BIOAGE

R&D Update
May 8, 2026

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Welcome & Introductions

Management attendees

Kristen Fortney, PhD
Co-Founder & CEO

Paul Rubin, MD
CMO & EVP Research

Dov Goldstein, MD, MBA
CFO

BIOAGE

Guest presenters

Matthias Geyer, PhD
Director, Institute of Structural Biology

University of Bonn

Michael Davidson, MD
Clinical Professor of Medicine
Director, Lipid Clinic

University of Chicago
Pritzker School of Medicine

Brian Hafler, MD, PhD
Associate Professor
Ophthalmology & Visual Science

Yale School of Medicine

David Boyer, MD
Senior Partner

Retina-Vitreous Associates Medical Group

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Introduction		**Kristen Fortney, PhD** *Co-Founder & CEO*	
BGE-102			
Structural biology insights		**Matthias Geyer,** PhD *University of Bonn*	
Phase 1 results		**Paul Rubin,** MD *CMO & EVP Research*	
Cardiovascular		**Michael Davidson,** MD *University of Chicago*	
Ophthalmology	**Paul Rubin,** MD *CMO & EVP Research*	**Brian Hafler,** MD, PhD *Yale University*	**David Boyer,** MD *Retina-Vitreous Associates*
Closing remarks		**Kristen Fortney, PhD** *Co-Founder & CEO*	
Q&A		**Management Team** **Michael Davidson, MD** **Brian Hafler, MD, PhD**	

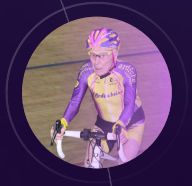
BIOAGE

Introduction

Kristen Fortney, PhD
Co-Founder & CEO

BIOAGE

We are harnessing the biology of human aging to develop new therapies for cardiometabolic diseases



The BioAge discovery platform: from human data to therapeutics for metabolic aging

Validated platform: ongoing partnerships with Novartis & Lilly to discover drugs and drug targets

>150M molecular data points: one of the world's largest collections of longitudinal human aging data and functional outcomes

BGE-102: oral brain-penetrant NLRP3 inhibitor

Potential "pipeline in a pill" targeting efficacy in-line with injectable anti-inflammatories

CV risk: potential best-in-class profile for hsCRP reduction

- 86% reduction in hsCRP in obese subjects
- 87-93% of subjects achieved normalized hsCRP <2 mg/L

Ophthalmology: therapeutic retinal exposure enables oral treatment of diseases including DME, where intravitreal anti-IL-6 has shown benefit

Anticipated catalysts: CV risk POC H2:2026, DME POC mid-2027

APJ agonism: exercise mimetic for obesity

Obesity: potential to double weight loss & fully restore body composition when combined with an incretin in preclinical models

Anticipated catalysts: IND submission 2026 YE

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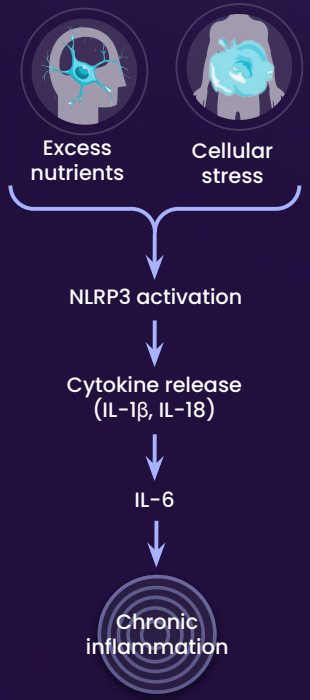
Chronic NLRP3 activity drives disease & predicts poor human longevity

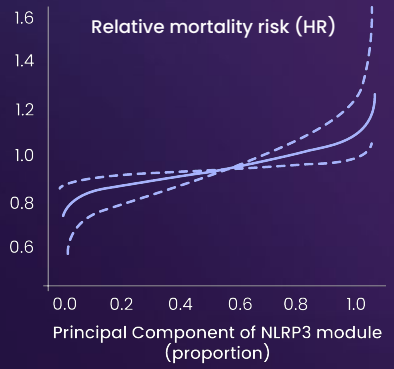
NLRP3 overview

- Normal NLRP3 function: innate immune response to danger signals
- In pathology: cellular stress & nutrient excess stimulates chronic activation
- Resulting chronic inflammation drives a range of diseases



NLRP3 in human longevity & disease

Reduced NLRP3 activity is associated with longevity



Strong human genetic evidence for NLRP3 in cardiometabolic disease

Mendelian randomization: NLRP3 levels strongly predictive of heart failure (↑ 1 SD expression = up to ↑ 70% risk)

GoF mutations ↓ lean mass & body composition ↑ atherosclerosis

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Our lead program, BGE-102, is well positioned to address diseases driven by inflammation in both the CNS and the periphery

Key attributes

Potential best-in-class potency based on Phase 1 trial results

1.8 nM IC_{90} by human ex vivo whole blood stimulation

24h IC_{90} coverage at 60 mg QD provided 24-hour IL-1β suppression ≥90%

86% hsCRP reduction; 87-93% of subjects achieved normalized hsCRP (<2 mg/L)*
in line with injectable anti-IL-6 drugs

CNS penetrant
~0.7 Kp,uu_{CSF}
(120 mg MAD, day 14)

Attractive safety & tolerability
All AEs to date mild / moderate, self-limited, with no dose dependency

50-97x safety margin for 60 mg dose**
based on 3-month GLP tox

Strong IP position
2045 composition of matter & claims for novel NLRP3 binding site

*Note: * in MAD cohorts - obese subjects with baseline hsCRP > 3 mg/L; ** based on Phase 1 healthy volunteer MAD at Day 14*

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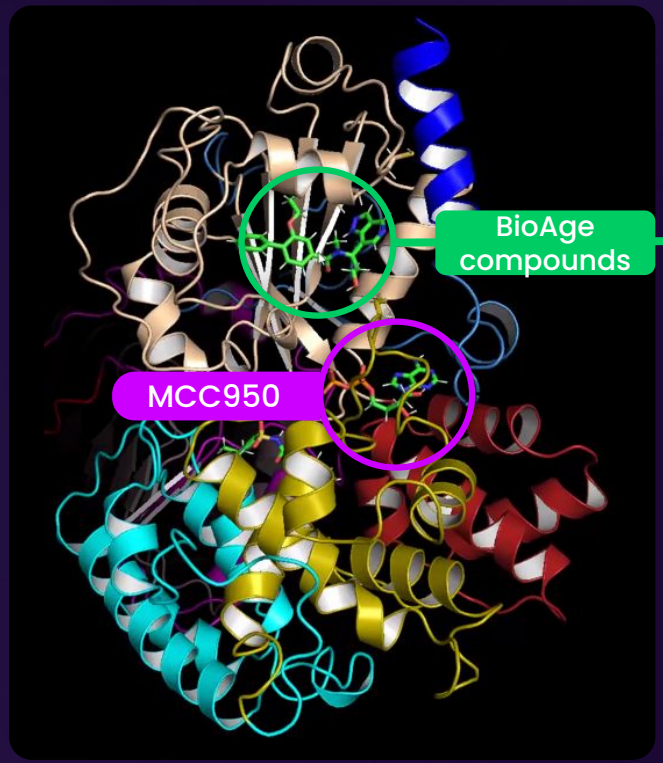
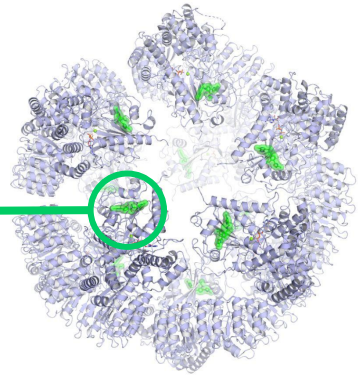
BioAge NLRP3 inhibitors: unique, patented binding site & novel mechanism

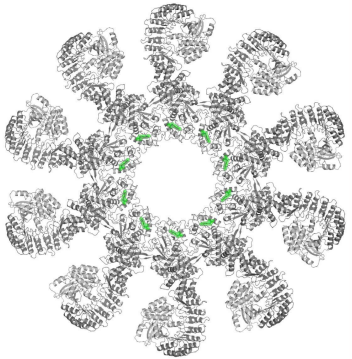
Unique binding site



Our inhibitors bind both the active & inactive inflammasome, unlike other NLRP3 inhibitors



Inactive decamer



Active inflammasome

Discovery of potent and selective inhibitors of human NLRP3 with a novel mechanism of action

JEM Journal of Experimental Medicine

The discovery of novel and potent indazole NLRP3 inhibitors enabled by DNA-encoded library screening

Bioorganic & Medicinal Chemistry Letters

Inhibition of NLRP3 by a CNS-penetrating indazole scaffold

bioRχiv

Collaboration with Matthias Geyer, PhD, Director, Institute of Structural Biology University of Bonn

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NLRP3

Structural biology insights

Phase 1 results

Cardiovascular

Ophthalmology

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Matthias Geyer, PhD

Director, Institute of Structural Biology
University of Bonn

A new way to inhibit NLRP3

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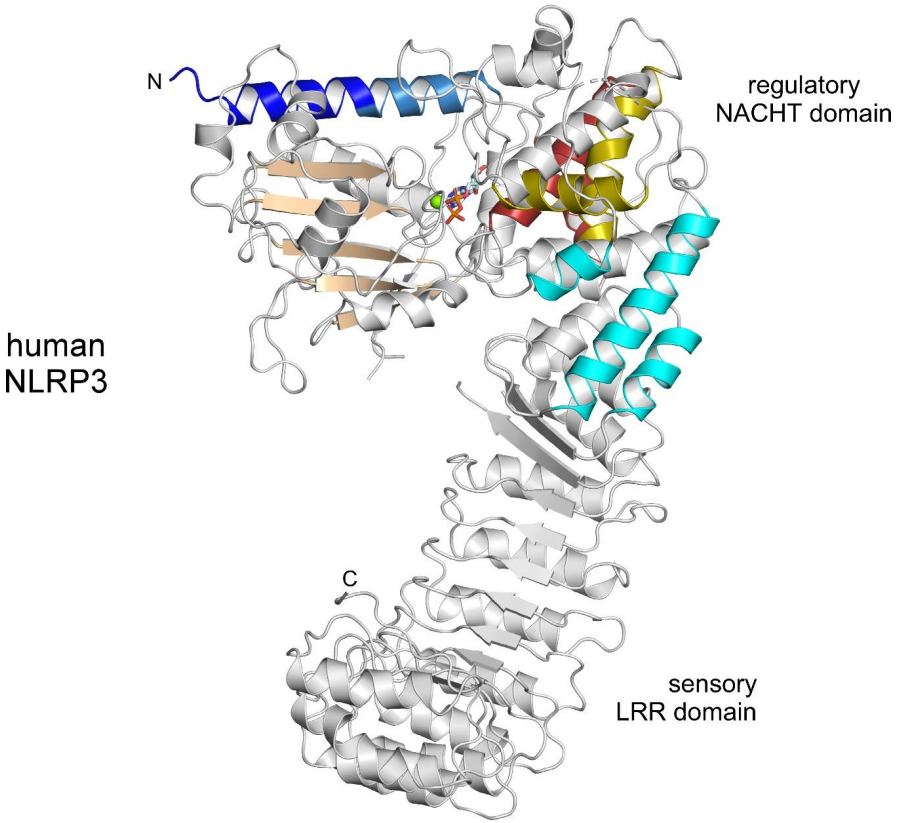
BGE-102 targets a different site on NLRP3 than other inhibitors in development



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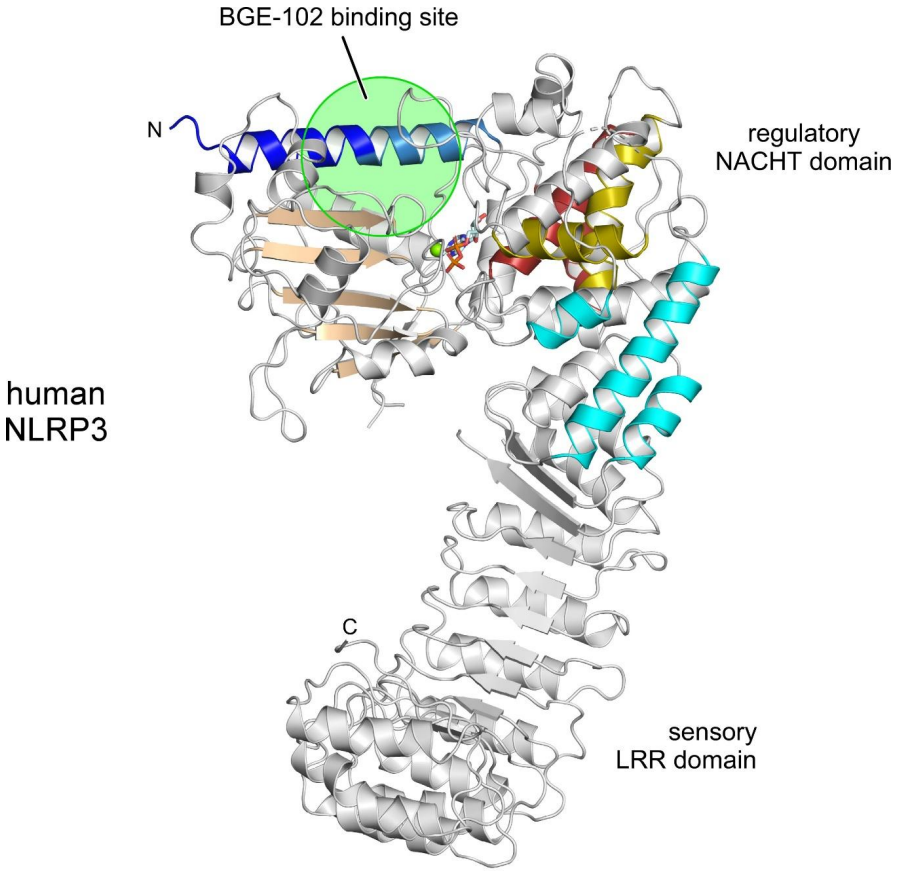

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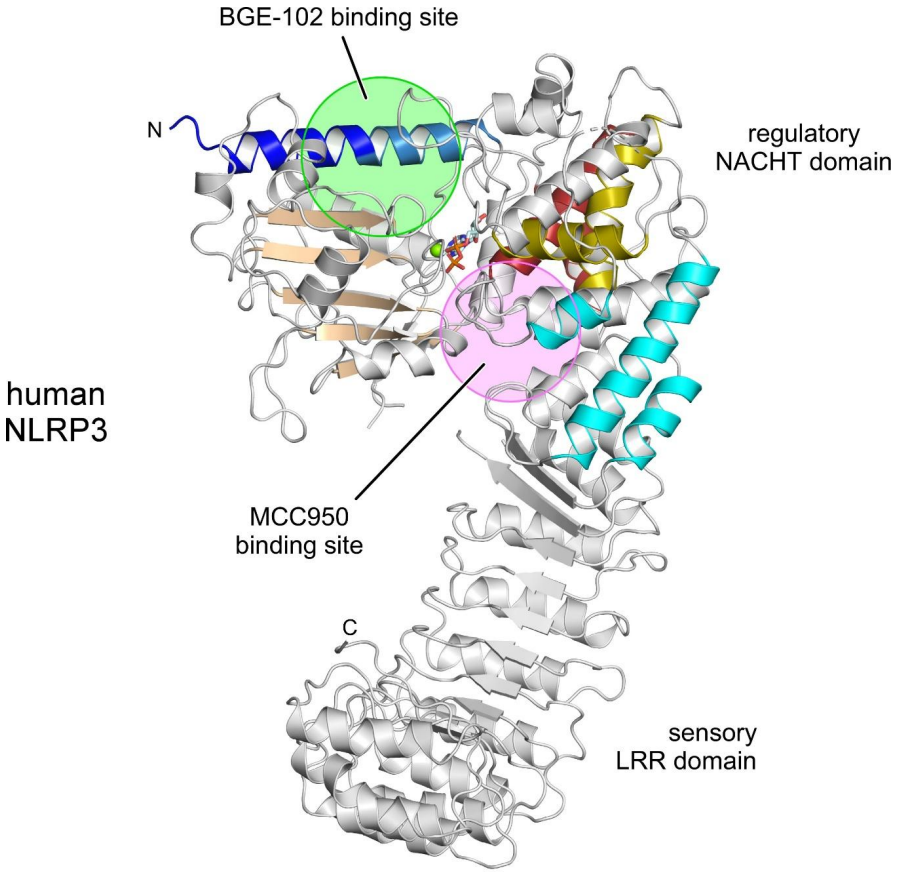

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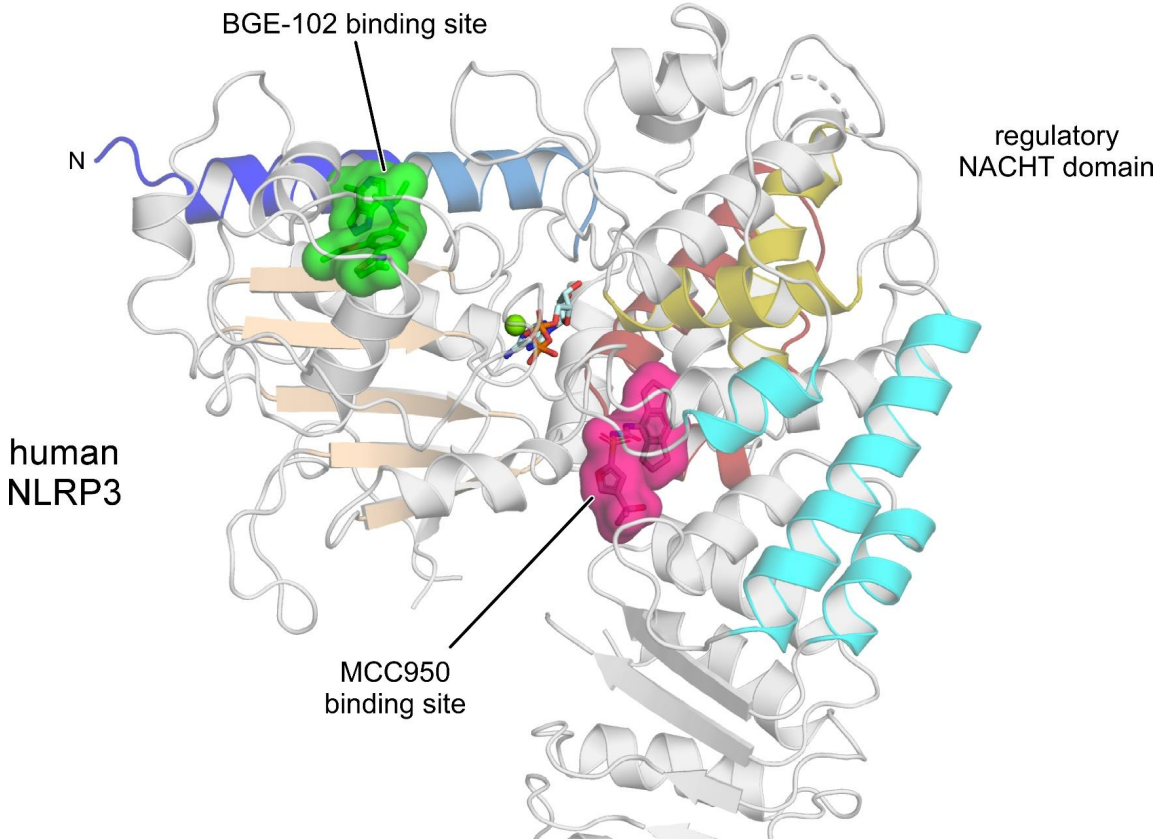
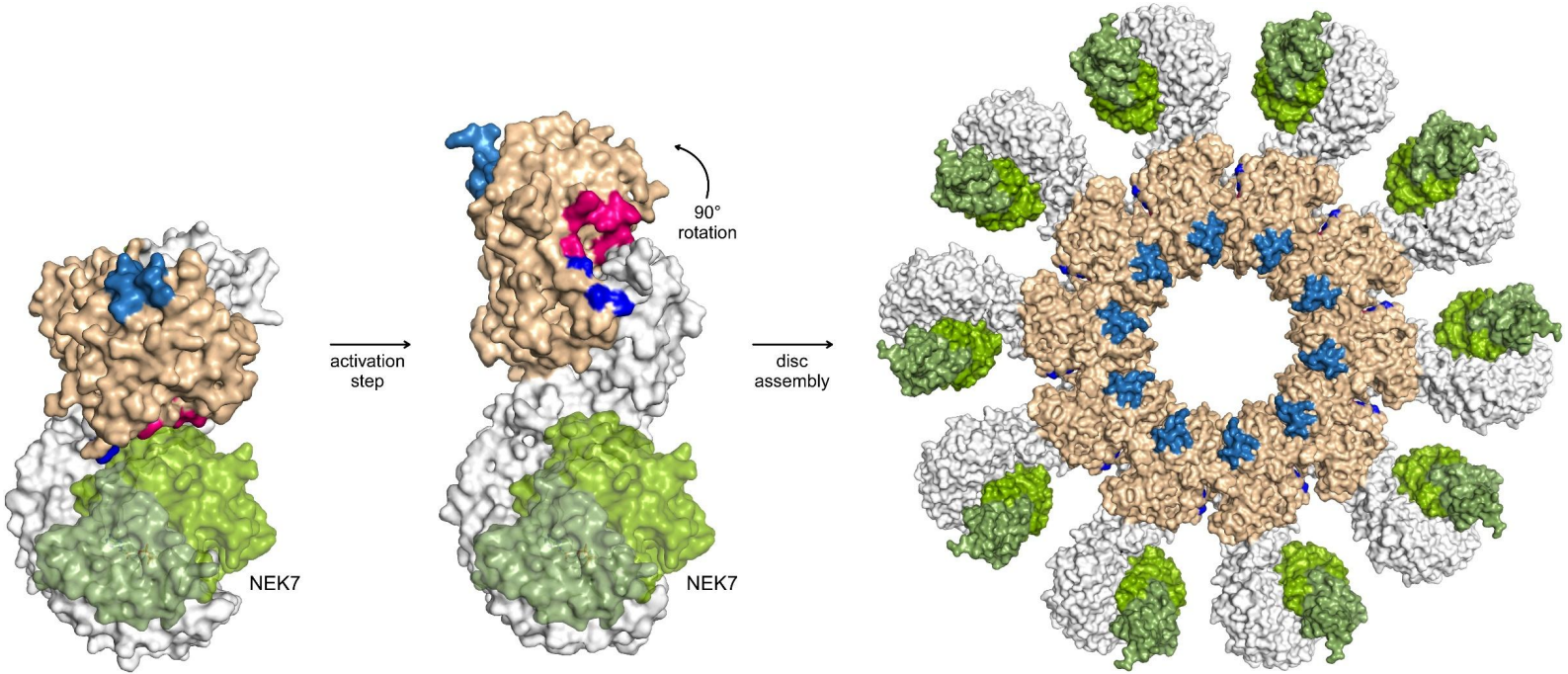


BGE-102 inhibits both forms of NLRP3 – the resting state and the active inflammasome



NLRP3•ADP

inactive

NLRP3•ATP

active

NLRP3•ATP inflammasome

signaling-competent assembly

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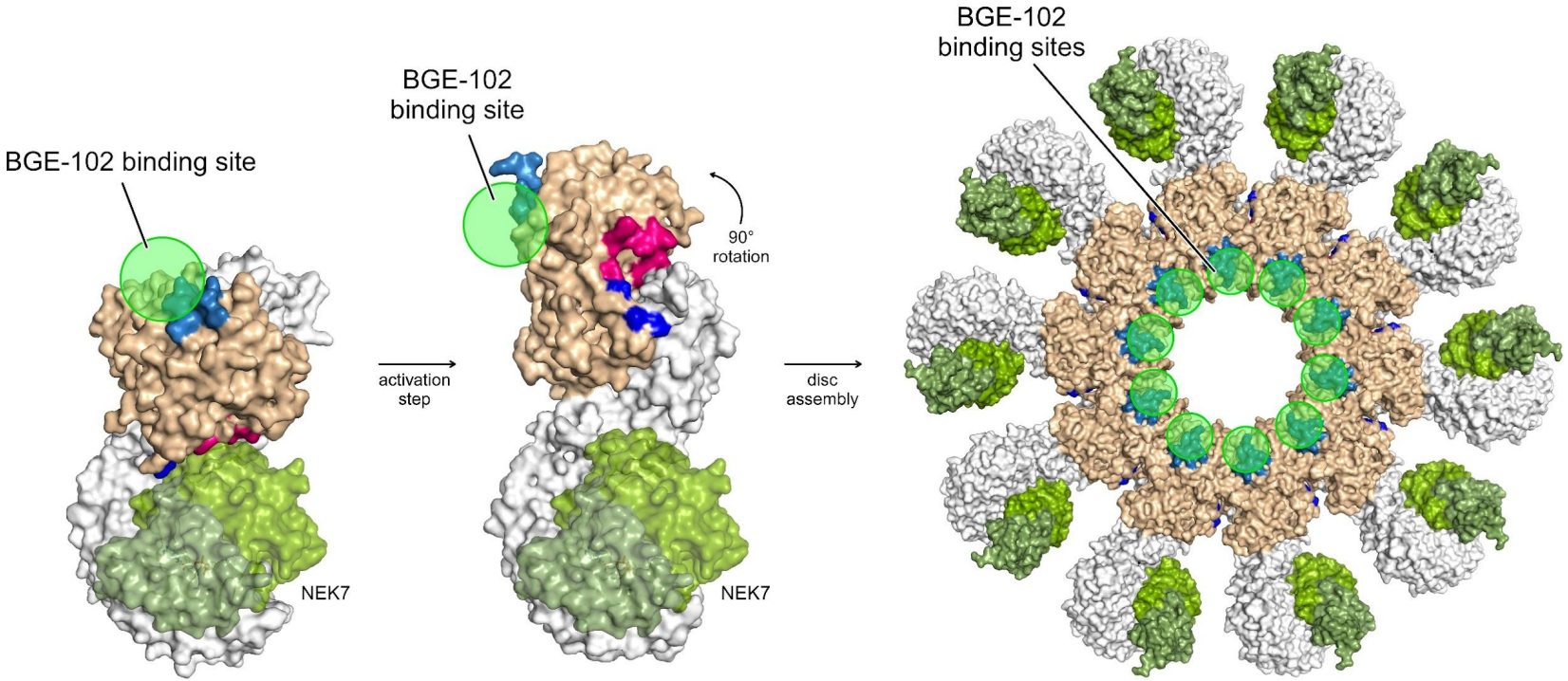


NLRP3•ADP

inactive

NLRP3•ATP

active

NLRP3•ATP inflammasome

signaling-competent assembly

BGE-102 is selective for NLRP3

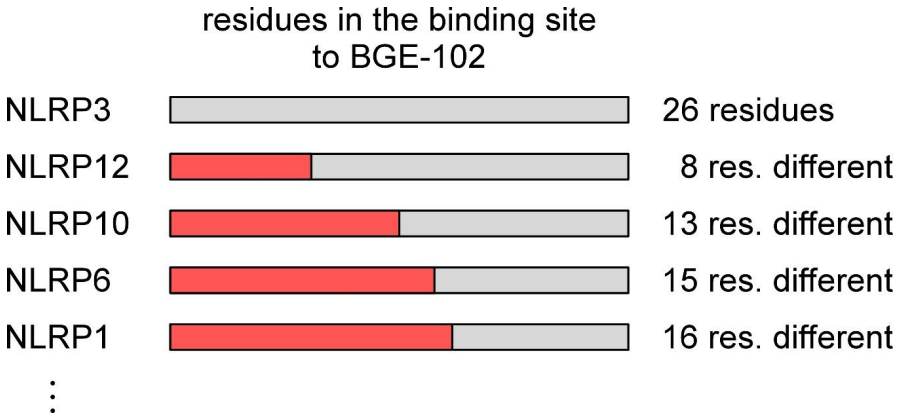
Subfamily	Protein	Function	Domain architecture
NLRP	NLRP1	inflammasomal signaling	
	NLRP2,-7,-9	inflammasomal signaling, embryonic development	
	NLRP3	inflammasomal signaling	
	NLRP5	embryonic development	
	NLRP6	inflammasomal signaling, NF-κB and MAPK signaling inhibition, type I IFNs	
	NLRP8,-13	unknown	
	NLRP10	NF-κB regulation	
	NLRP11	NF-κB and MAPK signaling inhibition	
	NLRP12	inflammasomal signaling, NF-κB and MAPK signaling inhibition	
	NLRP14	spermatogenesis	

Subfamily	Protein	Function	Domain architecture
NLRA	CIITA	MHC II expression	
NLRB	NAIP	T3SS and flagellin recognition	
NLRC	NOD1	NF-κB and MAPK signaling, autophagy	
	NOD2	NF-κB and MAPK signaling, autophagy, type I IFN	
	NLRC3	negative regulator of T-cell activation	
	NLRC4	inflammasomal signaling with NAIP	
	NLRC5	MHC I expression, antiviral response	
	NLRX1	antiviral response, type I IFN inhibition, ROS regulation, autophagy	

There are 22 human NOD-like receptors

BGE-102 is selective for NLRP3

The BioAge compound binding site is in pocket of NLRP3 that is different from every other inflammasome



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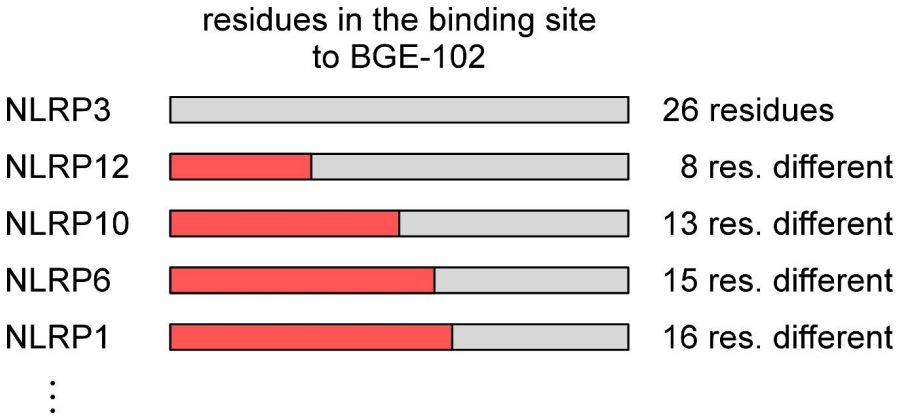
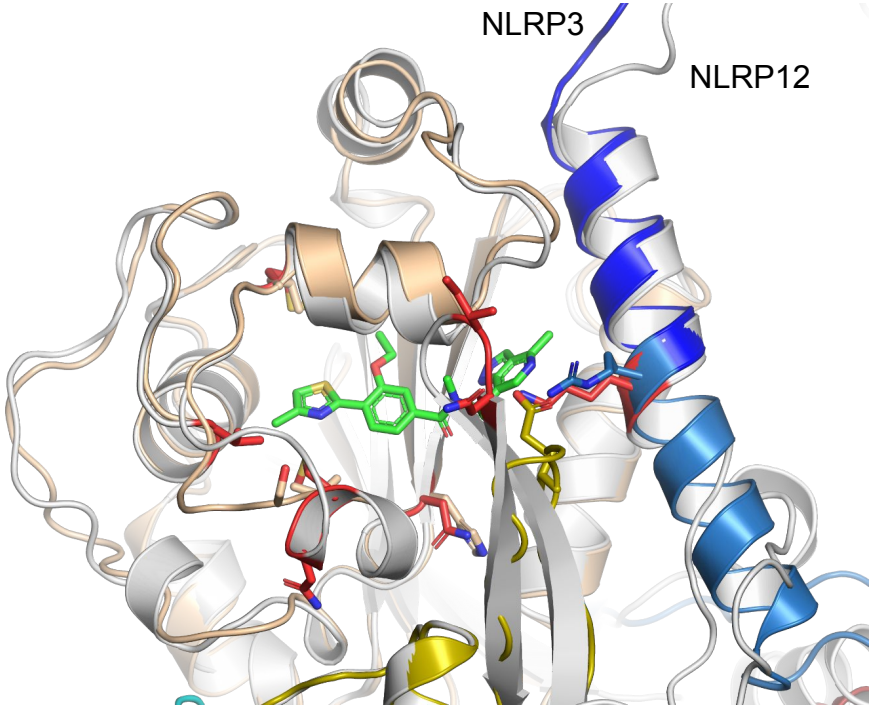
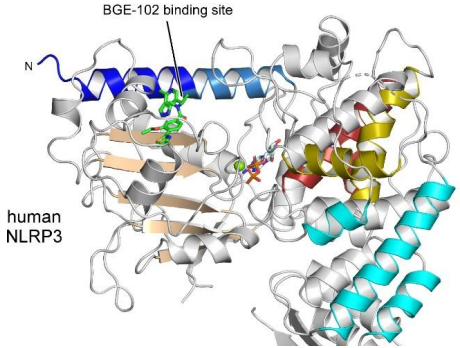


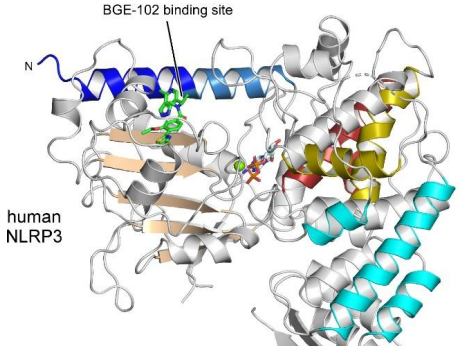


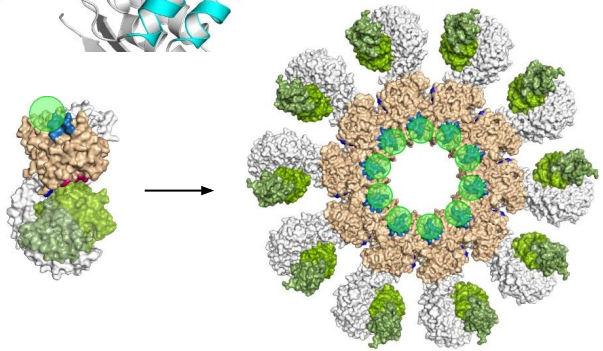
Three structural features set BGE-102 apart



The BGE-102 binding site on NLRP3 is **unique** from every other inhibitor in the clinic

Three structural features set BGE-102 apart

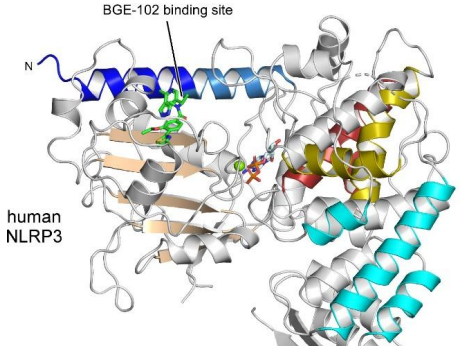




The BGE-102 binding site on NLRP3 is **unique** from every other inhibitor in the clinic

The BGE-102 binding site is accessible in the **resting** and **active state** of NLRP3

Three structural features set BGE-102 apart



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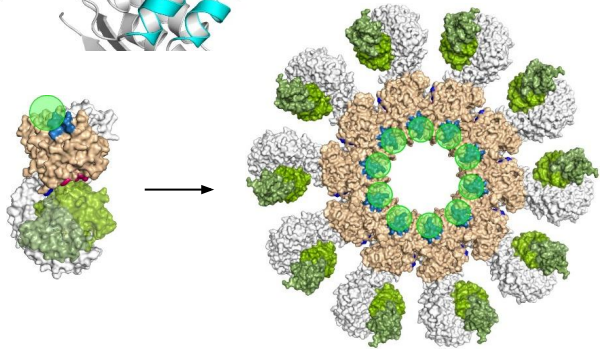

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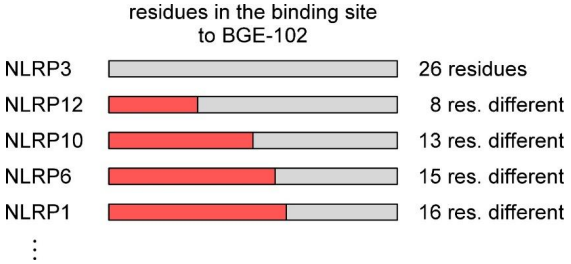

The BGE-102 binding site is **selective for NLRP3 alone** within the inflammasome family

NLRP3

Structural biology insights

Phase 1 results

Cardiovascular

Ophthalmology

Paul Rubin, MD
CMO & EVP Research

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PHASE 1 RESULTS

Phase 1 clinical trial design

	Single Ascending Dose HVs & obese	**Multiple Ascending Dose** HVs - 14 days of dosing	**Multiple Ascending Dose** Obese - 14-21 days of dosing
Objectives	Safety & tolerability, pharmacokinetics, pharmacodynamics (IL-1β, hsCRP)		
Subjects	HVs (N=36) & obese subjects (N=9)	HVs (N=18)	Obese subjects with hsCRP > 3 mg/L (N=41)
Design	5 cohorts x  HVs: 10 mg | 30 mg | 60 mg | 120 mg Obese: 60 mg (fed / fasted)	2 cohorts x  60 mg | 120 mg	60 mg  21 days 120 mg  14 days

Note: HVs = healthy volunteers *active* *placebo*
All cohorts conducted in a clinical trial unit; for post-baseline visits, window +/- 1 day

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PHASE 1 RESULTS

BGE-102 met key trial objectives

Safety / tolerability

- Well tolerated with all mild / moderate, self-limited AEs
- No dose-limiting toxicities

Pharmacokinetics

- Dose-proportional exposure with $T_{1/2}$ supporting QD dosing

Pharmacodynamics

After 14 days of dosing at 60 mg QD in healthy volunteers:

- ≥90% suppression of IL-1β for 24 hours
- Drug levels in CSF > IC_{90}

Efficacy

After 21 days of dosing at 60 mg QD in obese subjects:

- 86% reduction in hsCRP
- 87% of subjects achieved normalized hsCRP (<2 mg/L)

After 14 days of dosing at 120 mg QD in obese subjects:

- 86% reduction in hsCRP
- 93% of subjects achieved normalized hsCRP (<2 mg/L)

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PHASE 1 RESULTS

BGE-102 was well tolerated

- Only mild / moderate treatment-emergent AEs (TEAEs); all self-limited with no dose dependency
- No serious AEs
- No TEAEs leading to discontinuation
- No clinically meaningful adverse changes in vital signs, laboratory values, or ECGs

TEAEs	All BGE-102 (N=82)	All placebo (N=22)
Subjects with at least 1 AE	50 (61.0%)	13 (59.1%)
Subjects with mild TEAEs	49 (59.8%)	12 (54.5%)
Subjects with moderate TEAEs	8 (9.8%)	3 (13.6%)

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PHASE 1 RESULTS

Obese MAD cohorts: baseline characteristics

Baseline characteristic	60 mg obese MAD (N=19)	120 mg obese MAD (N=14)	Obese placebo (N=8)
Age, years, mean (SD)	41.7 (9.8)	39.4 (9.8)	38.8 (6.7)
Male, n (%)	7 (36.8)	8 (57.1)	2 (25.0)
Female, n (%)	12 (63.2)	6 (42.9)	6 (75.0)
Race, n (%)			
White	10 (52.6)	11 (78.6)	2 (25.0)
Black or African American	6 (31.6)	2 (14.3)	3 (37.5)
American Indian or Alaska Native	1 (5.3)	0	0
Native Hawaiian or Other Pacific Islander	1 (5.3)	0	0
Multiple	1 (5.3)	1 (7.1)	3 (37.5)
Weight, kg, mean (SD)	99.9 (12.7)	99.0 (13.3)	107.2 (8.7)
BMI, kg/m^2, mean (SD)	34.9 (2.9)	35.1 (2.4)	36.0 (3.3)
hsCRP (mg/L), median (IQR)	6.30 (2.95; 7.80)	4.85 (3.68; 6.40)	5.35 (3.33; 7.10)
IL-6 (pg/mL), median (IQR)	2.23 (1.75; 2.65)	2.33 (1.88; 3.06)	1.85 (1.47; 2.55)

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PHASE 1 RESULTS

Obese MAD cohorts: safety and tolerability

TEAEs	60 mg obese MAD (N=19)	120 mg obese MAD (N=14)	Obese placebo (N=8)
Subjects with any related TEAE, n (%)	5 (26.3%)	2 (14.3%)	1 (12.5%)
Subjects with TEAE leading to discontinuation, n	0	0	0
Subjects with SAE or severe TEAE, n	0	0	0
TEAE: neutropenia, thrombocytopenia, or infections and infestations, n	0	0	0

The only related adverse event occurring in >1 subject on active treatment across pooled MAD cohorts (healthy volunteers and obese) was headache, which occurred in 6 subjects (12.8%)

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- Structural biology insights
- **Phase 1 results**
  - **Healthy volunteer SAD / MAD**
  - Obese MAD
- Cardiovascular
- Ophthalmology

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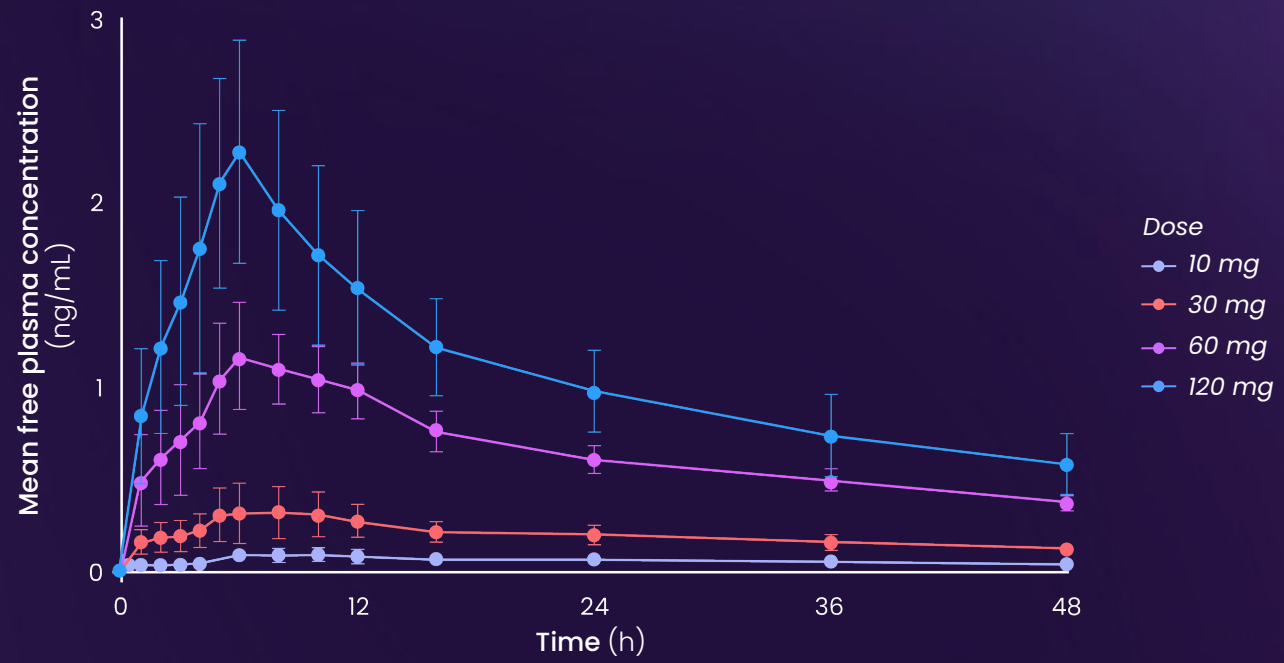
HEALTHY VOLUNTEER SAD / MAD

Plasma PK: dose-proportionality observed in SAD cohorts

SAD cohorts: mean plasma PK

BGE-102 and active metabolite* free concentration



*Note: * combined concentration of BGE-102 and its sole active metabolite M1, which acts as an NLRP3 inhibitor of comparable potency. M1 has been previously shown to have a large safety margin in rat & dog toxicology studies.*

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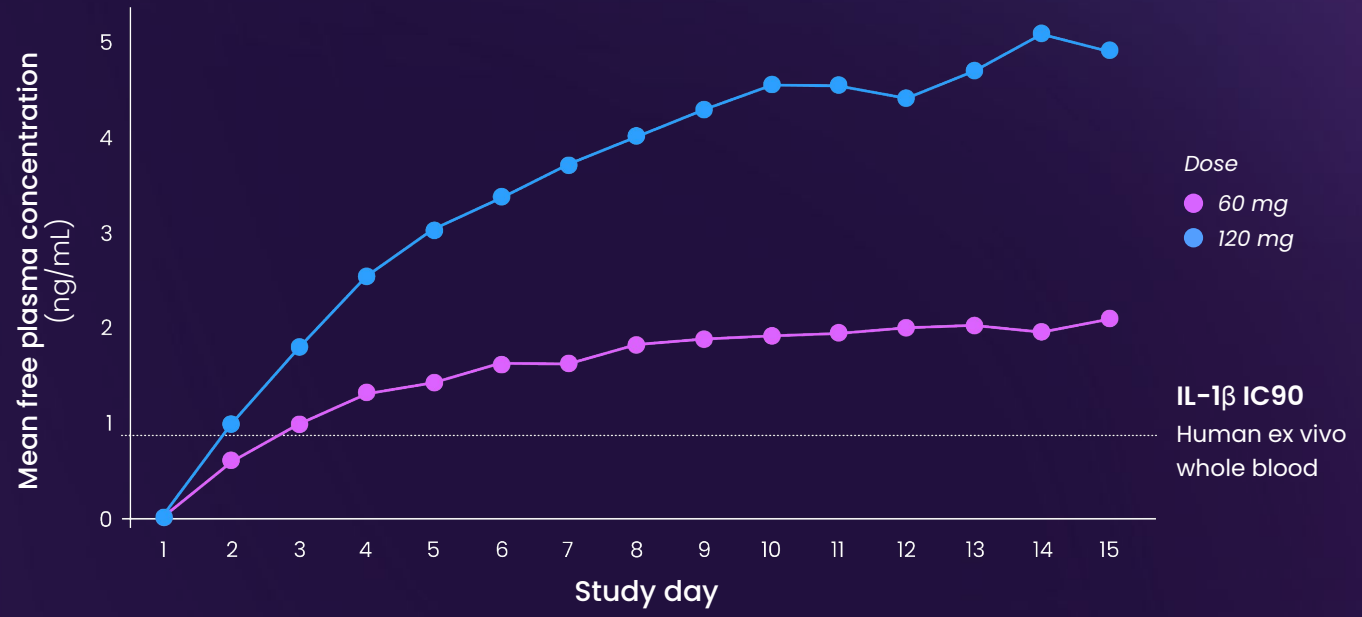
HEALTHY VOLUNTEER SAD / MAD

Plasma PK: MAD cohorts showed accumulation out to 14 days with near steady-state levels above the human IC_{90} for IL-1β inhibition

MAD cohorts: mean plasma PK at trough (pre-dose)

BGE-102 and active metabolite free concentration



*Note: * IC90 calculated from first 24 hours of treatment of healthy volunteers in SAD & MAD cohorts*

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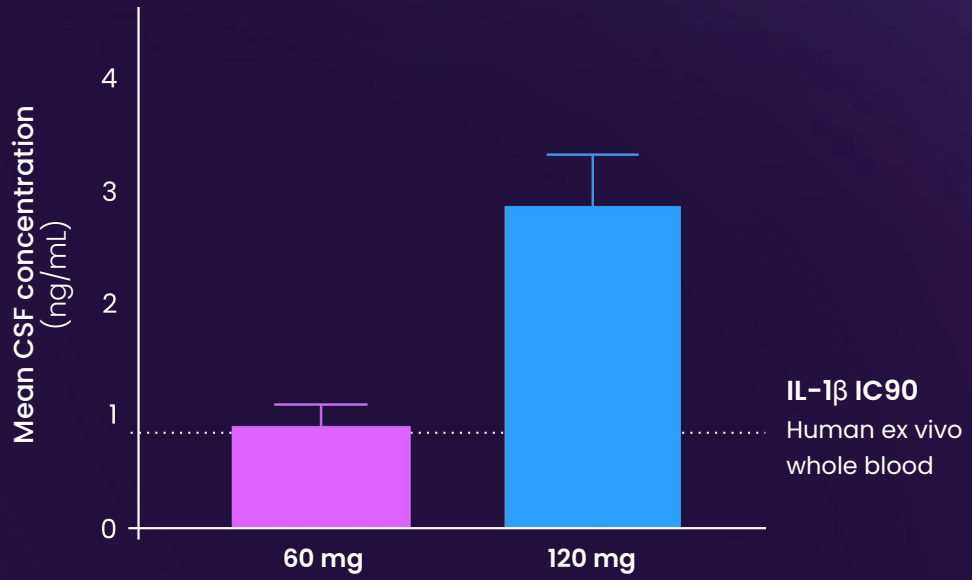
HEALTHY VOLUNTEER SAD / MAD

CSF PK: doses of 60 mg and above exceeded human IC_{90} at near steady state

MAD cohorts: Day 14 CSF PK

BGE-102 and active metabolite, 11-13 hours post-dose



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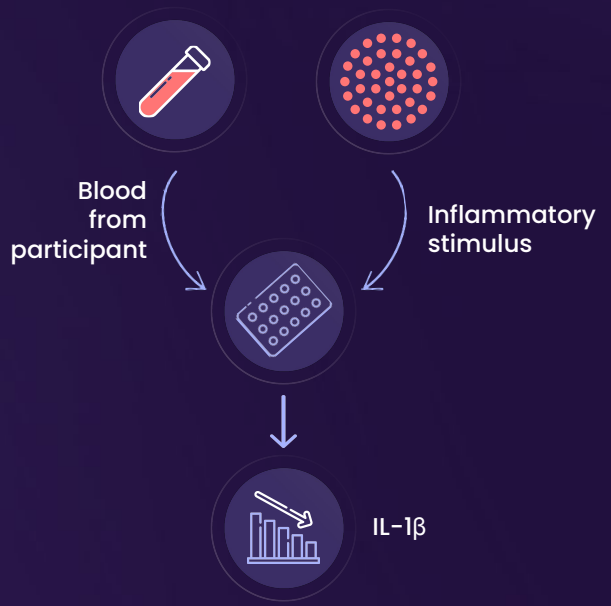
HEALTHY VOLUNTEER SAD / MAD

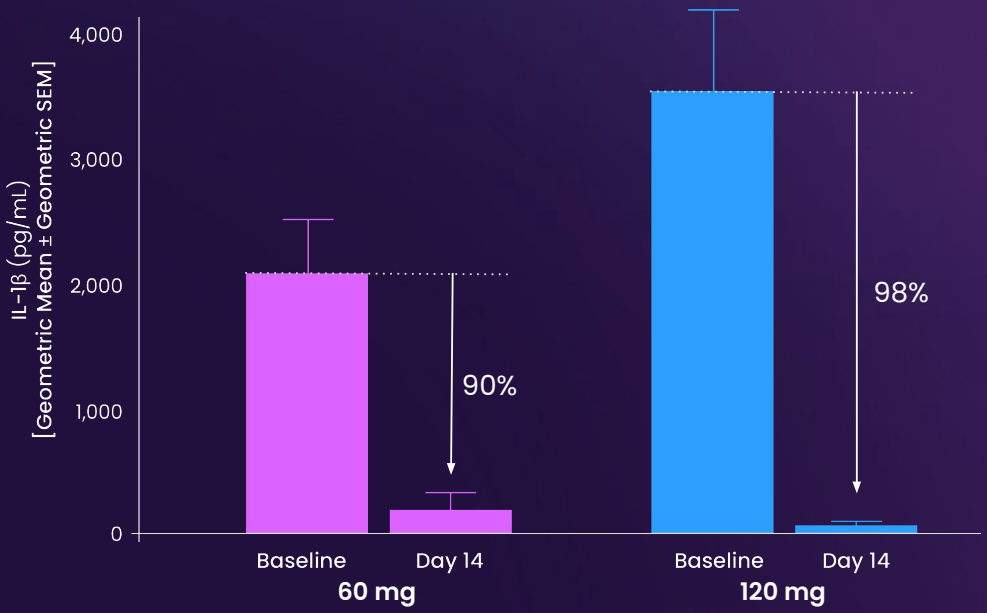
PK / PD: 90-98% suppression of IL-1β at BGE-102 trough in MAD cohorts

Ex vivo whole blood stimulation



MAD cohorts: mean IL-1β with ex vivo stimulation at trough

baseline vs Day 14

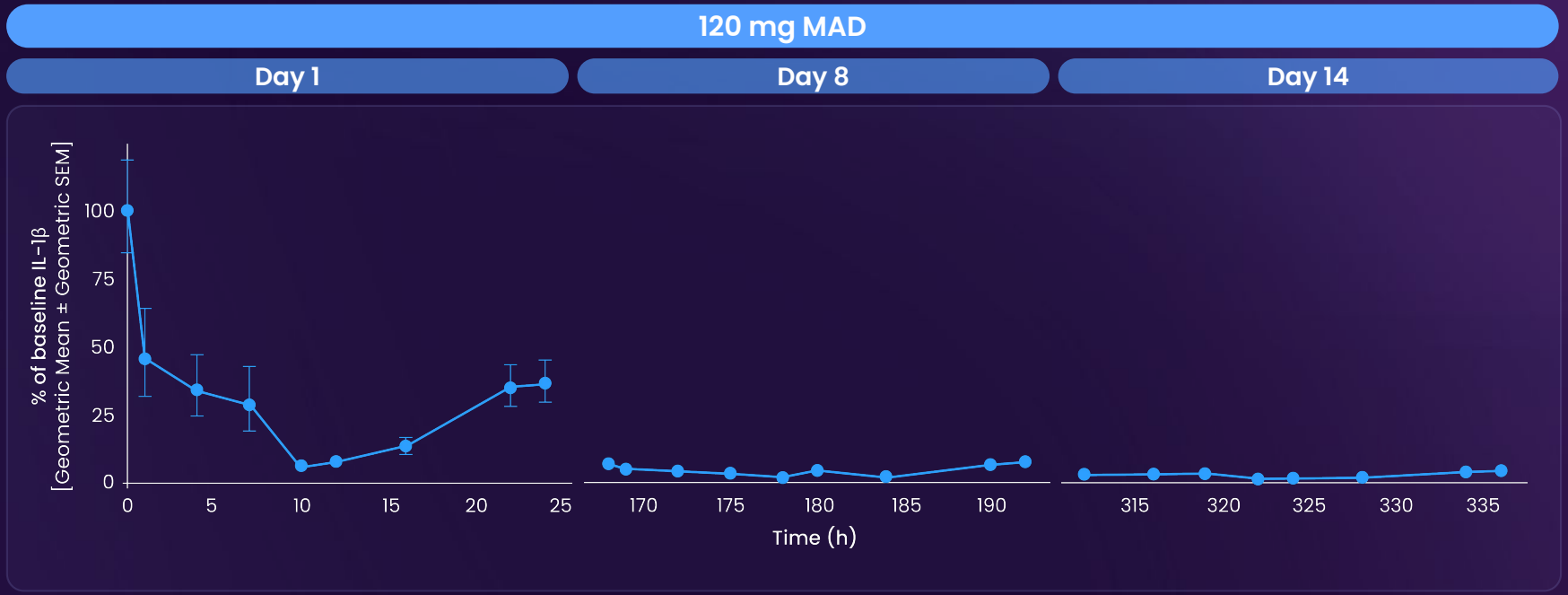


BIOAGE

NLRP3

HEALTHY VOLUNTEER SAD / MAD

PK / PD: 24 hours of near-maximal suppression of IL-1β achieved by Day 8



BIOAGE

NLRP3

Structural biology insights

Phase 1 results

- Healthy volunteer SAD / MAD
- Obese MAD

Cardiovascular

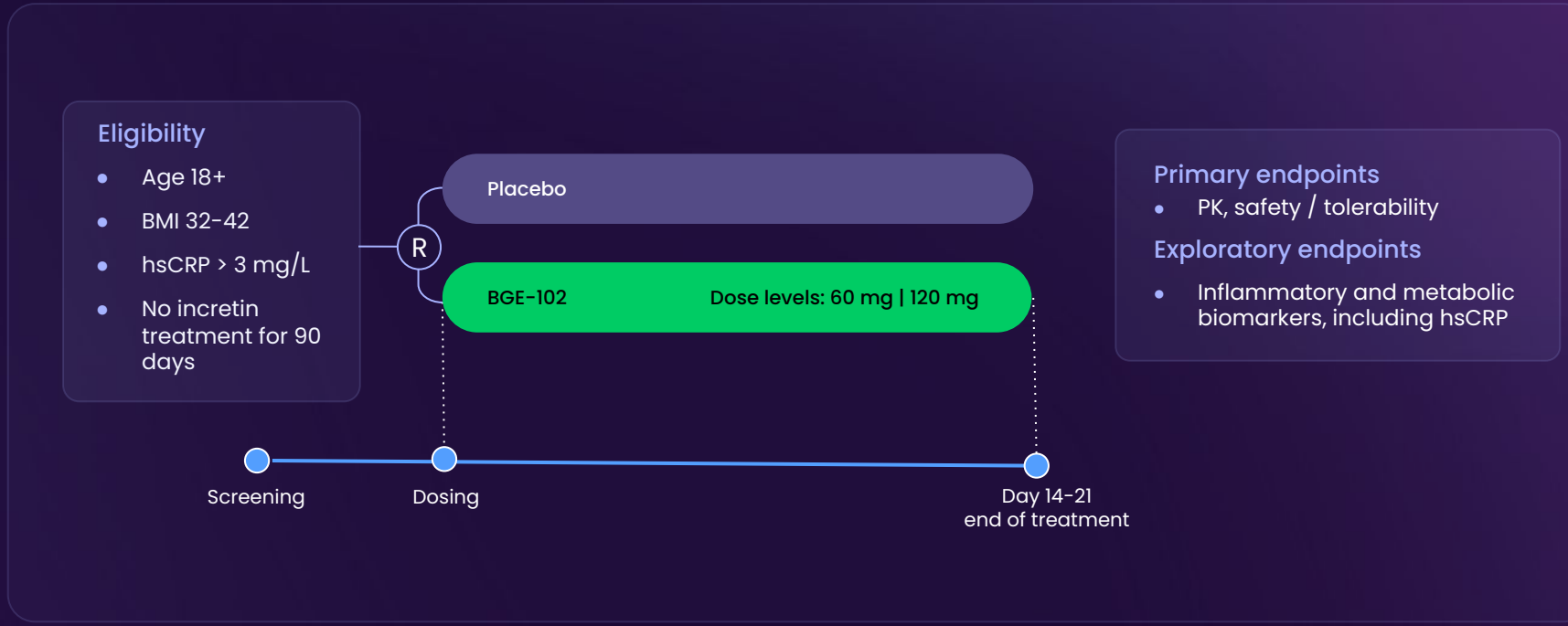
Ophthalmology

BIOAGE

NLRP3

OBESE MAD

The Phase 1 trial included 2 obese MAD cohorts, enabling evaluation of biomarkers including hsCRP

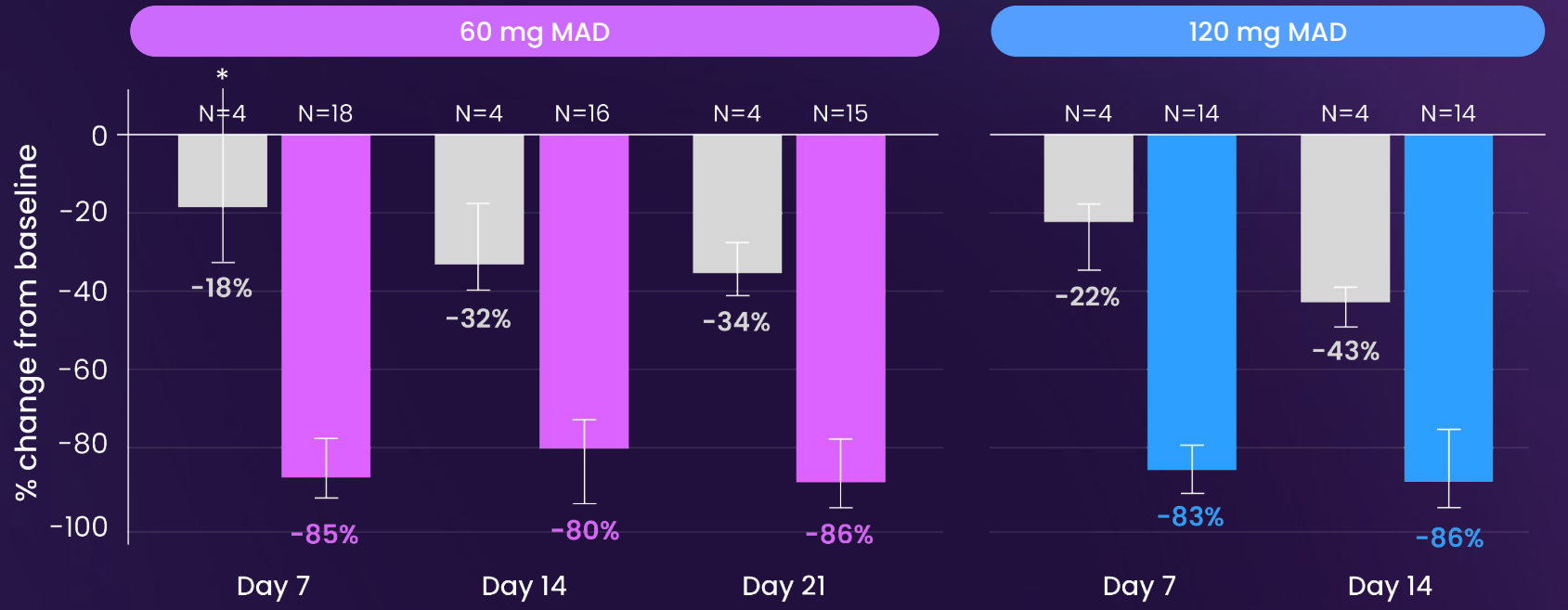


BIOAGE

NLRP3

OBESE MAD

60 mg & 120 mg doses of BGE-102 resulted in 86% reductions in hsCRP, consistent with best-in-class efficacy



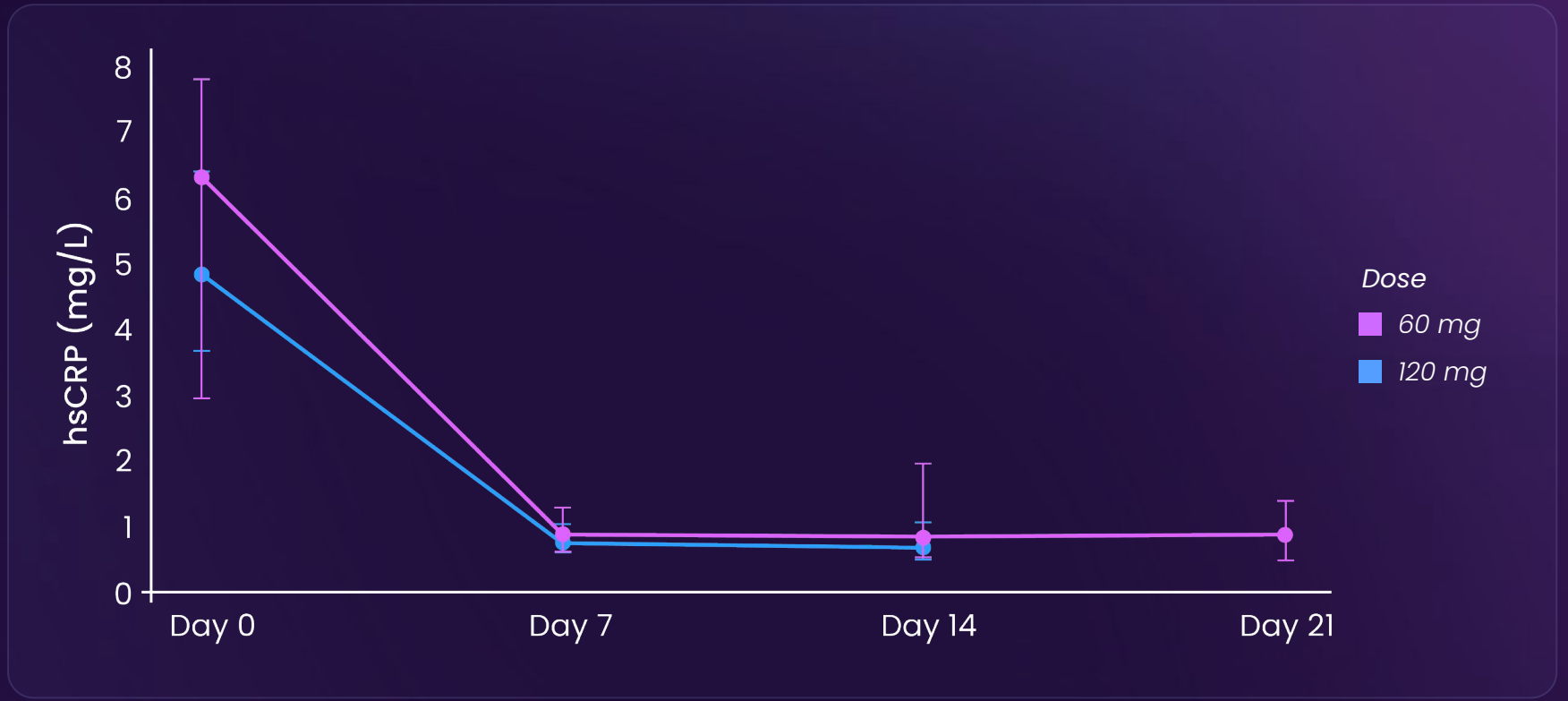
*Note: median values, error bars show IQR (Q1-Q3); * Day 7 placebo IQR upper limit is 115%; median baseline hsCRP 60 mg cohort 6.30 mg/L for both active treatment and placebo, 120 mg cohort 4.85 mg/L for active treatment and 4.25 mg/L for placebo*

BIOAGE

NLRP3

OBESE MAD

Similar hsCRP trajectories in both obese MAD cohorts



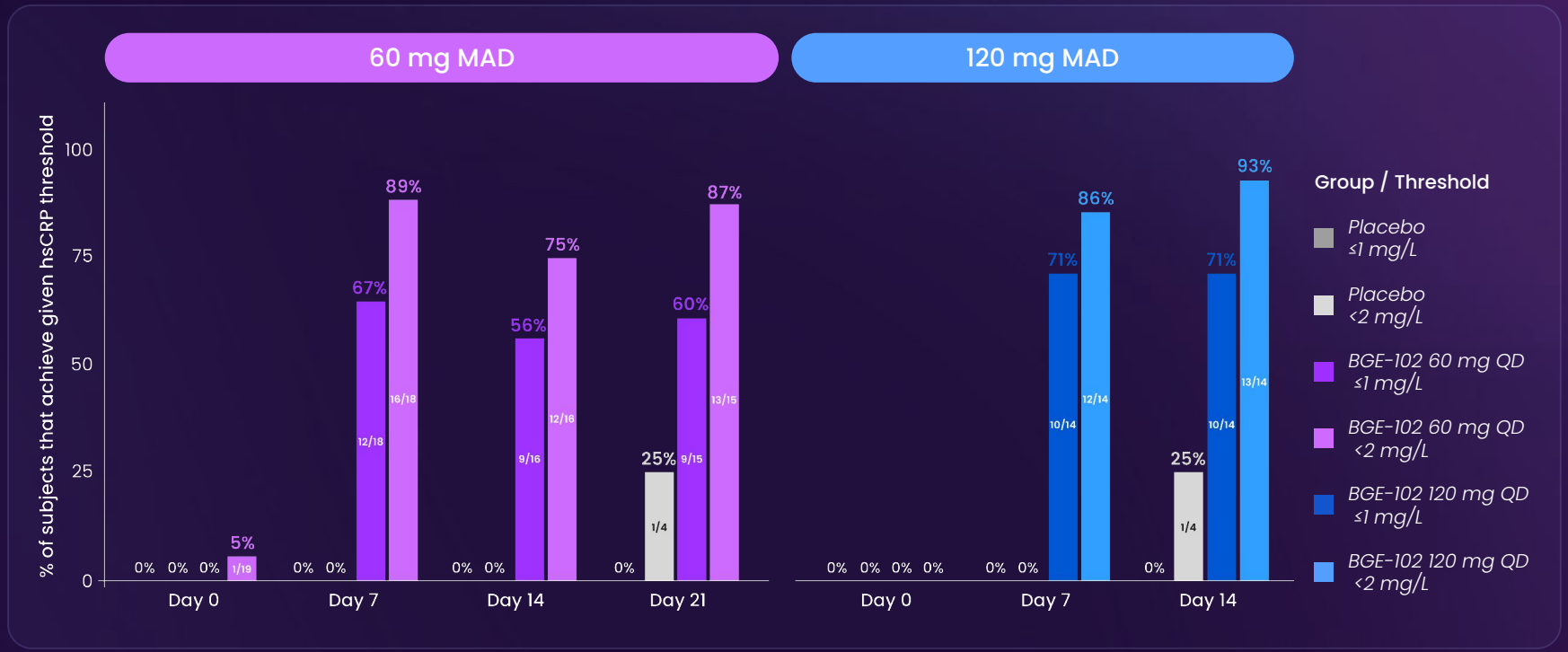
Note: median values, error bars show IQR (Q1-Q3); median baseline hsCRP for subjects on active treatment 60 mg cohort 6.30 mg/L, 120 mg cohort 4.85 mg/L

BIOAGE

NLRP3

OBESE MAD

87-93% of subjects on BGE-102 achieved normal hsCRP levels (<2 mg/L)



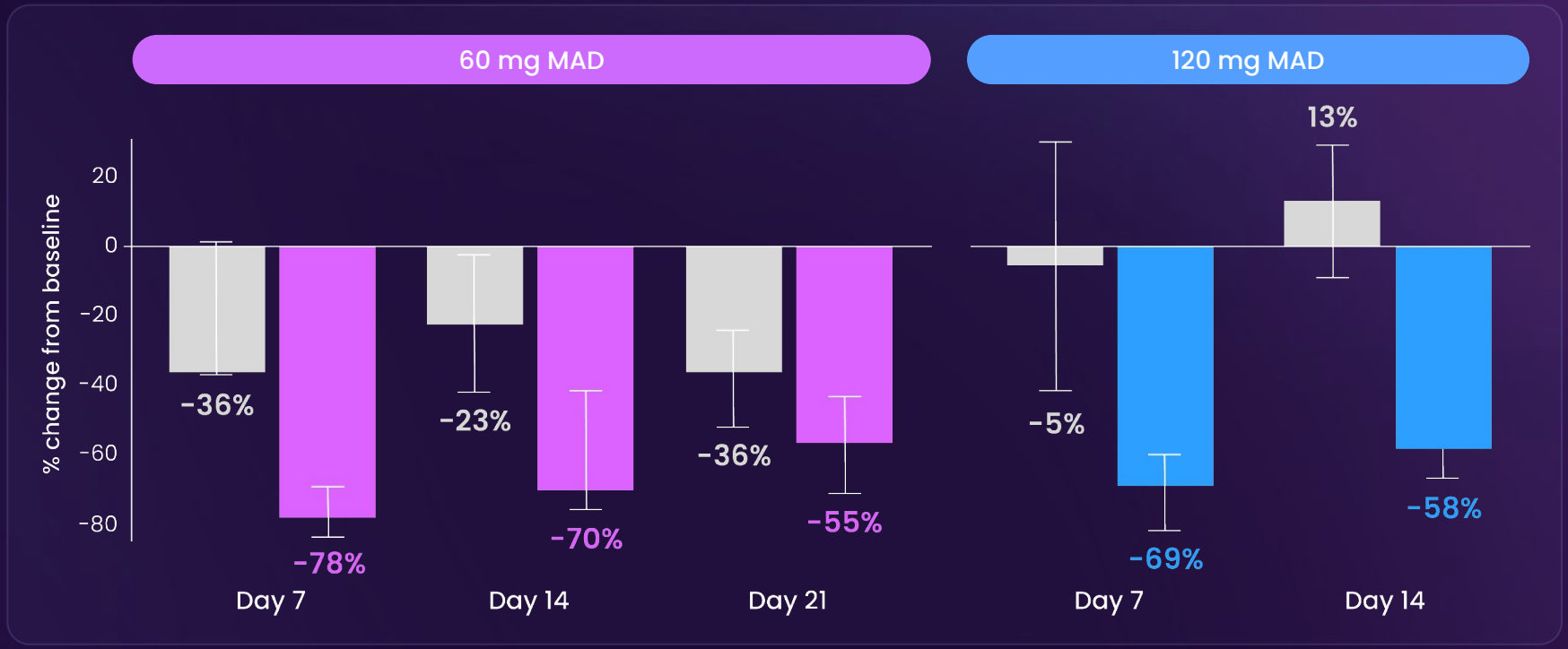
Note: median baseline hsCRP 60 mg cohort 6.30 mg/L for both active treatment and placebo, 120 mg cohort 4.85 mg/L for active treatment and 4.25 mg/L for placebo

BIOAGE

NLRP3

OBESE MAD

60 mg & 120 mg doses of BGE-102 resulted in comparable IL-6 reductions



Note: median values, error bars show IQR (Q1-Q3); median baseline IL-6 60 mg cohort 2.2 pg/mL for active treatment and 2.8 pg/mL for placebo 120 mg cohort 2.3 pg/mL for active treatment and 1.3 pg/mL for placebo

BIOAGE

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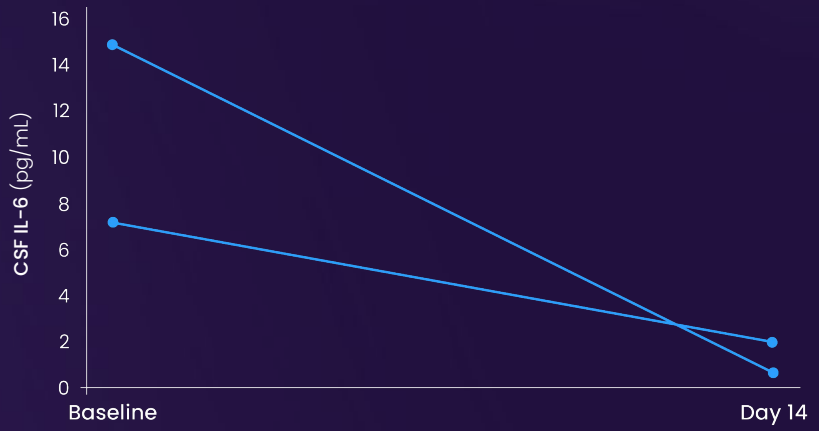
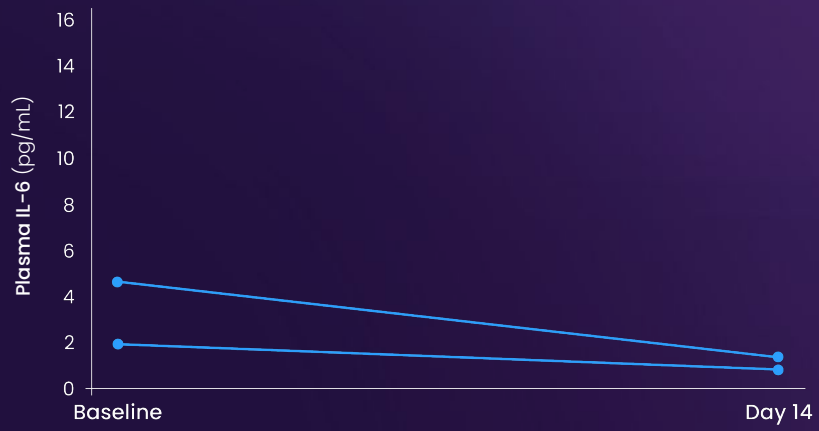
OBESE MAD

In the 2 subjects with elevated baseline CSF IL-6, BGE-102 reduced levels by 84% in the Day 14 CSF

CSF IL-6*



Plasma IL-6



*Note: * IL-6 levels >7 pg/mL in the CSF are considered elevated; corresponding decrease in the plasma was 62%; measurements performed using the Alamar NULISA platform*

BIOAGE

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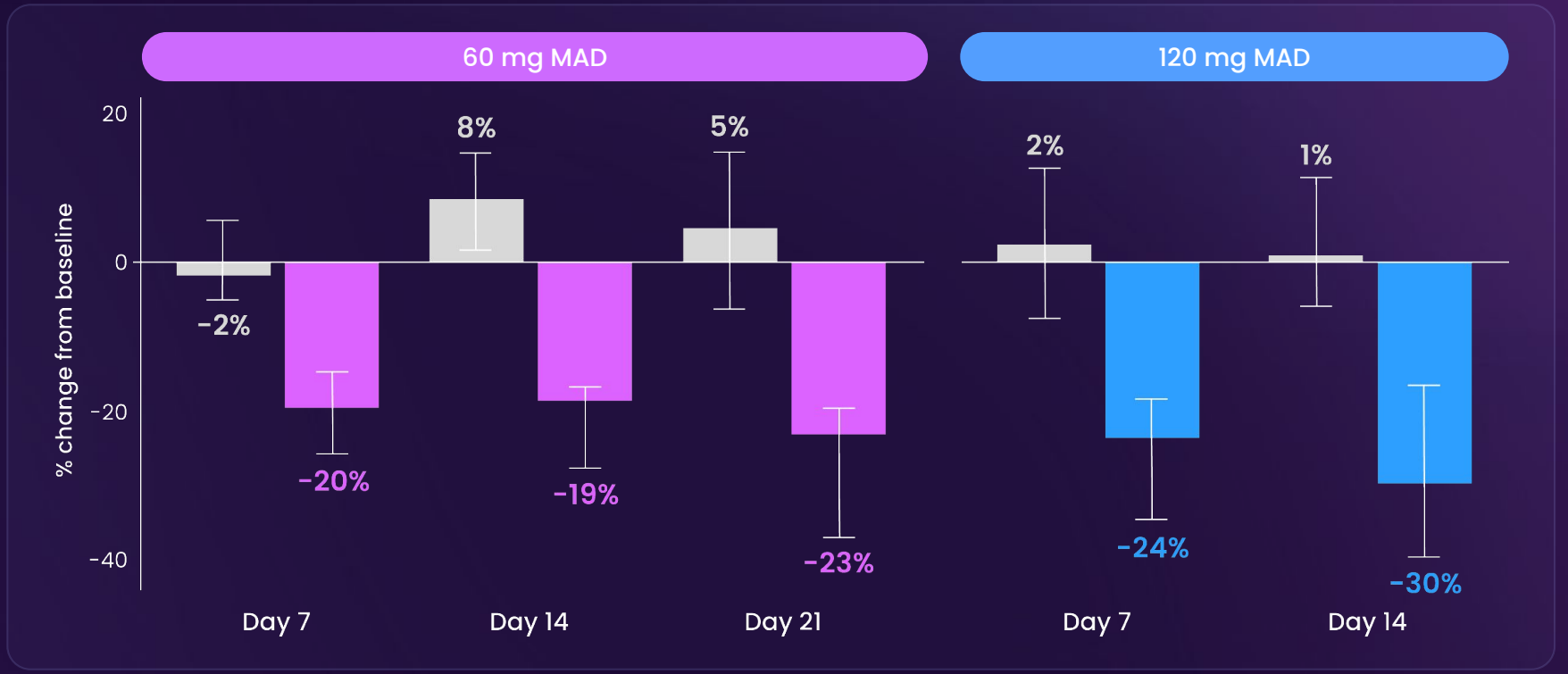
OBESE MAD

60 mg & 120 mg doses of BGE-102 resulted in comparable reductions in fibrinogen



Note: median values, error bars show IQR (Q1-Q3); median baseline fibrinogen 60 mg cohort 362 mg/dL for active treatment and 379 mg/dL for placebo, 120 mg cohort 331 mg/dL for active treatment and 290 mg/dL for placebo

BIOAGE

NLRP3

Structural biology insights

Phase 1 results

Cardiovascular

Ophthalmology

Paul Rubin, MD
CMO & EVP Research

BIOAGE

NLRP3

CARDIOVASCULAR

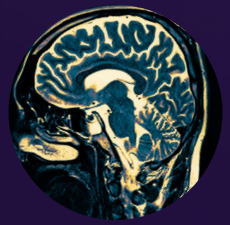
BGE-102 has the potential to address a range of cardiometabolic and neuroinflammatory disorders

Neuro-inflammation

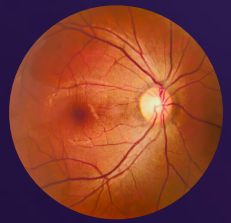
Peripheral inflammation

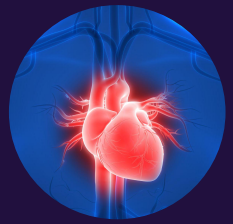
BGE-102 addressable diseases

Neurodegeneration

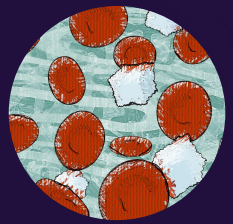


Ocular

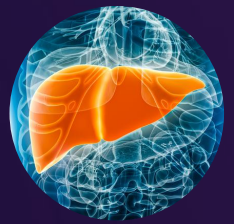




CVD (ASCVD, HF)



Insulin resistance



MASLD / MASH

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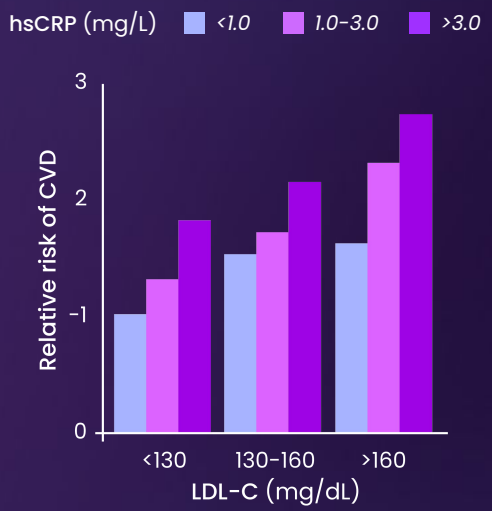
CARDIOVASCULAR

The CV opportunity

Inflammation is an independent risk factor for CVD



15M addressable patients in the US alone

~60%

of 25M ASCVD patients have elevated hsCRP

Orals are highly preferable

>80%

of statins are prescribed by PCPs

Potential for oral fixed-dose combinations with other CV risk mechanisms (e.g., PCSK9, GLP-1)

Lipid-lowering therapies set precedent for large market

$50B

expected global market size of lipid-lowering therapies by 2035

Source: Ridker 2002, Nguyen 2024, Mazhar 2024, broker reports, Towards Healthcare

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CARDIOVASCULAR

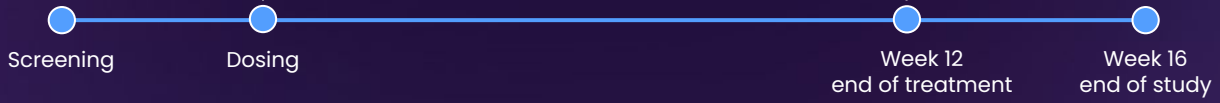
Planned CV risk POC

Key eligibility criteria

- Age 18+
- BMI 32-42
- hsCRP > 3 mg/L
- ≥1 additional CV risk factor

R

Placebo	N=40
BGE-102 30 mg QD	N=40
BGE-102 60 mg QD	N=40
BGE-102 90 mg QD*	N=40



Primary endpoint

- % change in hsCRP

Key secondary endpoints

- % normalization of hsCRP (<2 mg/L and ≤1 mg/L)

Exploratory endpoints

- Additional CV risk factors (e.g., Lp(a), fibrinogen, IL-6)
- MRI imaging
- Metabolic parameters (e.g., fasting glucose, HbA1c)
- Body weight

*Note: * PK modeling indicates that the 90 mg QD dose at steady state will reach or exceed exposures from the 120 mg QD dose after 1 week, where we observed maximal IL-1β inhibition.*

BIOAGE

Michael Davidson, MD

Clinical Professor of Medicine
Director, Lipid Clinic

University of Chicago
Pritzker School of Medicine

Anti-inflammatory strategies for ASCVD

BIOAGE

Role of inflammation in atherosclerosis?

- A fierce debate started in the 19th century -

Both observed cellular inflammatory changes in the atherosclerotic vessel walls

Carl von Rokitansky

"Inflammation accompanies atherosclerosis"

Rolitansky;
A manual of pathologic anatomy. 1852

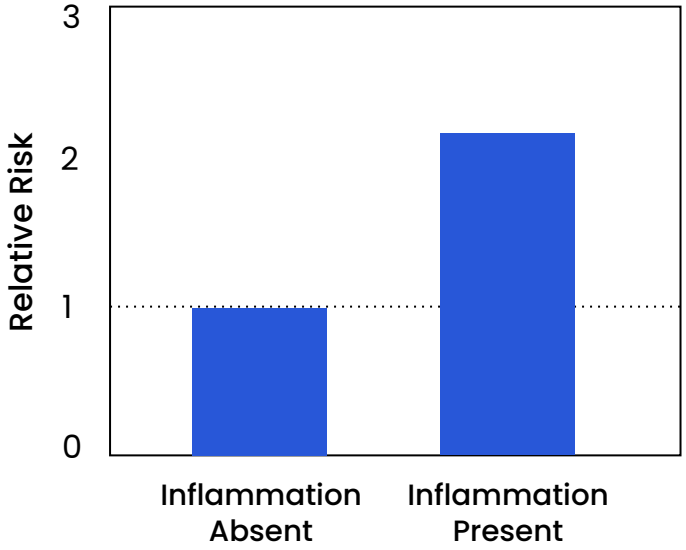
Rudolf Virchow

"Inflammation initiates atherosclerosis"

Virchow R. Der ateromatöse Prozess der Arterien. *Wien Med Wschr* 1856

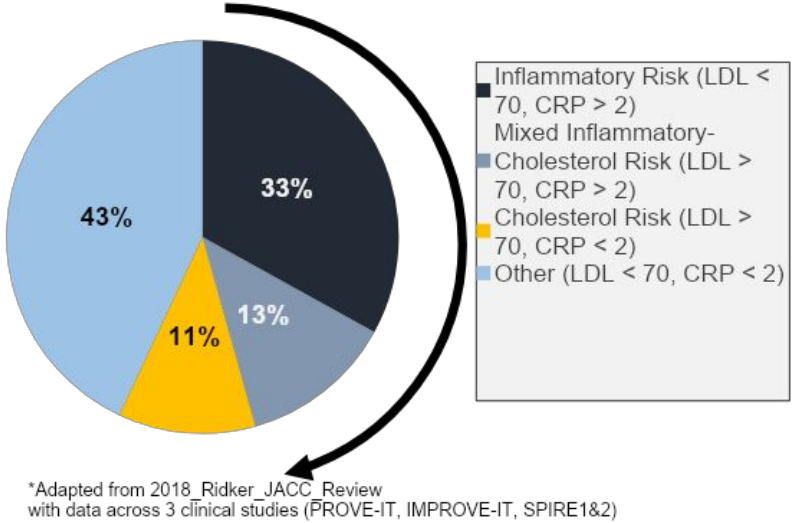
Chronic Inflammation is a Driver of Cardiovascular Risk

>2-fold increase in relative CV risk due to inflammation



Adapted from 1998_ridker_Circulation

~46% of patients with inflammation despite maximal lipid therapy (across 4 studies)

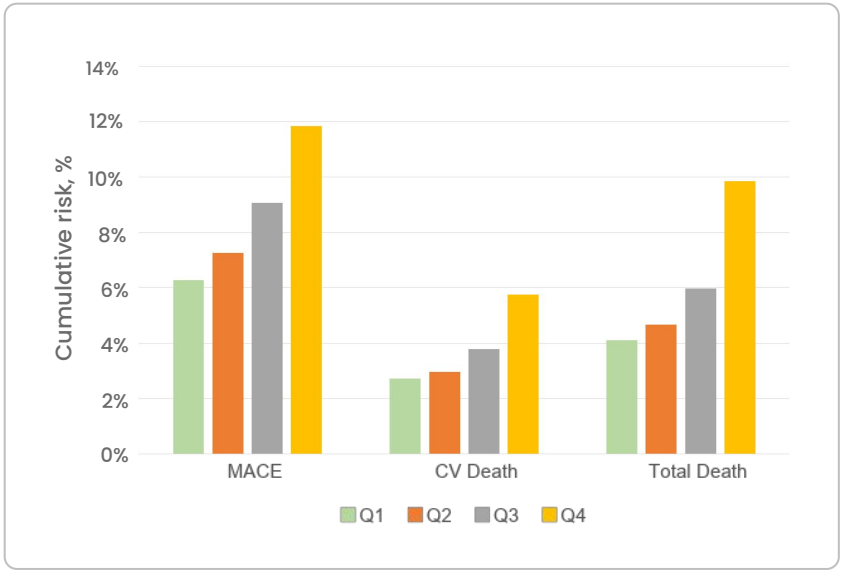


*Adapted from 2018_Ridker_JACC_Review with data across 3 clinical studies (PROVE-IT, IMPROVE-IT, SPIRE1&2)

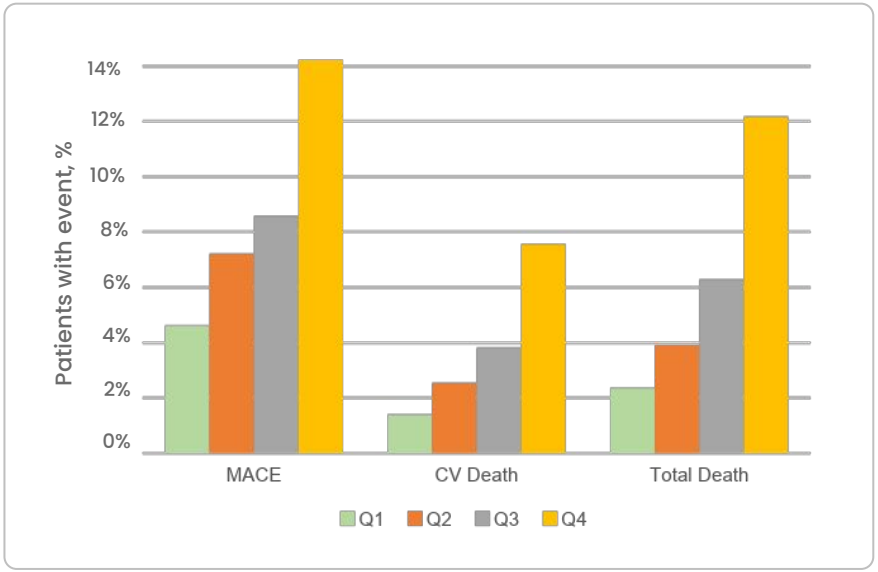
Source: 1998_Ridker_Circulation, 2019_Aday_Ridker_FCVM , 2018_Ridker_JACC_Review, 2019_Benjamin_Circulation, 2017_RIdker_NEJM, 2009_Ridker_CircOutcomes, 2018_Cao_BMJOpen, 2018_Bhatt_NEJM, Adapted from 2018_Ridker_JACC_Review

hsCRP and IL-6 predict MACE, CV Death, and Total Death: results from the STABILITY trial

MACE by baseline hsCRP quartile



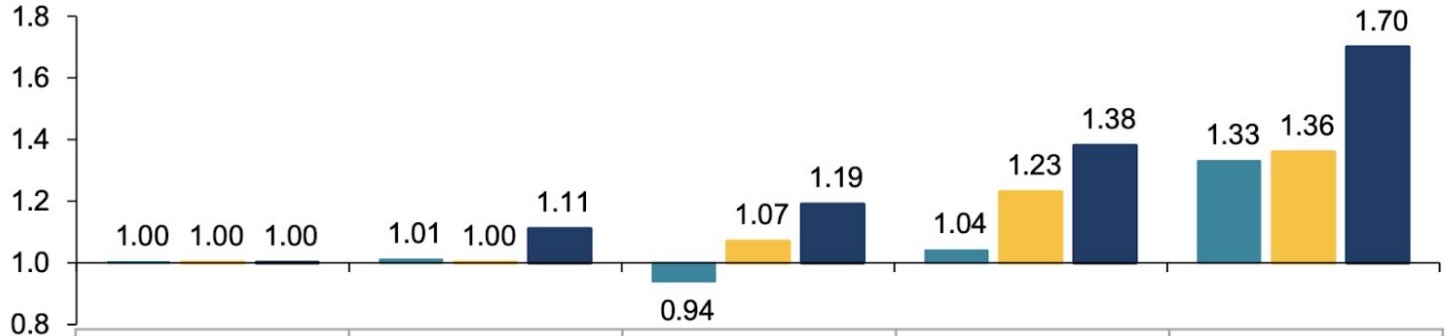
% of CHD patients with an event over 3 years by baseline IL-6 quartile



Emerging evidence suggests that hsCRP is more strongly associated with MACE than both LDL and Lp(a)

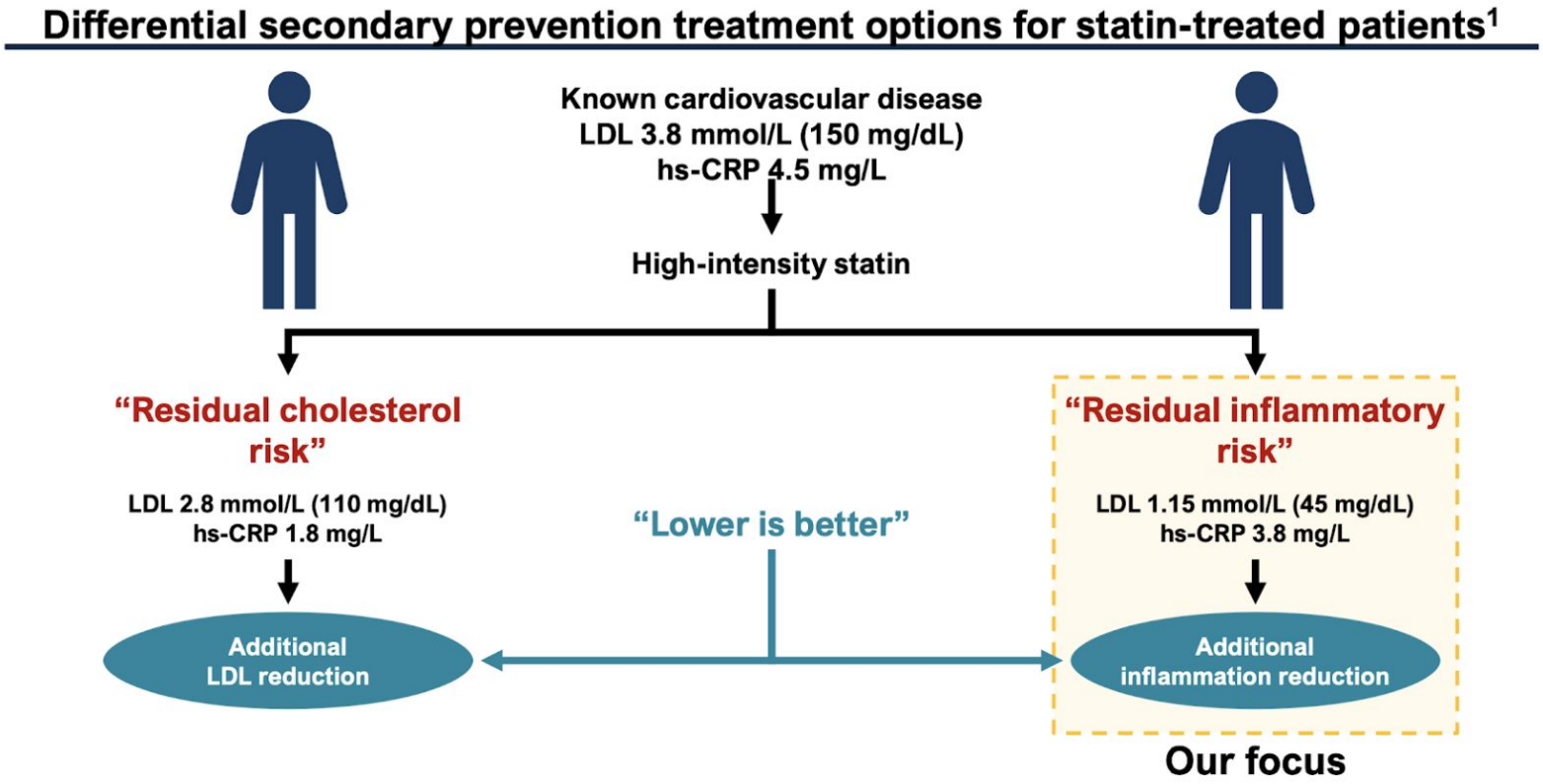
Late breaking data presented at European Society of Cardiology 2024 Congress and simultaneously published in the New England Journal of Medicine

30-year longitudinal data from the Women's Health Study[1]
(n=27,929)

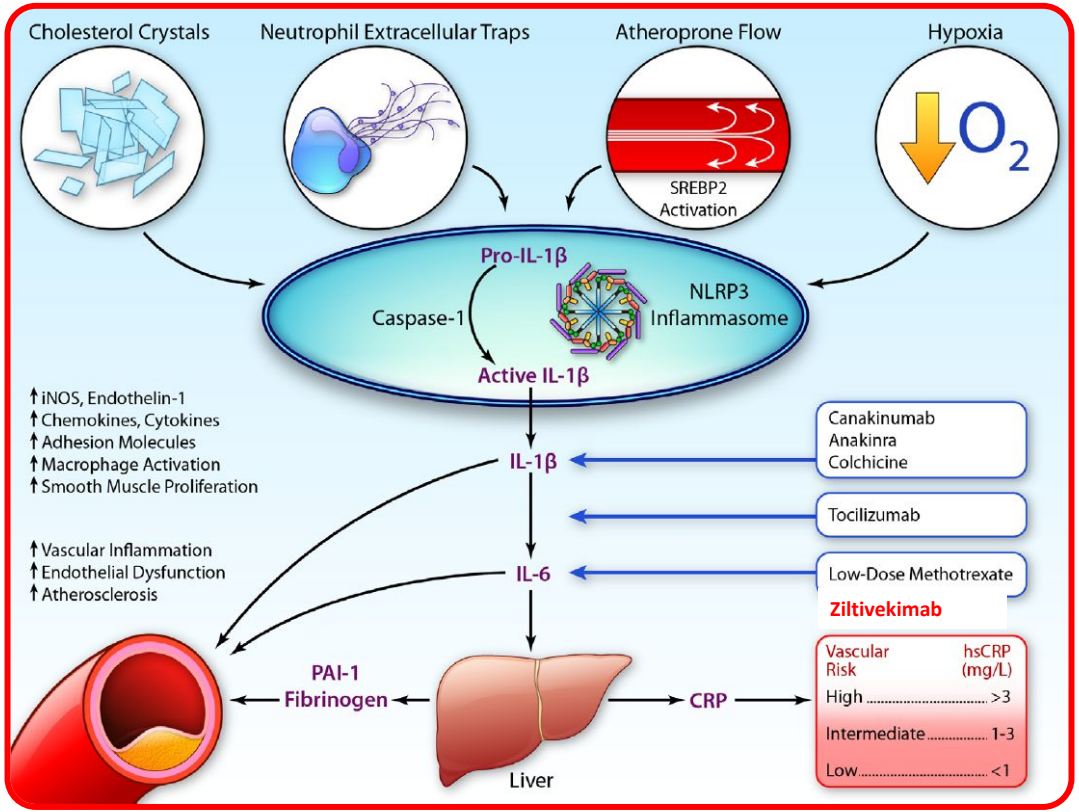


	Quintile (reference)	Quintile 2	Quintile 3	Quintile 4	Quintile 5
hs-CRP mg/L baseline	<0.65	0.65 to <1.47	1.47 to <2.75	2.75 to <5.18	≥5.18
LDL-C mg/dL baseline	<96.1	96.1 to <113.5	113.5 to <129.7	129.7 to <150.7	≥150.7
Lp(a) mg/dL baseline	<3.6	3.6 to <7.6	7.6 to <15.5	15.5 to <44.1	≥44.1

Many CV disease patients have residual inflammatory risk



Novel Targets for Reducing CRP for Atheroprotection



Ridker et al Circ Res 2016;118:145-156.

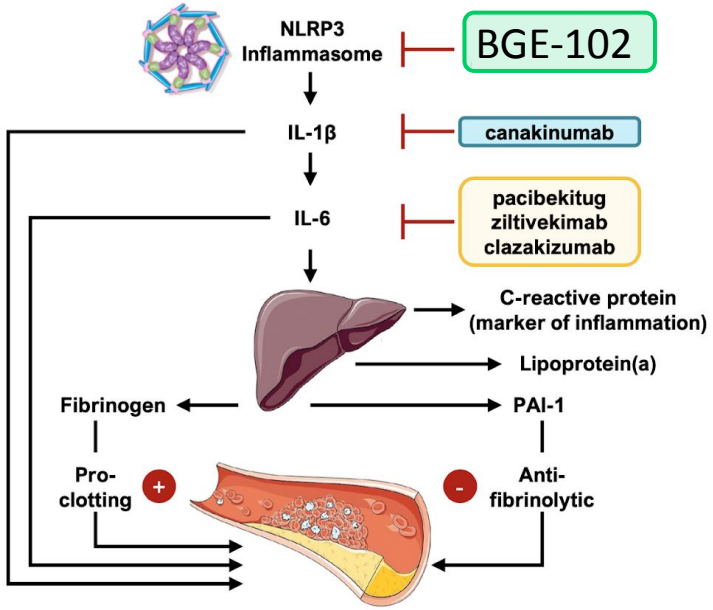
Canakinumab Anti-inflammatory Thrombosis Outcomes Study (CANTOS) Trial Design[1]

10,061 patients
- Stable CAD (post MI)
- On Statin, ACE/ARB, BB, ASA
- hs-CRP ≥2 mg/L

- Canakinumab 50 mg Q3M n = 2,170
- Canakinumab 150 mg Q3M n = 2,284
- Canakinumab 300 mg Q3M n = 2,263
- Placebo Q3M n = 3,344

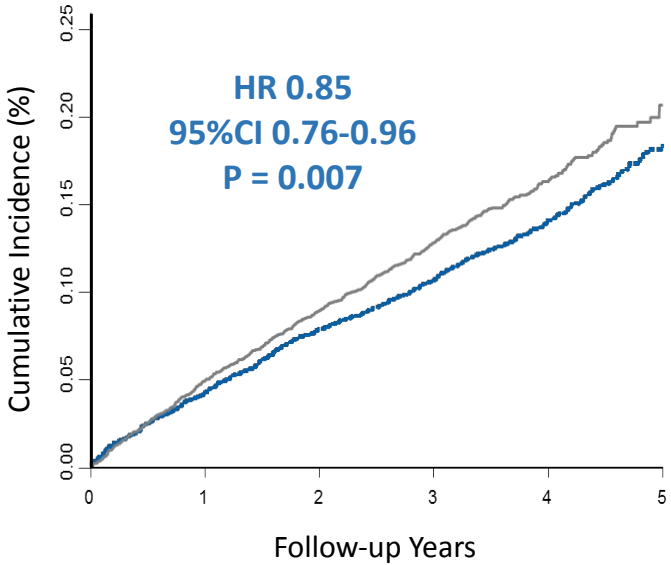
Primary endpoint:
Time to the first occurrence of MACE (CV death, non-fatal MI, or non-fatal stroke)

IL-1β is upstream of IL-6[2]

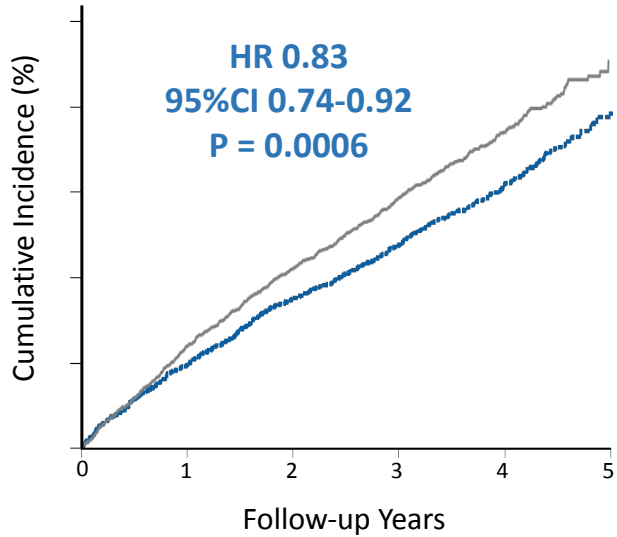


Placebo SC q 3 months
Canakinumab 150/300 mg SC q 3 months

MACE



MACE - Plus

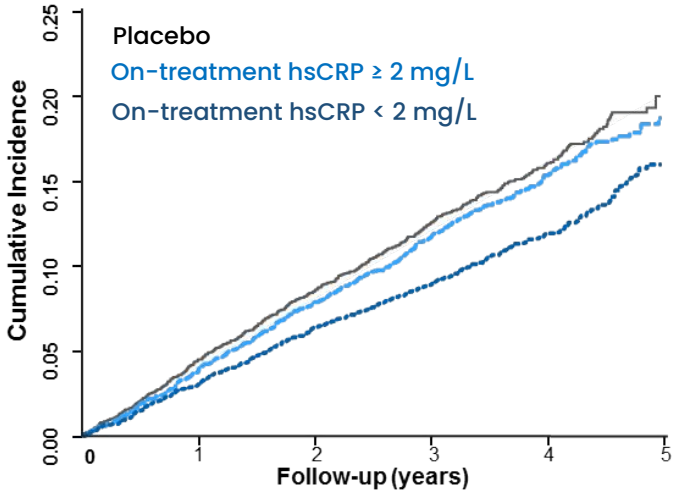


35 - 40% reductions in hsCRP and IL-6
No change in LDLC

Ridker PM et al. N Engl J Med. 2017;377:1119-31

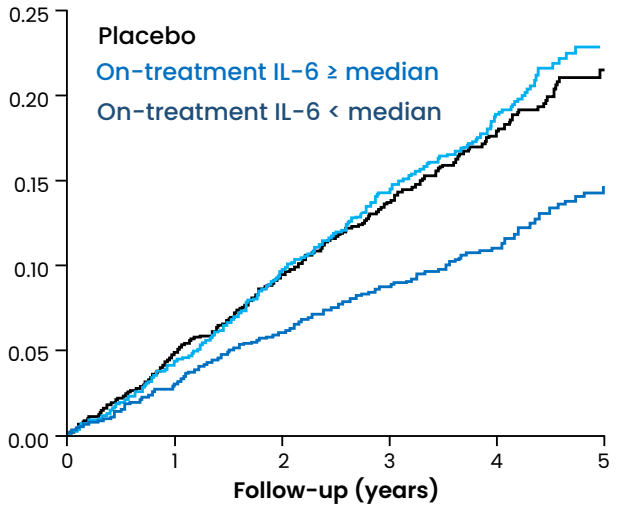
Achievement of CRP < 2.0mg/L Provide 50% reduction in Mortality if CANTOS like Responses can be Achieved in CVOT

On-Treatment hsCRP



MACE
25% reduction in risk for those achieving hsCRP < 2 mg/L
5 % reduction in risk for those achieving hsCRP ≥ 2 mg/L
(No change in LDL cholesterol)

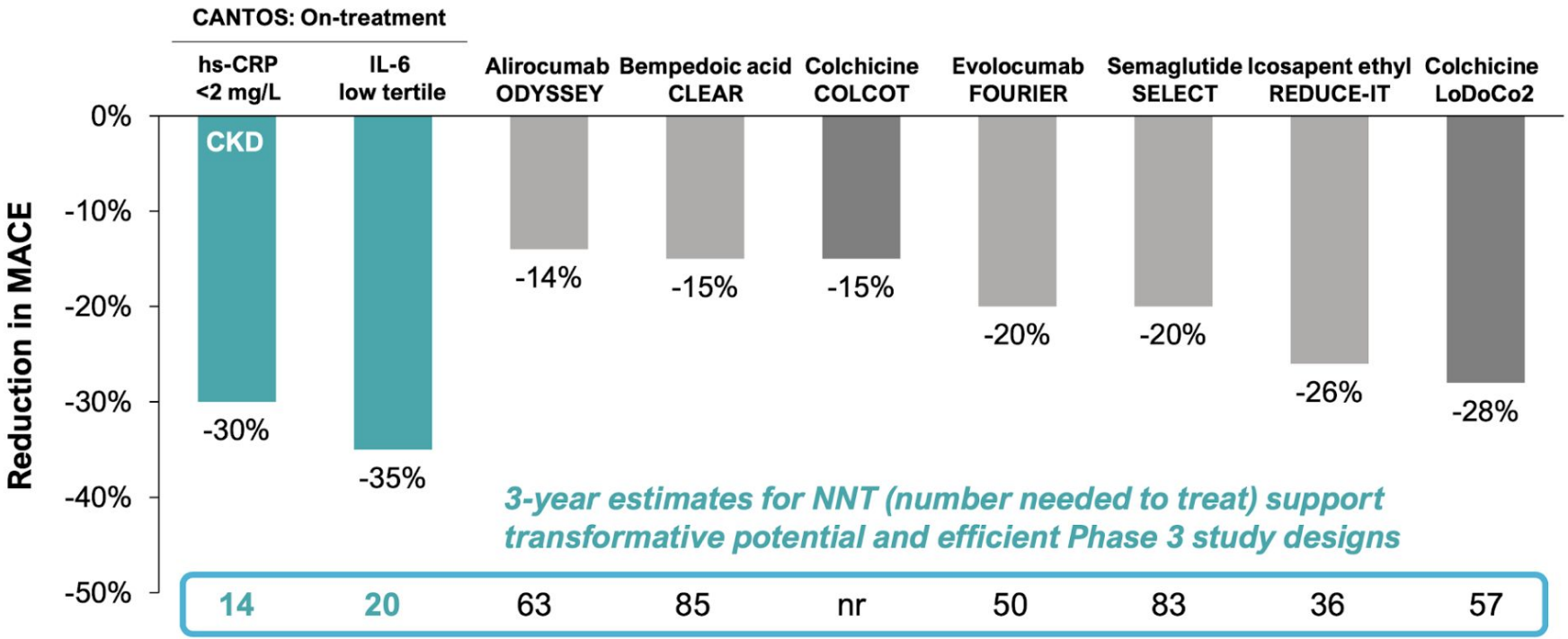
On-Treatment IL-6



MACE
36% reduction for those achieving IL-6 below median
No benefit for those achieving IL-6 above median
(No change in LDL cholesterol)

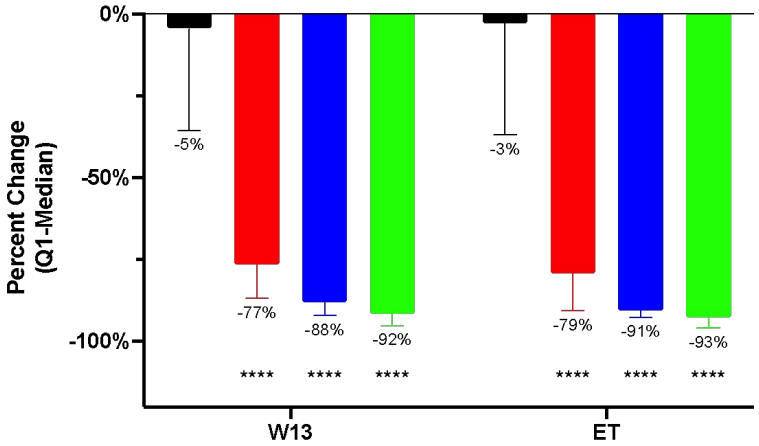
Source: Lancet 2018;391:319-328, Eur Heart J 2018;39:3499-3507
Canakinumab Antiinflammatory Thrombosis Outcome Study (CANTOS)

Lessons from canakinumab: Robust reduction of inflammation through IL-1B has transformative potential for ASCVD



Primary RESCUE End Point Achieved

Median Percent Change in hsCRP



Placebo
Ziltivekimab 7.5 mg
Ziltivekimab 15 mg
Ziltivekimab 30 mg

vs Placebo
**** $p < .0001$

linear trend: $p < .0001$

Median (Q1, Q3) hsCRP values (mg/L) at baseline, W13 and ET

Time	Ziltivekimab			
	Placebo	**7.5 mg**	**15 mg**	**30 mg**
Baseline	5.8 (3.3 ,9.9) (N=66)	5.5 (3.5 ,9.3) (N=66)	5.7 (3.5 ,8.1) (N=66)	5.8 (3.7 ,8.9) (N=66)
W13	5.1 (3.4 ,9.2) (N=57)	1.0 (0.6 ,2.8) (N=58)	0.6 (0.4 ,1.4) (N=61)	0.5 (0.2 ,0.8) (N=57)
ET	6.4 (3.5 ,10.0) (N=51)	0.9 (0.4 ,1.8) (N=52)	0.5 (0.3 ,1.4) (N=55)	0.4 (0.2 ,0.8) (N=53)

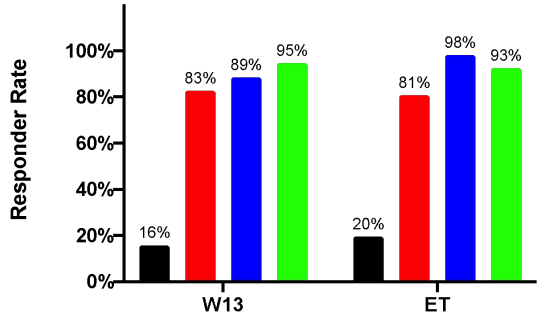
Note: Percent change is shown as Median (Q1/Q3)

ET (End of Treatments) is defined as the average of values at Week 23 and Week 24. For patients who discontinued after Week 13, ET is defined as the average of the values from the last 2 visits after Week 13 including Week 13

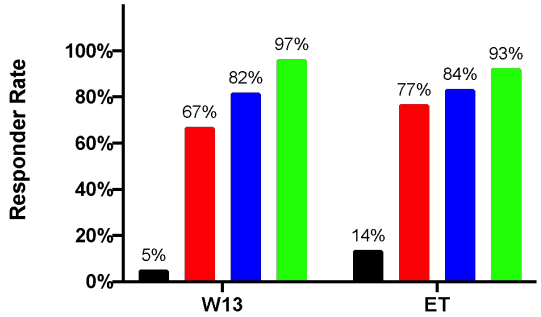
Increasing Responder Rate with Increasing Dose of Zilti

Superior hsCRP Responder Rate Relative to Canakinumab in CANTOS

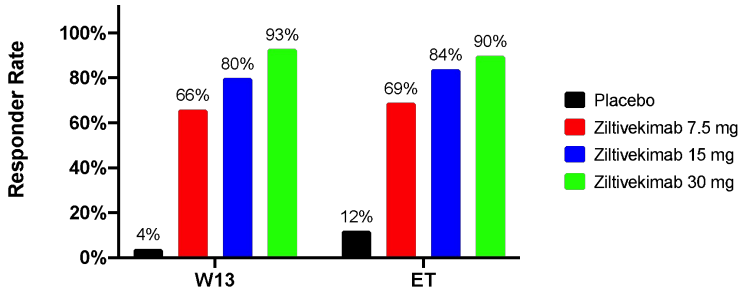
hsCRP suppression >= 50% from baseline



hsCRP suppression < 2 mg/L threshold

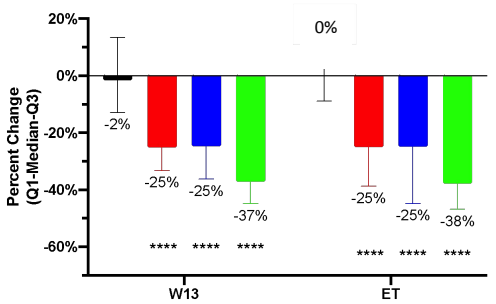


hsCRP suppression >= 50% from baseline AND below 2mg/L



Ziltivekimab Significantly Reduced Fibrinogen, SAA and Haptoglobin

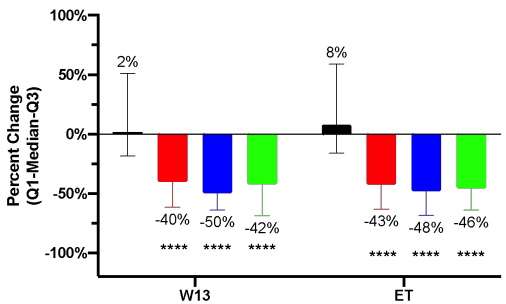
Fibrinogen



N (W13)= 55, 55, 60, 54
N(ET)= 50, 50, 54, 52

A 100 mg/dL increase is associated with a 2-fold increase in CV risk[+]

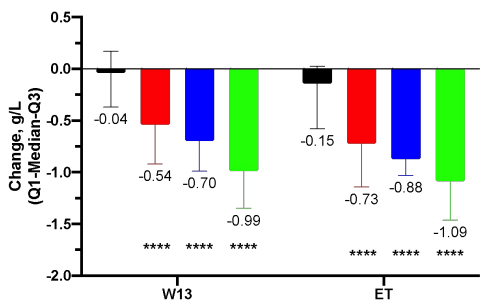
SAA



N (W13)= 57, 58, 60, 56
N(ET)= 52, 52, 55, 52

3.7 mg/L decrease with 15mg dose vs 0.05 mg/L increase with placebo

Haptoglobin



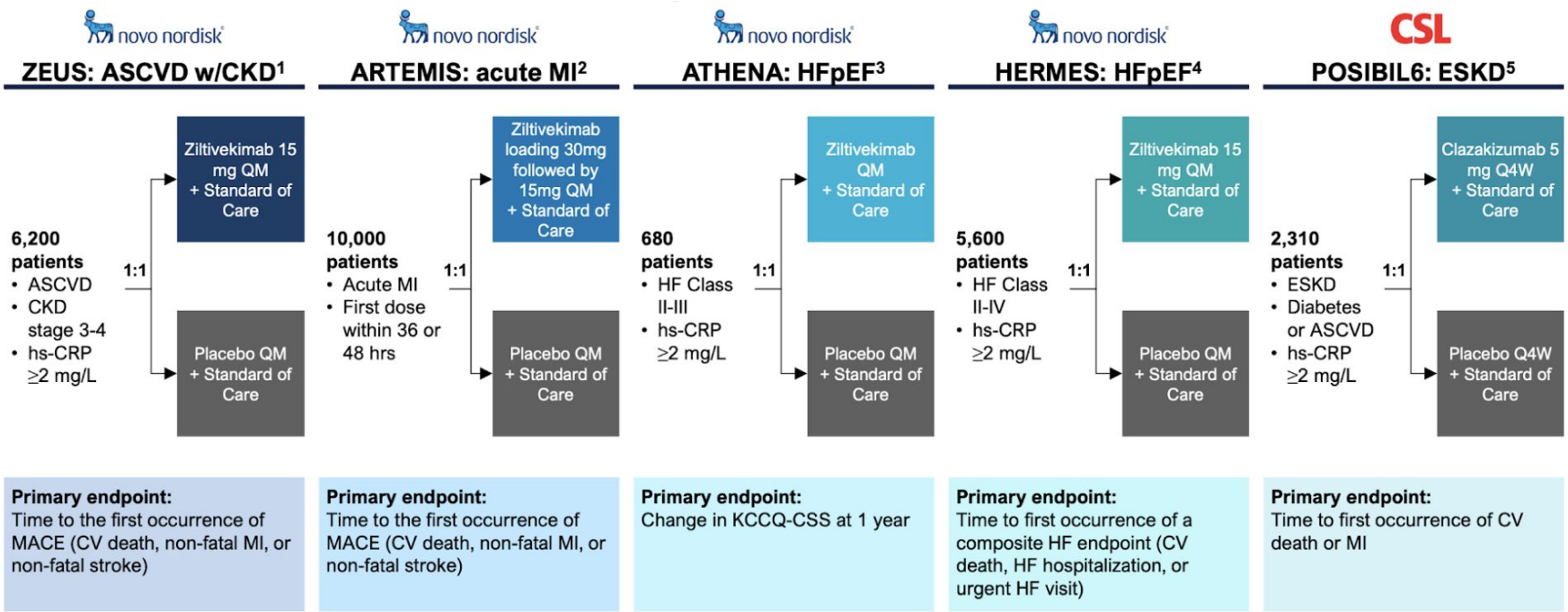
N (W13)= 57, 58, 59, 55
N(ET)= 52, 52, 54, 52

Placebo
Ziltivekimab 7.5 mg
Ziltivekimab 15 mg
Ziltivekimab 30 mg

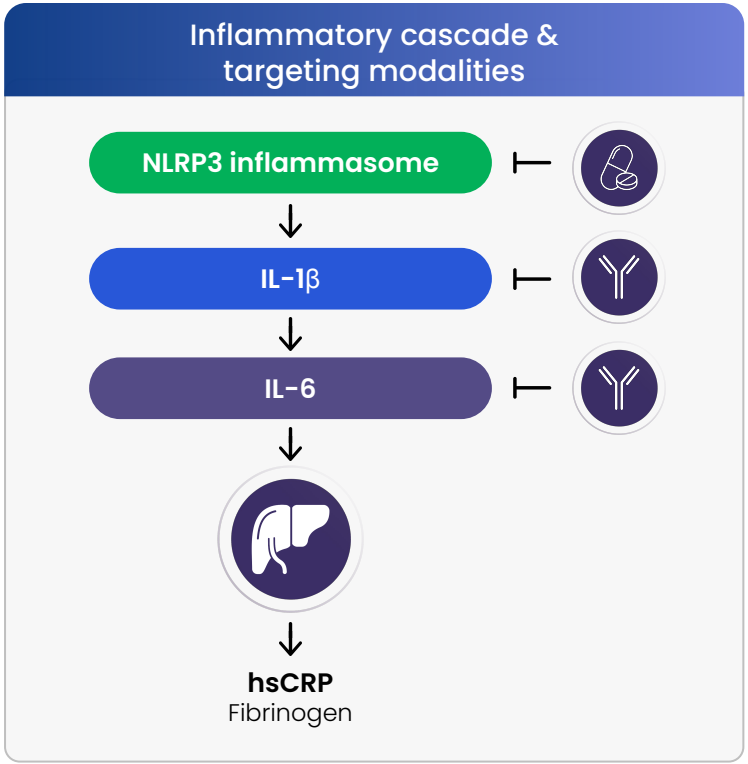
**** p <.0001

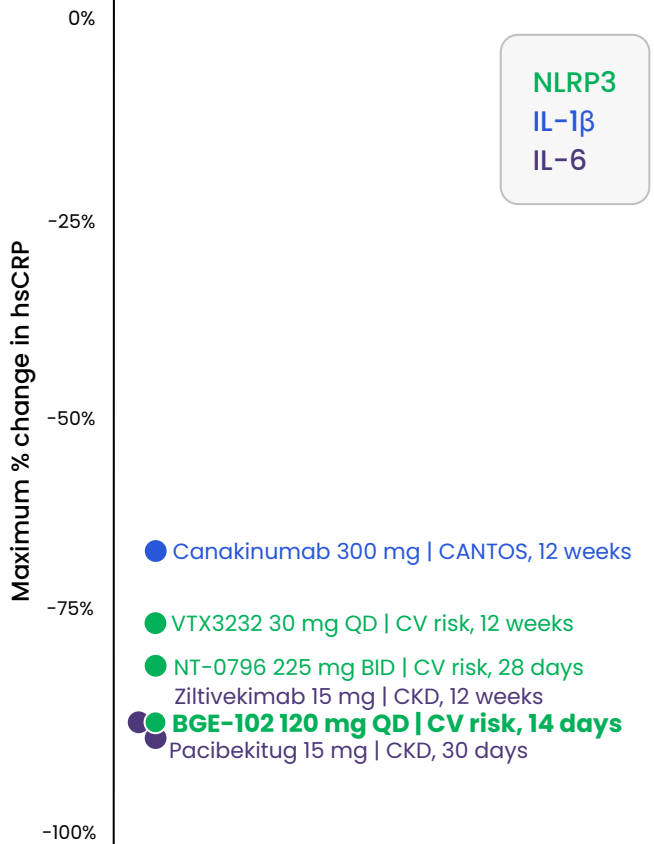
[+] *JAMA 2005 Oct 12;294(14):1799-809*
Note: Percent change is shown as Median (Q1/Q3)

Five Phase 3 CVOTs enrolling > 24,000 patients



NLRP3 inhibitors are a potential “oral IL-6” with comparable reductions in hsCRP

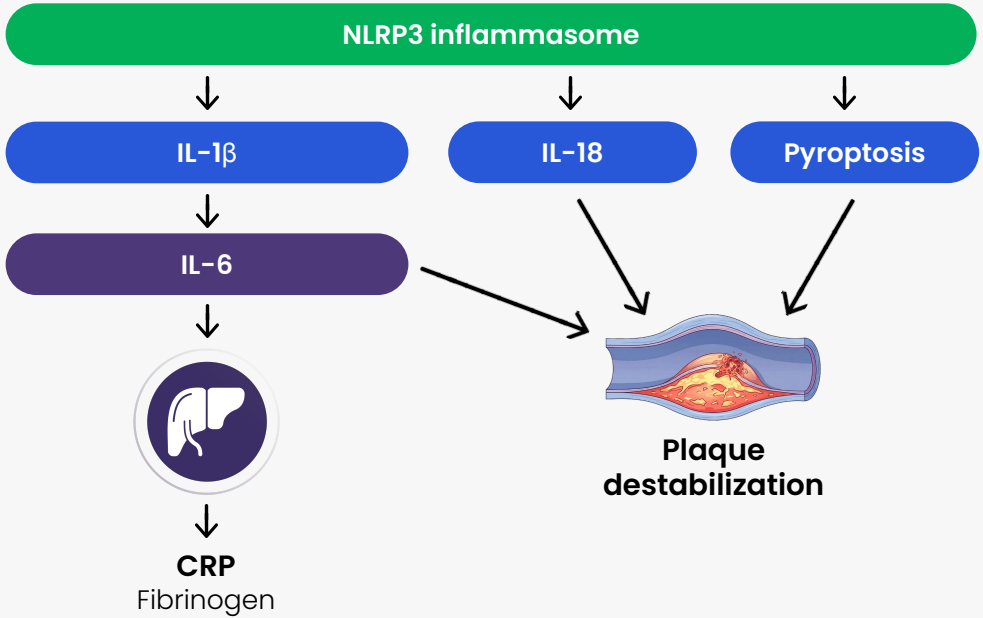




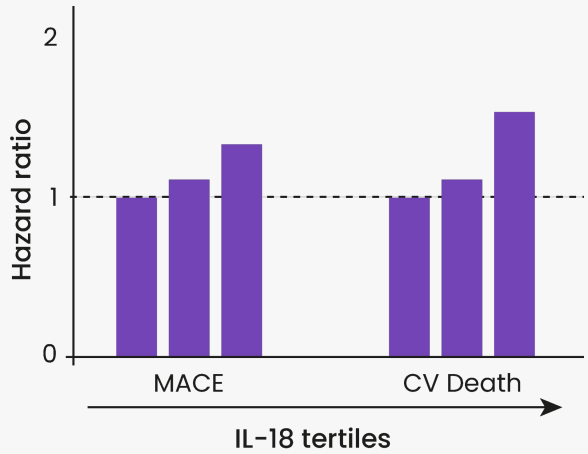
Sources: NodThera, Tourmaline Bio, Ventyx Biosciences, Wada 2023, Ridker 2017

NLRP3 inhibition also has potential benefits beyond IL-6, reducing IL-18 & cell death that contribute to plaque destabilization

NLRP3 drives ASCVD beyond IL-6



Baseline IL-18 is a CVD risk factor in the CANTOS trial



Source: Ridker 2020, Mallat 2001, Zeng 2021

NLRP3

Structural biology insights

Phase 1 results

Cardiovascular

Ophthalmology

Paul Rubin, MD

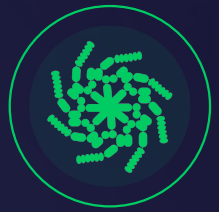
CMO & EVP Research

BIOAGE

NLRP3

OPHTHALMOLOGY

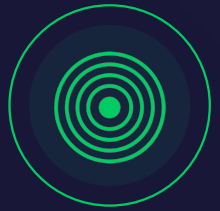
The retinal opportunity for BGE-102



Greater efficacy with new MOA

Oral treatment to lower treatment burden

Simultaneous treatment of bilateral disease



Ocular *and* systemic biology addressed

BGE-102 has shown therapeutic retinal exposure across species, including primates

BIOAGE

NLRP3

OPHTHALMOLOGY

NLRP3 activation is a central feature of retinal diseases DME & GA

Diabetic retinopathy / diabetic macular edema (DME)

hyperglycemia & oxidative stress

↓

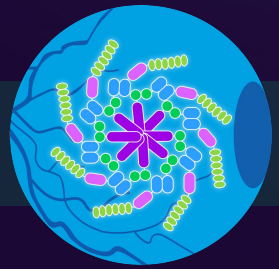
NLRP3 activation (IL-1β, IL-18)
endothelial cells | Müller cells, microglia

↓ pyroptosis

↓ IL-6

↓ VEGF

↓ vascular leakage & vision loss



Dry age-related macular degeneration (AMD) / geographic atrophy (GA)

accumulation of cellular debris
(drusen, lipofuscin) and Alu RNA

↓

NLRP3 activation (IL-1β, IL-18)
RPE cells | microglia

↓ pyroptosis

↓ chronic inflammation

↓ photoreceptor collapse & vision loss

Source: Zheng 2023

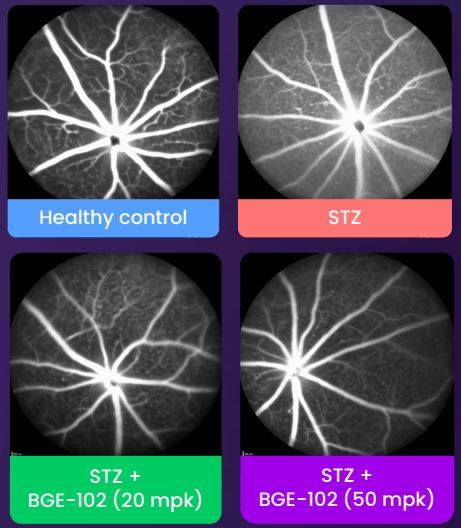
BIOAGE

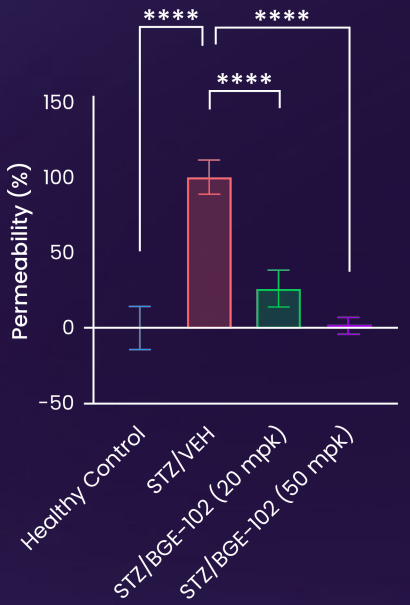
NLRP3

OPHTHALMOLOGY

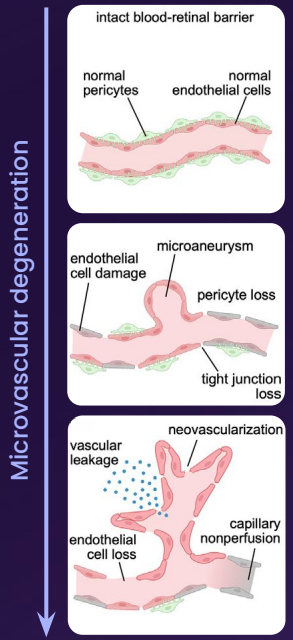
DME: oral delivery of BGE-102 preserved retinal vascular integrity in a preclinical model of diabetic retinopathy

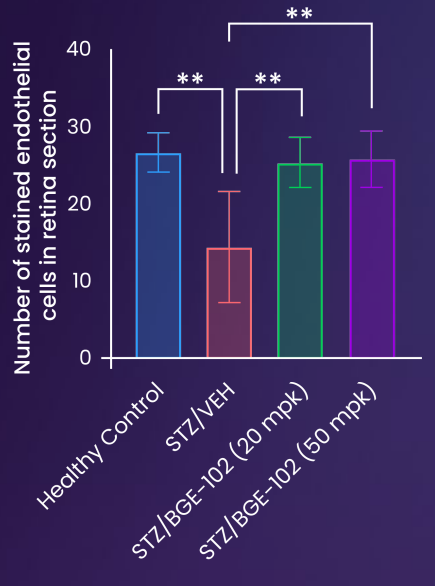
Retinal vascular permeability
measured via fluorescein angiography





Blood-retinal barrier integrity
claudin-5 IHC (endothelial tight junctions)





Note: Streptozotocin (STZ) diabetic mouse model; BGE-102 dosing initiated 11 days following last dose of STZ; assessments performed after 3 months of BGE-102 QD dosing; BGE-102 is more potent in human cells, for example 150–250x higher potency in microglia
Source: Wolf 2023

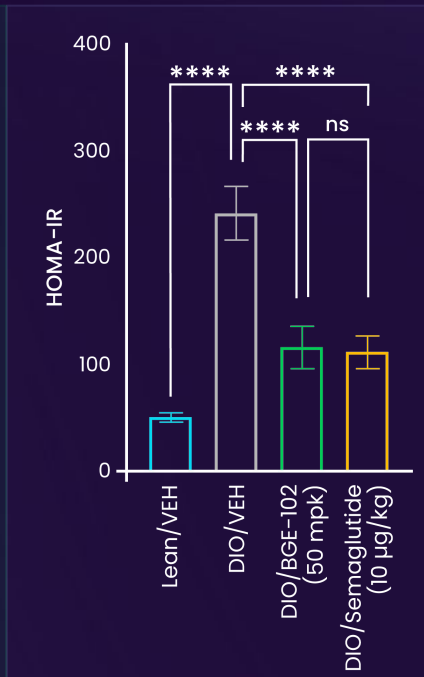
BIOAGE

NLRP3

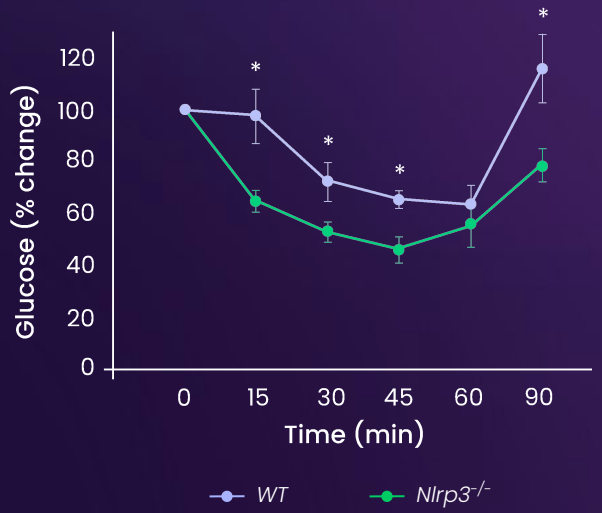
OPHTHALMOLOGY

DME: BGE-102 improved insulin sensitivity in obese mice, recapitulating mouse genetics & establishing potential to treat the key disease driver

BGE-102
HOMA-IR
(Day 26)



Nlrp3 -/-
Insulin tolerance test
(6 weeks)



Note: BGE-102 was dosed PO QD; semaglutide was dosed SC QD.
Source: Vandanmagsar 2011

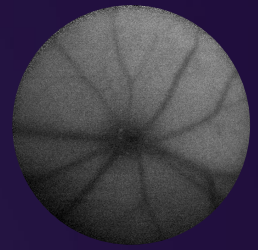
BIOAGE

NLRP3

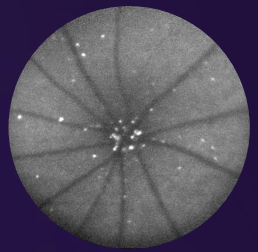
OPHTHALMOLOGY

GA: In a natural AMD preclinical model, oral delivery of a BGE-102 analog prevented age-related lipofuscin accumulation, a key disease feature

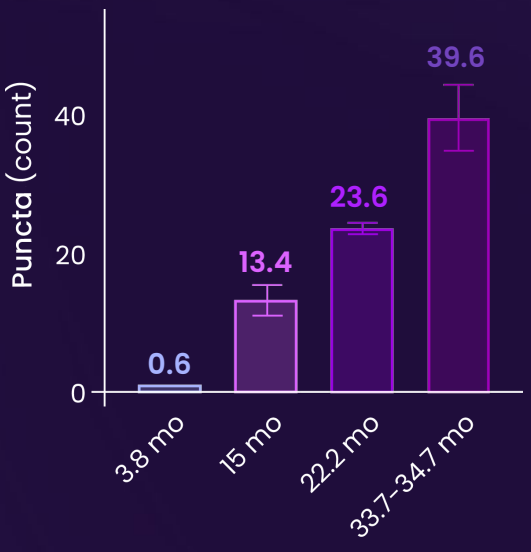
Natural AMD model
Lipofuscin punctae accumulate with age in outbred (HET3) mice



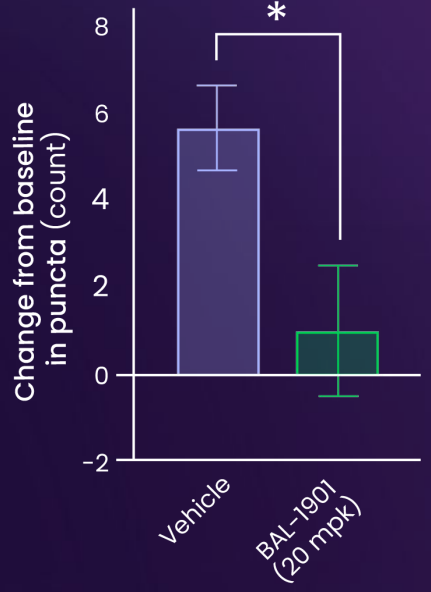
3 months



30 months



BAL-1901 prevented lipofuscin accumulation
with 3 mo of treatment beginning at 15 mo



BIOAGE

NLRP3

OPHTHALMOLOGY

Planned BGE-102 Phase 1b/2a POC in DME: goal is to demonstrate PD in the eye

Primary endpoint

- % change in intraocular IL-6

Exploratory

- Additional intraocular biomarkers
- Plasma biomarkers
- BCVA
- CST

BIOAGE

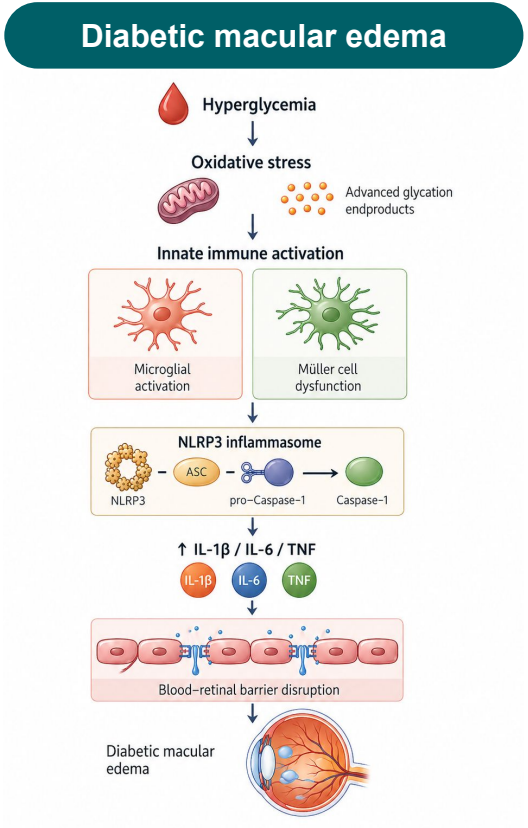
Brian Hafler, MD, PhD

Associate Professor
Ophthalmology & Visual Science,
Yale School of Medicine

Targeting the NLRP3 inflammasome in DME & GA

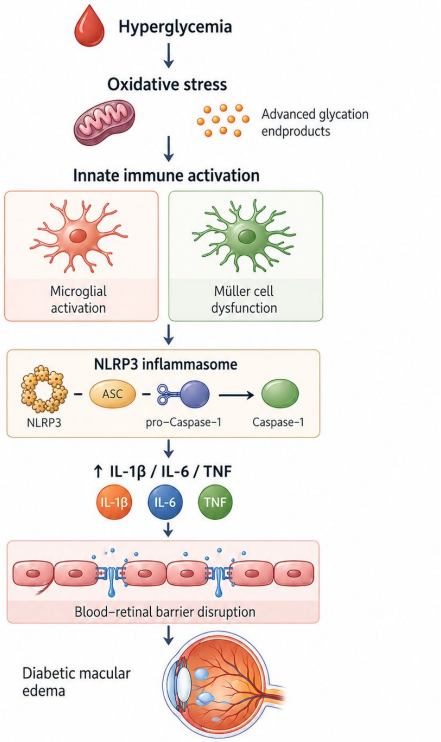
BIOAGE

The NLRP3 inflammasome plays a key role in DME & dry AMD / GA

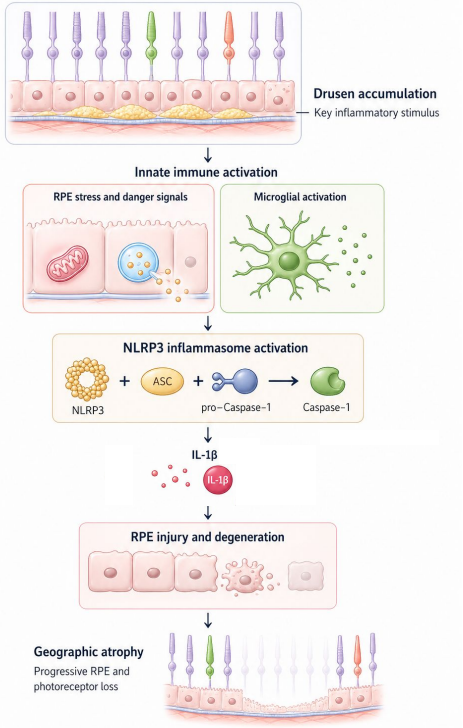


The NLRP3 inflammasome plays a key role in DME & dry AMD / GA

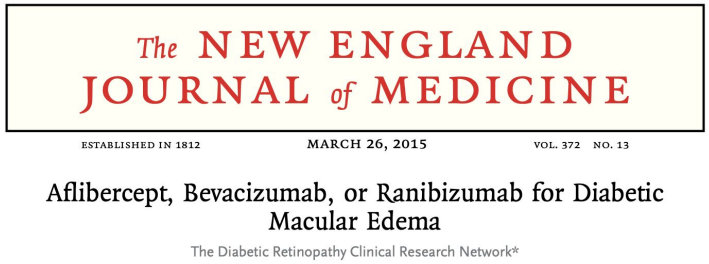
Diabetic macular edema



Geographic atrophy



Real-world DME remains burdensome despite anti-VEGF therapy



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MARCH 26, 2015 VOL. 372 NO. 13

Aflibercept, Bevacizumab, or Ranibizumab for Diabetic Macular Edema

The Diabetic Retinopathy Clinical Research Network*

Median of 9 intravitreal injections in year 1 across treatment arms

Treatment intensity and incomplete response still leave room for differentiated therapy

Why oral therapy matters

An effective oral adjunct or alternative could reduce procedure burden and expand options for incompletely controlled patients.

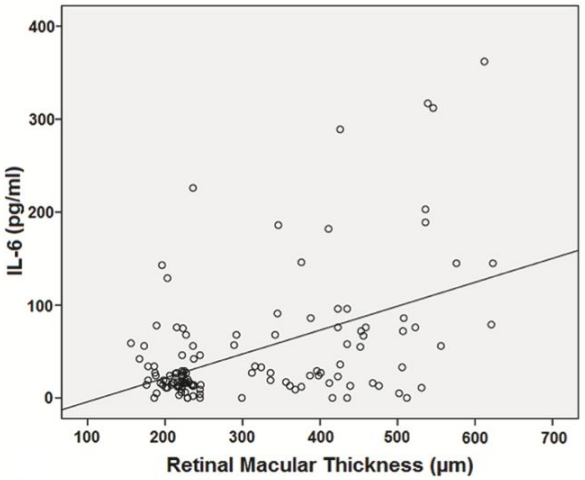
Varma et al., JAMA Ophthalmology, 2014; Payne et al., Eye, 2024; Fu DJ et al., Eye, 2025; Protocol T / NEJM 2015.
OCT morphology image from Kim K, Kim ES, Kwak HW, Yu SY. Long-term Outcomes of Diabetic Macular Edema Following Initial Intravitreal Ranibizumab Injection Based on Morphologic Pattern. 2016) under CC BY-NC.

Human aqueous humor data support inflammation-rich DME

Key findings

- Diabetic macular edema eyes showed higher IL-1β, IL-6, IL-8, MCP-1, IL-10, and VEGF than diabetic eyes without macular edema.
- These markers correlated with macular thickness, volume, and disease severity.
- The data demonstrate that diabetic macular edema is not only VEGF-driven but also inflammation-rich.

124-patient aqueous humor study

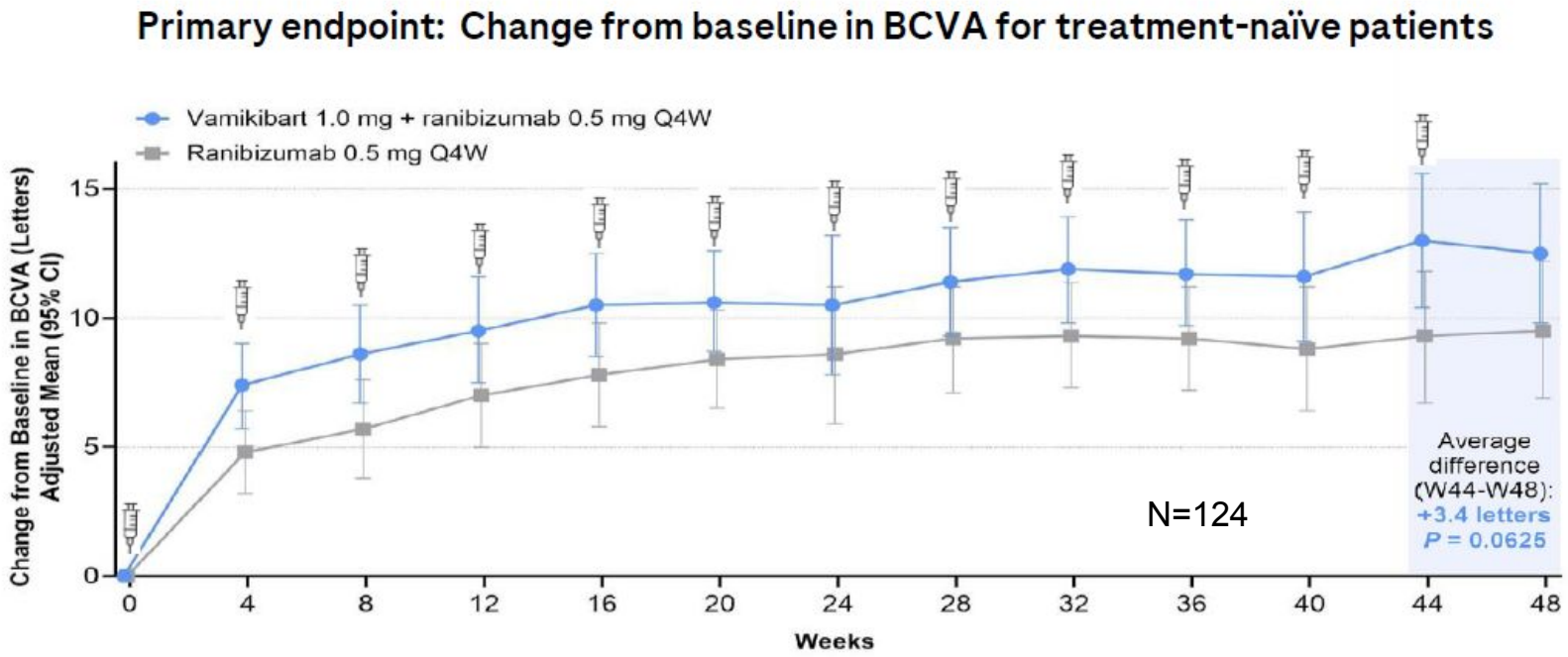


Dong N, Xu B, Chu L, Tang X. PLoS ONE. 2015;10(4):e0125329

Scientific implication

An upstream inflammasome-directed strategy is biologically plausible in inflammation-high diabetic macular edema.

Ph2 results for VEGF + IL-6 inhibition support role of inflammation in DME



Sources: Roche ASOPRS / AAO 2025 Virtual IR Event; ARVO 2026

Participants with ≥ 15 letter gain
44.7% ranibizumab + vamikibart vs. 28.6% ranibizumab

Exploratory clinical data support the inflammasome as a target in DME

Med

CellPress

Article

Oral lamivudine in diabetic macular edema: A randomized, double-blind, placebo-controlled clinical trial

Felipe Pereira,[1,8] Joseph Magagnoli,[2,8] Meenakshi Ambati,[3,8] Talita Fernandes de Oliveira,[1] Juliana Angélica Estevão de Oliveira,[1] Vinicius Oliveira Pesquero,[1] Lucas Zago Ribeiro,[1] Dante Akira Kondo Kuroiwa,[1] Fernando Korn Malerbi,[1] Sergio Atala Dib,[4] Nilva Bueno Moraes,[1] Michel Eid Farah,[1] Eduardo Buchele Rodrigues,[1] and Jayakrishna Ambati[5,6,7,9,*]

+9.8 letters

lamivudine at week 4

-1.8 letters

placebo at week 4

Interpretation

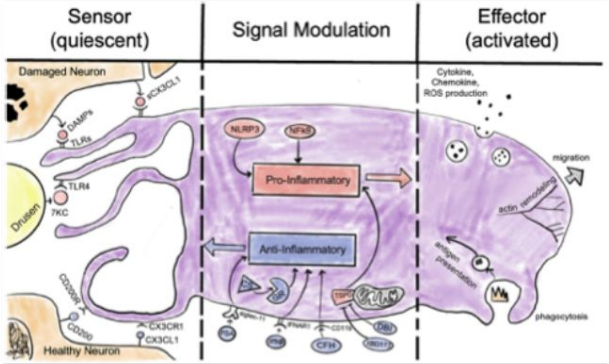
These data support the relevance of inflammasome modulation in diabetic macular edema

BGE-102 is positioned upstream of inflammatory drivers in DME

BGE-102

- Oral inhibitor of the NLRP3 inflammasome
- Intended to reduce inflammatory cytokines and vascular leakage
- Could complement anti-VEGF or potentially serve as an alternative in selected patients
- Oral dosing is the key differentiator if efficacy is shown

Mechanism



Seminars in Immunopathology (2022) 44:673–683
https://doi.org/10.1007/s00281-022-00939-3

REVIEW

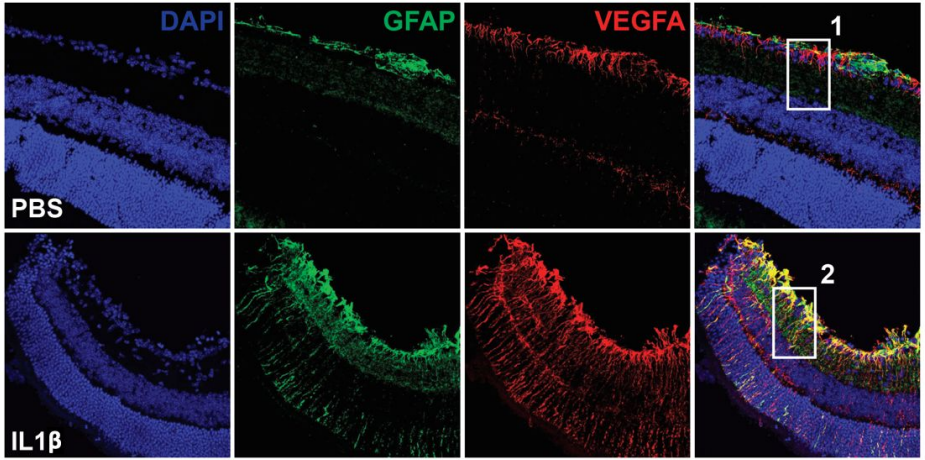
Check for updates

Glial-mediated neuroinflammatory mechanisms in age-related macular degeneration

Rahul M. Dhodapkar[1] · Diego Martell[2] · Brian P. Hafler[2,3]

Received: 1 December 2021 / Accepted: 14 April 2022 / Published online: 5 May 2022

IL-1β, a downstream effector of the NLRP3 inflammasome, drives retinal VEGF in vivo



nature communications

Article

https://doi.org/10.1038/s41467-023-37025-7

Single-cell analysis reveals inflammatory interactions driving macular degeneration

Received: 12 May 2022

Accepted: 27 February 2023

Published online: 05 May 2023

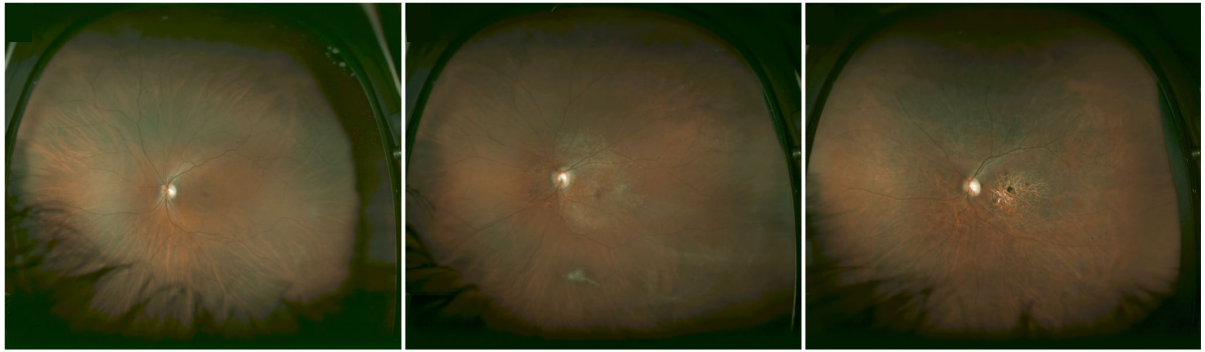
Check for updates

Manik Kuchroo[1,19], Marcello DiStasio[2,19], Eric Song[3,19], Eda Calapkulu[3], Le Zhang[1,4], Maryam Ige[5], Amar H. Sheth[5], Abdelilah Majdoubi[3], Madhvi Menon[6], Alexander Tong[7], Abhinav Godavarthi[8], Yu Xing[3], Scott Gigante[9], Holly Steach[10], Jessie Huang[7], Guillaume Huguet[11,12], Janhavi Narain[13], Kisung You[14], George Mourgkos[3], Rahul M. Dhodapkar[5], Matthew J. Hirn[15,16], Bastian Rieck[17], Guy Wolf[11,12], Smita Krishnaswamy[7,14,20] ✉ & Brian P. Hafler[2,3,18,20] ✉

Mechanistic implication

If upstream NLRP3 inhibition reduces IL-1β signaling, it may also reduce VEGFA-linked vascular leakage.

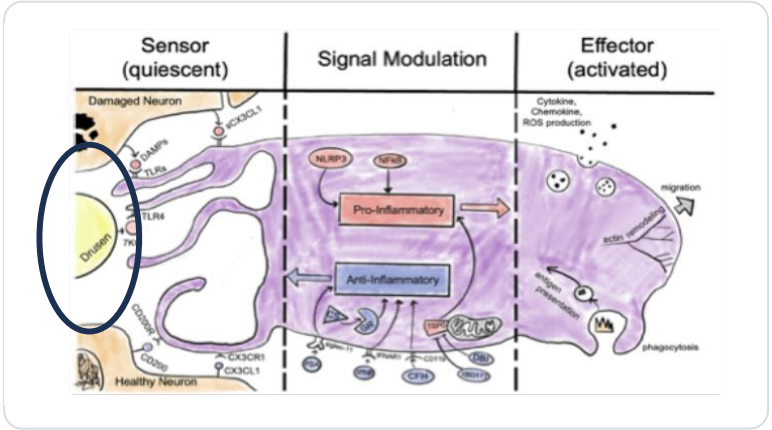
Drusen are inflammatory drivers of the NLRP3 inflammasome in AMD



Hafler BP. Retina. 37(3):417, 2017

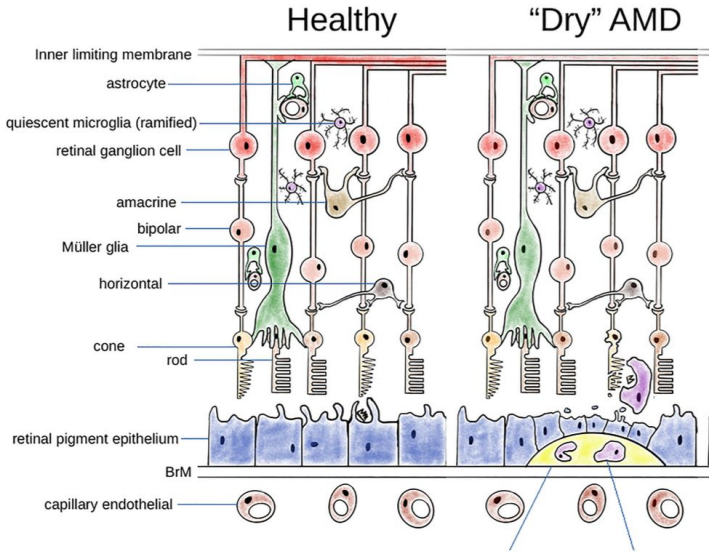
Drusen from donor AMD eyes activate the NLRP3 inflammasome.

Doyle, S., Campbell, M., Ozaki, E. et al. Nat Med 18, 791–798 (2012).



Dhodapkar RM, Martell D, Hafler BP. Glial-mediated neuroinflammatory mechanisms in age-related macular degeneration. Semin Immunopathol. 2022 Sep;44(5):673-683.

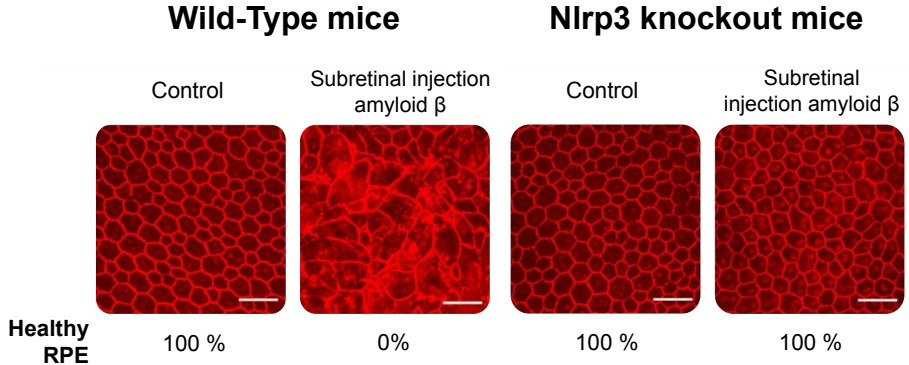
NLRP3 knockout rescues RPE degeneration in mouse model of GA



Drusen contain amyloid β

Pro-inflammatory component of geographic atrophy

Dhodapkar RM, Martell D, Hafler BP. Glial-mediated neuroinflammatory mechanisms in age-related macular degeneration. Semin Immunopathol. 2022 Sep;44(5):673-683.



Narendran, S., Pereira, F., Yerramothu, P. et al. Nucleoside reverse transcriptase inhibitors and Kamuvudines inhibit amyloid-β induced retinal pigmented epithelium degeneration. Sig Transduct Target Ther 6, 149 (2021).

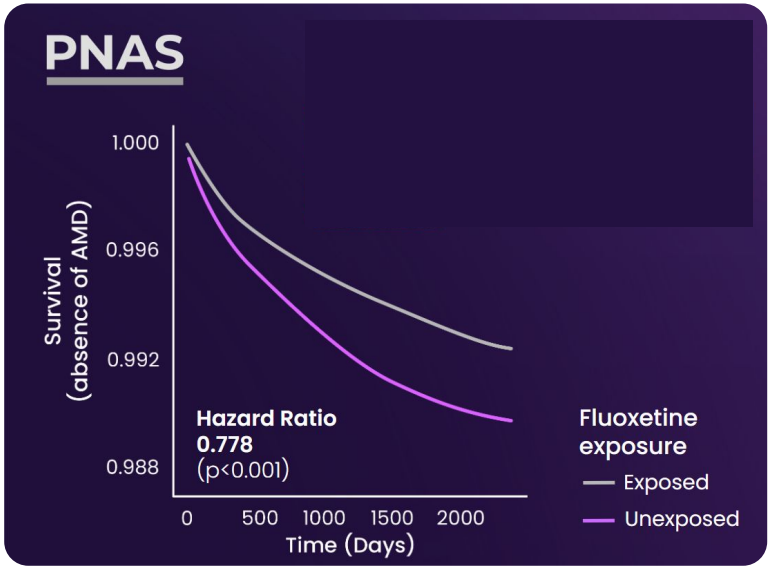
Inflammasome inhibition in humans: promising early signals of potential efficacy in GA

Phase 1 clinical trial suggests potential for inflammasome inhibitor intravitreal implant K8

53% reduction in geographic atrophy lesion growth with intravitreal K8 implant compared to untreated eyes at month 3 in low dose cohort (Inflammasome Therapeutics) n=10

Syfovre (C3 complement inhibitor) **17%** reduction in lesion growth at 12 months in Phase 3 trials OAKS and DERBY

Decreased incidence of AMD observed with fluoxetine, a weak NLRP3 inhibitor in a retrospective analysis



Ambati M, Apicella I, Wang SB, Narendran S, Leung H, Pereira F, Nagasaka Y, Huang P, Varshney A, Baker KL, Marion KM, Shadmehr M, Stains CI, Werner BC, Sadda SR, Taylor EW, Sutton SS, Magagnoli J, Gelfand BD. Identification of fluoxetine as a direct NLRP3 inhibitor to treat atrophic macular degeneration. Proc Natl Acad Sci U S A. 2021 Oct 12;118(41):e2102975118.

Source: https://inflam.com/images/pdfs-doc/GA_K9_First_Cohort_Low_Dose_Results.pdf

David Boyer, MD

Senior Partner

Retina-Vitreous Associates Medical Group

DME & GA treatment landscape

BIOAGE

Diabetic macular edema (DME)

DME is the leading cause of vision loss working aged adults

~27M patients globally

~25% of all diabetes patients develop DME within 10 years of diagnosis

60 years average age of diagnosis

Systemic disease, retinal manifestation

Elevated HbA1c is the largest risk factor for DME progression

Characterized by **central vision loss with distortion that impacts activities of daily living:** driving, reading, recognizing faces & work productivity

Anti-VEGFs are well-established but have many unmet needs

Anti-VEGFs are standard of care, however:

- Poor compliance given in-office monthly to bi-monthly intravitreal injections
- Response rates are lower in DME vs. wet AMD given prominent role of underlying inflammation

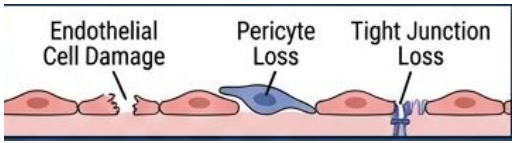
Sources: WESDR, Klein 1984, DCCT 1993, Cheung 2019, Angiogenesis Foundation

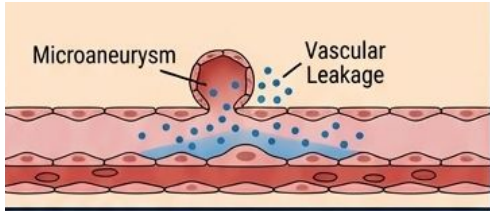
DME progresses due to hyperglycemia-driven vascular fluid leakage in the macula, directly causing central vision loss

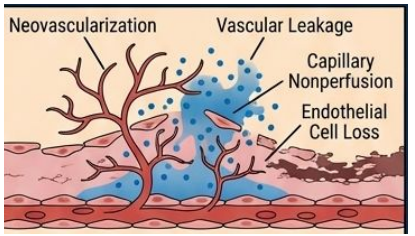
Chronic hyperglycemia causes **breakdown of the blood-retinal barrier**

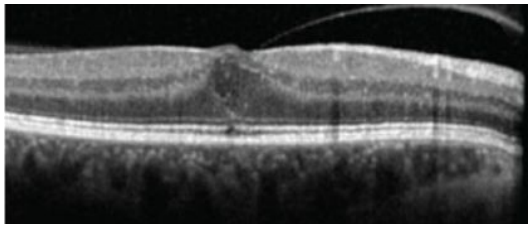
Fluid accumulates in the macula, visible on OCT

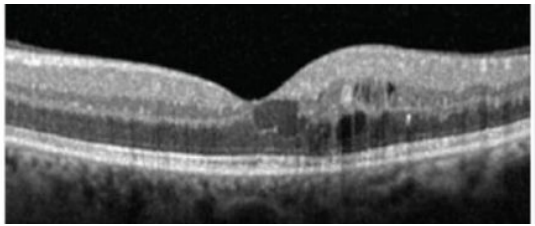
Persistent fluid **damages photoreceptor layer** (EZ)
Leads to central vision loss

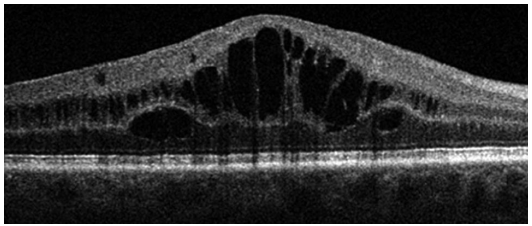












Sources: Ashraf 2020, Wittwer 2024

~45% of diagnosed patients are treated with anti-VEGFs; ~55% are observed and not actively treated

Diagnosed DME patients

~55% Observed

- Patients with **no to mild VA impairment,** but progressive structural damage on OCT scan
- Guidelines recommend observation-only; in these patients, **treatment burden outweighs benefit**

~45% Treated

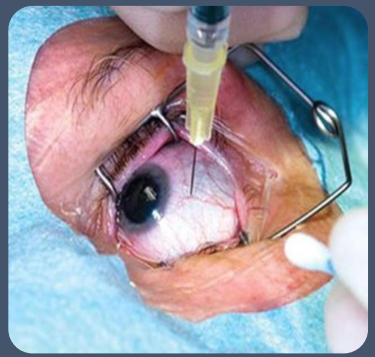
- Anti-VEGFs are generally initiated once disease becomes center-involving **with vision loss**

1st line	2nd line
IVT Anti-VEGF	**IVT steroids**

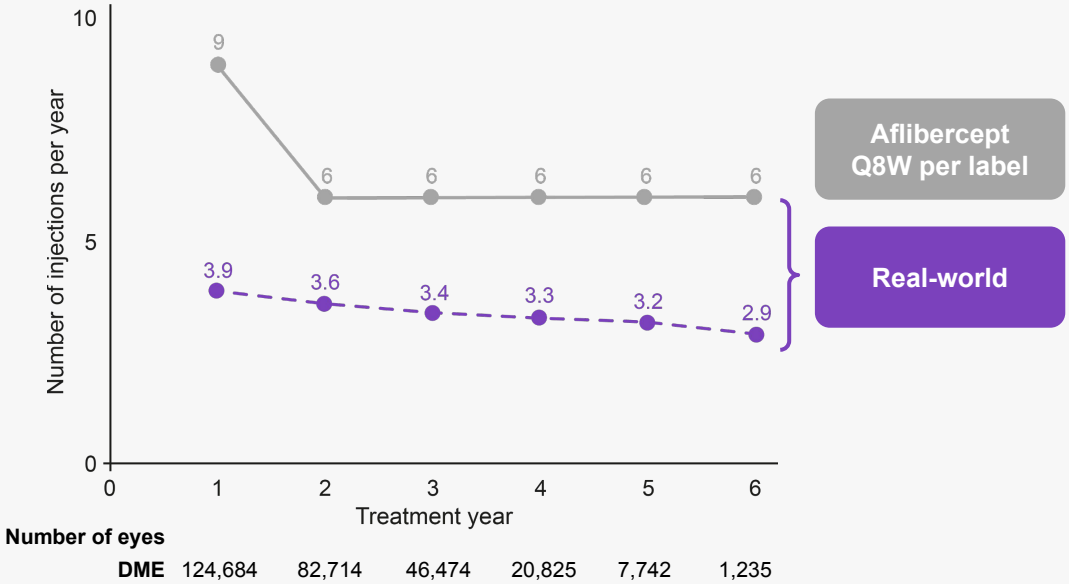
Sources: Baker 2019, AAO 2024

Compliance is a key challenge …

Injections are invasive and inconvenient; in the real world, they are not administered as frequently as they should be



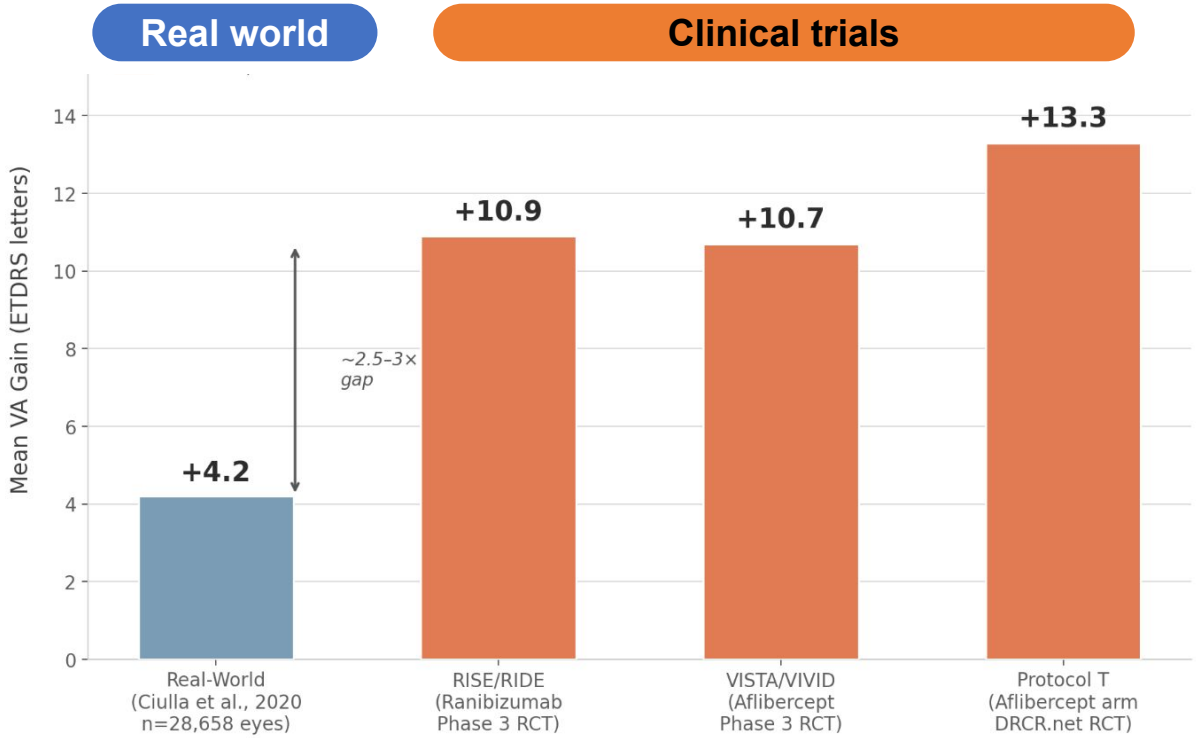
Number of anti-VEGF injections received
Real-world (IRIS) vs aflibercept 2 mg q8W label



Number of eyes						
DME	124,684	82,714	46,474	20,825	7,742	1,235

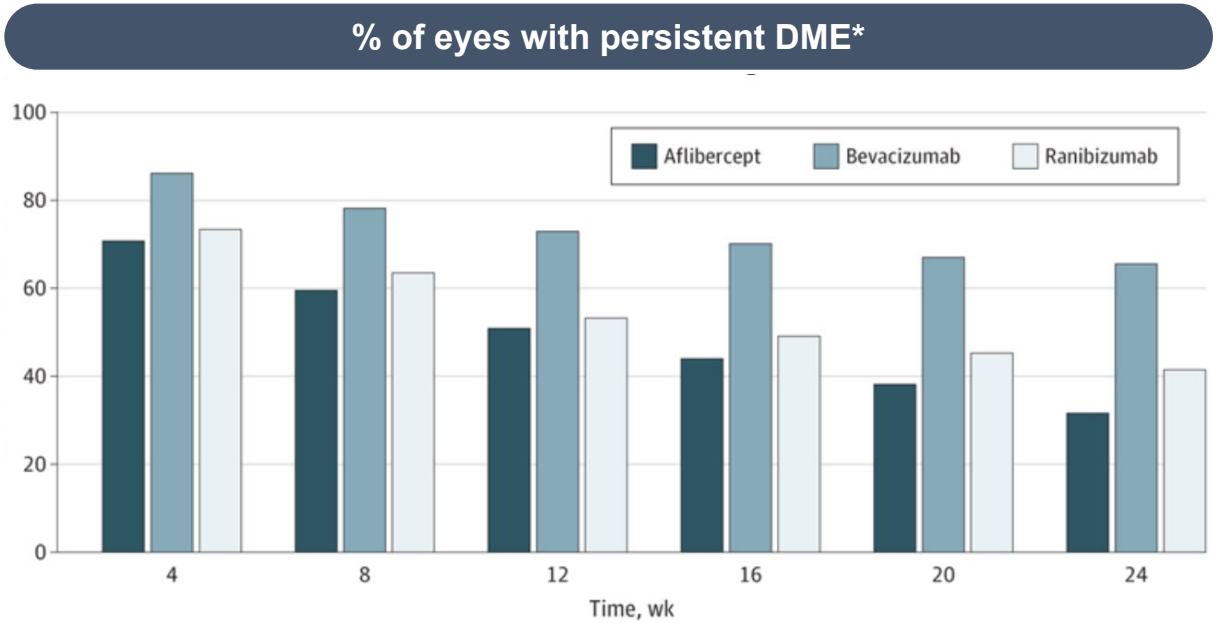
Sources: Kuo 2024, Lancet Photo Library

… that has led to 50-70% lower VA gains in the real world vs. in trials



Sources: Ciulla 2020, Wells 2015, Boyer 2015, Korobelnik 2014

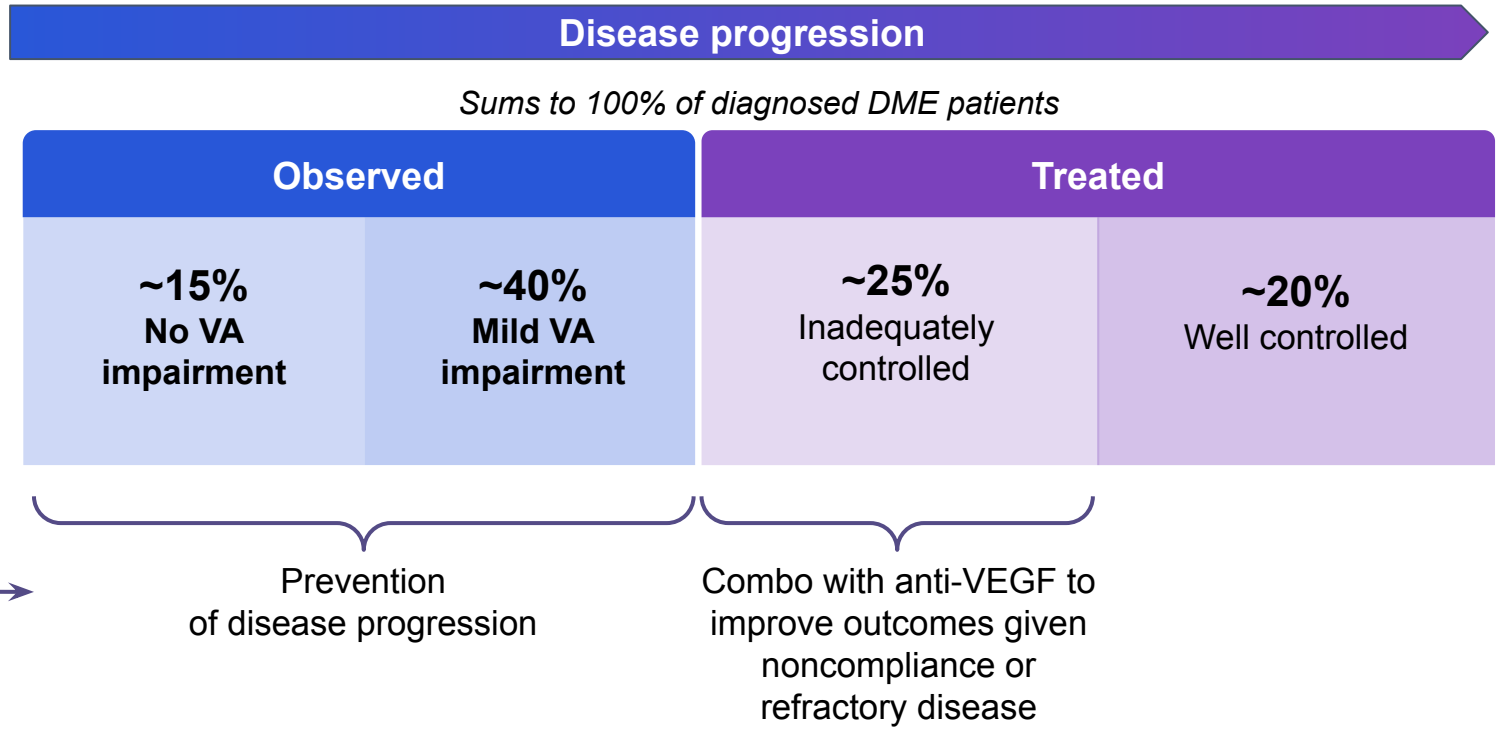
Even with strong compliance, ~50% of patients have persistent fluid on anti-VEGF therapy



**Note: Persistent DME was defined as central subfield thickness ≥ 250 µm*

Source: Bressler 2018

An effective oral therapy could benefit not only patients currently treated with anti-VEGFs but could allow for treatment of earlier-stage disease not currently treated



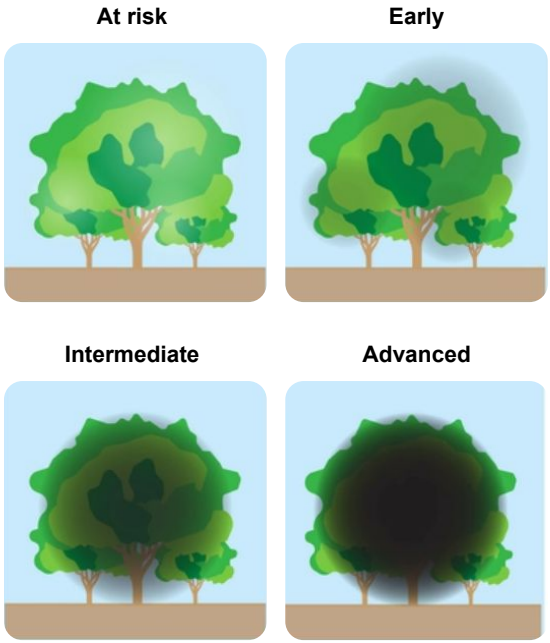
Source: Bressler 2018

Geographic atrophy (GA)

Geographic atrophy is a leading cause of blindness in the elderly, characterized by irreversible central vision loss

>8M patients globally	**Most severe form of AMD** (~20% of all cases) **Est. 18M patients by 2040:** rapidly growing **Age 75:** median age of onset
A leading cause of blindness	Characterized by **loss of central vision, poor low-light acuity, blurry or distorted vision** *From diagnosis:* **~5 years:** time to legal blindness (median) **~10 years:** time to total vision loss (median)
No treatments preserve VA	**No approved therapies have shown vision benefits to date**, unlike in other retinal diseases

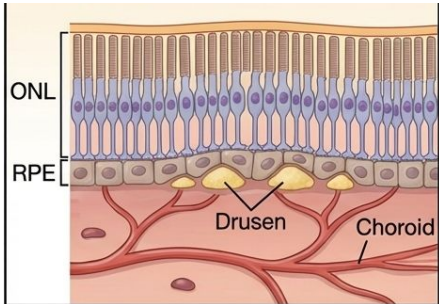
GA vision loss progression

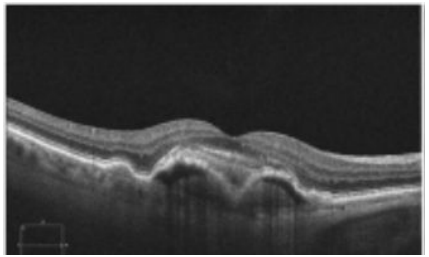


Sources: Wong 2014, Khanani 2023

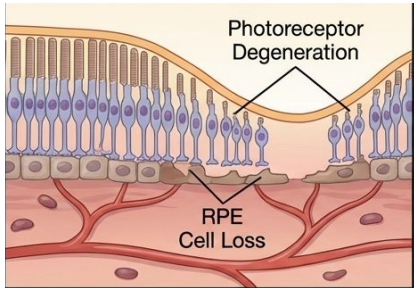
GA is characterized by continuous neurodegenerative decline

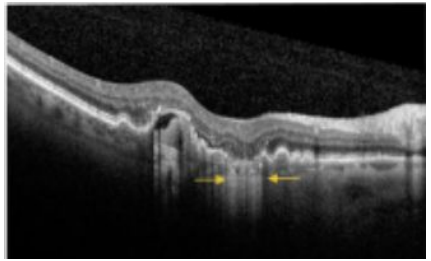
Drusen build up & initial RPE dysfunction



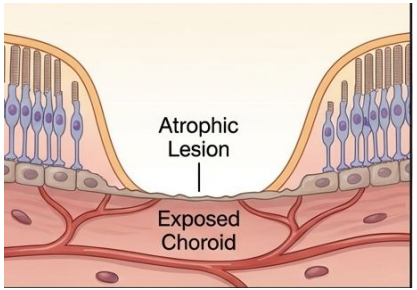


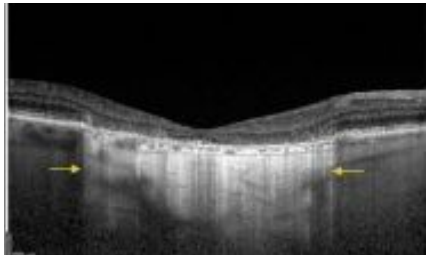
RPE cell death leads to photoreceptor degeneration
Photoreceptors convert light into vision signals





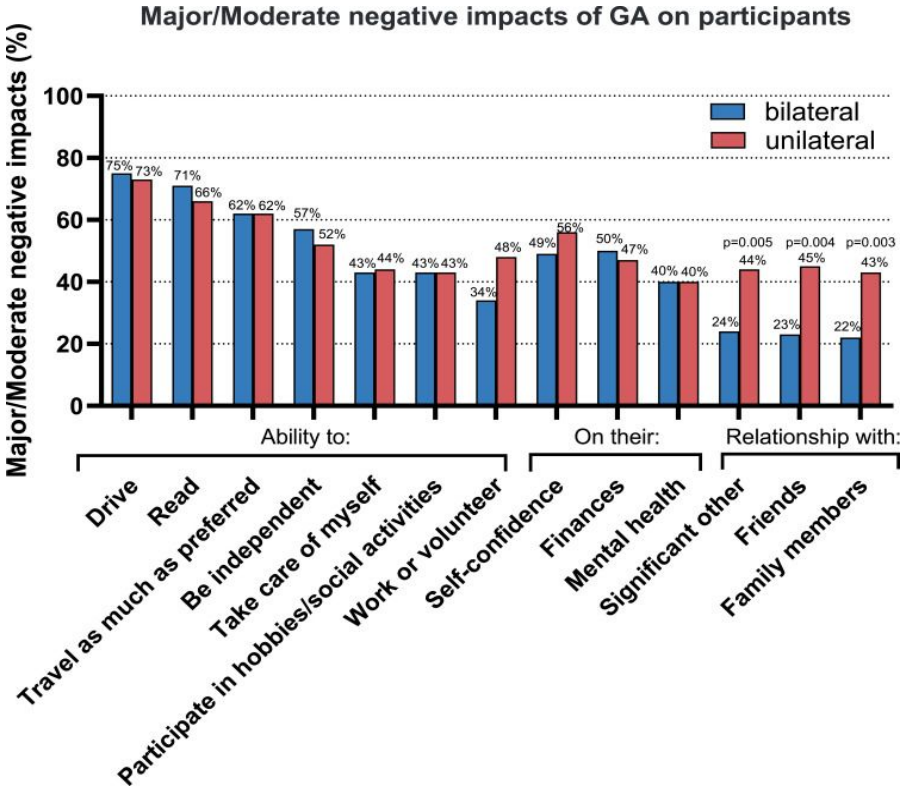
Expansion of atrophic lesions
Lesions signal established GA and lead to VA loss when they cover the fovea





Sources: Sadeghi 2026

The effects of GA on quality of life can be devastating, making it difficult or impossible for independent living

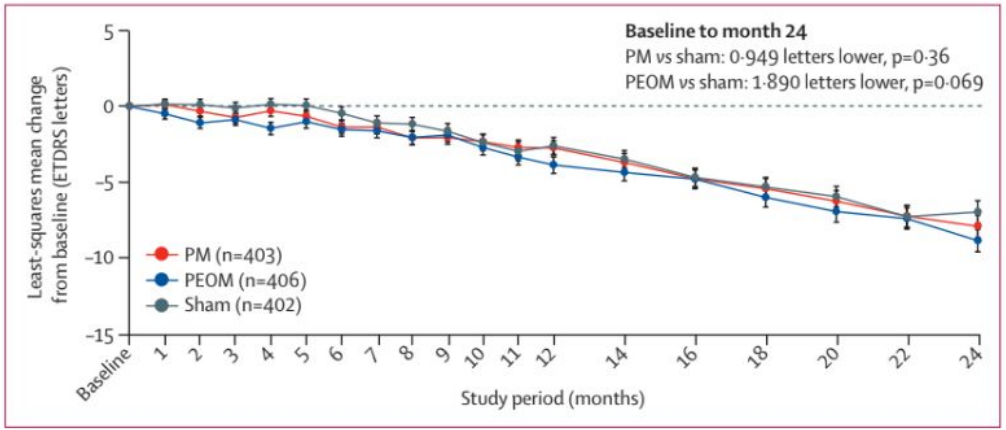


Source: Bakri 2024,: Global Geographic Atrophy Insights Survey (N=203)

Approved complement inhibitors have been shown to reduce lesion growth by 15-20%, no vision preservation shown to date

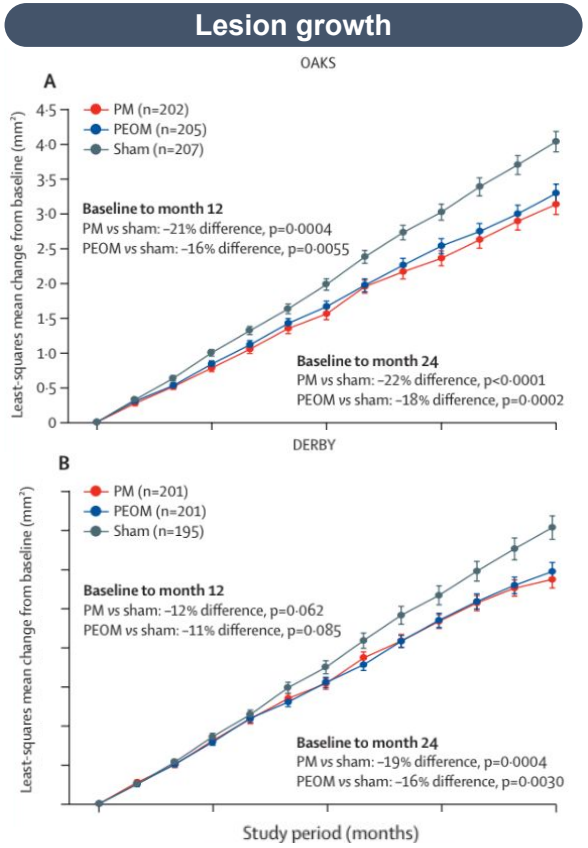
Syfovre OAKS/DERBY Pooled Analysis-
Showed declines in VA similar to sham
but significant lesion growth rate reduction

Visual acuity



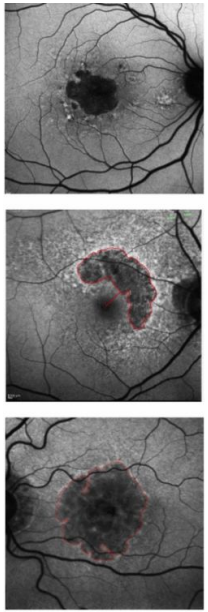
Source: Khanani 2023

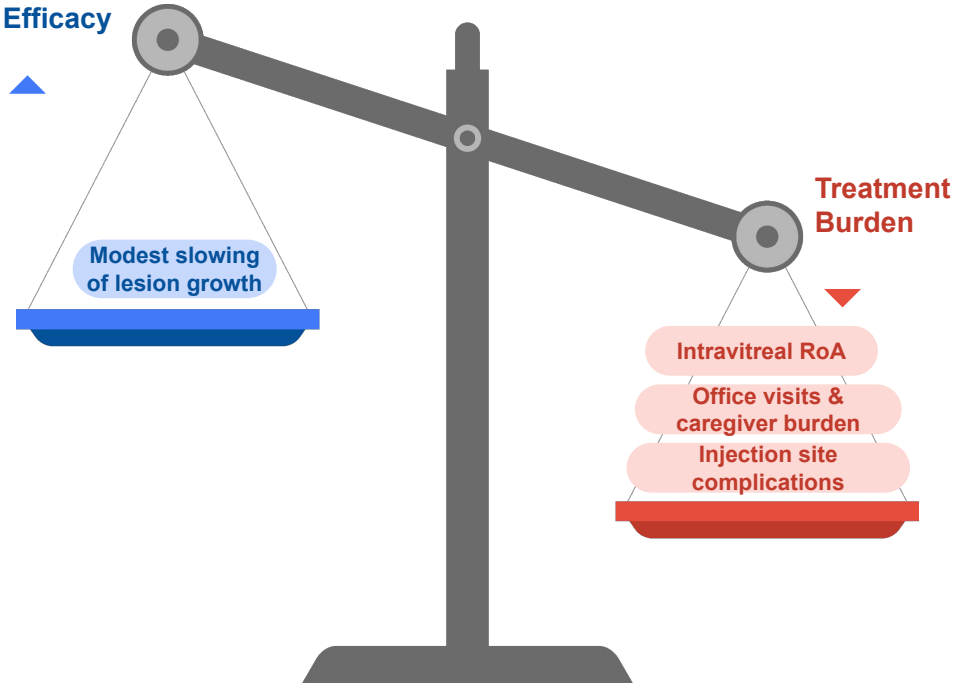
Lesion growth



Complement inhibitors are currently prescribed in ~10% of patients

Lesion growth in GA

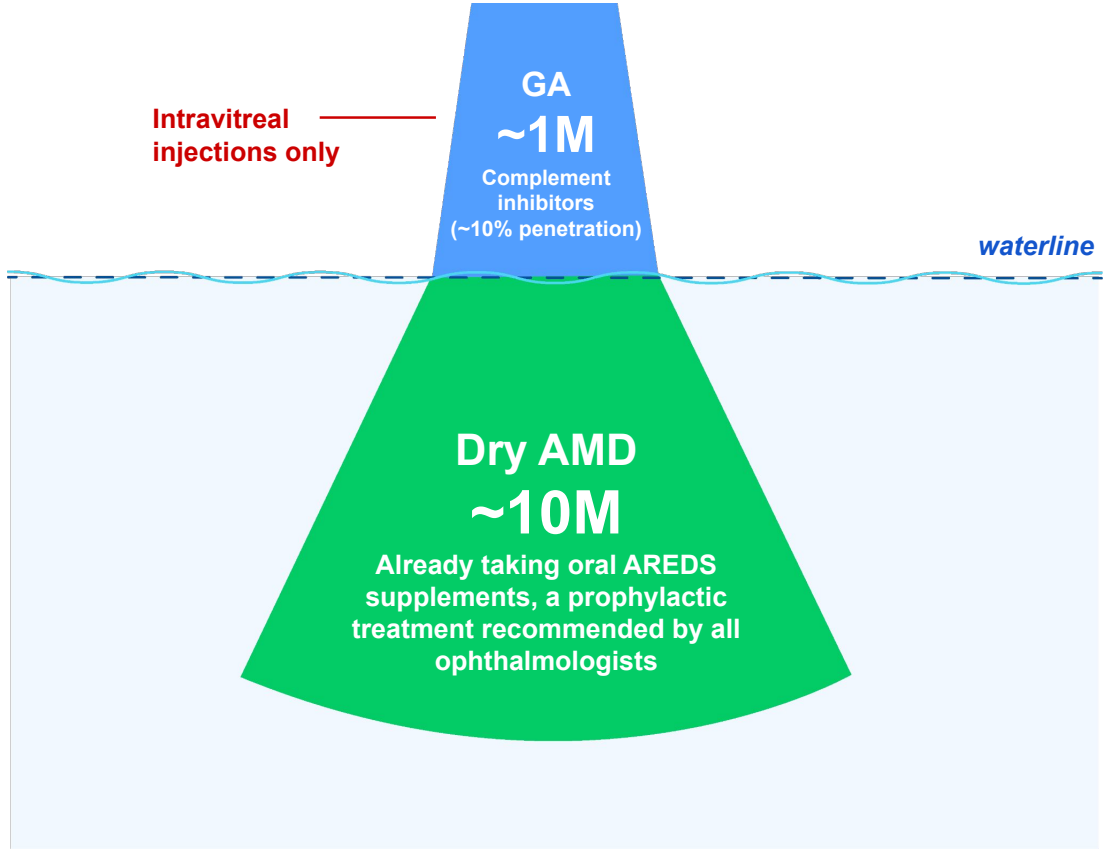




An effective oral therapy could rebalance efficacy and treatment burden

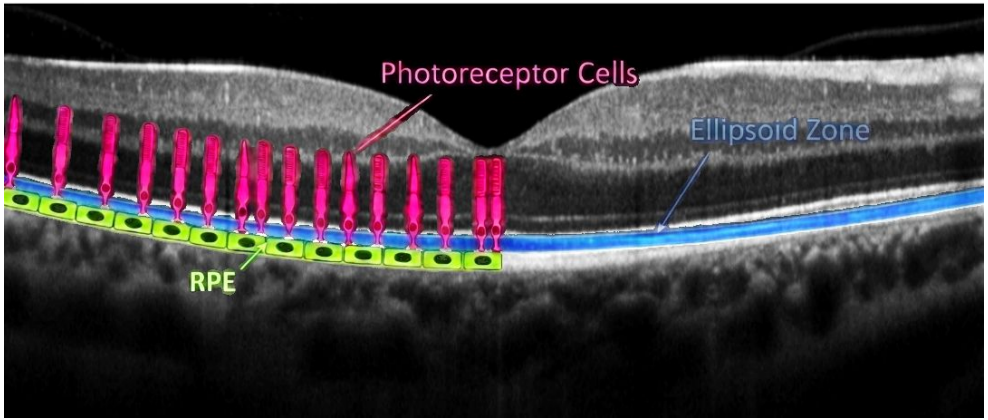
Source: Wong 2014, Khanani 2023, Mai 2024

An oral therapy could be used in earlier stages of dry AMD patients to slow GA progression



Emerging approvable endpoint: the ellipsoid zone (EZ)

- **Definition**: The EZ is a hyperreflective, photoreceptor rich band observed on OCT, serving as a crucial biomarker for assessing retinal health and vision loss
- **Clinical relevanc**e: EZ attenuation (loss) precedes GA formation and is the strongest predictor of visual function
- **Regulatory validation**: FDA has previously recognized EZ attenuation as an approvable endpoint:
  - Stealth Bio advanced to Ph3 directly because of EZ attenuation data in GA (a secondary endpoint)
  - Basis of ENCELTO approval (cell therapy for macular telangiectasia)
- **Adoption**: rapidly expanding across KOLs, clinical use & regulatory



Sources: Stoney Creek OCT 2026

Summary & Q&A

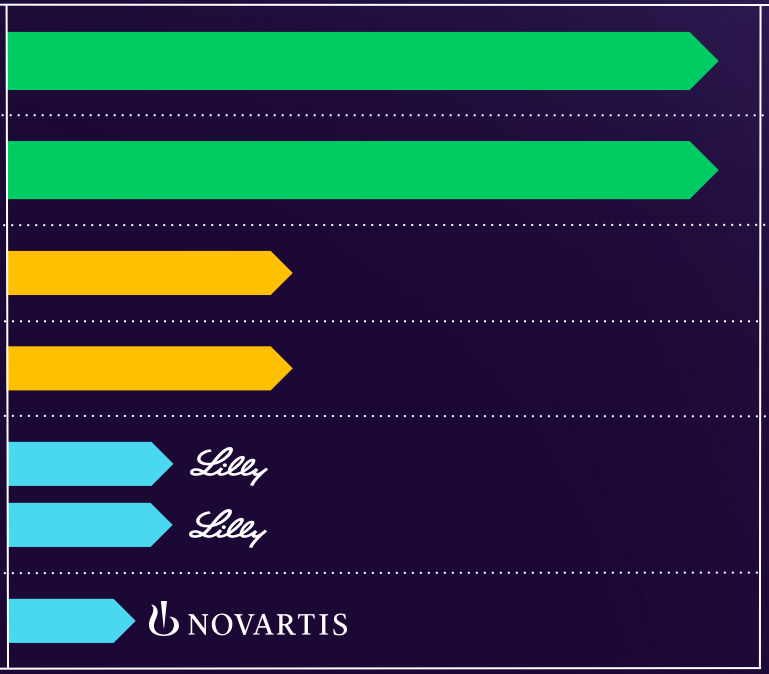
Kristen Fortney, PhD
Co-Founder & CEO

BIOAGE

Pipeline overview

Leveraging the BioAge platform to address key unmet needs in metabolic aging

Program	Mechanism of action	Target dosing	Indication	Anticipated milestones
BGE-102	NLRP3 inhibitor (CNS penetrant)	Oral QD	CV risk	**CV risk Phase 2 results** H2:2026
			Diabetic macular edema	**DME Phase 1b/2a initiation** mid-2026 **results** mid-2027
APJ	APJ agonist	Oral QD	Obesity	**IND submission** 2026 YE
		SQ QW	Obesity	
Program 1	Undisclosed		Cardio-metabolic - various	
Program 2	Undisclosed			
Target discovery	Multiple targets	-	-	



Strong balance sheet with $285.1M in cash, cash equivalents, and marketable securities as of December 31, 2025

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Q&A

BIOAGE