Tinlarebant 5mg (N=69)	Placebo (N=35)
TEAE	53 (76.8%)	8 (22.9%)
Severe TEAE	2 (2.9%)	0 (0.0%)
Serious TEAE	0 (0.0%)	0 (0.0%)
Study Drug-Related TEAE	49 (71.0%)	8 (22.9%)
Study Drug-Related Serious TEAE	0 (0.0%)	0 (0.0%)
TEAE Leading to Study Drug Discontinuation	4 (5.8%)	0 (0.0%)
TEAE Leading to Study Discontinuation	2 (2.9%)	0 (0.0%)
TEAE Leading to Death	0 (0.0%)	0 (0.0%)

- Most reported Ocular AEs: Xanthopsia, Delayed dark adaptation, and Night Vision Impairment
- The majority of the events were mild, and most resolved while on study
- There were no serious ocular TEAEs – 4 TEAEs lead to study drug discontinuation and 2 TEAEs lead to study discontinuation

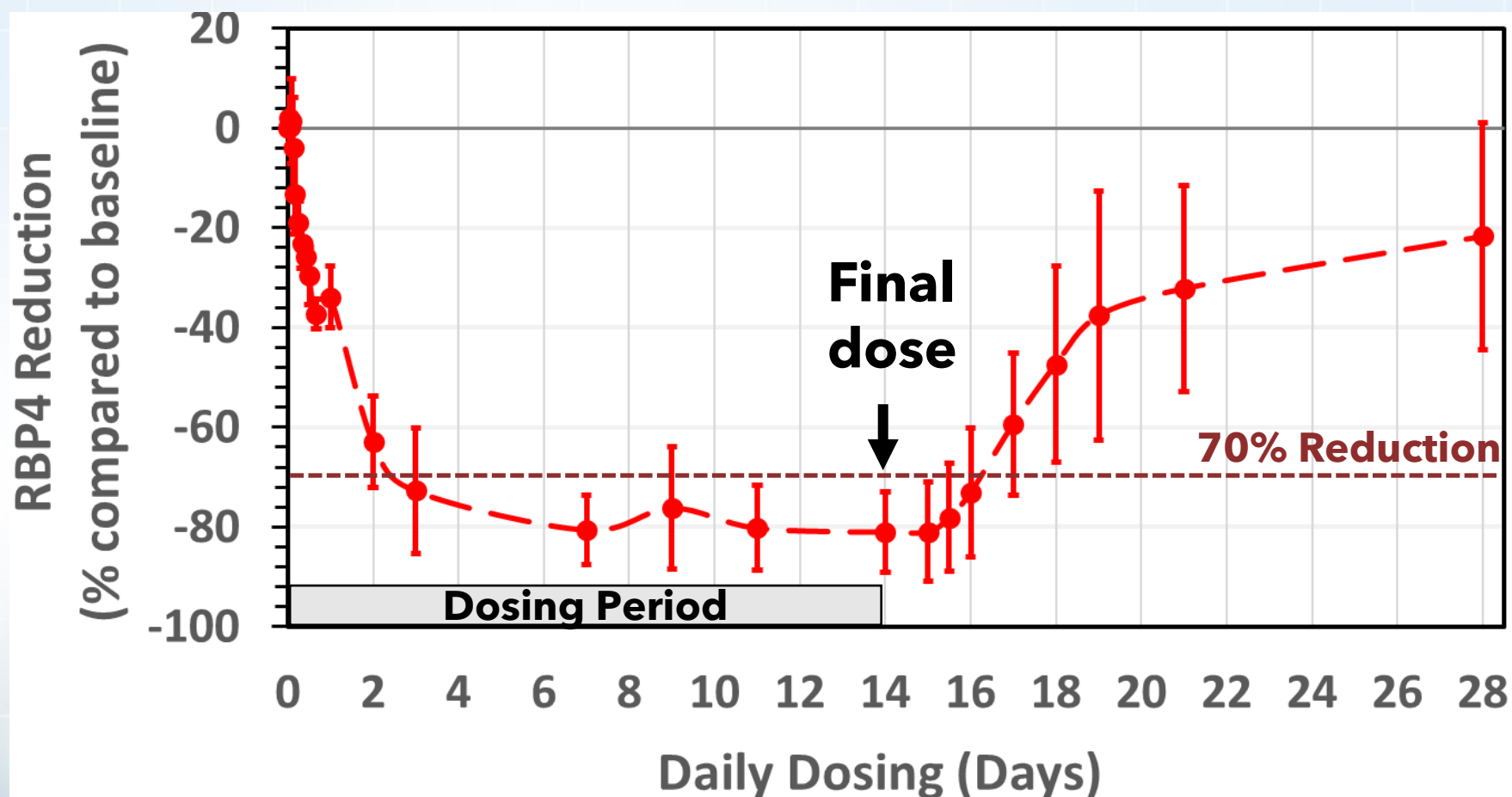


PHASE 3 PHOENIX TRIAL IN GEOGRAPHIC ATROPHY

LBS
008

TINLAREBANT: $\geq 70\%$ REDUCTION OF RBP4

Phase 1, 5mg Daily Dosing in Healthy Adults: Mean Percent Reduction of RBP4 (excludes placebo)





CLINICAL TRIAL DESIGN OVERVIEW IN GA

- **Established Efficacy Endpoint** – Reduction in atrophic lesion growth rate as measured by retinal imaging is the FDA accepted primary endpoint
- **Early Intervention** – Targeting patients with small lesion size to potentially slow disease progress at an early stage
- **Oral Once a Day Treatment** – well suited for long term treatment for chronic diseases
- **Broad Potential** – Primary focus on GA; potential to treat earlier stages (e.g., intermediate AMD)

	GA Phase 3 “PHOENIX” ⁽¹⁾
Enrollment	530 subjects
Sites	Global
Masking	Double Blind
Placebo	2:1 ratio (Tinarebant : Placebo)
Treatment duration	2 years
Primary measures	Slowing of atrophic lesion growth, safety & tolerability
Other measures	BCVA, SD-OCT, microperimetry
Interim analysis	Yes

⁽¹⁾ Additional Phase 3 study expected to be required prior to NDA filing



TINLAREBANT SUMMARY

SUMMARY OF RESULTS OF THE DRAGON TRIAL

- **The DRAGON trial met its primary endpoint:** A statistically significant slowing in DDAF lesion growth was observed in subjects treated with 5 mg/day oral Tinalarebant as compared to placebo
- **The treatment effect was 36%** and must be considered **clinically meaningful**
- The observed treatment effect was **supported by the fellow eye data** and the **key secondary endpoint: a reduction of DAF area growth**
- The **change in best-corrected visual acuity was minimal** in both the treatment and the placebo group – and is **in-line with natural history data**
- The biomarker of tinalarebant treatment, **RBP4 reduction, showed a sustained 80% reduction with very little variability**
- **Tinalarebant (5 mg p.o., daily) was well tolerated** in adolescent STGD1 patients

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TINLAREBANT HAS THE POTENTIAL TO BE THE FIRST-EVER APPROVED TREATMENT FOR STARGARDT DISEASE

- First-ever oral therapy in a retinal degenerative disease to demonstrate a **clinically meaningful slowdown of neurodegeneration**
- **36% reduction in DDAF lesion growth rate**, representing a robust and reproducible treatment effect in Stargardt disease
- **Excellent safety and tolerability profile** across two years of treatment
- **Addresses the root pathogenic mechanism** (bisretinoid accumulation), offering a rational, disease-modifying approach where no approved therapies currently exist
- **Broad applicability across disease stages**, from early *ABCA4*-mediated changes to more advanced atrophy
- **Personal clinical impact:** After >20 years caring for these patients, this represents a true game changer – a therapy I would confidently offer to all my Stargardt patients*

* Quoted from Dr. Hendrik Scholl, Chief Medical Officer of Belite Bio, based on his personal experience as an ophthalmologist treating STGD1 patients.



Lin BioScience

Bringing Hope to Incurable Diseases

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