BIOAGE

Corporate Presentation
April 2026

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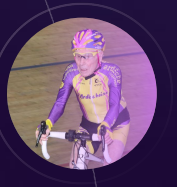
BIOAGE

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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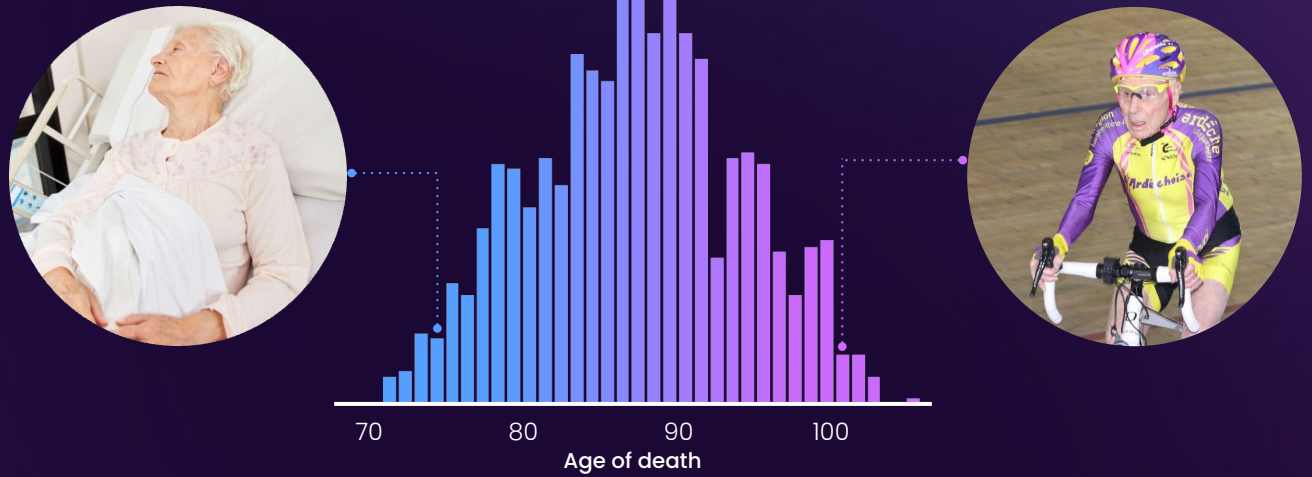
The BioAge Platform:
Harnessing human data to uncover molecular pathways and novel drug targets that drive metabolic aging and impact healthspan



A 50+ year natural human experiment

150M+	25K+	50+
Molecular data points	Profiles generated	Years of follow-up

Detailed healthspan trajectories

Physical function
- Grip strength
- Walking speed
- Mobility

Metabolism
- BMI
- Skinfold thickness
- Waist / hip circumference

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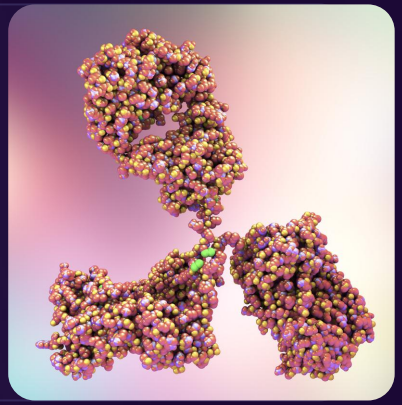
Validated platform with two ongoing pharma collaborations to advance novel aging targets

Lilly mAb discovery

mAb drug discovery collaboration for two novel BioAge targets

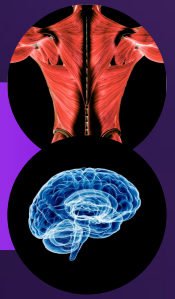


NOVARTIS Target discovery

Novel target discovery collaboration focused on the intersection of exercise & healthy aging

Exercise response

Longitudinal healthspan outcomes



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The BioAge leadership team: diverse professional experience across the biopharma ecosystem and an established track record of success

LEADERSHIP TEAM

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Peng Leong, PhD, MBA
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BJ Sullivan, PhD
Chief Strategy Officer

~800
Clinical trials*

~130
INDs*

~95
US regulatory approvals*

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Berkeley
University of California
University of Toronto
Buck
Live better longer.
Principia Biopharma
Schrödinger
LOXO Oncology at Lilly
XOMA
gsk
Takeda
Merck
AstraZeneca
Aisling Capital
LEK

* As of May 31, 2024

NLRP3

BGE-102 overview

Phase 1 results

Therapeutic areas

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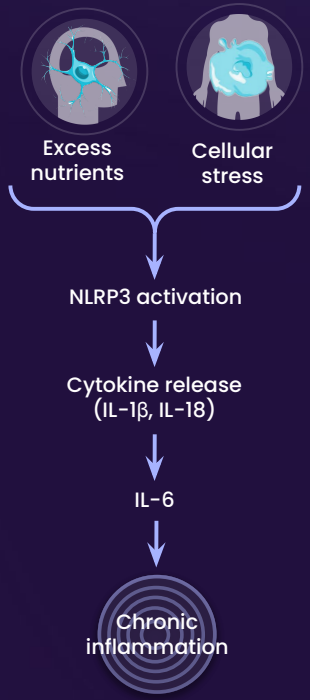
Chronic NLRP3 activity drives disease & predicts poor human longevity

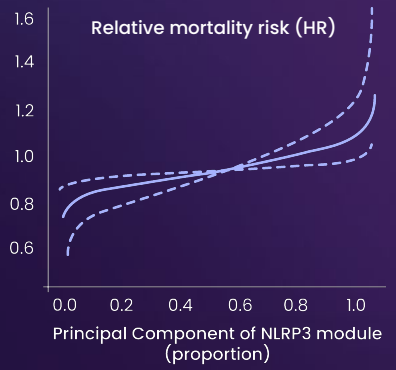
NLRP3 overview

- 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

NLRP3

BGE-102 OVERVIEW

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 CRP (<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 hsCRP > 3 mg/L; ** based on Phase 1 healthy volunteer MAD at Day 14*

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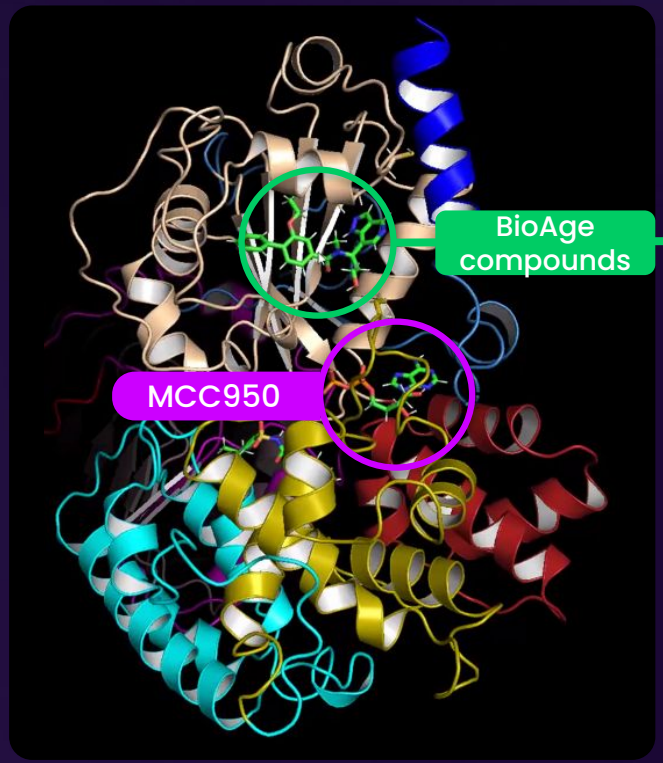
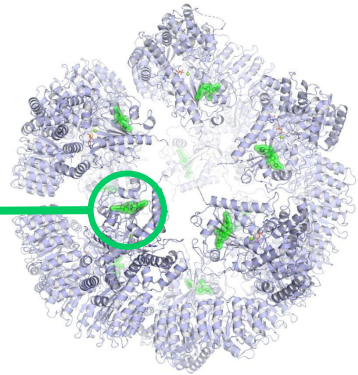
BGE-102 OVERVIEW

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

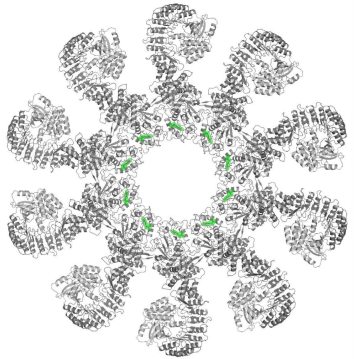
Unique binding site



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

BGE-102 overview

Phase 1 results

Therapeutic areas

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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 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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BGE-102 overview

Phase 1 results

- Healthy volunteer SAD / MAD
- Obese MAD

Therapeutic areas

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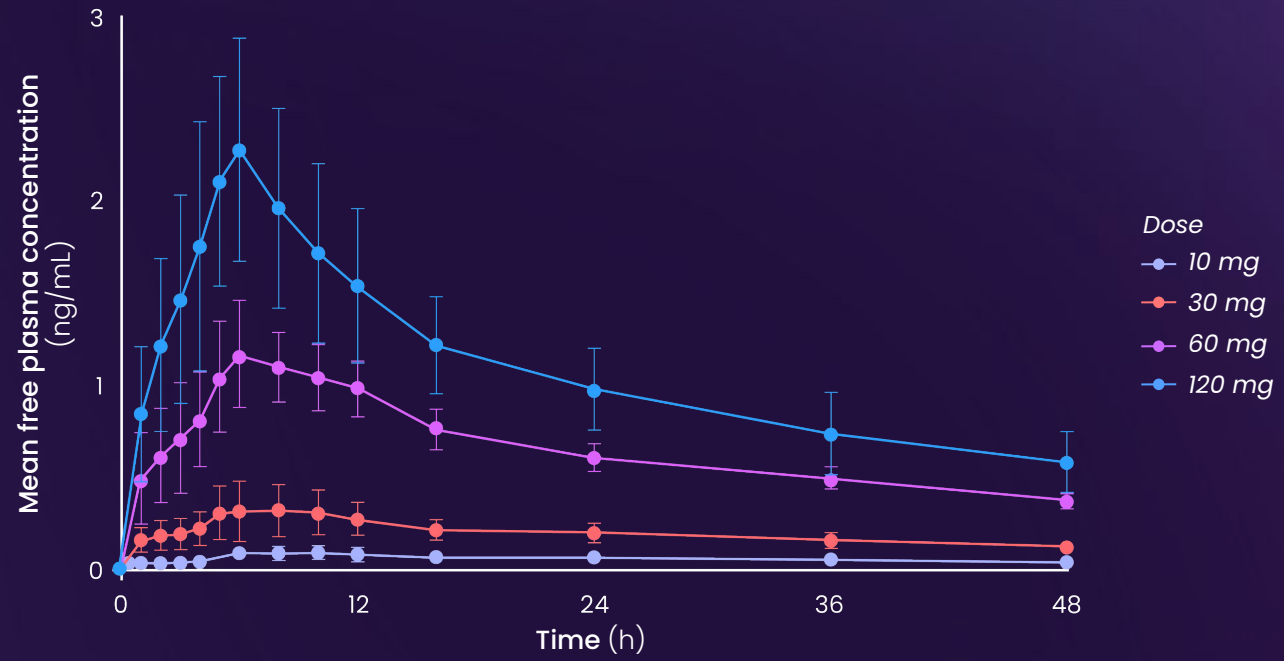
HEALTHY VOLUNTEER SAD / MAD

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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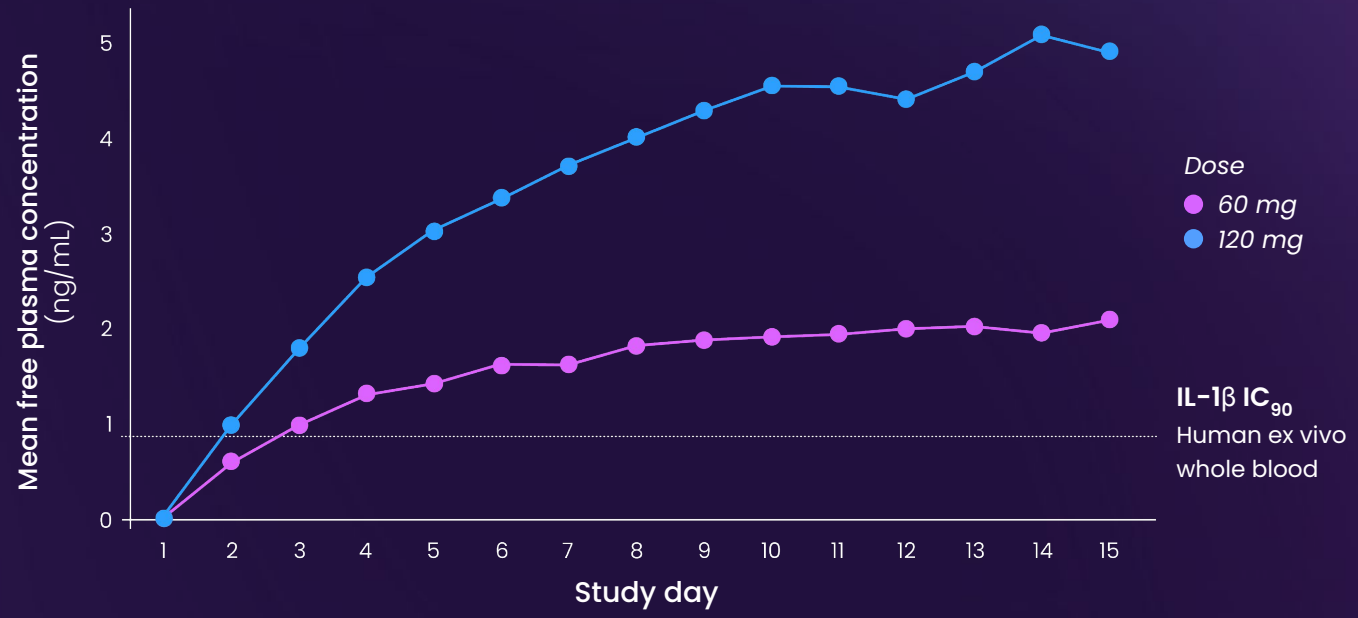
HEALTHY VOLUNTEER SAD / MAD

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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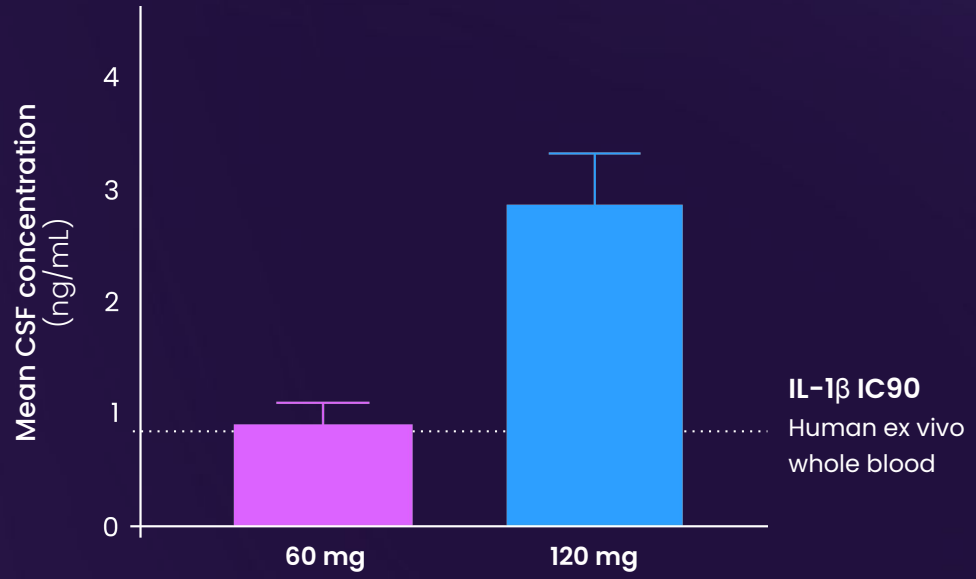
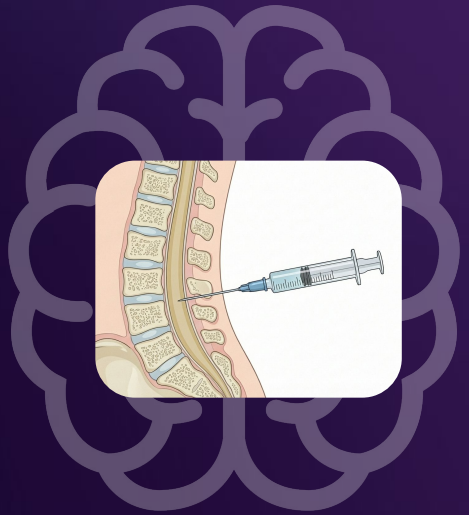
HEALTHY VOLUNTEER SAD / MAD

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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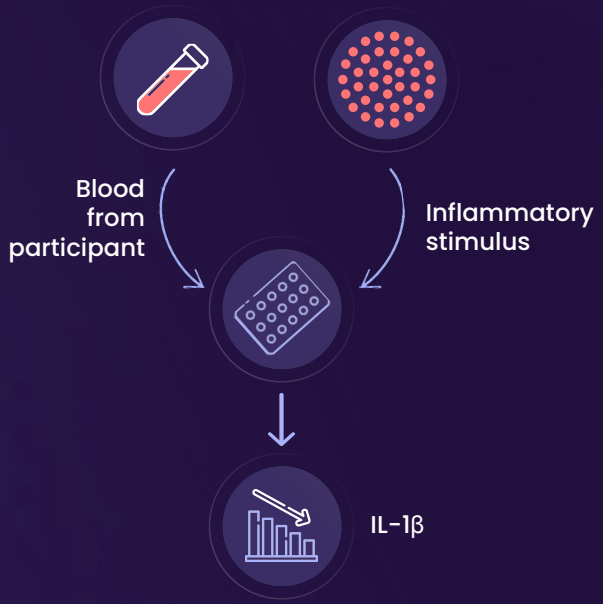
HEALTHY VOLUNTEER SAD / MAD

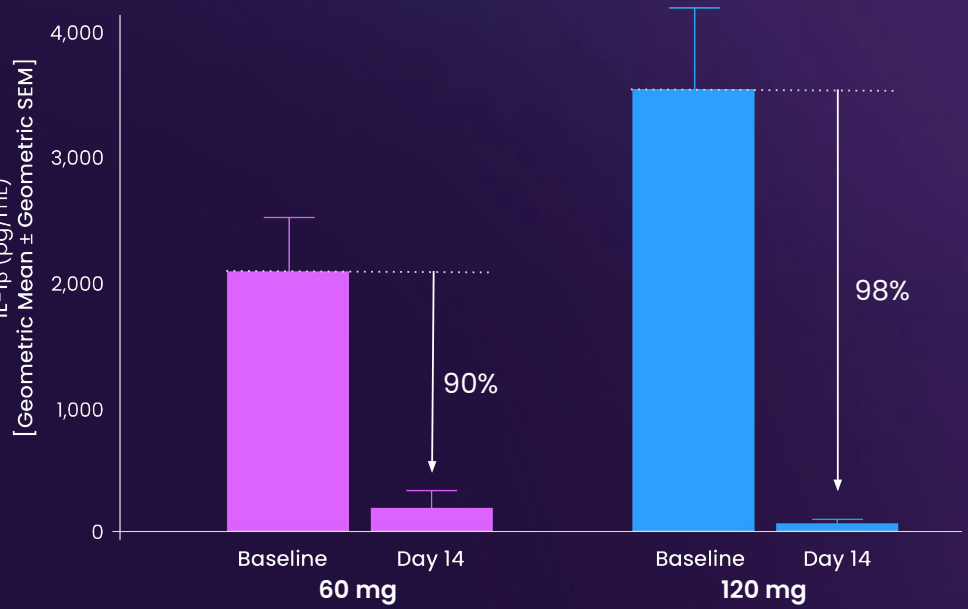
PK / PD: 90-98% suppression of IL-1β at BGE-102 trough in MAD cohorts

Ex vivo whole blood stimulation



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

baseline vs Day 14



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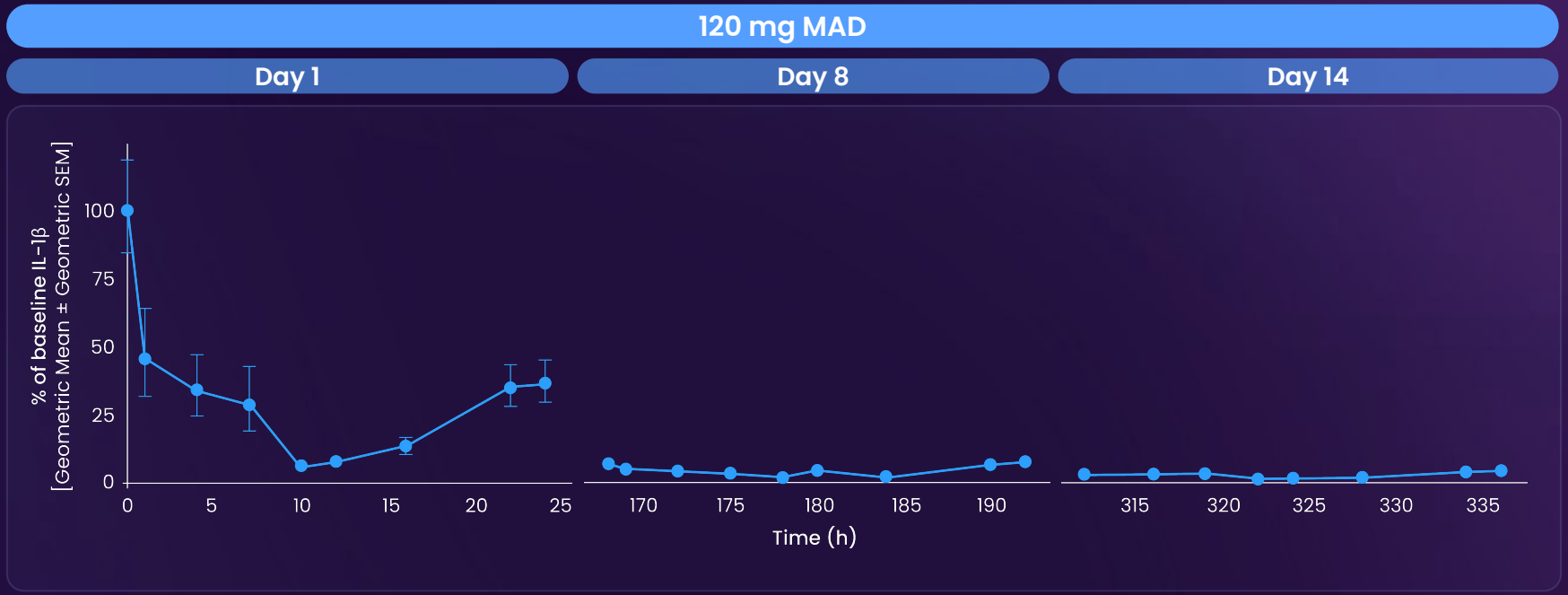
HEALTHY VOLUNTEER SAD / MAD

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



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BGE-102 overview

Phase 1 results

- Healthy volunteer SAD / MAD
- Obese MAD

Therapeutic areas

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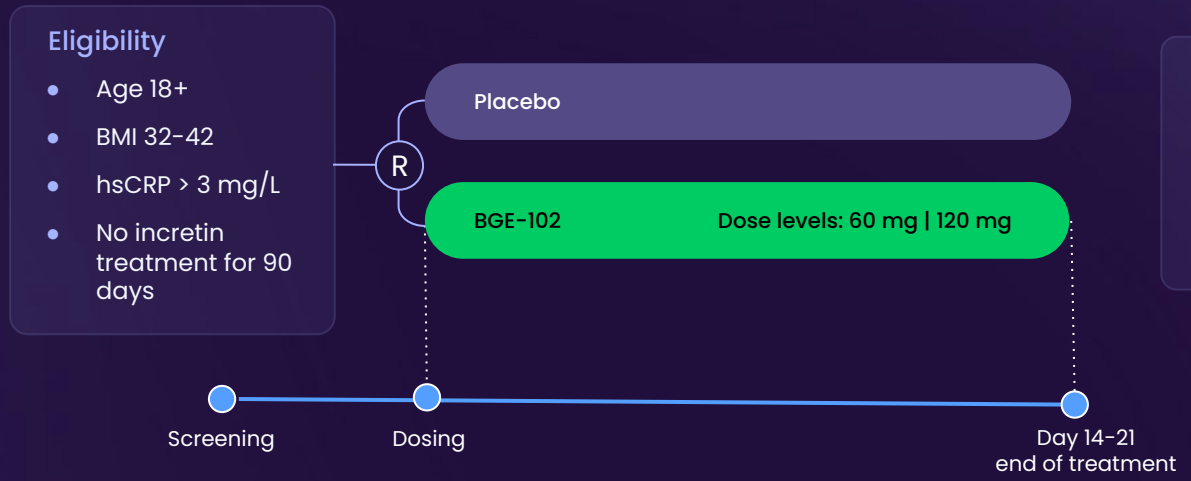
OBESE MAD

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



Primary endpoints

- PK, safety / tolerability

Exploratory endpoints

- Inflammatory and metabolic biomarkers, including hsCRP

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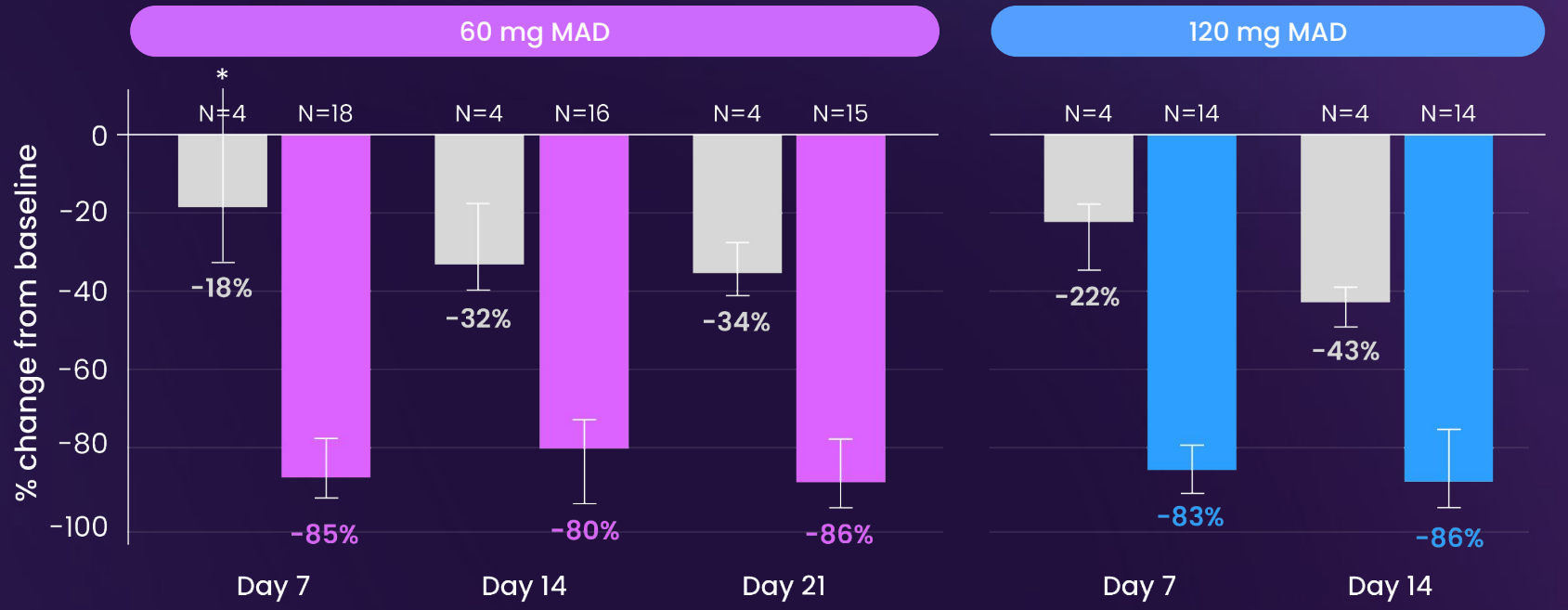
OBESE MAD

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*

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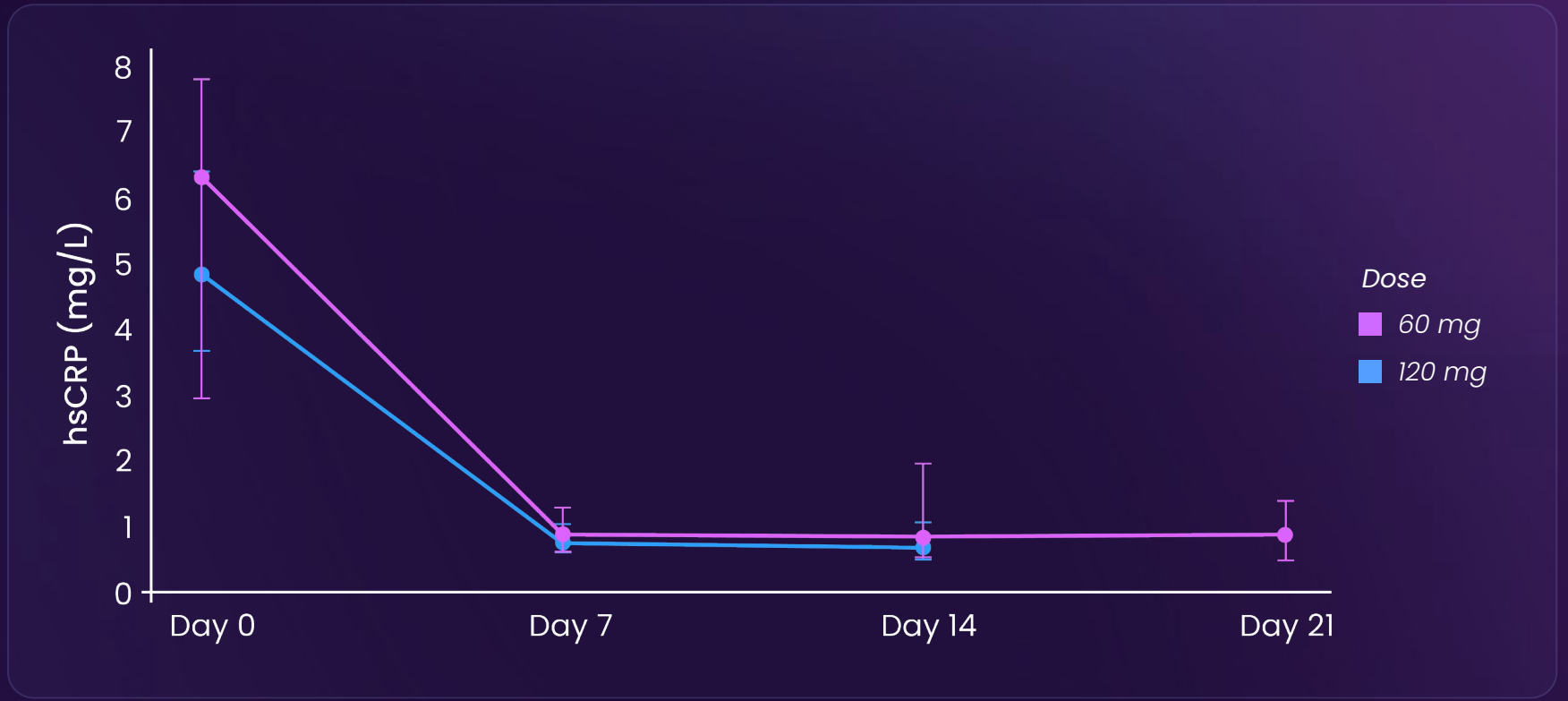
OBESE MAD

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

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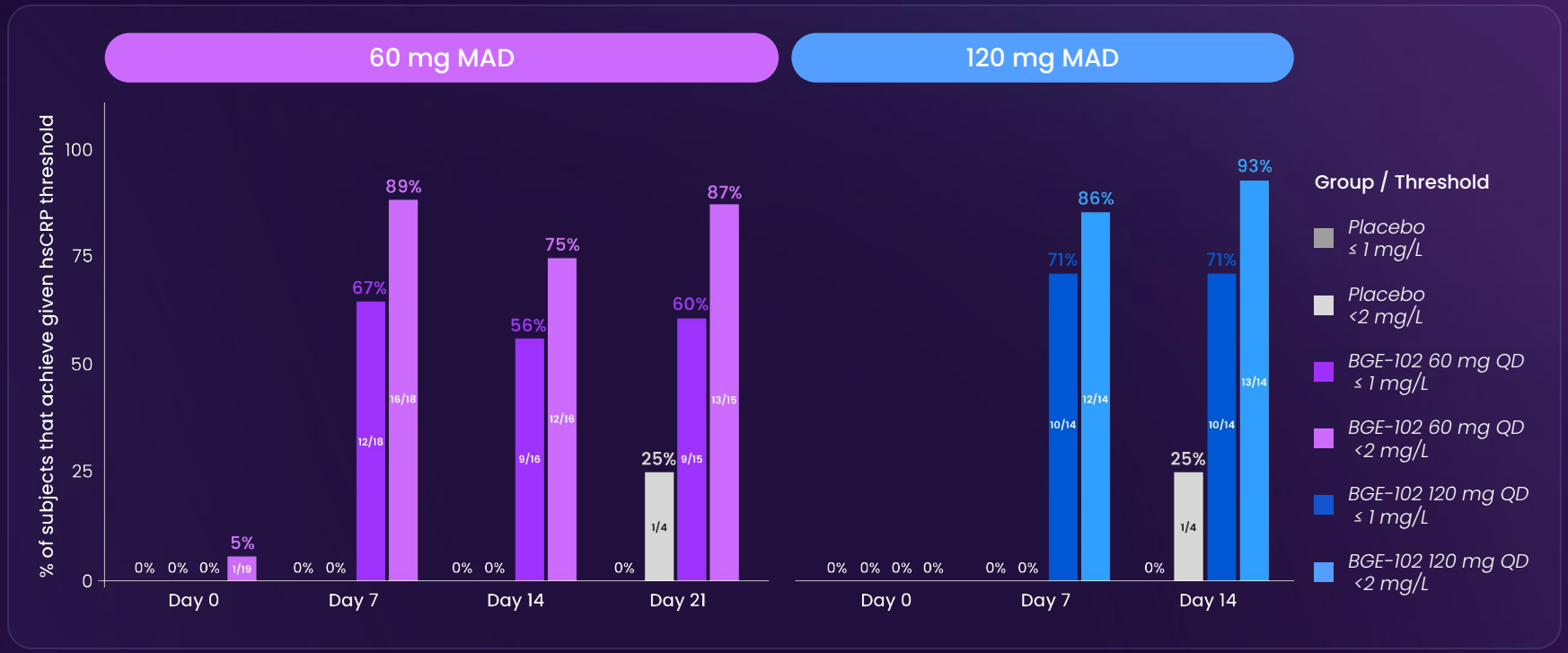
OBESE MAD

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

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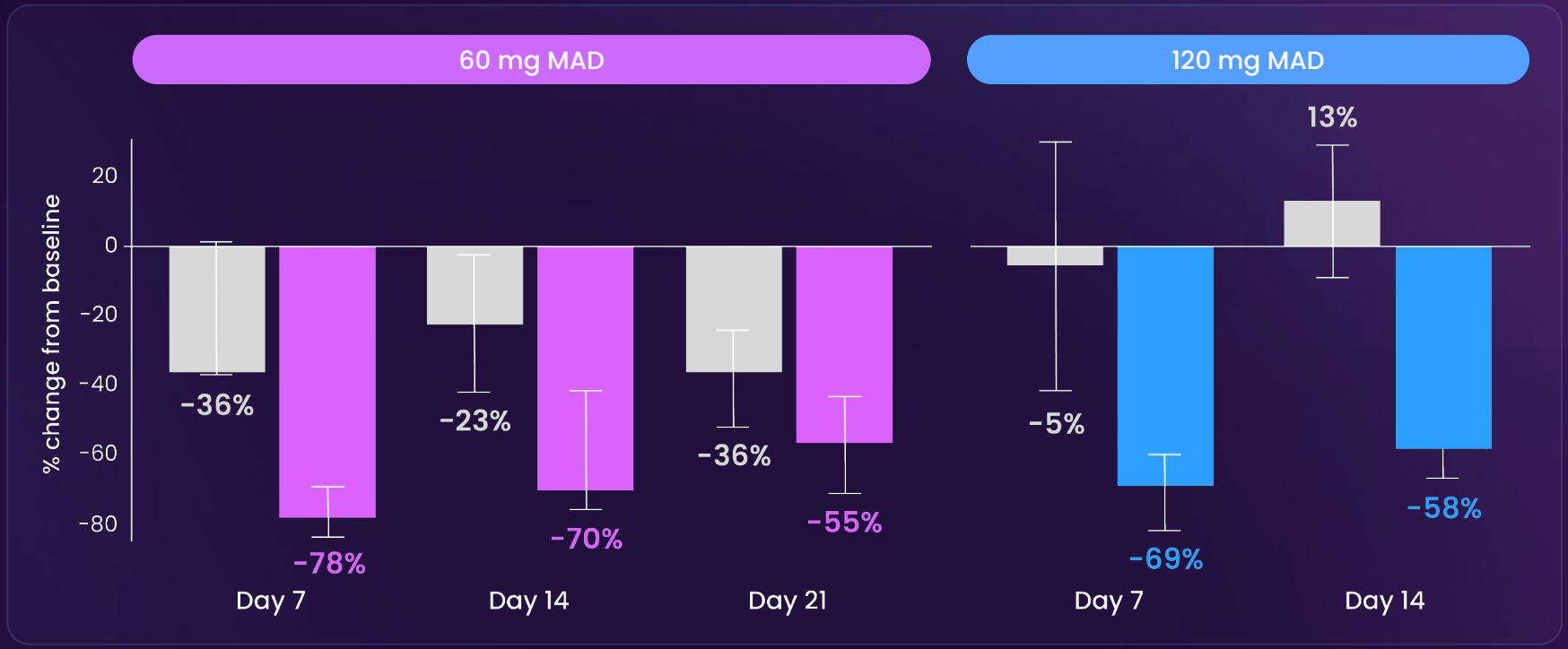
OBESE MAD

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

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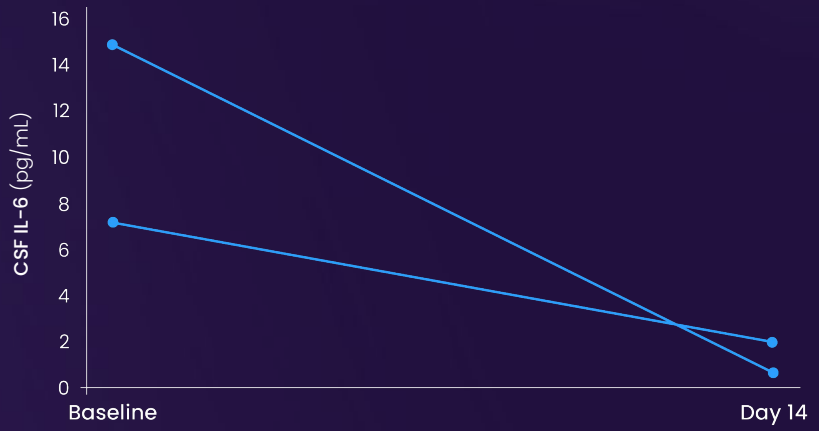
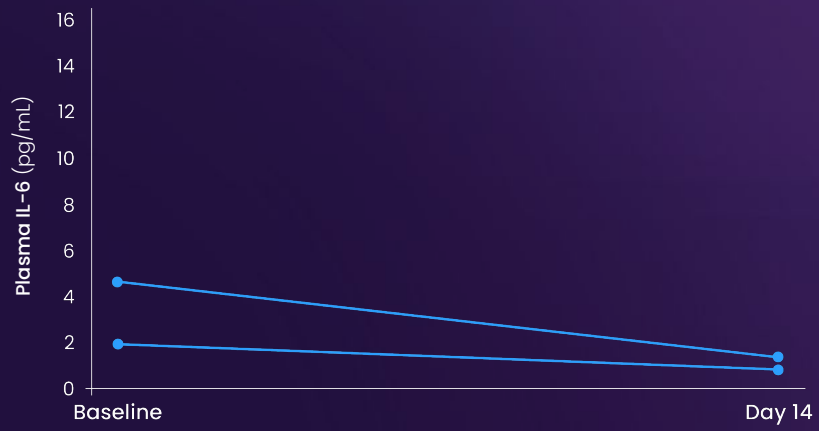
OBESE MAD

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*

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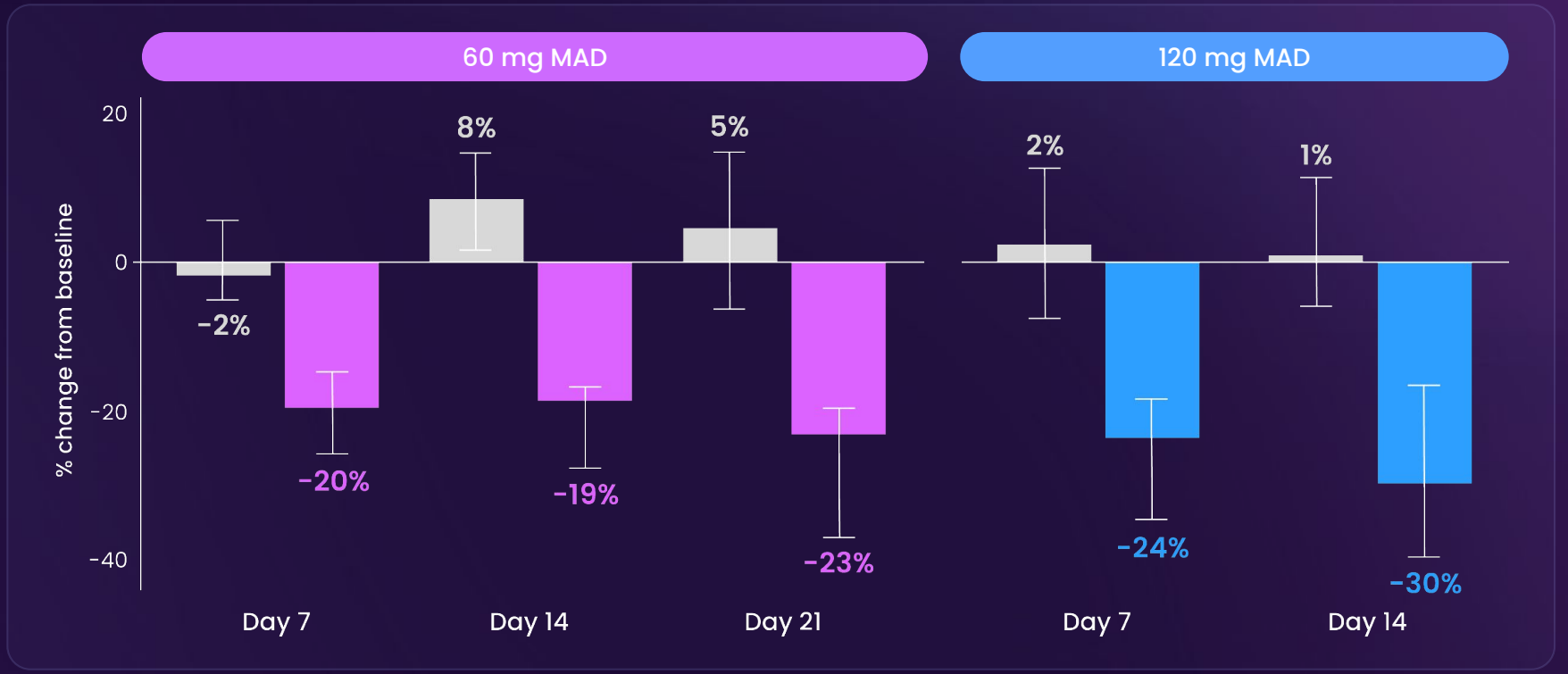
OBESE MAD

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 cohort active treatment and 290 mg/dL for placebo

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BGE-102 overview

Phase 1 results

Therapeutic areas

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THERAPEUTIC AREAS

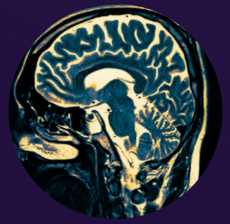
BGE-102 has the potential to address a range of cardiometabolic and neuroinflammatory disorders

Neuro-inflammation

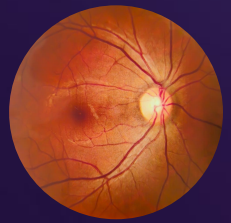
Peripheral inflammation

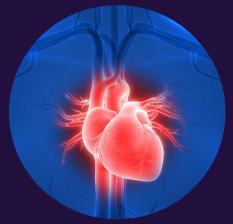
BGE-102 addressable diseases

Neurodegeneration

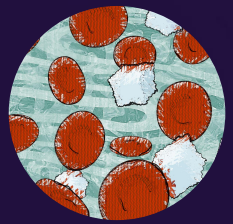


Ophthalmology

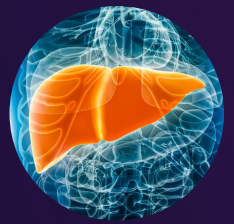




CVD (ASCVD, HF)



Insulin resistance



MASLD / MASH

BIOAGE

NLRP3

BGE-102 overview

Phase 1 results

Therapeutic areas

- Cardiovascular
- Ophthalmology

BIOAGE

NLRP3

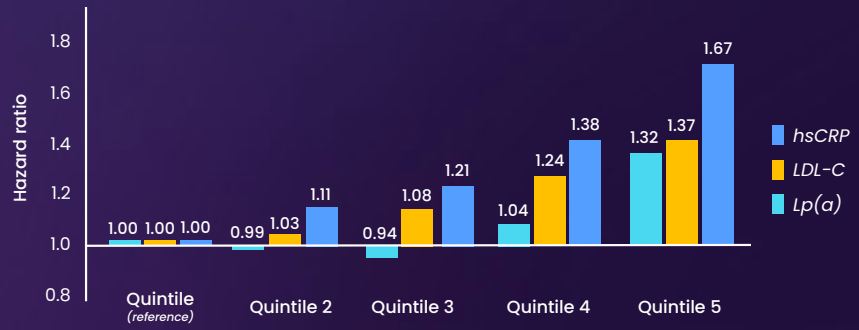
CARDIOVASCULAR DISEASE

Normalizing hsCRP (<2 mg/L) through IL-1β can drive a 25% MACE benefit

Inflammation (hsCRP) is more predictive of MACE than LDL or Lp(a)

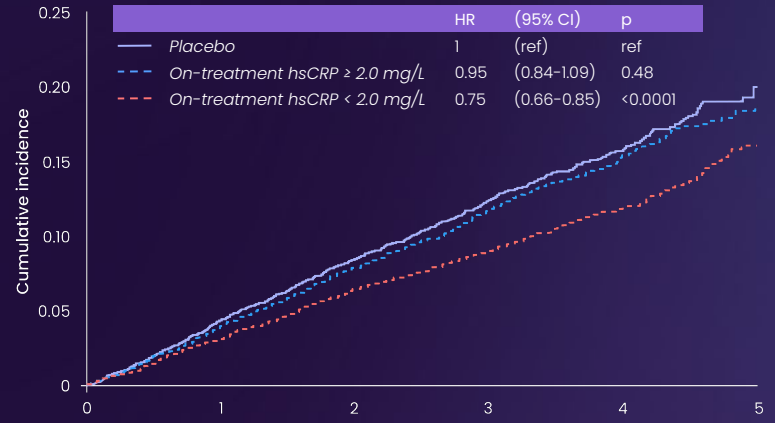
Risk of MACE in 30-year longitudinal data from the Women's Health Study

The NEW ENGLAND JOURNAL *of* MEDICINE



CANTOS trial validated the anti-inflammatory strategy

25% reduction in MACE for patients with hsCRP normalization



ACC SCIENTIFIC STATEMENT

Inflammation and Cardiovascular Disease: 2025 ACC Scientific Statement

A Report of the American College of Cardiology

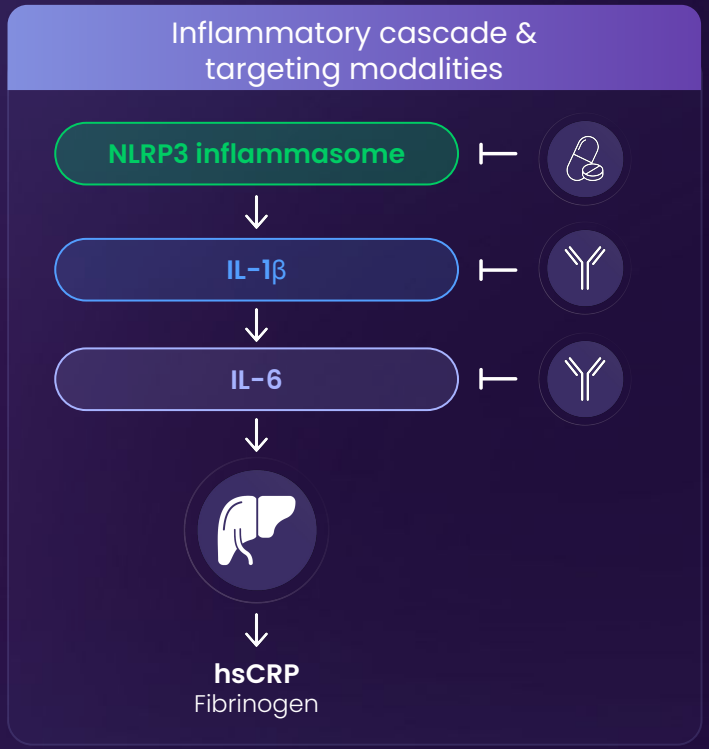
"In aggregate, the evidence linking inflammation with atherosclerotic CVD is no longer exploratory but is compelling and clinically actionable. The time for taking action has now arrived."

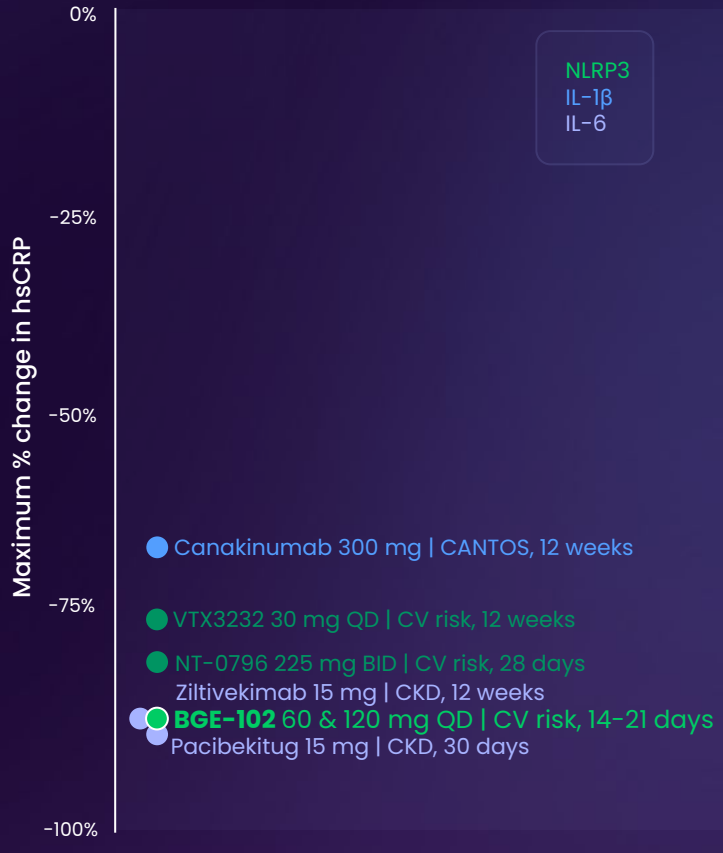
Source: Ridker 2024, Ridker 2018

BIOAGE

CARDIOVASCULAR DISEASE

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



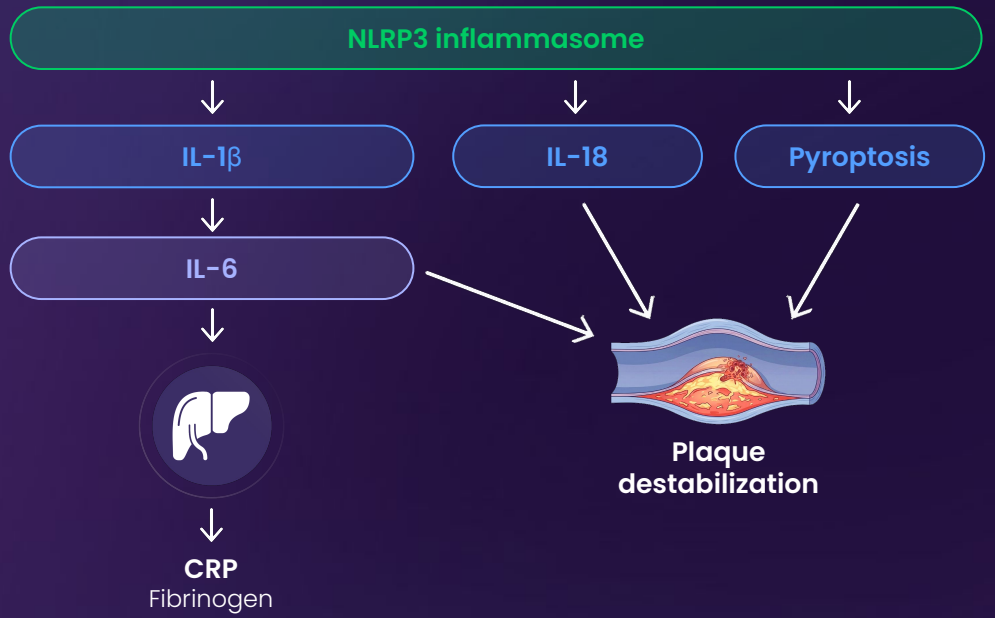


Sources: NodThera, Tourmaline Bio, Ventyx Biosciences, Wada 2023, Ridker 2017

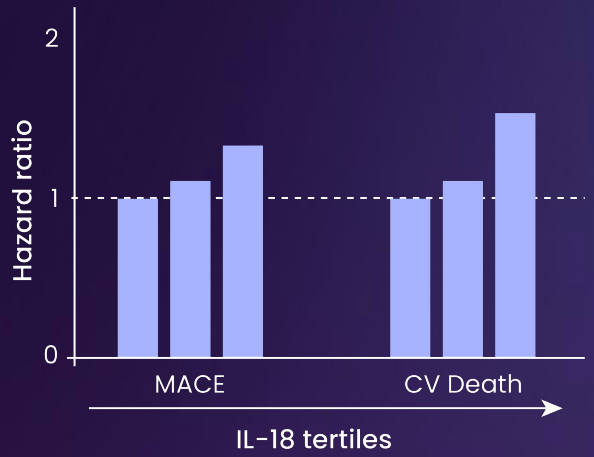
CARDIOVASCULAR DISEASE

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

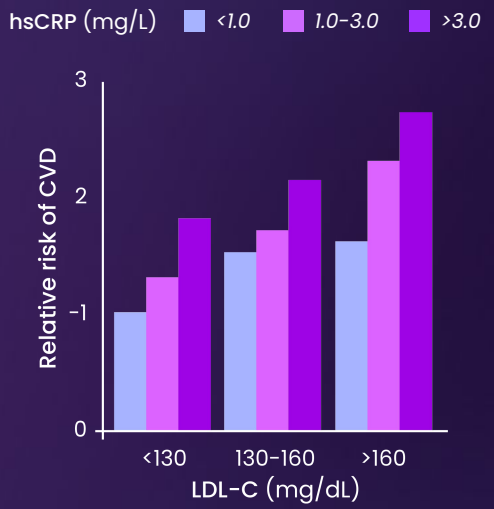
BIOAGE

NLRP3

CARDIOVASCULAR DISEASE

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

BIOAGE

CARDIOVASCULAR DISEASE

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

Screening — Dosing — Week 12 end of treatment — Week 16 end of study

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.*

NLRP3

BGE-102 overview

Phase 1 results

Therapeutic areas

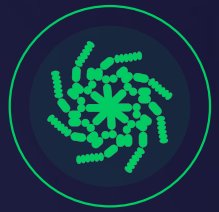
- Cardiovascular
- Ophthalmology

BIOAGE

NLRP3

OPHTHALMOLOGY

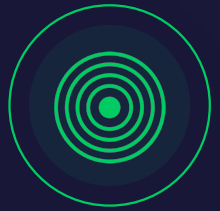
The retinal opportunity for BGE-102



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

OPHTHALMOLOGY

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

Diabetic retinopathy / diabetic macular edema (DME)

Hyperglycemia & oxidative stress

↓

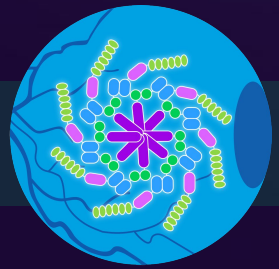
NLRP3 activation (IL-1β, IL-18)
endothelial cells | Müller cells, microglia

↓ 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

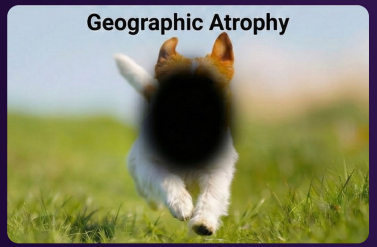
Source: Zheng 2023

NLRP3

OPHTHALMOLOGY

Prevalent retinal diseases with significant unmet needs

	DME	GA (late-stage AMD)
Description	• Vision loss due to vascular leakage • Leading cause of vision loss in adults <65	• Loss of central vision • 20% become legally blind within 1 year of diagnosis
Prevalence (US)	• 1M	• 1.2M
Key risk factors	• Hyperglycemia	• Age, inflammation, cardiometabolic disease
Approved therapies	• Intravitreal anti-VEGFs, intravitreal steroids, laser	• Intravitreal complement inhibitors (Syfovre, Izervay)
Key unmet needs	• **Reduced injection burden:** real world treatment outcomes are suboptimal given poor compliance • **New MOAs:** significant anti-VEGF refractory population	• **Disease control:** approved therapies offer limited efficacy



Sources: Al Sakini 2024, Lai 2024, Varma 2014, Seddon 2003, CDC, Mayo Clinic, Broker estimates

BIOAGE

NLRP3

OPHTHALMOLOGY

Key treatment challenges

Limited efficacy of approved therapies

DME

~45% of patients are refractory to anti-VEGFs despite compliance

GA

~90% are <u>not</u> treated given current drugs only slow progression 15-20%

High injection burden

~50% discontinue anti-VEGFs after an average of 6 months

(Bi)monthly injections required for approved therapies, often in elderly patients

Eyes treated individually

~70% develop bilateral disease within 1 year of diagnosis

~65% develop bilateral disease; treatment limited to 1 eye per visit

Systemic disease drivers not addressed

Insulin resistance is a key risk factor for refractory disease

Inflammation is both a risk factor & prognostic marker

Source: Kulkarni 2021, Kuo 2024, Bressler 2018, AREDs Research Group, Astellas / Apellis company reports, BioAge market research

NLRP3

OPHTHALMOLOGY

DME: intravitreal anti-IL-6 has validated the anti-inflammatory approach in multiple trials of retinal diseases driven by inflammation, including DME

Anti-IL-6 in DME

Rapid improvements in visual acuity & CST: seen within 4-8 weeks in Phase 2 trials

~1 line: Incremental vision with anti-VEGF, sustained through 1 year

Broad utility: efficacy in treatment-naïve and actively treated (anti-VEGF) patients

Roche

KODIAK

EYEPOINT

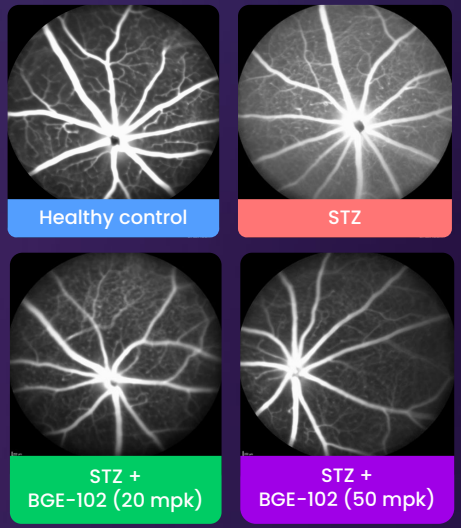
Orphan strategy: uveitic macular edema
Steroids are SOC and anti-IL-6 has shown robust efficacy
MEERKAT trial: up to 36.9% of patients achieved a ≥15 letter improvement from baseline in BCVA at week 16

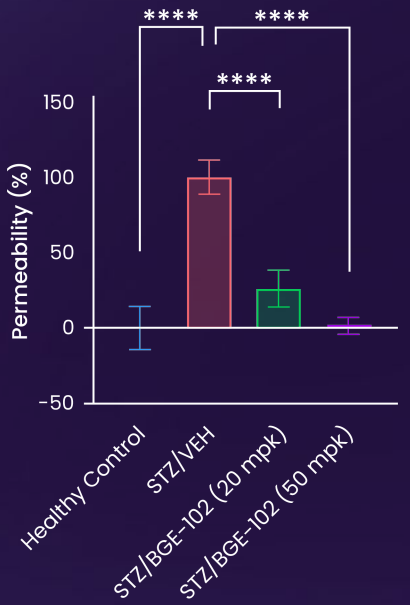
Source: Company Presentations (Kodiak, Eyepoint, Roche)

OPHTHALMOLOGY

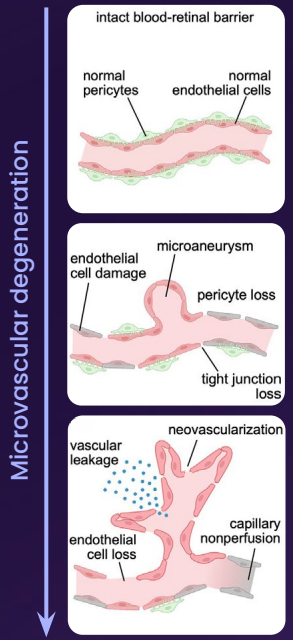
DME: oral delivery of BGE-102 preserved retinal vascular integrity in a preclinical model of diabetic retinopathy

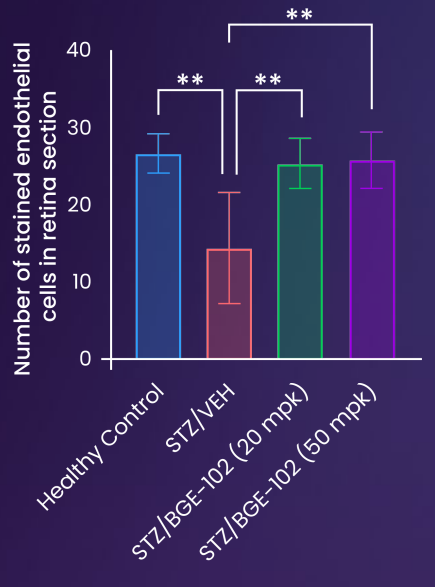
Retinal vascular permeability
measured via fluorescein angiography





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





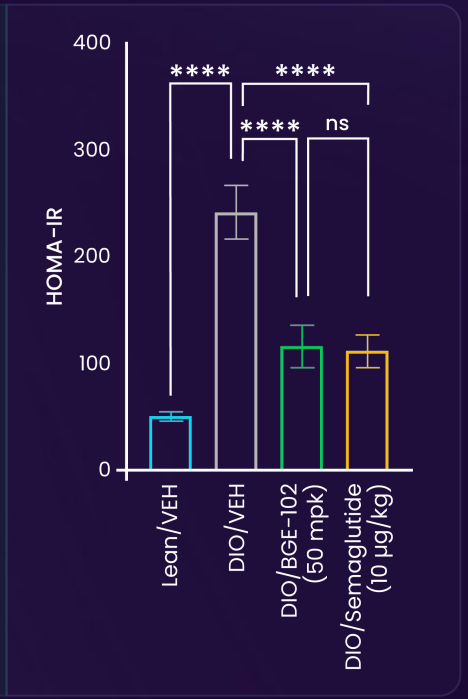
Note: Streptozotocin (STZ) diabetic mouse model; BGE-102 dosing initiated 11 days following last dose of STZ; assessments performed after 3 months of BGE-102 QD dosing; BGE-102 is more potent in human cells, for example 150–250x higher potency in microglia
Source: Wolf 2023

BIOAGE

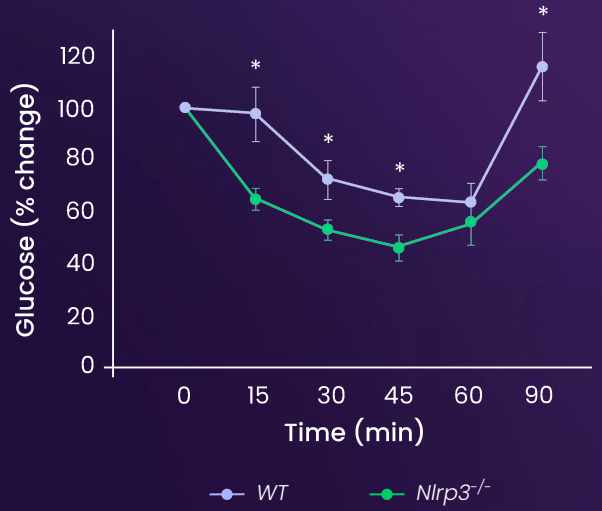
OPHTHALMOLOGY

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)

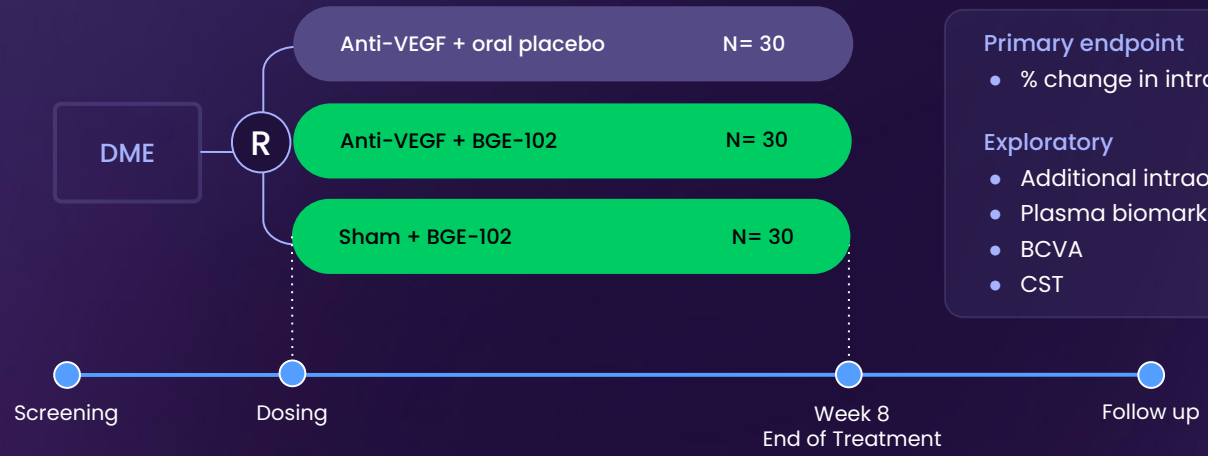


Note: BGE-102 was dosed PO QD; semaglutide was dosed SC QD.
Source: Vandanmagsar 2011

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

Key program advisors

David Boyer, MD
Senior Partner, Retina-Vitreous Associates Medical Group

Philip Rosenfeld, MD, PhD
Professor of Ophthalmology, Bascom Palmer Eye Institute, University of Miami

BIOAGE

Goals of DME POC:

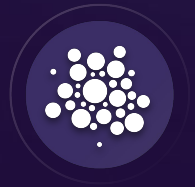
- Demonstrate PD in the eye with oral delivery of BGE-102
- Motivate subsequent development in additional ophthalmology indications, including GA

BIOAGE

OPHTHALMOLOGY

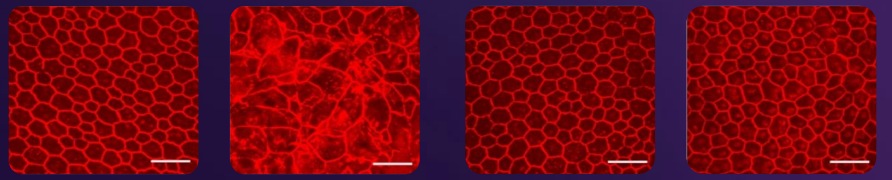
GA: NIrp3 knockout rescues integrity of the RPE in mice

Pro-inflammatory hallmarks of GA

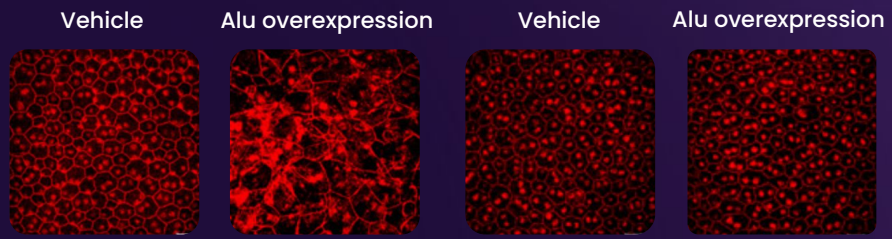


Drusen
(Aβ oligomers)

Alu RNA
(overexpression)



Healthy %	100	0	100	100



Healthy %	100	0	100	100

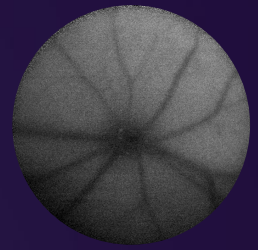
Source: Narendran 2021, Tarallo 2012

NLRP3

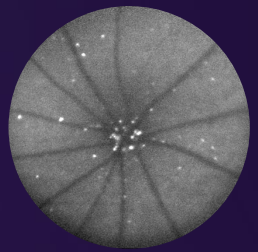
OPHTHALMOLOGY

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

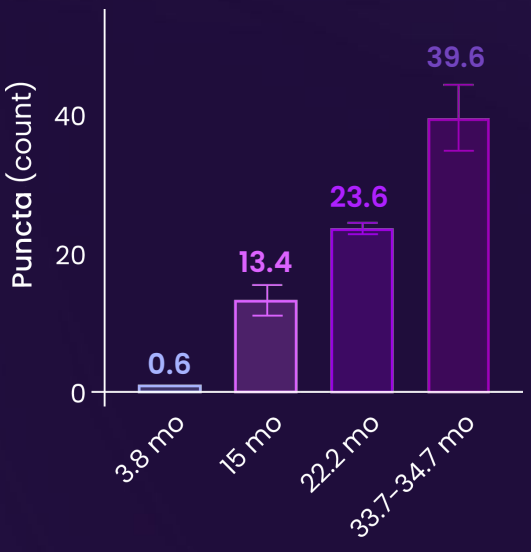
Natural AMD model
Lipofuscin punctae accumulate with age in outbred (HET3) mice



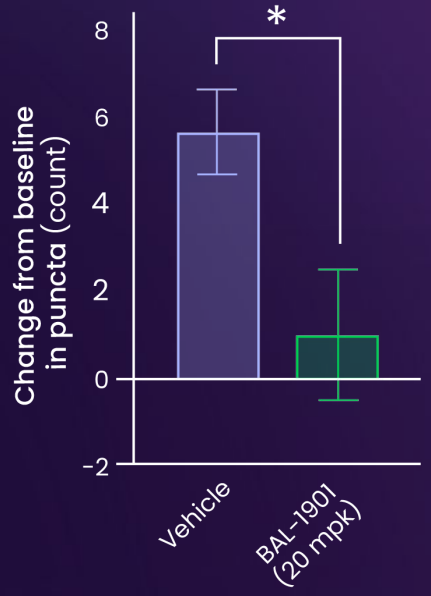
3 months



30 months



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



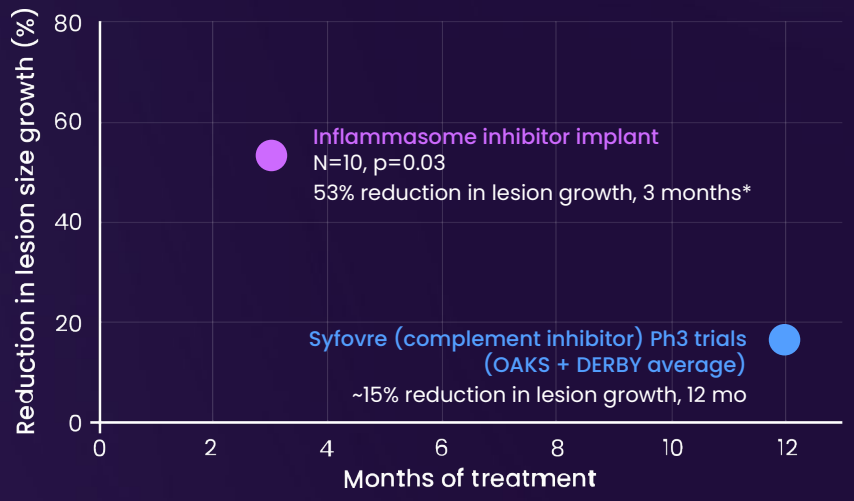
BIOAGE

OPHTHALMOLOGY

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

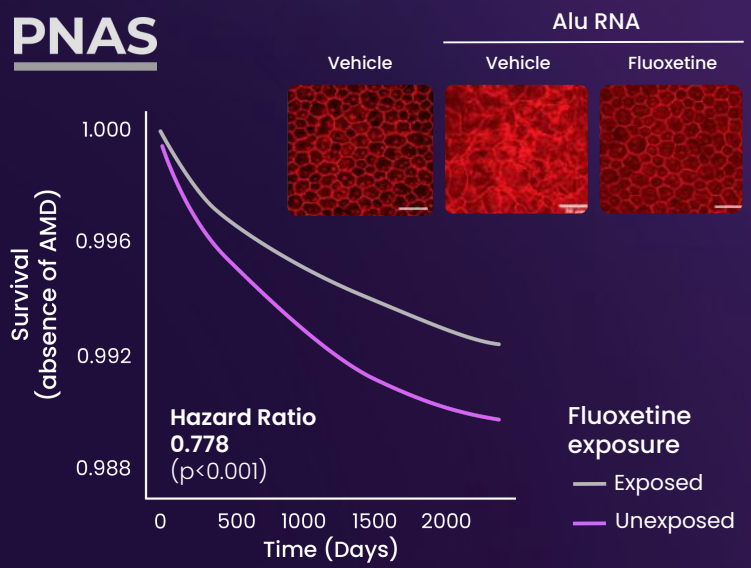
Pilot study shows potential for greater efficacy

Inflammasome inhibitor intravitreal implant, K8, vs. complement inhibition



Decreased incidence of AMD

observed with the SSRI fluoxetine, a weak NLRP3 inhibitor (µM potency), in retrospective analysis



*Note: * Comparison made to fellow contralateral eye*
Source: Ambati 2021, Heier 2023, Inflammasome Therapeutics

BIOAGE

Apelin / APJ

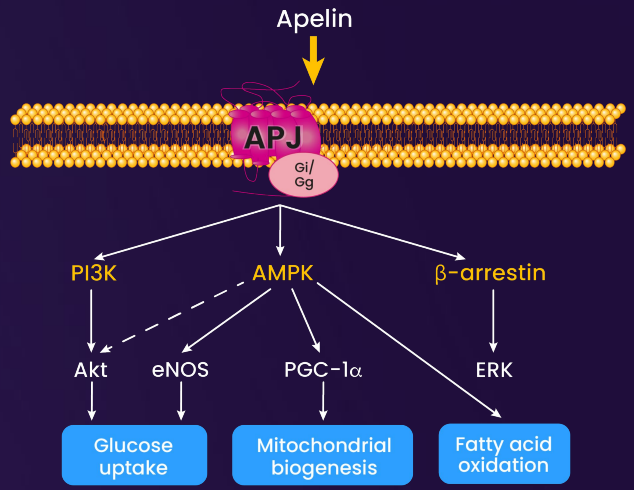
BIOAGE

APJ

Apelin is an exerkine and mimics many benefits of exercise

Shared biology between apelin & exercise

Exercise stimulates release of apelin into circulation



Systemic benefits

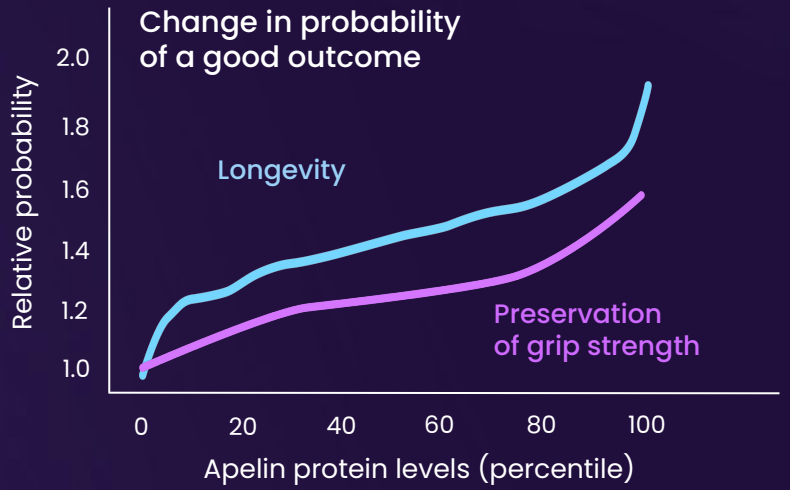
- Balance of lean & fat mass
- Basal metabolic rate
- Protein synthesis
- Mitochondrial biogenesis
- Insulin sensitivity / glucose metabolism

Source: Adapted from Bertrand 2015; Ceraudo 2014

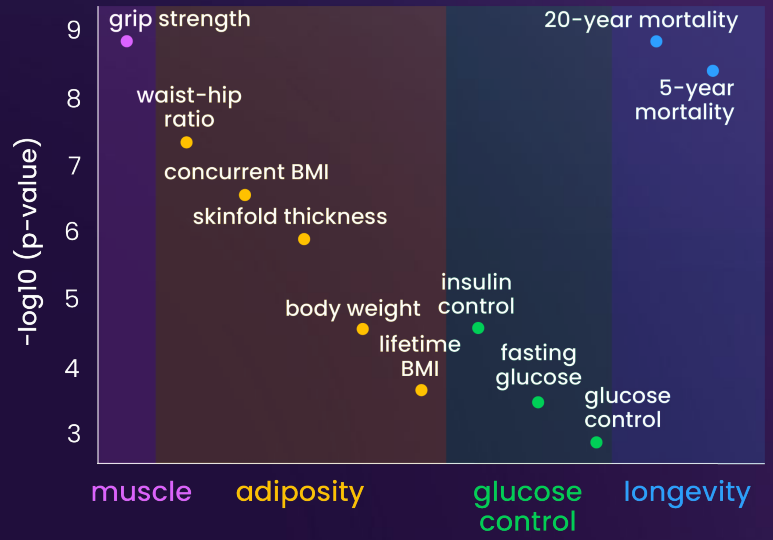
APJ

Human insights from the BioAge platform: Apelin signaling impacts muscle & metabolism

BioAge aging dataset



Human proteomic associations



BIOAGE

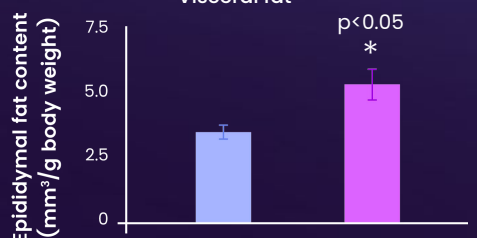
APJ

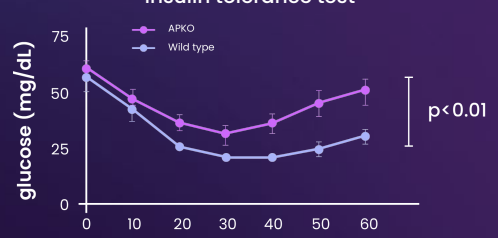
Apelin genetics reinforce beneficial role in systemic metabolism

Apelin KO

↑ adiposity

↑ insulin resistance



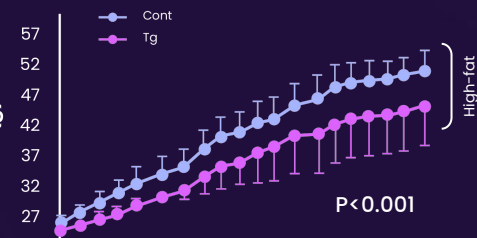


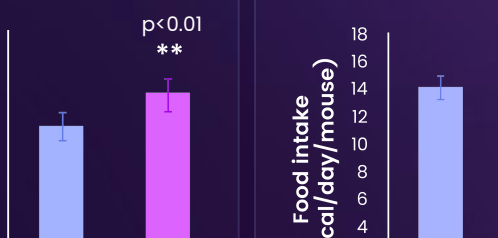
Apelin transgenic

↓ weight gain on high fat diet

↑ basal metabolic rate

No impact on energy intake





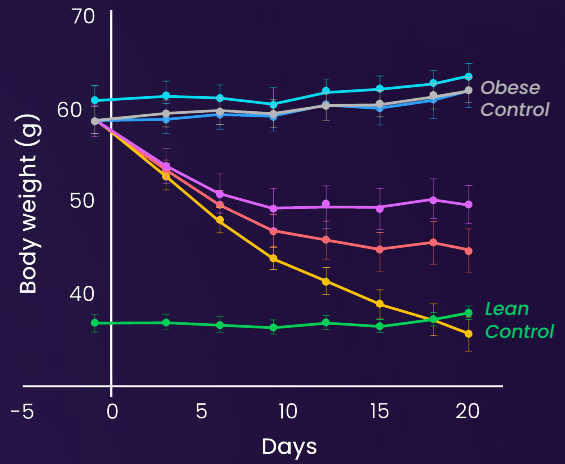
Consistent genetic evidence in humans:
Genome-wide significant associations for the apelin receptor APJ include BMI, lean mass, and serum lipids

Source: Yue 2010, Yue 2011, Yamamoto 2011, Pulit 2019, Sakaue 2021, Pei 2020, Richardson 2020, Wakil 2016

APJ

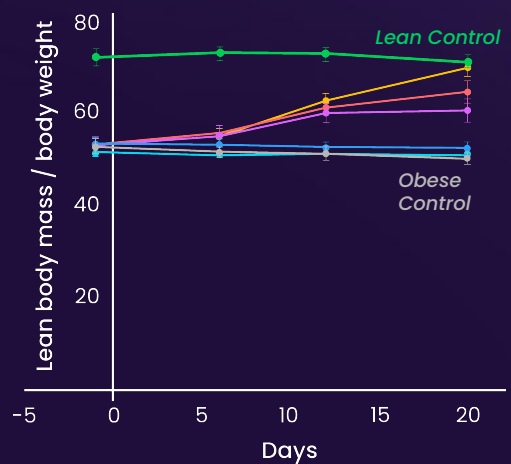
We previously demonstrated that APJ agonism can restore body weight & body composition to that of lean controls when combined with an incretin

Overall weight loss

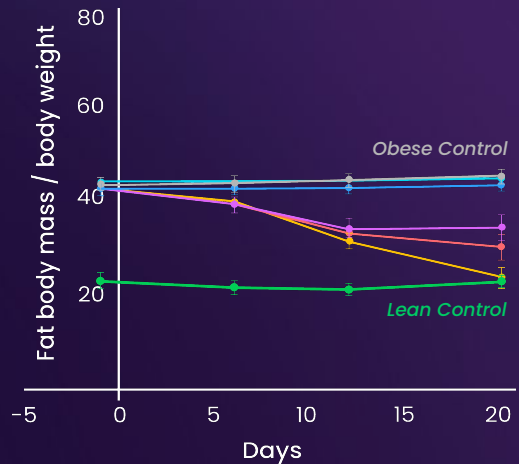


Body composition

% Lean Mass



% Fat Mass



Lean Control
Obese Control
Azelaprag (0.275g/L)
Azelaprag (1.1g/L)
Tirzepatide (10nmol/kg)
Tirzepatide (10nmol/kg)+ Azelaprag (0.275g/L)
Tirzepatide (10nmol/kg)+ Azelaprag (1.1g/L)

Note: Mice in all groups lose both lean mass and fat mass; with azelaprag, lean body composition (% lean mass) is restored. Lean and fat mass were measured with EchoMRI.

BIOAGE

APJ

In a clinical trial, APJ agonism resulted in significant muscle & metabolic benefits in older subjects on bed rest

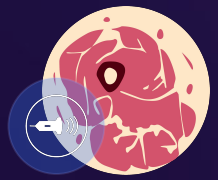
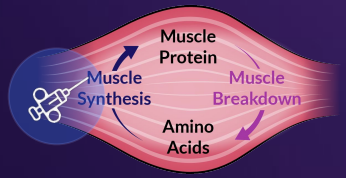
Ph1b design

10 days of bed rest & dosing

Healthy subjects 65+ (N=10 placebo, N=11 azelaprag)

Azelaprag (240mg) or placebo via daily IV infusion

Double-blind, non-randomized

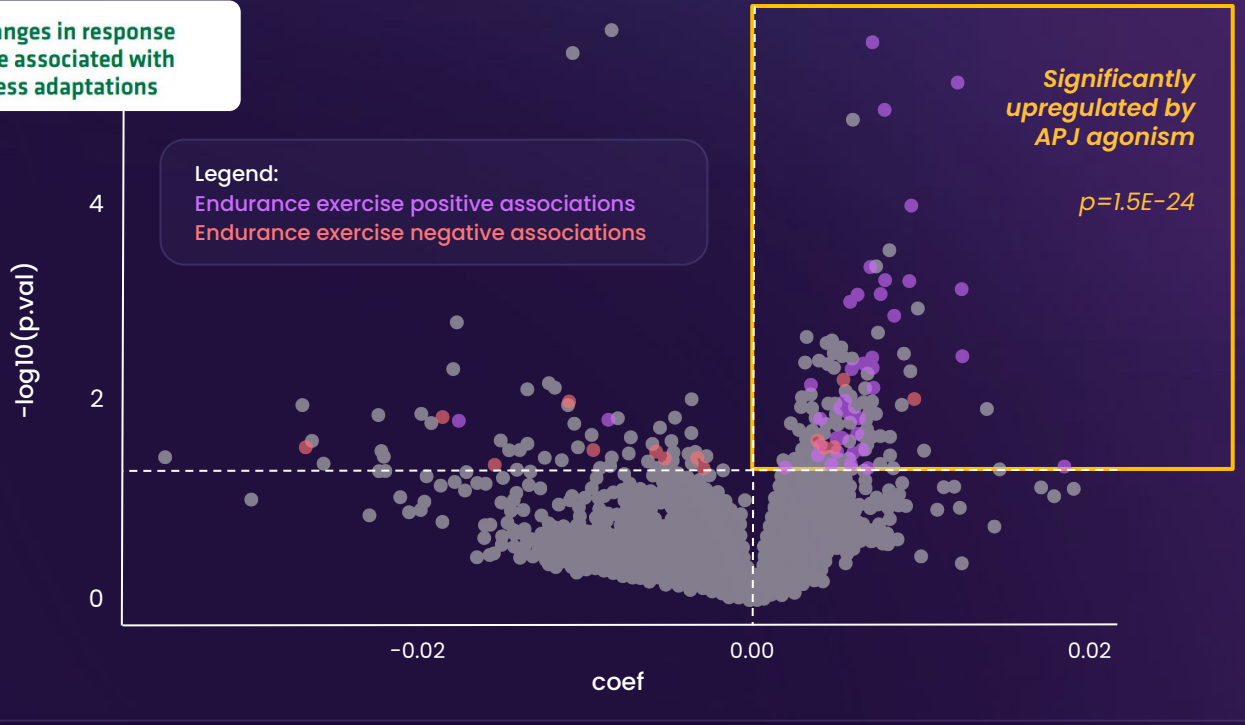
Muscle size			Fat infiltration	Metabolism
Thigh circumference	Muscle size (vastus lateralis)		Muscle degeneration	Muscle protein synthetic rate
				
Circumference	Diameter (thickness)	Cross-sectional area	Muscle grade (progression)	Cumulative protein synthesis
p<0.001	p<0.01	p<0.05	p<0.005	p<0.005

BIOAGE

In the trial, APJ agonism shifted the serum proteome consistent with recapitulating the benefits of exercise

JCI insight

Plasma proteomic changes in response to exercise training are associated with cardiorespiratory fitness adaptations



Source: Robbins 2023

BIOAGE

APJ

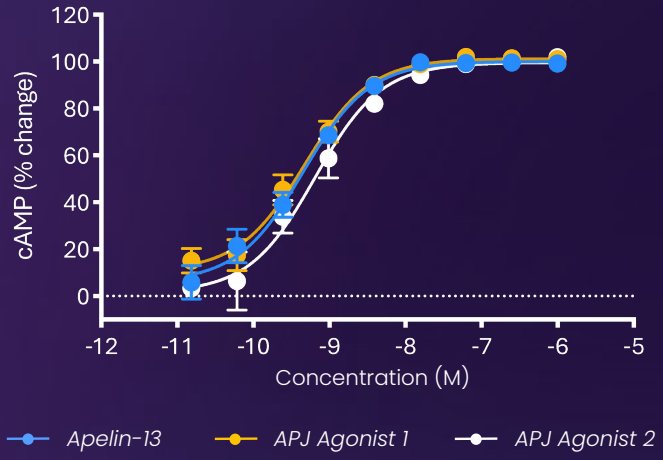
Our goal is to advance APJ agonists for both oral and parenteral delivery

Recent progress on APJ

Oral

- Composition of matter IP filed on small molecules with picomolar potency
- Scaffold distinct from azelaprag

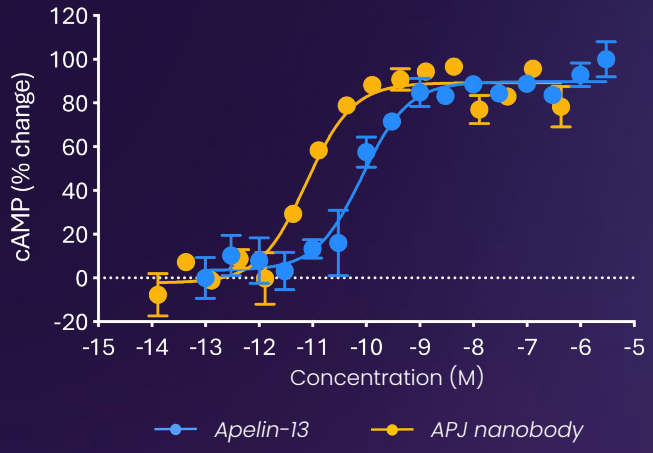
APJ agonist activity (cAMP)



Parenteral

- Exclusive option on APJ agonist nanobodies with potency 10x greater than apelin, the natural ligand

APJ agonist activity (cAMP)

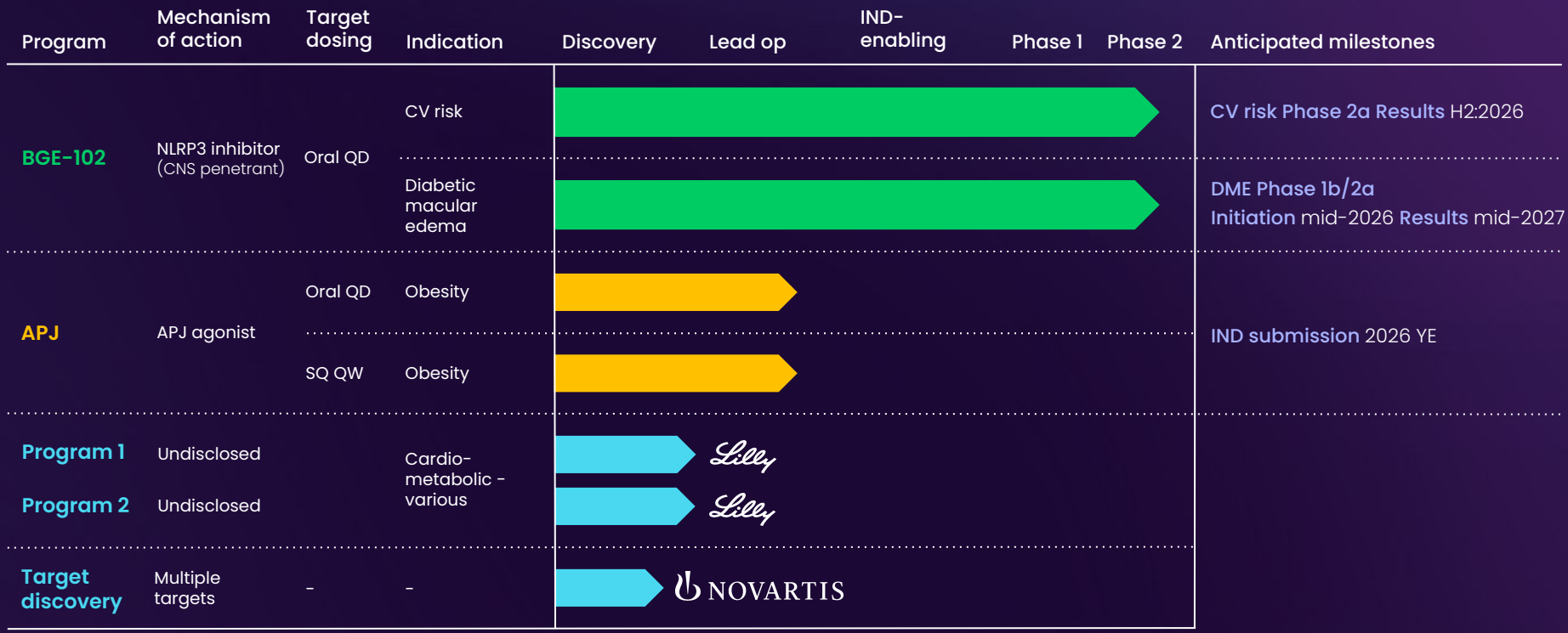


Collaboration with JiKang Therapeutics

BIOAGE

Pipeline overview

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



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

BIOAGE

BIOAGE