Developing Transformative Therapies to Overcome Chronic Debilitating Diseases

Corporate Presentation • May 2023

Forward-looking statements

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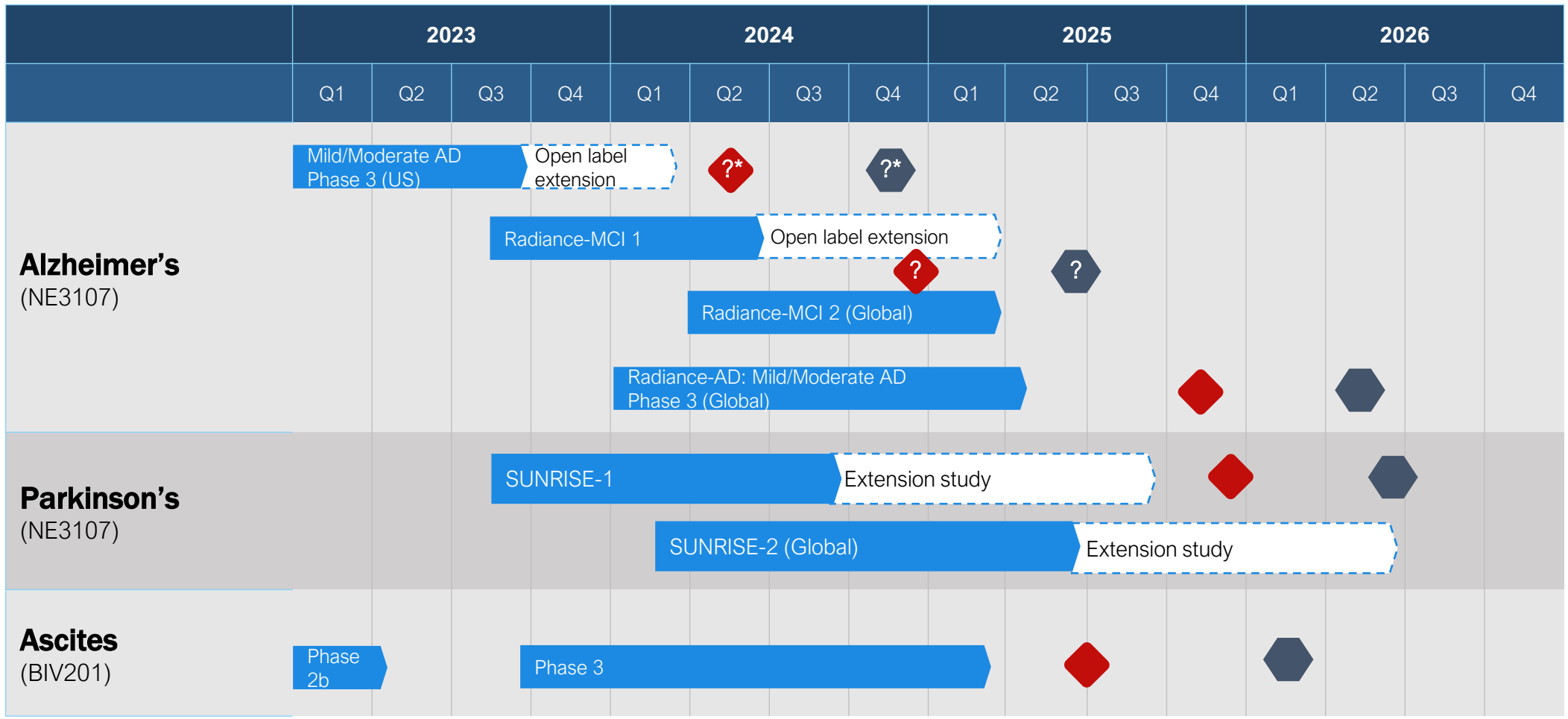
Overview

- We believe that inflammation is starting point for many things going wrong in the body
  - NE3107 is our drug candidate that modulates both inflammation and the associated insulin resistance
  - NE3107 is currently in clinical development for Alzheimer's Disease (AD) and Parkinson's disease (PD)
- In Alzheimer's, a Phase 2 exploratory biomarker trial found that patients treated with NE3107 for 3 months experienced:
  - Reversal of their cognitive decline as measured by multiple assessment tools
  - Reduction of TNFα in manner that's correlated with cognitive improvements
  - Reduction phospho-tau production and the ratio of phosphor-tau to amyloid beta (Aβ)
  - Improvements in one or more brain regions as seen from advanced functional MRI studies among patients with abnormal scans at baseline
- In Parkinson's, a Phase 2 trial found that patients treated with NE3107 for 28-days experienced:
  - Improvements of UPDRS part 3 score on Day 28 compared to Day 0 that is 3+ points better than those treated with levodopa alone at the 2- and 3-hour marks. This level of superiority is considered by PD experts to be clinically meaningful
  - Improvements of 6+ points among patients younger than 70 years old (a surrogate for less disease progression)
  - Significantly more NE3107-treated patients maintainedmorning "on" symptoms in the morning compared to none of levodopa-alone patients
- In liver disease, BIV201 is in Phase 2b for refractory ascites. In trials thus far, patients have experienced in ascites fluid build up and extension of time between paracenteses with no unexpected drug-related SAEs

Expected Catalysts & anticipated timelines



* 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.

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

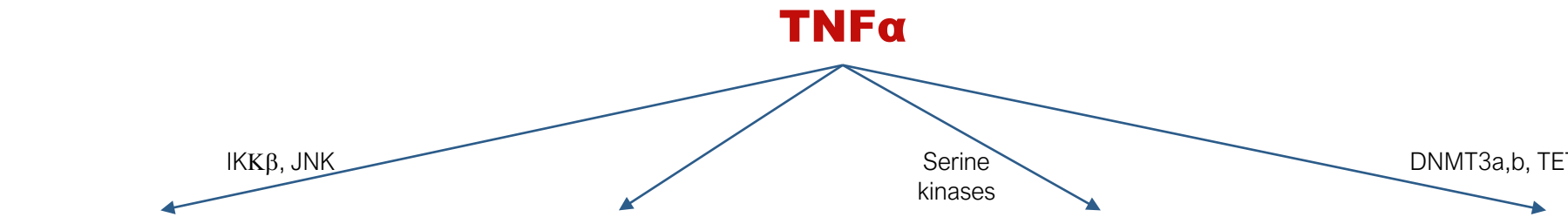
$3B

Annual sales for every
100,000 people treated

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

Source: Company estimates

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



Insulin resistance

Diabetes
Metabolic disorders
Parkinson's

Inflammatory factors

- Cytokines
- Chemokines
- APP, SNCA
- Others

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

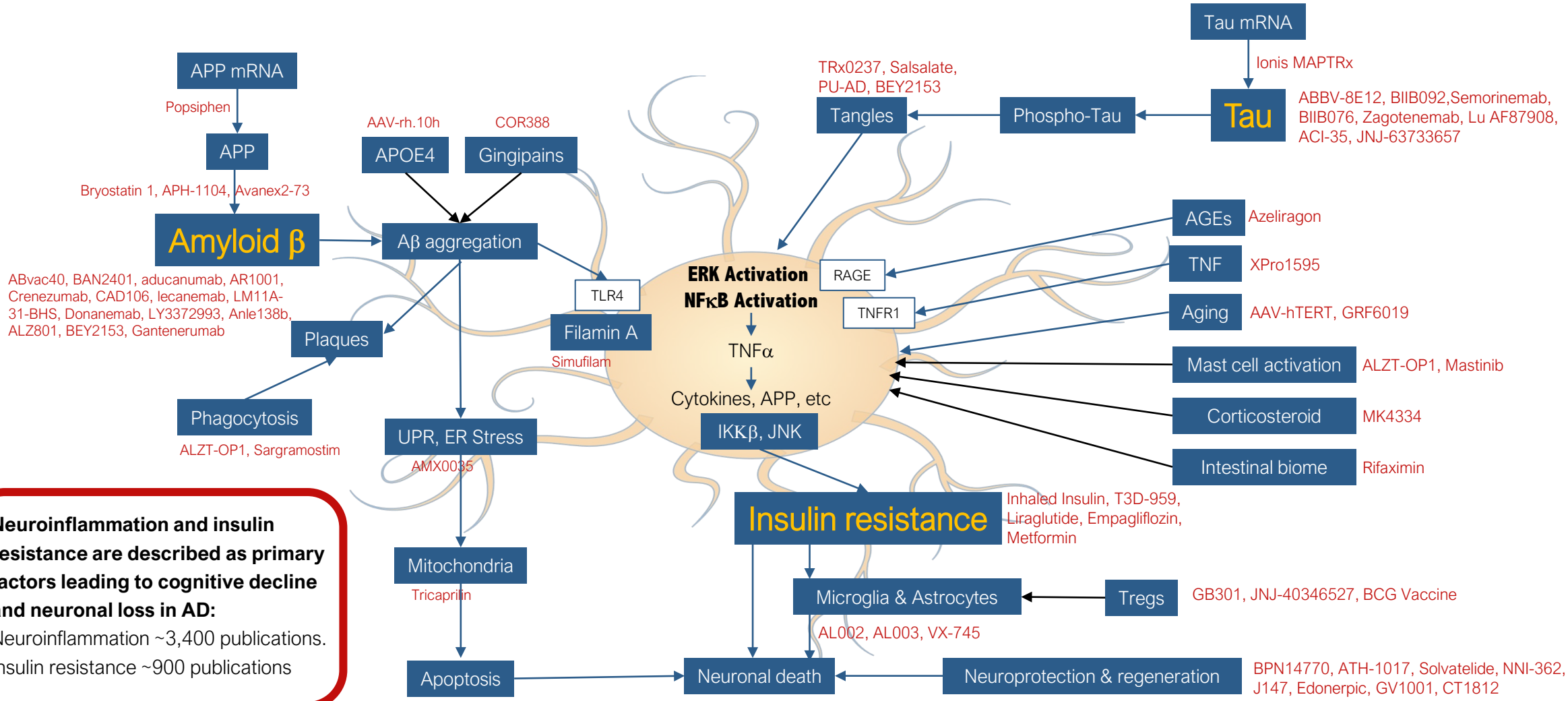
Tau phosphorylation

Alzheimer's

DNA Methylation

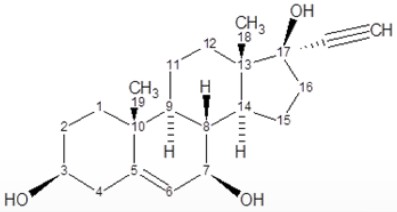
Systems dysregulation of gene expression
Accelerated aging
Various cancers
Cognitive impairment and dementia
Parkinson's disease
Cardiovascular disease
COPD and respiratory disease
Chronic kidney disease
Inflammatory bowel disease
Sepsis
Others

Alzheimer's Disease Pathways



Neuroinflammation and insulin resistance are described as primary factors leading to cognitive decline and neuronal loss in AD:
Neuroinflammation ~3,400 publications.
Insulin resistance ~900 publications

Background on NE3107



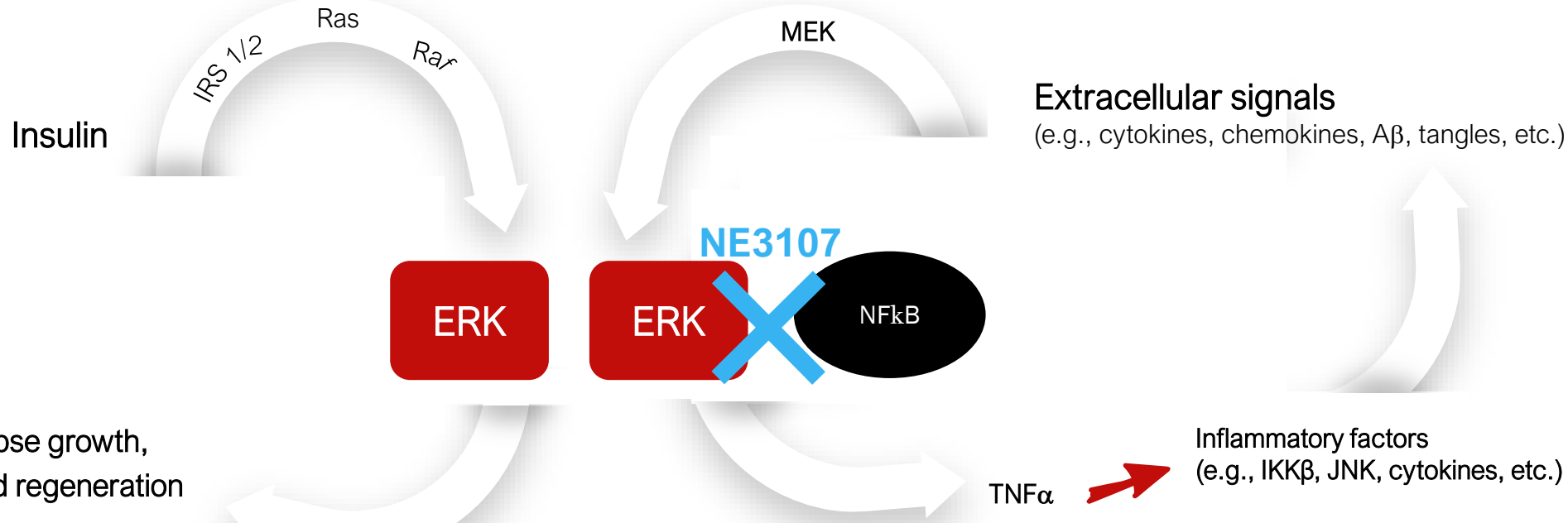
First-in-class molecule with desirable characteristics

- 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)

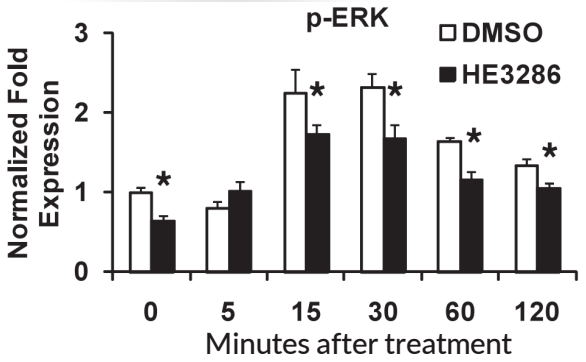
- Originated by Hollis-Eden Pharmaceuticals (renamed Harbor Therapeutics)
  - NE3107[1] is a synthetic analogue of a metabolite of the adrenal hormone DHEA
- Phase 1 and 2 trials in diabetic patients showed that NE3107:
  - Showed no differences in AEs compared to placebo
  - Decreased insulin resistance, 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

1. Previously known as HE3286

NE3107 blocks inflammatory (but not homeostatic) ERK and NFkB, which play pivotal roles in AD pathogensis[1]

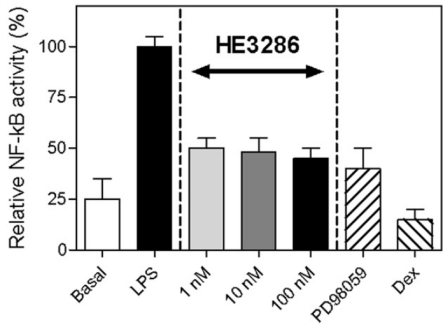


NE3107 Reduces ERK Activation



Lu 2010 Am J Physiol Endocrinol Metab 298 E1036

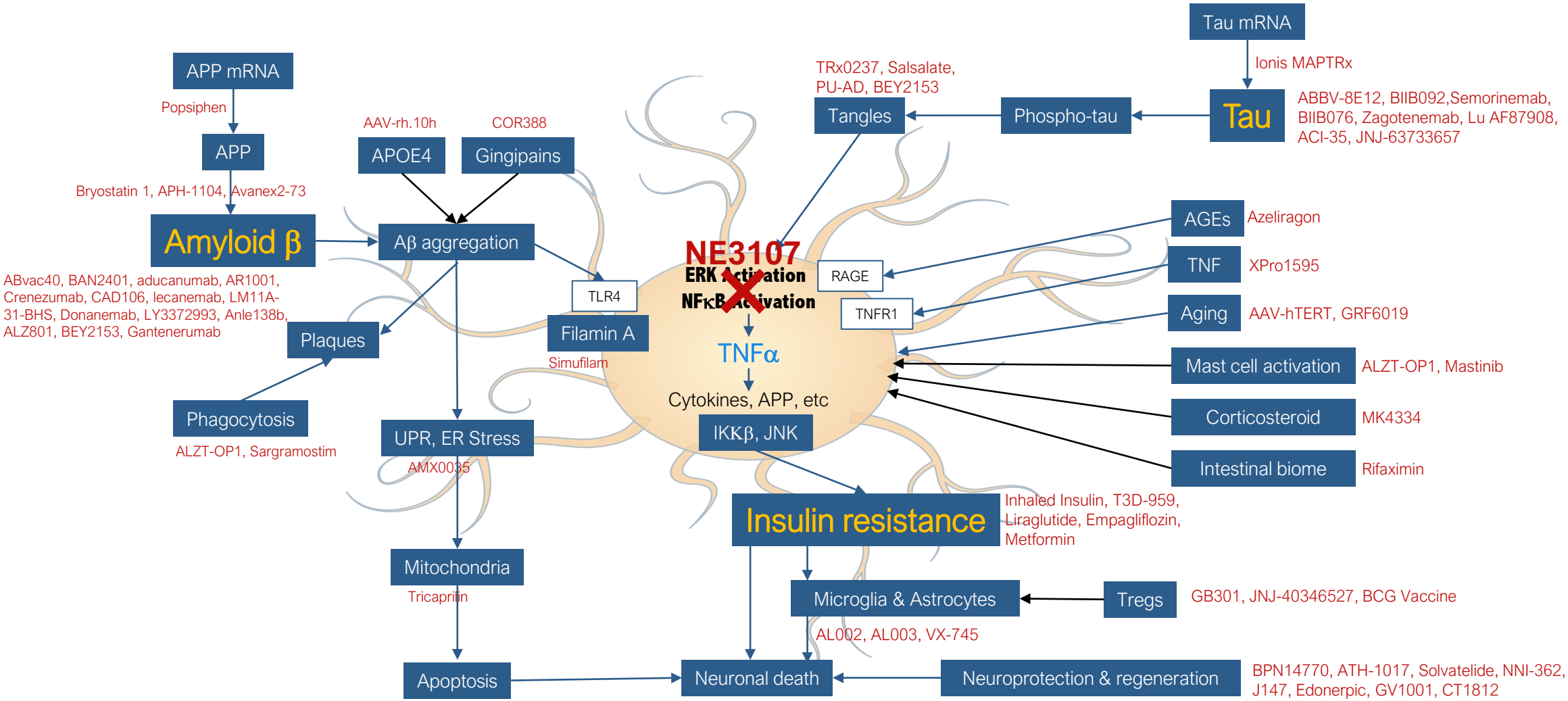
NE3107 Reduces NFkB Activity



Wang 2010 J Pharmacol Exp Ther 333 70

1. Sun et al. *Int. J. Mol. Sci.* 2022, 23, 8972.

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

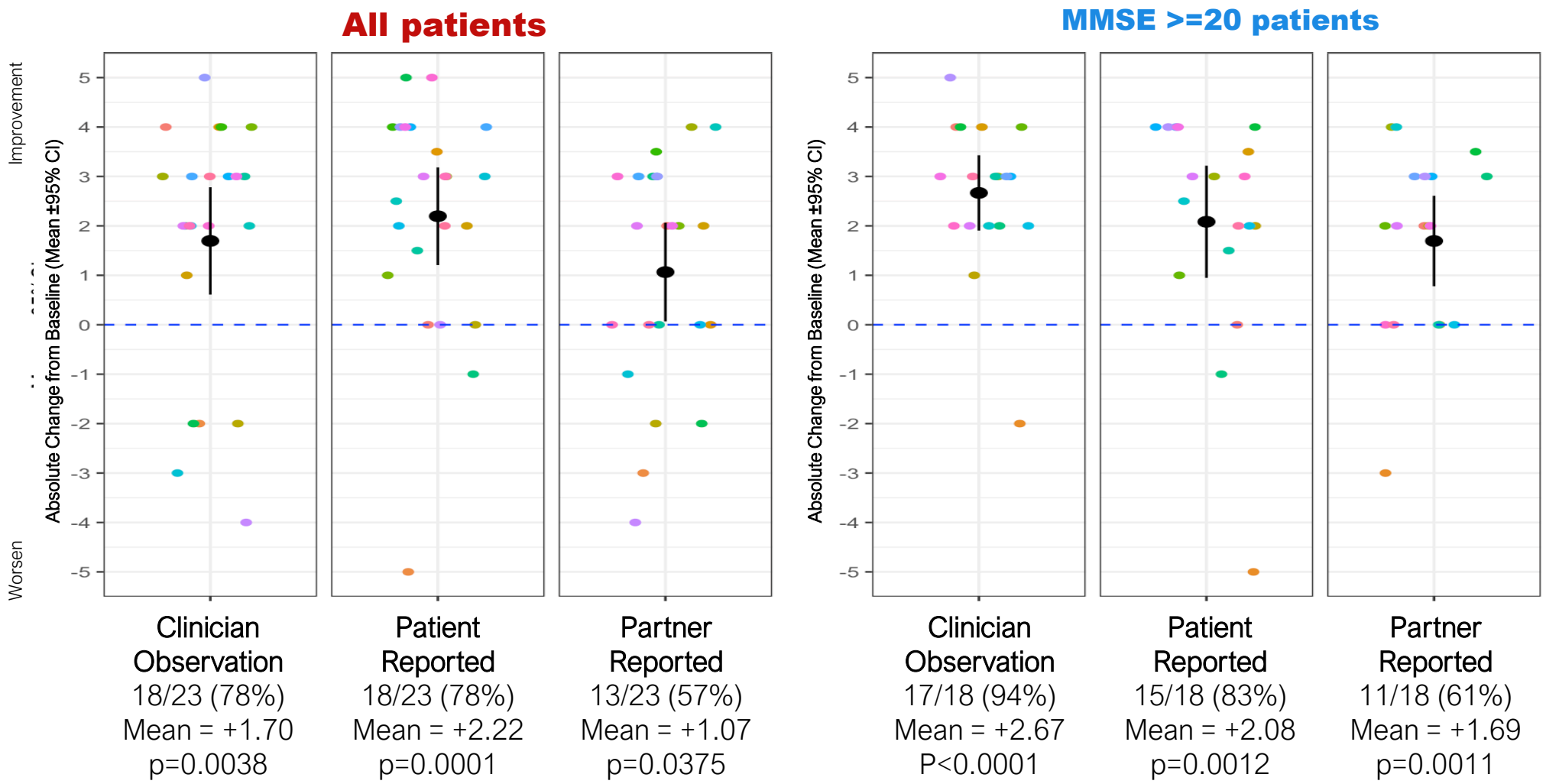


Investigator-Sponsored Trial in MCI and Mild Alzheimer's Disease, NCT05227820

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

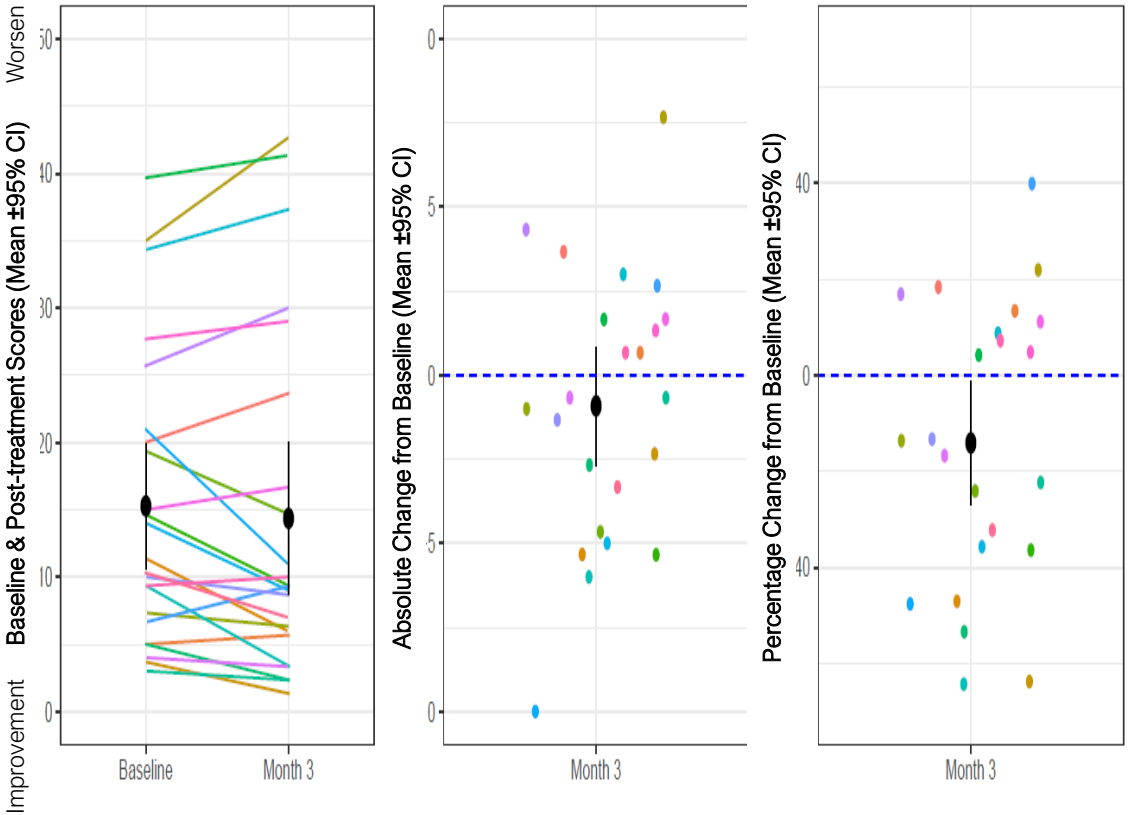
Large majority of patients improved significantly on the Global Rating of Change (overall impression)



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

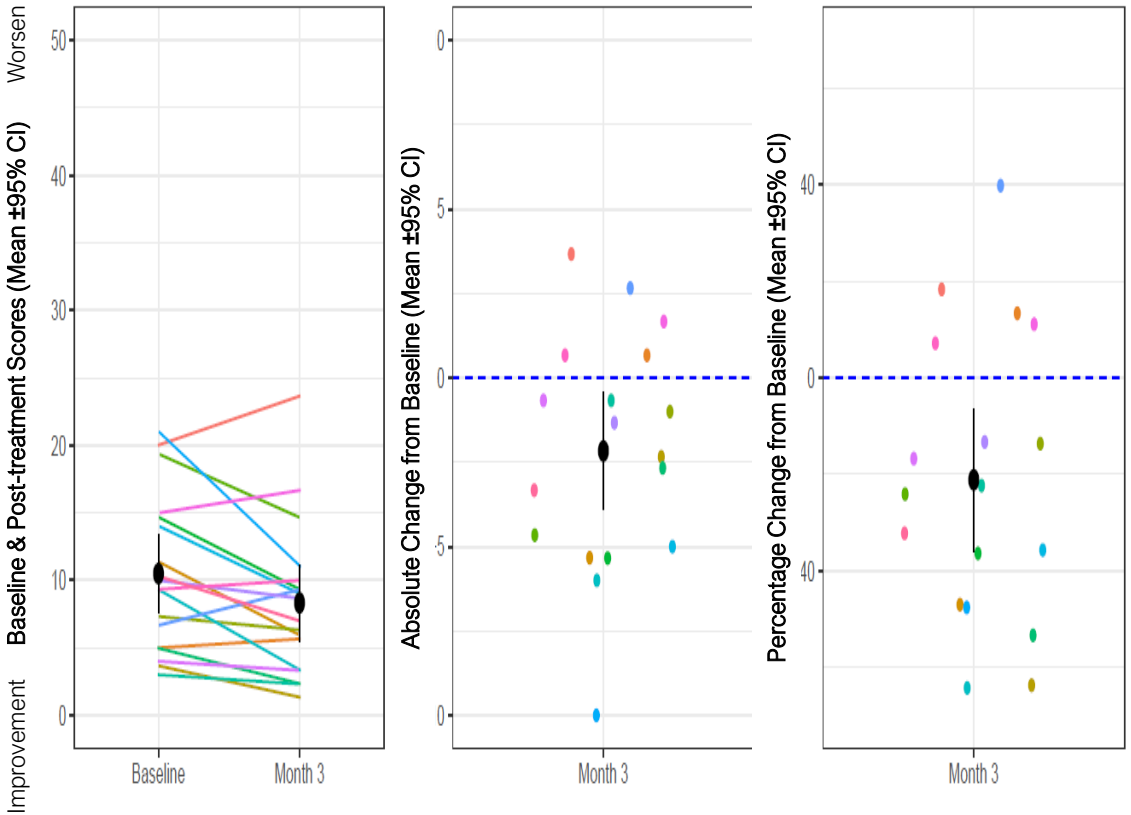
Significant improvements in ADAS-Cog12 assessment of cognition among MCI/Mild AD patients

All patients



13/23 improved (57%)
Mean Absolute Change = -0.91 (p=ns)
Mean % Change = -14.1% (p=0.0333)

MMSE >=20 patients



13/18 improved (72%)[1]
Mean Absolute Change = -2.2 (p=0.0173)
Mean % Change = -21.1% (p=0.0079)

1. Among responders: Mean Absolute Change = -3.718 (p=0.0003); Mean % Change = -36.2% (p<0.0001)

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***

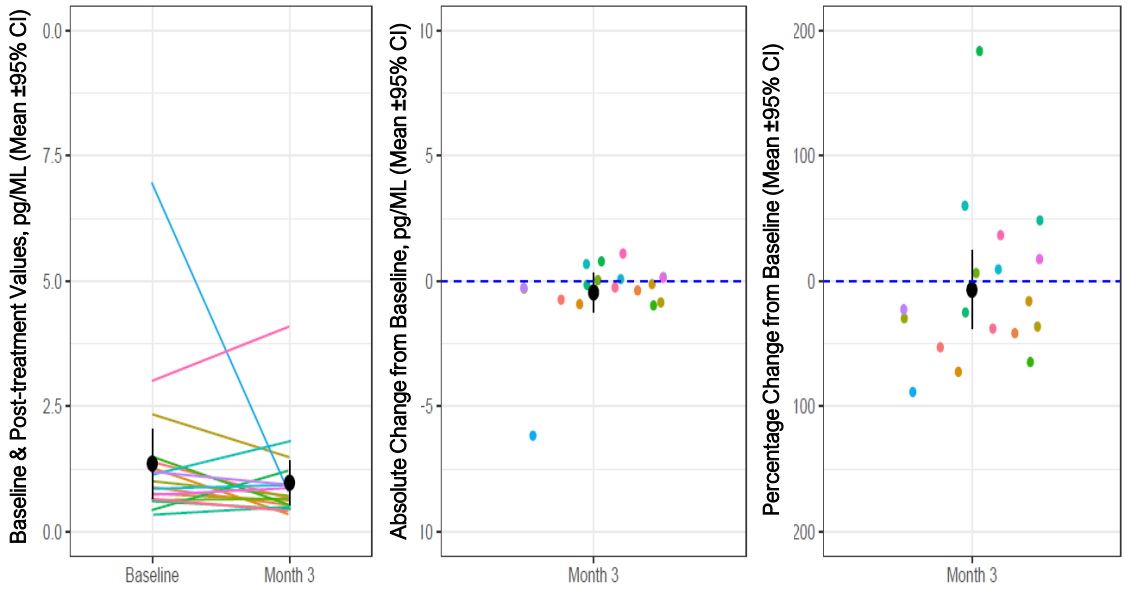
ADAS-Cog12 = Alzheimer's Disease Assessment Scale-Cognitive
MMSE = Mini-Mental State Examination
MoCA = Montreal Cognitive Assessment
QDRS = Quick Dementia Rating System
CDR = Clinical Dementia Rating scale
ADCOMS = Alzheimer's Disease Composite Score

Green = Improvement
* p<0.05

Improvements on TNFα among MCI/Mild AD patients

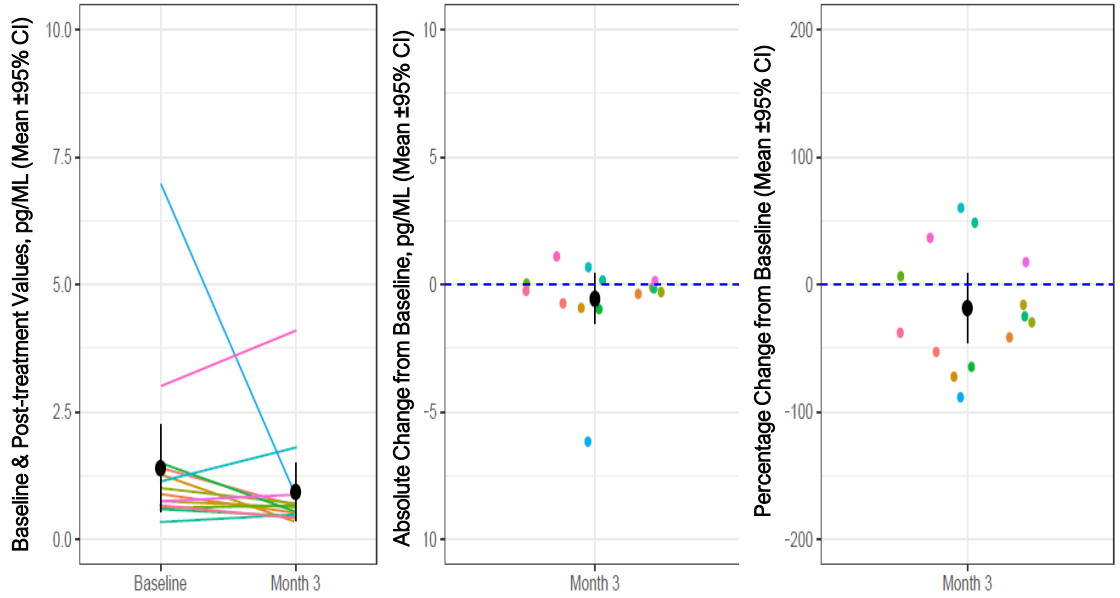
Tumor Necrosis Factor Alpha (TNFα) is a cytokine identified as a major regulator of inflammation whereby its excessive activation is associated with chronic inflammation[1]

All patients



11/18 improved (61%)
Mean Absolute Change = -0.45 (p=ns)
Mean % Change = -6.9%% (p=ns)

MMSE >=20 patients

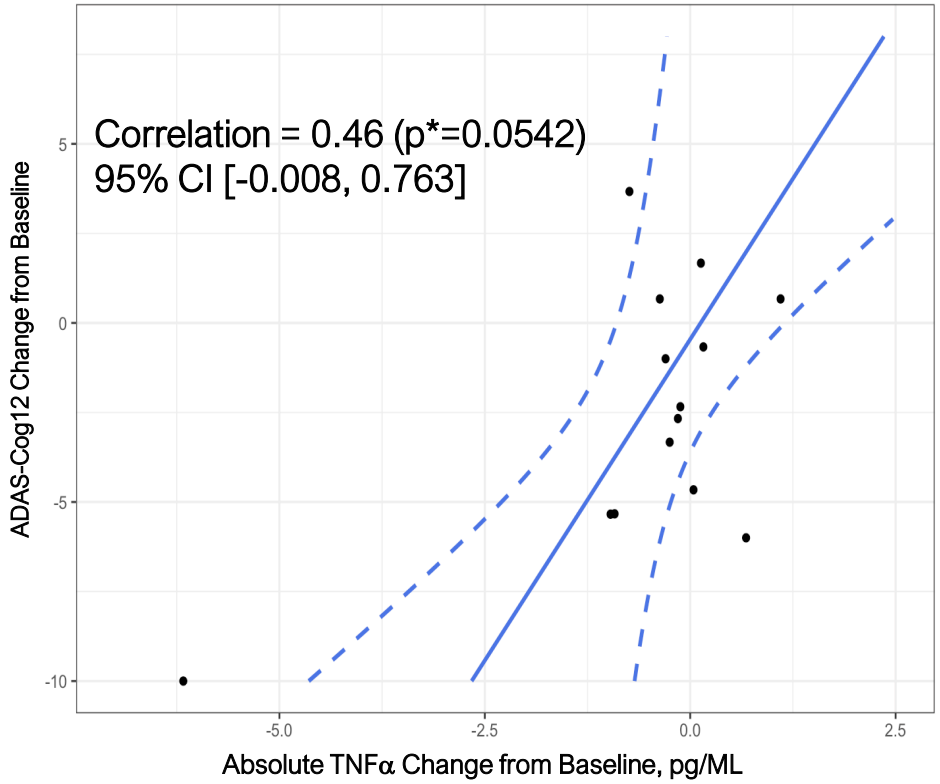


9/14 improved (64%)[2]
Mean Absolute Change = -0.563 (p=ns)
Mean % Change = -18.5% (p=ns)

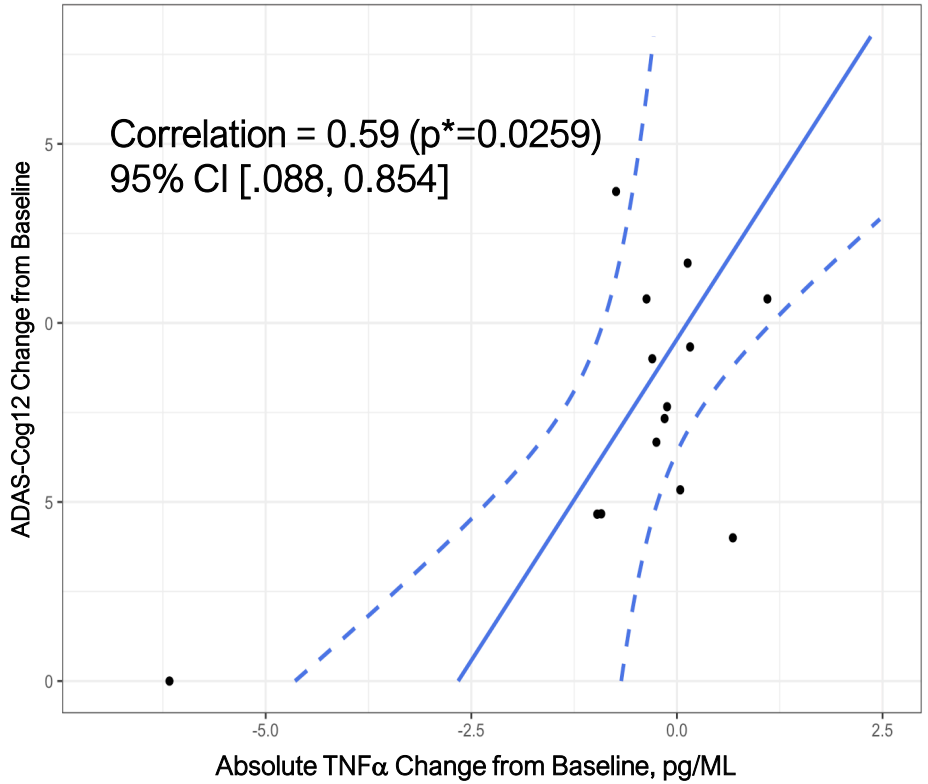
1. Jang et al. Int J Mol Sci. 2021 Mar; 22(5): 2719.
2. Among responders: Mean Absolute Change = -1.11 (p=ns); Mean % Change = -47.6% (p=0.003)

Improvements in TNFα significantly correlated to improvements in ADAS-Cog12

All patients



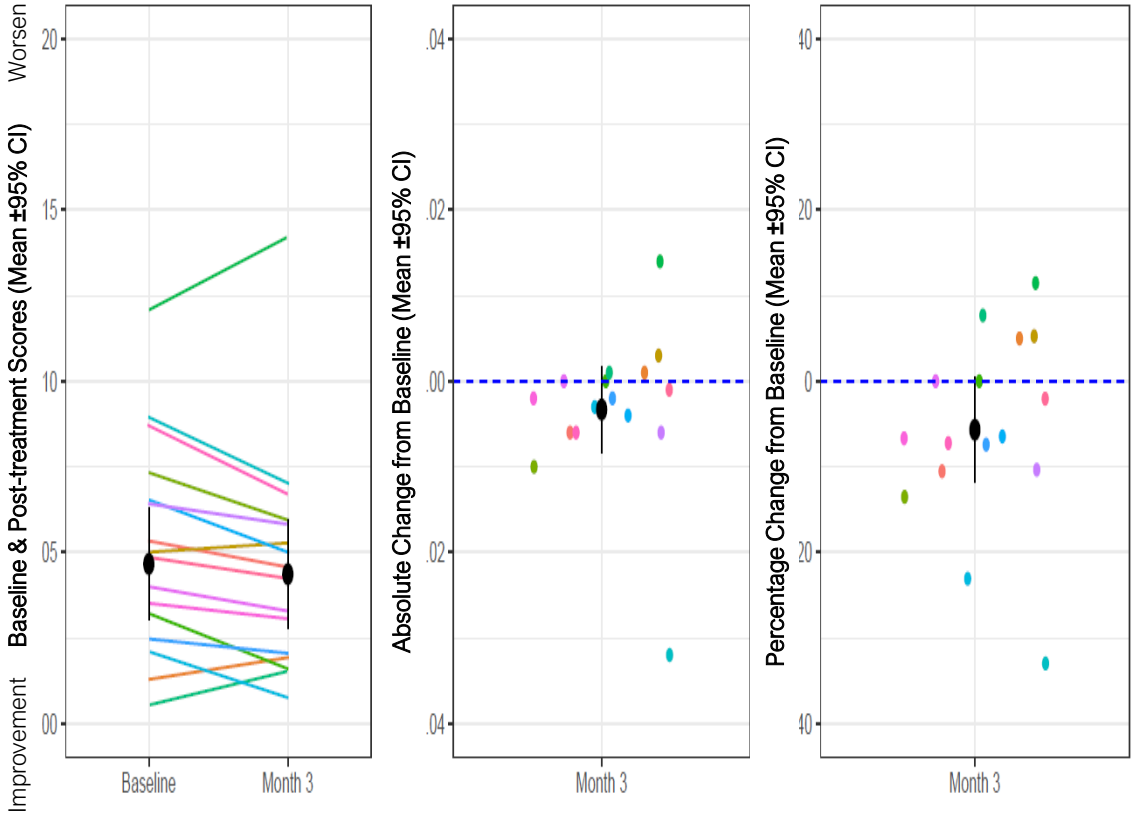
MMSE >=20 patients



* Pearson Correlation Test

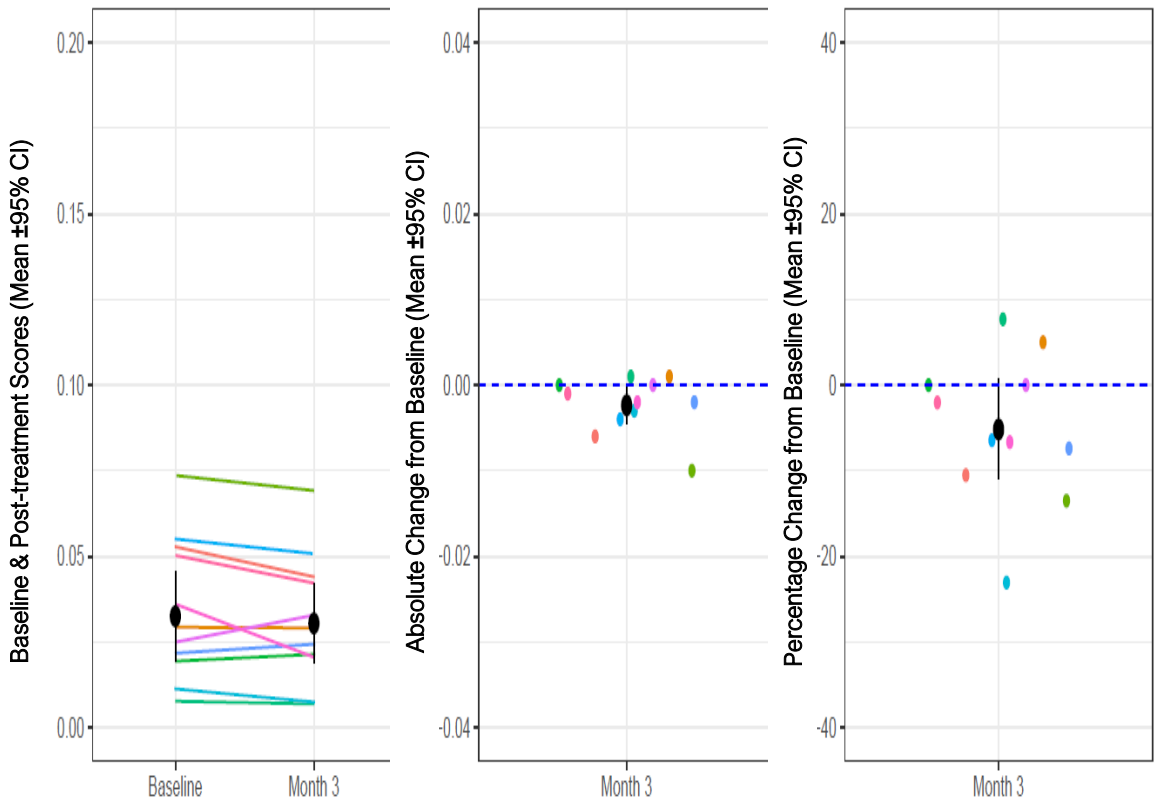
Significant improvements in the CSF p-tau : $A\beta_{42}$ Ratio, a predictive measure of PET amyloid status[1]…

All patients



10/16 improved (63%)
Mean Absolute Change = -0.0033 (p=0.1747)
Mean % Change = -5.67% (p=0.0648)

MMSE >=20 patients

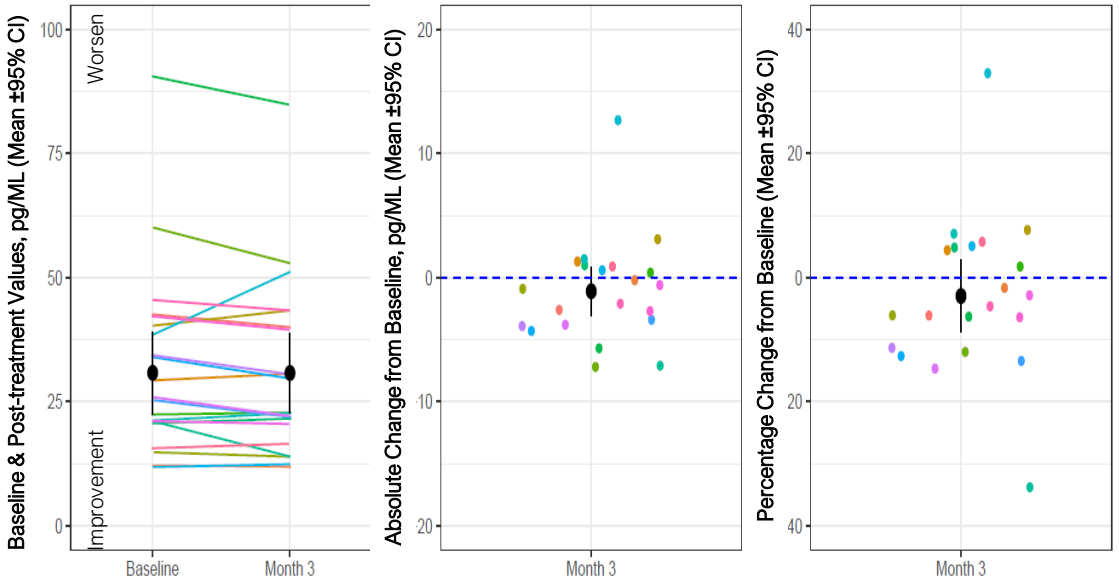


7/11 improved (64%)[2]
Mean Absolute Change = -0.0024 (p=0.0401)
Mean % Change = -5.18% (p=0.077)

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)

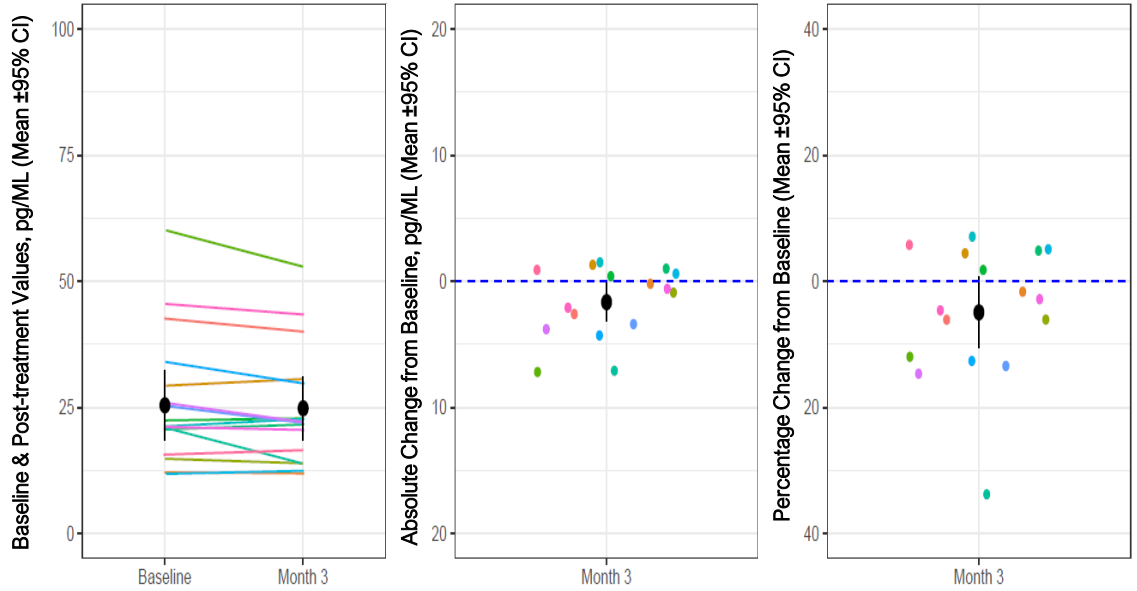
… Driven by a significant improvements in CSF p-tau

All patients



13/21 improved (62%)
Mean Absolute Change = -1.10 (p=0.2545)
Mean % Change = -2.96% (p=ns)

MMSE >=20 patients



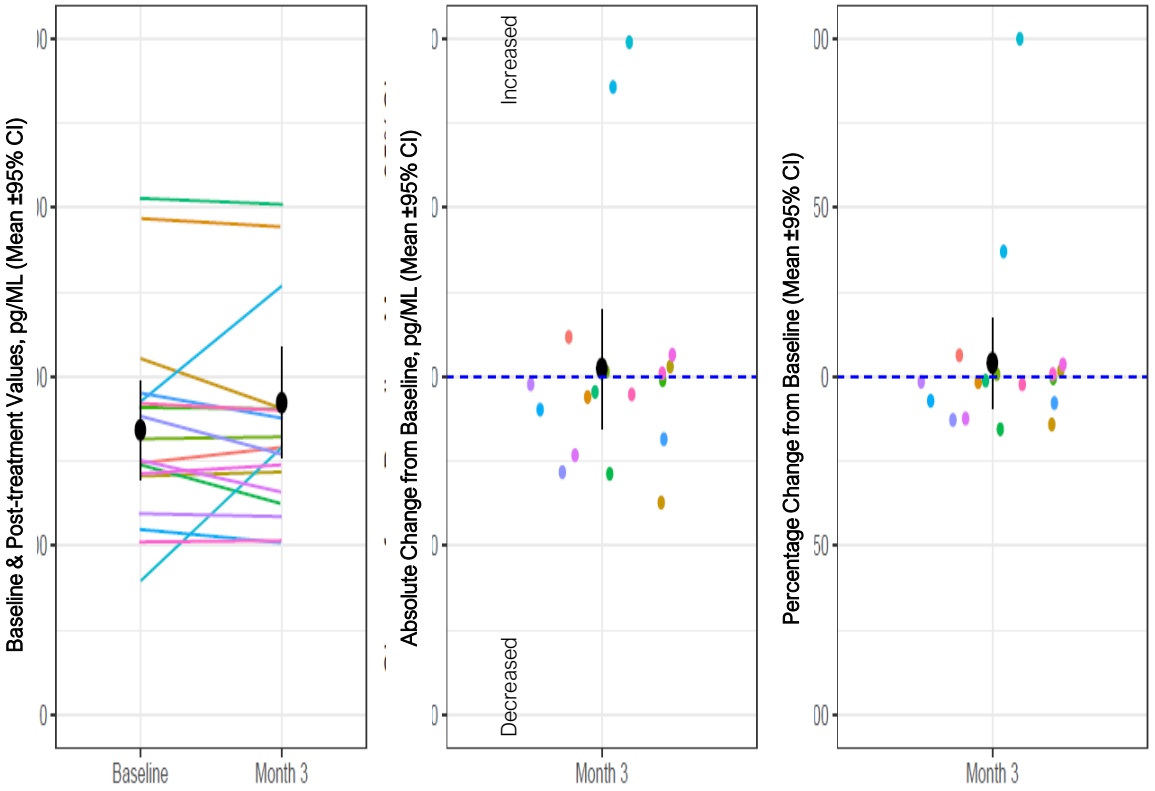
10/16 improved (63%)[1]
Mean Absolute Change = -1.66 (p=0.0343)
Mean % Change = -4.93% (p=0.0852)

- 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

1. Among responders: Mean Absolute Change = -3.22 (p=0.0027); Mean % Change = -10.8% (p=0.0053)

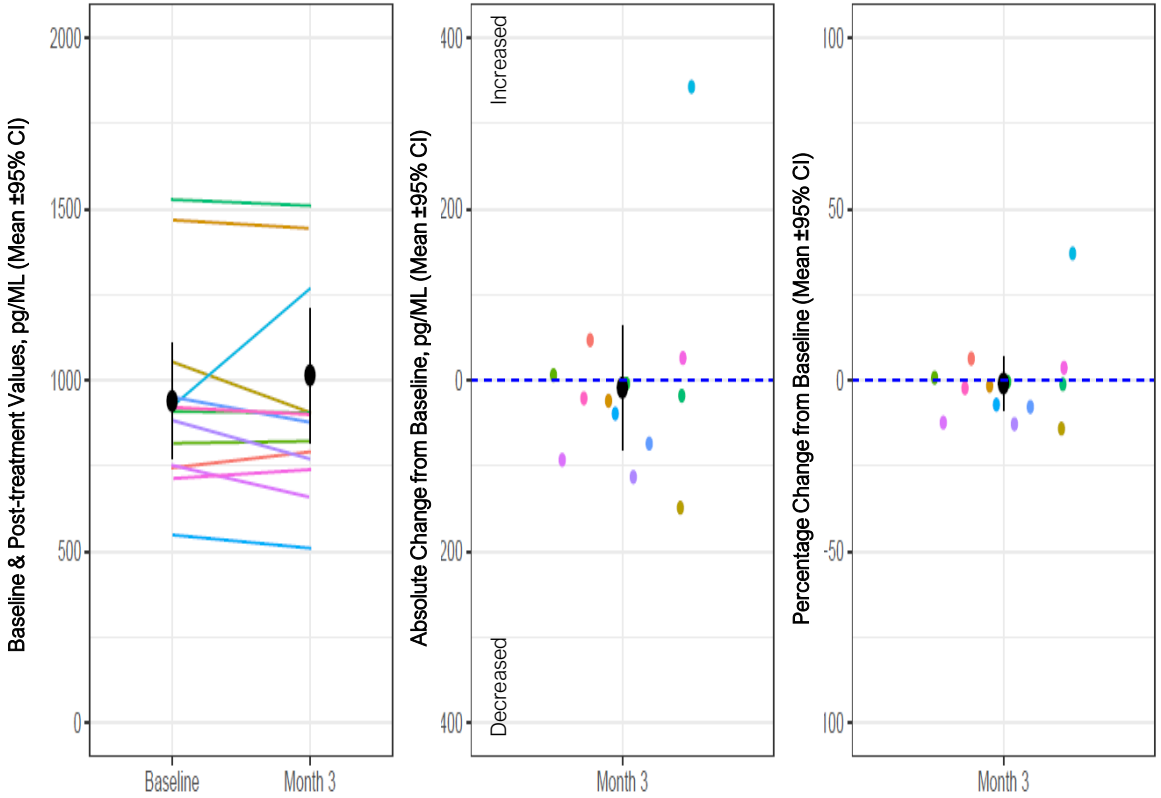
Modest improvements in CSF $A\beta_{42}$

All patients



11/18 decreased (61%)
Mean Absolute Change = 9.7 (p=ns)
Mean % Change = 4.1% (p=ns)

MMSE >=20 patients

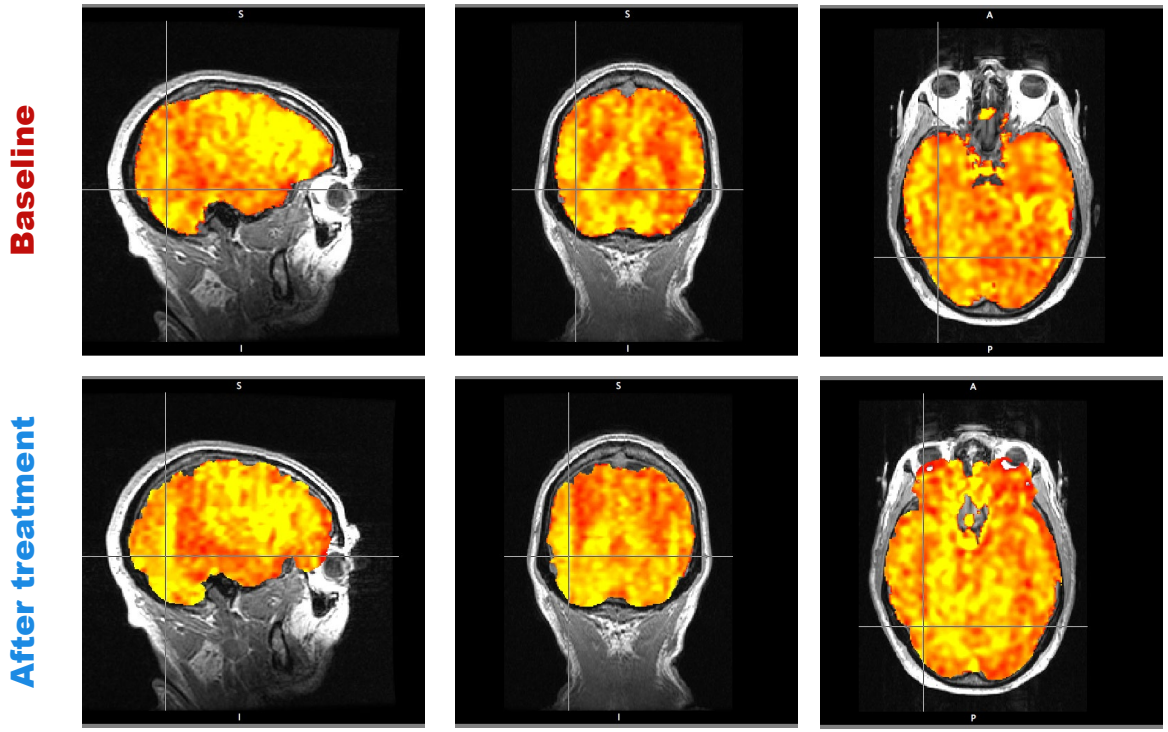


9/13 decreased (69%)
Mean Absolute Change = -8.69 (p=ns)
Mean % Change = -0.92% (p=ns)

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

- Arterial Spin Label Imaging looks at the flow of oxygenated hemoglobin
- Yellow indicates the most relative enhancement of flow while red indicates enhancement

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

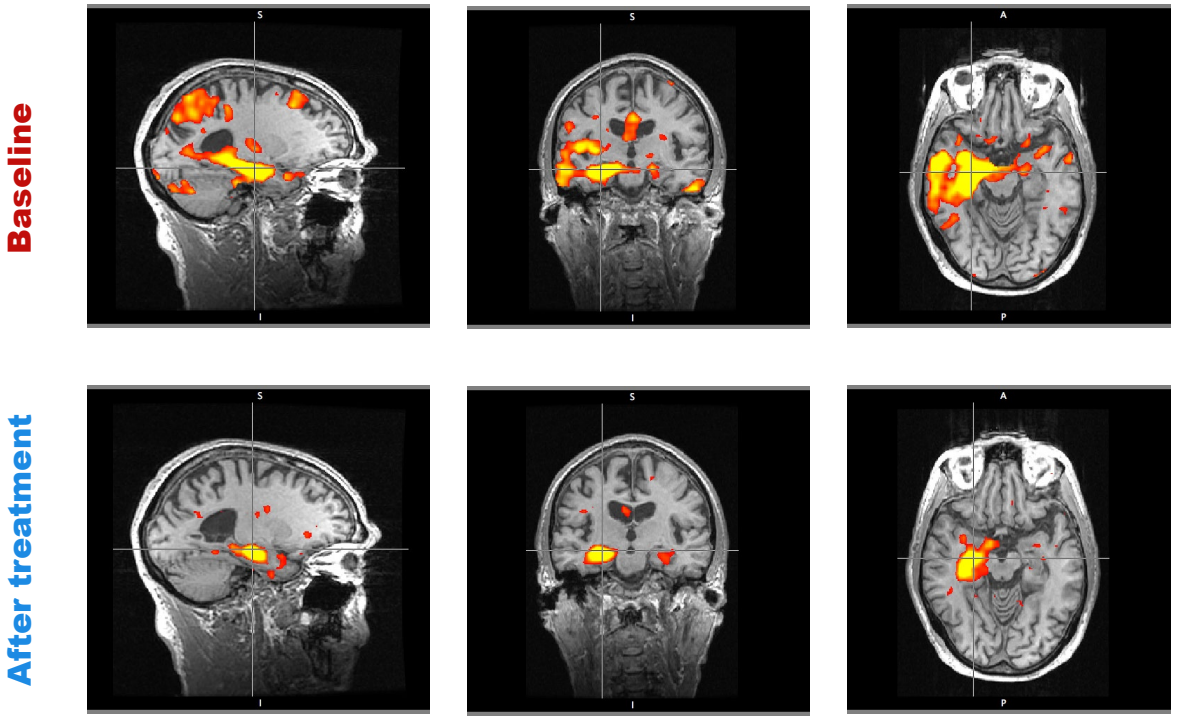


- 17% of 17 patients with MMSE >=20 had “signal” improvements in the ASL in relevant areas (temporal parietal occipital) compared to baseline. Increased blood flow serves as a marker for brain activity
- 12% of 17 patients declined

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

- 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

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



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: 20150429

The multifactorial nature of dementia pathology

- AD drug development at BioVie is based on the evidence that AD pathology is multifactorial in nature
  - While Aβ and p-tau are important factors, decades of research on dozens of agents have not conclusively correlated improvements on these individual factors to improvements in cognition
  - We believe additional factors such as inflammation, insulin resistance, metabolic dyshomeostasis, apoptosis, oxidative stress, and glucose utilization also play critical roles in AD etiology
- Data shows that NE3107's ability to reduce TNFα (the major regulator of inflammation) is highly correlated to improvements in cognition
  - We are pleased that the Principal Investigator observed these changes in 23 patients in only 3 months
  - We hypothesize that the modulation of TNFα levels and its inflammatory activation *via* TNFR1 lead to a multitude of changes among the many factors downstream from this master regulator, which collectively lead to improvements in neuronal health and cognition

NM101: Phase 3 trial in Mild to Moderate Alzheimer's Disease, NCT04669028

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

Remarkable Similarities Between Neurodegenerative Diseases

Neuroinflammation and oxidative stress are common features in the major neurodegenerative diseases, Alzheimer's, Parkinson's, frontotemporal lobar dementia and ALS

Remarkable parallels exist between AD and PD

- Activated microglia-inflammation
- Systemic inflammation driven
- TNF driven
- Insulin resistance, metabolic dysfunction
- Mitochondrial dysfunction and oxidative stress
- Endoplasmic reticulum stress- unfolded protein response
- Misfolded protein aggregates (alpha synuclein, Lewy bodies for PD)
- Controversial etiology, slow progression

Parkinson's Disease Clinical Development Program

NM201 Phase 2

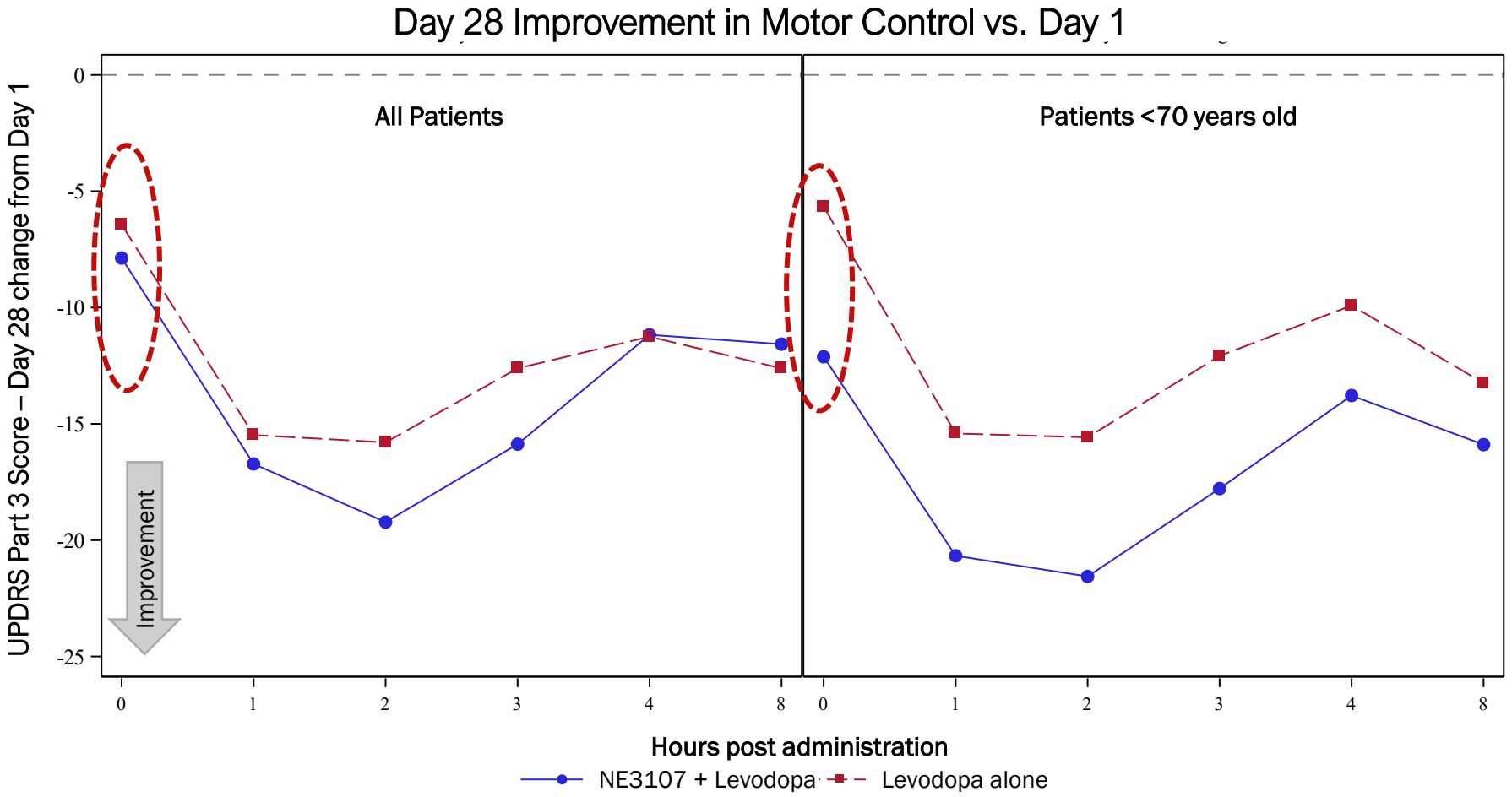
- Satisfying FDA requirement for drug-drug interaction study with L-dopa
- Detect efficacy signal for NE3107's pro-motoric activity

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

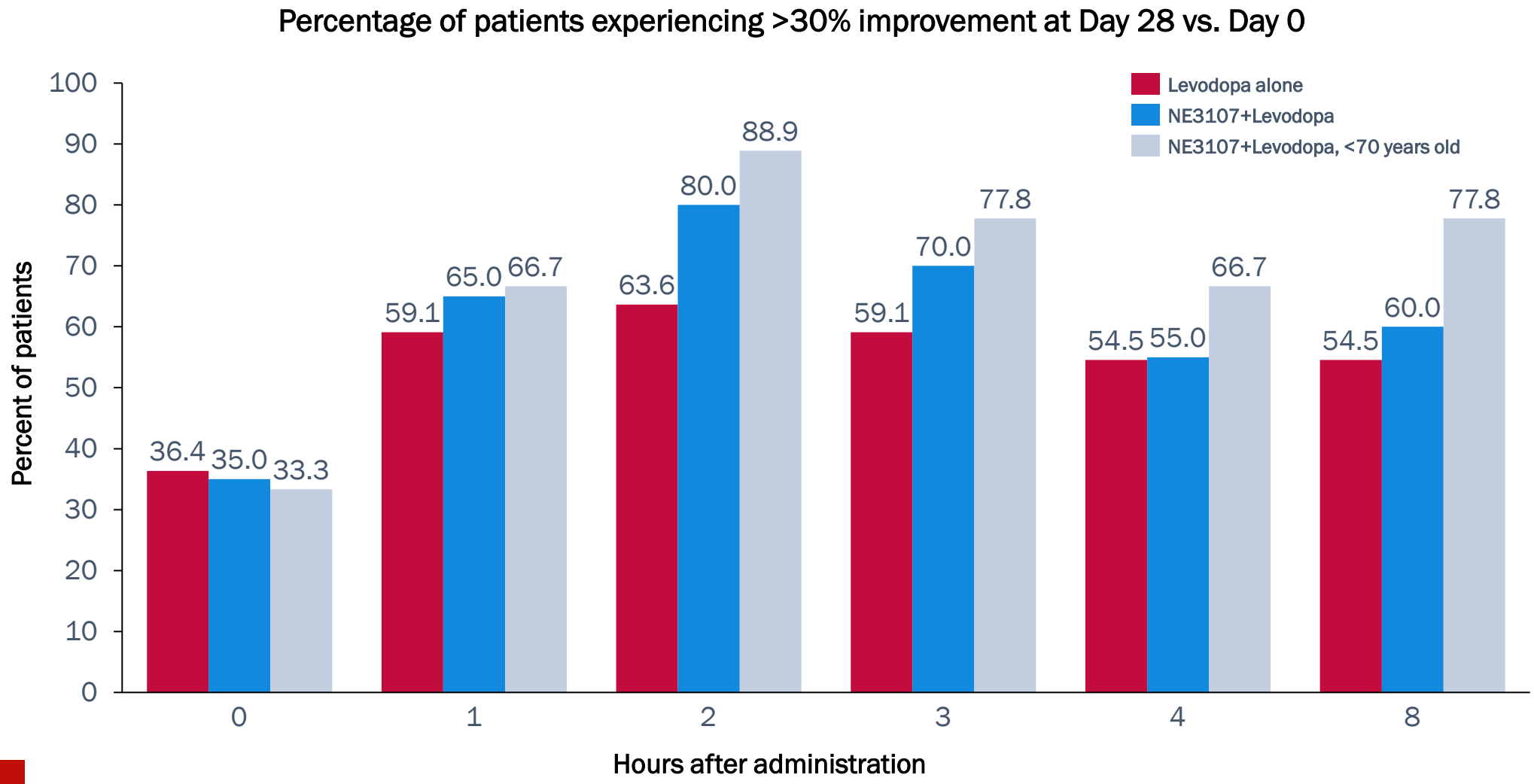
NE3107-treatment patients experienced fewer motor symptoms before morning drug administration



	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

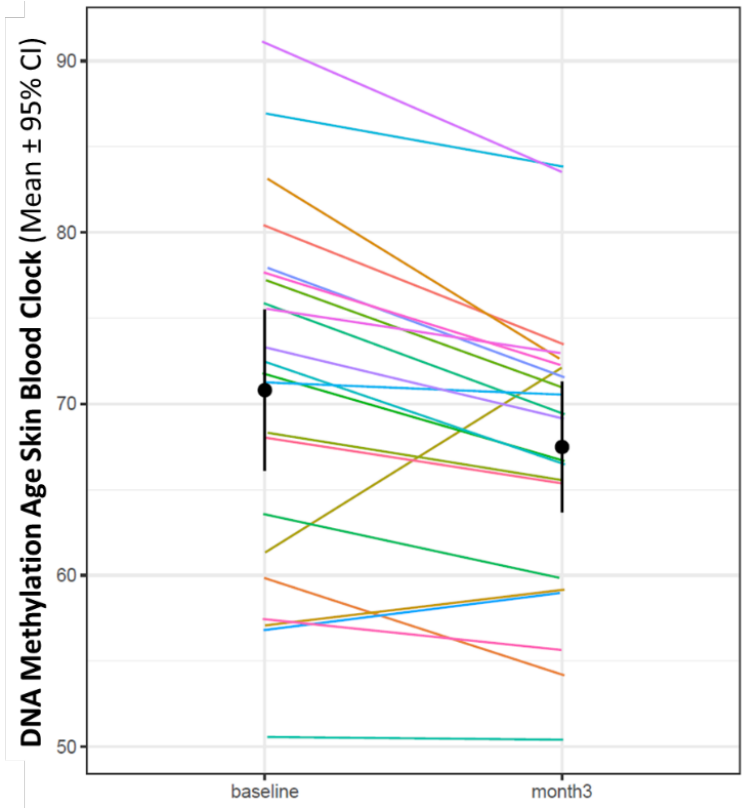


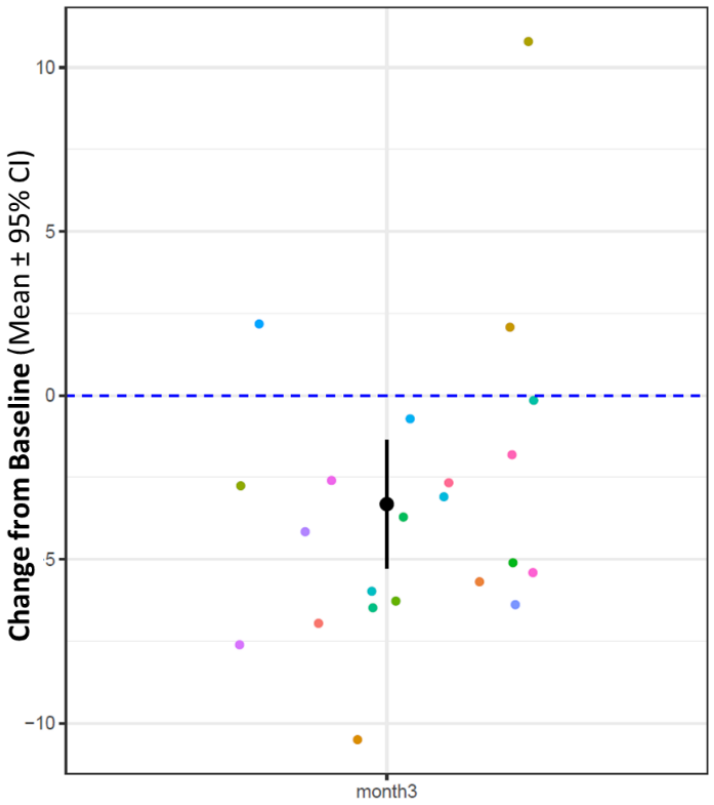
Background on biological aging

- Epigenetics is the study of how behavior (e.g., diet, exercise) and environment affect the way our genes work in addition to the genetic code itself
- Increasing body of evidence that signs of aging are epigenetic in nature
- DNA methylation is the process of how methyl groups are added or removed from DNA and thus regulate the expression of various genes in our bodies
- Genes become over- or under-methylated as we age, suggesting that the modulation of DNA methylation could modulate the aging process
- Dr. Steven Horvath, 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, is a leading authority on the study of DNA methylation, and the Horvath DNA methylation Skin & Blood clock predicts lifespan

NE3107's Impact on Biomarkers of Aging

- Blood samples were collected before and after 3 months of treatment with NE3107 in the completed Alzheimer's Investigator-Sponsored Phase 2 trial
- Samples analyzed by Dr. Horvath's team





19/22 decreased (86%)
Mean Absolute Change = -3.3 years (p=0.0021)

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

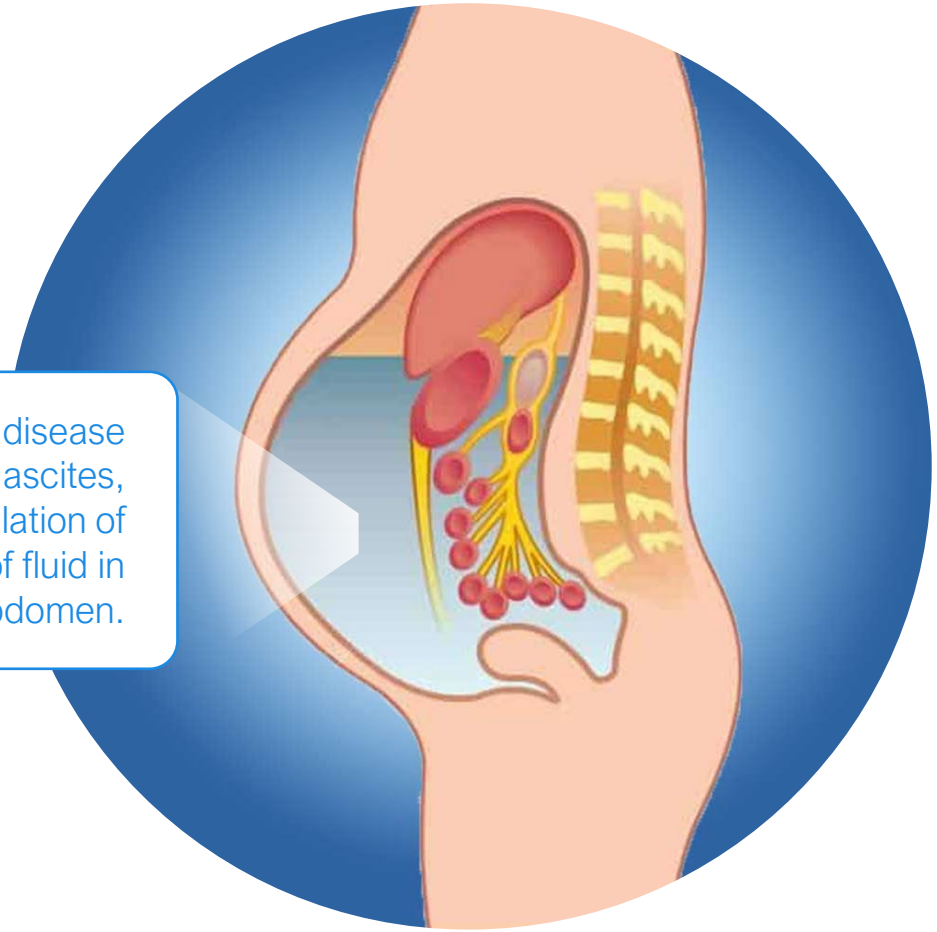
The kidneys are “burning out” by retaining massive quantities of salt and water

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



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

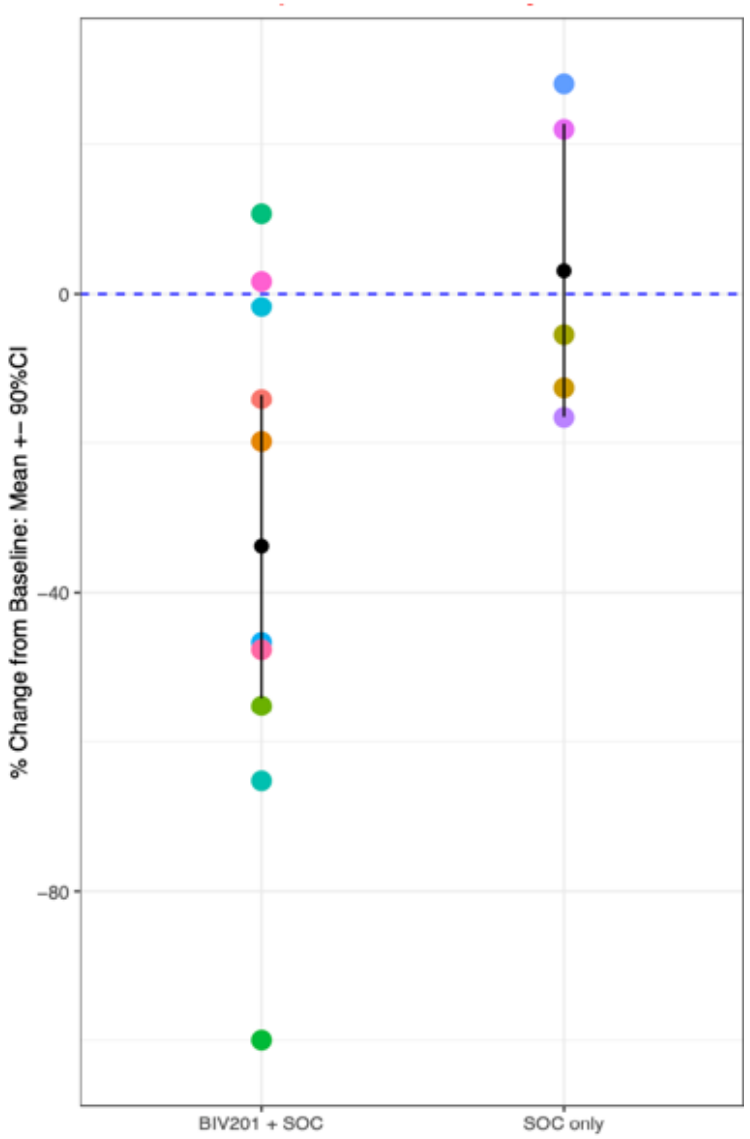
- Terlipressin administered as a continuous infusion
  - Outpatient treatment with small ambulatory infusion pump
- Targets the pathophysiology of ascites
  - Multiple small trials and Phase 2a support efficacy in reducing ascites
- Orphan and Fast Track Designations for the treatment of ascites due 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

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

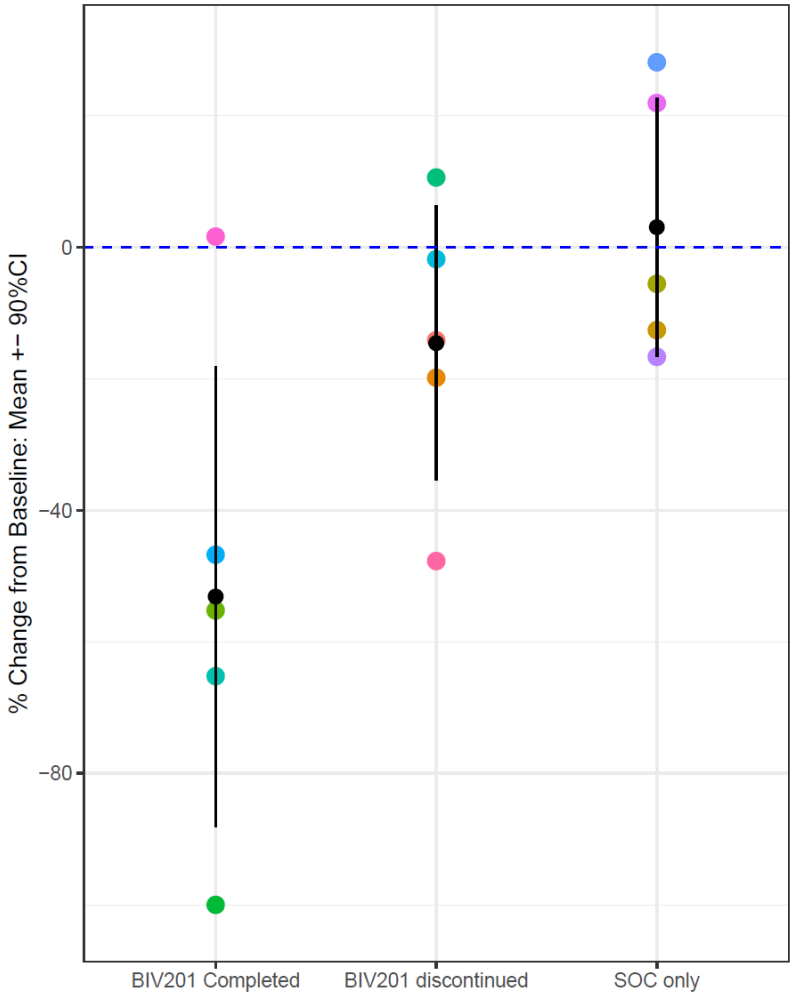


BIV201 + SOC
Mean: 34 % reduction
5/10 (50%) with >40% reduction
P=0.0046

SOC only
Mean: 3.1 % increase
0/5 (0%) with >40% reduction
P=0.8

BIV201 vs SOC
P value = 0.05 for difference

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
- $p<0.001$

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

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.

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

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

Clarence Ahlem , Neurodegenerative Disease Program
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 inflammation is starting point for many things going wrong in the body
  - NE3107 is our drug candidate that modulates both inflammation and the associated insulin resistance
  - NE3107 is currently in clinical development for Alzheimer's Disease (AD) and Parkinson's disease (PD)
- In Alzheimer's, a Phase 2 exploratory biomarker trial found that patients treated with NE3107 for 3 months experienced:
  - Reversal of their cognitive decline as measured by multiple assessment tools
  - Reduction of TNFα in manner that's correlated with cognitive improvements
  - Reduction phospho-tau production and the ratio of phosphor-tau to amyloid beta (Aβ)
  - Improvements in one or more brain regions as seen from advanced functional MRI studies among patients with abnormal scans at baseline
- In Parkinson's, a Phase 2 trial found that patients treated with NE3107 for 28-days experienced:
  - Improvements of UPDRS part 3 score on Day 28 compared to Day 0 that is 3+ points better than those treated with levodopa alone at the 2- and 3-hour marks. This level of superiority is considered by PD experts to be clinically meaningful
  - Improvements of 6+ points among patients younger than 70 years old (a surrogate for less disease progression)
  - Significantly more NE3107-treated patients maintainedmorning "on" symptoms in the morning compared to none of levodopa-alone patients
- In liver disease, BIV201 is in Phase 2b for refractory ascites. In trials thus far, patients have experienced in ascites fluid build up and extension of time between paracenteses with no unexpected drug-related SAEs

Thank You

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

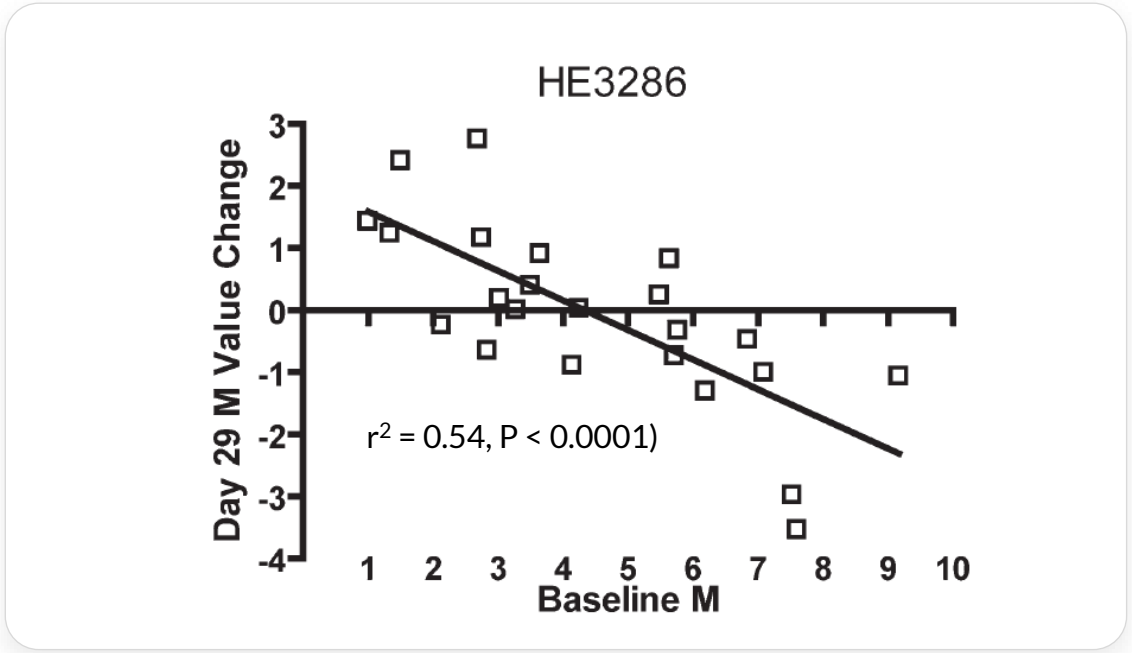
NE3107:

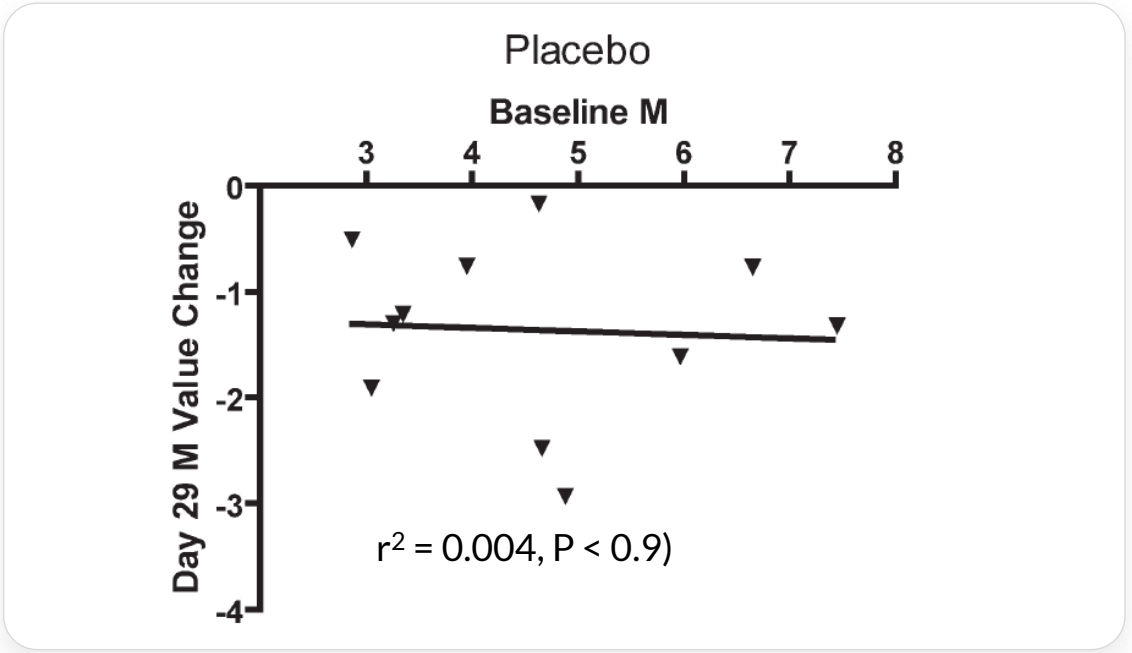
- Decreased insulin resistance, postprandial glucose and HbA1c compared to placebo
- Decreased inflammation-driven systems dysregulation of inflammatory, hematopoietic and metabolic parameters compared to placebo[4]
- Showed no differences in AEs compared to placebo

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





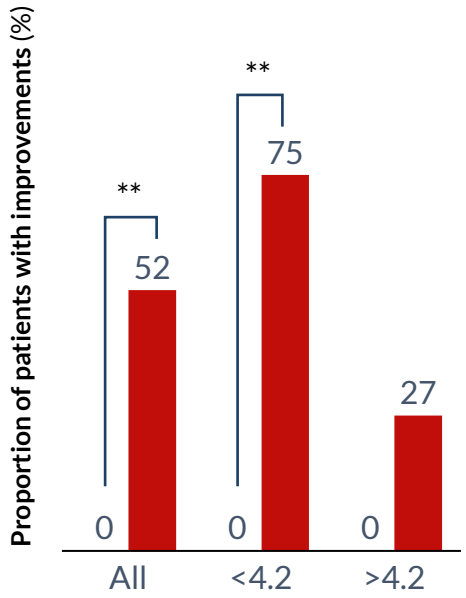
Day 29 Glucose disposal rate (M, measured in mg/kg/min in glucose clamp studies) value change as a function of baseline M

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

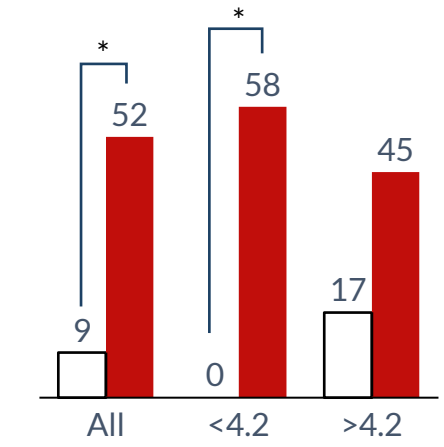
Insulin sensitizing improvement also brought improvements in AD indicators

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

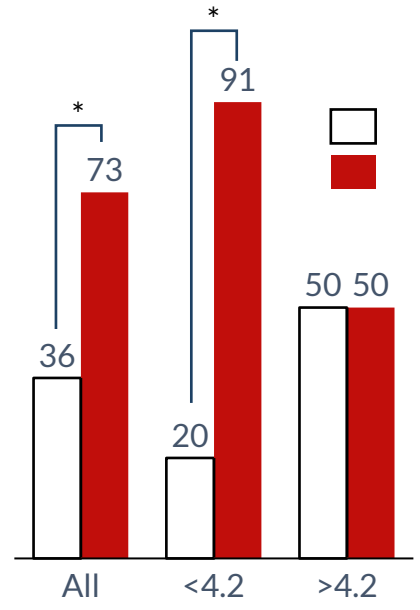
Increased insulin sensitivity …



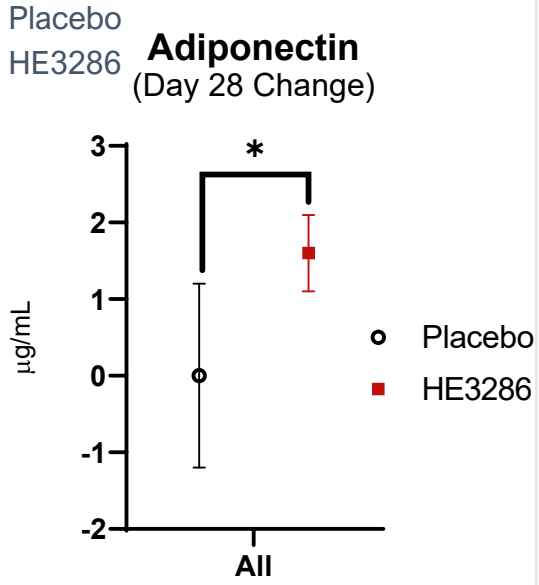
… increased HDL …



… decreased CRP …



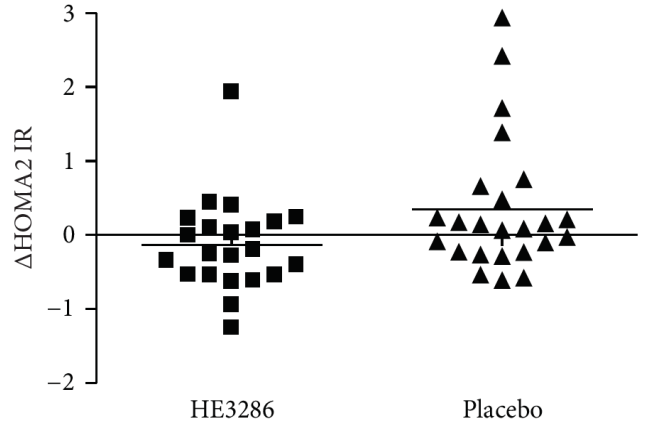
… and increased adiponectin



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

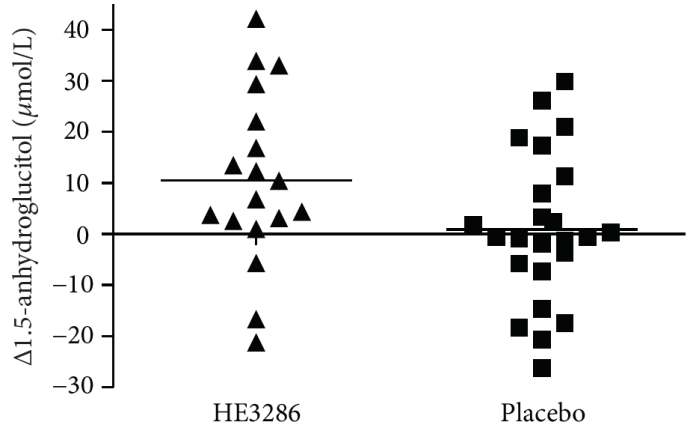
NE3107's ability to enhance insulin sensitivity reproduced in humans in Phase 2 trial with T2D patients

Improved the homeostatic model assessment of insulin resistance …



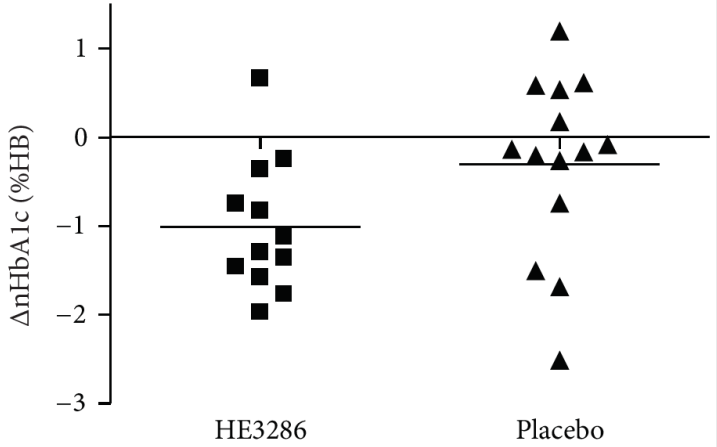
Cohort 1 baseline MCP-1 > 40 pmol/L individual subject changes in homeostatic model assessment of insulin resistance at Day 84

… decreased postprandial glucose …



Pooled cohorts 1 and 2 changes at Day 84

… decreased HbA1c …



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

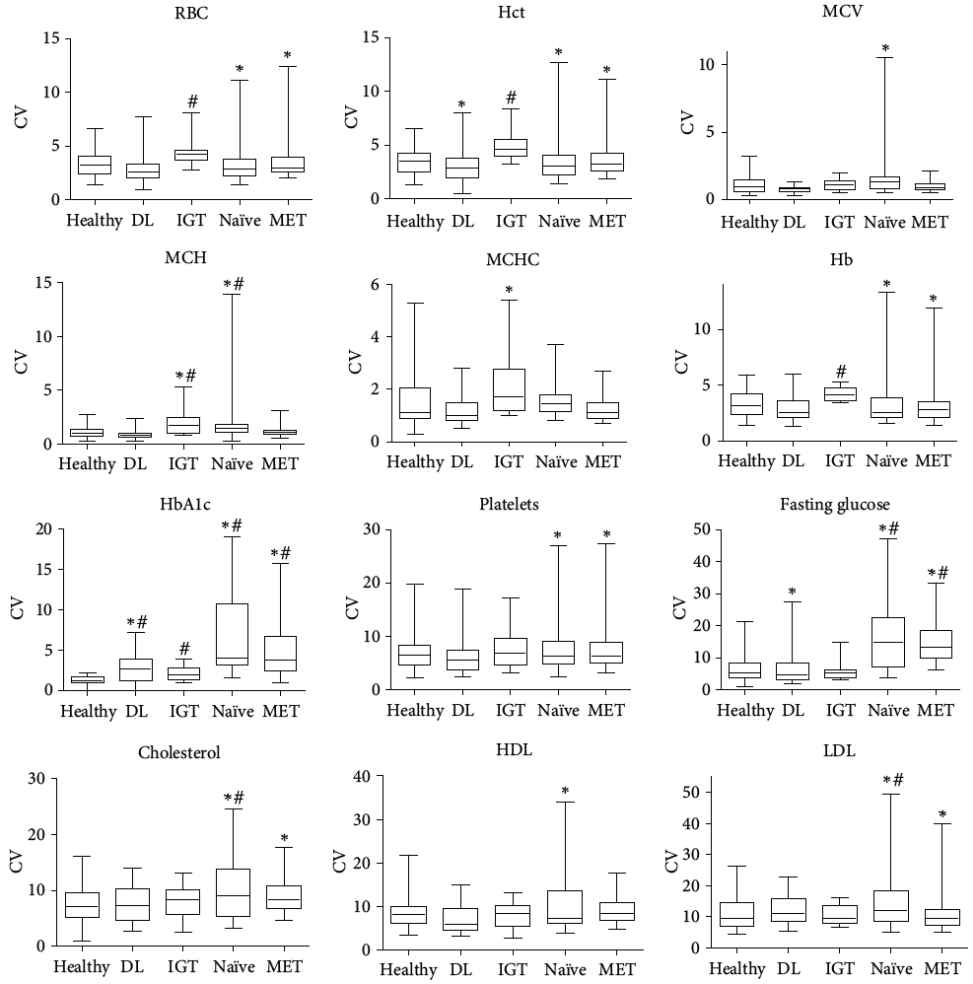
… decreased insulin resistance in inflamed T2D patients

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.

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

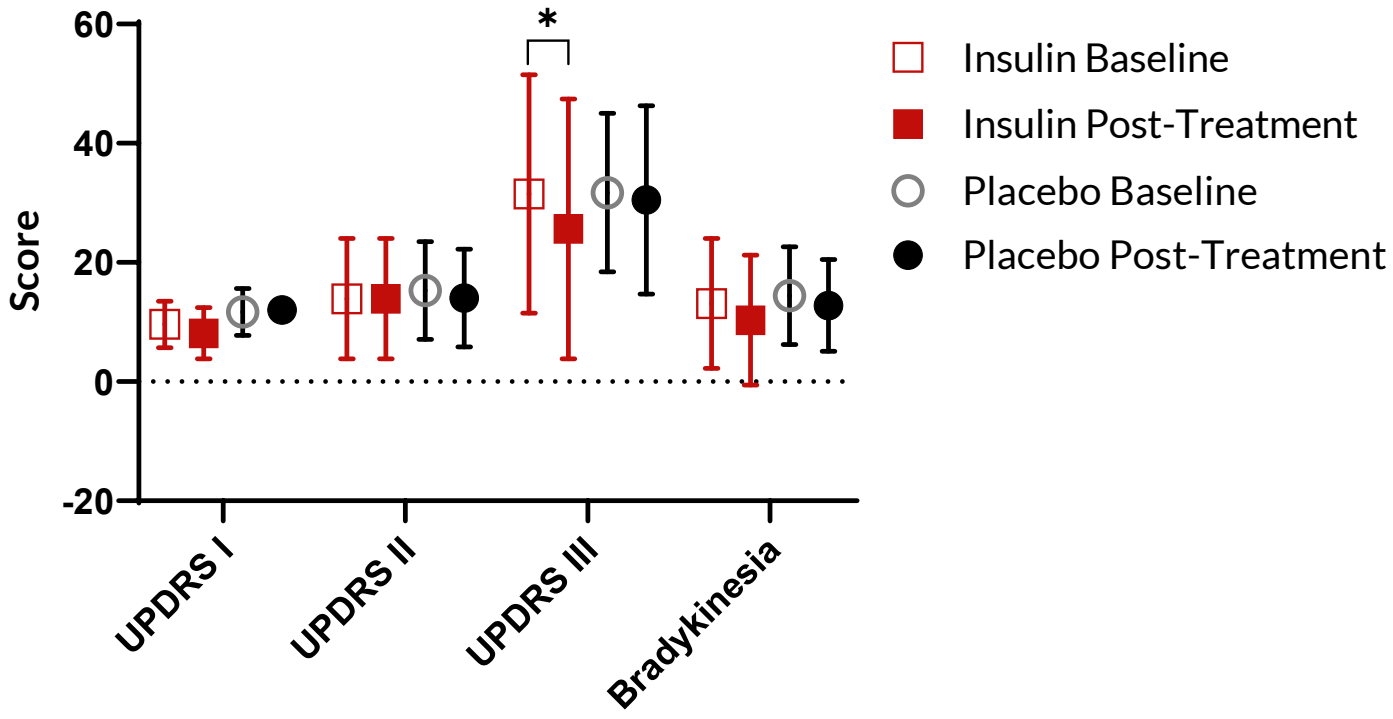
Group	Day	Parameter	HE3286 *W* test *P*	Placebo *W* test *P*
Cohort 1	84	ΔInsulin[d]	>0.1	**<0.0001**
		ΔC-peptide	>0.1	**<0.0001**
		ΔFasting glucose	>0.1	**0.02**
		ΔHOMA2 %B	>0.1	**<0.0001**
		ΔHOMA2 IR	>0.1	**0.002**
		Δleptin	>0.1	**0.005**
Cohort 1 MCP-1 > 40[b]	84	ΔHbA1c	>0.1	**0.006**
		ΔFasting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

The Role of Reduced Insulin Signaling in Parkinson's Disease

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

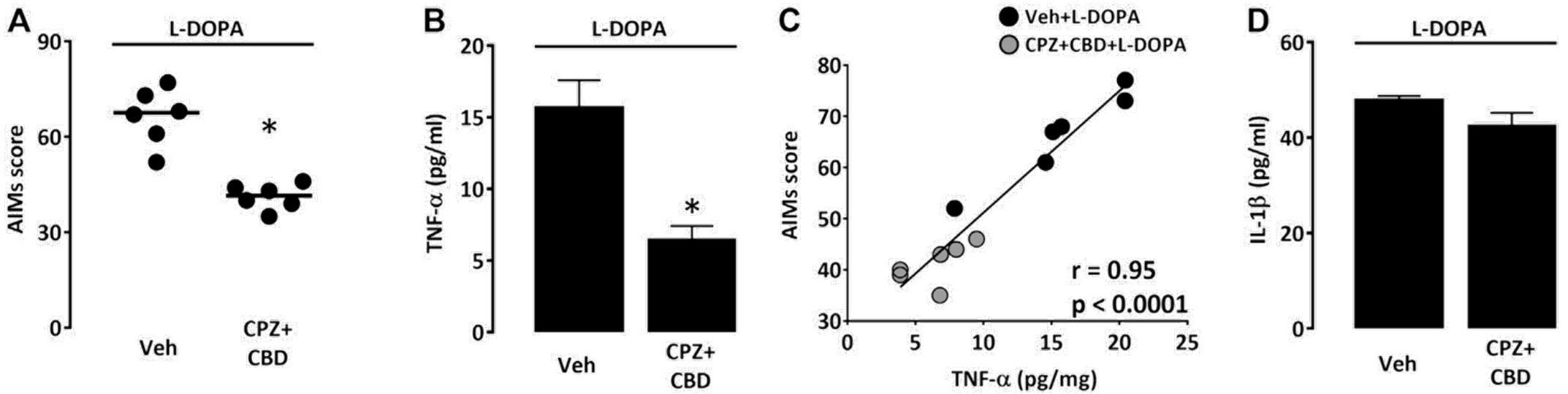
Intranasal Insulin Treatment Reduced Inflammation and Improved Motor Activity



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

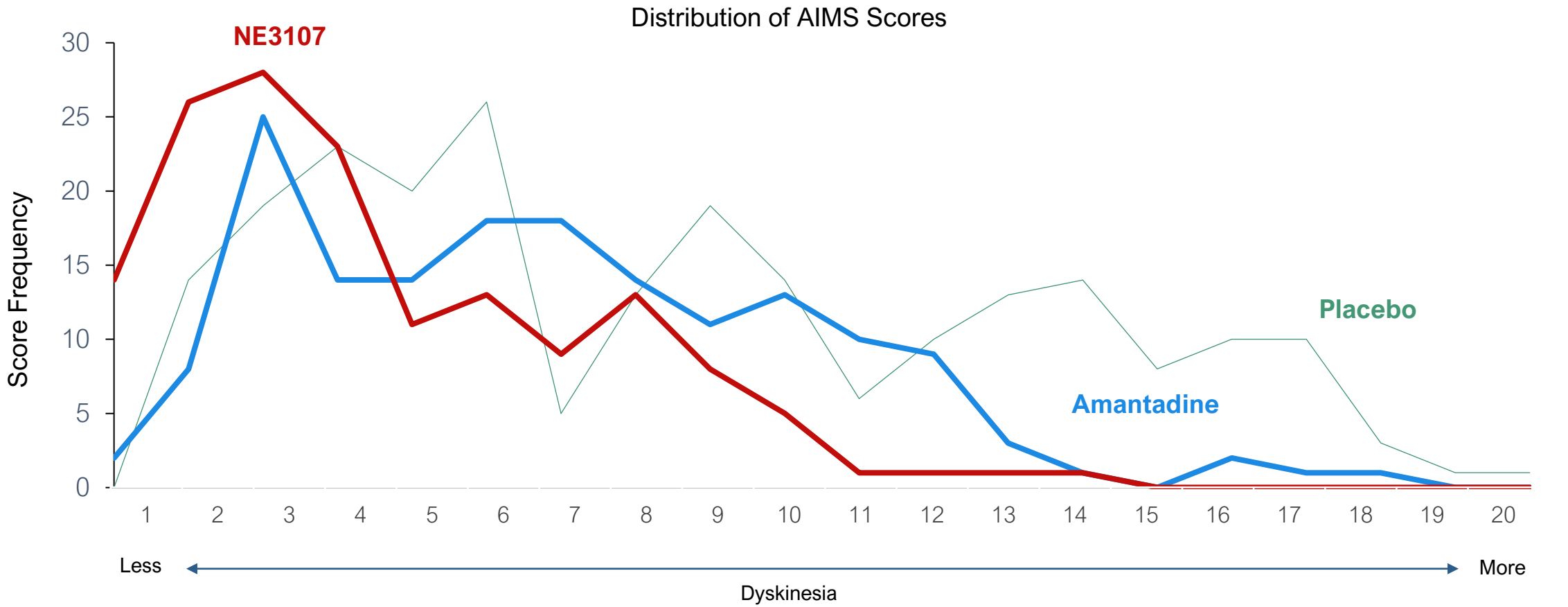
Reduction of TNF-induced inflammation reduces LID



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

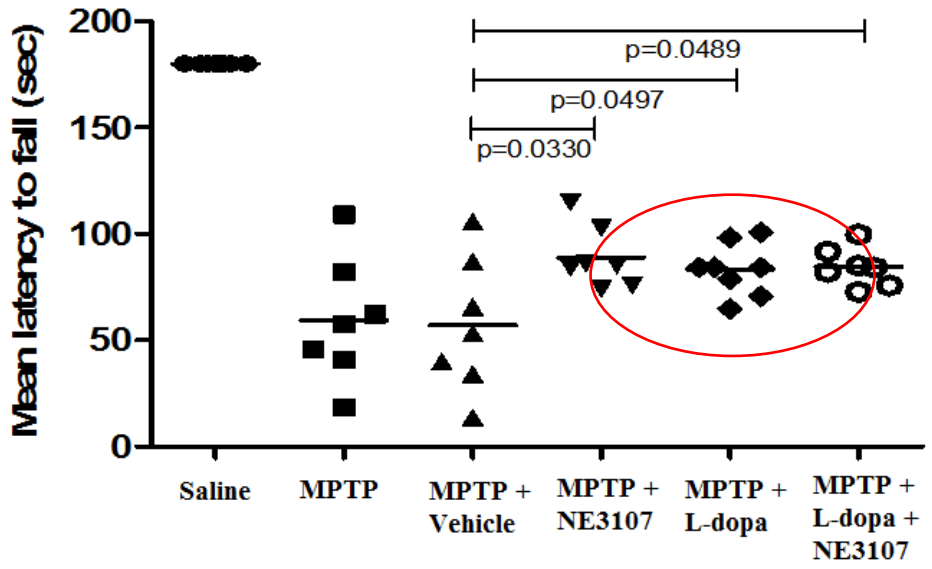
NE3107 decreases L-Dopa-induced dyskinesia (LID) in marmosets



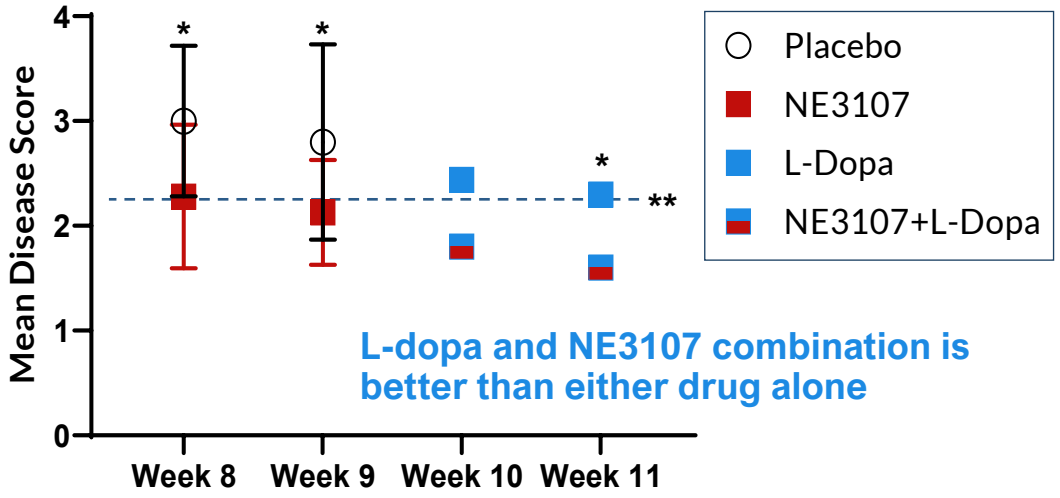
Abnormal Involuntary Movement Scale (AIMS)

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

MPTP Mouse



MPTP Marmoset treated at Week 8

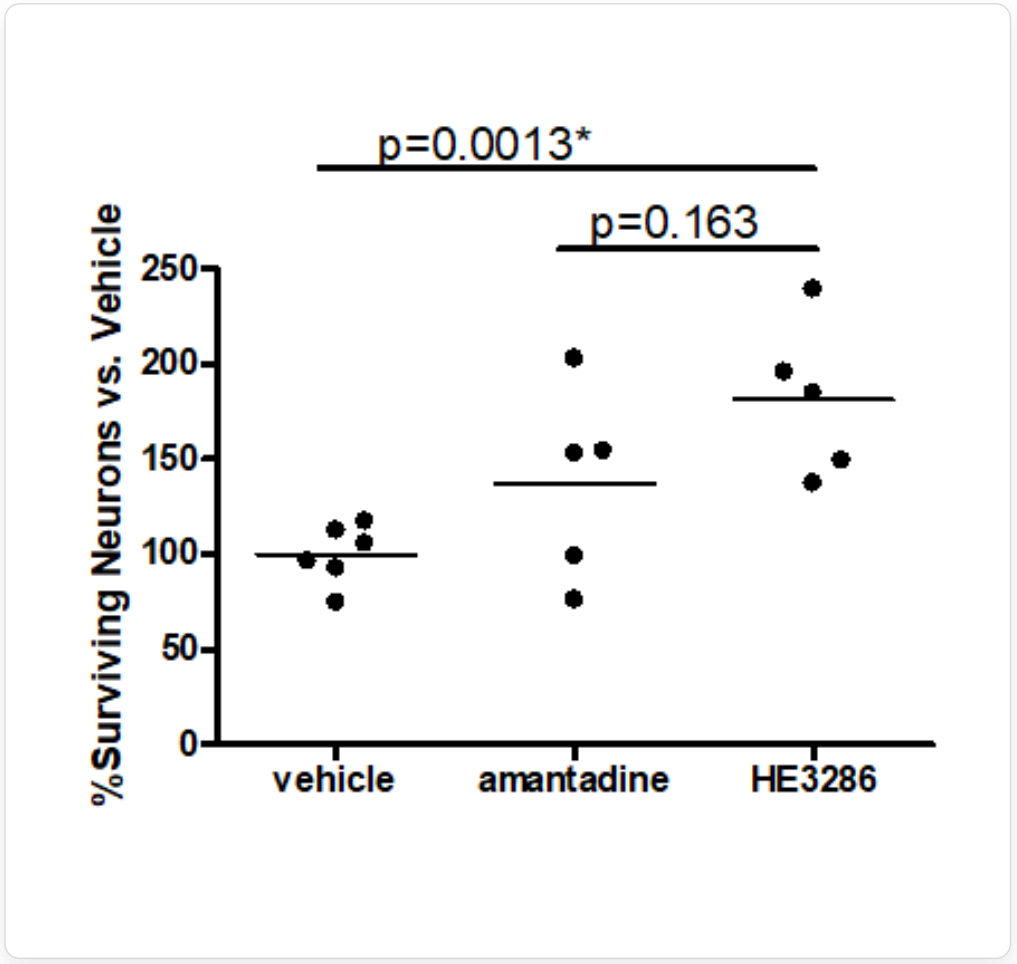


*p<0.001 compared to other treatment arm in time period
** L-Dopa at week 11 not statistically different from NE3107 at weeks 8 and 9

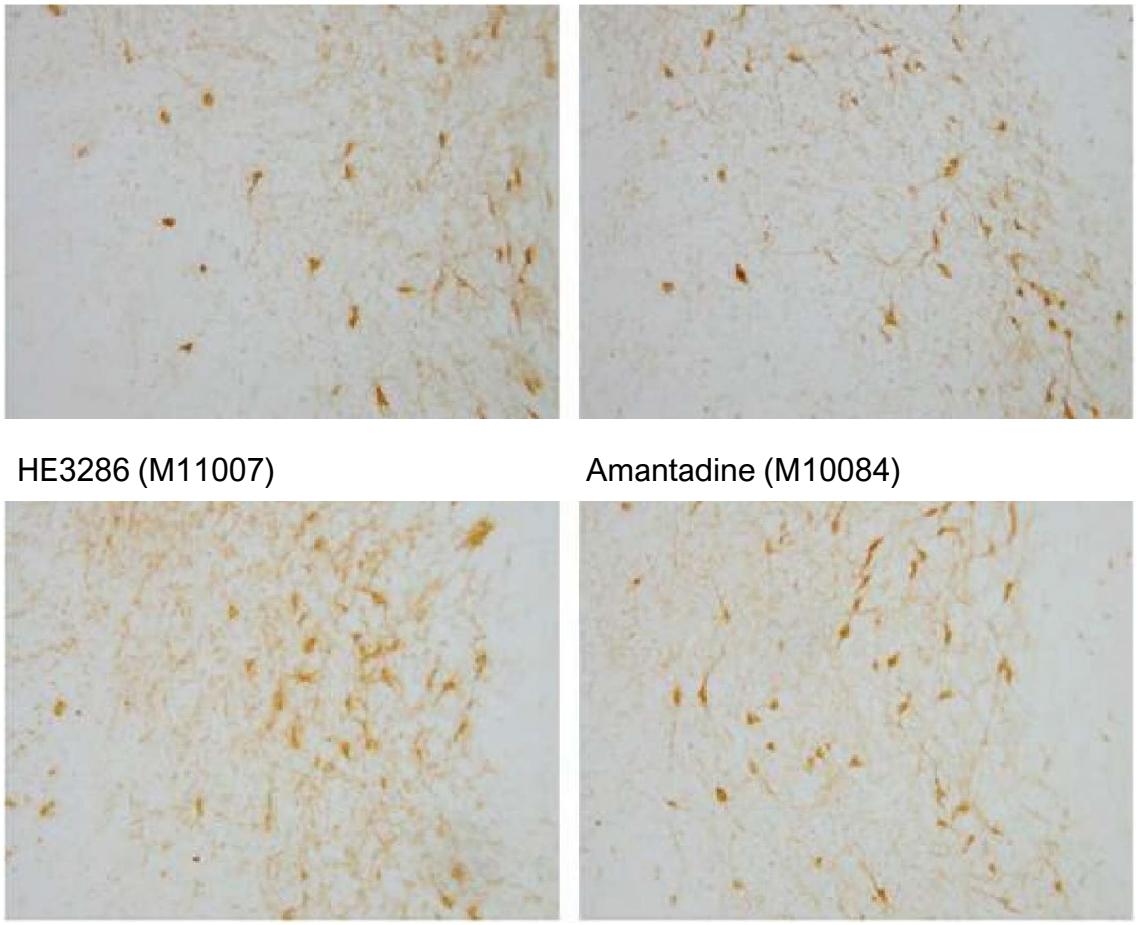
NE3107's promotoric effects observed within 4 days of treatment

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)

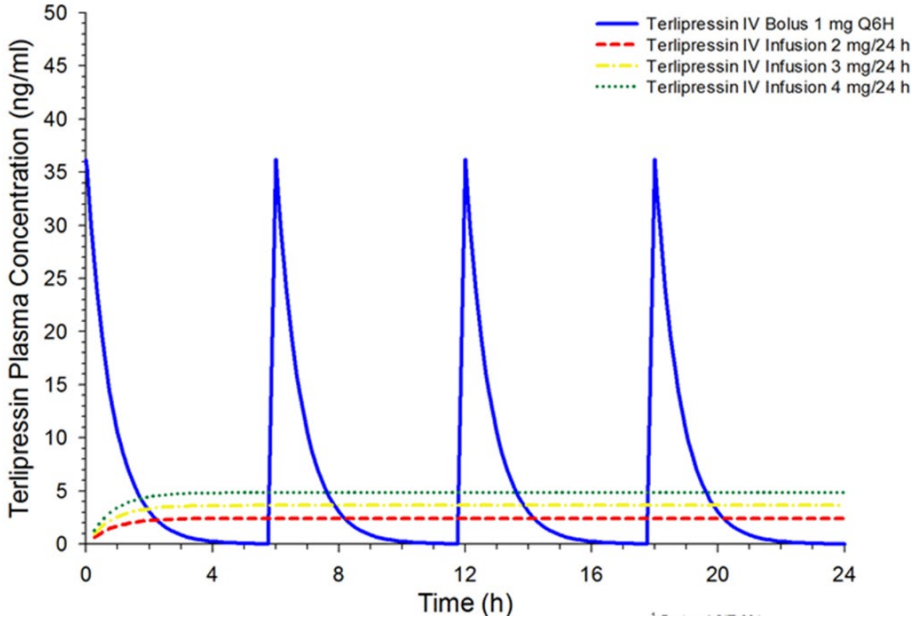
Amantadine (M10084)

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

BioVie Phase 2a trial results: BIV201 Pharmacokinetics

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

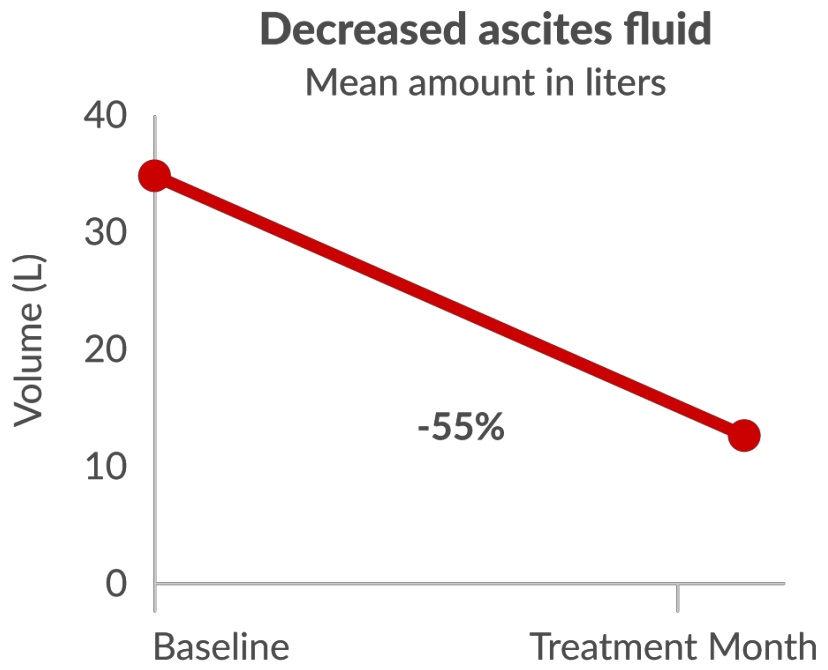
Comparison of the PK Profile of Terlipressin Administered by Continuous Infusion or Intermittent IV Boluses.

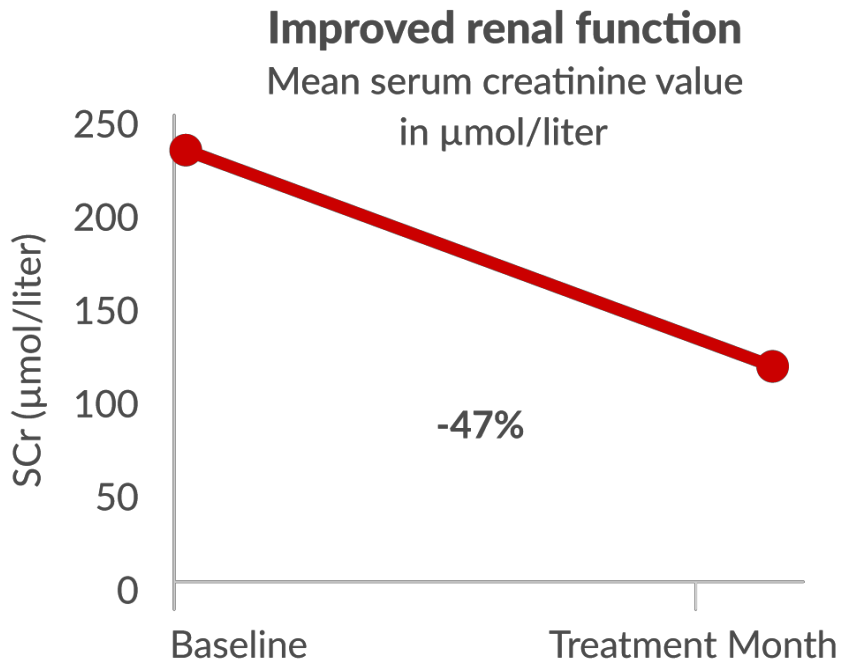


Simulated terlipressin plasma concentrations following IV bolus of 1 mg every 6 hours 1 [Lucassin, AusPAR-cer 2011] and IV continuous infusions of 2 mg, 3 mg, or 4 mg per day

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

Continuous Infusion Terlipressin in 6 Refractory Ascites/HRS Patients*





*(Data licensed from Dr. P. Angeli); Source: Adapted from BioVie US Patent application 16/379,446 Angeli et al. Mean results during one month of therapy compared to month prior to starting therapy.. No serious drug-related side effects were reported in this study. Terlipressin is not available in the US

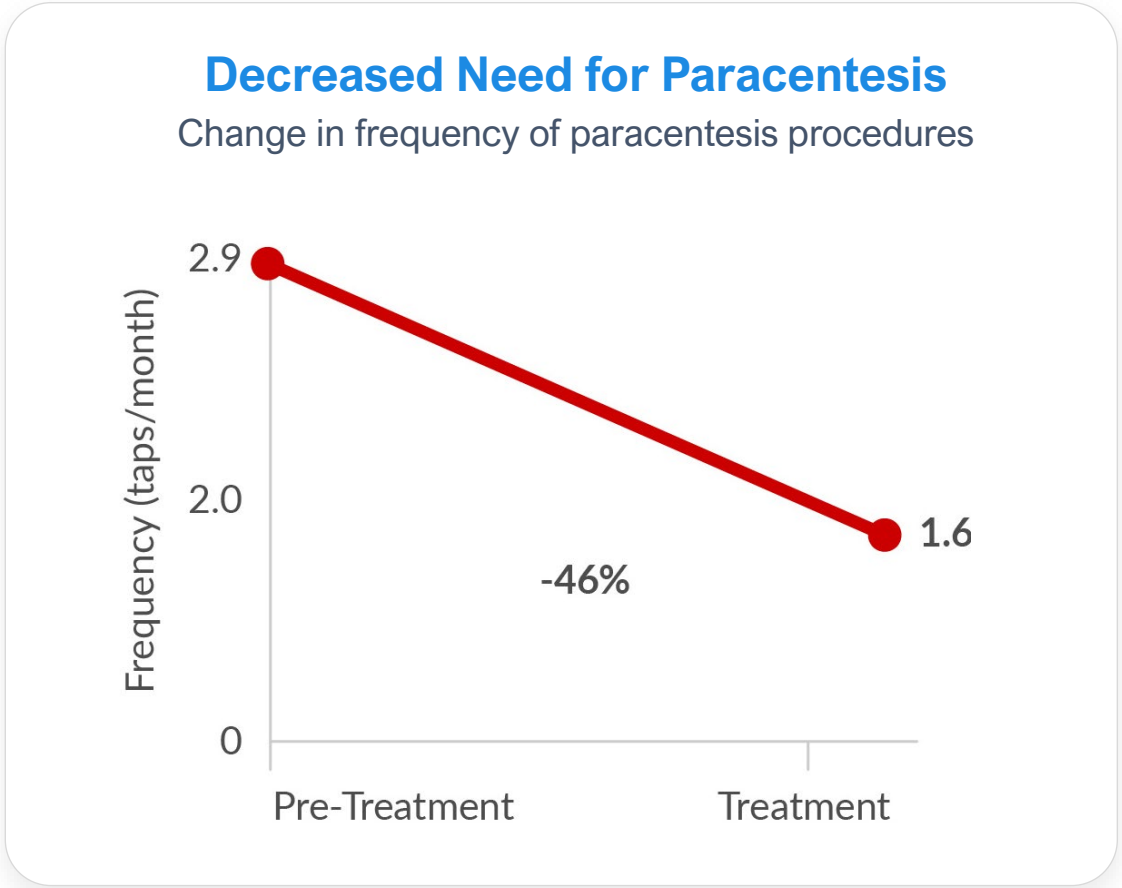
Continuous Infusion Terlipressin in 19 Refractory Ascites/HRS Patients

Pre-therapy:

- 70% of patients required weekly large volume paracentesis (LVP)
- 63% poor muscle strength

Results:

- Median duration of CI terlipressin treatment: 51 days
- 46% average reduction in frequency of paracentesis
- Significantly improved muscle strength and nutritional intake
- No complications directly attributable to terlipressin



Source: JHEP 2019. Frequency of paracentesis over 30 days compared to average over 30 days during 3-month period prior to therapy; (Chapman et al.. Terlipressin is not available in the US)

