

BIOAGE

Corporate Presentation
June 2026

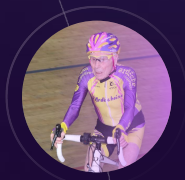
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We are harnessing the biology of human aging to develop new therapies for cardiometabolic diseases



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: QUELL-CV H2:2026, QUELL-DME mid-2027

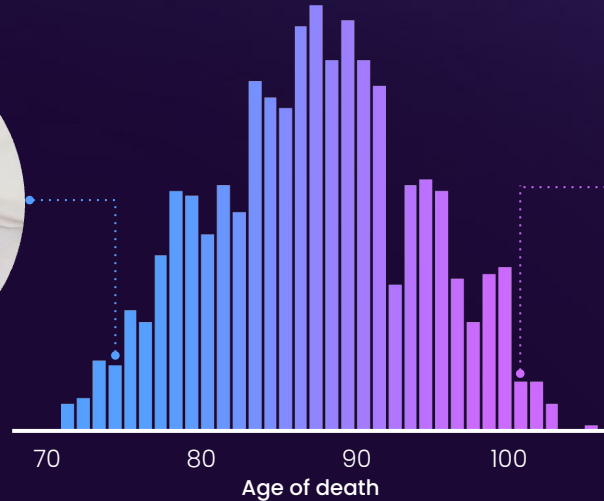
APJ agonism: exercise mimetic for obesity

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

Anticipated catalysts: IND submission 2026 YE

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+

Molecular data points

25K+

Profiles generated

50+

Years of follow-up

Detailed healthspan trajectories



Physical function

- Grip strength
- Walking speed
- Mobility



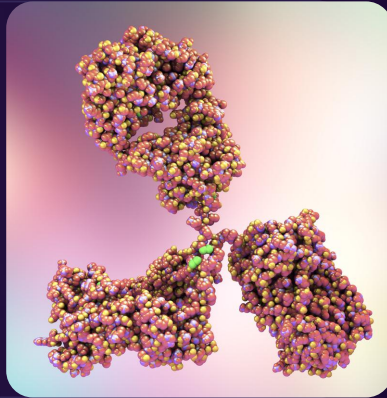
Metabolism

- BMI
- Skinfold thickness
- Waist / hip circumference

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

Lilly mAb discovery

mAb drug discovery collaboration for two novel BioAge targets



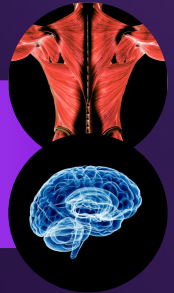
 NOVARTIS Target discovery

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



Exercise response

Longitudinal healthspan outcomes



We are supported by an accomplished team of board members and advisors

BOARD MEMBERS



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Chair
Former CEO, GSK



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CEO / CFO experience



Vijay Pande, PhD
Managing Partner,
VZVC

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

LEADERSHIP TEAM



Kristen Fortney, PhD
Co-Founder, CEO



Eric Morgen, MD
Co-Founder, COO



Paul Rubin, MD
CMO & EVP Research



Ann Neale
CDO



Dov Goldstein, MD, MBA
CFO



Peng Leong, PhD, MBA
CBO & TA Head,
Brain Aging



BJ Sullivan, PhD
Chief Strategy
Officer

~800
Clinical trials*

~130
INDs*

~95
US regulatory
approvals*

Stanford

Berkeley
UNIVERSITY OF CALIFORNIA

UNIVERSITY OF
TORONTO

Buck
Live better longer.

PRINCIPIA
BIOPHARMA

SCHRÖDINGER

LOXO
ONCOLOGY *Lilly*

XOMA

gsk

Takeda

MERCK

AstraZeneca

AISLING
CAPITAL

LEK™

NLRP3

BGE-102 overview

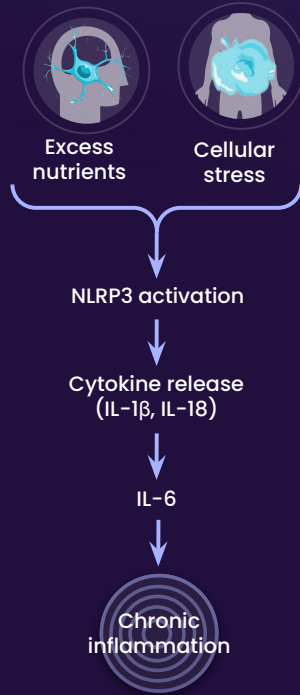
Phase 1 results

Therapeutic areas

Chronic NLRP3 activity drives disease & predicts poor human longevity

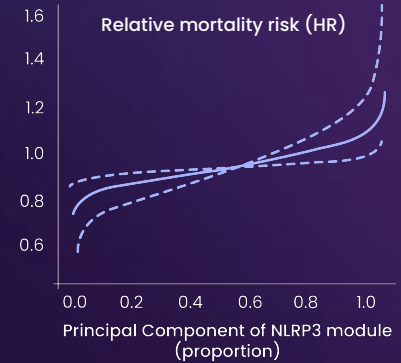
NLRP3 overview

- 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 (\uparrow 1 SD expression = up to \uparrow 70% risk)

GoF mutations \downarrow lean mass & body composition \uparrow atherosclerosis

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 IC₉₀ by human ex-vivo whole blood stimulation

24h IC₉₀ coverage at 60 mg QD provided 24 hour IL-1 β suppression \geq 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

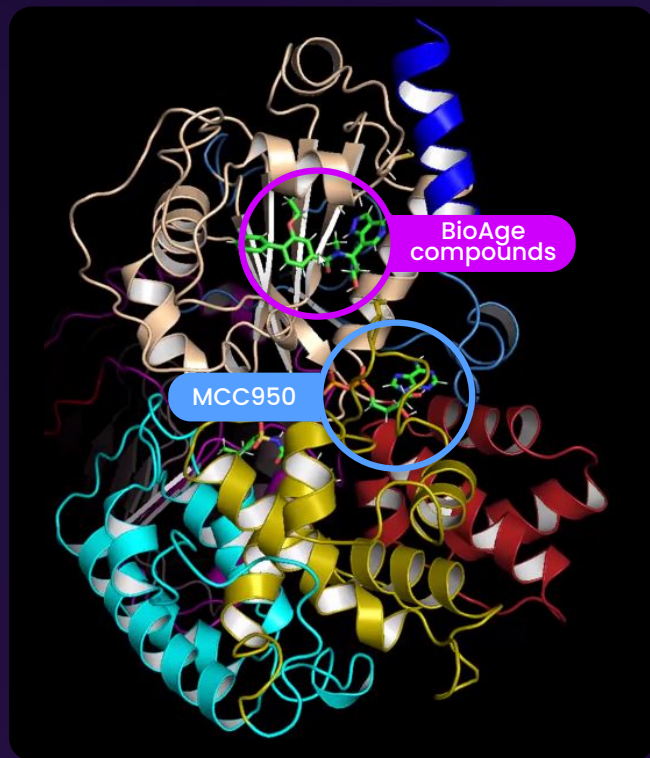


Strong IP position

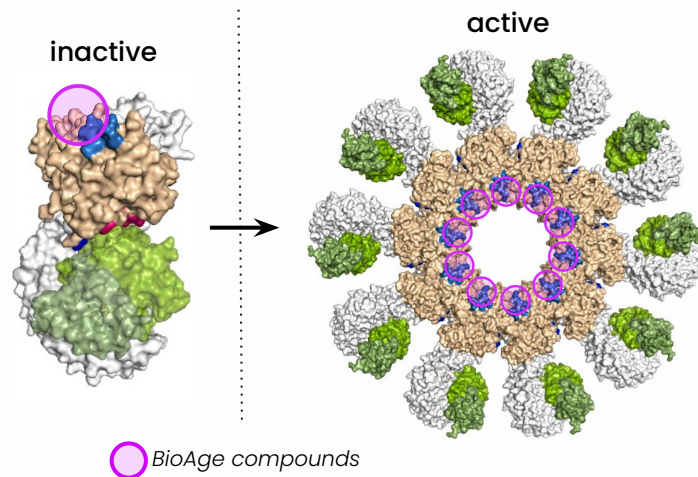
2045 composition of
matter & claims for
novel NLRP3 binding site

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

Unique binding site



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



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



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

bioRxiv

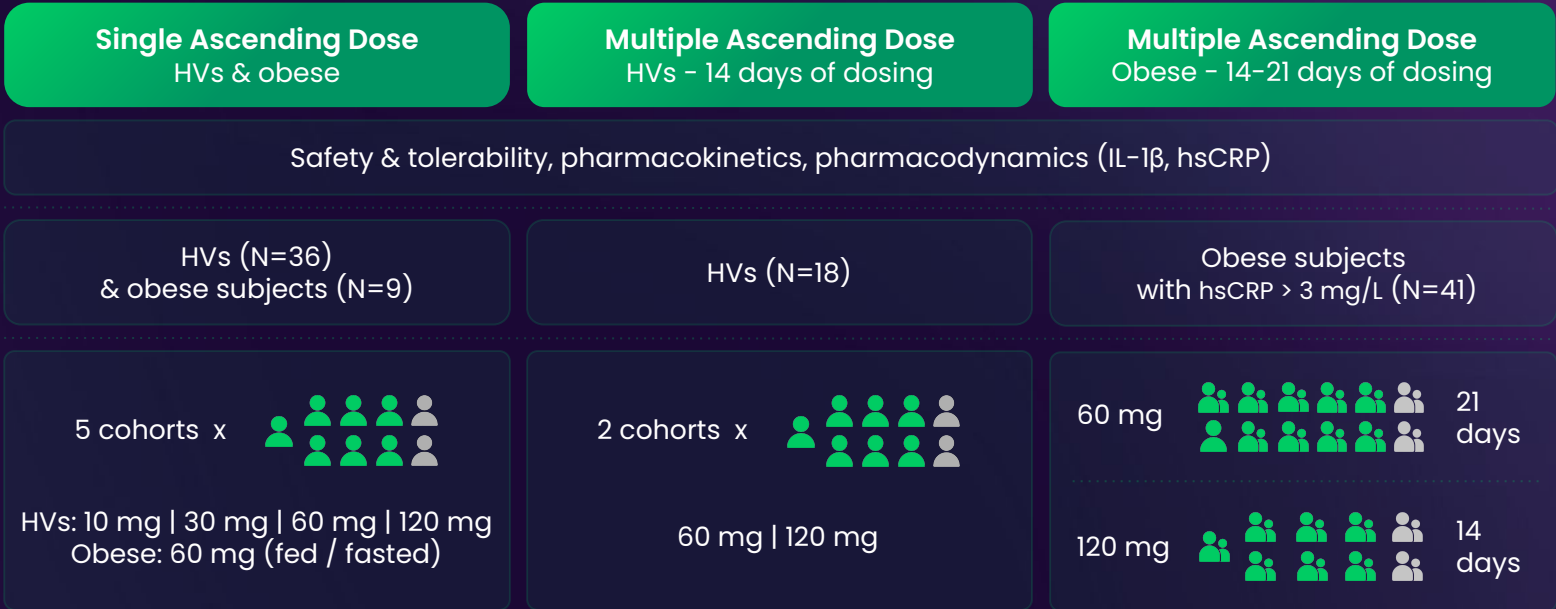
NLRP3

BGE-102 overview

Phase 1 results

Therapeutic areas

Phase 1 clinical trial design



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:

- $\geq 90\%$ suppression of IL-1 β 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)

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%)

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)

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%)

NLRP3

BGE-102 overview

Phase 1 results

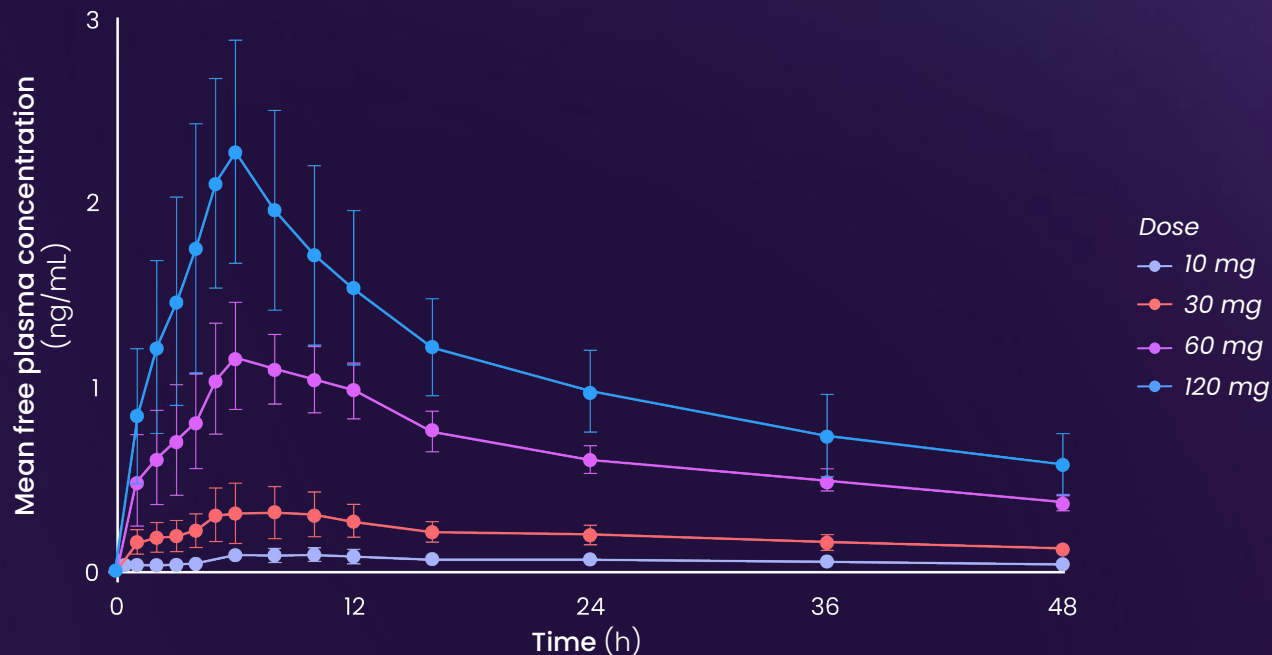
Healthy volunteer SAD / MAD

Obese MAD

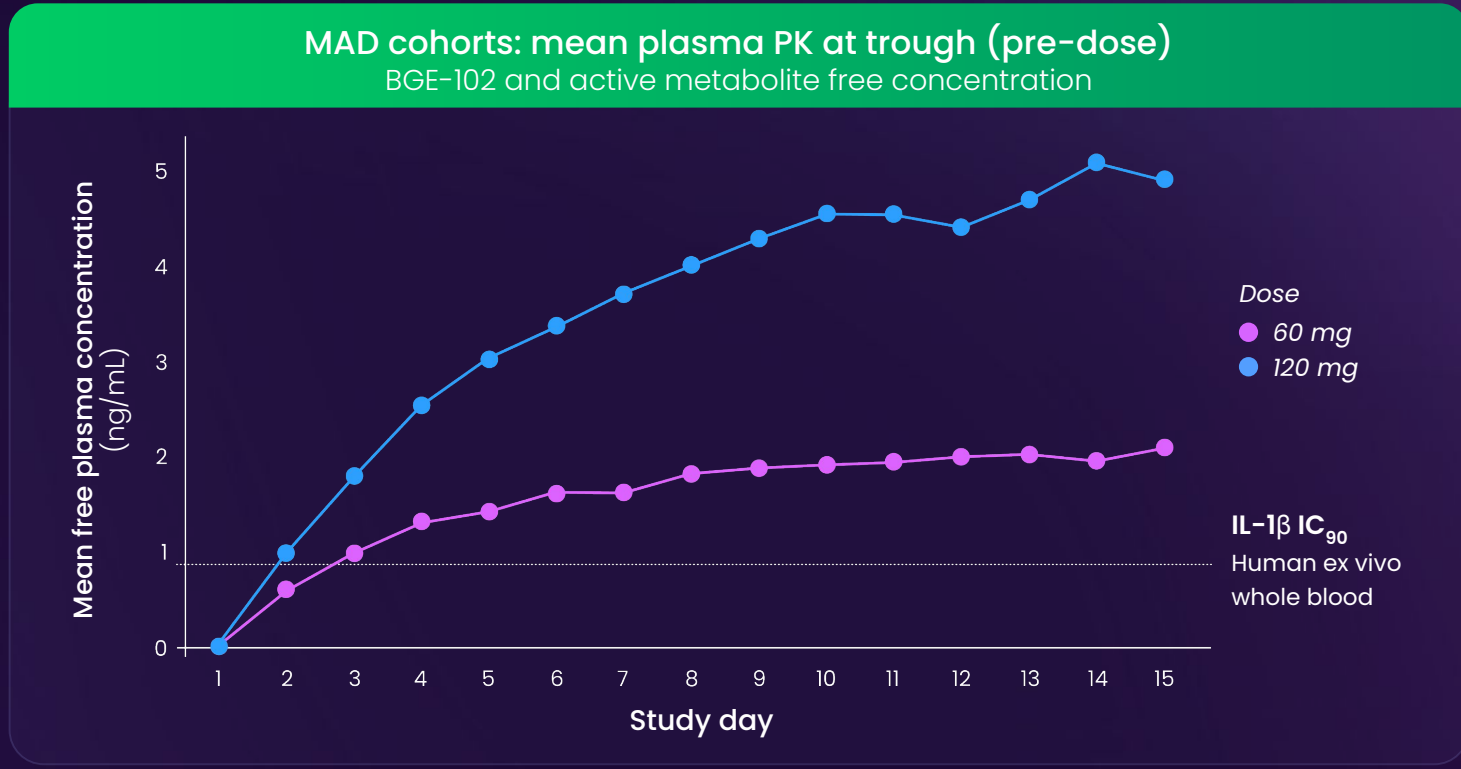
Therapeutic areas

Plasma PK: Dose-proportionality observed in SAD cohorts

SAD cohorts: mean plasma PK
BGE-102 and active metabolite* free concentration

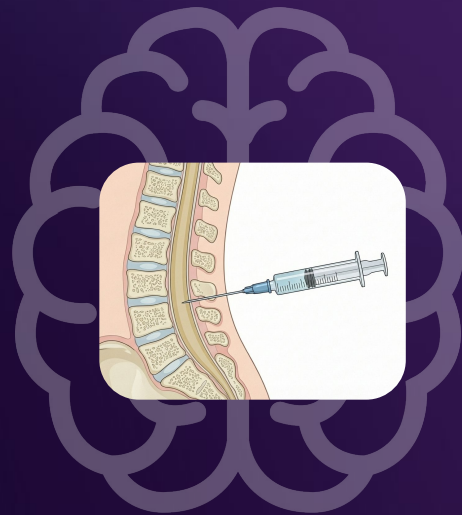
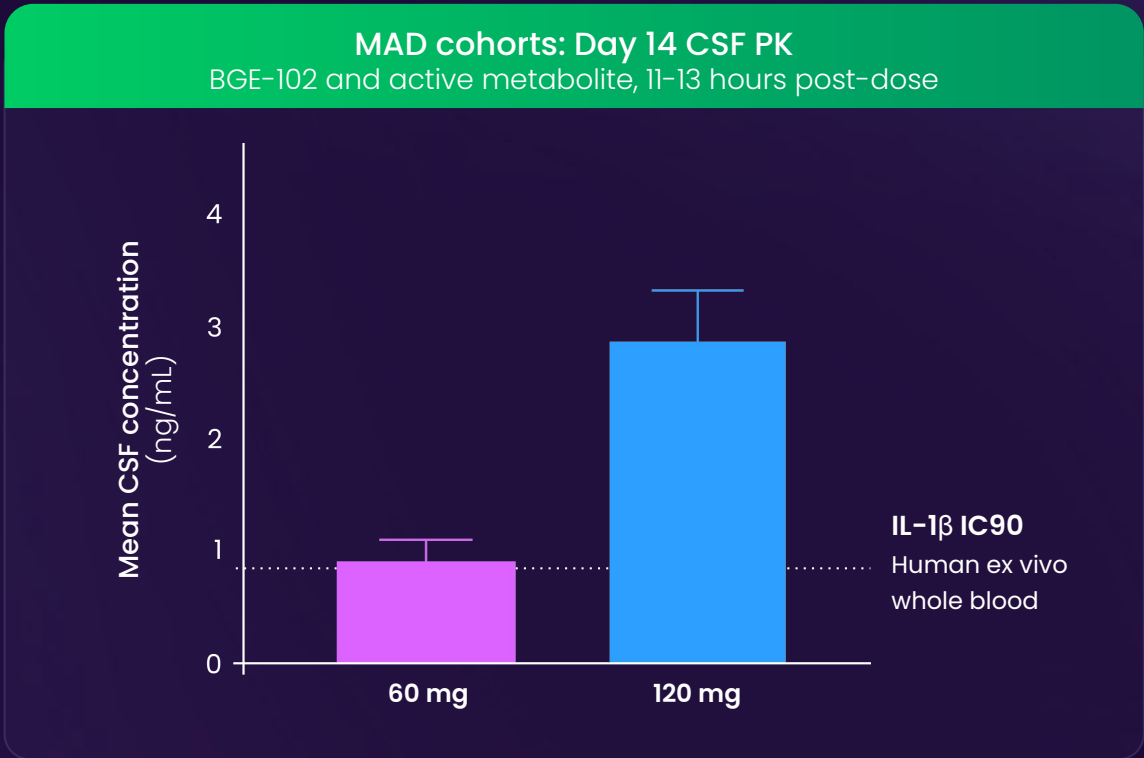


Plasma PK: MAD cohorts showed accumulation out to 14 days with near steady-state levels above the human IC₉₀ for IL-1 β inhibition



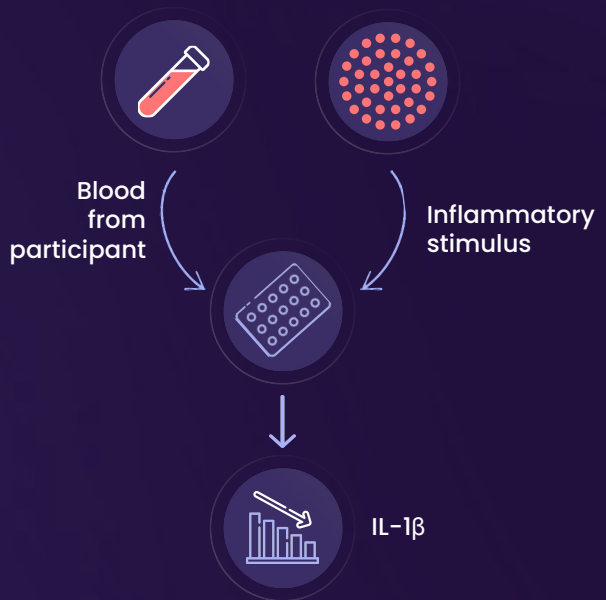
20 Note: * IC90 calculated from first 24 hours of treatment of healthy volunteers in SAD & MAD cohorts

CSF PK: doses of 60 mg and above exceeded human IC₉₀ at near steady state

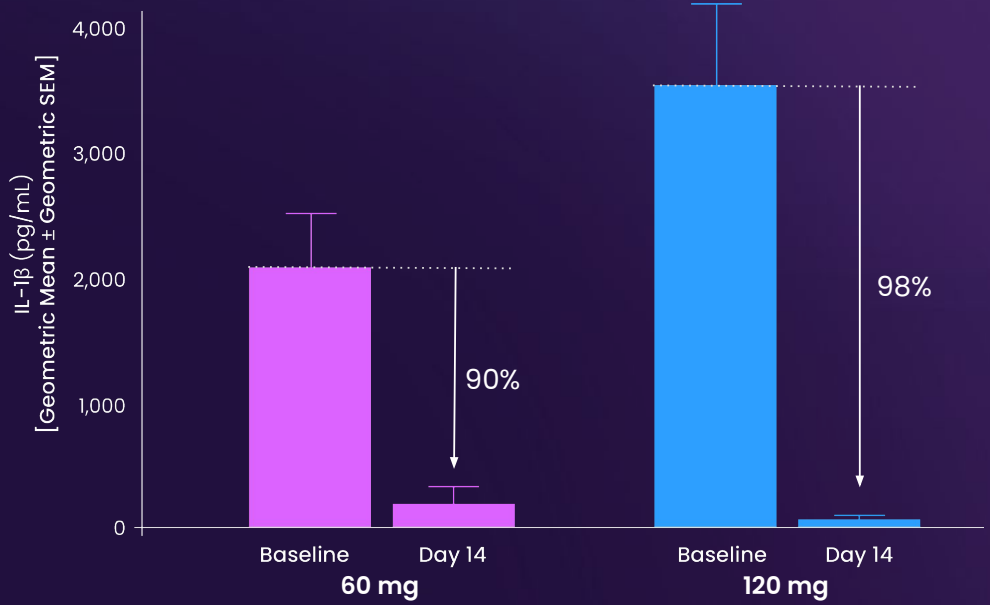


PK / PD: 90-98% suppression of IL-1 β at BGE-102 trough in MAD cohorts

Ex vivo whole blood stimulation



MAD cohorts: Mean IL-1 β with ex vivo stimulation at trough baseline vs Day 14



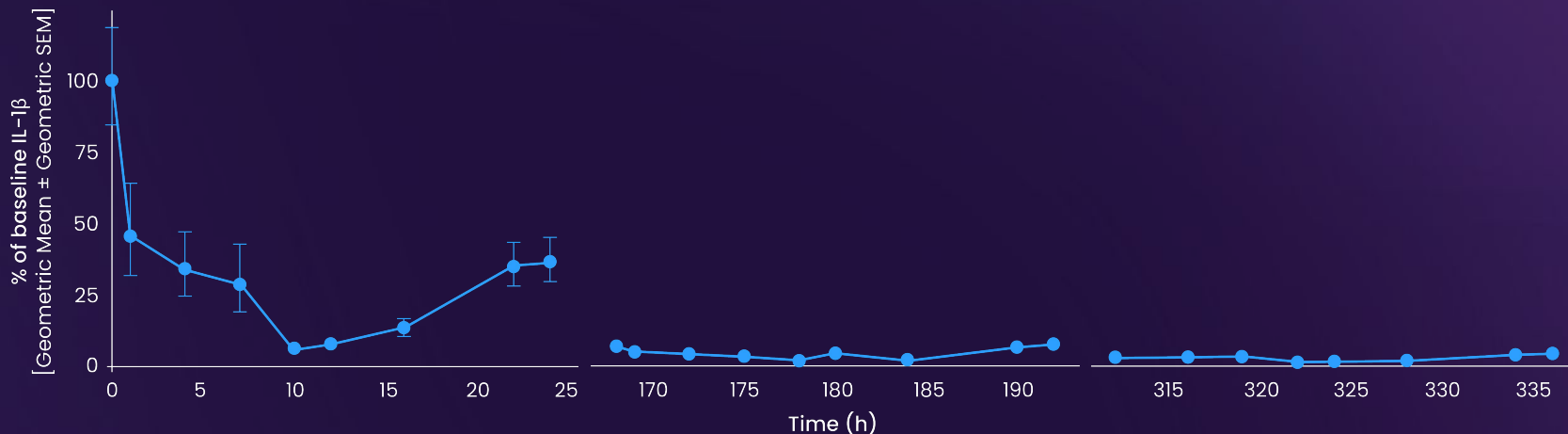
PK / PD: 24 hours of near maximal suppression of IL-1 β achieved by Day 8

120 mg MAD

Day 1

Day 8

Day 14



NLRP3

BGE-102 overview

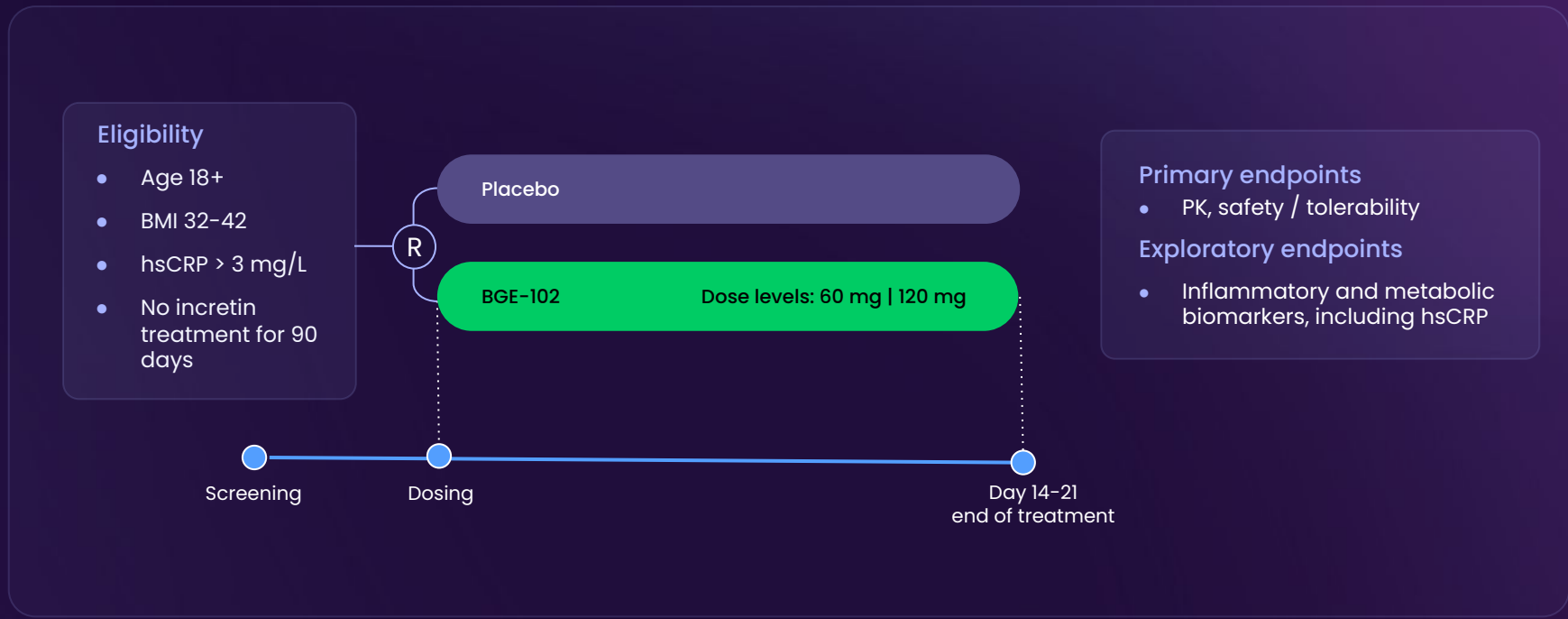
Phase 1 results

Healthy volunteer SAD / MAD

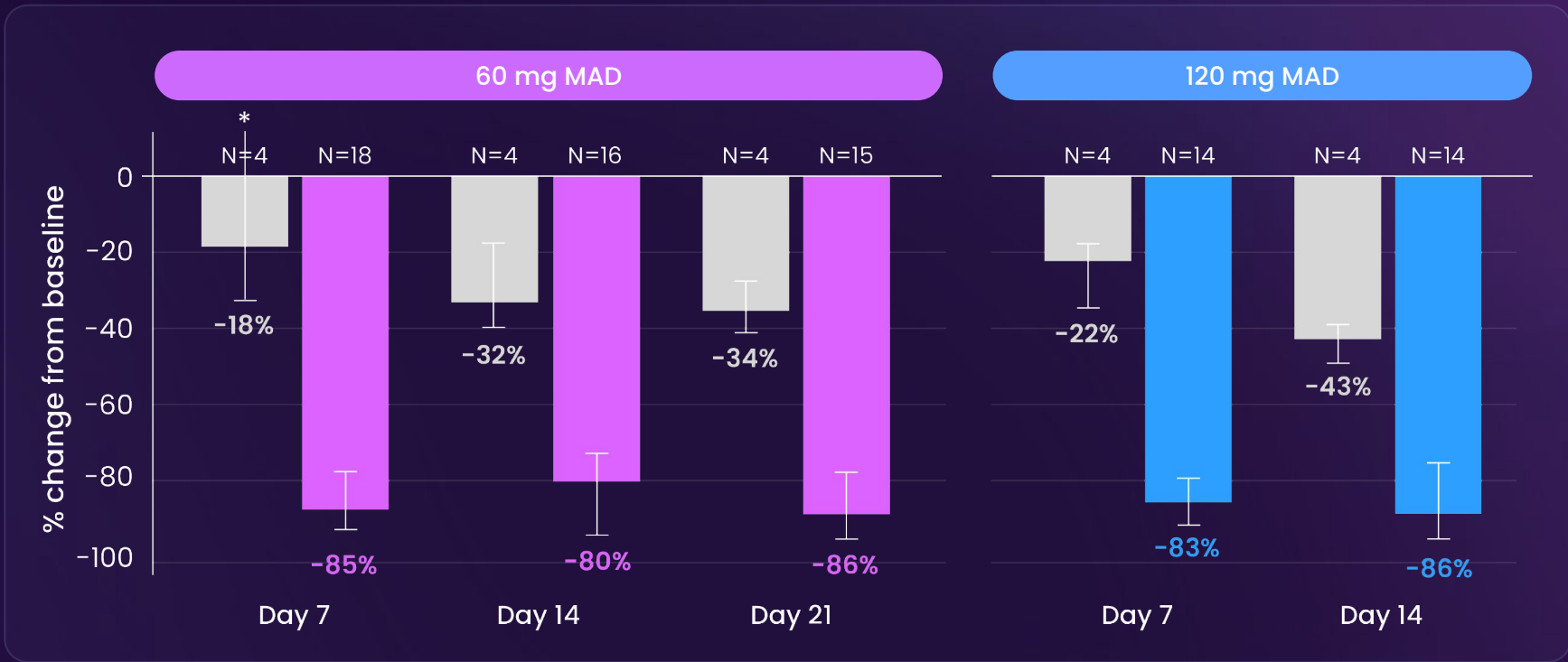
Obese MAD

Therapeutic areas

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

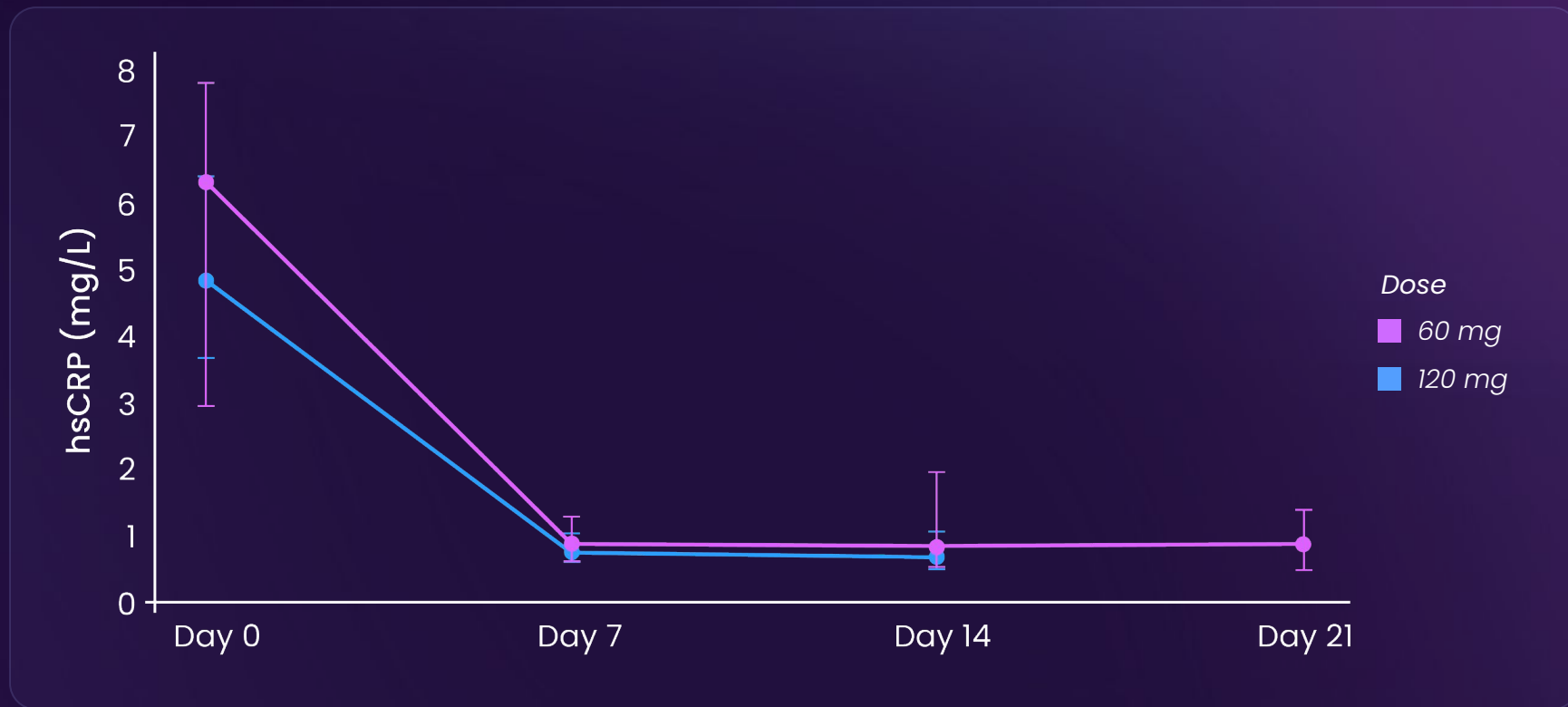


60 mg & 120 mg doses of BGE-102 resulted in 86% reductions in hsCRP, consistent with best-in-class efficacy

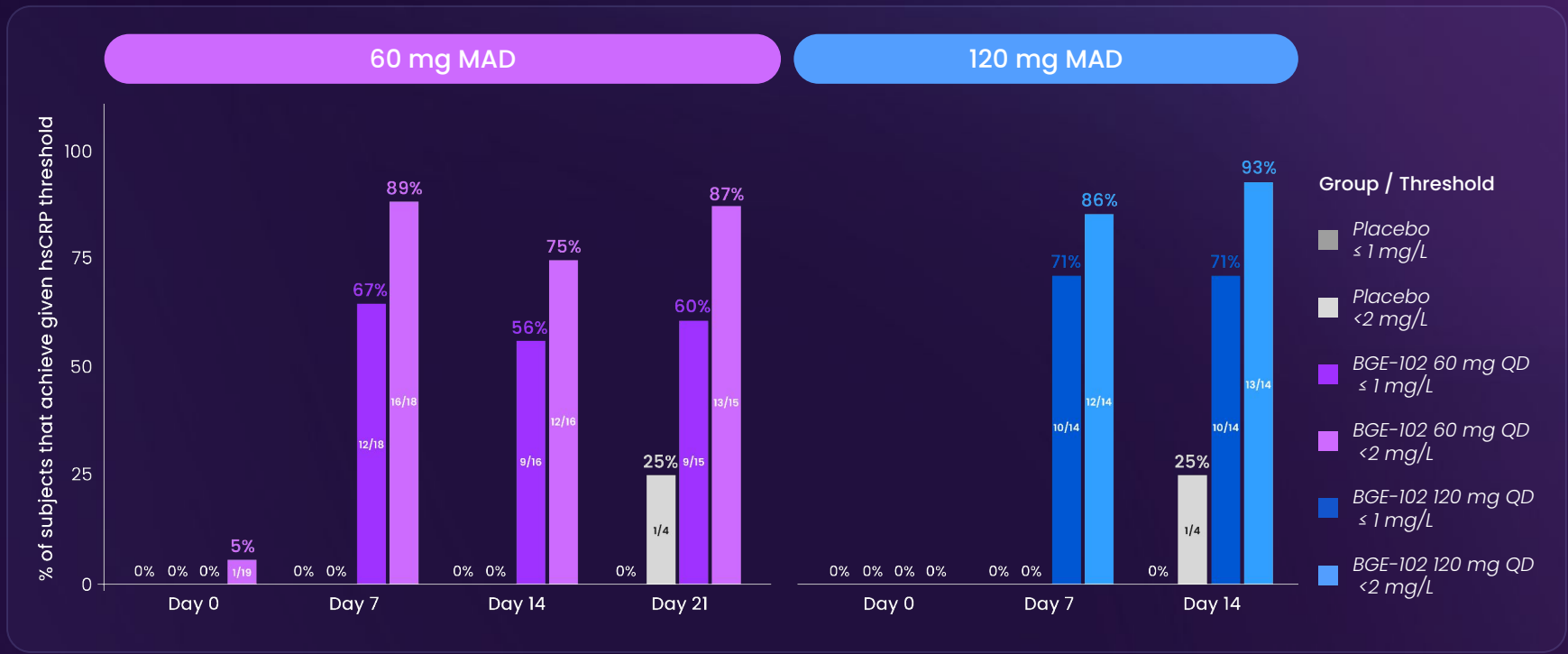


Note: median values, error bars show IQR (Q1-Q3); * Day 7 placebo IQR upper limit is 115%; median baseline hsCRP 60 mg cohort 6.30 mg/L for both active treatment and placebo, 120 mg cohort 4.85 mg/L for active treatment and 4.25 mg/L for placebo

Similar hsCRP trajectories in both obese MAD cohorts

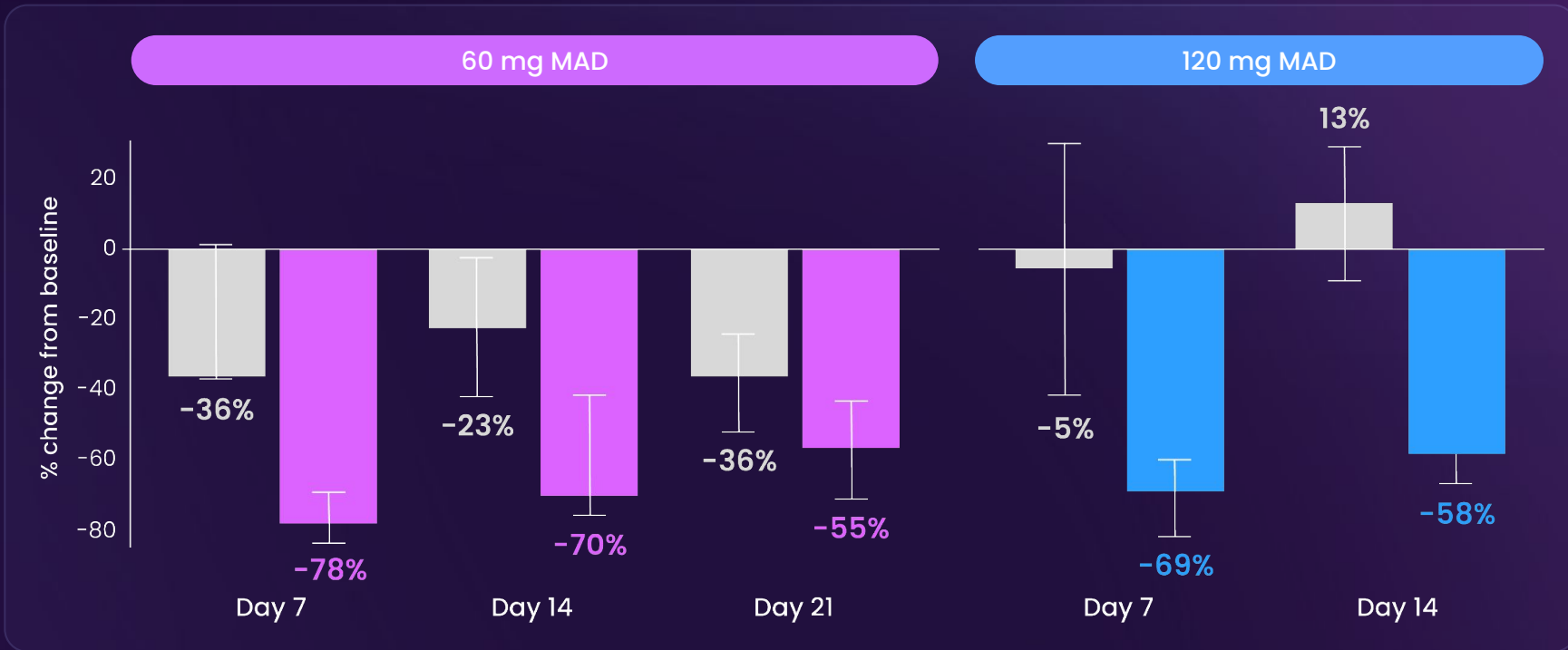


87%–93% of subjects on BGE-102 achieved normal hsCRP levels (<2 mg/L)



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

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

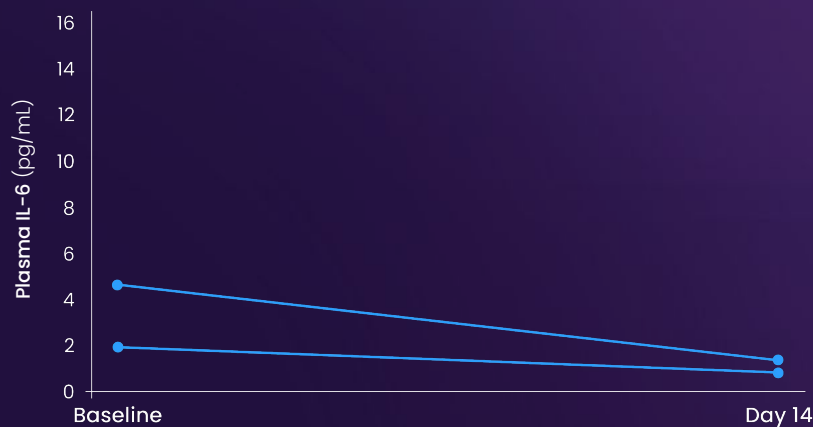


In the 2 subjects with elevated baseline CSF IL-6, BGE-102 reduced levels by 84% in the Day 14 CSF

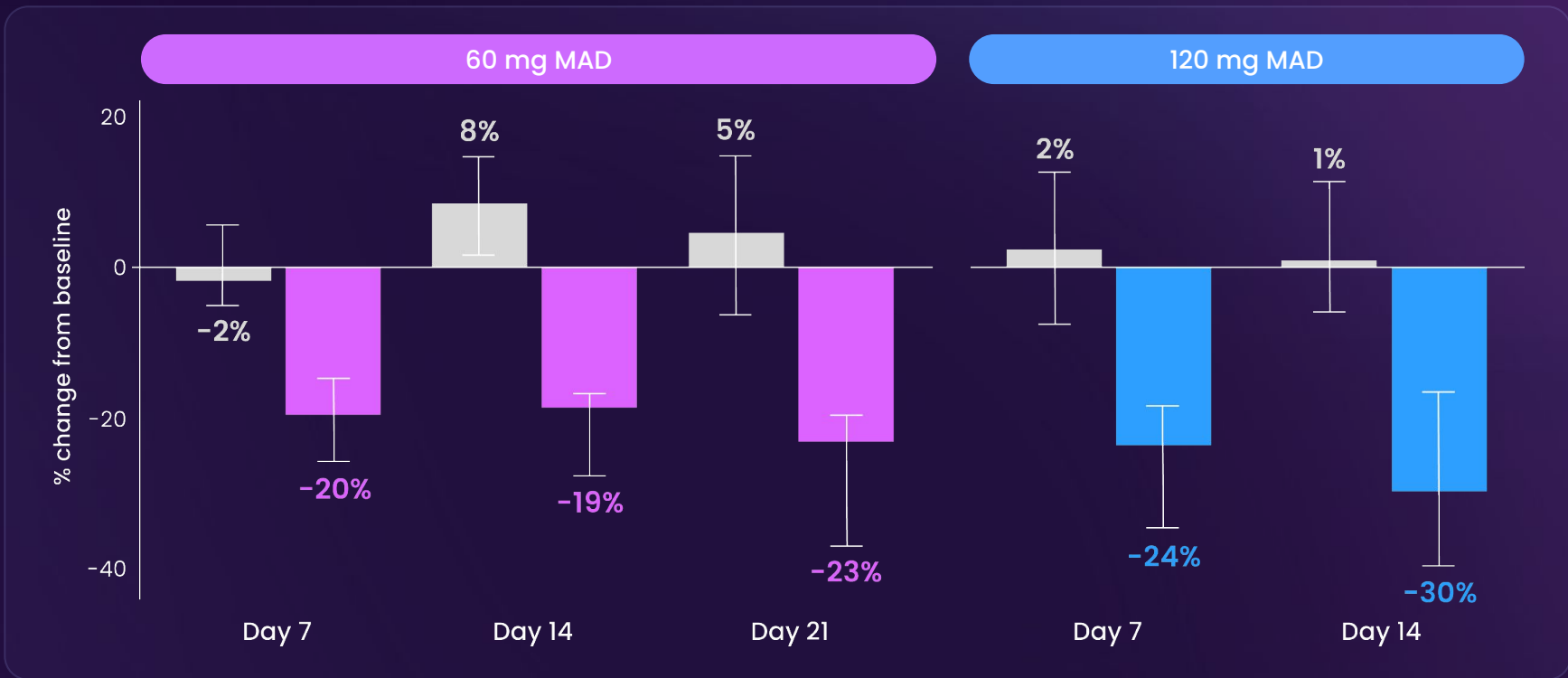
CSF IL-6*



Plasma IL-6



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



NLRP3

BGE-102 overview

Phase 1 results

Therapeutic areas

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

Neuro-inflammation

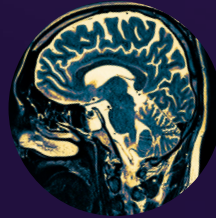


Peripheral inflammation



BGE-102 addressable diseases

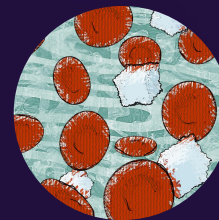
Neurodegeneration



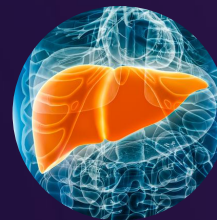
Ophthalmology



CVD
(ASCVD, HF)



Insulin
resistance



MASLD /
MASH

NLRP3

BGE-102 overview

Phase 1 results

Therapeutic areas

Cardiovascular

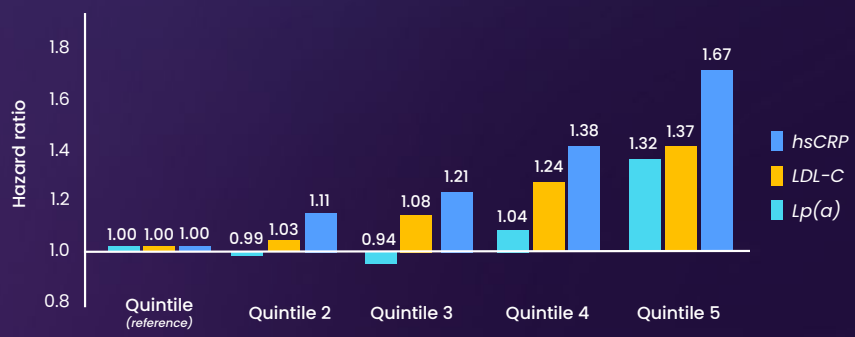
Ophthalmology

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

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

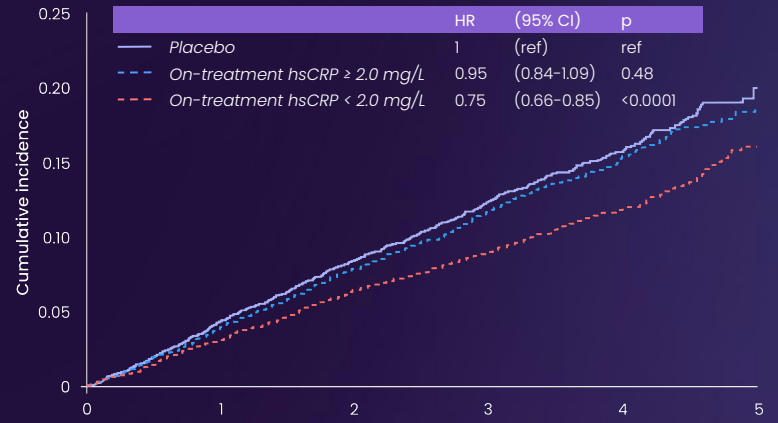
Risk of MACE in 30-year longitudinal data from the Women's Health Study

The NEW ENGLAND JOURNAL of MEDICINE



CANTOS trial validated the anti-inflammatory strategy

25% reduction in MACE for patients with hsCRP normalization



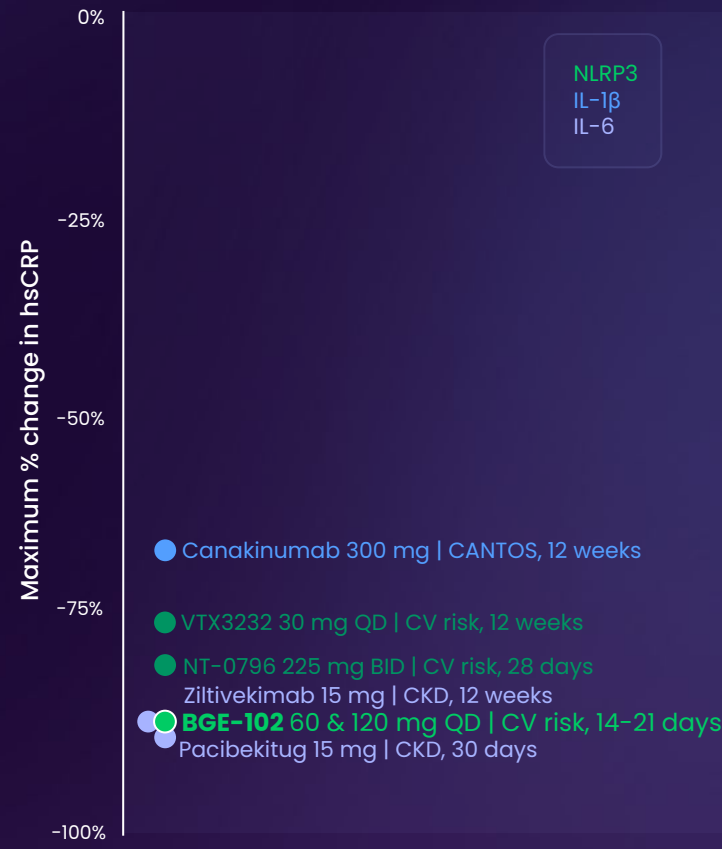
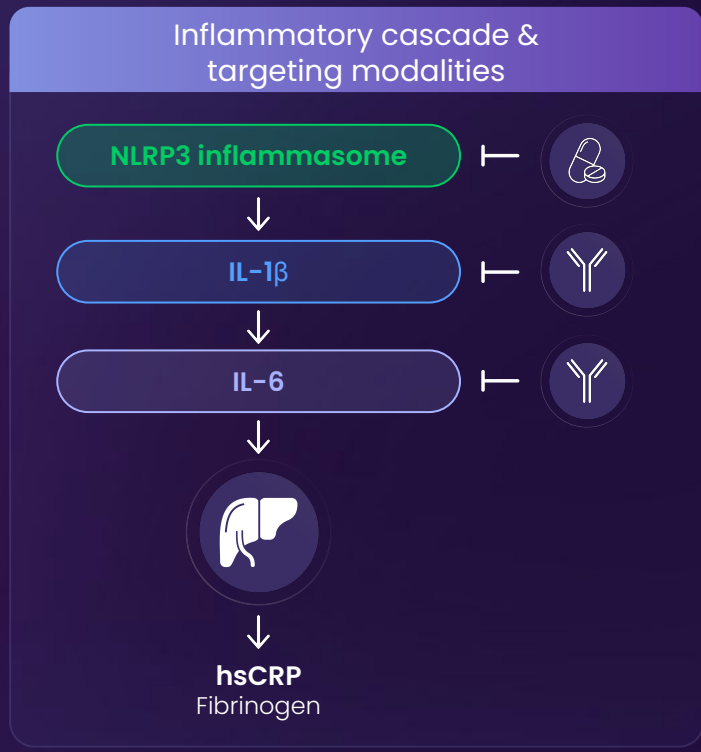
ACC SCIENTIFIC STATEMENT

Inflammation and Cardiovascular Disease: 2025 ACC Scientific Statement

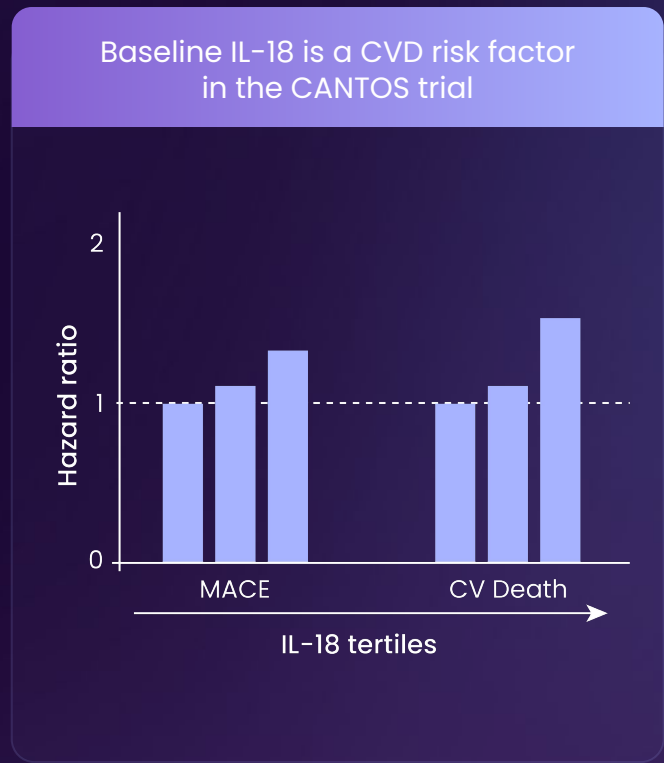
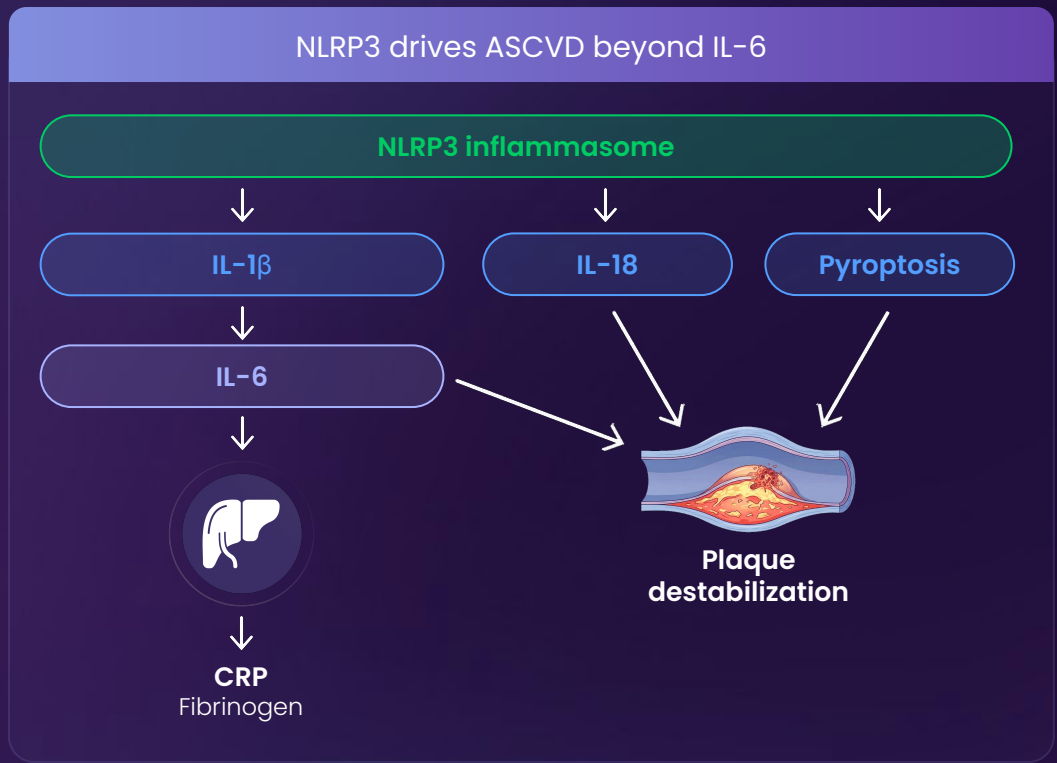
A Report of the American College of Cardiology

"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."

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



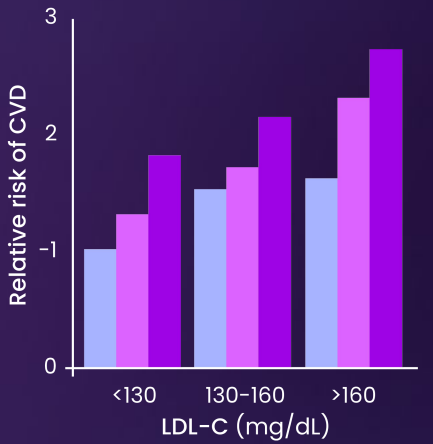
NLRP3 inhibition also has potential benefits beyond IL-6, reducing IL-18 & cell death that contribute to plaque destabilization



The CV opportunity

Inflammation is an independent risk factor for CVD

hsCRP (mg/L) ■ <1.0 ■ 1.0-3.0 ■ >3.0



15M addressable patients in the US alone

~60%
of 25M ASCVD patients have elevated hsCRP

Orals are highly preferable

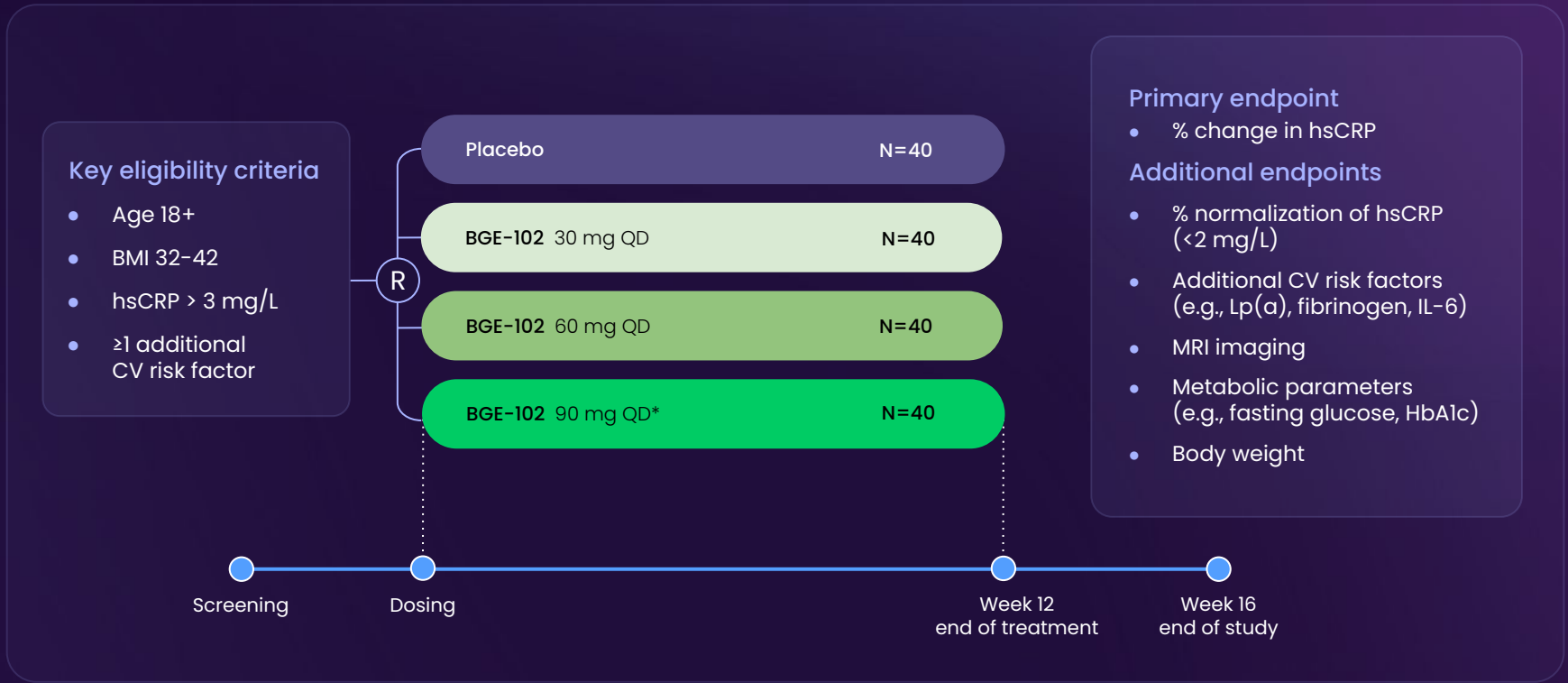
>80%
of statins are prescribed by PCPs

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

QUELL_{CV} Phase 2 design



39 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

The retinal opportunity for BGE-102



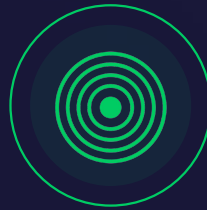
**Greater efficacy
with new MOA**



**Oral treatment to lower
treatment burden**



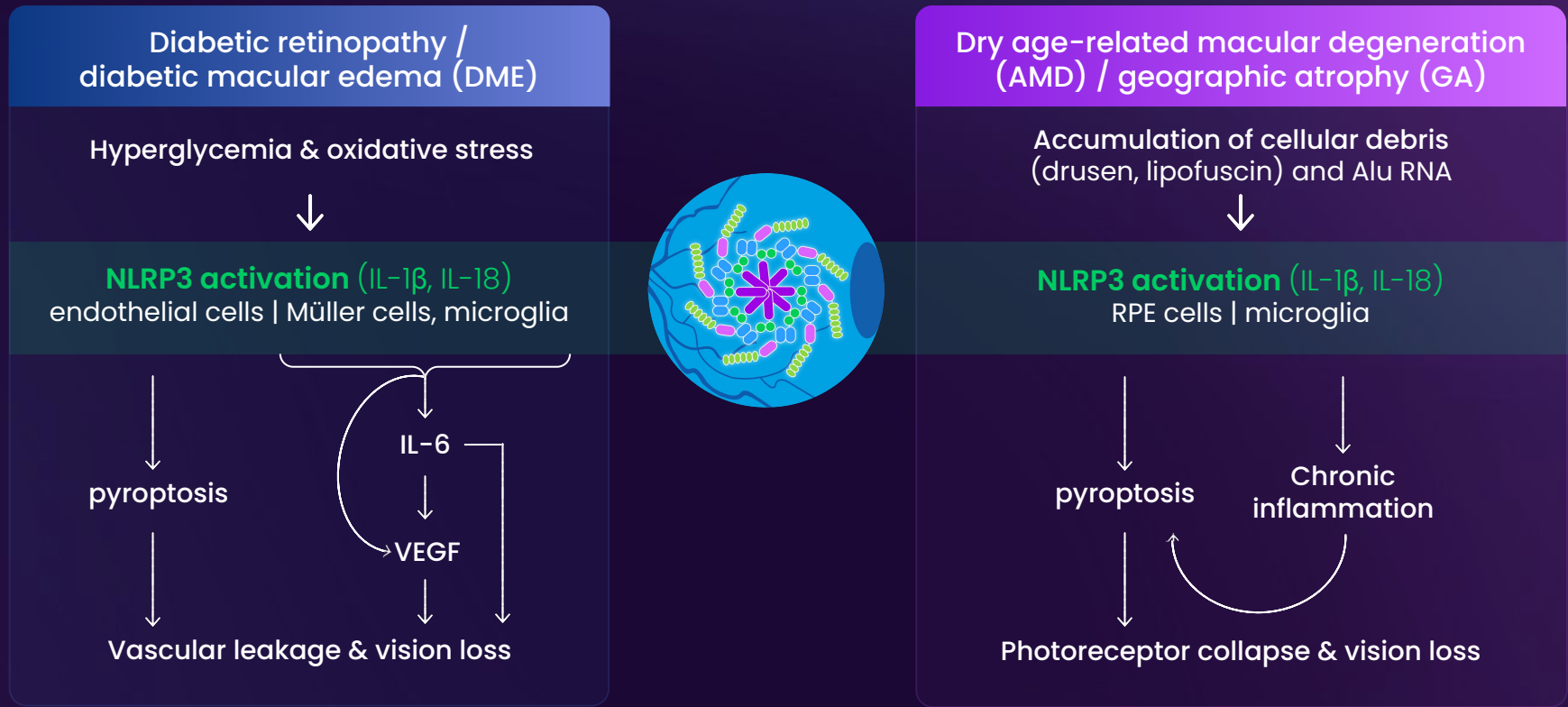
**Simultaneous treatment
of bilateral disease**



**Ocular *and* systemic
biology addressed**

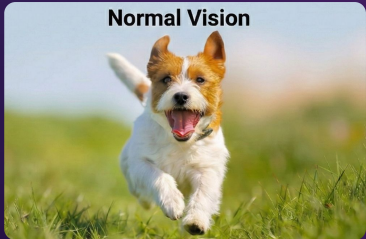
BGE-102 has shown therapeutic retinal exposure across species, including primates

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







Prevalent retinal diseases with significant unmet needs

	DME	GA (late-stage AMD)
Description	<ul style="list-style-type: none"> Vision loss due to vascular leakage Leading cause of vision loss in adults <65 	<ul style="list-style-type: none"> Loss of central vision 20% become legally blind within 1 year of diagnosis
Prevalence (US)	<ul style="list-style-type: none"> 1M 	<ul style="list-style-type: none"> 1.2M
Key risk factors	<ul style="list-style-type: none"> Hyperglycemia 	<ul style="list-style-type: none"> Age, inflammation, cardiometabolic disease
Approved therapies	<ul style="list-style-type: none"> Intravitreal anti-VEGFs, intravitreal steroids, laser 	<ul style="list-style-type: none"> Intravitreal complement inhibitors (Syfovre, Izervay)
Key unmet needs	<ul style="list-style-type: none"> Reduced injection burden: real world treatment outcomes are suboptimal given poor compliance New MOAs: significant anti-VEGF refractory population 	<ul style="list-style-type: none"> Disease control: approved therapies offer limited efficacy



Key treatment challenges

 <p>Limited efficacy of approved therapies</p>	 <p>High injection burden</p>	 <p>Eyes treated individually</p>	 <p>Systemic disease drivers not addressed</p>
<p>DME</p> <ul style="list-style-type: none"> ~45% of patients are refractory to anti-VEGFs despite compliance <p>GA</p> <ul style="list-style-type: none"> ~90% are <u>not</u> treated given current drugs only slow progression 15-20% 	<ul style="list-style-type: none"> ~50% discontinue anti-VEGFs after an average of 6 months <p>(Bi)monthly injections required for approved therapies, often in elderly patients</p>	<ul style="list-style-type: none"> ~70% develop bilateral disease within 1 year of diagnosis <ul style="list-style-type: none"> ~65% develop bilateral disease; treatment limited to 1 eye per visit 	<ul style="list-style-type: none"> Insulin resistance is a key risk factor for refractory disease Inflammation is both a risk factor & prognostic marker

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



KODIAK



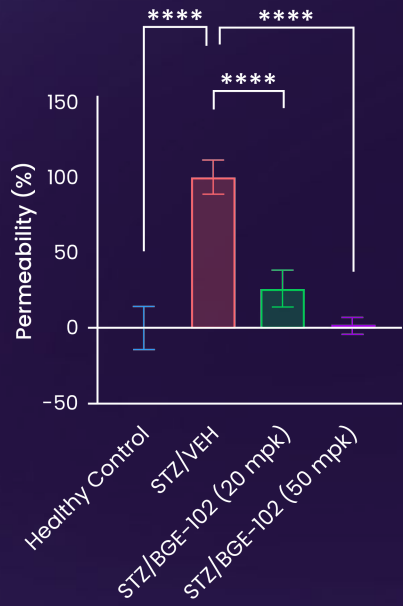
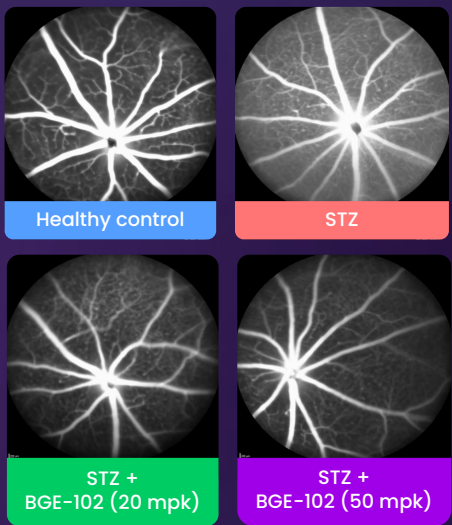
Orphan strategy: uveitic macular edema

Steroids are SOC and anti-IL-6 has shown robust efficacy

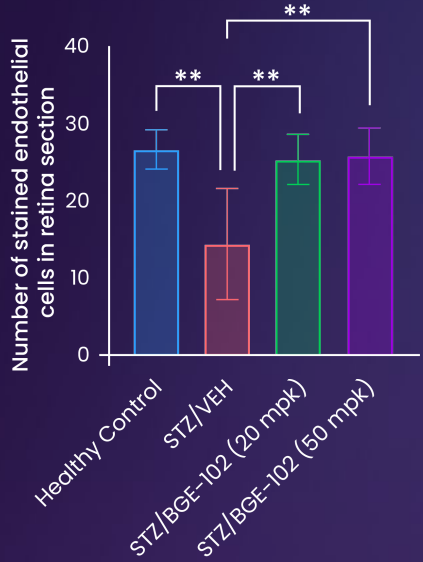
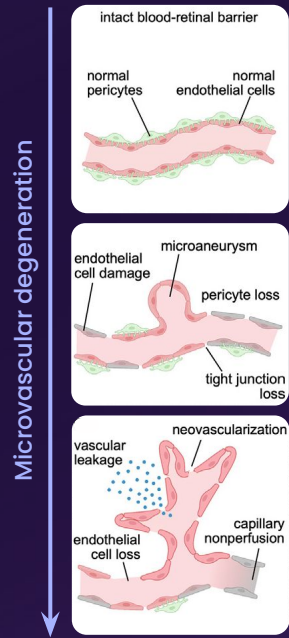
MEERKAT trial: up to 36.9% of patients achieved a ≥15 letter improvement from baseline in BCVA at week 16

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

Retinal vascular permeability measured via fluorescein angiography



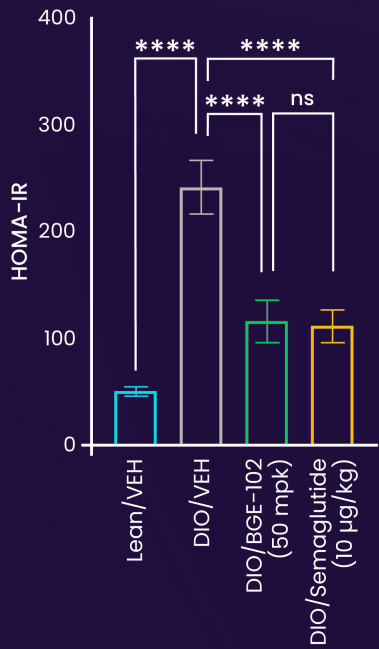
Blood-retinal barrier integrity claudin-5 IHC (endothelial tight junctions)



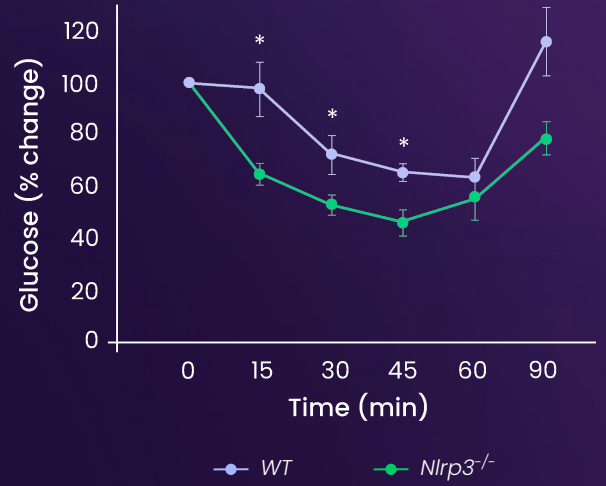
46 Note: Streptozotocin (STZ) diabetic mouse model; BGE-102 dosing initiated 11 days following last dose of STZ; assessments performed after 3 months of BGE-102 QD dosing; BGE-102 is more potent in human cells, for example 150-250x higher potency in microglia
Source: Wolf 2023

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)

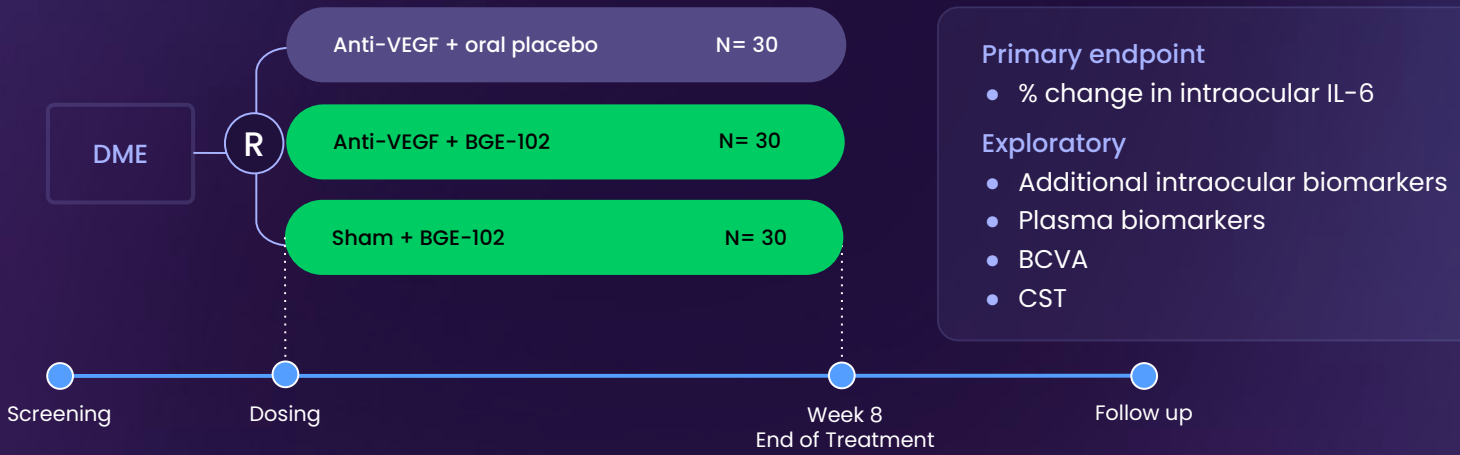


Nlrp3 -/-
Insulin tolerance test
 (6 weeks)



QUELL_{DME} Planned Phase 1b/2a design

Goal: demonstrate PD in the eye



Goals of DME POC:

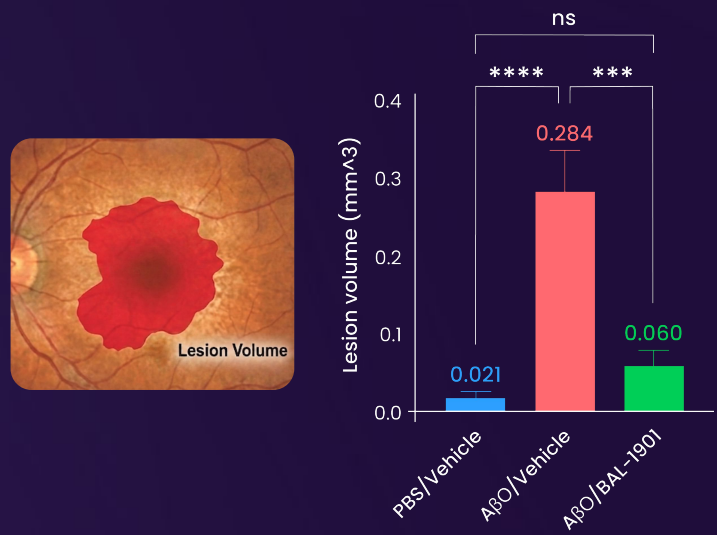


- Demonstrate PD in the eye with oral delivery of BGE-102
- Motivate subsequent development in additional ophthalmology indications, including GA

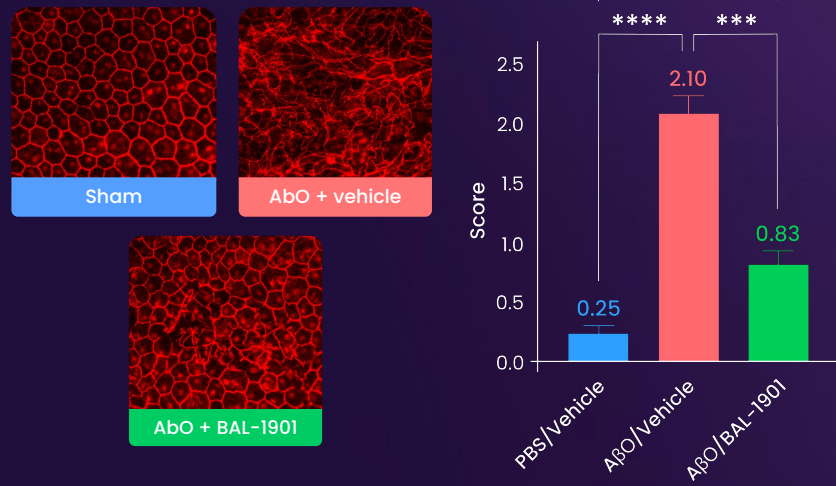
GA: oral delivery of BGE-102 analog prevented AβO-driven lesion formation & improved RPE morphology in a mouse model

GA model: subretinal injection of amyloid β oligomers (AβOs)
 AβOs are a major drusen component in dry AMD/GA and drive RPE degeneration

Lesion volume in line with controls (day 7)

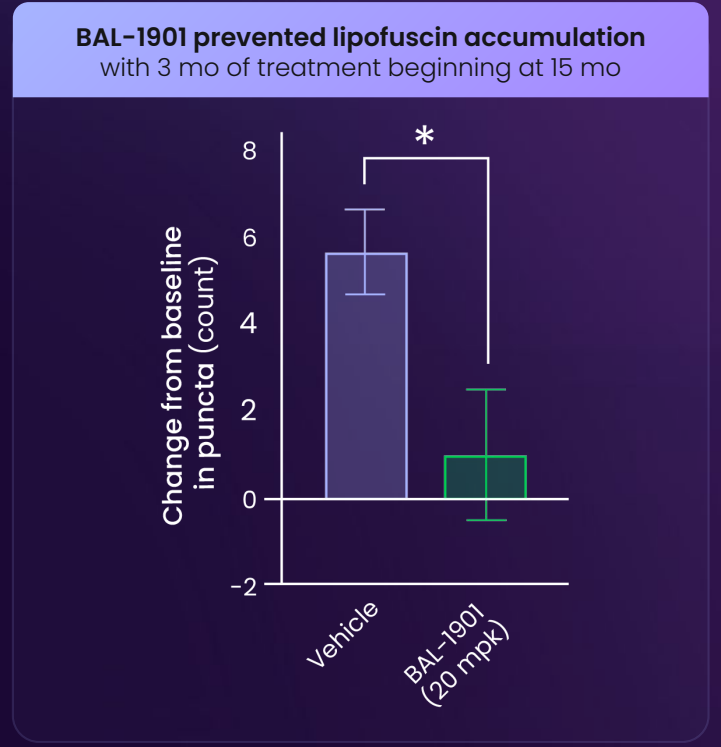
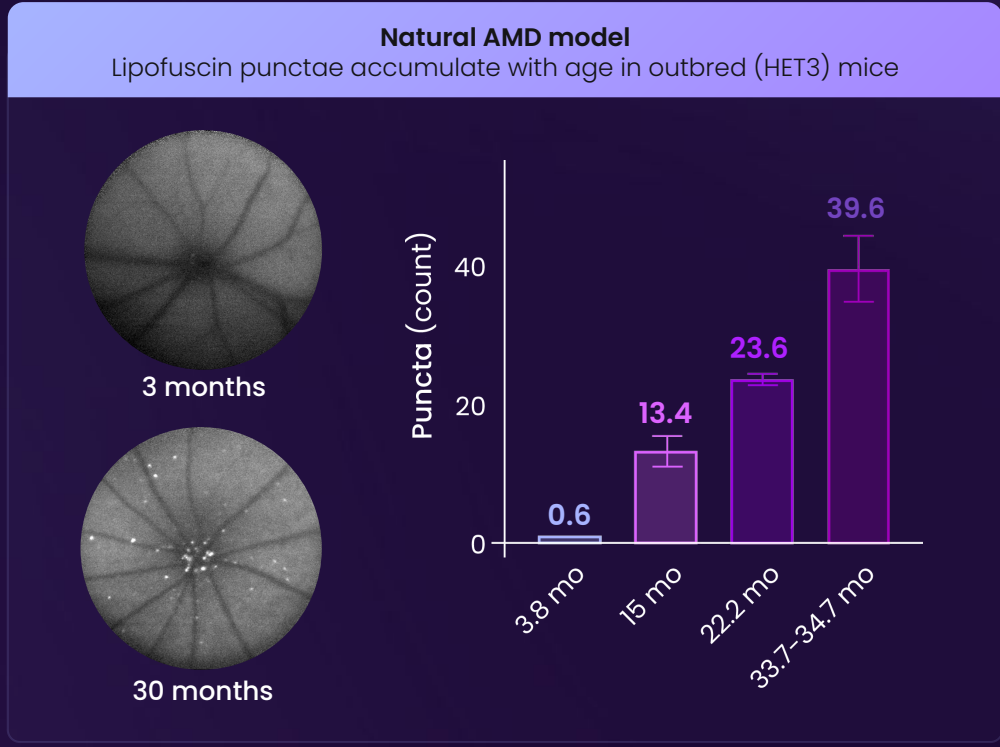


RPE morphology (day 8)



Note: 3-month-old humanized NLRP3 mice were treated with oral BAL-1901 (20 mpk PO QD) initiated 3 days prior to AβO injection and continued through Day 7. Similar results reported in Nlrp3 knockout mice (Narendran 2021)

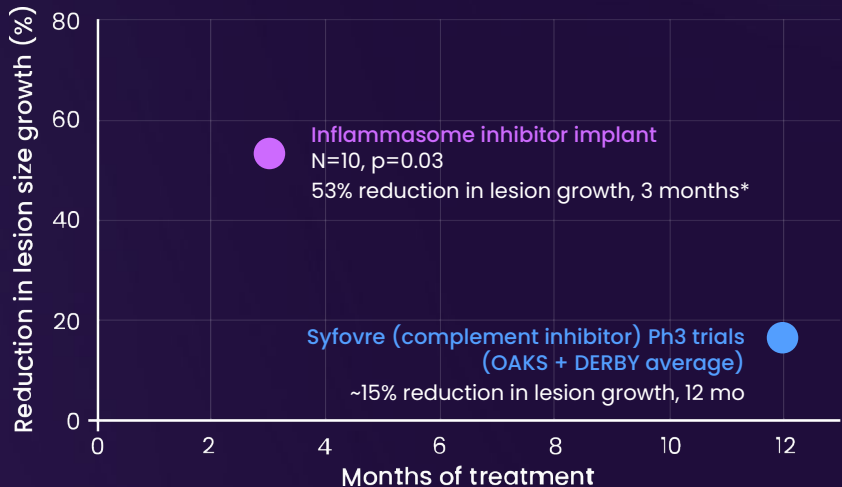
GA: In a natural AMD preclinical model, oral delivery of a BGE-102 analog prevented age-related lipofuscin accumulation, a key disease feature



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

Pilot study shows potential for greater efficacy

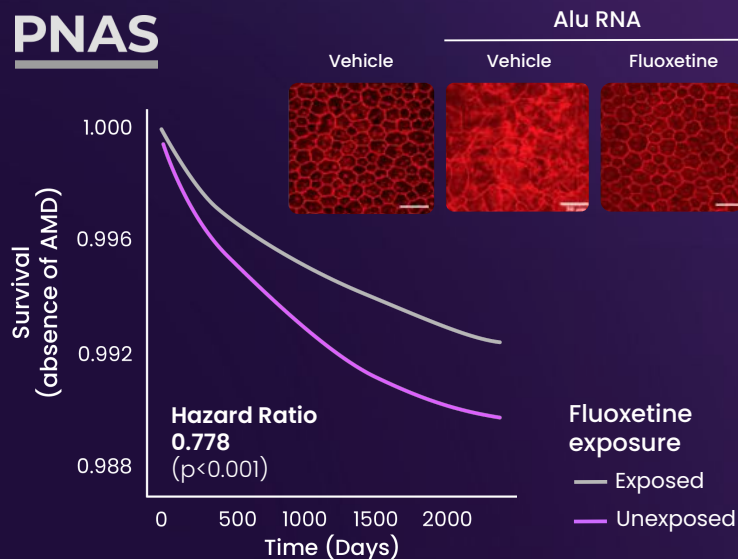
Inflammasome inhibitor intravitreal implant, K8, vs. complement inhibition



Decreased incidence of AMD

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

PNAS



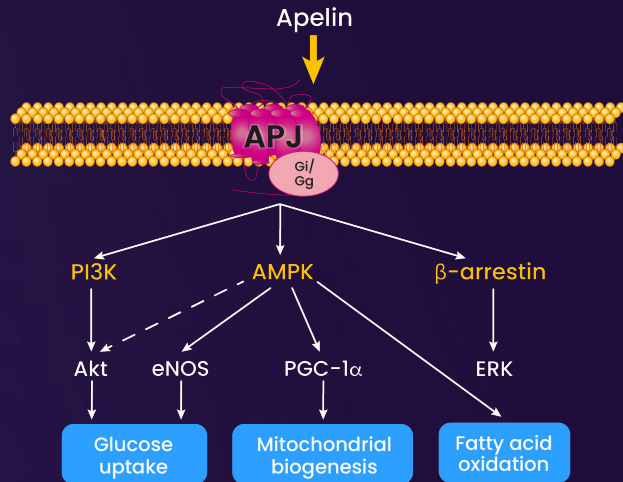
Apelin / APJ

Apelin is an exerkin and mimics many benefits of exercise

Shared biology between apelin & exercise



Exercise stimulates release of apelin into circulation



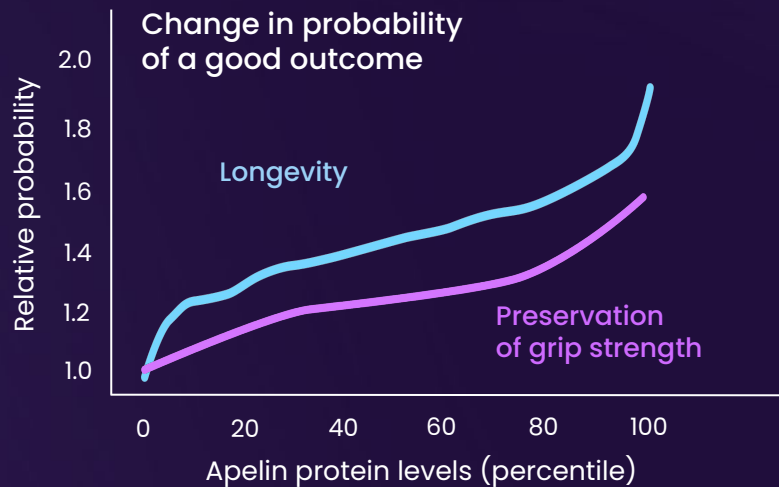
Systemic benefits

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

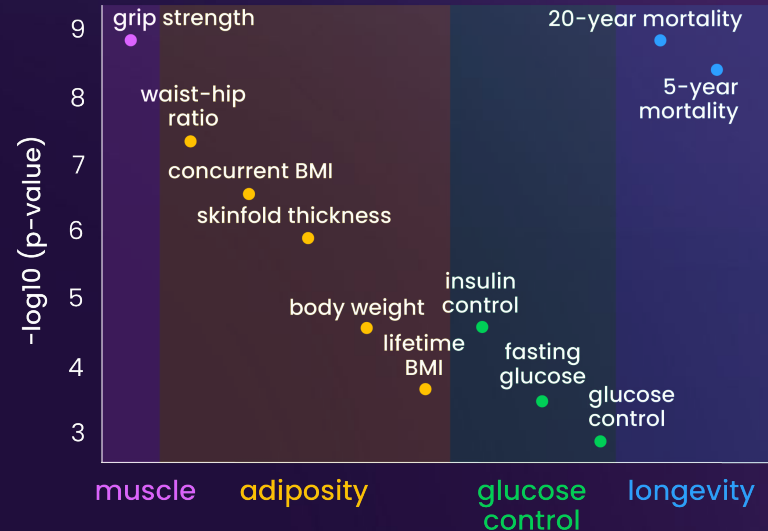


Human insights from the BioAge platform: Apelin signaling impacts muscle & metabolism

BioAge aging dataset



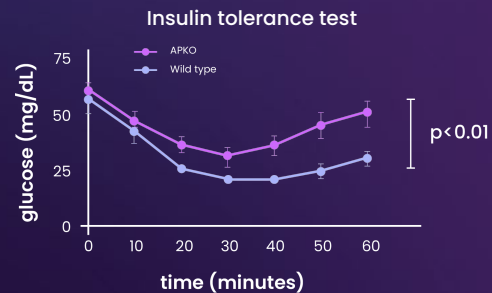
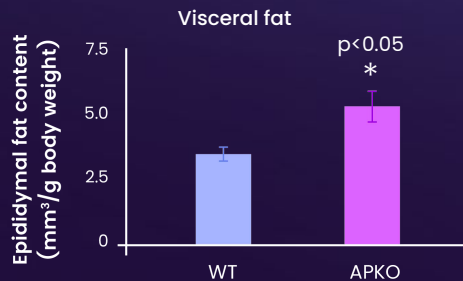
Human proteomic associations



Apelin genetics reinforce beneficial role in systemic metabolism

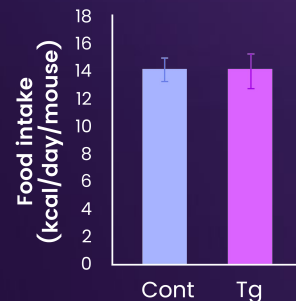
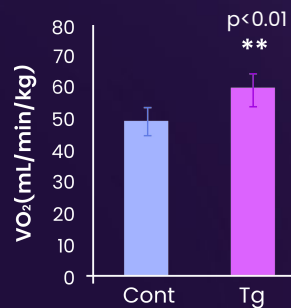
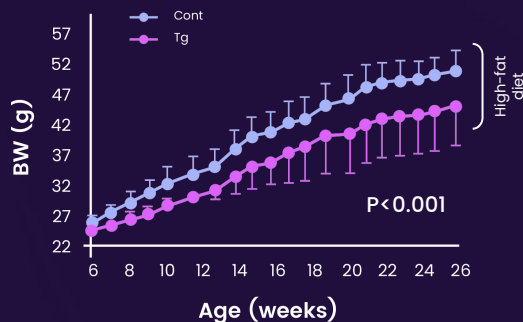
Apelin KO

- ↑ adiposity
- ↑ insulin resistance



Apelin transgenic

- ↓ weight gain on high fat diet
- ↑ basal metabolic rate
- No impact on energy intake

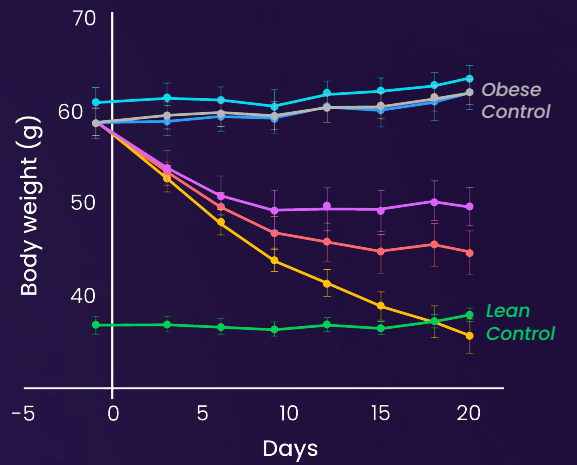


Consistent genetic evidence in humans:

Genome-wide significant associations for the apelin receptor APJ include BMI, lean mass, and serum lipids

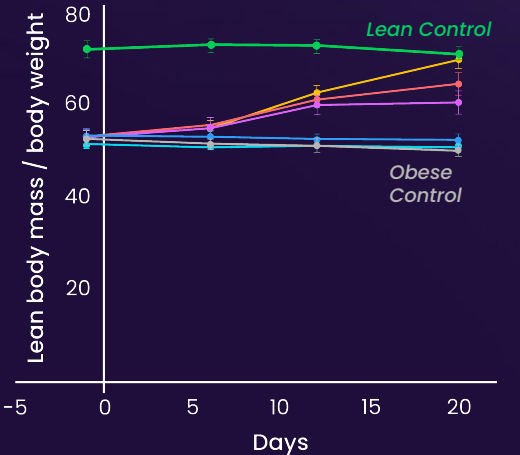
We previously demonstrated that APJ agonism can restore body weight & body composition to that of lean controls when combined with an incretin

Overall weight loss

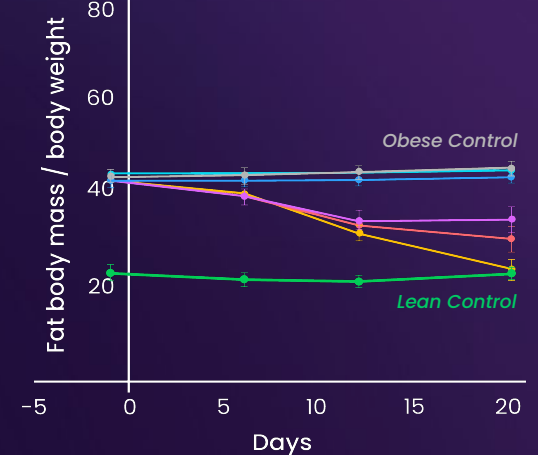


Body composition

% Lean Mass



% Fat Mass



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

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

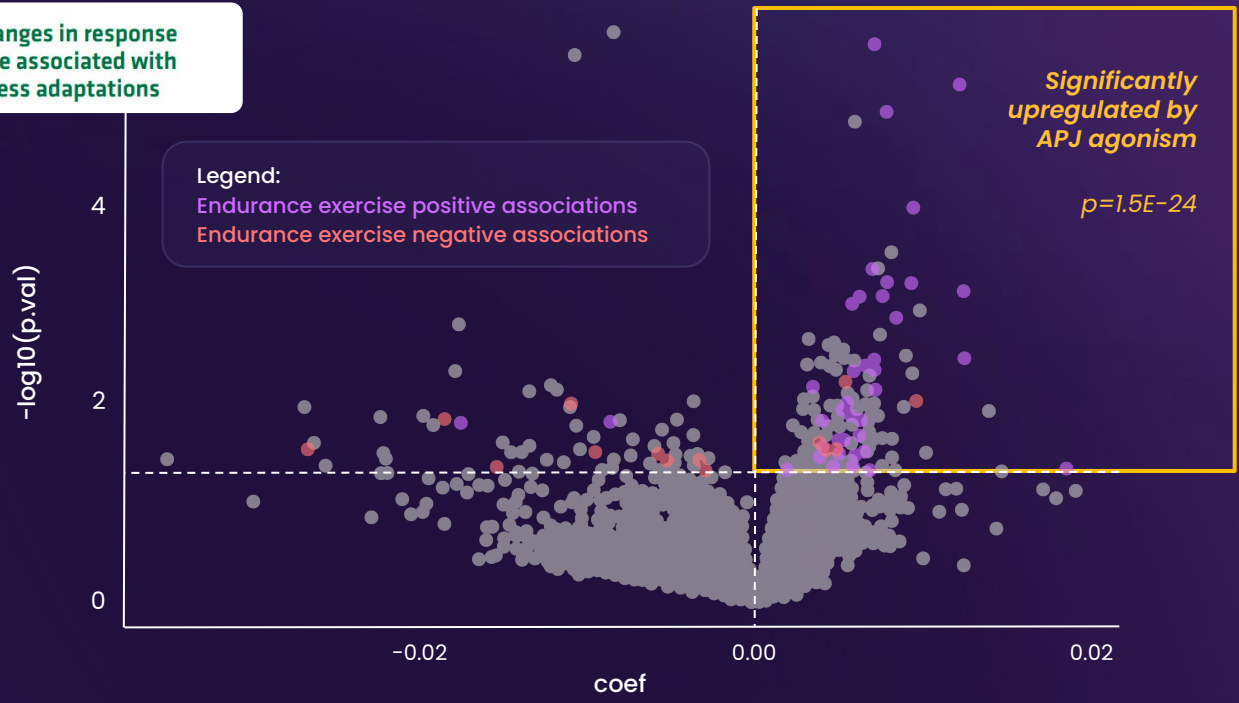
Metabolism

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

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



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



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

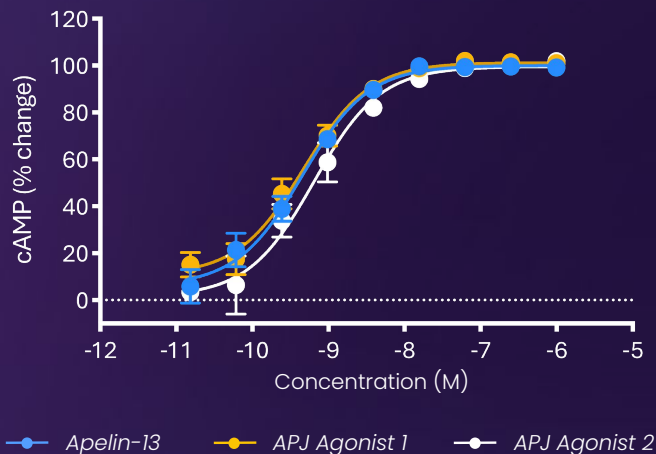
Recent progress on APJ



Oral

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

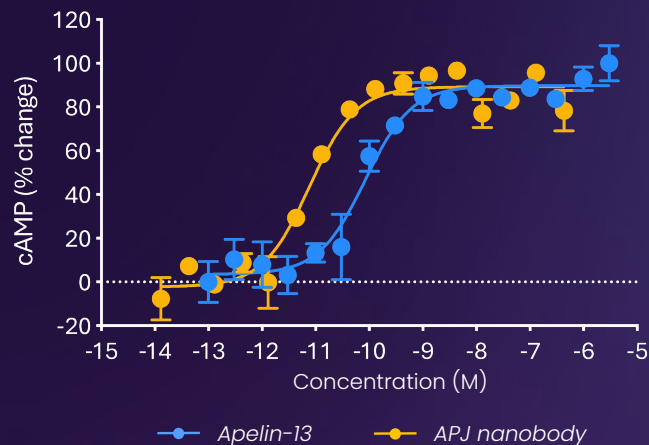
APJ agonist activity (cAMP)



Parenteral

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


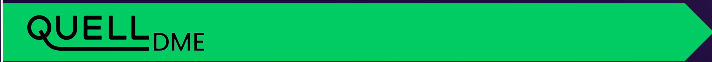





APJ agonist activity (cAMP)



Collaboration with JiKang Therapeutics

Pipeline overview

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

Program	Mechanism of action	Target dosing	Indication	Discovery	Lead op	IND-enabling	Phase 1	Phase 2	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	 Lilly					
Program 2	Undisclosed			 Lilly					
Target discovery	Multiple targets	-	-	 NOVARTIS					

BIOAGE