BIOAGE

Corporate Presentation
4Q24

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

BJ Sullivan, PhD
Chief Strategy Officer

~800
Clinical trials*

~130
INDs*

~95
US regulatory approvals*

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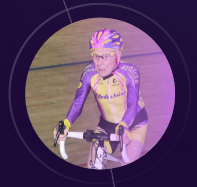
We are harnessing the biology of human aging to develop new therapies for metabolic diseases



Azelaprag: an oral exercise mimetic for obesity in Ph2

3 key potential benefits: increased weight loss, improved tolerability, improved body composition

- Mechanism: small molecule apelin receptor APJ agonist
- Core value proposition: potential injectable-like weight loss (20%+) in an all-oral incretin combination
- Potential for significant upside: improved body composition & tolerability
- Clinical results: promoted muscle metabolism in Ph1b; well tolerated in 265 subjects
- Preclinical results: 2x overall weight loss with incretins; strong synergy with other satiety mechanisms
- 3x Ph2 trials planned: STRIDES with Zepbound (ongoing); STRIDES 2 with Wegovy; STRIDES T2D in type 2 diabetes

Oral NLRP3 inhibitor

- Internally discovered compound
- High potency & brain penetration
- Novel structure, mechanism, and binding site

BioAge platform

- Target discovery platform focused on muscle and metabolic health
- Proprietary multi-omics spanning 50+ years of human aging

Pipeline overview

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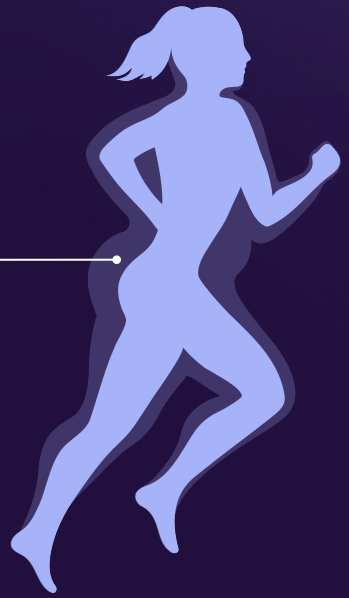
Exercise mimetics can address key unmet needs in obesity

Exercise mimetics for obesity

Benefits of targeting exercise

- Safe way to increase energy expenditure
- Highly translational benefits



Key potential clinical value propositions

- Increased oral weight loss
- Improved body composition
- Improved tolerability

Therapeutic approach

Incretins +
exercise mimetic

Potential pharmacological parallel to diet and exercise

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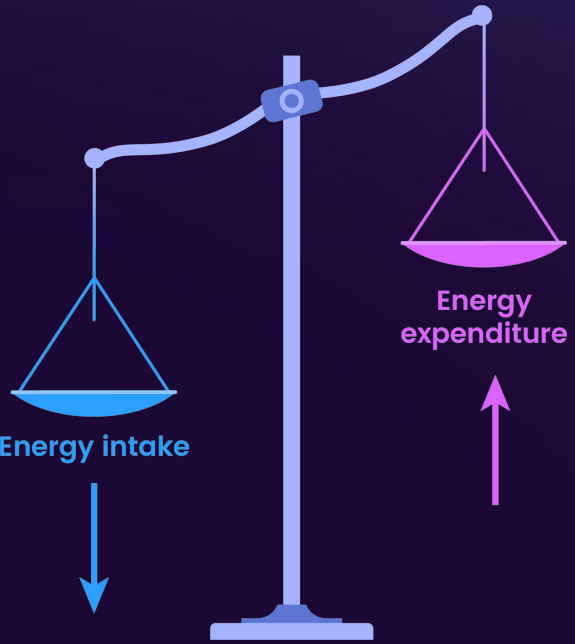
Optimizing energy balance: the vast majority of oral development in obesity targets energy intake; far fewer target energy expenditure

Clinical-stage oral obesity programs

Incretins
*GLP-1, GIP, amylin**
19

Pfizer · AstraZeneca · novo nordisk · Roche · Lilly

Other mechanisms
13



Exercise mimetics
2

Azelaprag BIOAGE

Other mechanisms
6

- Mitochondrial uncoupling
- AMPK
- Androgen receptor
- MAS receptor

Energy intake + energy expenditure complementary mechanisms that can be combined to enhance weight loss quantity and quality

*Note: * Amycretin is an oral co-agonist of GLP-1 and amylin*
Source: GlobalData

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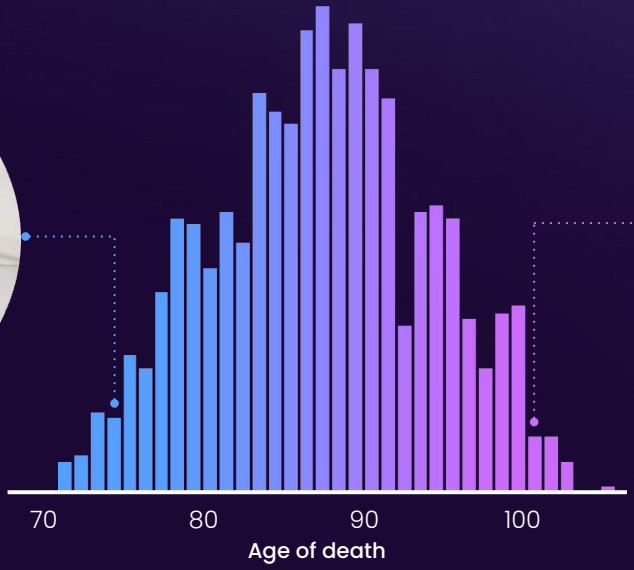
Azelaprag

Obesity

We know there are many pathways that impact human lifespan and metabolic health; it is our mission to find them and develop therapeutics to target them



A 50+ year natural human experiment

50M+	10K+	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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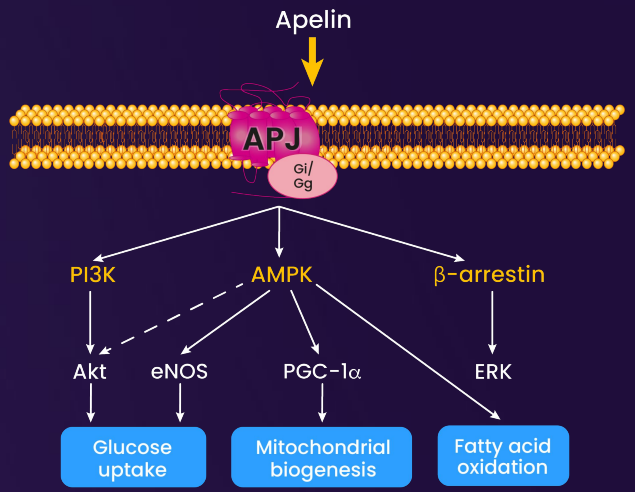
Apelin is an exerkine and mimics many benefits of exercise

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

AZELAPRAG: TOLERABILITY

Azelaprag has a favorable tolerability profile in 265 subjects across Phase 1 studies

Treatment-emergent adverse events with >3% incidence

		Placebo (N=62)	Azelaprag (N=265)
Severe None have been reported	Headache	0	0
	Dizziness	0	0
	Back Pain	0	0
Moderate Only headaches observed in both active and placebo groups	Headache	1 (1.6%)	1 (0.4%)
	Dizziness	0	0
	Back Pain	0	0
Mild	Headache	2 (3.2%)	21 (7.9%)
	Dizziness	2 (3.2%)	5 (1.9%)
	Back Pain	2 (3.2%)	2 (0.8%)

Toxicology coverage

- No observed adverse effect level = highest dose tested
- 5-10x safety margin for Ph2 doses

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AZELAPRAG: PH1B RESULTS

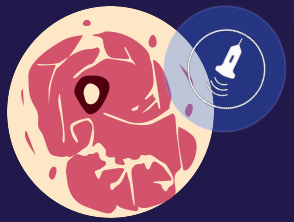
Azelaprag significantly improved muscle atrophy and metabolism in our Ph1b trial of older subjects bed rest for 10 days

	Healthy volunteers age 65+	N=10 placebo N=11 azelaprag 240mg *delivered daily via IV*	10 days of bed rest & dosing

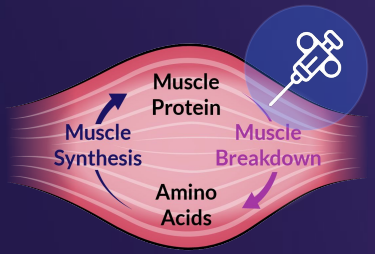
Thigh circumference

Muscle size

Fat infiltration



Muscle metabolism



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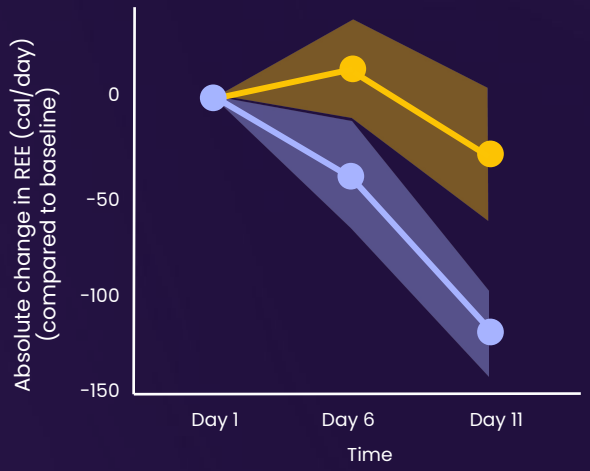
Azelaprag induced shifts in the serum proteome that are indicative of preserved resting energy expenditure and VO_2 max

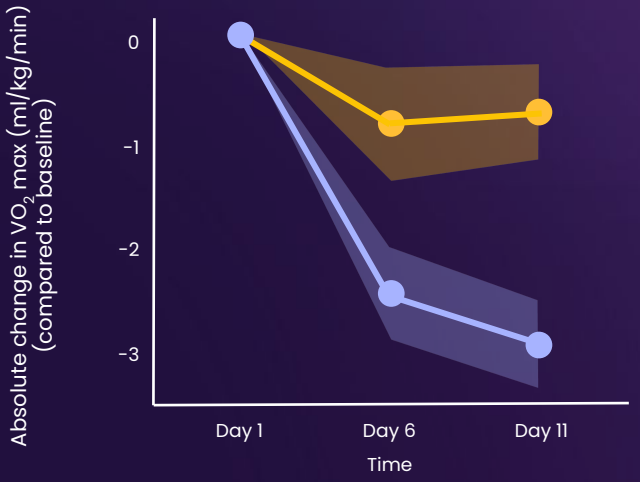
Predicted resting energy expenditure

End of treatment: p=0.03



Predicted cardiorespiratory fitness: VO_2 max

End of treatment: p=0.004



Treatment Group — *Placebo* — *Azelaprag*

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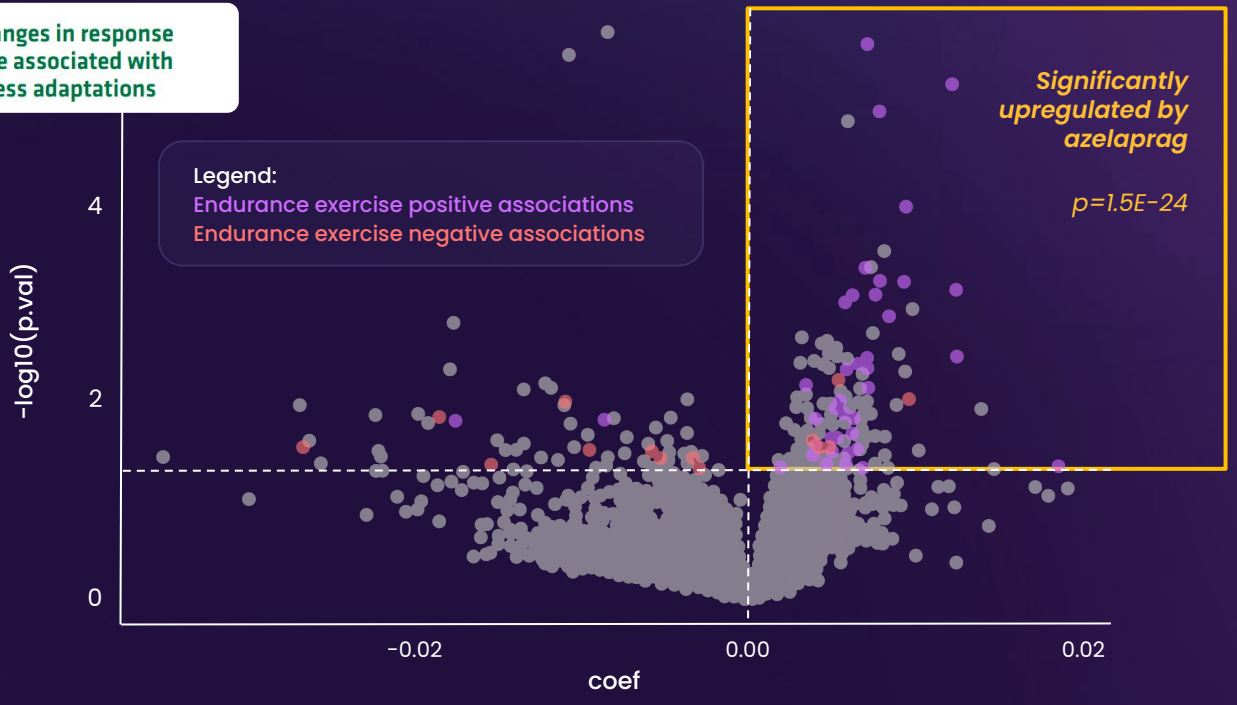
Azelaprag shifted the serum proteome consistent with being an exercise mimetic

JCI insight

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



Source: Robbins 2023

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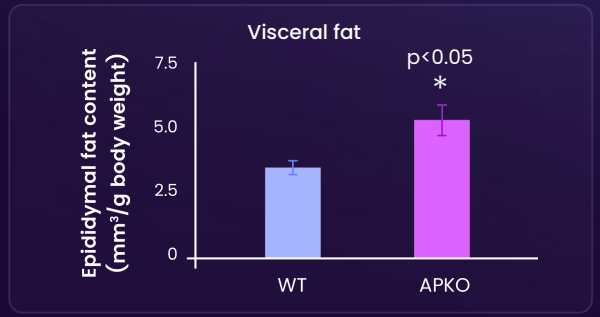
AZELAPRAG IN OBESITY

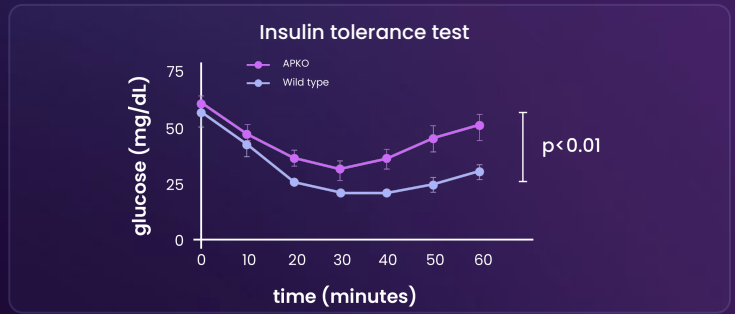
Apelin genetics reinforce beneficial role in systemic metabolism

Apelin KO

↑ adiposity

↑ insulin resistance



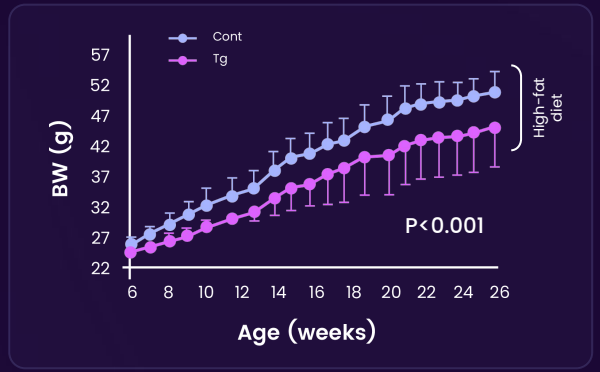


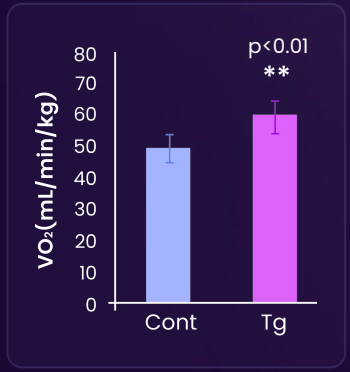
Apelin transgenic

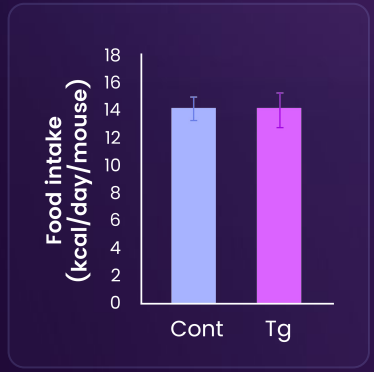
↓ weight gain on high fat diet

↑ 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

AZELAPRAG IN OBESITY

Many ways to win: azelaprag, in combination with an incretin, has the potential to address key unmet needs, including oral weight loss, tolerability, and body composition

Target performance: azelaprag + incretin drug	Target	Upside
Route of administration	Oral co-administration	
Primary endpoint	**20% overall weight loss** Clear regulatory path: ~5% improvement vs. GLP-1 alone*	**25% - 30% overall weight loss** Preclinical models show potential for higher magnitude of benefit
Secondary endpoints / additional differentiation		Improvements in: • Body composition *Prevention of rebound weight gain & frailty* • Glucose control • Tolerability • GLP-1 titration needs

*Note: * Oral GLP-1 orforglipron (Lilly) achieved up to 14.7% avg. weight loss at week 36 across doses in Ph2; oral semaglutide (Novo) achieved 15.1% avg. weight loss at 68 weeks in Ph3a*
Source: Evercore research; Wharton 2023 (Lilly orforglipron Ph2 results); Novo Press Release (semaglutide Ph3 results)

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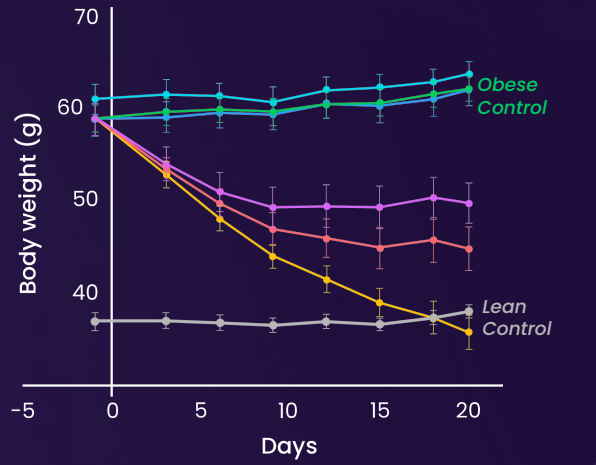
AZELAPRAG IN OBESITY: PRECLINICAL RESULTS

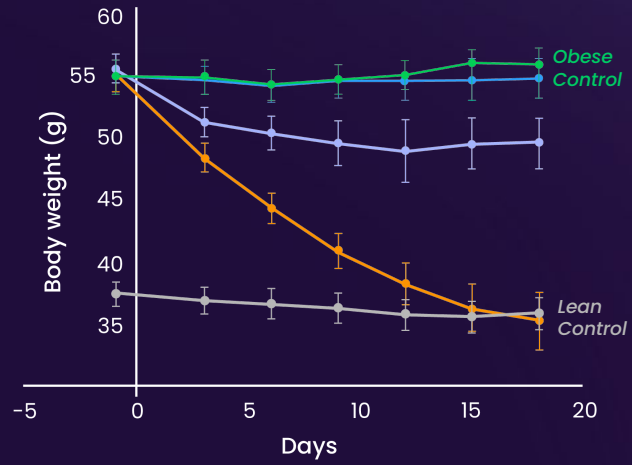
In preclinical obesity models, azelaprag more than doubled weight loss with incretins; similar results observed with other satiety mechanisms

Overall weight loss

Tirzepatide



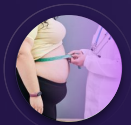
Semaglutide



Similar azelaprag exposures (AUC) achieved

DIO model
1.1g/L in water
(higher dose)

Ph1b bedrest
240mg IV QD



Ph2 obesity
300 mg QD &
BID

Lean Control — *Azelaprag 0.275g/L)* — *Tirzepatide (10nmol/kg)* — *Tirzepatide (10nmol/kg)+ Azelaprag (1.1g/L)* — *Semaglutide (30nmol/kg)*

Obese Control — *Azelaprag (1.1g/L)* — *Tirzepatide (10nmol/kg)+ Azelaprag (0.275g/L)* — *Semaglutide (30nmol/kg) + Azelaprag (1.1g/L)*

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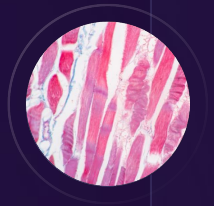
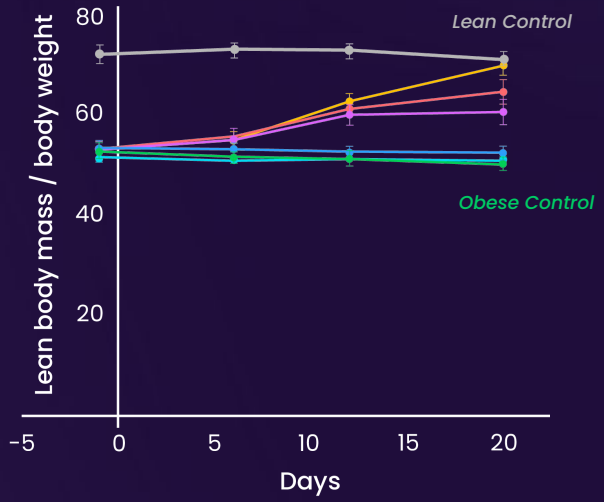
AZELAPRAG IN OBESITY: PRECLINICAL RESULTS

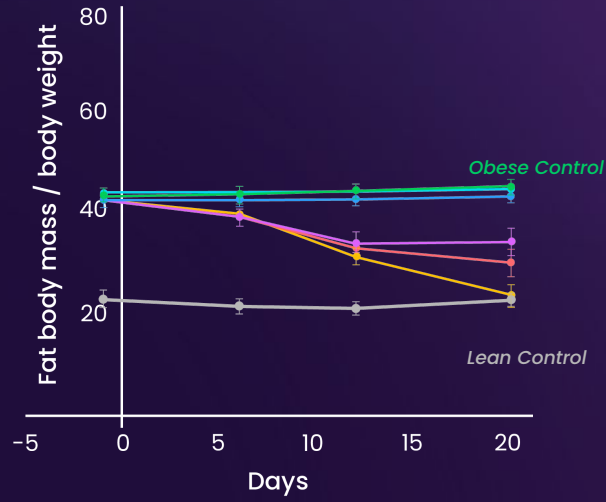
Addition of azelaprag to tirzepatide restored body composition to that of lean controls in a dose-dependent fashion

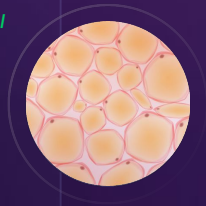


% Lean Mass



% 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.

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AZELAPRAG IN OBESITY

We intend to initiate 3 clinical trials with azelaprag

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AZELAPRAG IN OBESITY

Ongoing Ph2 STRIDES trial of azelaprag + tirzepatide in older obese patients

- 90% power for weight loss corresponding to an approvable difference
- 5mg dose of tirzepatide mimics the magnitude of current oral weight loss

STRIDES Azelaprag + tirzepatide Ph2 trial in obesity

Adults with obesity

- Age 55+
- BMI 30-40
- Exclude T2D/NASH
- GLP-1 naive

TZP 5mg SC QWK	Placebo PO QD	N=66
TZP 5mg	Azelaprag 300 mg QD*	N=66
TZP 5mg	Azelaprag 300 mg BID*	N=66
Placebo	Azelaprag 300 mg BID*	N=22

Primary endpoint

% change in overall weight loss

Exploratory endpoints

- Metabolic parameters (e.g., fasting glucose, HbA1c)
- PROs & QoL
- Body composition (DEXA)
  - % Lean mass
  - % Fat mass
  - Bone density
- Biomarkers
- Wearables (activity, sleep)

Power: ~90% power to detect a 3.3% improvement in weight loss over TZP monotherapy. Corresponds to >5% overall weight loss at 1 year.

*Note: double-blind and placebo-controlled; * oral bioavailability of 300 mg QD and BID doses previously evaluated in a Phase 1 PK trial*

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AZELAPRAG IN OBESITY

We are collaborating with Lilly on the Ph2 STRIDES trial

Lilly & Lilly Chorus are providing:

- Tirzepatide supply at no cost
- Therapeutic area expertise (e.g., regulatory, clinical science)
- Study execution (e.g., sites, vendors)

AZELAPRAG IN OBESITY

We also plan to initiate a second Ph2 trial with semaglutide; topline anticipated by end of 2026

Goal
Maximize program value & post-Ph2 optionality by demonstrating:

- An incretin class effect
- Efficacy in younger patients
- Full year of weight loss kinetics

Adults with obesity

- Age 18+
- BMI 30-40
- Exclude T2D/NASH
- GLP-1 naive

SEMA 2.4mg SC QWK	Placebo PO QD	N=100
SEMA 2.4mg	Azelaprag 300 mg QD	N=100
SEMA 2.4mg	Azelaprag 300 mg BID	N=100

Primary endpoint
Mean % change in body weight

Exploratory endpoints

- Metabolic parameters (e.g., fasting glucose, HbA1c)
- PROs & QoL
- Body composition (DEXA)
- Biomarkers
- Wearables (activity, sleep)

Power: ~90% power to detect a 5% improvement (approvable difference) in weight loss over semaglutide alone

Note: double-blind and placebo-controlled

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AZELAPRAG IN OBESITY: DEVELOPMENT PATHWAYS

Base case: We are targeting a class label as an incretin add-on therapy

Incretin-agnostic add-on therapy

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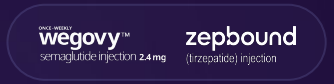
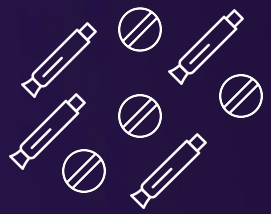




- **Target profile:** oral product that can be added to *any* incretin to increase weight loss and improve tolerability & body composition
- **Flexible potential use:** could be combined with oral and injectable incretins
- **Key addressable patients:** high BMI; poor incretin response (efficacy, tolerability)

Ph3 development

Financeable from follow-on post Ph2 data

Approvable efficacy
+5% overall weight loss at 1 year

Database required for approval
placebo-controlled trials with 3K patients on active treatment

Potential trial designs to support incretin-agnostic label:

1. Azelaprag + tirzepatide
   vs. tirzepatide monotherapy
2. Azelaprag + semaglutide
   vs. semaglutide monotherapy

AZELAPRAG IN OBESITY: DEVELOPMENT PATHWAYS

There is an additional attractive path as part of an all-oral fixed-dose combination

Oral fixed-dose combination
(likely through pharma partnership)

- **Target profile:** potential best-in-class oral weight loss in a single product
- **Key addressable patients:** Broad - 77% who prefer orals
- **Best positioned partners:** pharma with an oral *small molecule* incretin or appetite-suppressing agent
- **Commercial advantages:** LOE extension given new formulation IP

Ph3 development

Approvable efficacy & database required for approval are the same between development paths

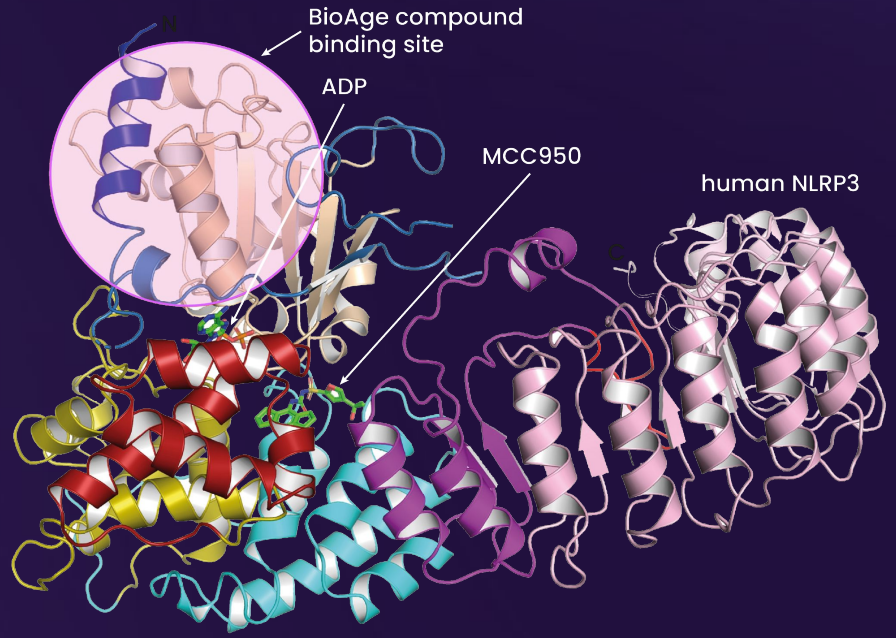
Source: Boye 2020

NLRP3

Neuroinflammation

NLRP3: NOVEL BINDING SITE

We have discovered structurally and mechanistically novel NLRP3 inhibitors bind in distinct region



Collaborator profile

Matthias Geyer, PhD

Director, Institute of Structural Biology University of Bonn

Expert in NLRP3 structural biology

Identified the structure of NLRP3 decamer bound to MCC950

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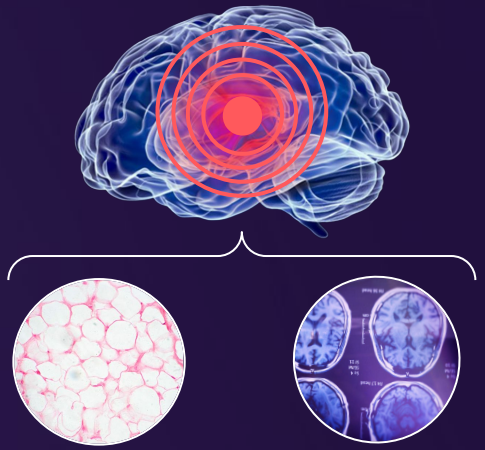
NLRP3: KEY ATTRIBUTES

We are developing NLRP3 inhibitors well positioned to address the neuroinflammation that drives metabolic and neuro diseases

Neuroinflammation is linked to both metabolic disease and neurodegeneration



Metabolic disease
(obesity, diabetes)

Neuro-degeneration
(e.g., AD, PD)

Key attributes

First patent granted July 18, 2023

Potential best-in-class potency
Low single-digit nanomolar IC_{90} in human microglia

Selective, reversible inhibition of NLRP3

Ph1 SAD / MAD goals

Human potency & duration of effect

CNS penetration

CNS penetrant
Brain / plasma ratio up to ~1 & high free fraction

Strong in vitro safety profile

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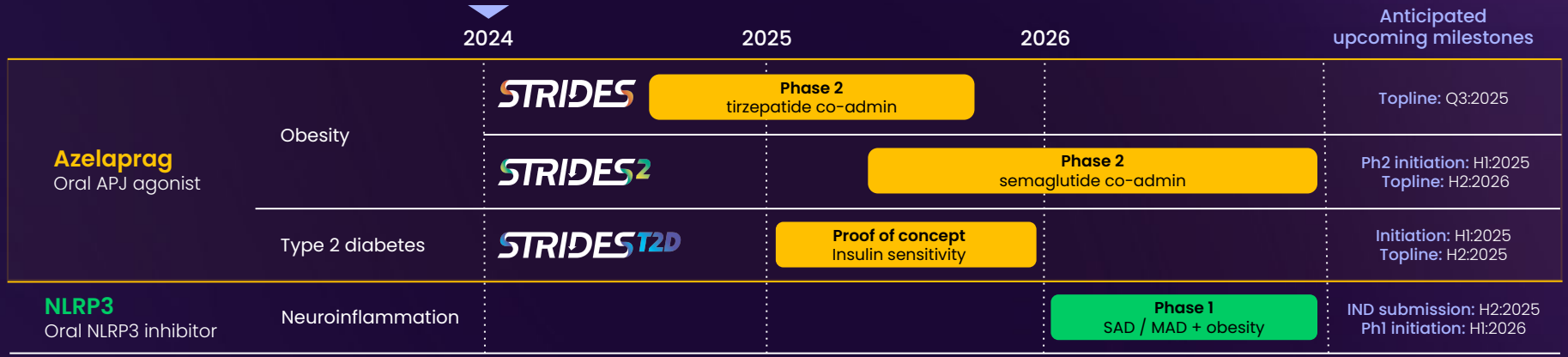
Highlights & key milestones

Azelaprag, an oral exercise mimetic for obesity: potential for improved weight loss, tolerability, and body composition

1. **Amgen asset with meaningful clinical experience**
   Well tolerated in 265 subjects across eight Ph1 studies; metabolic benefits consistent with exercise in Ph1b
2. **Established APJ biology & genetics highlight promise of azelaprag in obesity and metabolic indications**
   2x overall weight loss with incretins in preclinical studies
3. **Highly differentiated opportunity to create an all-oral weight loss regimen**
   Well tolerated, complementary oral mechanism that enhances weight loss with oral GLP-1

NLRP3: highly potent, CNS penetrant, oral compounds for neuroinflammation; emerging promise in obesity



Platform: we are currently building molecules against 2 novel targets

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