Unlocking the science of longevity to develop transformative therapies

Corporate Presentation • September 2023

Forward-looking statements

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Overview

We believe that TNFα-mediated inflammation

- Initiates and perpetuates a forward-feeding pro-inflammatory cycle
- Leads to insulin resistance
- Accelerates the “DNA methylation” and the aging process

Our lead asset NE3107 modulates the production of TNFα. In clinical trials, many patients treated with NE3107 experienced:

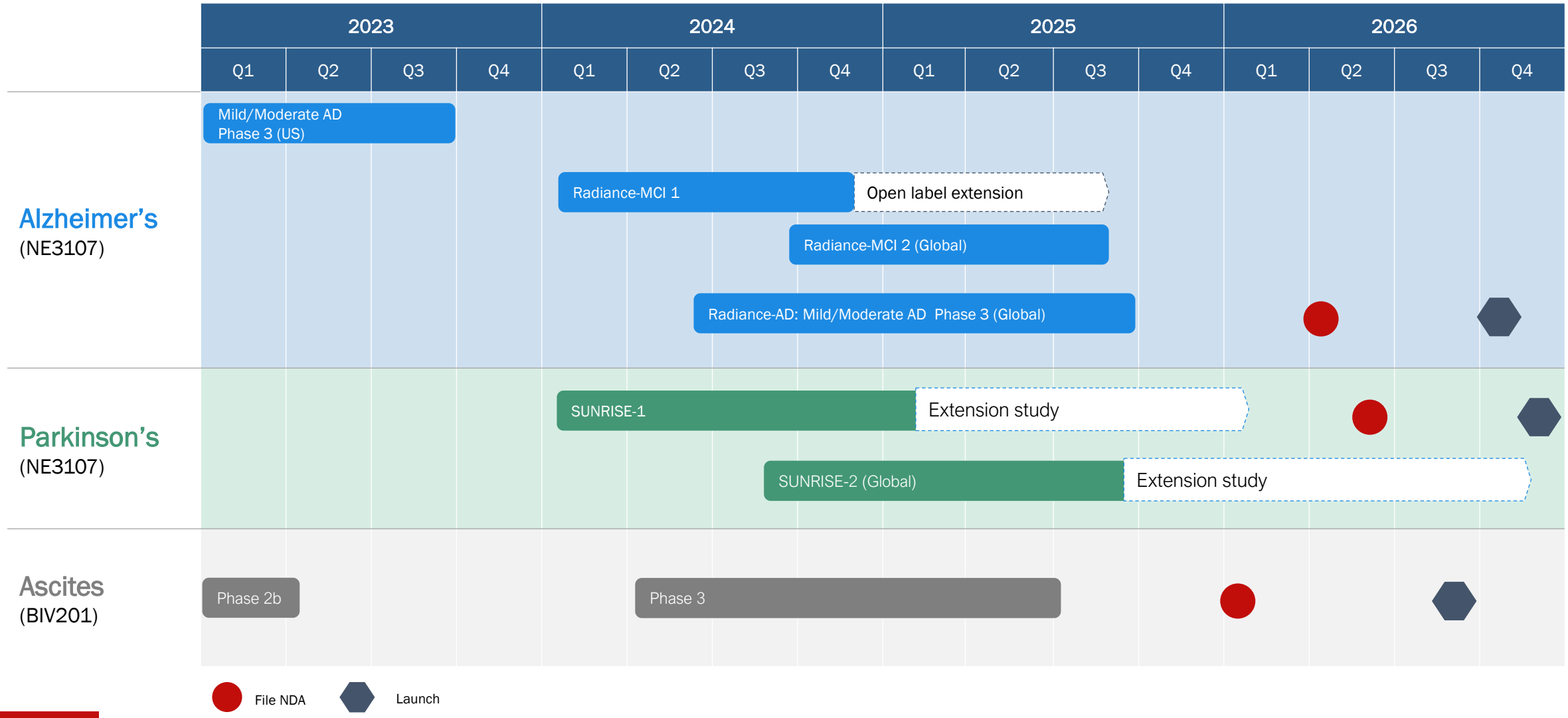
- Reduced inflammation and the associated insulin resistance
- Improved cognition, lowered p-tau levels, and improved brain imaging scans in Alzheimer’s Disease (AD)
- Improved motor control and “morning on” symptoms in Parkinson’s disease (PD)
- Lowered DNA methylation levels

NE3107 may change the expression of specific genes in a manner that is significantly correlated to observed cognitive and biomarker changes

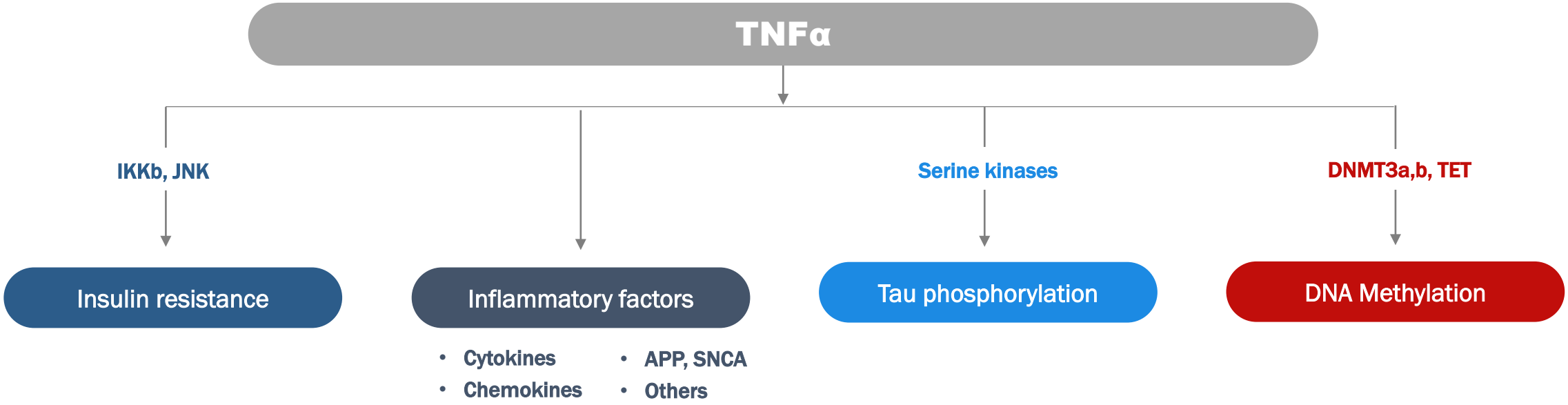
- Provides epigenetic basis to explain improvements observed in AD and PD trials
- Gives optimism for what we may see when Phase 3 AD trial reads out in Q4 2023

BIV201 reduces fluid build up and has the potential to become the first therapeutic for ascites, a condition with 50% mortality rate within 12 months. Discussions with FDA underway to finalize Phase 3 trial design

Expected catalysts and anticipated timelines



Far-reaching impact of TNFα-mediated chronic low-grade inflammation



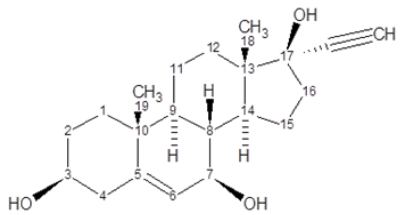
Diabetes
Metabolic disorders
Parkinson's

Alzheimer's
Parkinson's
Metabolic disorders
Various cancers
Rheumatoid arthritis
Various autoimmune diseases

Alzheimer's

Systems dysregulation of gene expression
Accelerated aging
Various cancers
Cognitive impairment and dementia
Parkinson's disease
Cardiovascular disease
COPD and respiratory disease
Chronic kidney disease
Inflammatory bowel disease
Sepsis
Others

NE3107's mechanism of action

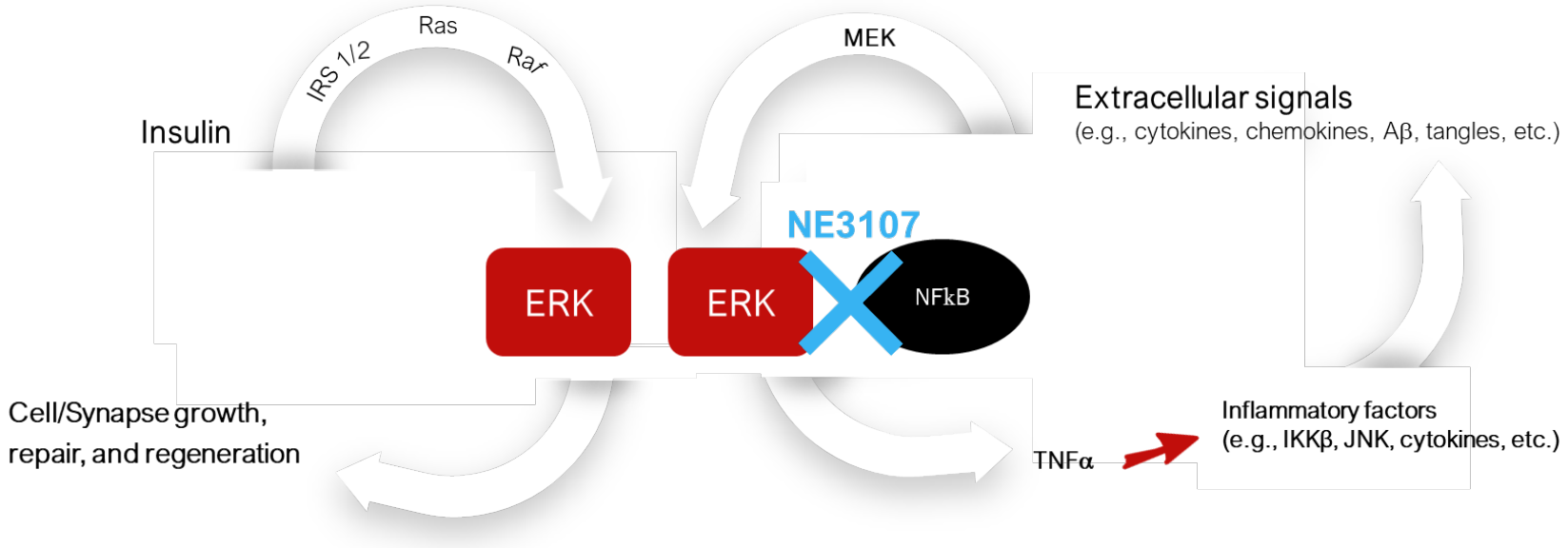


First-in-class molecule with desirable characteristics

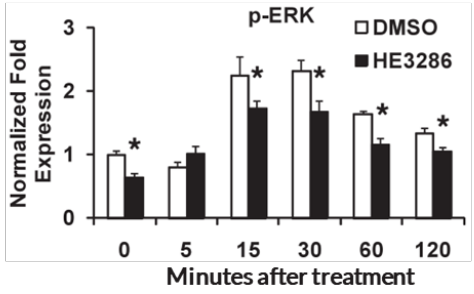
Small molecule; orally bioavailable

Crosses blood-brain barrier, thus CNS and peripheral applications

No safety issues identified to date in pre-clinical and clinical trials (up to Phase 2)

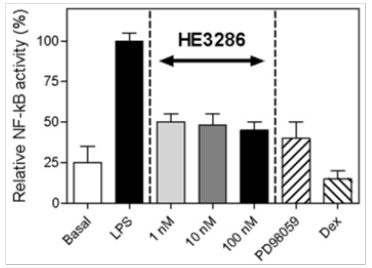


NE3107 Reduces ERK Activation



Lu 2010 Am J Physiol Endocrinol Metab 298 E1036

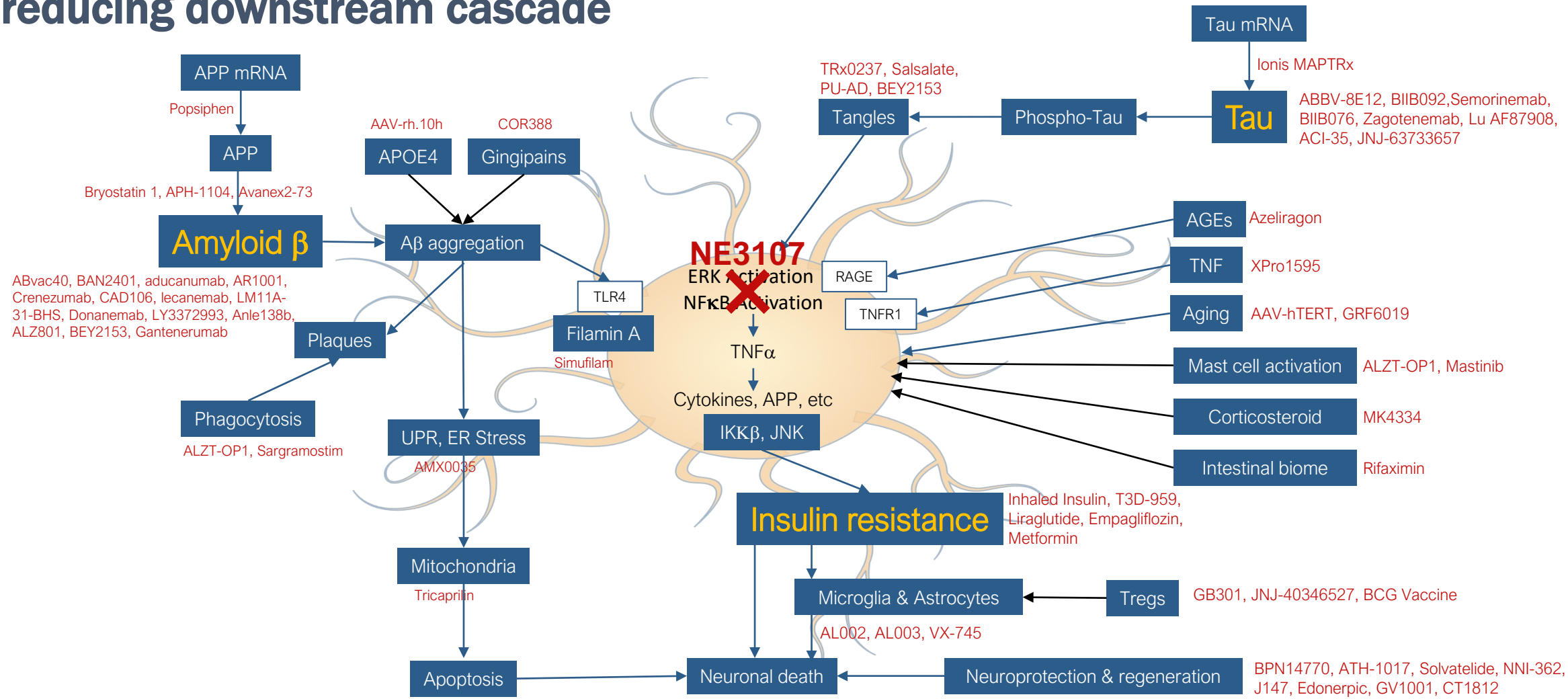
NE3107 Reduces NFkB Activity



Wang 2010 J Pharmacol Exp Ther 333 70

NE3107
in Alzheimer's Disease

NE3107 modulates inflammation at the central hub, thereby potentially reducing downstream cascade



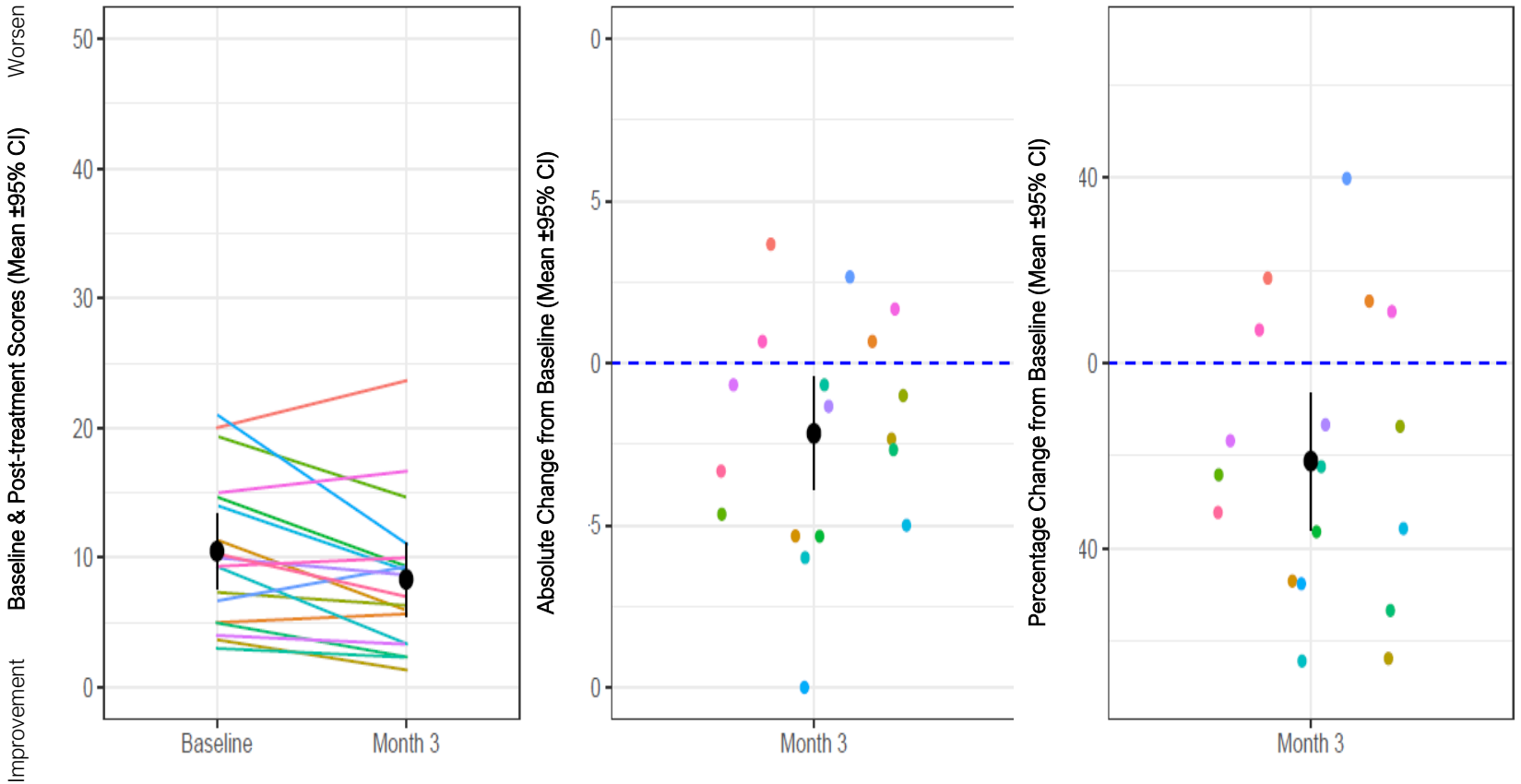
Investigator-Sponsored Trial in MCI and Mild Alzheimer's Disease, NCT05227820

An open-label, Investigator-Initiated exploratory biomarker study in patients who have MCI to Mild AD

- 55-89 years old, males and females experiencing cognitive decline with Clinical Dementia Rating (CDR) score of 0.5 to 1, suggesting mild cognitive impairment to mild dementia
- Study seeks to measure changes in cognition through verbal and visual test procedures and changes in biomarkers of Alzheimer's disease and inflammatory and metabolic parameters that can be measured in the CNS with advanced neuroimaging techniques in patients before and after treatment with 20 mg of NE3107 twice daily for 3 months

- **Use advanced functional magnetic resonance imaging (fMRI) to assess change from baseline to completion**
  - Change in glutathione levels will be analyzed as measured by Magnetic Resonance Spectroscopy (MRS)
  - Change in Diffusion Tensor Imaging - Neurite Orientation Dispersion Density Imaging (DTI-NODDI) to assess stabilization and or improvement in dendritic density
  - Change in Arterial Spin Labeling (ASL); change in functional connectivity of the nucleus basalis of meynert (NBM) with both hippocampi as well as between both hippocampi; and change in Neurovascular Coupling (NVR) as measured on BOLD imaging
- **Additional exploratory endpoints**
  - Clinical Dementia Rating Change as calculated from the Quick Dementia Rating Scale (QDRS) Change
  - Montreal Cognitive Assessment (MoCA) Change
  - Alzheimer's Disease Assessment Scale - Cognitive Subscale (ADAS-Cog11) Change
  - Mini-Mental State Examination (MMSE) Change
  - Glucose Serology/Metabolic Level Change
  - DNA methylation Change

Significant improvements in ADAS-Cog12 assessment of cognition among MCI/Mild AD patients



MMSE >=20 patients

13/18 improved (72%)[1]

Mean Absolute Change = -2.2 (p=0.0173)

Mean % Change = -21.1% (p=0.0079)

Cognitive improvements consistent across multiple assessment scales

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*

ADAS-Cog12
= Alzheimer's Disease Assessment Scale-Cognitive

MMSE
= Mini-Mental State Examination

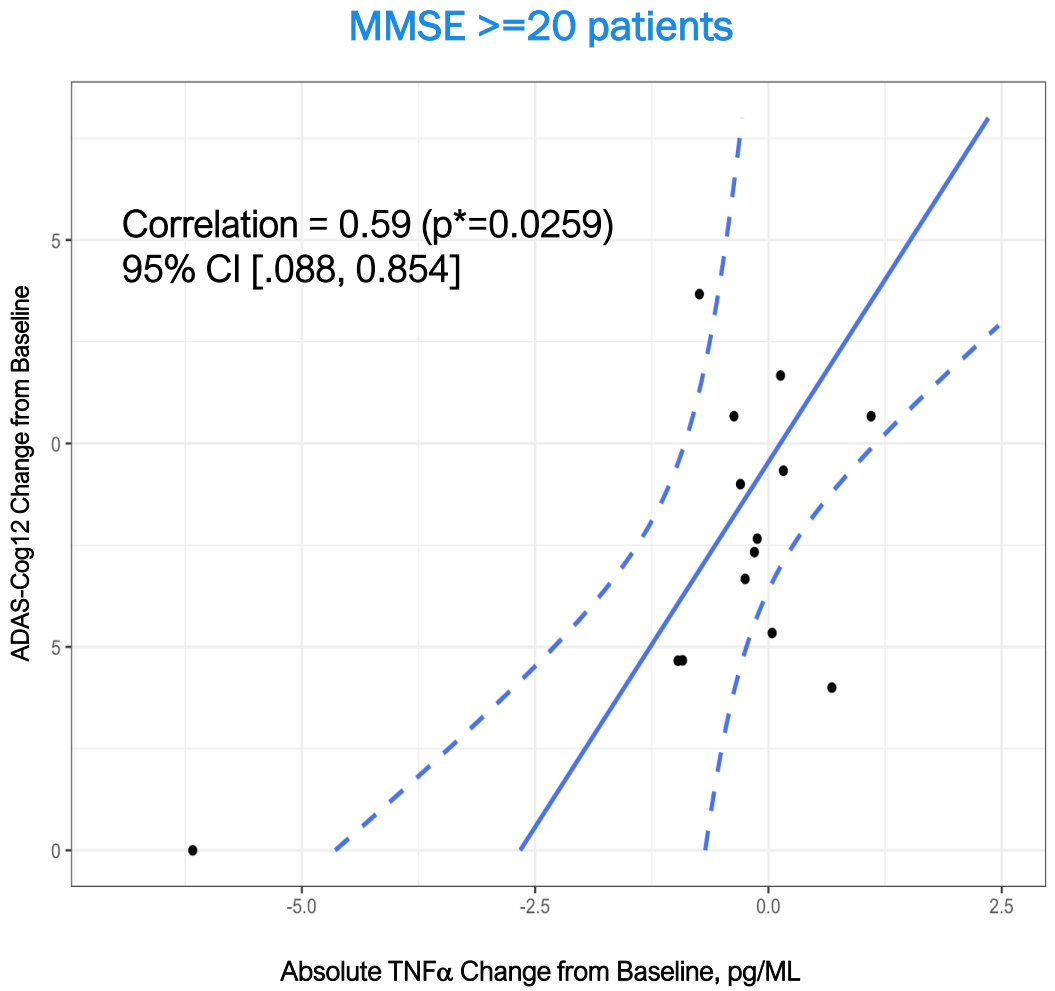
MoCA
= Montreal Cognitive Assessment

QDRS
= Quick Dementia Rating System

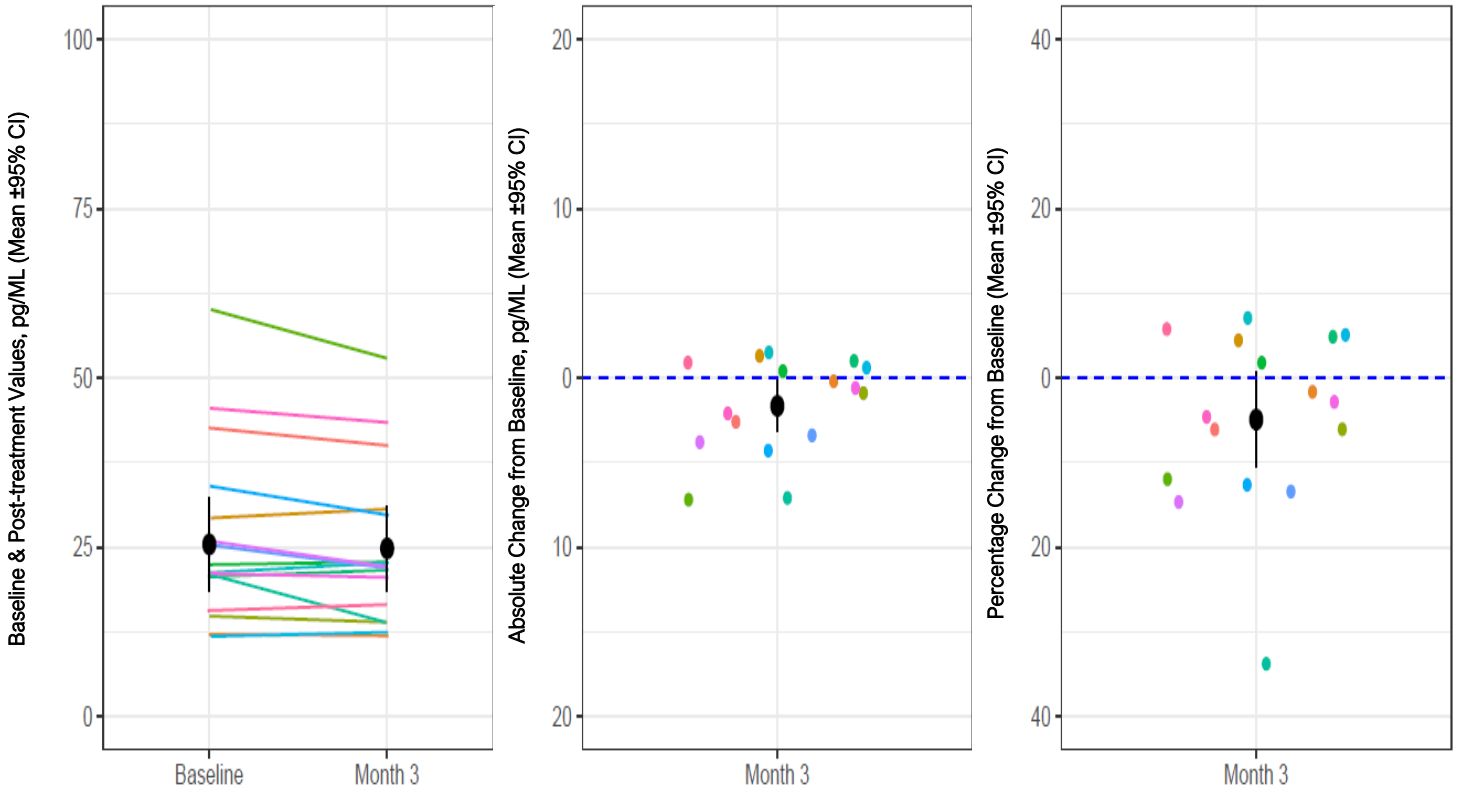
CDR
= Clinical Dementia Rating scale

ADCOMS
= Alzheimer's Disease Composite Score

Improvements in TNFa significantly correlated to improvements in ADAS-Cog12



Significant improvements in CSF p-tau



NE3107 decreased CSF p-tau by 5% over 3 months among MCI/Mild AD patients

Due to NE3107's mechanism, reduction in p-tau levels are expected to increase and accumulate over time

MMSE >=20 patients

10/16 improved (63%)[1]

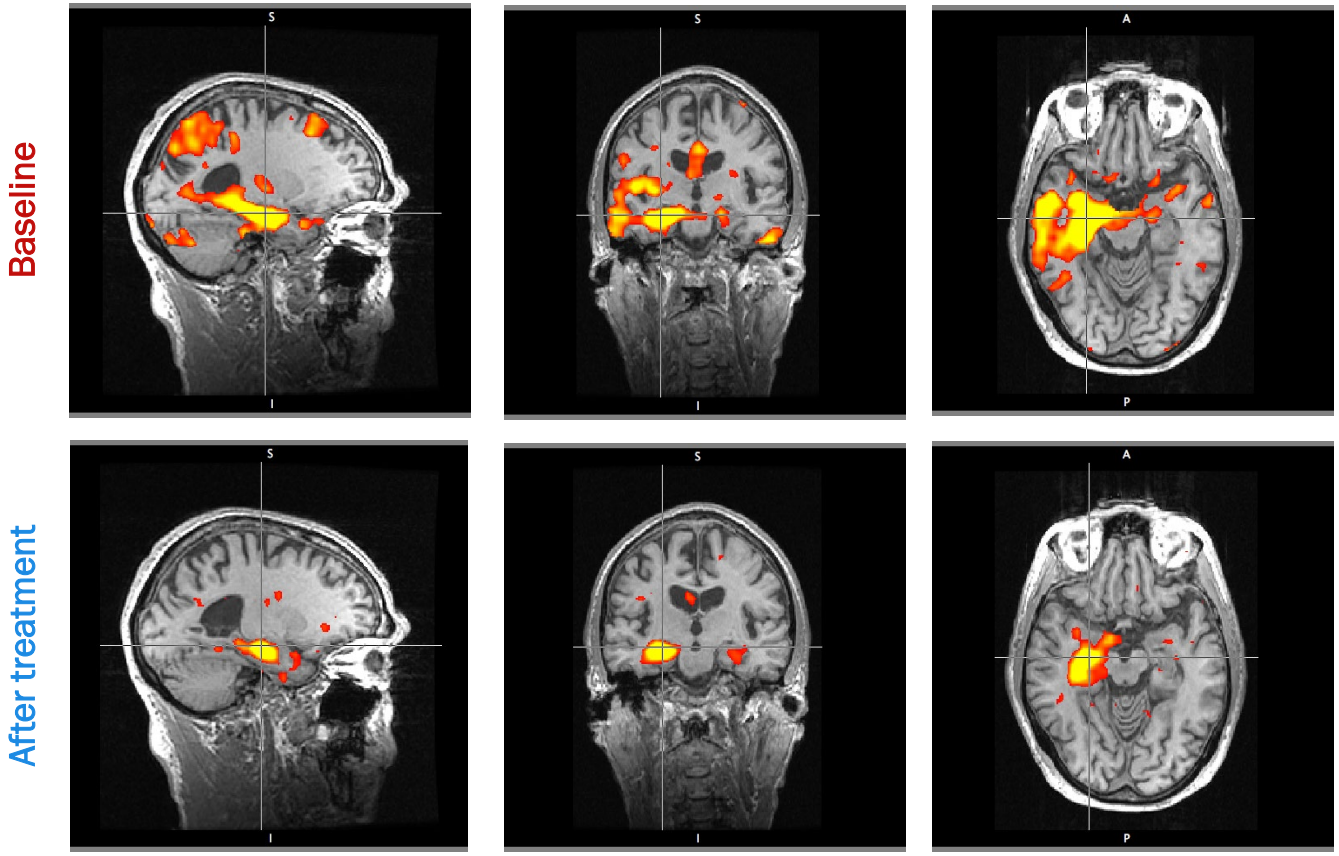
Mean Absolute Change = -1.66

(p=0.0343)

Mean % Change = -4.93% (p=0.0852)

Blood Oxygen Level-Dependent (BOLD) imaging shows NE3107 can reduce hyperactivation of the hippocampus[1] towards normal

Patient N08 – Global Rating of Change +3.5 (Partner Reported)



BOLD imaging looks at the release of oxygen from blood to the tissues and characterizes network connectivity

Image coloration indicates hyperactivation by stressed cells seeking help

Among patients with MMSE >=20

Left lobe

44% of 17 patients improved while 6% declined

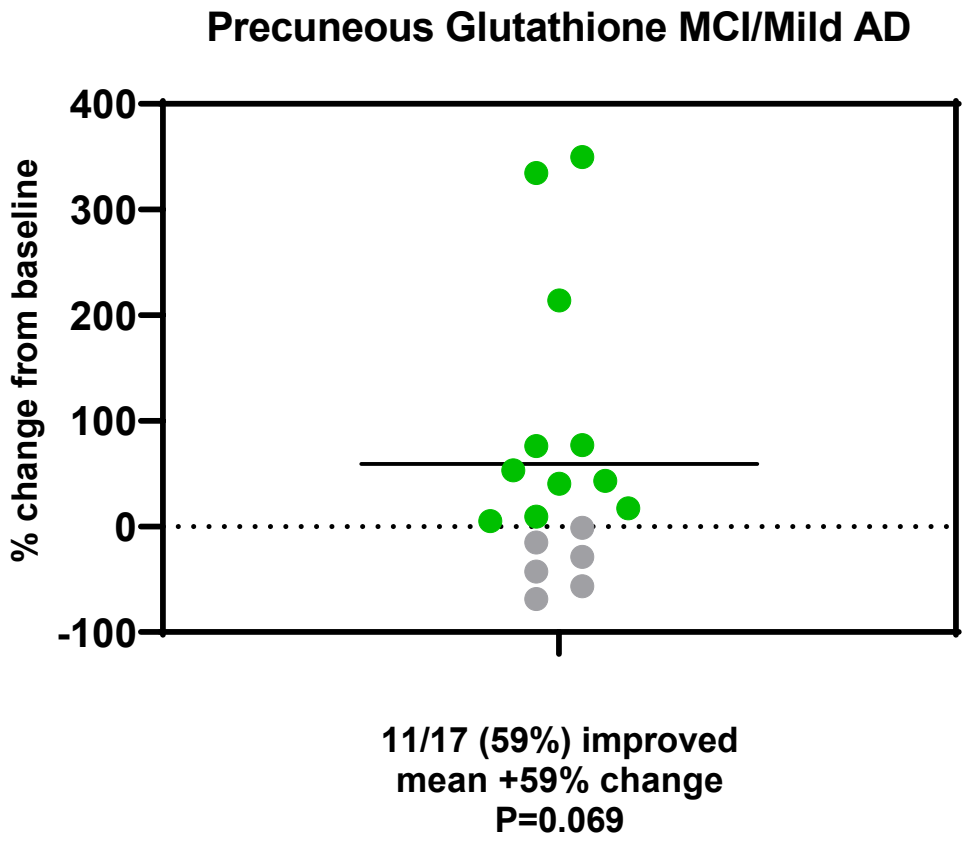
Right lobe

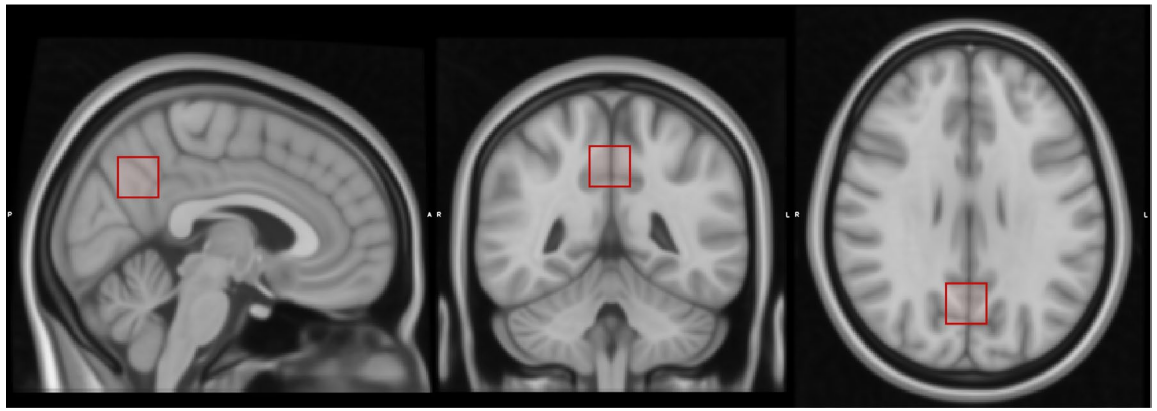
39% of 17 patients improved while 6% declined

1. Aβ-induced change causes hyperactivity in cortical and hippocampal neurons as well as network hypersynchrony. Busche and Konnerth. 2016 Phil. Trans. R. Soc. B 371: 2015042

NE3107 May Be Associated With Reduced Oxidative Stress in the Brain

% change from baseline in brain glutathione assessed by MRS of precuneus





For all patients, there were significant correlations between glutathione and TNF-α (r=−0.44) and glutathione and ADAS-Cog12 (r=−0.45)

Current understanding provides optimism for the Phase 3 trial in Mild to Moderate Alzheimer's expected to read out in Q4 2023

A Phase 3, Double-blind, Randomized, Placebo-controlled, Parallel Group, Multicenter Study of NE3107 in 316 to 400 Patients who have Mild to Moderate AD

- Pivotal study for Alzheimer's disease. Two weeks each of 5 mg and 10 mg BID dose titration followed by 26 weeks of 20 mg twice daily vs. placebo, approximately 160 subjects in each arm, 80% power
- Diagnosed with AD and without evidence of a vascular contribution. Mild to moderate disease. CDR 1-2. MMSE 14-24.
- 60-85 years old, males and females
- Randomization stratified by MMSE and Homeostatic Model Assessment 2 Insulin Resistance (HOMA2)

- **Co-primary endpoints**
  - Mean change from Baseline to Week 30 in the twelve-question form of the Alzheimer's Disease Assessment Scale-Cognitive (ADAS-Cog 12) comparing the NE3107 group to the placebo group
  - Mean change from Baseline to Week 30 in Alzheimer's Disease Cooperative Study-Clinical Global Impression of Change (ADCS-CGIC) comparing the NE3107 group to the placebo group
- **Secondary endpoints**
  - ADCS-ADL (functional), ADCOMS (4 Alzheimer's Disease Assessment Scale–cognitive subscale items, 2 Mini-Mental State Examination items, and all 6 Clinical Dementia Rating-Sum of Boxes items), NPI-12 (care-giver rating of behavioral changes), MMSE, CDR
  - Glycemic control: HOMA2, Mean Amplitude of Glycemic Excursion (MAGE) using continuous glucose monitoring, fructosamine levels, post-prandial glucose and fasting blood glucose vs time.
  - MRI total hippocampus volume change, baseline to end of treatment in a subset of approximately 50% of active and placebo subjects
  - Target engagement assessed in a small subset of active and placebo subjects using PET to quantify cortical glucose utilization

NE3107
in Parkinson's Disease

Parkinson's Disease Clinical Development Program

NM201 Phase 2

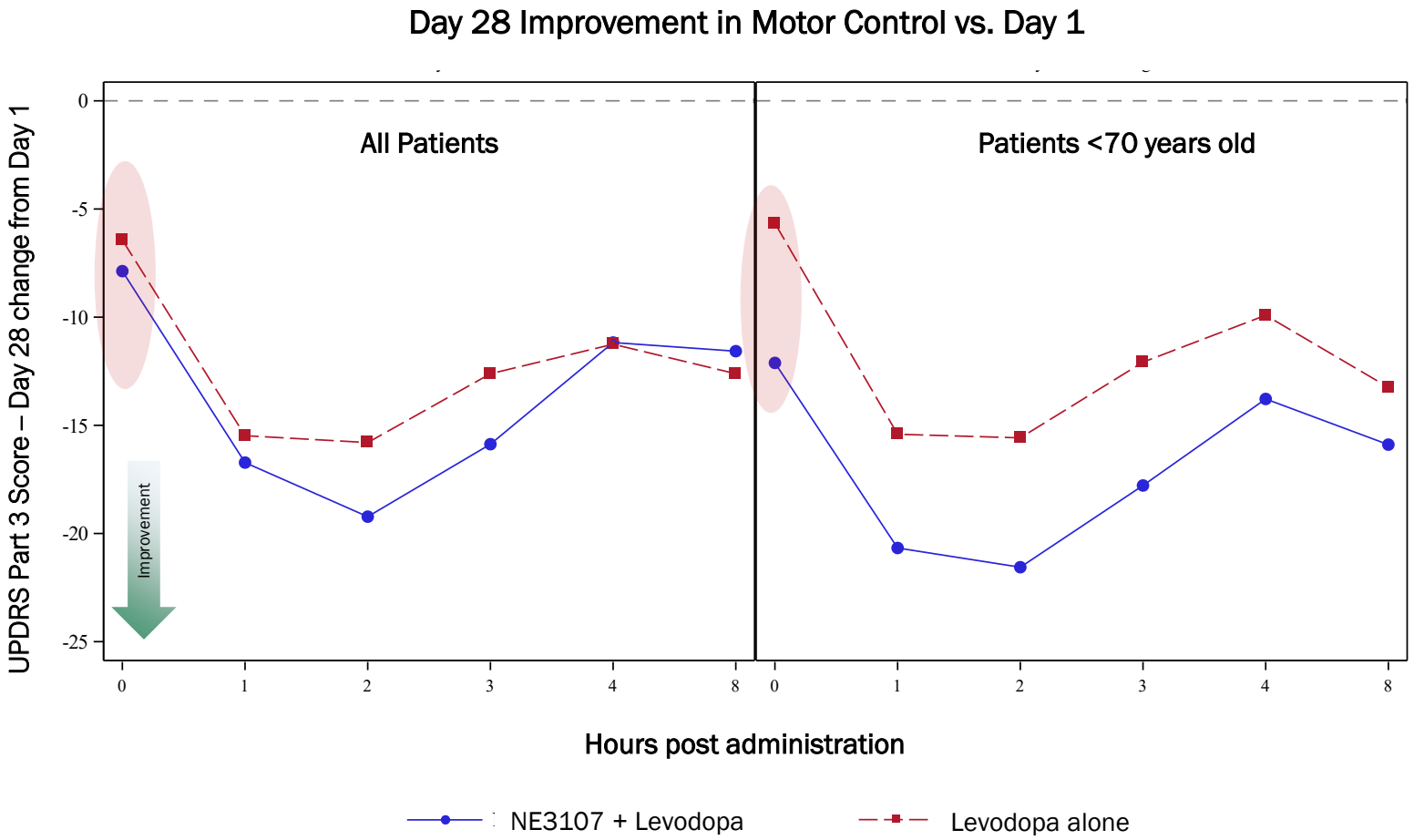
- Satisfying FDA requirement for drug-drug interaction study with L-dopa
- Detect efficacy signal for NE3107's pro-motoric activity

40 patients with defined L-dopa "off state", 1:1 active: placebo, 20 mg BID for 28 days

- **Safety assessments**: Standard measures of patient health, L-dopa PK and DDI
- **Efficacy assessments:** MDS-UPDRS* parts 1-3, Hauser ON/OFF Diary, Non-Motor Symptom Scale

* Motor Disease Society Unified Parkinson's Disease Rating

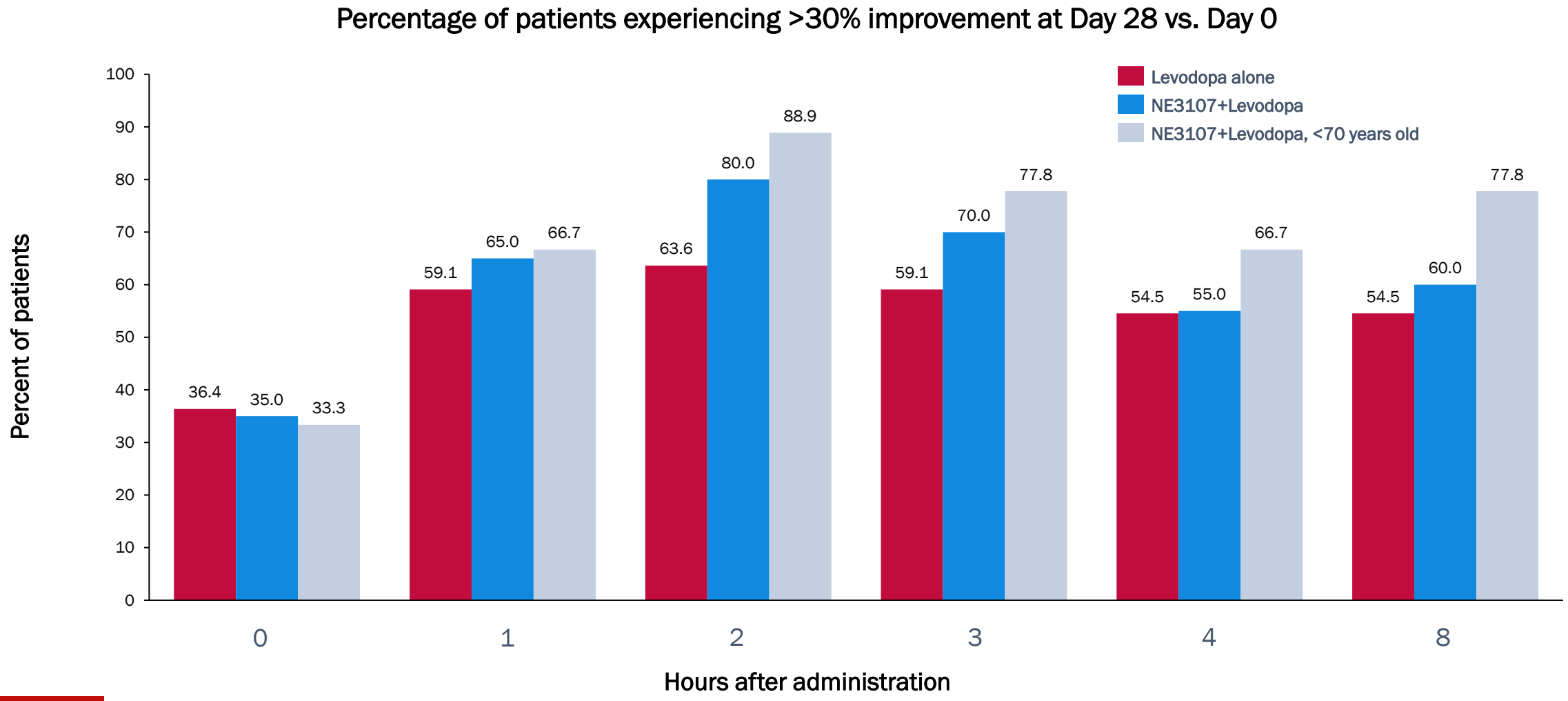
NE3107-treatment patients experienced fewer motor symptoms before morning drug administration



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

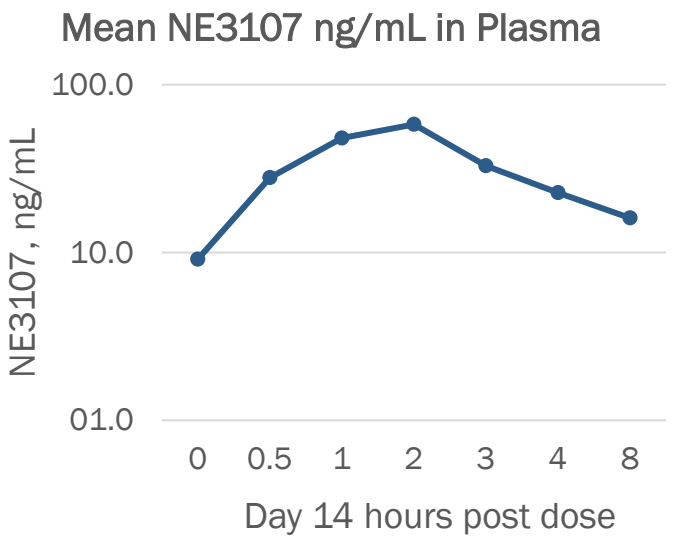
* Fisher's exact test

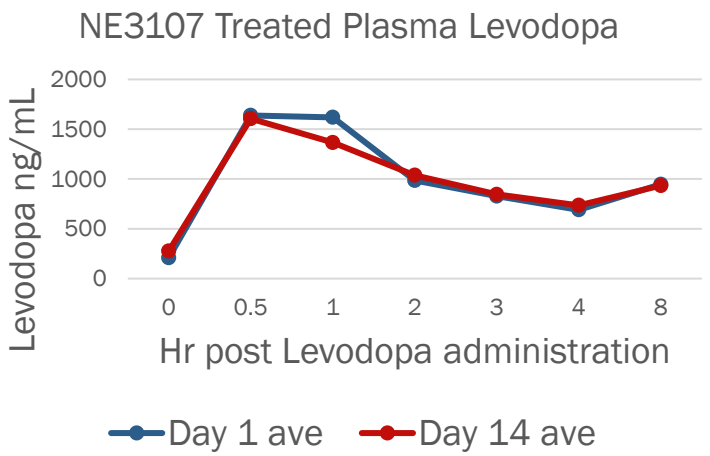
Larger proportion of patients treated with NE3107 had >30% improvements in motor control

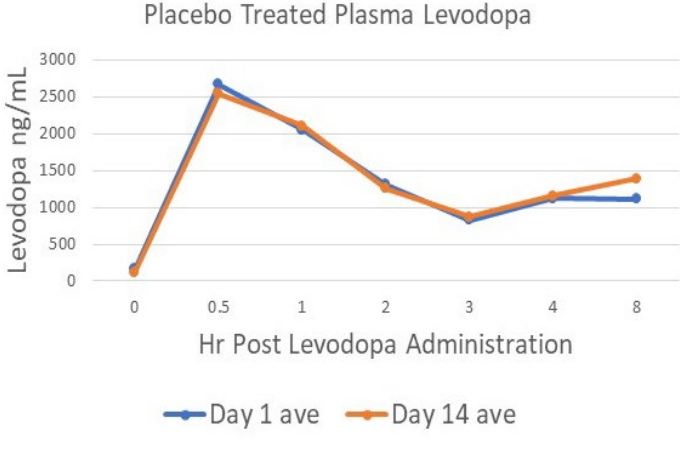


Desirable pharmacokinetics – no observed DDI

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

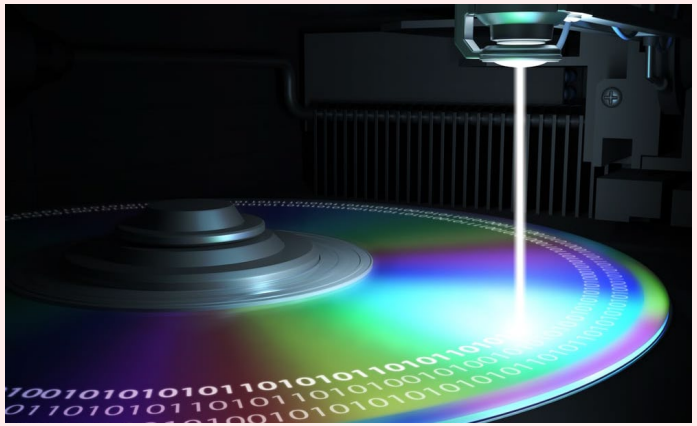






NE3107
in Longevity

Clean DVD leads to clear picture and sound



Scratches & smudges lead to skips and blurs

Impact of wear & tear on a laser's ability to decode DVDs

Quality of picture is dependent on the laser's ability to clearly decode the disk …

The same thing happens in our body

DNA methylation

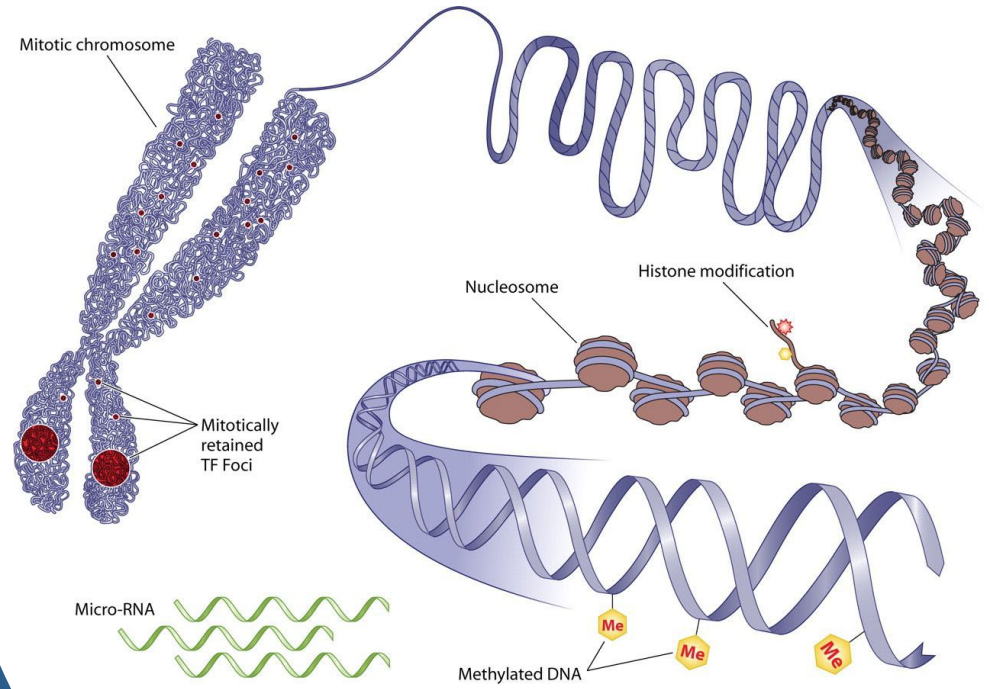
DNA methylation happens when methyl groups are added to our DNA

- DNA methyltransferases add methyl groups to DNA
- Functionally the equivalent of scratches and smudges on a DVD surface
- The methyl groups interfere with RNA polymerase's ability to decode DNA

DNA methylation may happen where a cytosine is positioned next to guanine and is separated by a phosphate group (CpG)

- 28 million CpGs in genome

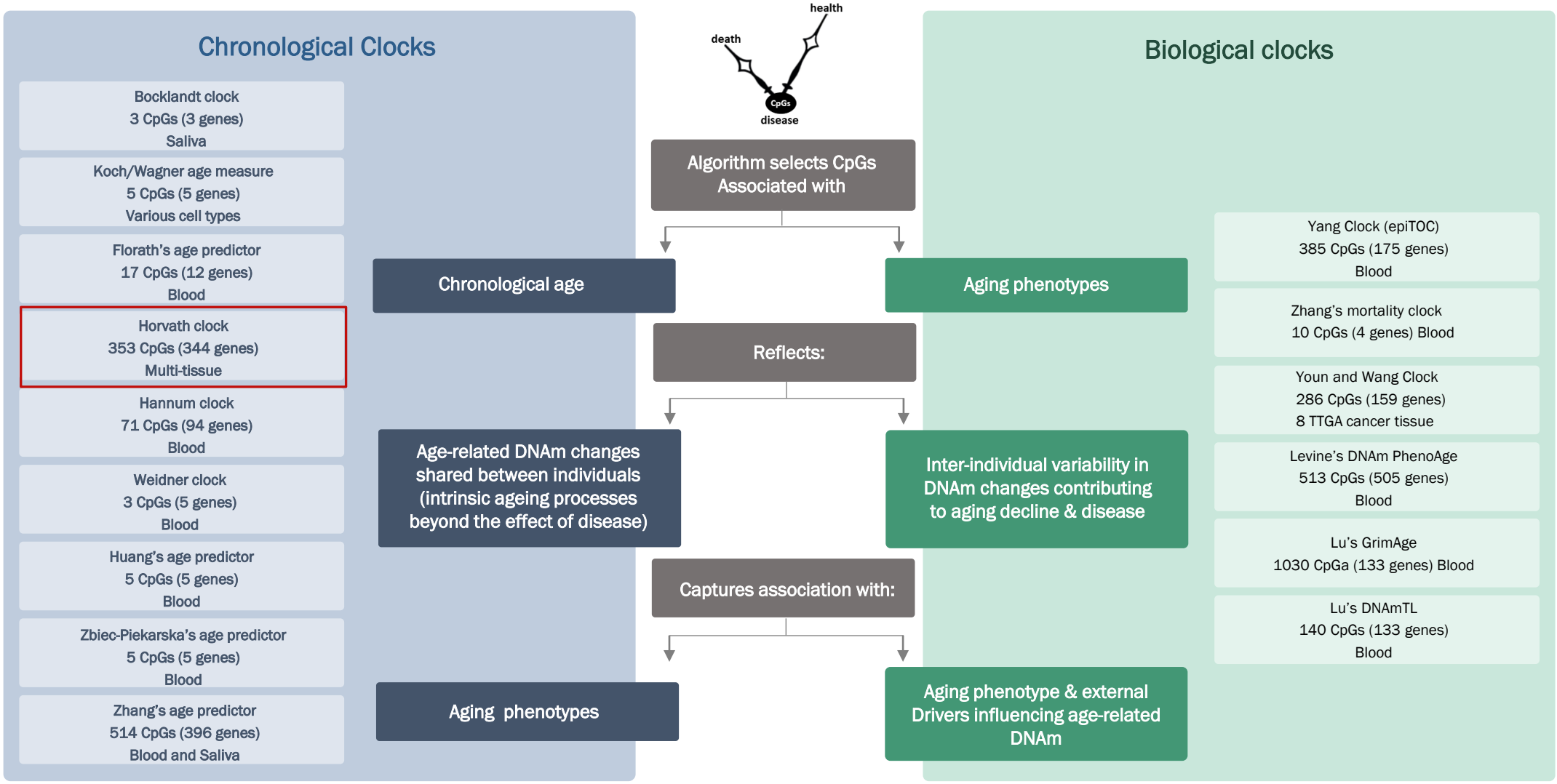
Hypermethylation of DNA is associated with many disease conditions



Observations about DNA methylation

- DNA methylation increases as we age
- DNA methylation can be affected by behavioral (diet, exercise) and environmental factors
- DNA hypermethylation is associated with a large number of disease conditions, including various forms of cancers, age-related cognitive impairment and dementia, Parkinson's disease, cardiovascular disease, COPD and respiratory disease, chronic kidney disease, inflammatory bowel disease, sepsis, and many others*
- Inflammation has been shown to be a driver of hypermethylation of DNA**
- Extent of DNA methylation can be measured by various "clocks"

DNA methylation “clocks” measure extent of aging and biological function

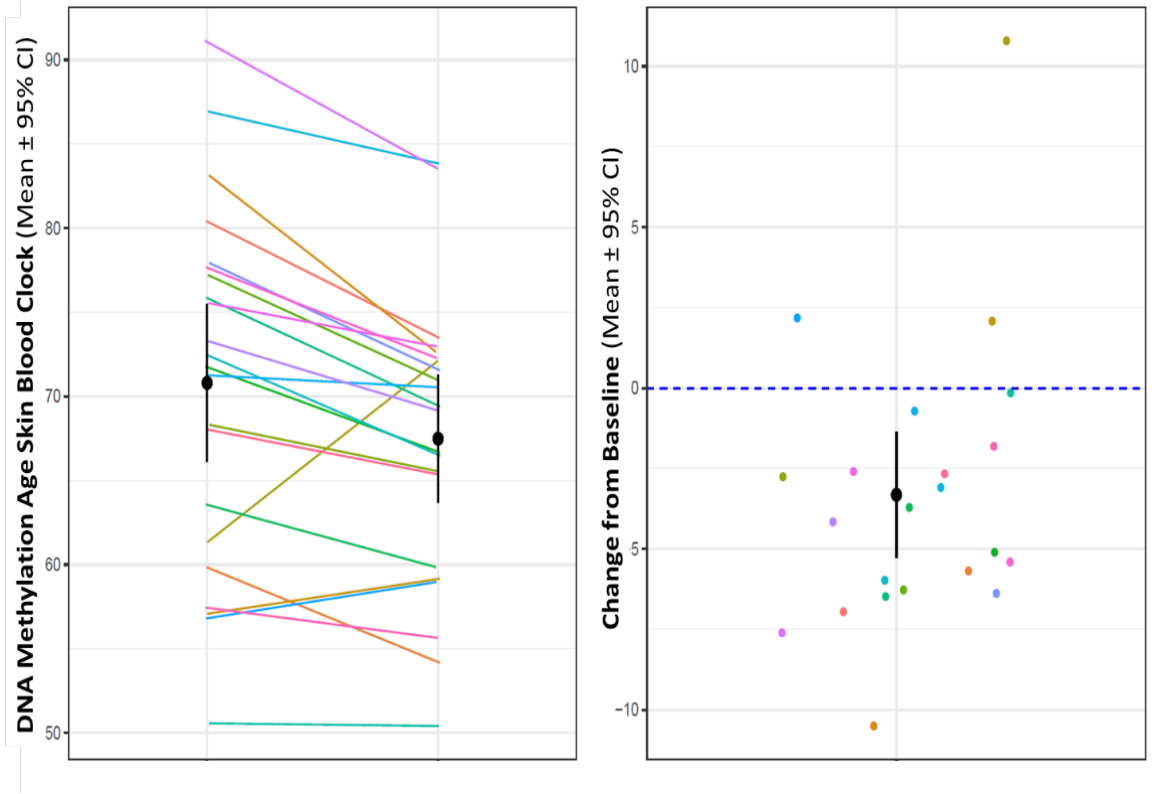


NE3107 significantly reduced DNA methylation as measured by the SkinBloodAge Clock

Dr. Steve Horvath* developed an extremely precise Biological age DNA methylation clock, the DNAmethylation SkinBloodAge.

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%)

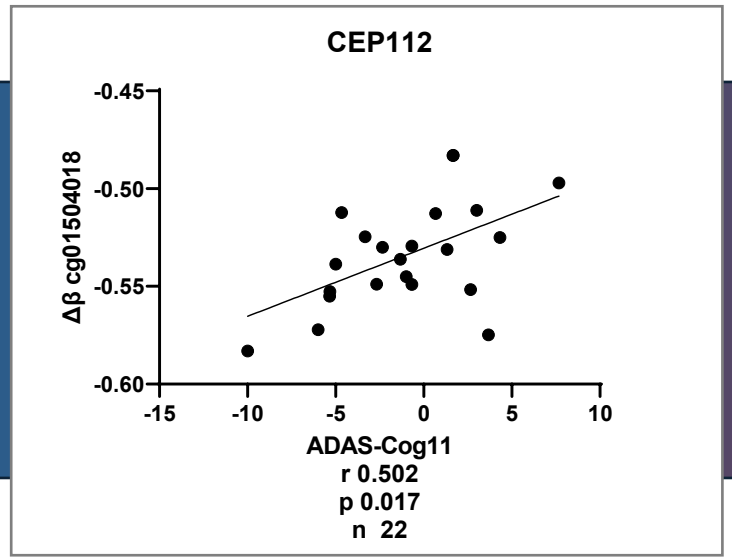


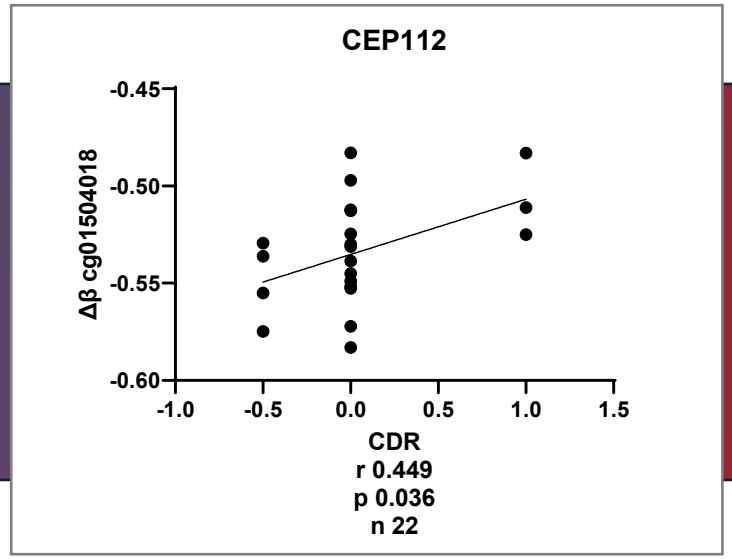
19/22 decreased (86%)

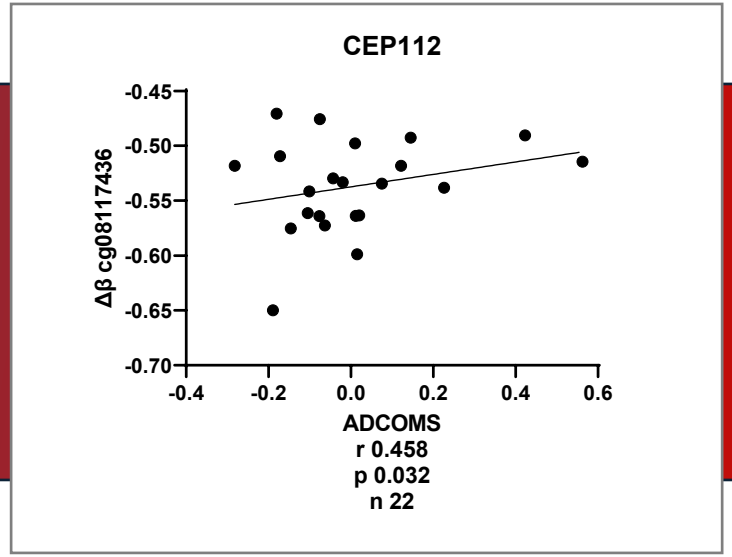
Mean Absolute Change = -3.3 years (p=0.0021)

* Professor of Human Genetics at the UCLA David Geffen School of Medicine and Professor of Human Genetics & Biostatistics at the UCLA Field School of Public Health

Lower DNA methylation of the CEP112 gene significantly correlated with measures of cognition







- CEP112 encodes a coiled-coil domain containing protein that belongs to the cell division control protein 42 effector protein family. In neurons, it localizes to the cytoplasm of dendrites and is also enriched in the nucleus where it interacts with the RNA polymerase III transcriptional repressor Maf1 to regulate gamma-aminobutyric acid A receptor surface expression.
- CEP112 was identified as a hub gene expressed in control compared to Alzheimer's disease in modeling of cognitive reserve.* It is thought to be important in the maintenance of cognitive reserve, and its is decreased in AD.
- Decreasing DNA methylation of CEP112 may result in increased expression, and this would be consistent with the correlations of CEP112 DNAm and ADAS-Cog11, CDR and ADCOMS scores.

* Zhang 2020 Alzheimers Res Ther 12 165

>3,000 correlations between reductions in DNAm of various CpGs and cognitive, biomarker and neuroimaging endpoints

Frequency of significant Spearman correlations between changes in DNAm (individual CpG residues) and clinical measures after 14 weeks of treatment

Clinical measure	Insulin signaling[a]	Anti-oxidant[b]	Anti-inflammatory[c]	Anti-apoptotic[d]	Anti-amyloid[e]	Neuro-stimulatory[f]
MRI neuroimaging						
Hippocampus	16	24	9	4	3	1
Grey matter[g]	91	78	24	24	22	26
Frontal lobe	238	152	44	49	36	69
Temporal lobe	181	112	26	28	13	49
Parietal lobe	91	154	40	47	14	74
Occipital lobe	42	28	9	2	2	5
Glutathione	67	43	27	14	8	20
Cognitive assessments						
CDR	15	23	13	5	5	10
MMSE	31	29	18	7	3	5
ADAS-Cog11	26	16	12	4	9	8
ADCOMS	13	17	4	3	5	8
MoCA	23	8	8	2	2	6
QDRS	10	19	6	3	1	8
PDQ-9	71	49	22	6	17	18
Biomarkers						
pTau	22	26	6	6	3	12
pTau/Aβ42	46	43	19	6	5	15
Aβ42	11	24	9	7	1	15
Tau	34	38	10	2	2	15
TNF-α	23	17	14	7	8	8

aINS, INSR, IGF, IGFR, IRS, AKT, and PI3K; bGST, GPX, GSS, GSR, AATF, ARSB, ATM, NEDD4, ZMAT, TXN, TXNRD, and PRDX; cIL6, IL4, IL10, IL13, NFKBIB, SIRT, and LRRFIP1; dIAP and BIRC; eSORL1, PIGK, UBA1, and ZNF331; f NEUROD1, BDNF, NGF, NTRK, NTF3, and CEP112; Subcortical grey matter

Summary of findings

01

We believe the data suggest that observed clinical findings and measured changes in DNA methylation and biomarkers are not accidental

02

Data show that patients treated with NE3107 experienced reduced DNA methylation

03

Data also show that NE3107 may have changed the expression of specific genes in a manner that is significantly correlated to observed cognitive and biomarker changes

BIV201
in Ascites

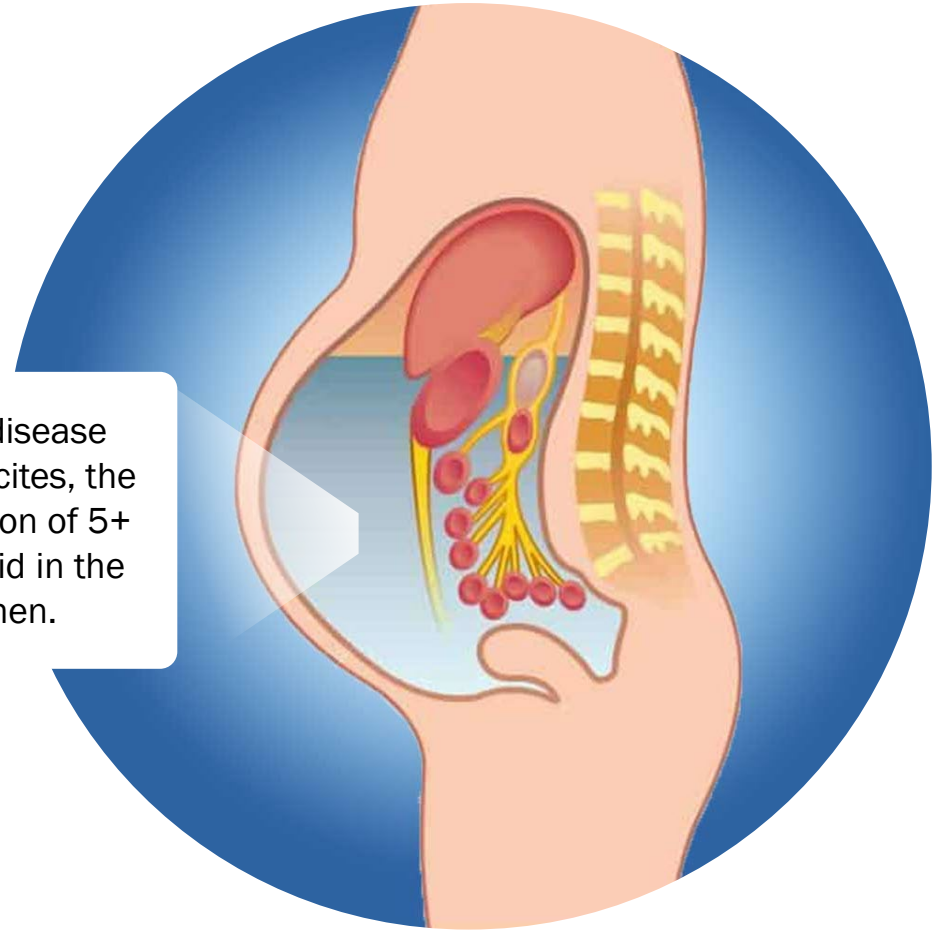
BIV201 Disease Target: Refractory Ascites

Refractory ascites patients typically undergo **paracentesis** to remove ascites fluid every week to 10 days

Paracentesis:

- Withdrawal of 5–10L of ascites fluid (on average) from abdomen using a large bore needle
- Provides a few days of symptomatic relief
- The kidneys are “burning out” by retaining massive quantities of salt and water
- Patients suffer frequent life-threatening complications
- No remaining options except for TIPS[1] surgery or liver transplant
- Estimated $670 million addressable US market with 20,000[2] targeted patients

No drugs ever approved by FDA to treat ascites



Our first disease target is ascites, the accumulation of 5+ liters of fluid in the abdomen.

1. TIPS = transjugular intrahepatic portosystemic shunt to channel blood flow around the liver
2. Derived from Scaglione J Clin Gastroenterol.49(8):690-6; D'Amico Journal of Hepatology, Volume 44, pp. 217-231; D'Amico Aliment Pharmacol Ther. 39(10):1180-93; Samonakis World Journal of Hepatology, 6(7), pp. 504-512; Sivanathan Dtsch Med Wochenschr, Volume 139, pp. 1758-1762 and Gines New England Journal of Medicine, 350(16), pp. 1646-1654.

Prefilled Syringe with Patent-pending Liquid Formulation

BIV201*	Our liquid formulation of terlipressin. We will seek patent protection in the US, Europe, China and Japan
Accurate dosing	Eliminates mixing minute quantities of powder terlipressin that could result in medication errors or sterility loss
Enhanced convenience	Simply inject fluid into the saline bag and attach to pump

BIV201 Prefilled Syringe
Stable for 18+months at room temp.

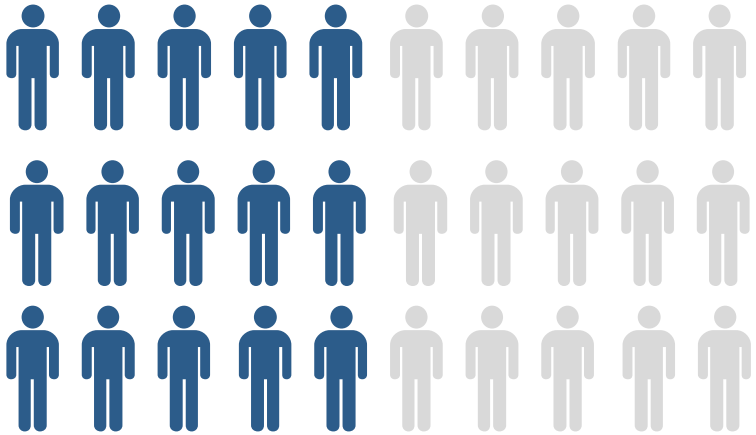
Needle or Connector

50 mL bag of saline for insertion into pump

Portable pump
Carried in small satchel

*In clinical trials; not approved by FDA

BIV201 Phase 2b trial

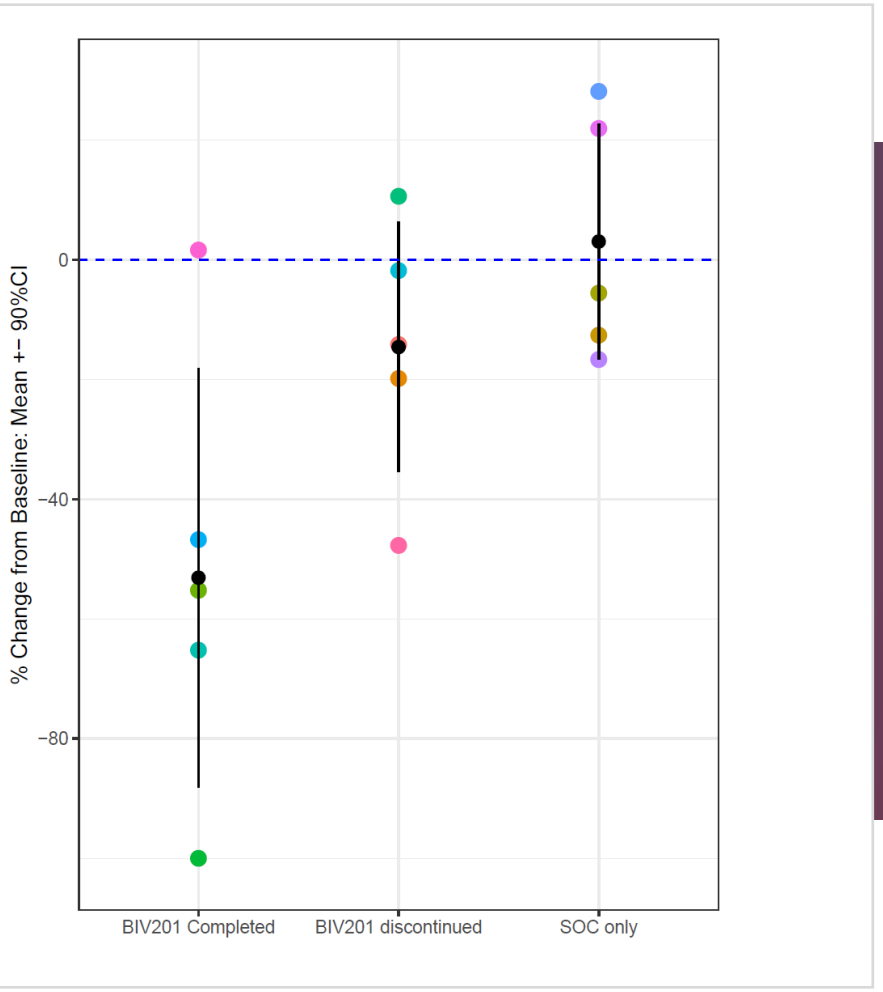


Originally targeted 30 patients randomized 2:1

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

Change in ascites volume 28d pre- vs post-treatment



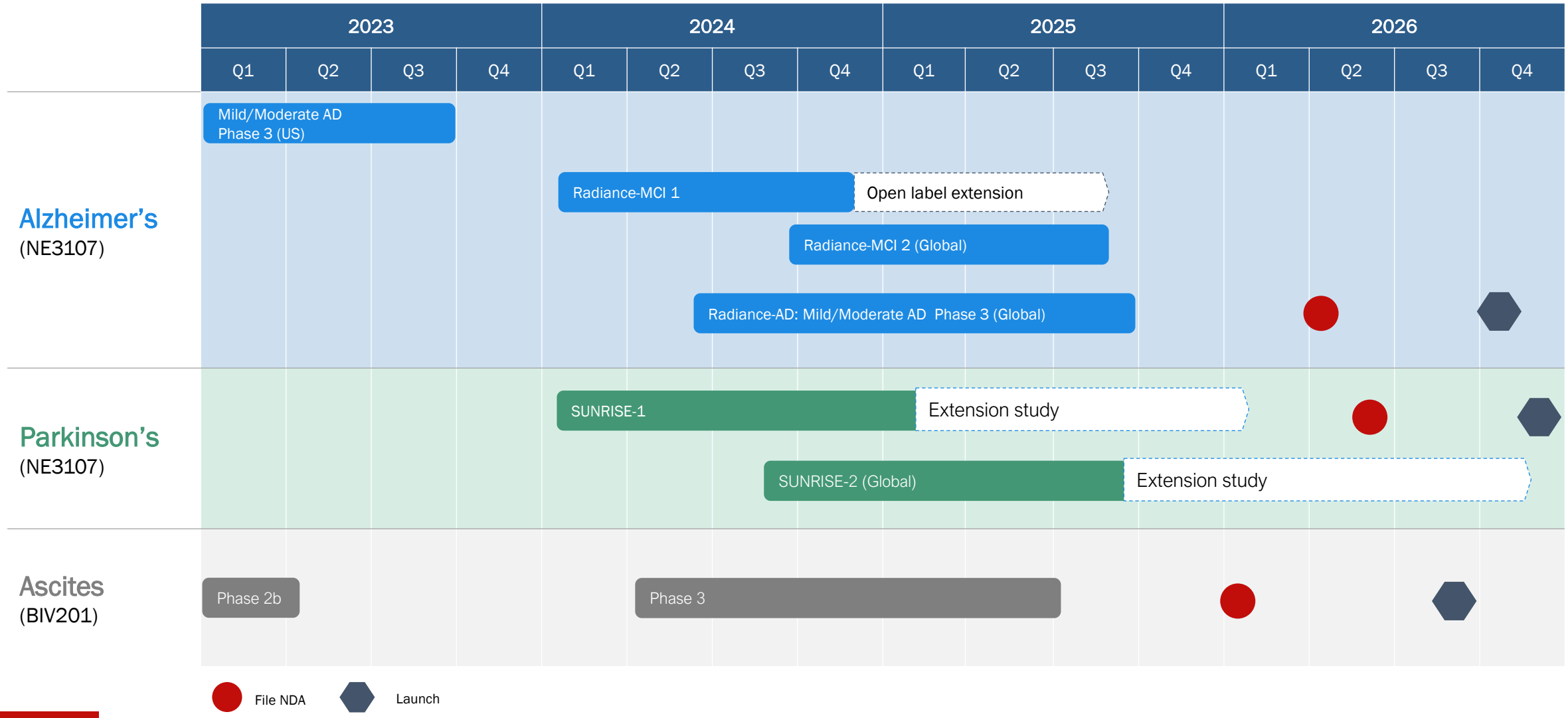
53% reduction in ascites volume among patient completing BIV201 treatment

15% reduction among patients who started but did not complete treatment

3.1% increase for SOC patients

p<0.001

Expected catalysts and anticipated timelines



Commercial potential in US market alone*

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% market penetration

$30K/year

Ascites

$1.6B

US peak sales

45% market penetration

$45K/year

2026 launch

2032 peak sales

Leadership Team

Deep expertise provides a strong foundation for success

Cuong Do, President & Chief Executive Officer
30+ years in biopharma & technology
President, Samsung Global Strategy Group
Chief Strategy Officer for Merck, Senior partner at McKinsey & Company

Chris Reading, PhD, Neurodegenerative Disease Program
40+ years in biopharma
Chief Scientific Officer, Hollis-Eden Pharmaceuticals
VP of Product and Process Dev. for Systemix
U Texas Dept. of Tumor Biology

David Morse, Chief Regulatory Officer
35 years experience Regulatory Affairs and multi-region product development strategy
Former VP with two top-5 international CRO's
Former Associate Director CDER, FDA

Sarah Hoit, Chief Social Impact Officer
30+ years in Social Impact, healthcare and technology
CEO & Co-Founder for Connected Living, Inc
CEO & Founder for Explore, Inc
Deputy Director of AmeriCorps in White House

Joseph Palumbo, MD, Chief Medical Officer
30+ years treating patients; 25+ years in biopharma
CMO, Zynerba
Global Head of Medical Science & Translational Research, Global Head & Psychiatry Franchise Medical Leader, J&J

Penelope Markham, Liver Cirrhosis Program
25 years in biopharma drug development
Lead Scientist Terlipressin (LATPharma/ BioVie 11 years)
Head Research Biology Protez Pharma
Co-founder/Director of Research Influx Inc.

Clarence Ahlem , Operations
35+ years in biopharma
Vice President, Product Development Harbor Therapeutics
Director, Product Development, Hollis-Eden Pharmaceuticals
US San Diego

J. Wendy Kim, Chief Financial Officer
35 years in finance/ accounting
As CFO managed corporate finance and operations groups
Closed M&A transactions and secured financings
Combined 22 years at KPMG and BDO LLP

Recap

We believe that TNFα-mediated inflammation

- Initiates and perpetuates a forward-feeding pro-inflammatory cycle
- Leads to insulin resistance
- Accelerates the “DNA methylation” and the aging process

Our lead asset NE3107 modulates the production of TNFα. In clinical trials, many patients treated with NE3107 experienced:

- Reduced inflammation and the associated insulin resistance
- Improved cognition, lowered p-tau levels, and improved brain imaging scans in Alzheimer’s Disease (AD)
- Improved motor control and “morning on” symptoms in Parkinson’s disease (PD)
- Lowered DNA methylation levels

NE3107 may change the expression of specific genes in a manner that is significantly correlated to observed cognitive and biomarker changes

- Provides epigenetic basis to explain improvements observed in AD and PD trials
- Gives optimism for what we may see when Phase 3 AD trial reads out in Q4 2023

BIV201 reduces fluid build up and has the potential to become the first therapeutic for ascites, a condition with 50% mortality rate within 12 months. Discussions with FDA underway to finalize Phase 3 trial design

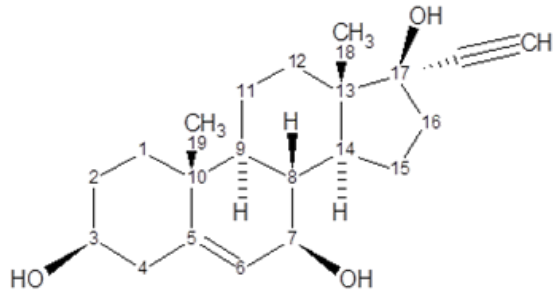
Thank You

Background on NE3107

First-in-class molecule with desirable characteristics

- Small molecule; orally bioavailable
- Crosses blood-brain barrier, thus CNS and peripheral applications
- No safety issues identified to date in pre-clinical and clinical trials (up to Phase 2)

- Originated by Hollis-Eden Pharmaceuticals (renamed Harbor Therapeutics)
  - NE3107[1] is a synthetic analogue of a metabolite of the adrenal hormone DHEA
- Phase 1 and 2 trials in diabetic patients showed that NE3107:
  - Showed no differences in AEs compared to placebo
  - Decreased insulin resistance, postprandial glucose and HbA1c compared to placebo
  - Decreased inflammation-driven systems dysregulation of inflammatory, hematopoietic and metabolic parameters compared to placebo
- Neurmedix eventually bought the assets and hired the lead scientists with the mandate to:
  - Determine NE3107's mechanism of action
  - Get the drug back into the clinic
- NE3107 turns out to have a very unique and unexpected mechanism of action



Prior NE3107 Clinical Studies

Phase 1[1]

Obese, impaired glucose tolerant healthy volunteers

NE3107:

Improved insulin-stimulated glucose disposal, assessed by hyperinsulinemic, euglycemic glucose clamp procedures

Decreased C-reactive protein (CRP[2]) and increased HDL and adiponectin (both associated with benefit in AD)

Showed no differences in AEs compared to placebo

Phase 2[3]

Obese and inflamed type 2 diabetes subjects

NE3107:

Decreased insulin resistance, postprandial glucose and HbA1c compared to placebo

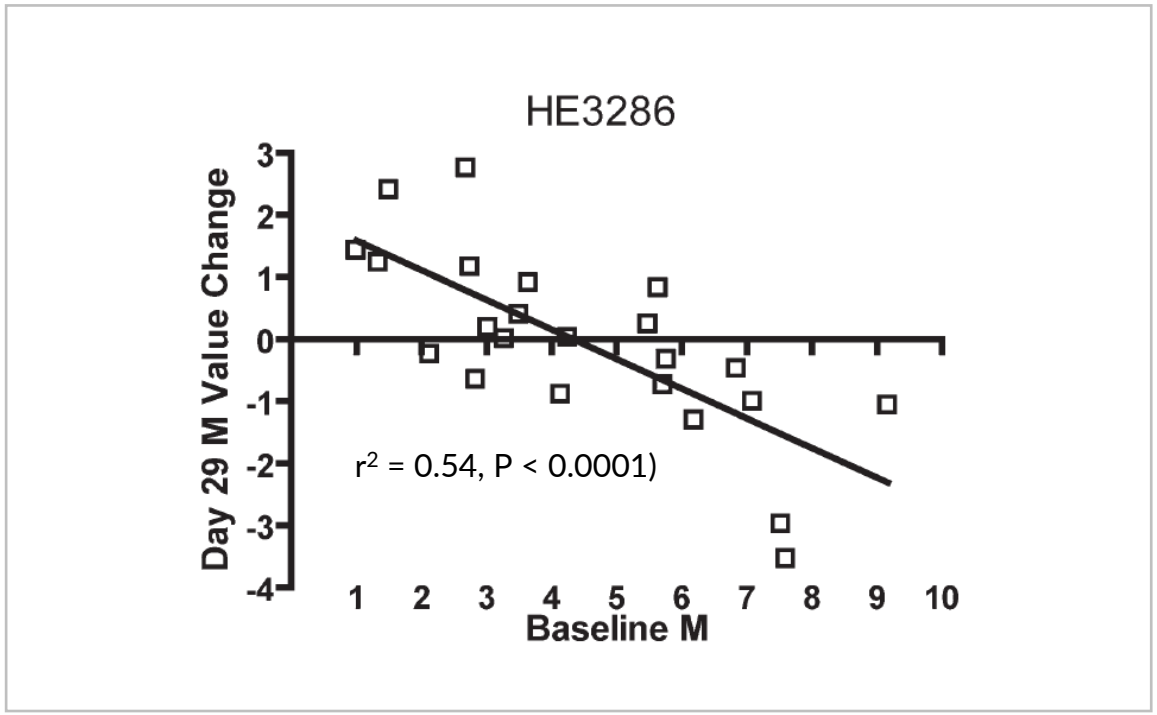
Decreased inflammation-driven systems dysregulation of inflammatory, hematopoietic and metabolic parameters compared to placebo[4]

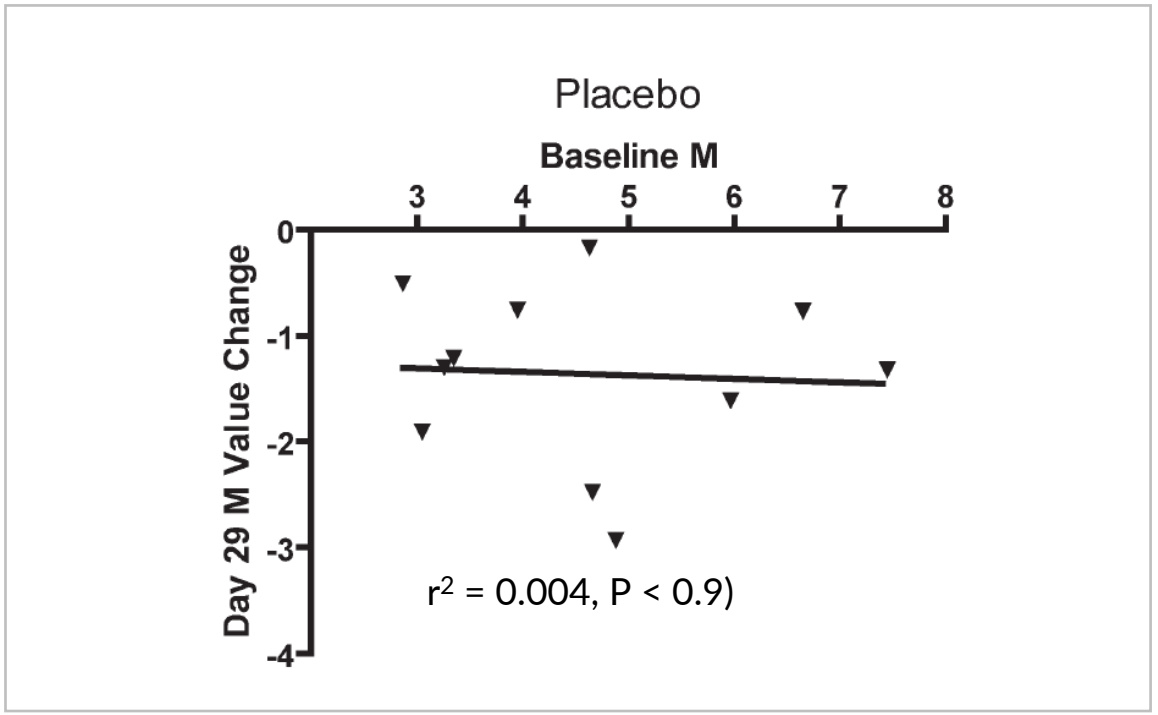
Showed no differences in AEs compared to placebo

1. Reading Mediators Inflamm 2013 814989
2. CRP is a nonspecific marker of inflammation that is increased in the brain and serum of patients with Alzheimer's disease (AD), and has been associated with increased risk of developing dementia
3. Reading 2013 Obesity 21 E343
4. Systems dysregulation in diabetes has been shown to increase risk for AD, and similar systems dysregulation of laboratory and clinical parameters is correlated with AD progression

NE3107's ability to enhance insulin sensitivity reproduced in humans in Phase 1 trial with impaired glucose tolerance patients

Improved insulin-dependent glucose disposal in 48 obese, inflamed subjects





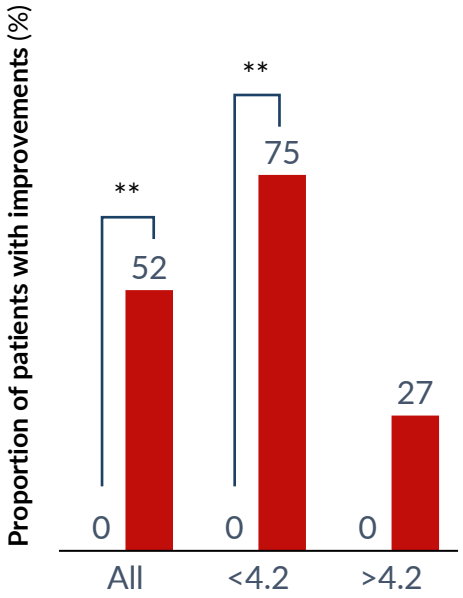
Day 29 Glucose disposal rate (M, measured in mg/kg/min in glucose clamp studies) value change as a function of baseline M

Reading 2013 Obesity 21 E343. Note: NE3107 was originally known as HE3286 from Hollis Eden Pharmaceuticals

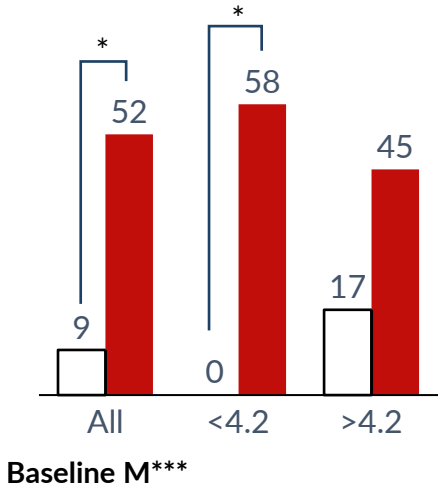
Insulin sensitizing improvement also brought improvements in AD indicators

In a Phase 1 study 48 obese subjects with impaired glucose tolerance, NE3107 …

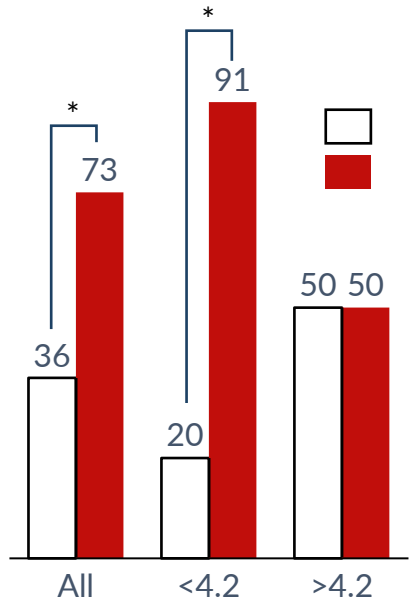
Increased insulin sensitivity …



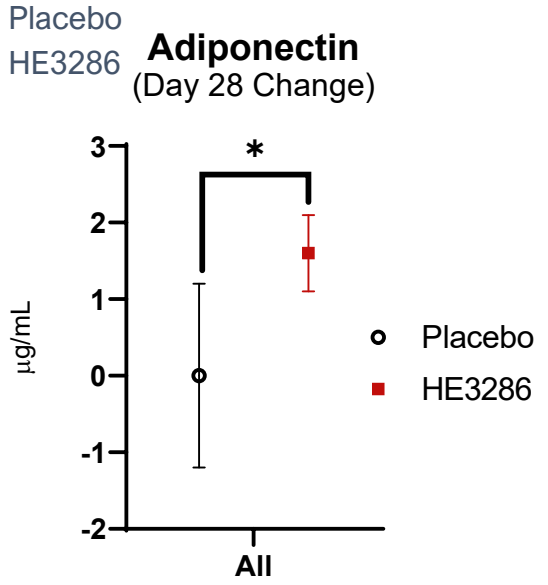
… increased HDL …



… decreased CRP …



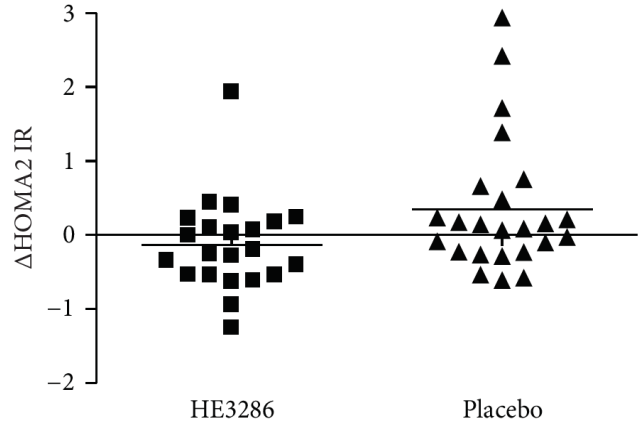
… and increased adiponectin



Fischer's exact test p<0.05 **p<0.01 *** Baseline glucose disposal rate (M) measured in mg/kg/min. Median baseline is 4.2
Source: Reading 2013 Obesity 21 E343. Note: NE3107 was originally known as HE3286 from Hollis Eden Pharmaceuticals

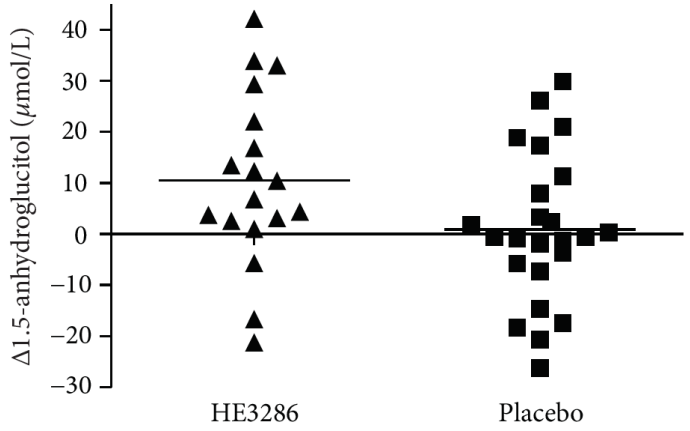
NE3107's ability to enhance insulin sensitivity reproduced in humans in Phase 2 trial with T2D patients

Improved the homeostatic model assessment of insulin resistance …



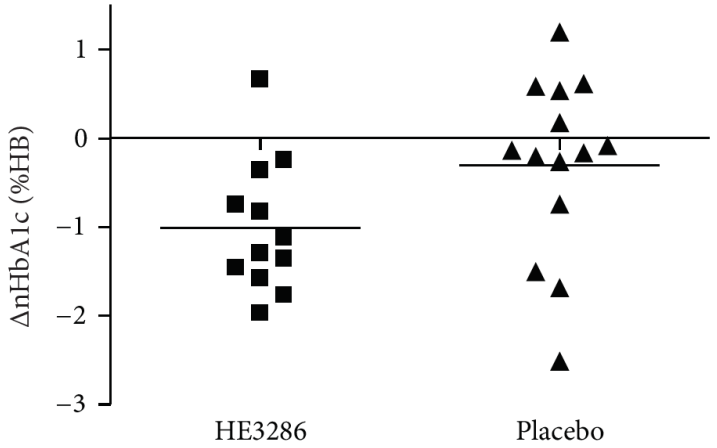
Cohort 1 baseline MCP-1 > 40 pmol/L individual subject changes in homeostatic model assessment of insulin resistance at Day 84

… decreased postprandial glucose …



Pooled cohorts 1 and 2 changes at Day 84

… decreased HbA1c …



Day 112 cohort 2 baseline BMI > 31 kg/m^2

… decreased insulin resistance in inflamed T2D patients

Effect	Value	Change		*P*	Test[g]
		HE3286	Placebo		
ΔHOMA2 IR[c]	Day 84 mean	−0.1	+0.4	0.02	*t*-test

Reading 2013 Mediators Inflamm 814989. Note: NE3107 was originally known as HE3286 from Hollis Eden Pharmaceuticals

Inflammation drives systems dysregulation

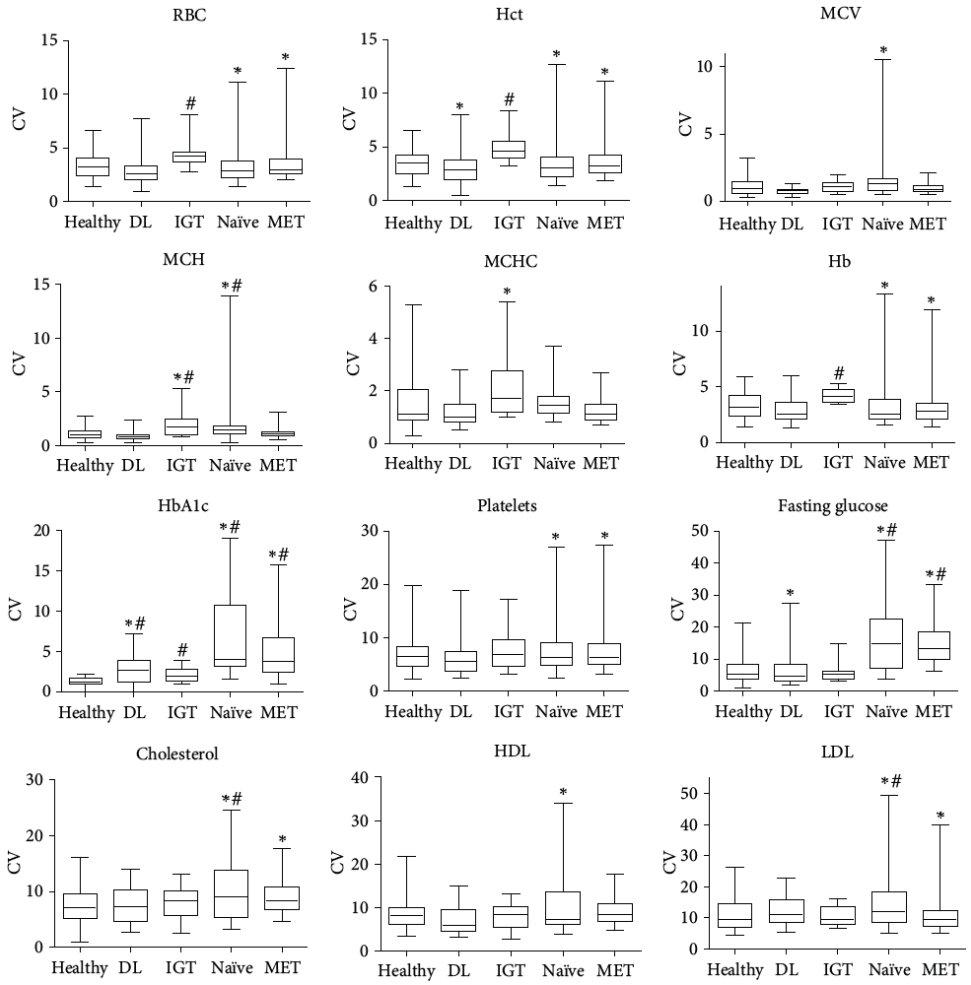
Patients with increasing metabolic disease show greater variability for individual hematology, metabolic and chemistry values

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

Hb: hemoglobin, Hct: hematocrit, RBC: red blood cell count, MCV: mean cell volume, MCH: mean corpuscular hemoglobin, MCHC: mean corpuscular hemoglobin concentration, 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 in advanced T2D patients

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	Cohort 1	MCP-1 > 40	Cohort 2	BMI > 31
HE3286	n=44	n=22	n=35	n=12
Placebo	n=51	n=25	n=34	n=15

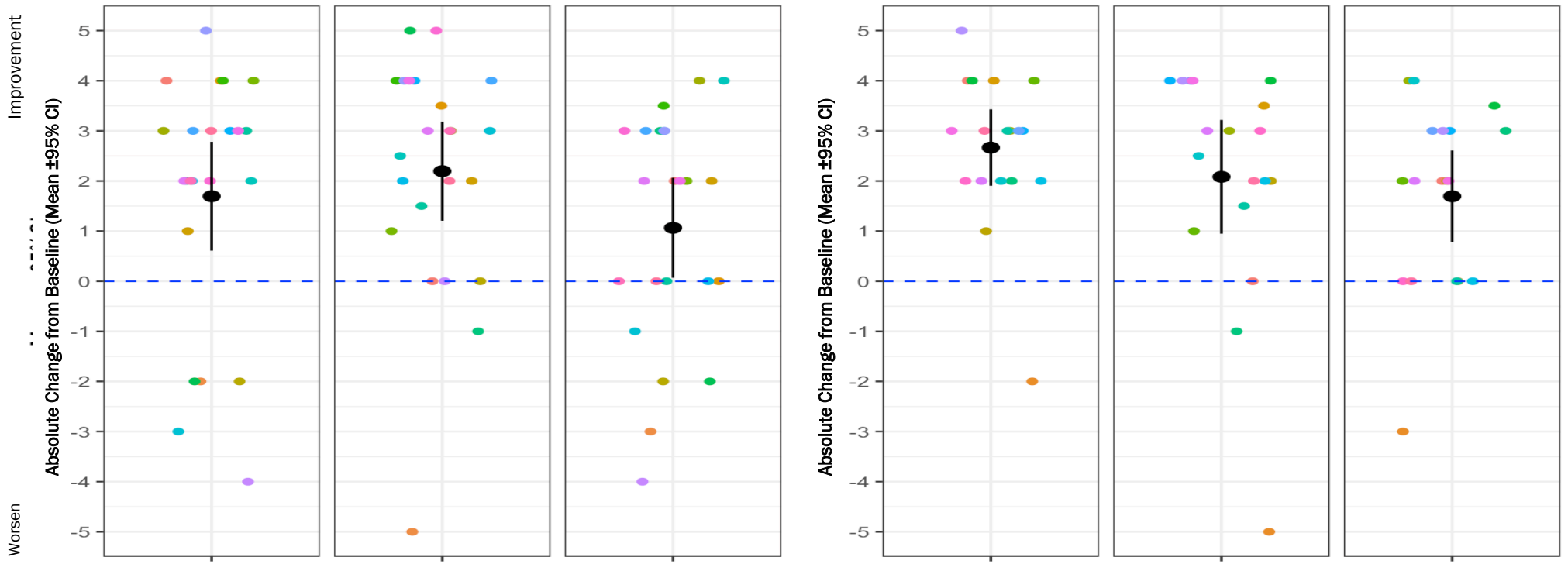
Group	Day	Parameter	HE3286 *W* test *P*	Placebo *W* test *P*
Cohort 1	84	ΔInsulin[d]	>0.1	**<0.0001**
		ΔC-peptide	>0.1	**<0.0001**
		ΔFasting glucose	>0.1	**0.02**
		ΔHOMA2 %B	>0.1	**<0.0001**
		ΔHOMA2 IR	>0.1	**0.002**
		Δleptin	>0.1	**0.005**
Cohort 1 MCP-1 > 40[b]	84	ΔHbA1c	>0.1	**0.006**
		ΔFasting 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**
		ΔTriglycerides	>0.1	**<0.0001**
	112	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

Large majority of patients improved significantly on the Global Rating of Change (overall impression)

All patients

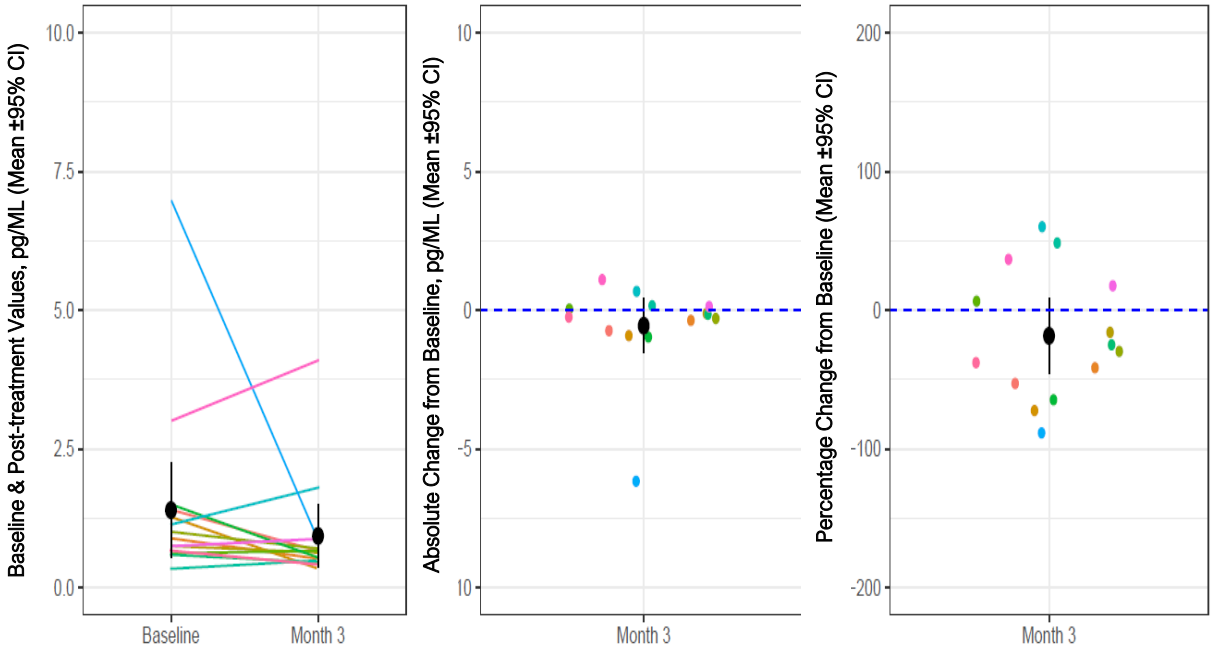
MMSE >=20 patients



Clinician Observation	Patient Reported	Partner Reported	Clinician Observation	Patient Reported	Partner Reported
18/23 (78%)	18/23 (78%)	13/23 (57%)	17/18 (94%)	15/18 (83%)	11/18 (61%)
Mean = +1.70	Mean = +2.22	Mean = +1.07	Mean = +2.67	Mean = +2.08	Mean = +1.69
p=0.0038	p=0.0001	p=0.0375	P<0.0001	p=0.0012	p=0.0011

GRC Scale: +5 = completely recovered; -5 = very much worse

Improvements on TNFa among MCI/Mild AD patients



Tumor Necrosis Factor Alpha (TNFα) is a cytokine identified as a major regulator of inflammation whereby its excessive activation is associated with chronic inflammation[1]

MMSE >=20 patients

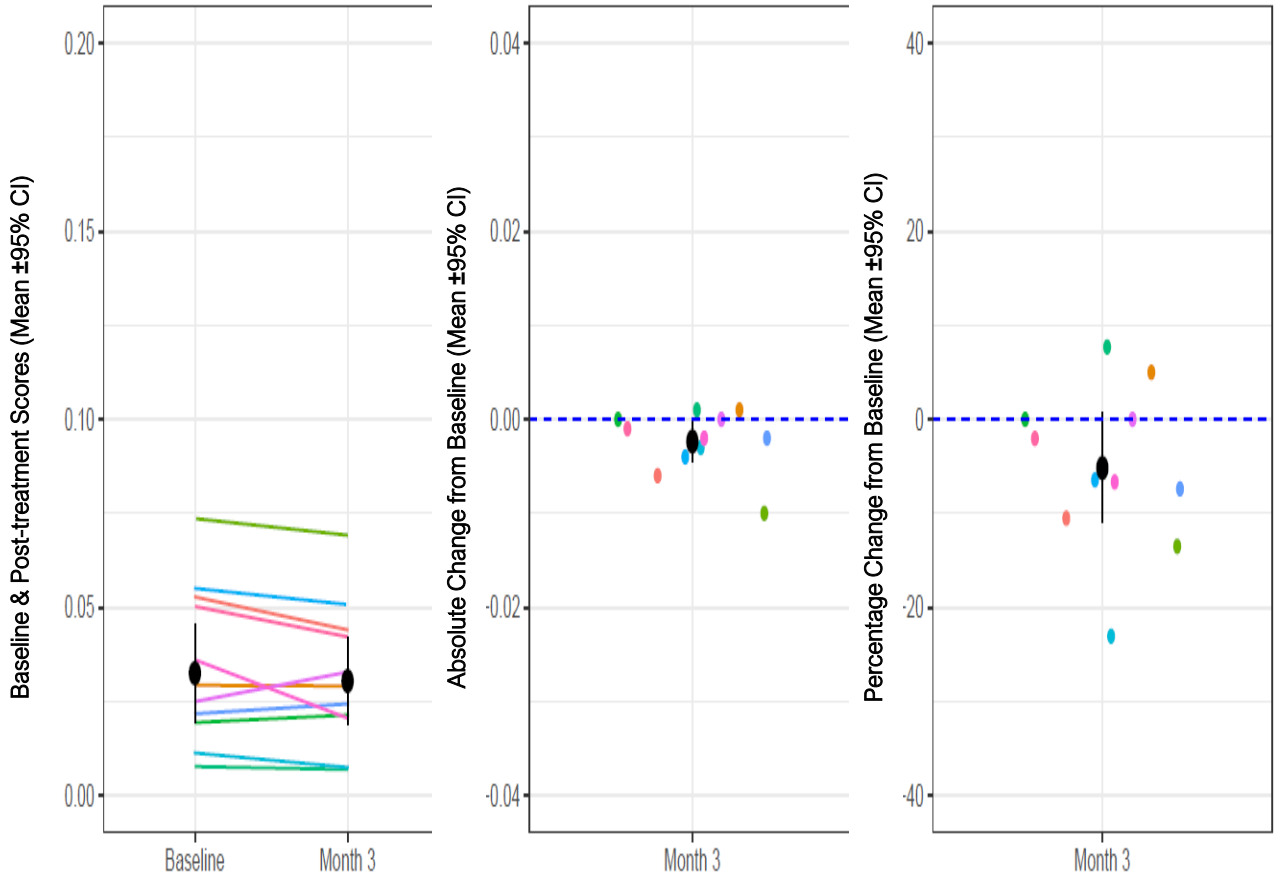
9/14 improved (64%)[2]

Mean Absolute Change = -0.563 (p=ns)

Mean % Change = -18.5% (p=ns)

1. Jang et al. Int J Mol Sci. 2021 Mar; 22(5): 2719.
2. Among responders: Mean Absolute Change = -1.11 (p=ns); Mean % Change = -47.6% (p=0.003)

Significant improvements in the CSF p-tau : Ab42 Ratio, a predictive measure of PET amyloid status[1]...



MMSE >=20 patients

7/11 improved (64%)[2]

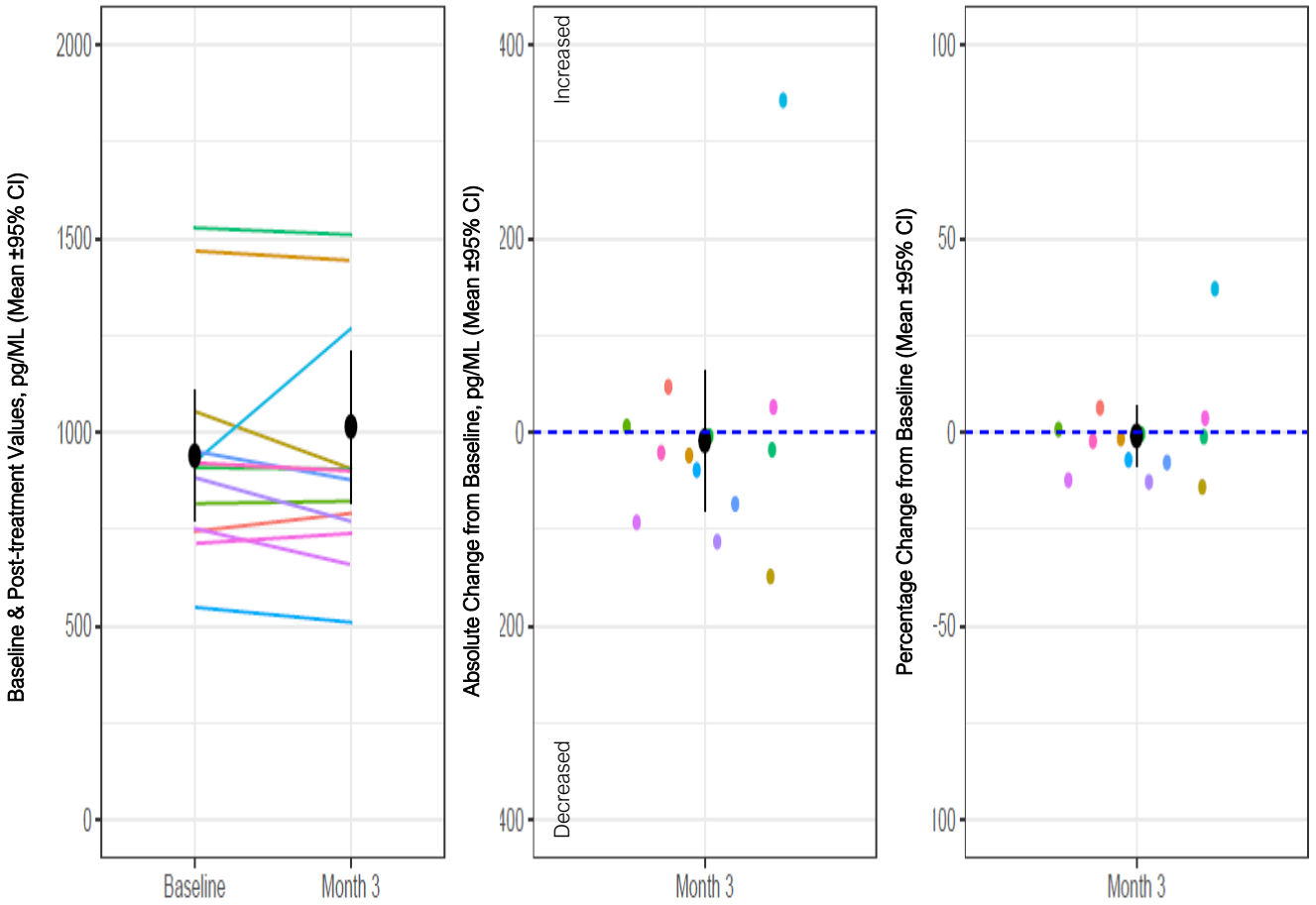
Mean Absolute Change = -0.0024

(p=0.0401)

Mean % Change = -5.18% (p=0.077)

1. Campbell et al. Alzheimers Dement (Amst). 2021; 13(1): e12190
2. Among responders: Mean Absolute Change = -0.0040 (p=0.0144); Mean % Change = -10.0% (p=0.0082)

Modest improvements in CSF Ab_{42}



MMSE >=20 patients

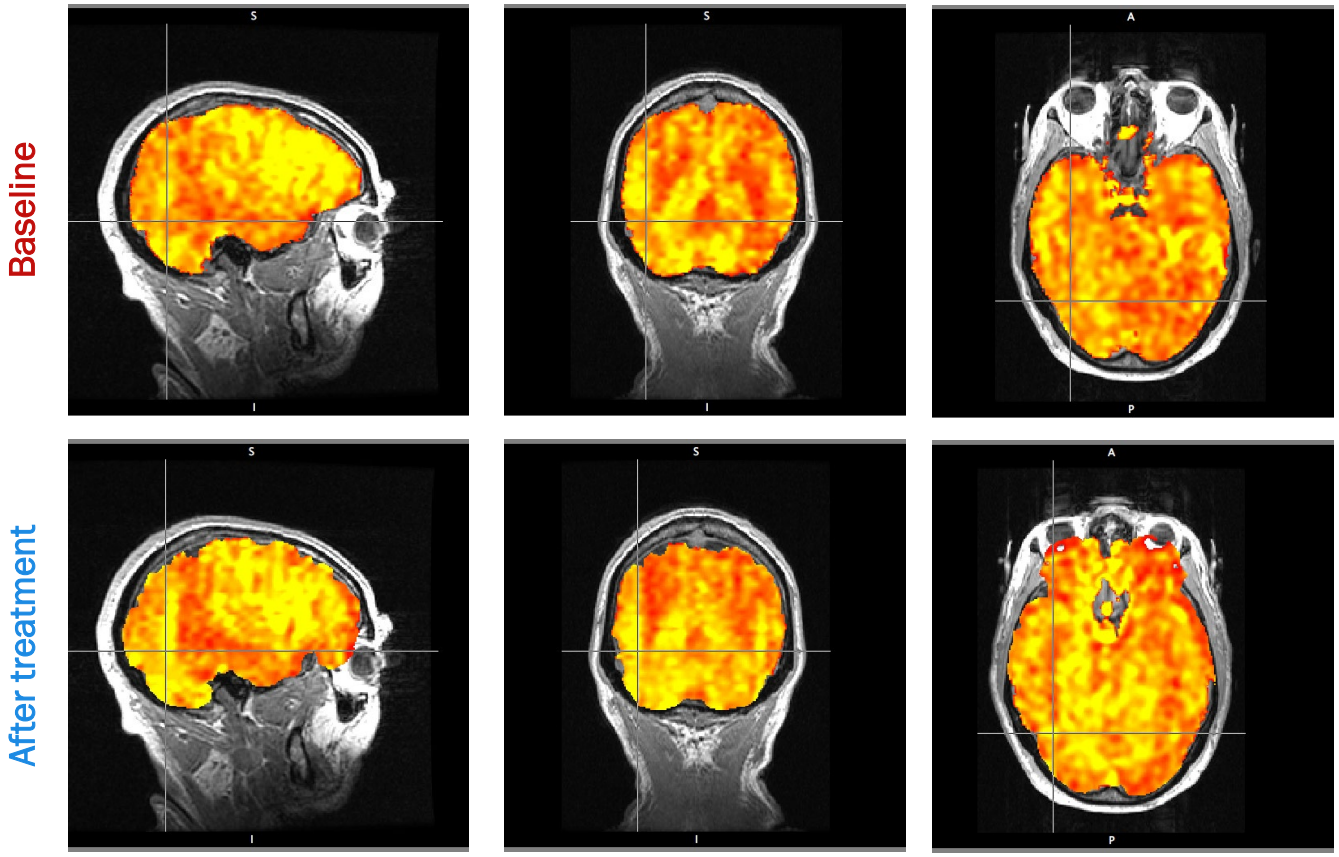
9/13 decreased (69%)

Mean Absolute Change = -8.69 (p=ns)

Mean % Change = -0.92% (p=ns)

Arterial Spin Label Imaging suggests NE3107 enhances blood flow in the brain, a marker for brain activity

Patient N08 – Global Rating of Change +3.5 (Partner Reported)



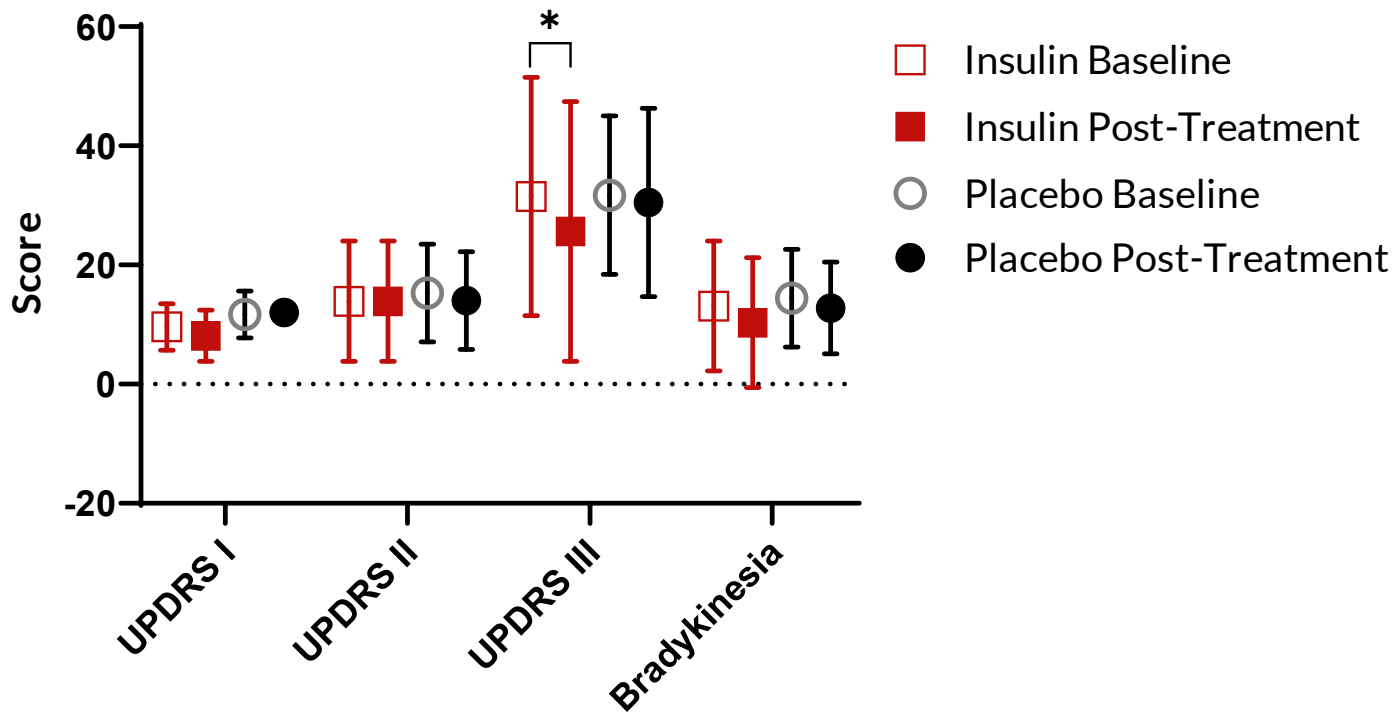
Arterial Spin Label Imaging looks at the flow of oxygenated hemoglobin

Yellow indicates the most relative enhancement of flow while red indicates enhancement

The Role of Reduced Insulin Signaling in Parkinson's Disease

Stimulation of insulin signaling improves motor function in Parkinson's patients

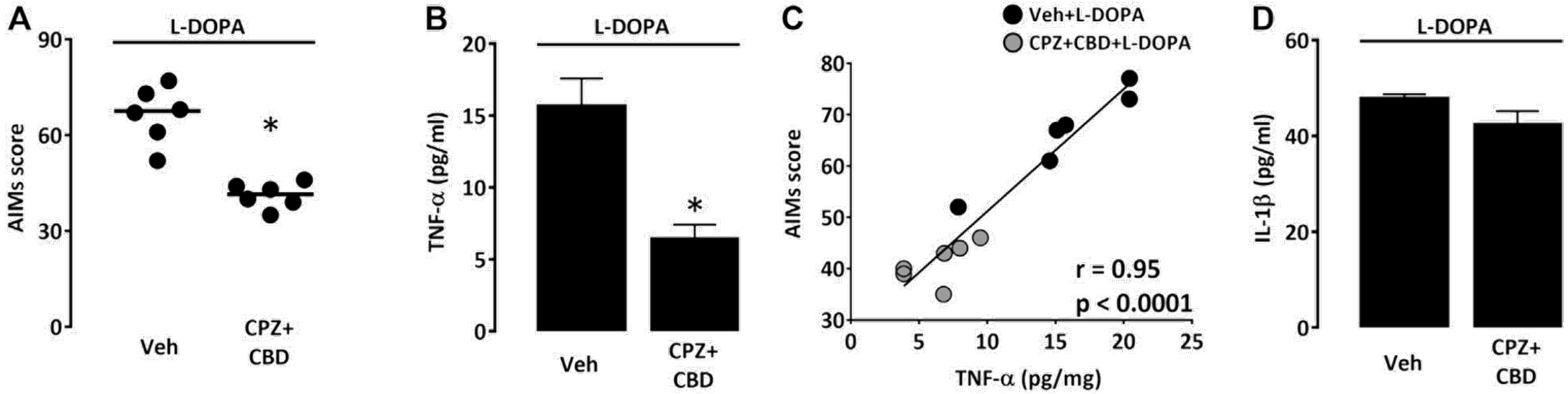
Intranasal Insulin Treatment Reduced Inflammation and Improved Motor Activity



UPDRS: Unified Parkinson's Disease Rating Scale; Part I (mentation, behavior and mood); Part II (activities of daily living); Part III (Motor)
From: Novak 2019 *J Pone 0214364*

Inflammation's Role in LID

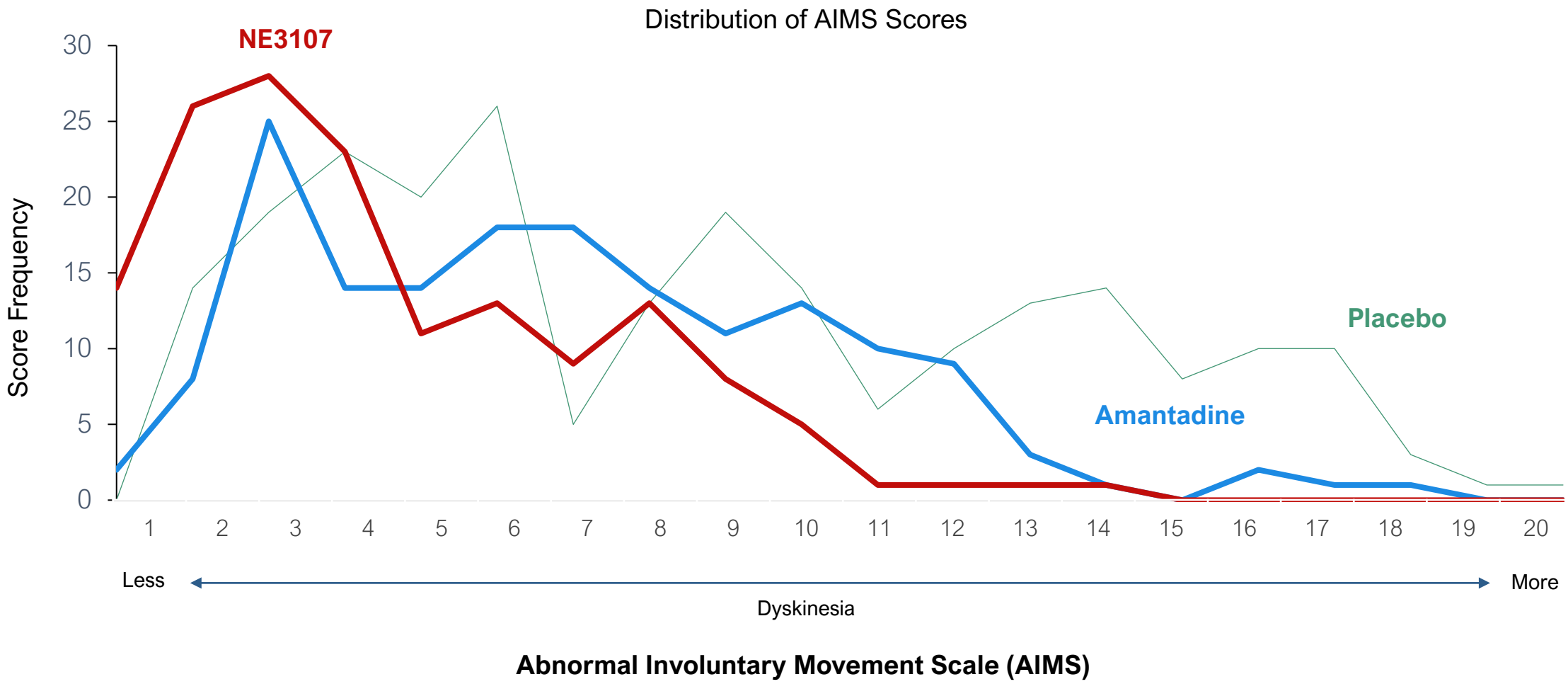
Reduction of TNF-induced inflammation reduces LID



6-OHDA: 6-hydroxydopamine-lesioned mice rendered dyskinetic; AIM: Abnormal Involuntary Movement Scale (lower is better); CPZ-CBD: capsazepine (CPZ) with cannabidiol (CBD), anti-inflammatory agents

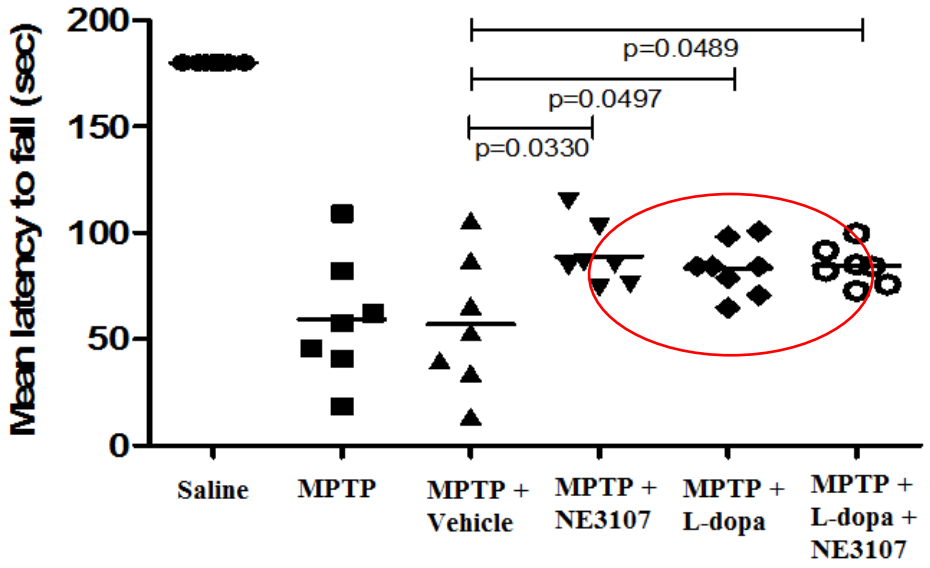
Pereira 2021 F Phar 617085

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

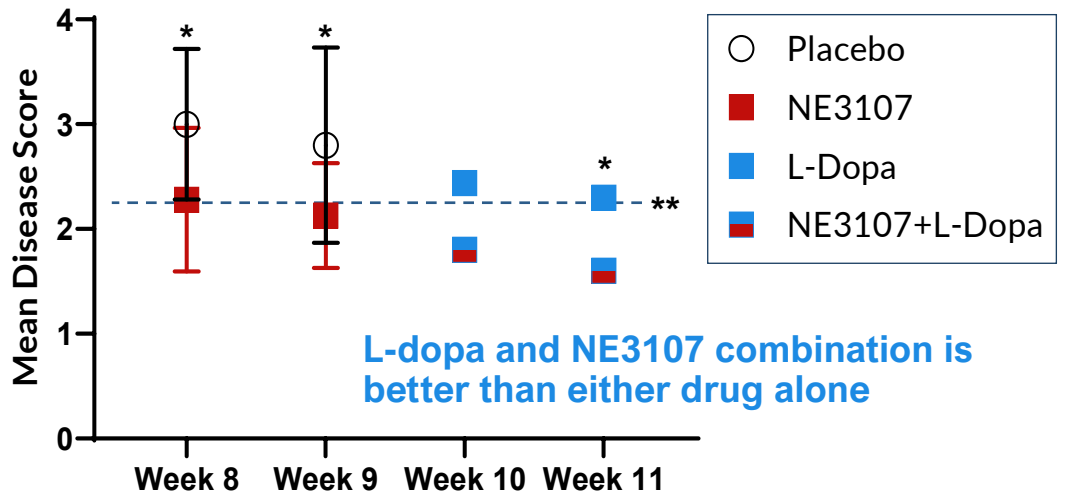


NE3107 has similar promotoric activity to L-dopa in rodent and marmoset models

MPTP Mouse



MPTP Marmoset treated at Week 8

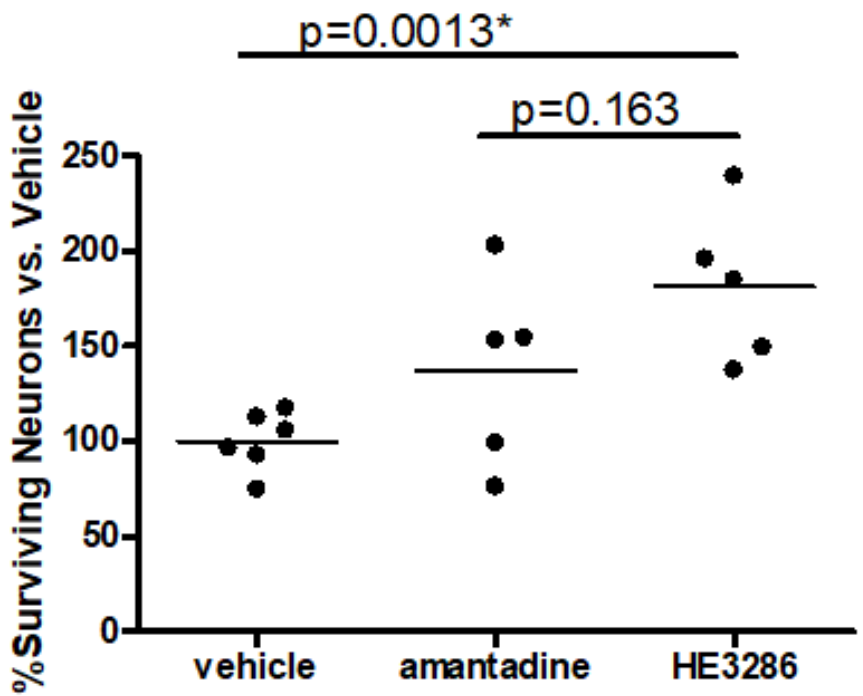


*p<0.001 compared to other treatment arm in time period
** L-Dopa at week 11 not statistically different from NE3107 at weeks 8 and 9

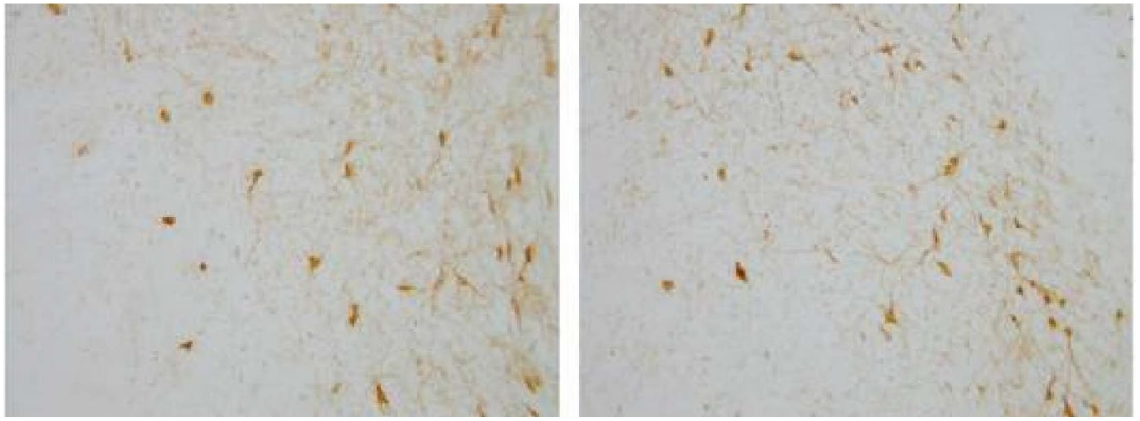
NE3107's promotoric effects observed within 4 days of treatment

MPTP: 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine, a toxin used to mimic many aspects of PD

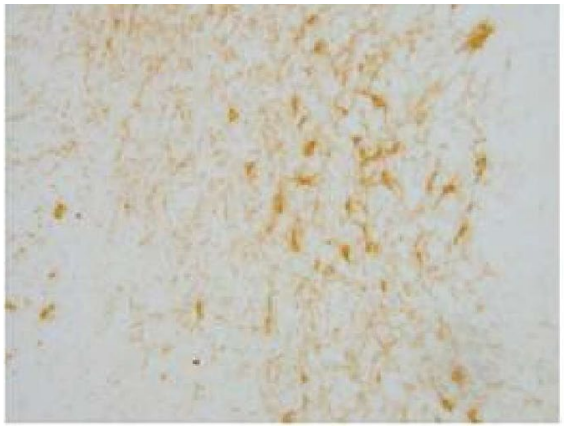
NE3107 preserved TH+ neurons in MPTP marmosets



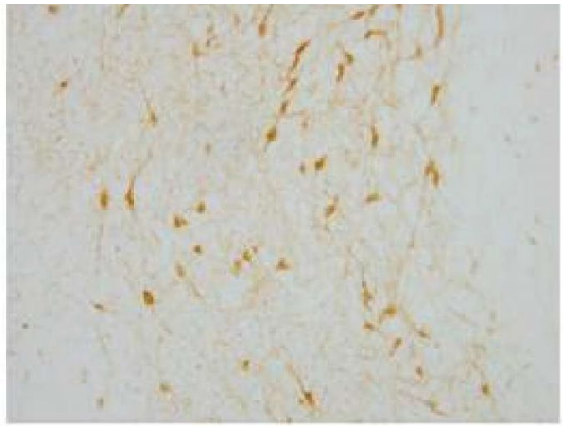
Control moneys (M09100 and M11008)



HE3286 (M11007)



Amantadine (M10084)



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

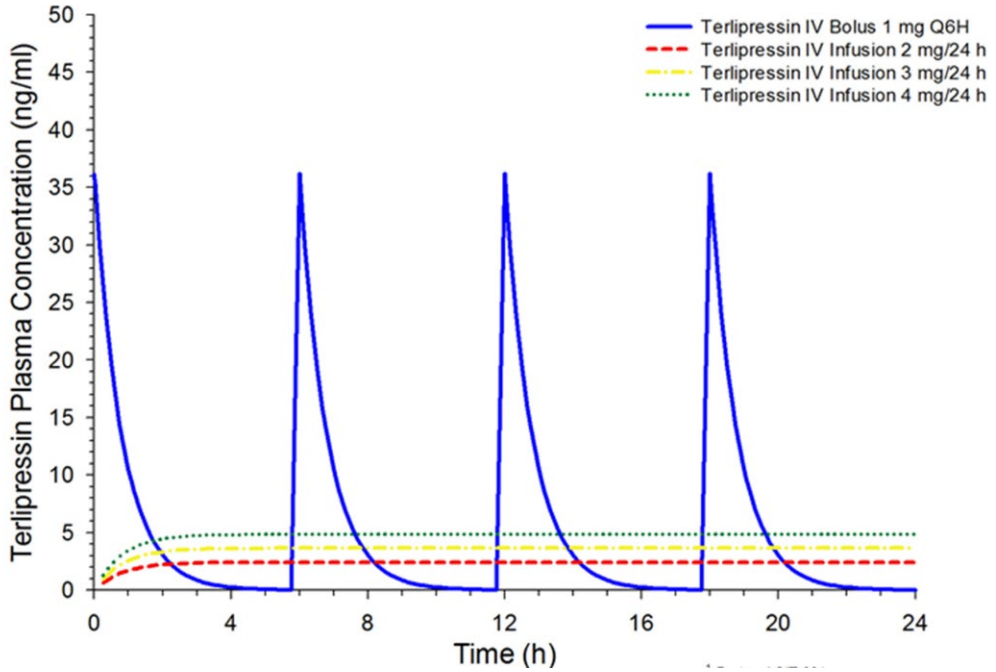
BIV201

- 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))

BioVie Phase 2a trial results: BIV201 Pharmacokinetics

Continuous low blood levels of terlipressin versus spikes with IV-bolus dosing

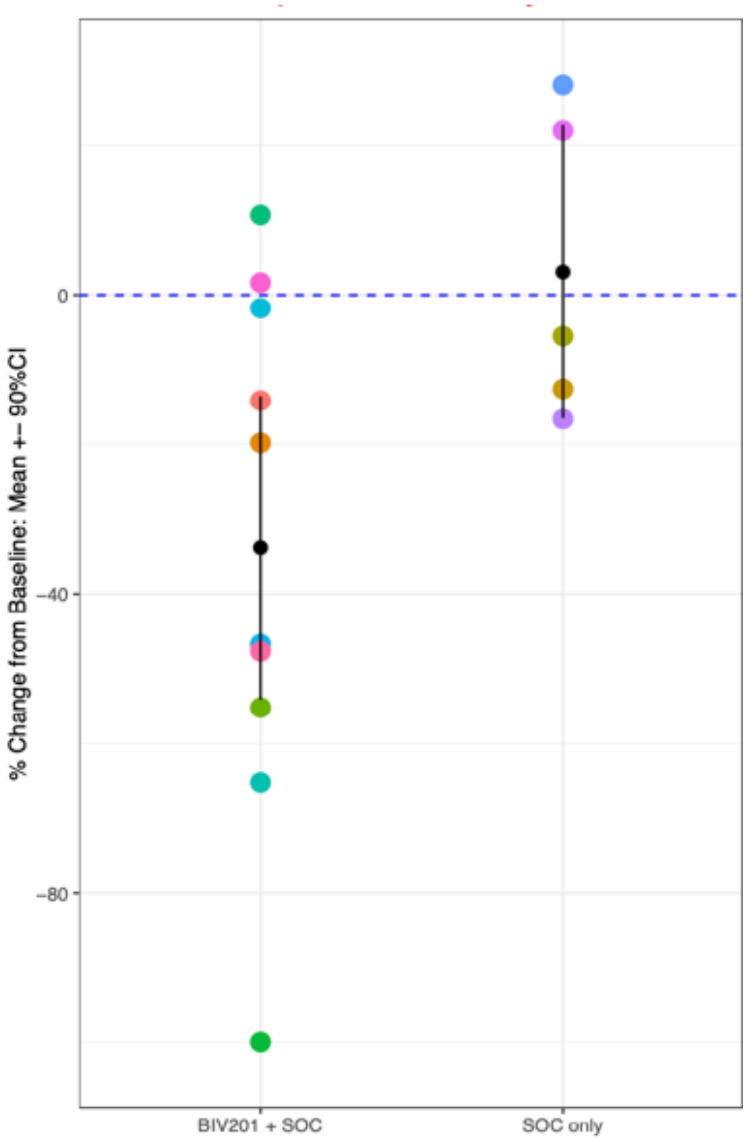
Comparison of the PK Profile of Terlipressin Administered by Continuous Infusion or Intermittent IV Boluses.



Simulated terlipressin plasma concentrations following IV bolus of 1 mg every 6 hours 1 [Lucassin, AusPAR-cer 2011] and IV continuous infusions of 2 mg, 3 mg, or 4 mg per day

Source: BioVie poster presentation at AASLD 2019. BIV201 is an investigational therapy

Ascites volume (L) 28d pre- vs 28d post-treatment



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