

BioVie Day Presentation

March 23, 2023

Forward-looking statements

This document contains forward-looking statements made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995 that involve risks, uncertainties and assumptions that could cause BioVie's actual results and experience to differ materially from anticipated results and expectations expressed in these forward-looking statements. BioVie has in some cases identified forward-looking statements by using words such as "anticipates," "believes," "hopes," "estimates," "looks," "expects," "plans," "intends," "goal," "potential," "may," "suggest," and similar expressions. Among other factors that could cause actual results to differ materially from those expressed in forward-looking statements are: BioVie's need for, and the availability of, substantial capital in the future to fund its operations and research and development. Other risks are that BioVie's compounds may not successfully complete pre-clinical or clinical testing or be granted regulatory approval to be sold and marketed in the United States or elsewhere. BioVie cannot guarantee the effectiveness of its patents or Orphan Drug designations. A more complete description of these risk factors is included in BioVie's filings with the Securities and Exchange Commission. In addition to the risks described above and in BioVie's filings with the Securities and Exchange Commission, other unknown or unpredictable factors also could affect BioVie's results. No forward-looking statements can be guaranteed and actual results may differ materially from such statements. You should not place undue reliance on any forward-looking statements. BioVie undertakes no obligation to release publicly the results of any revisions to any such forward-looking statements that may be made to reflect events or circumstances after the date that these slides are posted to BioVie's website or to reflect the occurrence of unanticipated events, except as required by applicable law or regulation.

Agenda

- Overview
- Chronic Low-grade Inflammation Science
- Development programs
 - Alzheimer's Disease
 - Parkinson's Disease
 - Epigenetics & Longevity
 - Liver Disease
- Clinical & Regulatory Strategy
- Commercialization Roadmap
- Social Impact
- Recap

Overview

Executive Summary

- The company is in the early stages of multiple late-stage data readouts for multiple indications addressing great unmet medical needs
- Early results in Alzheimer's Disease (AD), Parkinson's Disease (PD) and Ascites have all been encouraging, and our KOLs/SAB have encouraged us to move more assertively
- Potentially pivotal registrational trials are underway or will begin in 2023 for all indications
- Topline results from our Phase 3 AD trial expected to be announced October 2023. Regulatory submissions expected in 2024 and 2025
- We believe our portfolio, if approved, will create a platform of great benefit to patients and economic value to shareholders
- All development and commercialization options (including partnering) being considered
- Our support of Social Impact Partners allows us to bring better patient-centered support to affected individuals/families, of which our future medication will be an important part

Our journey thus far

- BioVie started with a single program in refractory ascites with the backdrop that terlipressin (or any treatment) has never been approved in the US
- Acquisition of the Neurmedix assets (including NE3107) expanded our horizons
 - The team that discovered NE3107 sought to develop a new therapy for diabetes
 - Their understanding of systems biology and metabolic syndromes led to the insights on NE3107's unique MOA and value in areas beyond diabetes, including AD and PD
- We took a more assertive approach than big-pharma
 - Proceeded straight to a potentially pivotal Phase 3 in mild- to moderate-AD as our first trial in AD
 - Supported exploratory biomarker Phase 2 started afterwards that associated NE3107 with reversed cognitive decline and reduced p-Tau in MCI and mild-AD
 - Conducted larger PD DDI study than required to seek an efficacy signal in humans, which revealed impact on greater motor control and morning “on time”
- Strong data and counsel from KOLs/SAB leading us to be more expansive with our aspirations than originally plan

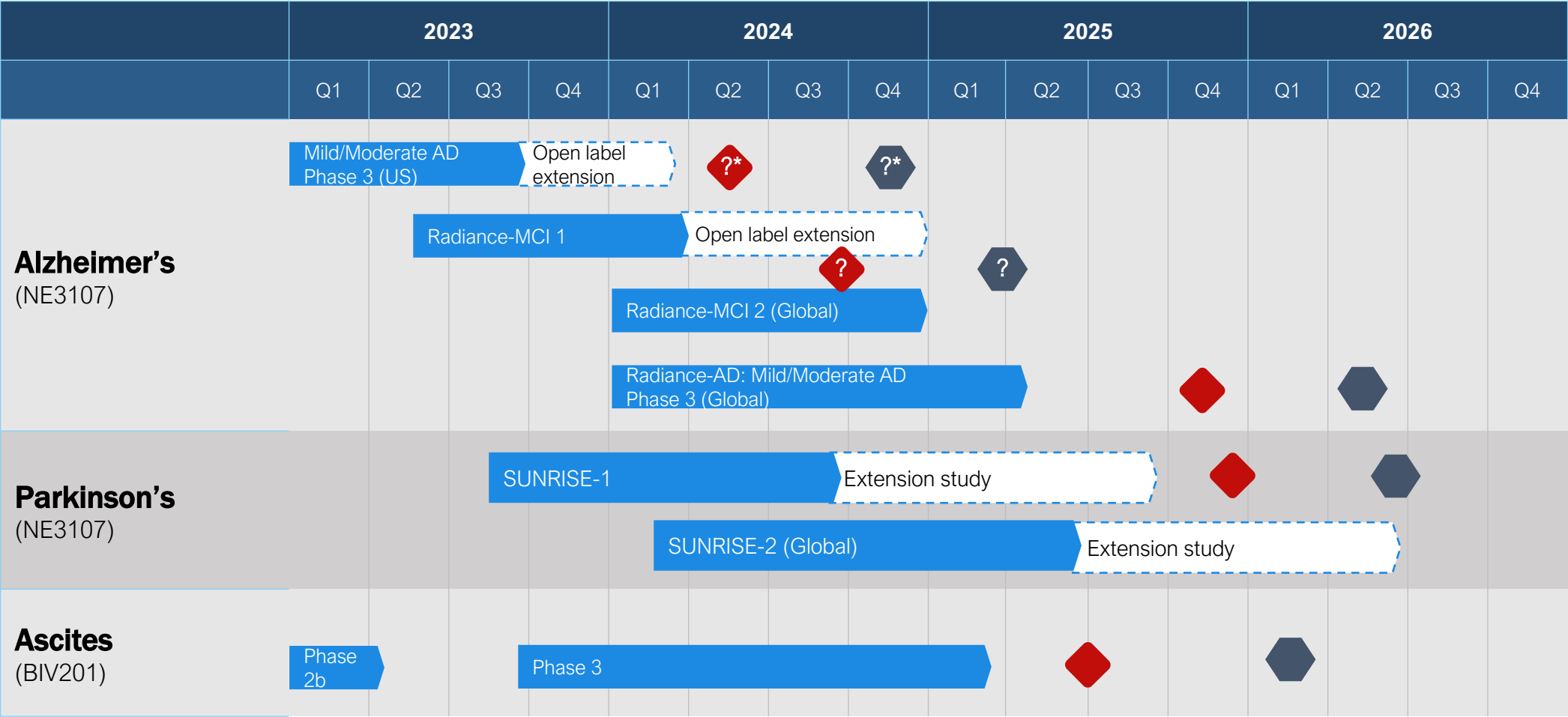
Our journey ahead



File NDA



Launch



* Two Phase 3 trials are usually required for registration. However, the FDA has allowed filing based on strong data from a single pivotal trial in indications with few therapeutic options.

Chronic Low-grade Inflammation Science

Chronic low-grade inflammation

- Inflammation is the result of the body's natural protective processes

Insults

- Pathogens
- Trauma
- Ischemia
- Stress
- Radiation
- Obesity
- Cancer

Responders

- Blood proteins
- Platelets
- Granulocytes
- Monocytes
- Macrophages
- Microglia
- Endothelial cells

Master Alarms

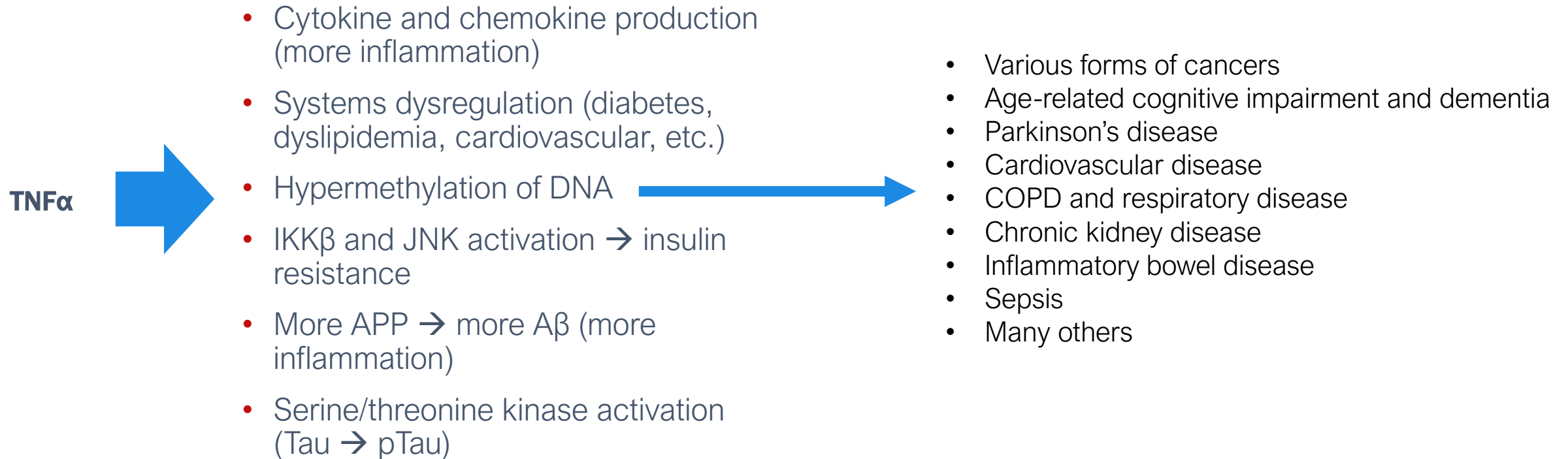
- NFκB transcription factors
- Oxidative stress
- Reactive oxygen
- Reactive nitrogen

Pro-inflammatory factors

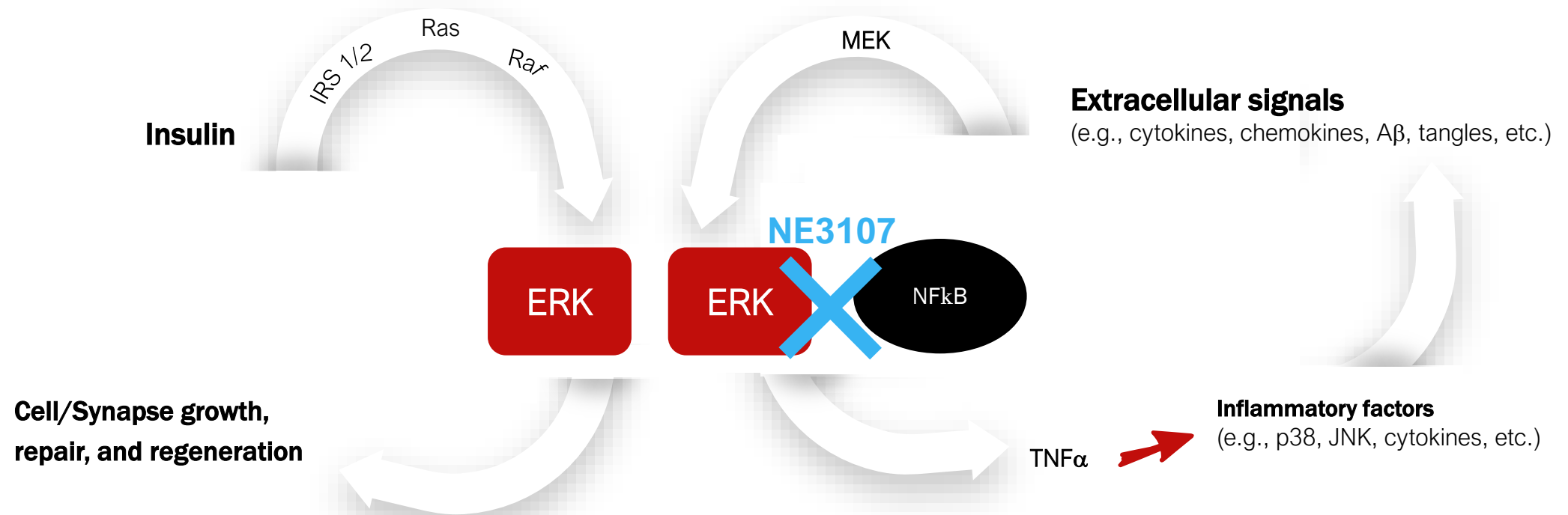
- Cytokines
- Chemokines
- Receptors
- Complement factors
- Metalloproteinases
- Cell adhesion molecules

- If insults and pro-inflammatory factors eliminated, pathology addressed
- If not, low grade chronic inflammation and associated insulin resistance ensues
- Inflammation and insulin resistance go hand-in-hand

TNF α -mediated inflammation leads to many downstream consequences



NE3107 blocks inflammatory (but not homeostatic) ERK and NFkB, which play pivotal roles in AD pathogenesis

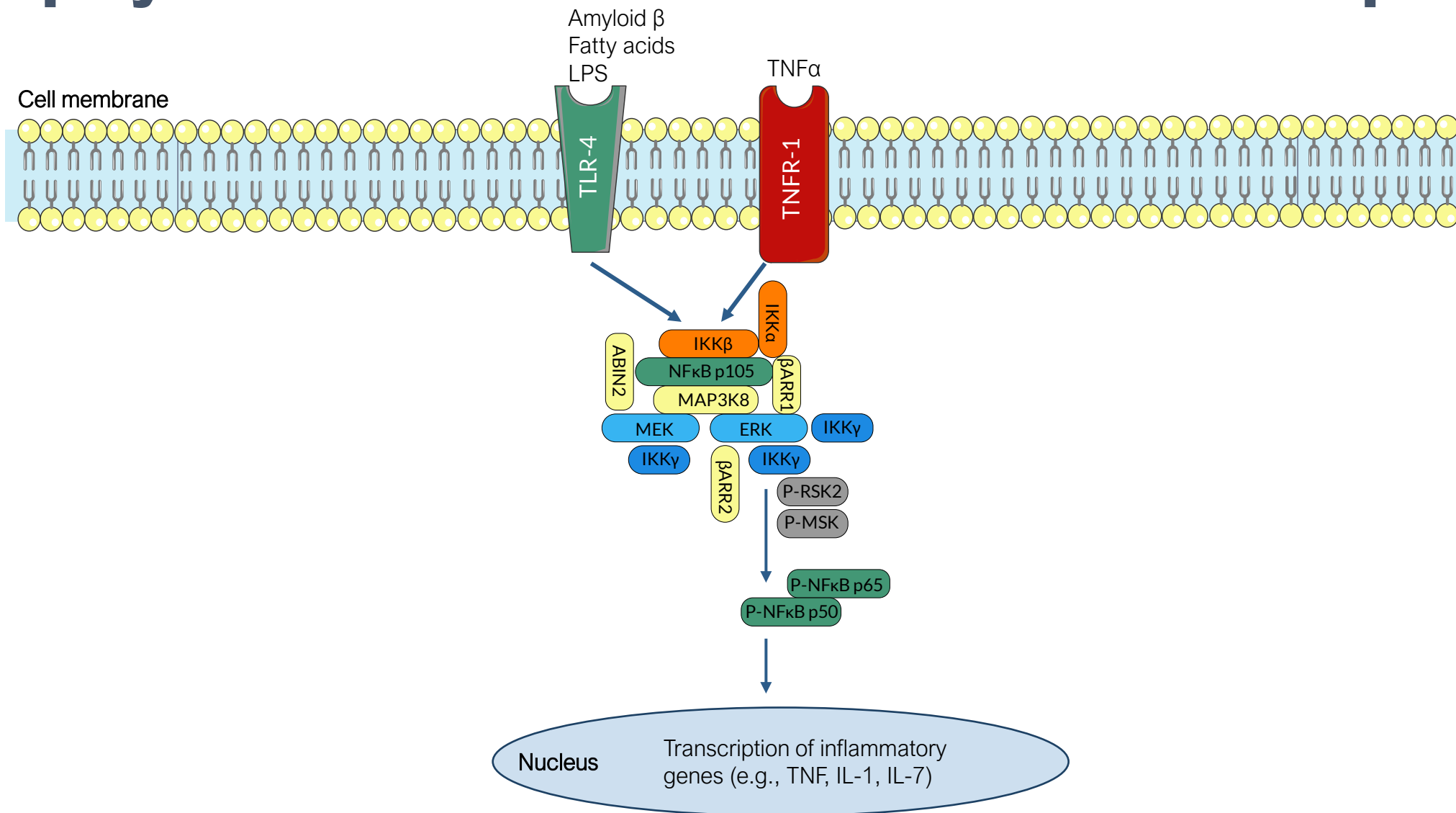


Activation of Inflammatory Signaling

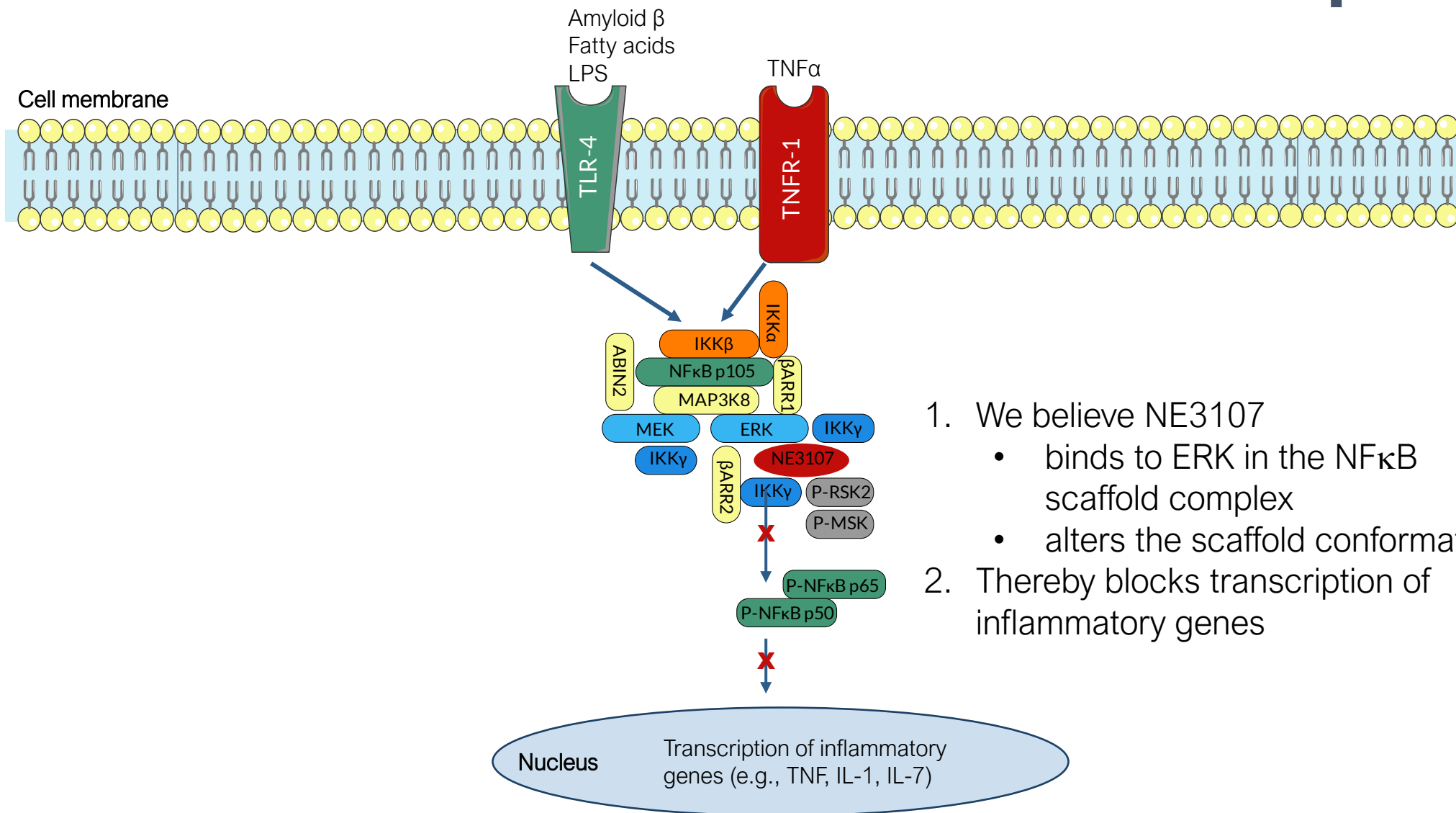
Inflammatory signals (Amyloid beta, TNF, LPS, fatty acids, others) interact with toll-like receptor 4 (TLR4) and with TNF Receptor 1 (TNFR1) leading to:

- 01 Activation of I kappa B kinase beta (IKK β)
- 02 Phosphorylation of NF κ B p105, which is degraded to p50
- 03 Release of MAP3K8 phosphorylation of MEK. ERK, RSK, MSK, NF κ B3 (RelA p65)
- 04 Nuclear localization of p50 and phospho-p65
- 05 Transcription of kappa B (κ B) **promoter** sites for inflammatory genes (e.g., TNF, IL-1, IL-6)
- 06 Translation, maturation and secretion of inflammatory mediators (e.g., TNF)
- 07 TNF binding to TNFR1
- 08 MAP3K8-dependent ERK phosphorylation of TNFR1
- 09 Internalization of the TNF/TNFR1 complex leading to further inflammatory signaling

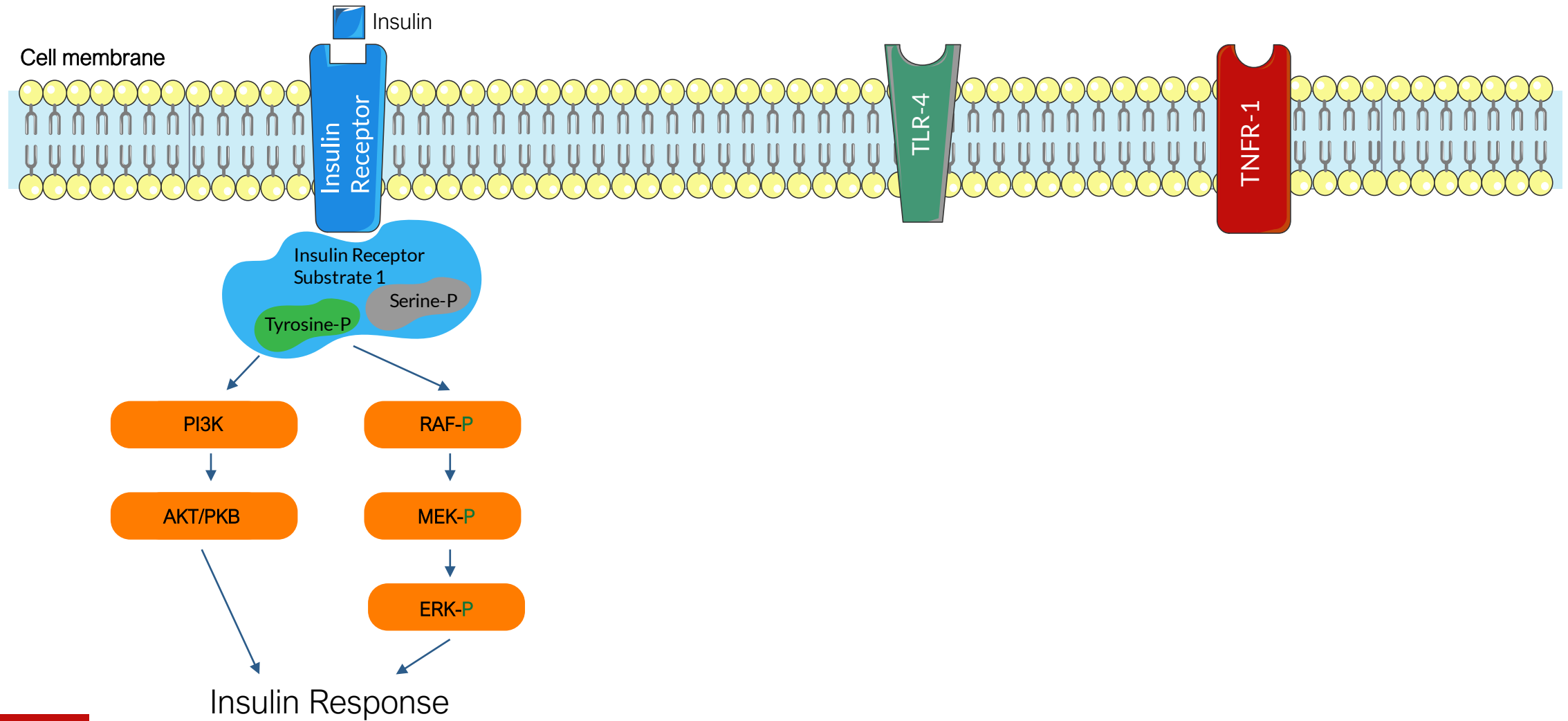
ERK plays a critical role in the NF κ B scaffold complex



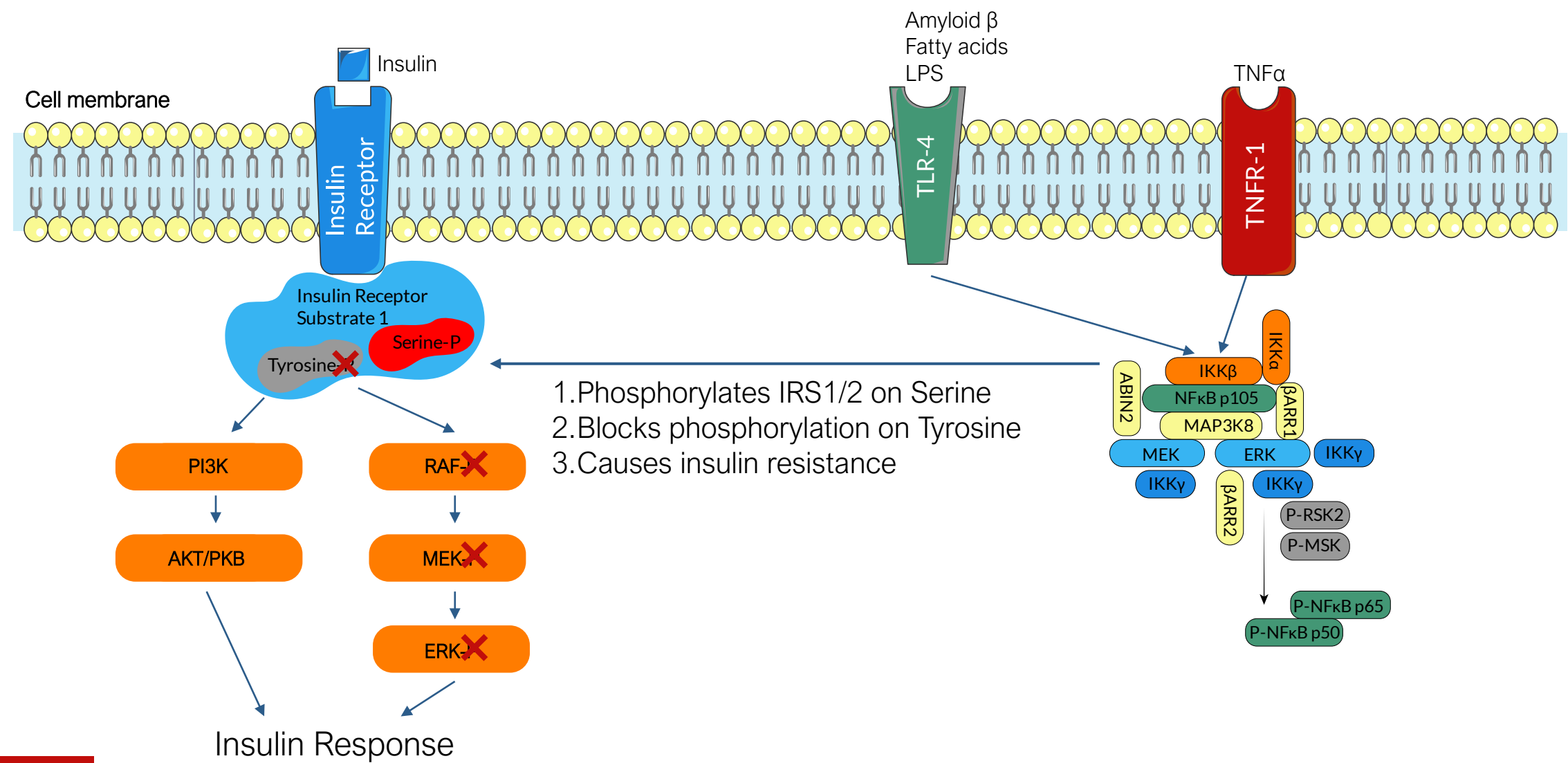
We believe NE3107 alters the NF κ B scaffold complex



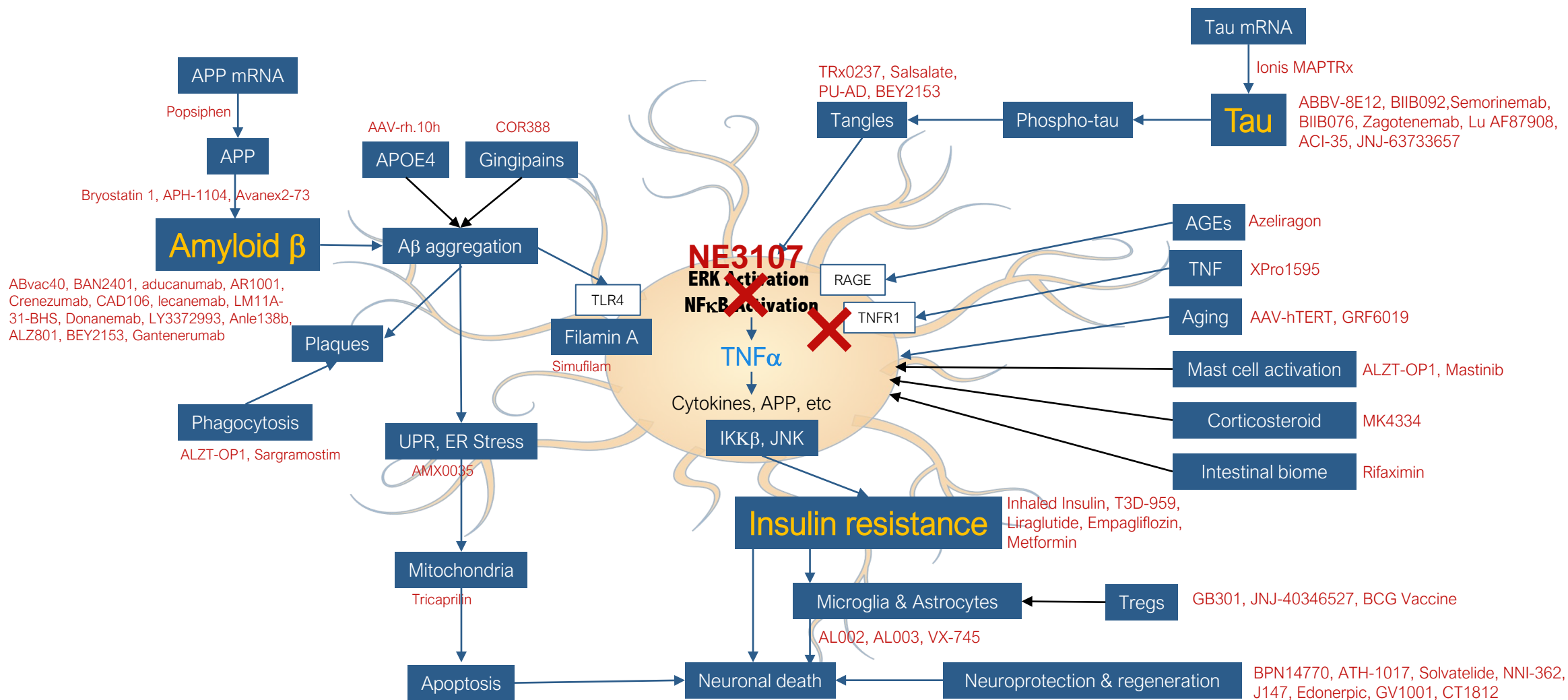
Normal insulin signaling



Inflammation-mediated insulin resistance



NE3107 believed to modulates inflammation at the central hub, thereby reducing downstream inflammatory cascade



Expanding beyond NE3107

- HE3291 is a sister molecule with similar activity profile as NE3107
- We are resuming discovery research to explore additional molecules and indications
 - We now have a much better understanding of NE3107's presumed MOA
 - Enables even more precise medicinal chemistry to target binding sites
- Focus areas will be communicated in due course

Alzheimer's Disease

Alzheimer's Disease program overview

- Results from NE3107 Phase 2 exploratory biomarker trial in Mild Cognitive Impairment (MCI)/mild-AD reported at the CTAD Annual Conference in December 2022
 - First data set showing impact on cognition, p-tau, and imaging biomarkers
 - Impact on biological aging created new program on epigenetics & longevity
- NM101 Phase 3 in mild- to moderate-AD fully enrolled
 - Last patient visit expected in September 2023. Topline data readout expected October 2023
- Future clinical trials
 - Radiance-MCI 1 being launched by June 2023 to focus on US registration
 - Follow-on trials for global registrations in MCI and mild/moderate AD targeted for early 2024

NE3107 Phase 2 exploratory biomarker trial

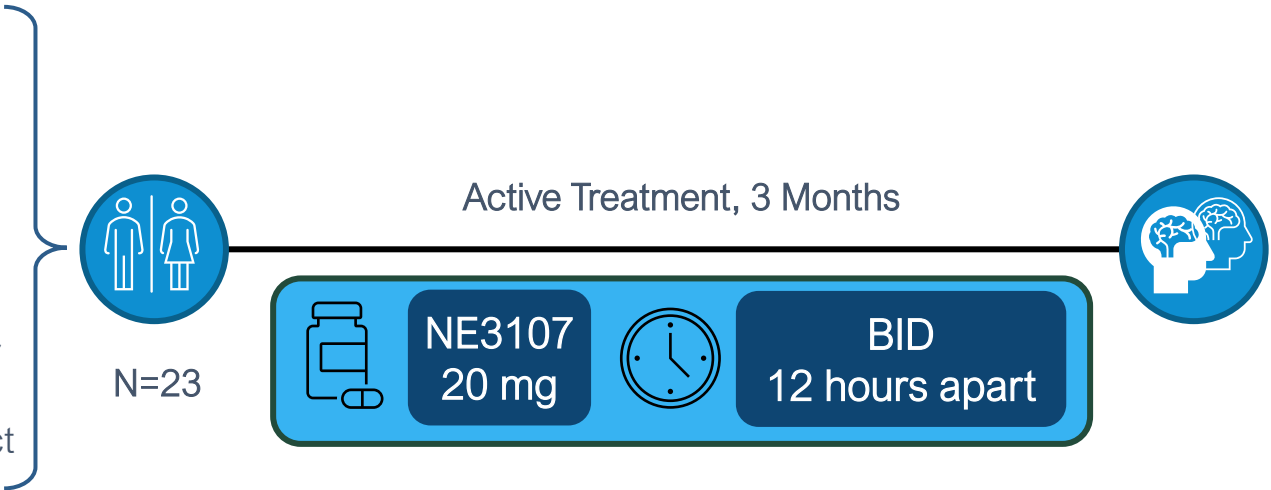
Open-Label Single-Arm 3-Month Study

Inclusion criteria

- 50-89 years old
- Cognitive decline due to dementia
- QDRS score: 1.5-12.5
- CDR score: 0.5 (MCI) or 1 (mild dementia)

Exclusion criteria

- Prior imaging inconsistent with AD
- History of stroke that resulted in cognitive or motor deficits
- MRI/CT evidence of moderate/large cerebral infract



**Change from
baseline
assessments**

Neuroimaging	Clinical Assessments	Biomarker Assessments
MRS Relative CBF Anatomical imaging BOLD imaging* DTI-NODDI†	MMSE ADAS-Cog12 MoCA QDRS and CDR ADCOMS	Plasma TNF-α CSF Aβ42 CSF p-tau P-tau:Aβ42 ratio Brain glutathione (by MRS)

*Preliminary data; †Data analysis ongoing.
ADAS-Cog, Alzheimer's Disease Assessment Scale-Cognitive Subscale; ADCOMS, AD Composite Score; ASL, arterial spin labeling; BID, twice per day; BOLD, blood-oxygen level dependent; CBR, cerebral blood flow; CDR, Clinical Dementia Rating; CSF, cerebrospinal fluid; DTI-NODDI, diffusion tensor imaging – neurite orientation dispersion and density imaging; MMSE, Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment; MRS, magnetic resonance spectroscopy; NMR, nuclear magnetic resonance; QDRS, Quick Dementia Rating Scale.

Clinical outcomes assessments

NE3107 Was Associated With Improvements From Baseline in Clinical Outcomes of Neuropsychological and Cognitive Assessments after 3 months of Treatment

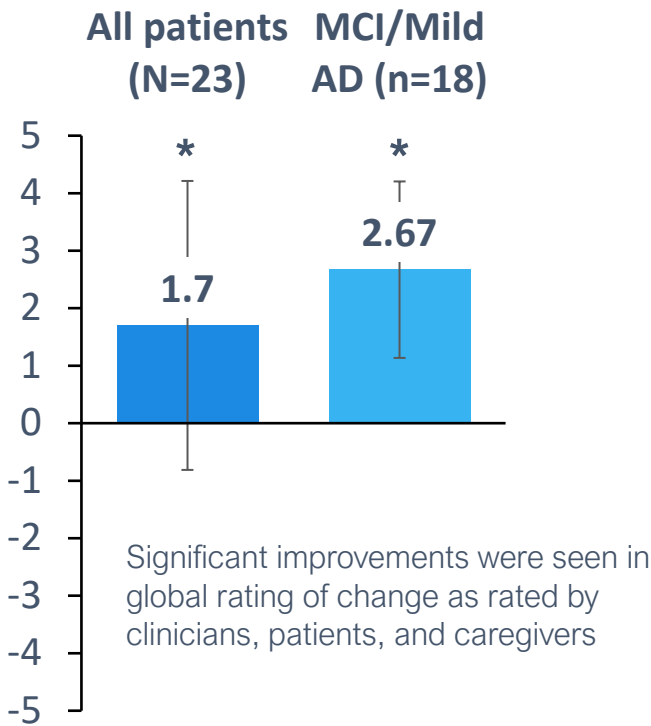
Changes from Baseline in Clinical Assessments

Assessment	All Patients (N=23)	MCI/Mild AD (n=18)
ADAS-Cog12	-0.913	-2.167*
MMSE	-0.74	-0.39
MoCA	-0.04	0.56
QDRS	-0.54	-1.56*
CDR	0.04	-0.11*
ADCOMS	0.0049	-0.07*

In the patients with baseline MMSE ≥20 (indicating MCI or mild dementia), NE3107 was associated with statistically significantly improved cognitive functioning vs baseline, in ADAS-Cog12, QDRS, CDR, ADCOMS,

Green=Improvement

Clinician-Rated Global Rating of Change



Biomarker improvements

Changes from baseline in biomarkers

Assessment	All Patients (N=23)	MCI/Mild AD (n=18)
TNF- α , pg/mL	-0.452	-0.563
CSF p-tau, pg/mL	-1.1	-1.66*
P-tau:A β 42 ratio	-0.0033	-0.0024*

In the MMSE \geq 20 patients, NE3107 was associated with statistically significant improvements in p-tau and the p-tau:A β 42 ratio

Green=Improvement

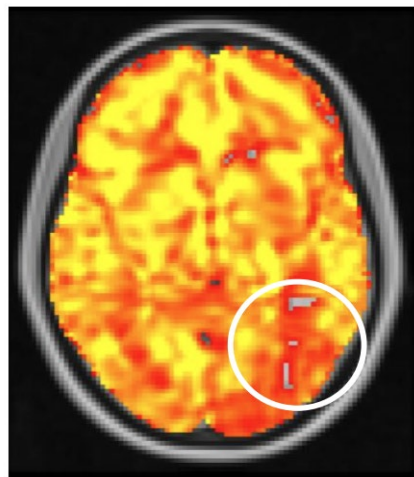
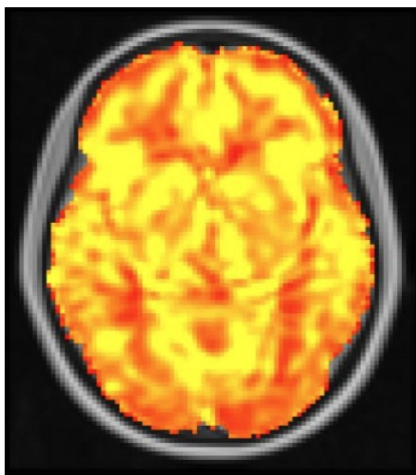
Correlations between biomarkers and clinical outcomes

	All Patients	MMSE \geq 20 n=18
ADAS-Cog12		
TNF- α	r=0.46	r=0.59*
ADCOMS		
A β 42	r=0.53*	r=0.31
P-tau	r=0.49*	r=0.37

NE3107 was associated with statistically significant correlations between several clinical assessments and biomarkers

Neuroimaging methodology

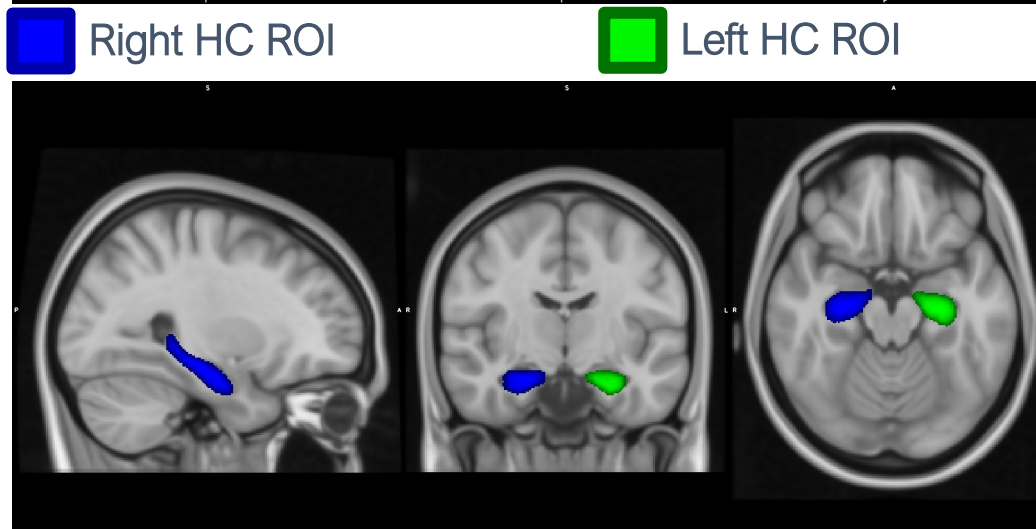
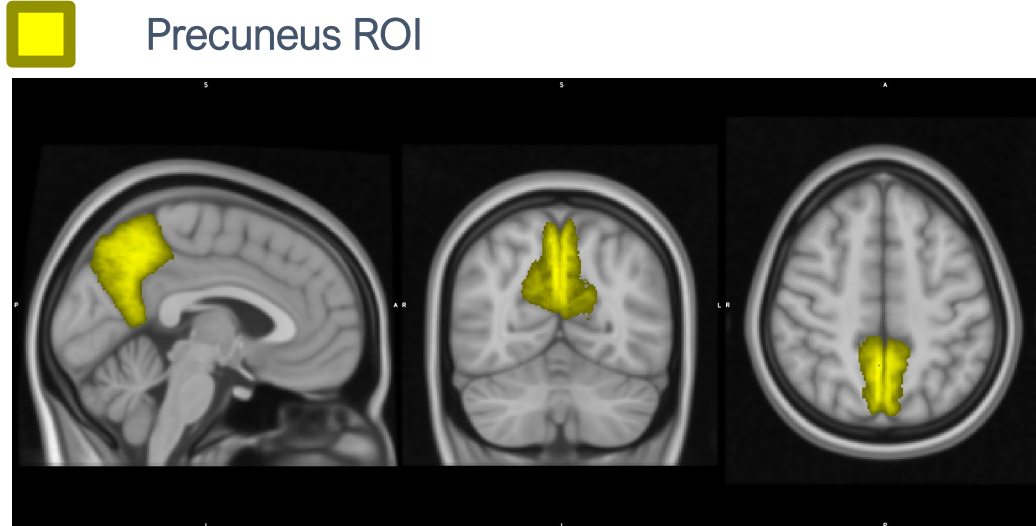
- Functional MRI data were submitted to blinded review and scoring by two independent clinician-readers. Inter-rater reliability was 96%; in the event of disagreement between raters, data were subjected to additional review and ultimately scored by consensus
- Criteria for “Improved,” “Stable,” or “Declined”
 - Improved: No longer meets criteria for abnormality
 - Declined: Previously ‘normal’ scan met criteria for abnormality at follow-up OR ▪ Abnormality becomes more extensive in an abnormal patient
 - Stable: No significant change between baseline and follow-up scans



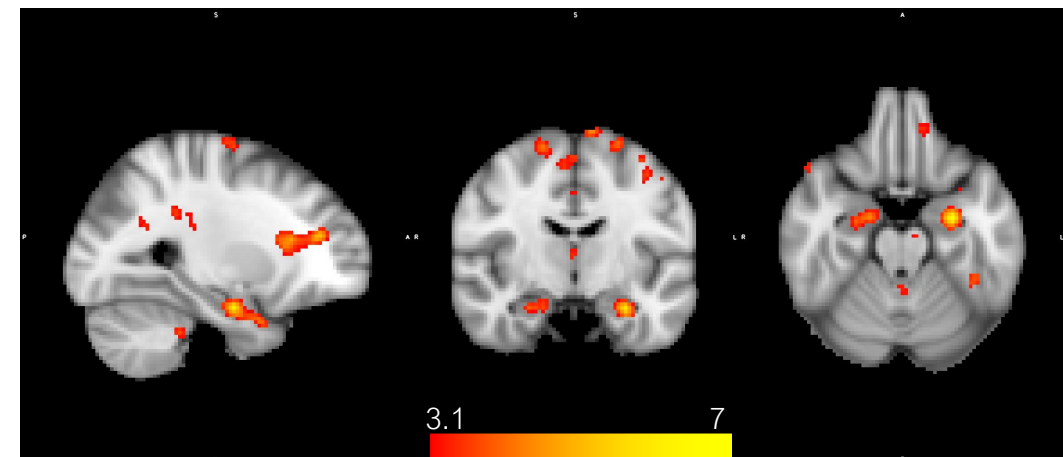
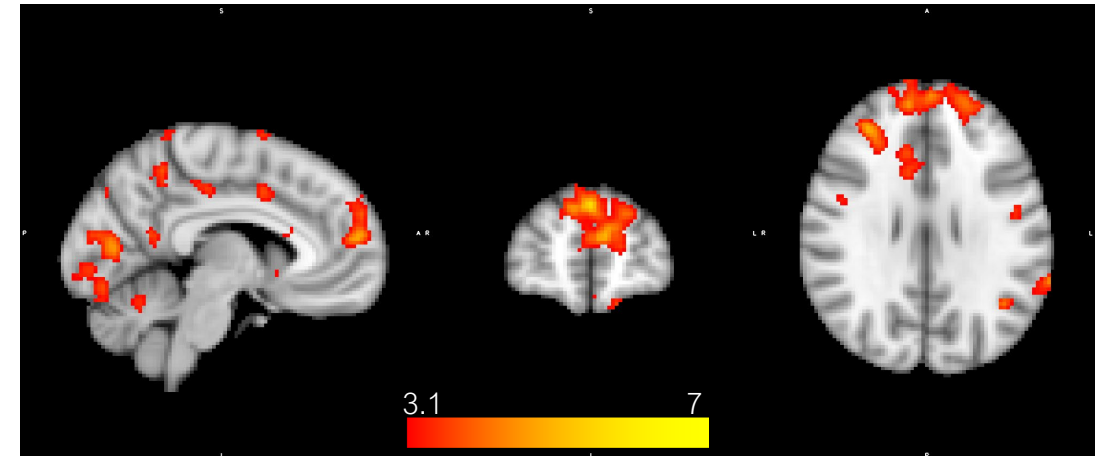
Criteria for Abnormality: Perfusion / Relative cerebral blood flow (CBF): 30% decrease in signal intensity in the temporal, parietal, or occipital lobes (in at least one hemisphere) was scored as abnormal.

Examples shows a subject that had decreased perfusion (abnormal) that improved at follow-up

NE3107 increased functional connectivity from baseline in group analysis using BOLD imaging

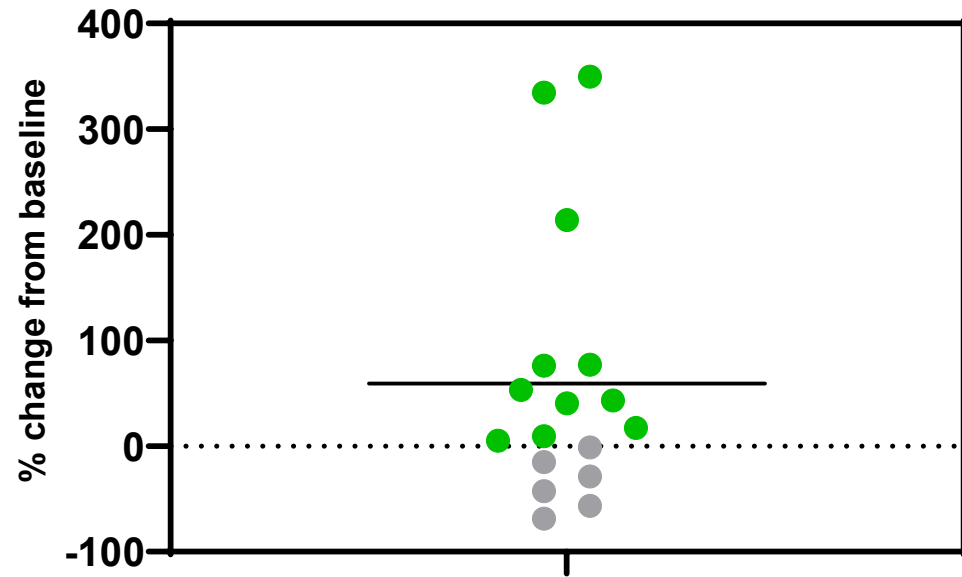


Statistically significant regions of increased connectivity

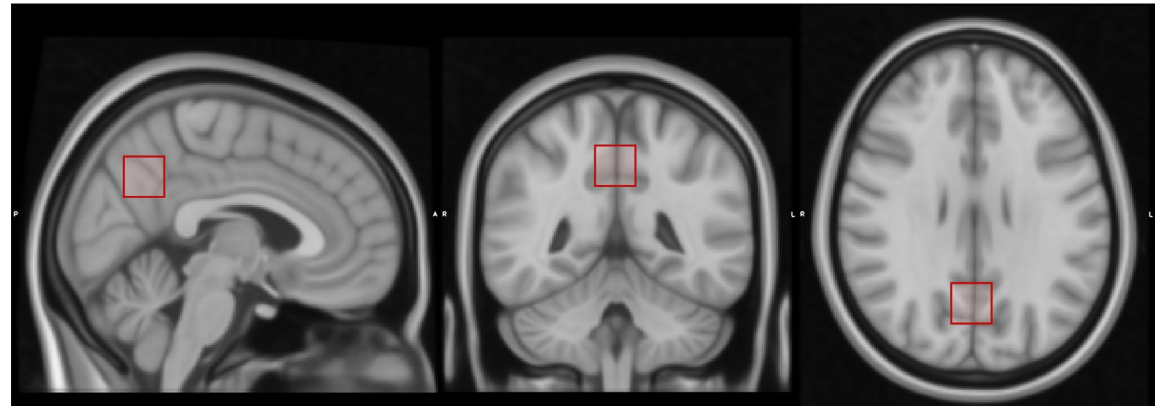


NE3107 associated with reduced oxidative stress in the brain

Brain glutathione assessed by MRS of Precuneus



11/17 (59%) improved
mean +59% change
P=0.069



Significant correlation of Glutathione with:
TNF- α ($r=-0.44$)
ADAS-Cog12 ($r=-0.45$)

Update on NM101 Phase 3 in Mild/Moderate

- Trial fully enrolled as of February 28, 2023
 - 100 patients completed treatment at that time
 - Top-line data readout expected in October 2023
- Trial continues to have a good safety profile and low discontinuation rate
- Blinded baseline data shows evidence of metabolic inflammation in amyloid β positive and negative, and APOE ϵ 4 positive and negative subjects submitted for presentation at the American Diabetes Association's 83rd Scientific Sessions in San Diego, June 23-26, 2023

Radiance-MCI 1 Trial

- A potentially pivotal Phase 2/3, double-blind, randomized, placebo-controlled, parallel group, multicenter study of NE3107 in MCI subjects
 - MCI patients have a measurable loss of memory, but have not yet shown significant functional impairment
 - MCI is defined by Stage 2 and Stage 3 diagnostic criteria in Draft Guidance for Industry- Early AD: Developing Drugs for Treatment (February 2018)
 - Most patients with MCI continue to degenerate to AD. Inflammation drives both conditions
 - Arresting/reversing at the MCI stage (CDR less than 0.5) has the potential for the greatest long term patient benefit
- Patients 50-80 years old qualify with computer-based tests of cognitive impairment
- Patients will receive 20 mg NE3107 or placebo for 6 months for CDR-SB primary endpoint
 - Patients randomized to NE3107 will continue receiving drug for 1 year to observe effect on disease progression
 - Patients randomized to placebo at the start crosses over to NE3107 for 6 months (delayed start)
- Anticipate first patient enrollment by June 2023
- Trial approach provides maximum flexibility to work with FDA for approval
 - Single MCI trial in combination with mild-/moderate-AD Phase 3 for full approval in MCI in the US
 - A second MCI trial to be started in 2024 for as second trial for full approval in MCI globally

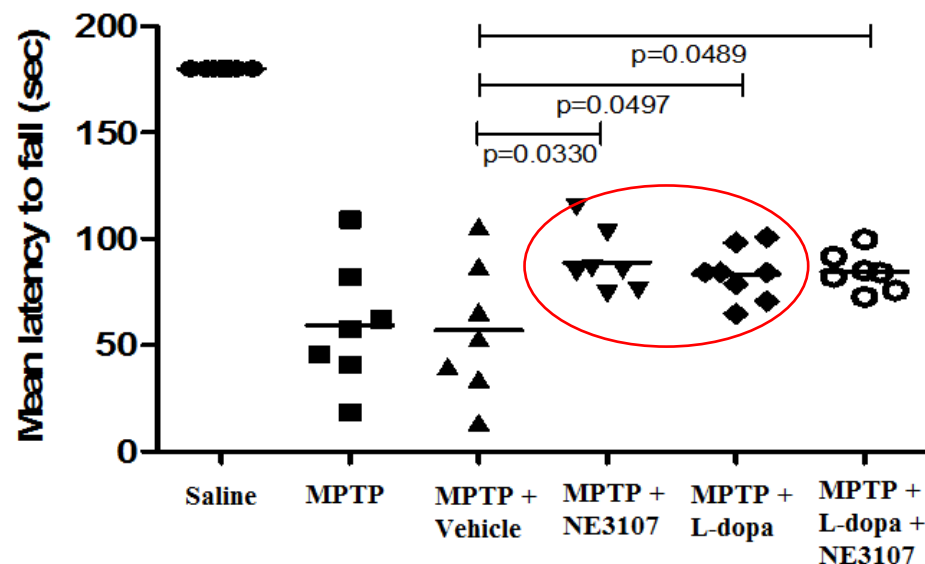
Parkinson's Disease

Parkinson's Disease program overview

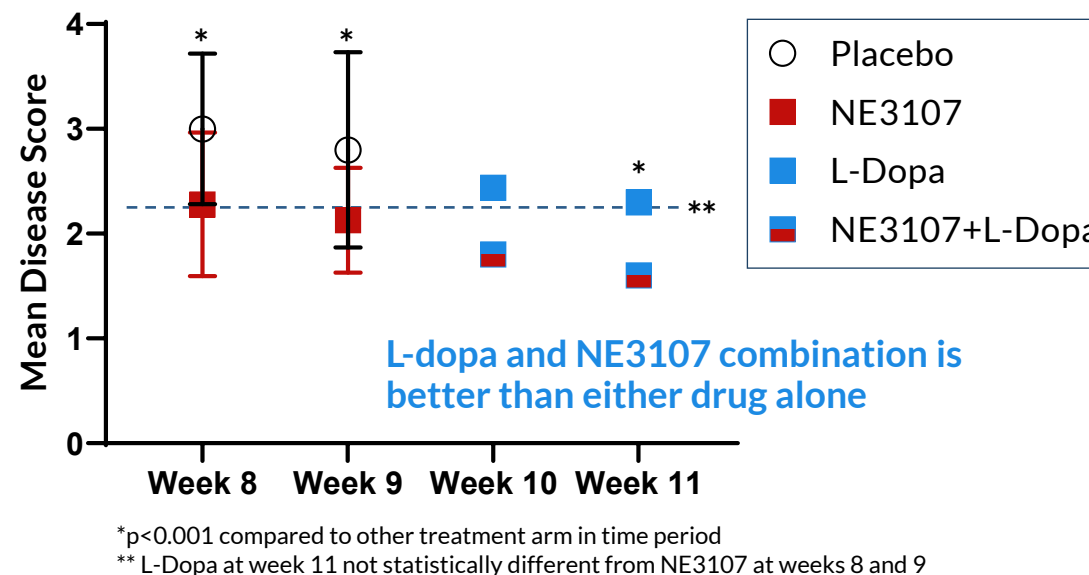
- Clinical development was initiated based on strong data from preclinical studies
- Phase 2 NM201 trial launched with 2 main objectives
 - Primary: safety and drug-drug-interaction study (as requested by FDA) to demonstrate the absence of adverse interactions with levodopa
 - Secondary: detect efficacy signal in humans
 - Small study not powered for statistical significance
- NM201 objectives were both met
 - 3+ points greater motor control vs. placebo on the UPDRS Part 3 score. 6+ point among <70 years old
 - Statistically significant fraction of patients experienced morning “on” state with levodopa withheld overnight and prior to receiving their morning medication
- Clinical studies in levodopa naïve patients being launched
 - SUNRISE-1 is a potentially pivotal Phase 2b starting 3Q2023
 - SUNRISE-2 is a potentially pivotal Phase 3 starting late 1Q2024

NE3107 has similar promotoric activity to L-dopa in rodent* and marmoset* models

MPTP Mouse



MPTP Marmoset treated at Week 8



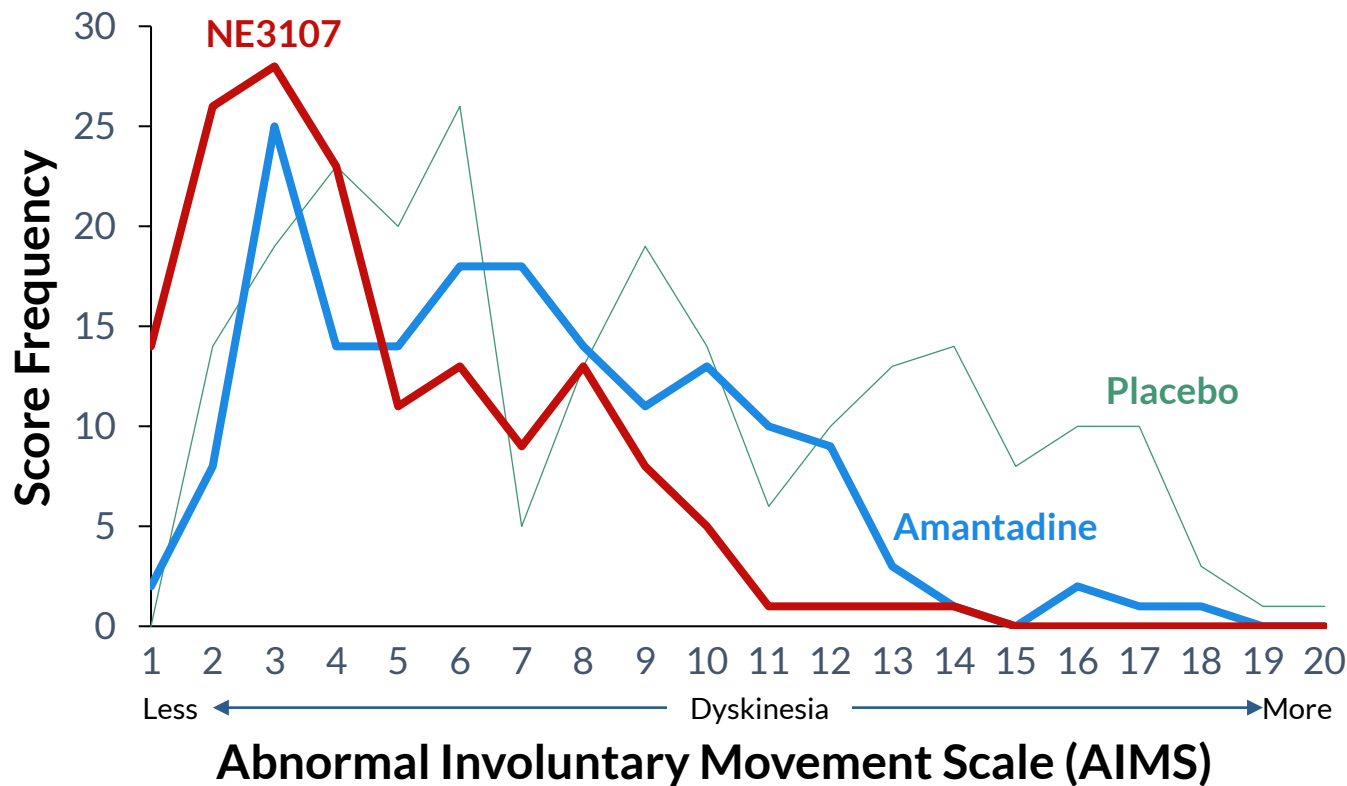
NE3107's promotoric effects observed within 4 days of treatment

NE3107 decreases L-Dopa-induced dyskinesia (LID) in marmosets*

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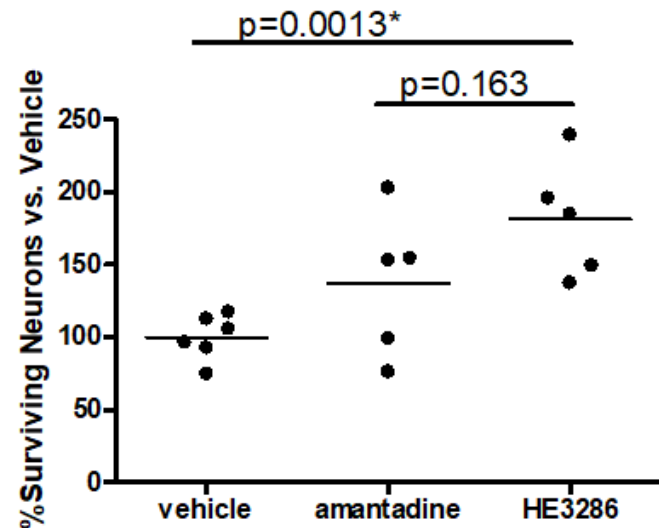
Distribution of AIMS Scores



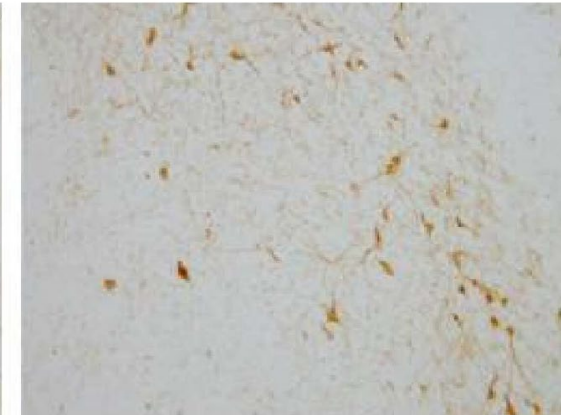
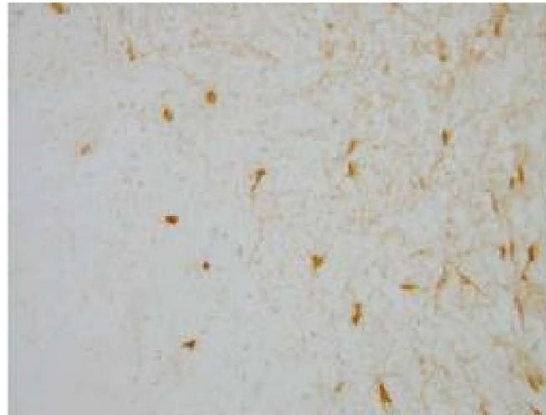
NE3107 preserved ~2X TH+ (dopaminergic) neurons in MPTP marmosets*

Funded by

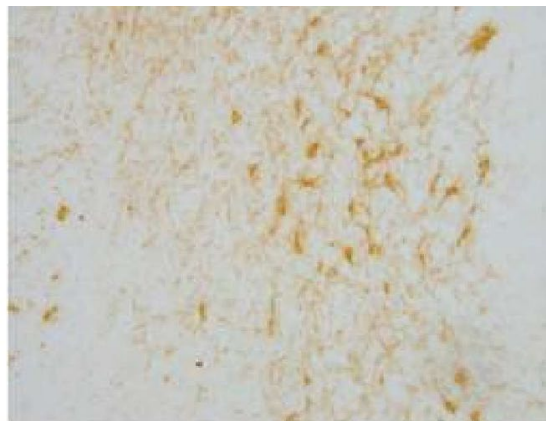
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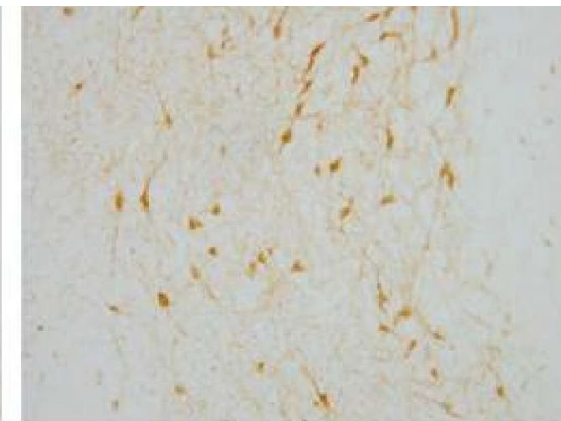
Control moneys (M09100 and M11008)



HE3286 (M11007)



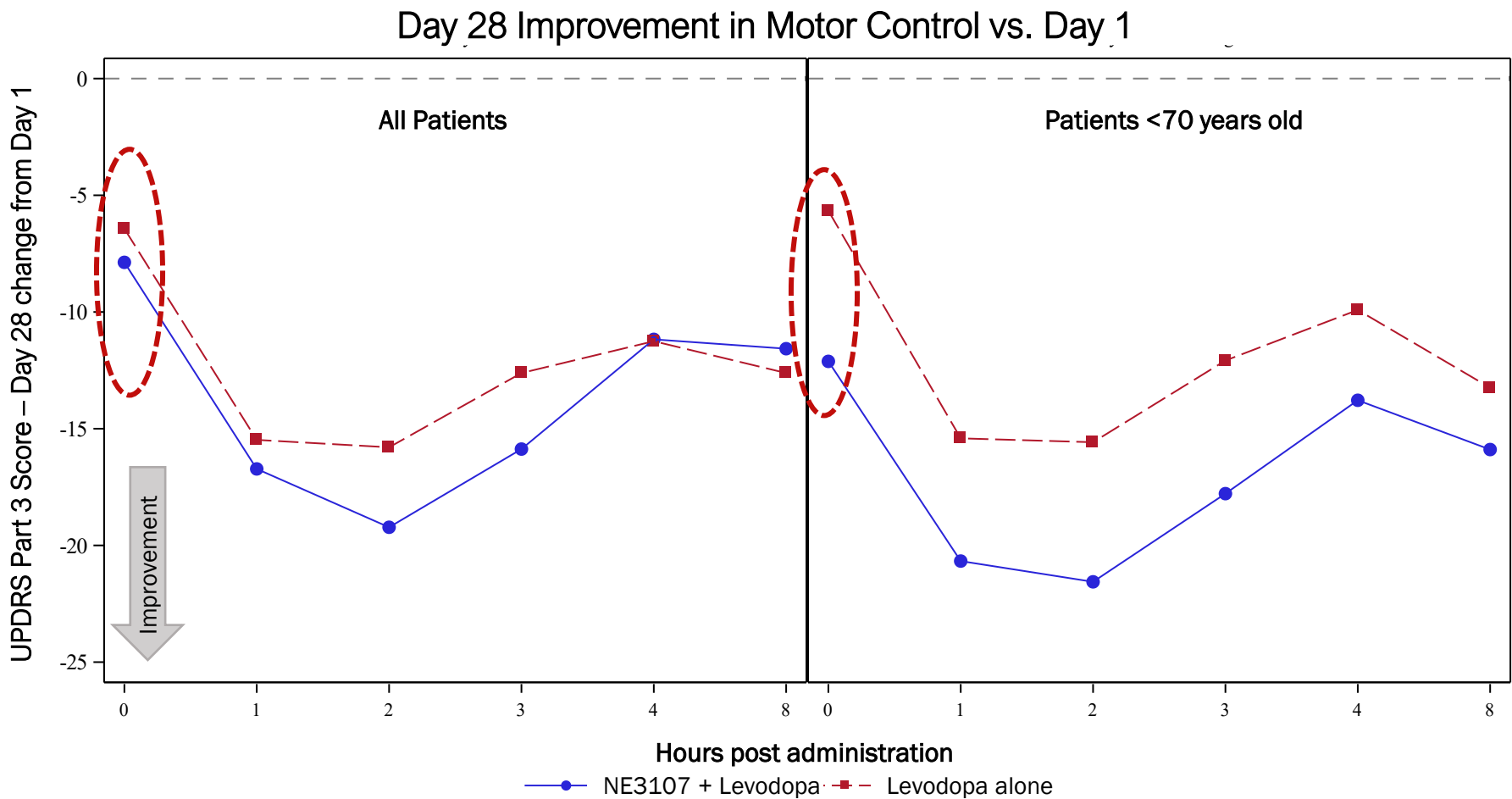
Amantadine (M10084)



Parkinson's disease clinical development program

- NM201 sought to satisfy FDA requirement for drug-drug interaction (DDI) study with L-Dopa and assess NE3107 promotoric and anti-LID activity
- 45 patients with defined L-Dopa “off state” randomized 1:1 active:placebo, 20 mg BID for 28 days
- Primary endpoints: Safety, L-Dopa PK and DDI
- Secondary endpoint: MDS-UPDRS* part 1-3 and NMSS**

NE3107-treatment patients experienced fewer motor symptoms before morning drug administration

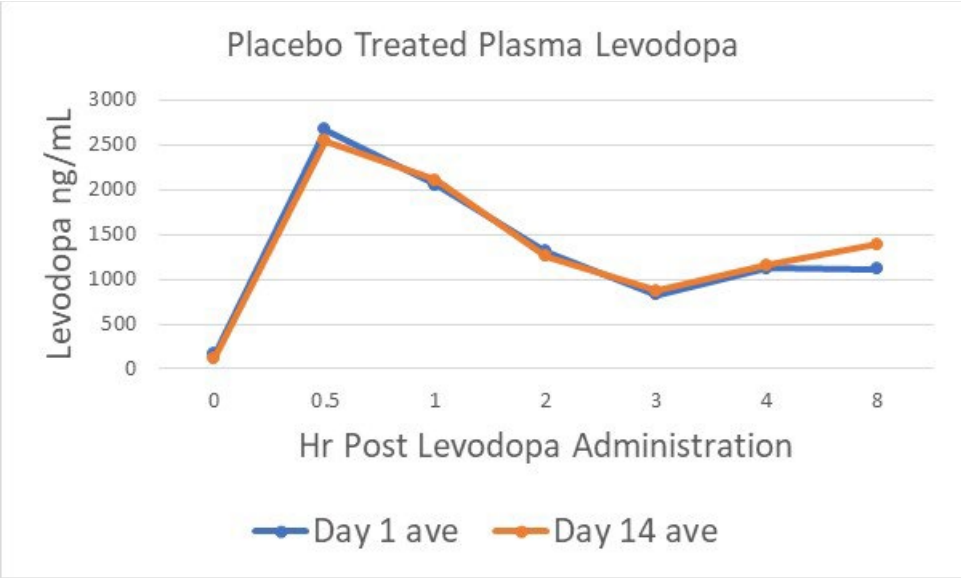
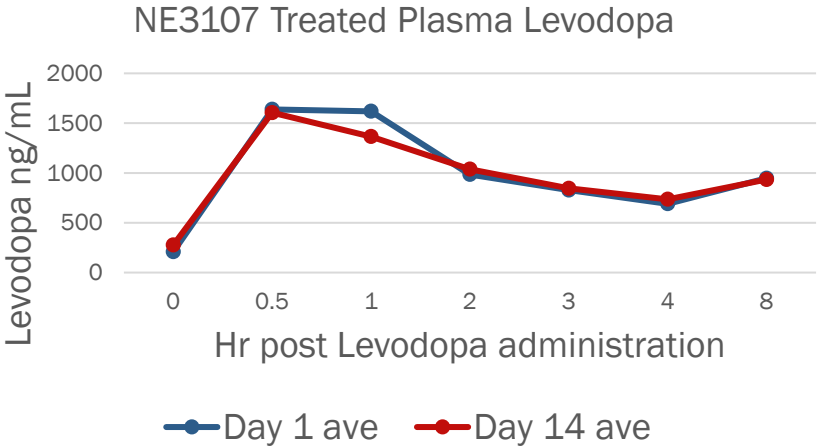
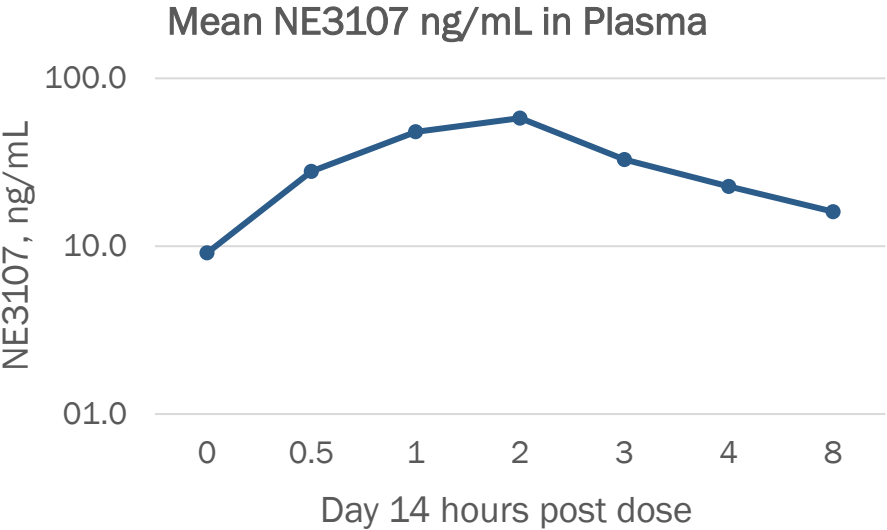


	NE3107	Placebo
“On” at t=0	6	0
Total patients	20	19
P-value*	0.02	

* Fisher’s exact test

Desirable pharmacokinetics – no observed DDI

0-8 hr 231 ngh/mL (SD 22.38)



Collaboration with Parkinson Study Group



- The largest, pro-active, not-for-profit scientific network of Parkinson Centers in North America for over 30 years
- Strict credentialing process, with over 150 Credentialed PSG Centers in North America, Hawaii, and the Caribbean
- Over 350 credentialed investigators and 245 coordinators and growing
- Strong partnerships with both the **Michael J Fox Foundation** and the **Parkinson's Foundation**
- International collaborations in India, China, Scandinavia, & Germany
- Partners receive support from credentialed coordination & biostatistics centers: **CTCC** at the University of Rochester, **NCRI** at Massachusetts General Hospital, and the **Columbia University Biostatistics Center**

BioVie's Parkinson's Advisory Committee

- **Anthony Lang** (co-chair), MD. Author of 950+ peer-reviewed papers and 100+ book chapters in Parkinson's Disease
 - Director, the Morton and Gloria Shulman Movement Disorders Clinic at Toronto Western Hospital
 - Jack Clark Chair for Parkinson's Disease Research at the University of Toronto
 - Lily Safra Chair in Movement Disorders at the University Health Network in Toronto
 - Director, the University Health Network and the University of Toronto Edmond J. Safra Program in Parkinson's Disease
 - Director, Division of Neurology at the University of Toronto
- **Hubert Fernandez** (co-chair), MD. Director, Center for Neurological Restoration, Cleveland Clinic
- **Tanya Simuni**, MD, FAAN. Arthur C. Nielsen Jr Professor of Neurology and the Director of the Parkinson's disease and Movement Disorders Center at Northwestern University Feinberg School of Medicine
- **Rajesh Pahwa**, MD. Laverne and Joyce Rider Professor of Neurology, chief of the Parkinson's and Movement Disorder Division and director of Parkinson Foundation Center of Excellence at the University of Kansas Medical Center
- **Sheng Luo**, PhD. Professor of Biostatistics & Bioinformatics, Duke University

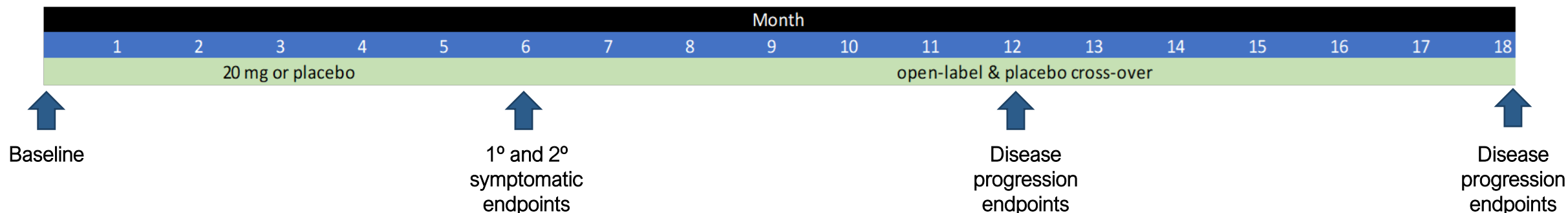
Strong support from advisors for a more aggressive plan to address significant unmet medical needs

*“The Parkinson’s Study Group (PSG) has provided guidance to many industry partners who have successfully brought to market Parkinson’s therapeutics that address the unmet needs of patients. **The PSG members unanimously recommended that the company accelerate plans to explore the potential efficacy of NE3107 in levodopa naïve Parkinson’s patients due to the current unmet need.** With great enthusiasm, the PSG advisory members look forward to collaborating with BioVie and seeing the results of clinical trials in this patient population”*

Dr. Hubert Fernandez on behalf of the Parkinson’s Study Group,
Co-Chair PSG and Director of the Center for Neurological Restoration at Cleveland Clinic

March 15, 2023

SUNRISE 1 and 2 in Treatment Naïve PD Patients



- SUNRISE-1 and Sunrise-2 have the same design in levodopa naïve PD patients
- 20 mg NE3107 or placebo BID, rescue medication permitted as directed by the investigator
- 6-month to primary endpoint (UPDRS Part 3 score) and secondary endpoint PADLS*
 - Placebo-cross over to NE3107 for 12-month extension to demonstrate disease modification
- Sunrise-1 to enroll 100 patients randomized 1:1, first patient enrollment 3Q2023
 - Blinded interim at 3 months to repower if needed
- Sunrise-2 expected to enroll an estimated 200 patients (to be repowered from Sunrise-1)
 - Start late 1Q2024 with primary endpoint readout 3Q2025
 - Sunrise-1 results in 3Q2024 will be used to re-power SUNRISE-2 Phase 3, if necessary

Additional endpoints

Secondary endpoints

- Parkinson's Disease Activities of Daily Living Scale* (PADLS)
- Time to initiation of PD therapy
- Parkinson's sleep scale

Exploratory endpoints

- Rate of disease progression defined by:
- Change in MDS-UPDRS Part 3 score compared to baseline after withholding all PD and study medications for 24 hours in treatment compared to placebo groups at 26, 52, and 56 weeks
- Change in MDS-UPDRS Part 3 score compared to baseline after withholding all PD and study medications for 24 hours in NE3107 patients after 26 and 52 weeks of treatment compared to historical controls, and at week 56
- Plasma levels of inflammatory biomarkers (adiponectin, leptin, CRP, ceruloplasmin, C1q, IFN γ , IL1 β , IL6, IL17, IL23, MCP1, RANTES, and TNF α)
- Change in heart rate variability compared to baseline
- Changes in DNA methylation compared to baseline

Epigenetics & Longevity

Epigenetics & Longevity program overview

- Early stages of program initiated based on early findings that NE3107 may affect the biological aging process
- We have identified the nucleus of academic collaborators to steer this program
 - Ekaterina Rogaeva, Ph.D., Professor, Department of Medicine University of Toronto; Chair Research on Dementia with Lewy Bodies; Tanz Center for Research in Neurodegenerative Diseases.
 - Nikolaos Daskalakis Ph.D., M.D., Assist. Prof. Psychiatry; Director Neurogenomics and Translational Bioinformatics Lab, McLean Hospital, Harvard Medical School

Genome reacts to environment, mutations & aging: epigenetics

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DNA

- 25,000 genes
- 6 billion base pairs
- 4% code for proteins
- 100 million variations

DNAm controls gene-expression

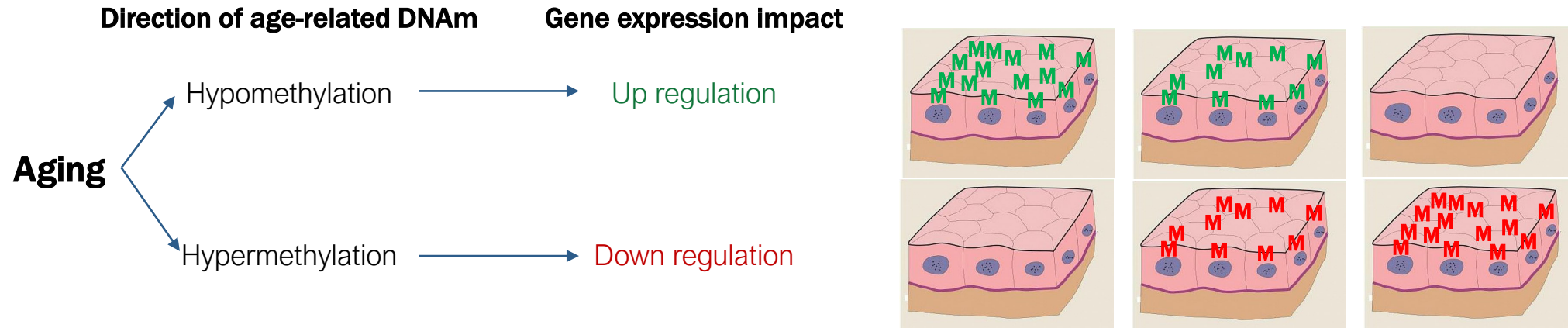
- 28 million CpGs in genome
- DNAm arrays: <1 million CpGs
- DNAm levels at some CpGs are age-related, affecting expression of >2000 genes

Protein

RNA

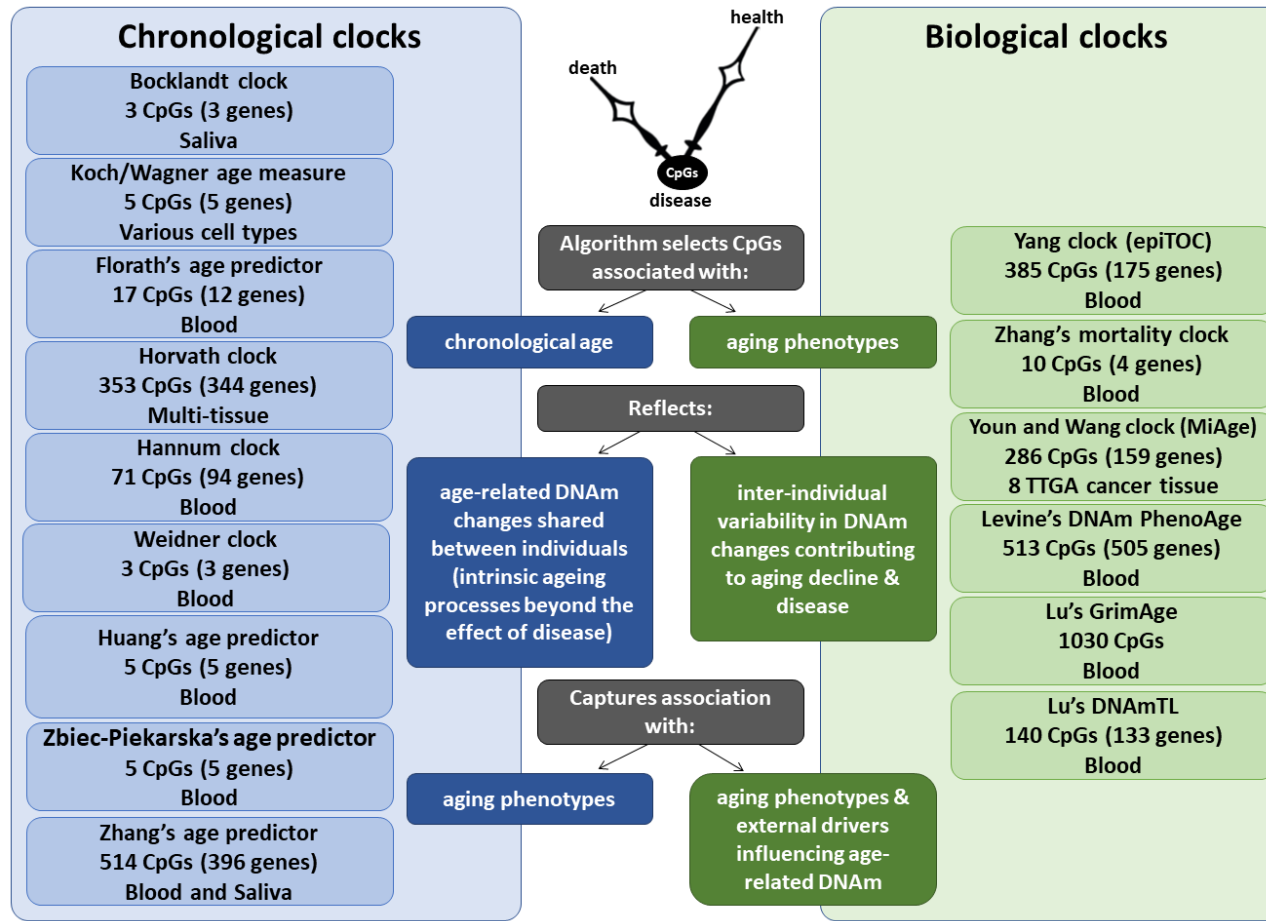
- Diet and exercise remains the most accepted tool for influencing epigenetics
- Clinicians have experimented with metformin and rapamycin

DNA methylation clocks and their predictive capacity for aging phenotypes and health span



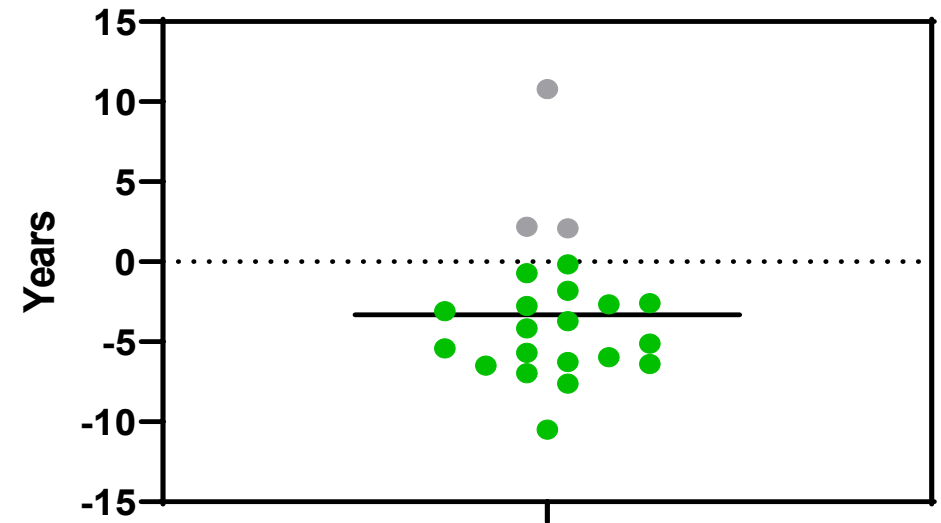
- DNAm clocks are based on cumulative assessment of the methylation levels of many age-related CpGs
- DNAm clocks reflect biological aging, the pace of which varies among individuals
- 15 reported DNAm clocks reflect different mechanisms of aging

Each DNAm clock is built according to a unique training method



NE3107 decreased biological age in MCI/AD subjects

- Dr. Steve Horvath* developed an extremely precise Biological age DNA methylation clock, the **DNAmSkinBloodAge**
- The biological clock age was in close agreement with the chronological age (72.3 vs 71.6; +0.98%) at baseline
- After 3 months treatment with NE3107 there was a decrease in DNA methylation commensurate with 3.3 years reduction on the Skin Blood Clock (68.1 vs 71.6; -4.9%)



mean -3.3 Years
95% CI -5.28 to -1.35
p=0.002
19/22 (86%) decreased

Next steps

- Additional results of NE3107 modulation of DNA methylation pertaining to immunomodulatory cells, endocrine mediator expression, and their correlates will be presented at the Alzheimer's Association International Conference (AAIC) July 16-20, 2023
- Along with Drs. Rogaeva and Daskalakis, we are assembling longevity advisors, collaborators and team
 - To deepen Biovie's current epigenetic analyses in three diseases of aging: MCI, Mild-moderate AD, and PD
 - To broaden to additional areas of exploration

Liver Disease

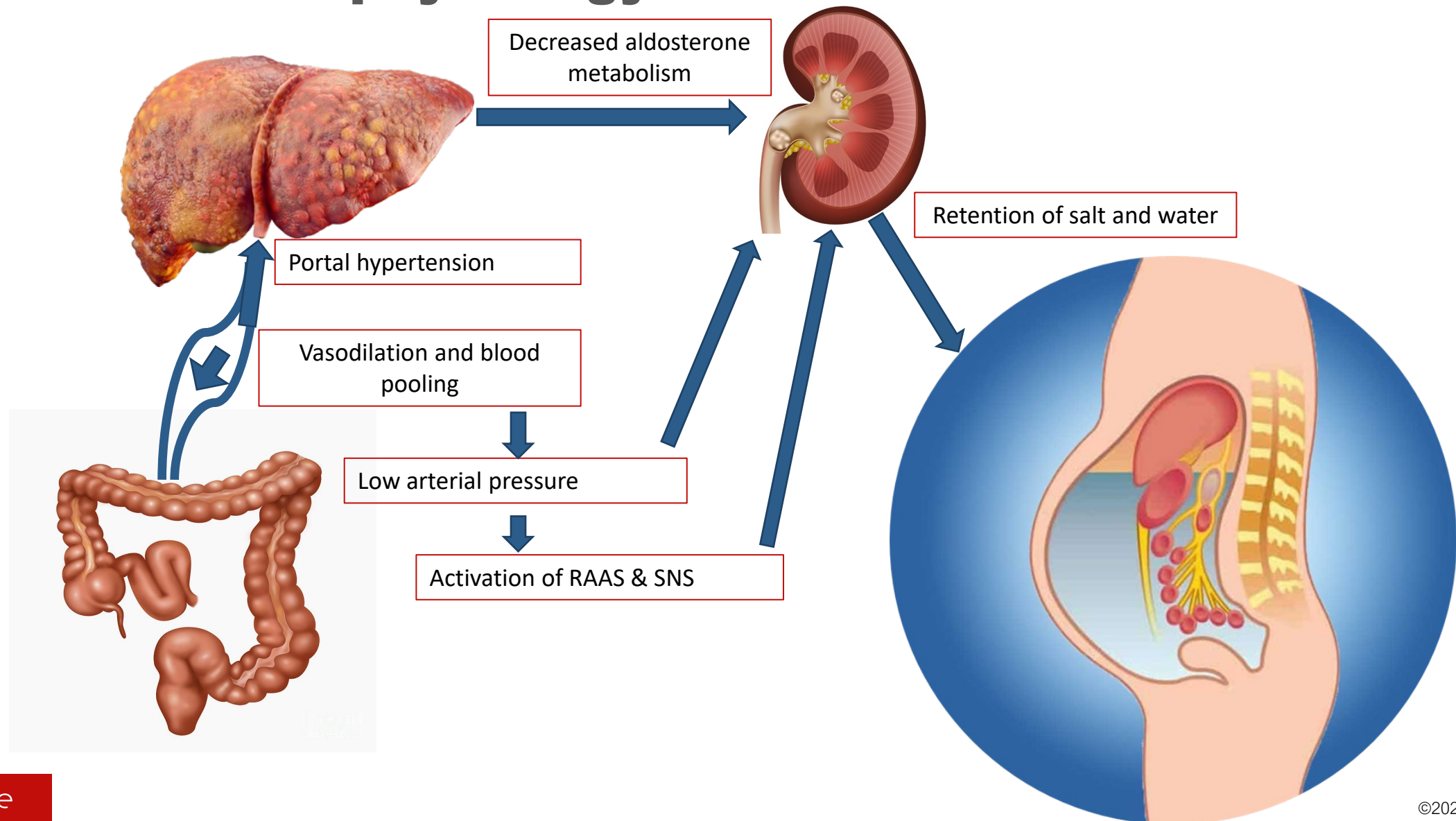
Refractory Ascites

- Fluid build-up in the abdomen of patients with advanced cirrhosis
 - Cannot be controlled with an effective dose of diuretics
- No approved pharmacological treatments – unmet need
- First line treatment is repeated mechanical draining by paracentesis
 - Surgically implanted shunts an option in select patients
- Mortality > 40% in 12 months*



Very poor quality of life
Not typically hospitalized

Ascites Pathophysiology



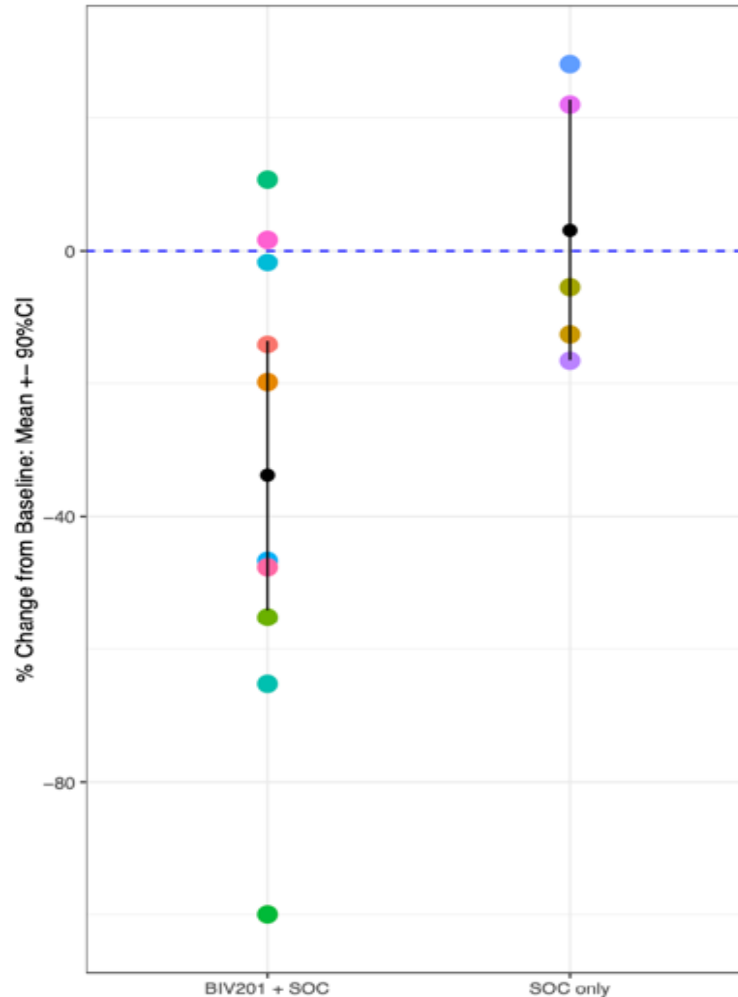
BIV201

- Terlipressin administered as a continuous infusion
 - Outpatient treatment with small ambulatory infusion pump
- Targets the pathophysiology of ascites
 - Multiple small trials and Phase 2a support efficacy in reducing ascites
- Orphan and Fast Track Designations for the treatment of ascites due to all etiologies except cancer
- Mallinckrodt's Terlivaz approved in US 2022 indicated *to improve kidney function in adults with hepatorenal syndrome with rapid reduction in kidney function*. Does not impact BIV201 Clinical Program
 - Approved for different indication – Orphan market exclusivity for HRS
 - Administered in conjunction with daily albumin
 - Different dosage form and administration (intermittent bolus injections)
 - Restricted to hospital setting - black box warning
- Impacts BIV201's regulatory pathway and non-clinical package for NDA (505(b)(1))

BIV201 Program Update

- Phase 2b - Originally targeted 30 patients randomized 2:1
- Paused enrollment based on encouraging data from the first 15 patients informing next steps
 - 10 randomized to BIV201; 5 randomized to standard of care
 - 5 completed 2 X 28-day cycles
 - 5 discontinued treatment during or at end of Cycle 1

BIV201-treated patients experienced a 37% advantage in ascites fluid reduction compared to SOC



BIV201 + SOC

Mean: 34 % reduction

5/10 (50%) with >40% reduction

P=0.0046

SOC only

Mean: 3.1 % increase

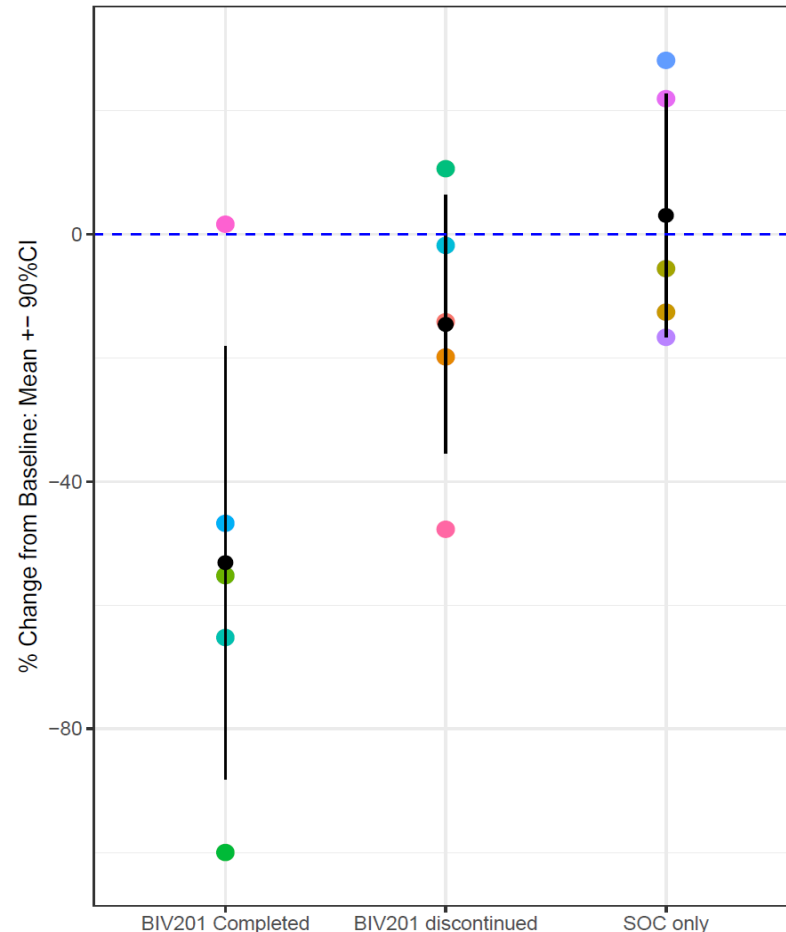
0/5 (0%) with >40% reduction

P=0.8

BIV201 vs SOC

P value = 0.05 for difference

Patients completing BIV201 treatment experienced 56% advantage in ascites fluid reduction compared to SOC



- 53% reduction in ascites volume among patient completing BIV201 treatment
- 15% reduction among patients who started but did not complete treatment
- 3.1% increase for SOC patients
- $p < 0.001$

Safety

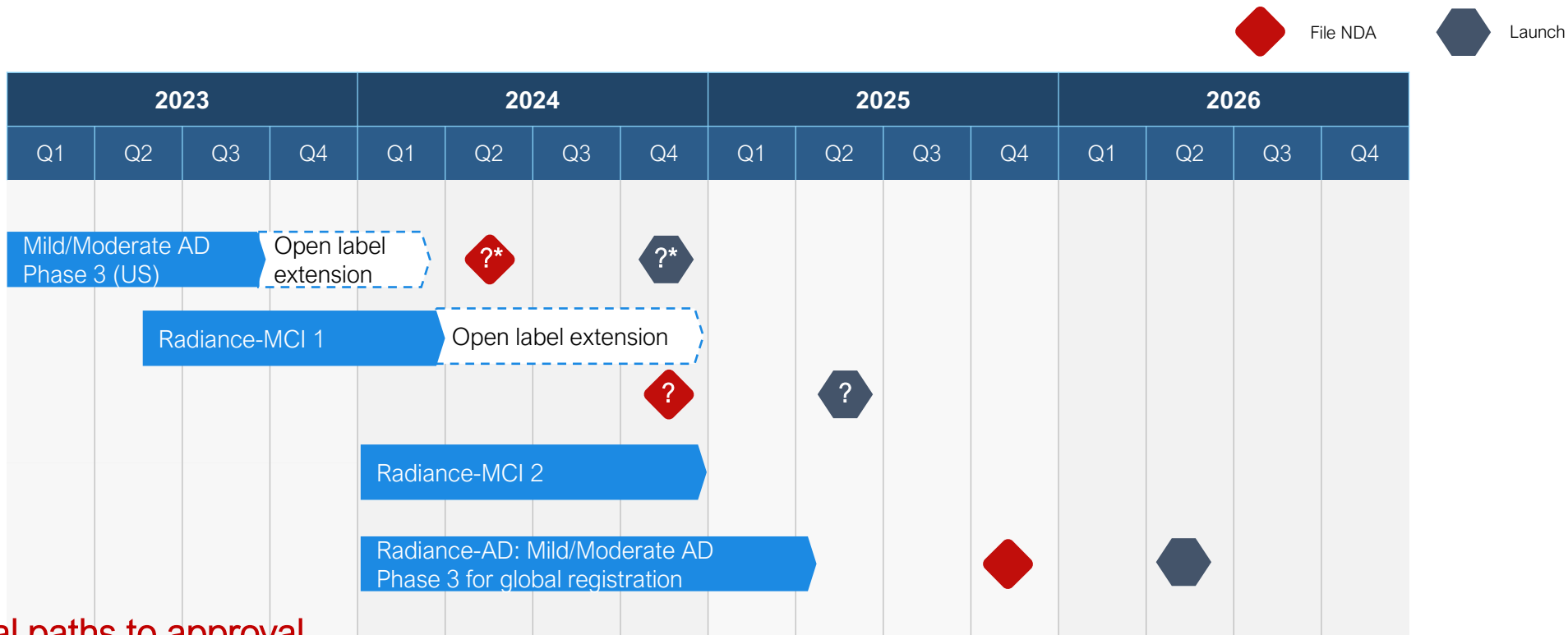
- Incidence of treatment emergent adverse events similar in both groups
- Overall safety consistent with patient population with advanced cirrhosis
- No unexpected SAEs

Conclusions

- BIV201 treatment resulted in a reduction in ascites
- Treatment appears to be well tolerated
- Engaging FDA in discussions on next steps

Clinical & Regulatory Strategy

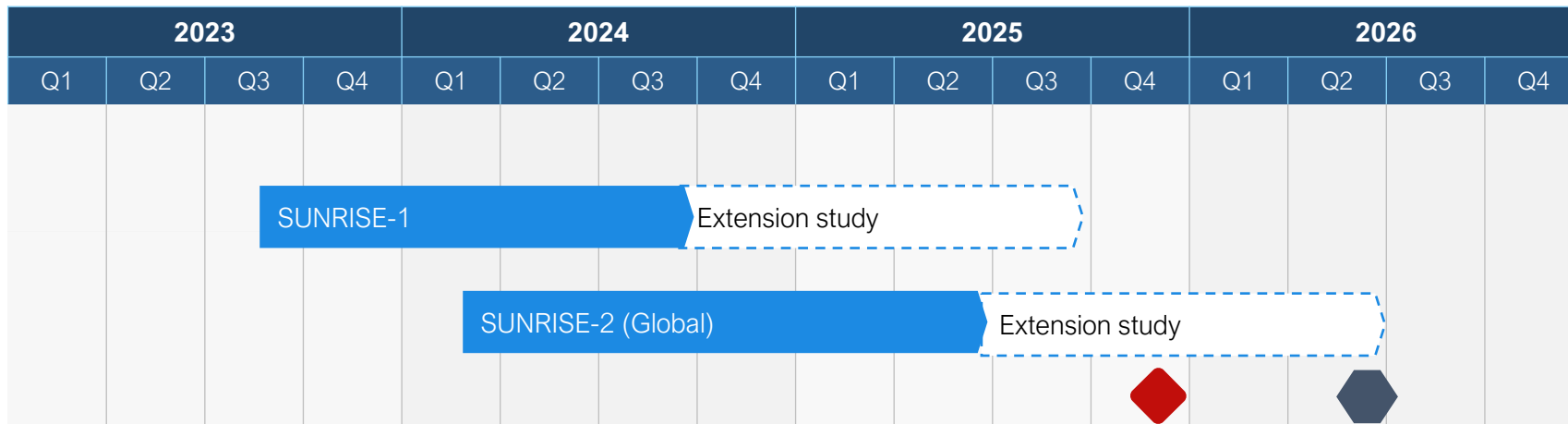
Alzheimer's Clinical Program creates maximum optionality for approval



Potential paths to approval

- Conditional approval based on strong clinical, functional, and objective results from single US Phase 3 pivotal in mild/moderate AD
- Regular approval for broad indication in dementia (MCI through Moderate) based on combination of positive outcomes from Phase 3 in mild/moderate and Sunrise-MCI 1
- Regular approval for mild/moderate AD from US and Global Phase 3 trials in mild/moderate

Parkinson's clinical program is straight forward



- Both SUNRISE-1 and SUNRISE-2 are designed to serve as pivotal studies
 - Primary end-point of UPDRS Part 3 (motor) scores at 6 months
 - Extension to 18 months to demonstrate disease modification
- SUNRISE-1 adaptive design enables sample size adjustment (if needed) at 3 months
- SUNRISE-2 sample adjustment (if needed) based on SUNRISE-1 results

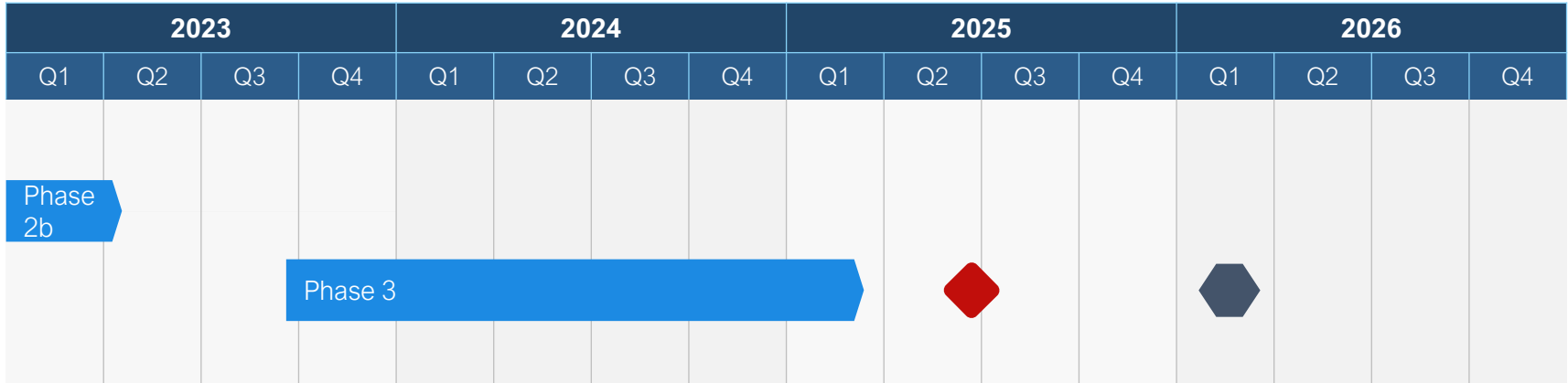


File NDA



- Launch

Ascites program



- Phase 2b paused with only one-half targeted patients enrolled due to statistically significant results demonstrating reduction of ascites fluid with BIV201 treatment
- Discussions initiated with FDA on path forward



File NDA



Launch

Commercialization Roadmap

Commercialization approach

- All options are open
- Partnering is an option
 - Initial conversation have been held
 - Some companies interested in global collaboration
 - Others in geographic rights
 - Most companies are intrigued and awaiting Phase 3 data readout
- Go-alone is always an option

Commercial potential in US market alone

Ascites

\$1.6B

US peak sales

- \$45K/year
- 45% market penetration
- 2026 launch
- 2032 peak sales

Alzheimer's

\$30B

Annual sales for every
1 million people treated

- 15% market penetration
- \$30K/year – much lower
all-in cost vs. competition

Parkinson's

\$3B

Annual sales for every
100,000 people treated

- 10% penetration of US
market
- \$30K/ year

Current finances

- \$44 million cash balance as of February 28, 2023
- We have resources to fund operations through most of the year and could extend through first part of 2024 if necessary

Social Impact

BioVie is the founding backer of Social Impact Partners



Mission:

To combat Alzheimer's and other neurodegenerative conditions and enable people to lead better and healthier lives by 1) working collaboratively, 2) raising awareness, and 3) mobilizing resources that drive innovation and new solutions.

SIP's Goals



Mobilizing Resources & Driving Innovation

1. Mobilize 1 million new healthcare workers to support individuals and families.
2. Increase public/private funding of Alzheimer's medical research 3x in 5 years and expand Medicaid/Insurance Coverage for services needed.
3. Invest and grow in 10 companies annually who advance innovative solutions.



Working Collaboratively

4. Connect a global coalition of partners and individuals to promote a public and private collaboration to positively impact healthy aging and brain health.
5. Create college campus networks with Youth Ambassadors across the all countries.
6. Encourage industry collaboration (PPP's sharing preclinical models and resources).



Raising Awareness & Education

7. Implement a significant public service ad campaign to build universal awareness of Alzheimer's.
8. Draft legislation with "Youth Board" in conjunction with consortium to present in DC & globally.
9. Support "turning passion into action" with speaker series and educational programs for universities.

Meet our global consortium and youth board schools



Recap

Executive Summary

- The company is in the early stages of multiple late-stage data readouts for multiple indications addressing great unmet medical needs
- Early results in Alzheimer's Disease (AD), Parkinson's Disease (PD) and Ascites have all been encouraging, and our KOLs/SAB have encouraged us to move more assertively
- Potentially pivotal registrational trials are underway or will begin in 2023 for all indications
- Topline results from our Phase 3 AD trial expected to be announced October 2023. Regulatory submissions expected in 2024 and 2025
- We believe our portfolio, if approved, will create a platform of great benefit to patients and economic value to shareholders
- All development and commercialization options (including partnering) being considered
- Our support of Social Impact Partners allows us to bring better patient-centered support to affected individuals/families, of which our future medication will be an important part

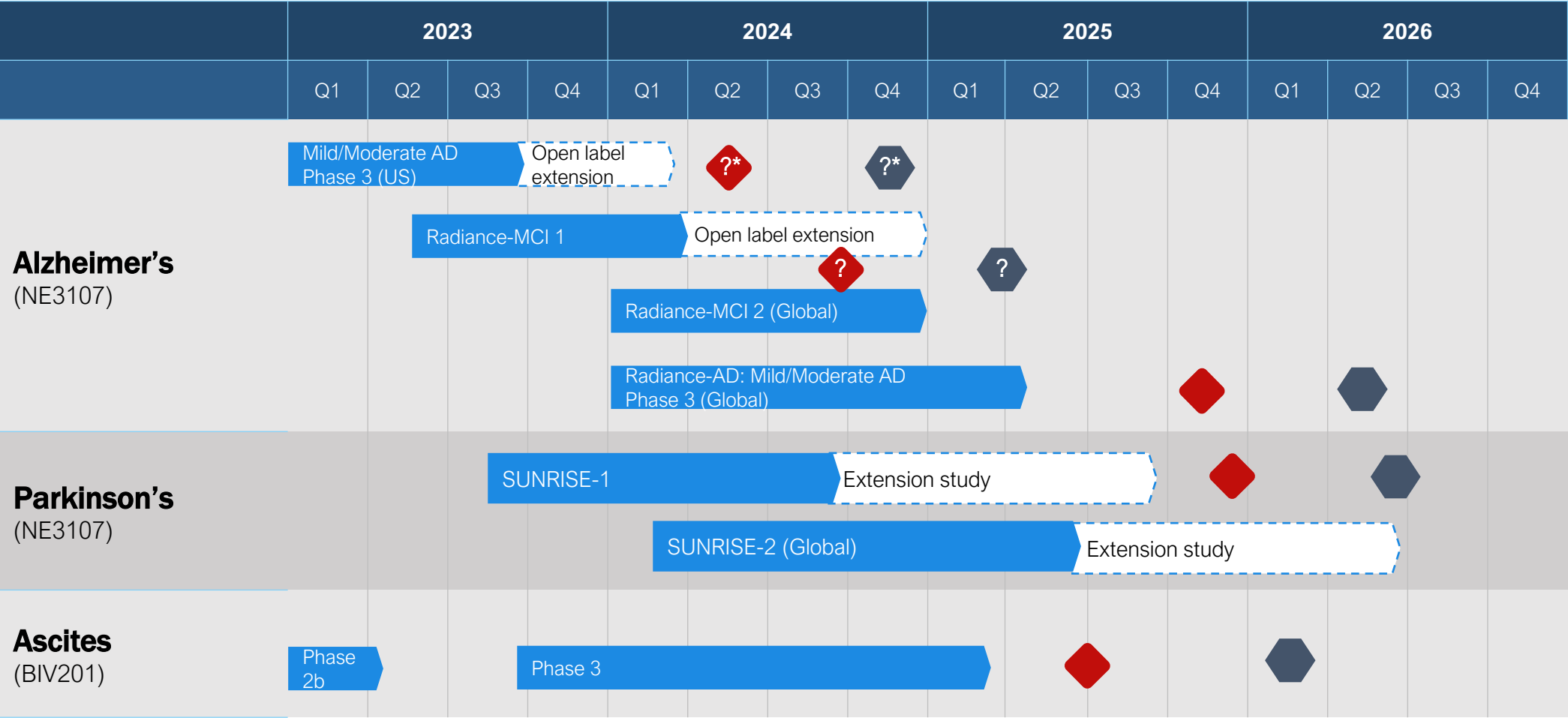
Expected Catalysts & anticipated timelines



File NDA



Launch

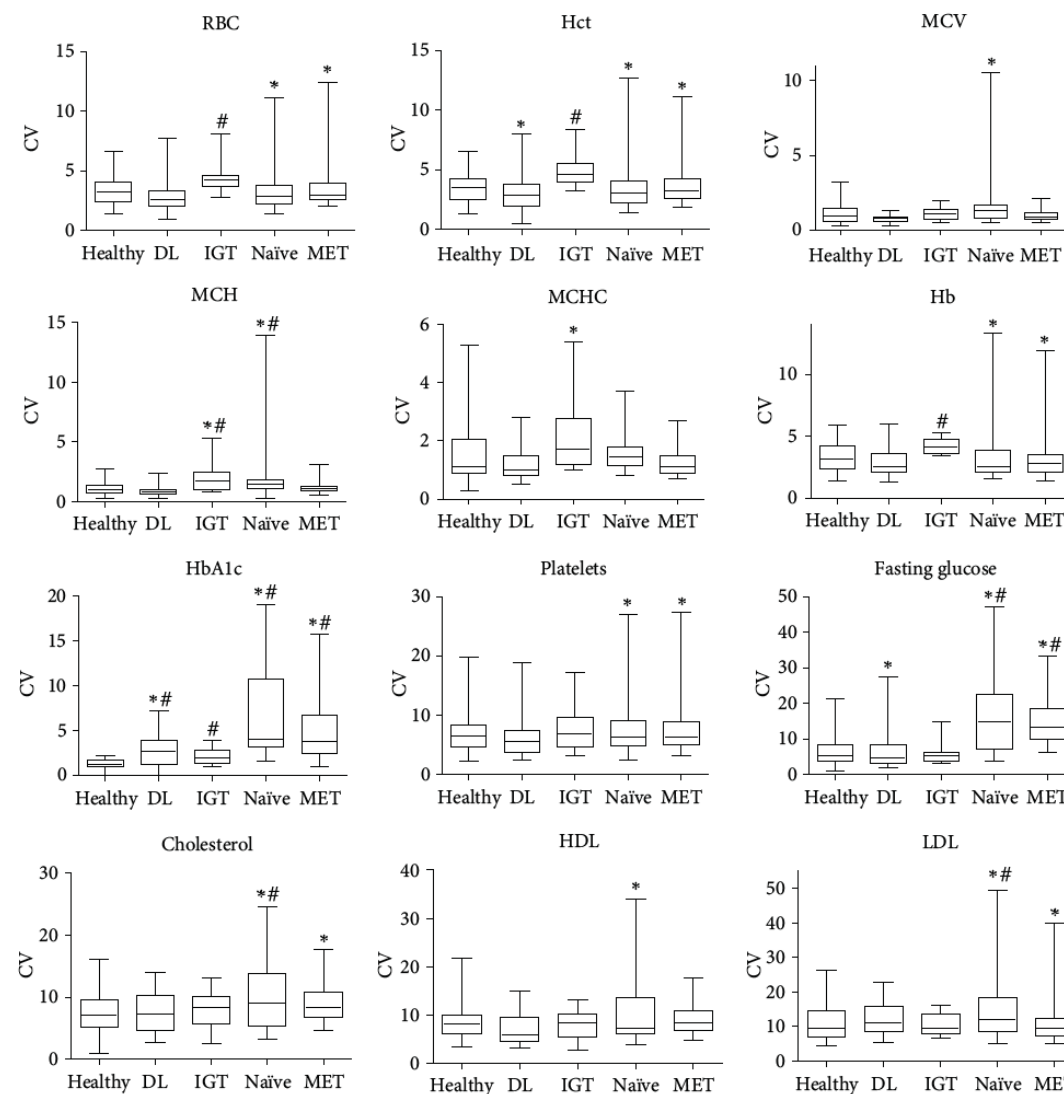


* Two Phase 3 trials are usually required for registration. However, the FDA has allowed filing based on strong data from a single pivotal trial in indications with few therapeutic options.

Appendix

Inflammation drives systems dysregulation

- Patients with increasing metabolic disease show greater variability for individual hematology, metabolic and chemistry values
- Indicates increasing dysregulation



DL: dyslipidemic placebo group, IGT: impaired glucose tolerant placebo group, Naïve: treatment-naïve uncontrolled T2DM placebo group, MET: uncontrolled T2DM placebo group on a stable dose of metformin

RBC: red blood cell count, Hct: hematocrit, MCV: mean cell volume, MCH: mean corpuscular hemoglobin, MCHC: mean corpuscular hemoglobin concentration, Hb: hemoglobin, HbA1c: hemoglobin A1c, HDL: high density lipoprotein cholesterol, LDL: low density lipoprotein cholesterol.

Statistically Significant Welch ANOVA).

* Statistically significant 2-sided FF test.

Reading 2013 *Mediators Inflamm* 814989

NE3107 decreased systems dysregulation in a Phase 2 study

- Heteroscedasticity* investigated by analyzing data distributions for normality (Shapiro-Wilks *W* test)
 - Deviations from normal distribution represents dysregulation
- Placebo patients had significantly abnormal data distributions after 84 days of treatment whereas NE3107 subjects did not
 - Placebo patients had many parameters that significantly deviated from normal distribution (i.e., dysregulated)
 - NE3107 treatment reduced systems dysregulation

Group	Day	Parameter	HE3286 <i>W</i> test <i>P</i>	Placebo <i>W</i> test <i>P</i>
Cohort 1	84	ΔInsulin ^d	>0.1	<0.0001
		ΔC-peptide	>0.1	<0.0001
		ΔFasting glucose	>0.1	0.02
		ΔHOMA2 %B	>0.1	<0.0001
		ΔHOMA2 IR	>0.1	0.002
		Δleptin	>0.1	0.005
Cohort 1 MCP-1 > 40 ^b	84	ΔHbA1c	>0.1	0.006
		ΔFasting glucose	>0.1	0.02
		ΔHOMA2 %B	>0.1	<0.0001
Cohort 2	84	ΔnHbA1c	>0.1	0.04
		ΔInsulin	>0.1	>0.1
		ΔFasting glucose	>0.1	0.03
		ΔHOMA2 %B	>0.1	>0.1
		ΔMCP-1	>0.1	0.005
	112	ΔTriglycerides	>0.1	<0.0001
		ΔnHbA1c	>0.1	0.0007
		ΔInsulin	>0.1	>0.1
		ΔFructosamine	>0.1	0.002
		ΔHOMA2 %B	>0.1	<0.0001
Cohort 2 BMI > 31 ^c	84	ΔHOMA2 %B	>0.1	0.007
		ΔMCP-1	>0.1	>0.1
		ΔTriglycerides	>0.1	>0.1
	112	ΔInsulin	>0.1	<0.0001
		ΔC-peptide	>0.1	<0.0001
		ΔHOMA2 %B	>0.1	<0.0001
		ΔHOMA2 IR	>0.1	<0.0001

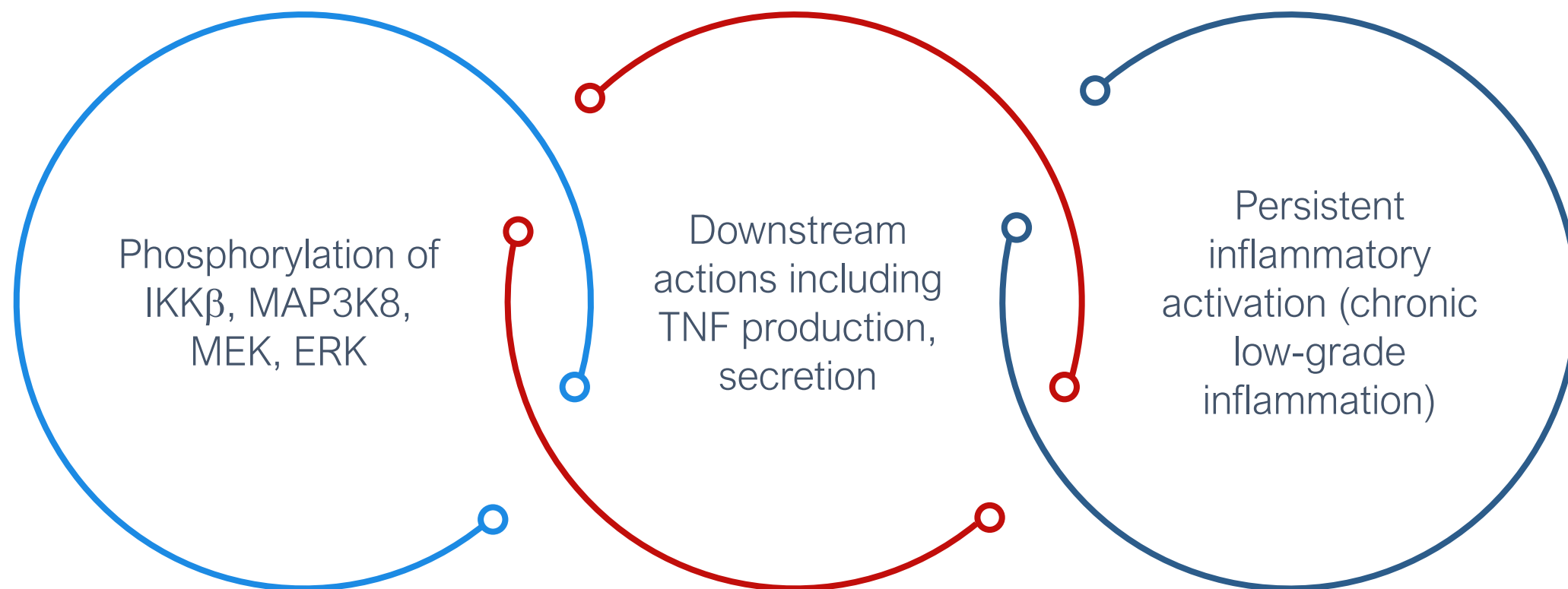
* Heteroscedasticity describes differences in variances between groups.

Reading 2013 *Mediators Inflamm* 814989

Note: NE3107 was originally known as HE3286 from Hollis Eden Pharmaceuticals

NE3107's inhibition of inflammatory signaling

NE3107 binds to ERK in the NF κ B scaffold complex, and alters the scaffold conformation, inhibiting



Homeostatic insulin signaling



01

Hyperglycemia increases insulin, which binds to the insulin receptor (IR) on cells in muscle, liver, adipose tissue, brain, pancreas, etc.

02

Insulin binding to IR triggers IR phosphorylation of insulin receptor substrate 1 & 2 (IRS1/2) on tyrosine, activating Phospho-inositol 3 kinase (PI3K) and MAPKs RAF, MEK and ERK

03

PI3K feeds back to phosphorylate IRS1/2 on serine, inhibiting further stimulation as glucose is taken up in cells in response to insulin

Inflammatory inhibition of Insulin signaling

As in macrophage lineage cells, the NF κ B resting scaffold is present in these insulin target cells

Inflammatory mediators (e.g., Amyloid β , alpha-synuclein, TNF, fatty acids) activate NF κ B, IKK β , as in macrophages

IKK β is a serine kinase, and phosphorylates serine on IR, blocking PI3K and ERK homeostatic signaling and resulting in insulin resistance

NE3107 restoration of Insulin sensitivity

