

Developing Transformative Therapies to Overcome Chronic Debilitating Diseases

Corporate Presentation • January 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.

Overview

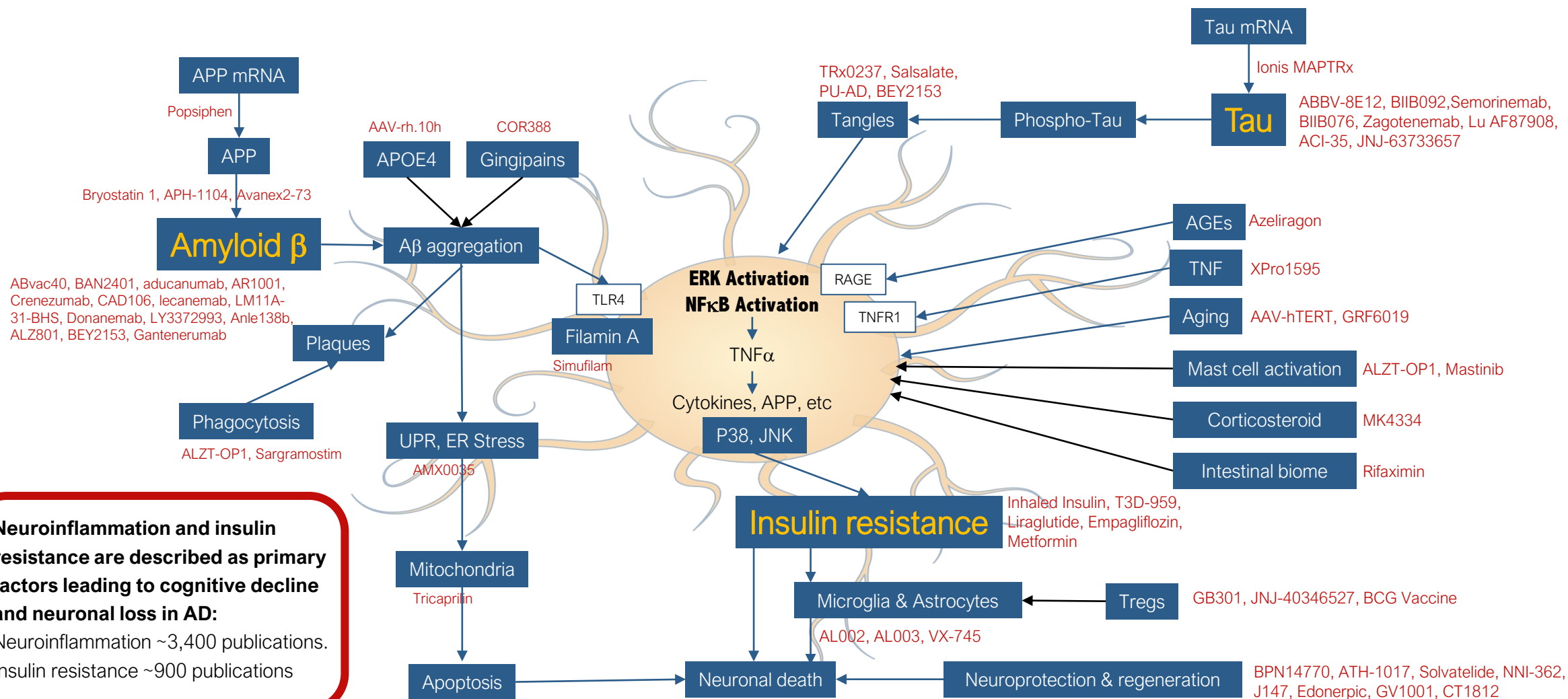
- We believe that inflammation is starting point for many things going wrong in the body
 - NE3107 is our drug candidate that modulates both inflammation and the associated insulin resistance
 - NE3107 is currently in clinical development for Alzheimer's Disease (AD) and Parkinson's disease (PD)
- In 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 TNF α in manner that's correlated with cognitive improvements
 - Reduction phospho-tau production and the ratio of phosphor-tau to amyloid beta (A β)
 - 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)
- In liver disease, BIV201 is in Phase 2b for refractory ascites. In trials thus far, patients have experienced:
 - Reductions in ascites fluid build up and extension of time between paracenteses
 - No drug-related SAEs

Expected Catalysts & anticipated timelines (US)

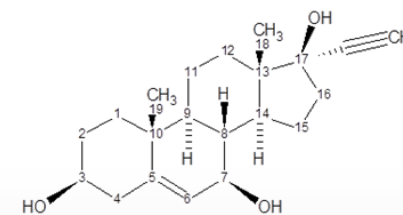


 File NDA
  Launch

Alzheimer's Disease Pathways



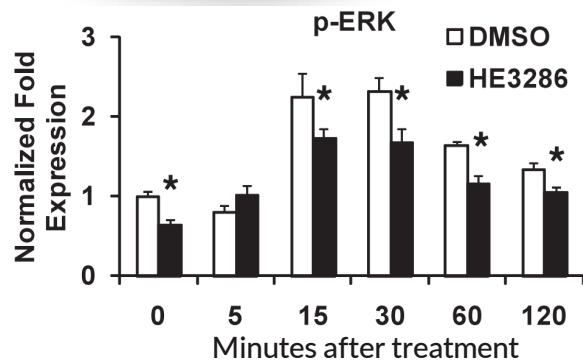
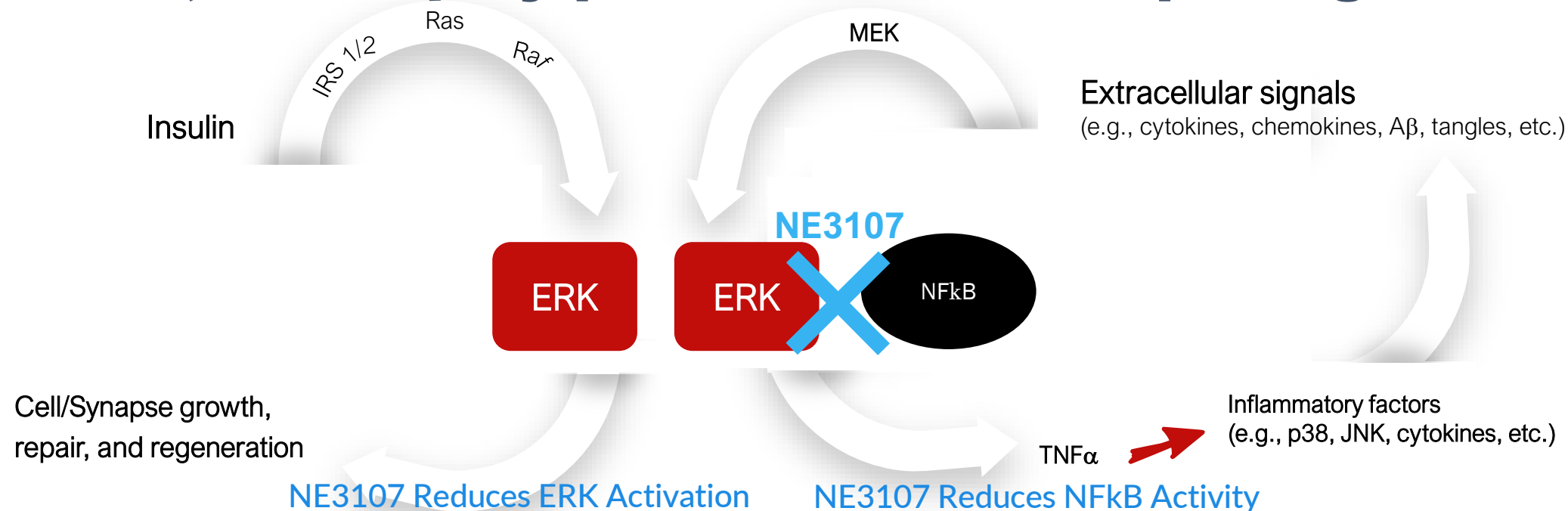
Background on NE3107



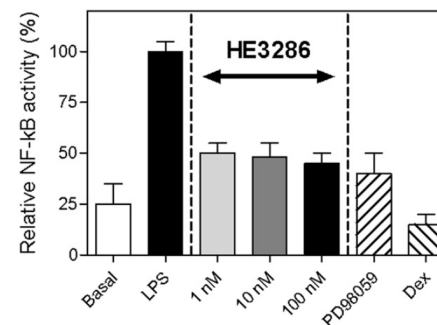
First-in-class molecule with desirable characteristics

- 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)
 - NE3107¹ 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

NE3107 blocks inflammatory (but not homeostatic) ERK and NFkB, which play pivotal roles in AD pathogenesis¹



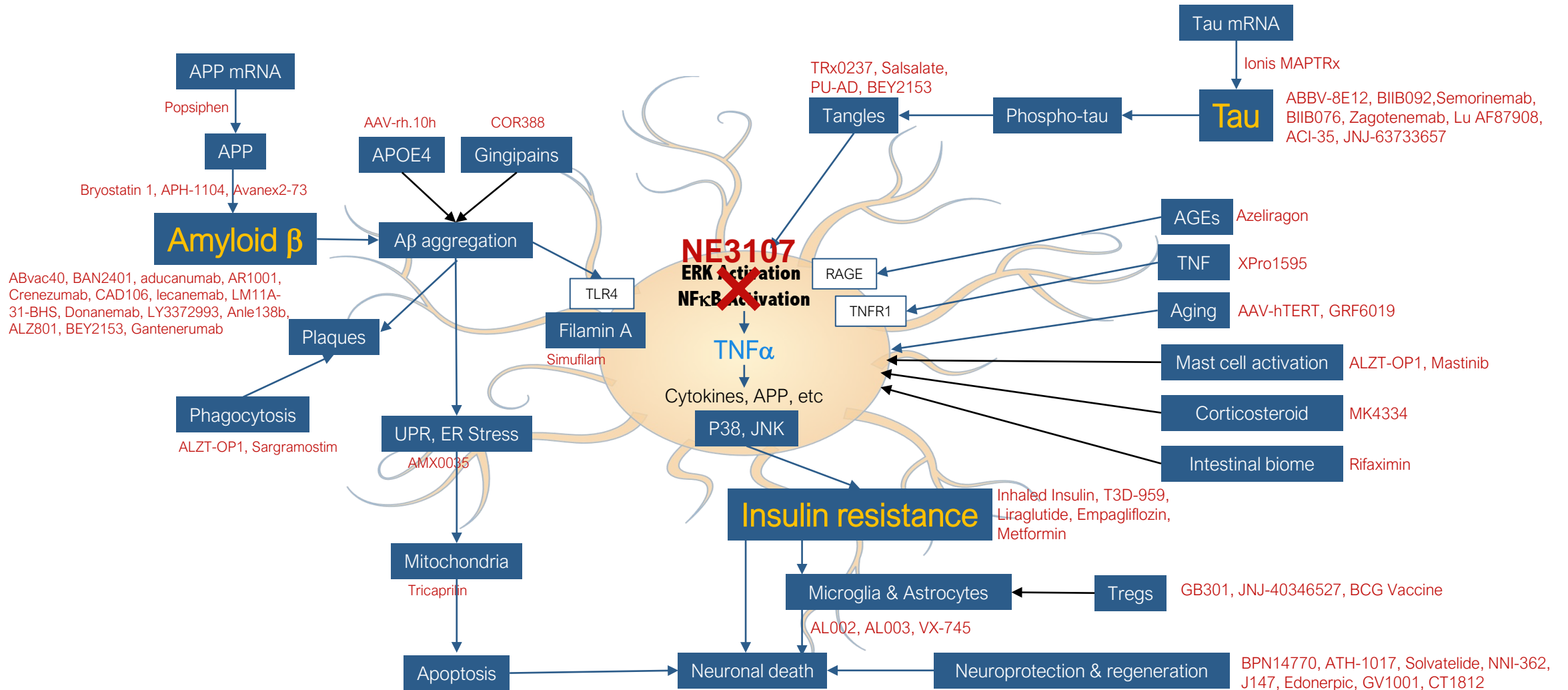
Lu 2010 Am J Physiol Endocrinol Metab 298 E1036



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

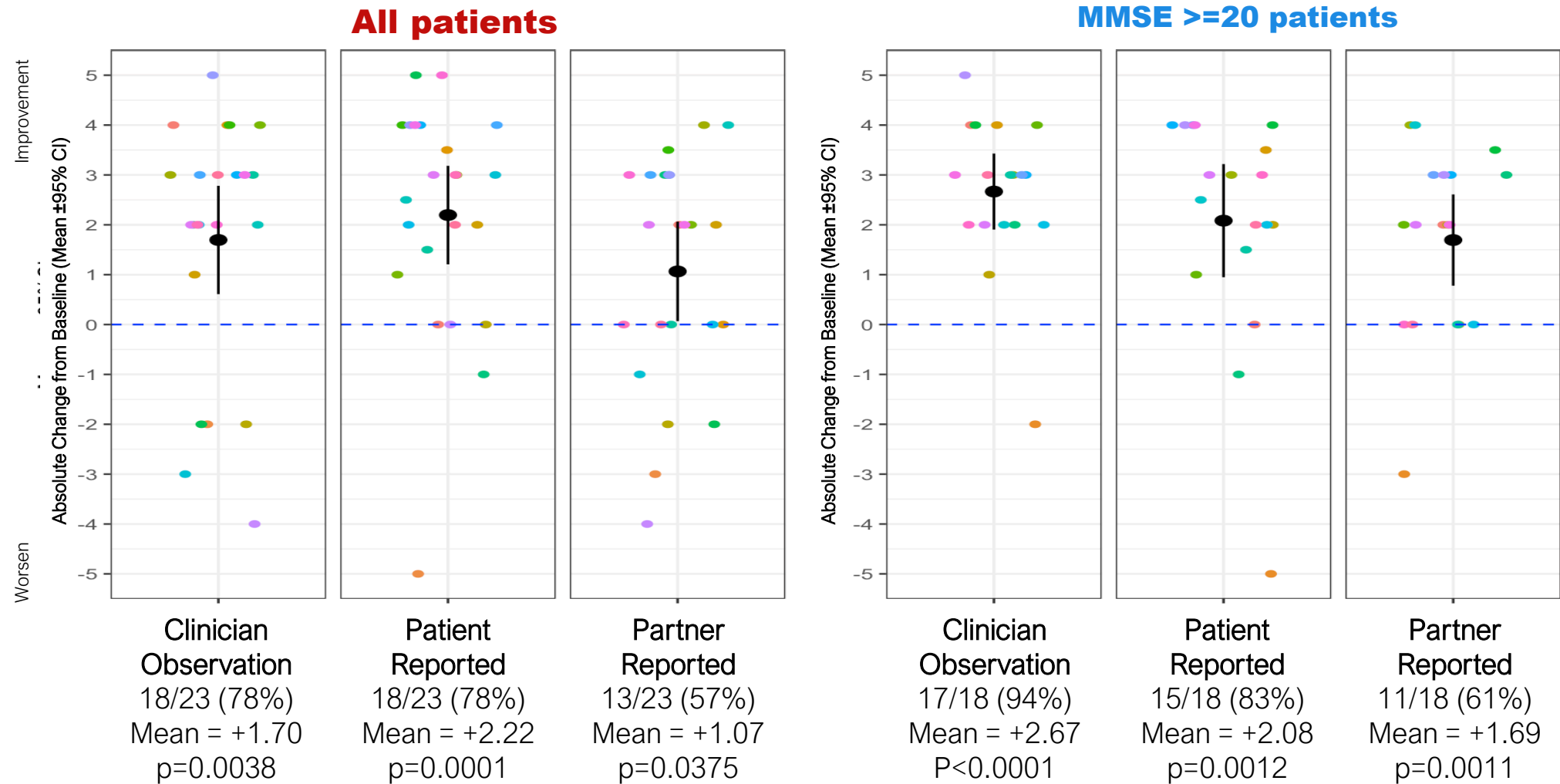


Investigator-Sponsored Trial in MCI and Mild Alzheimer's Disease, NCT05227820

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

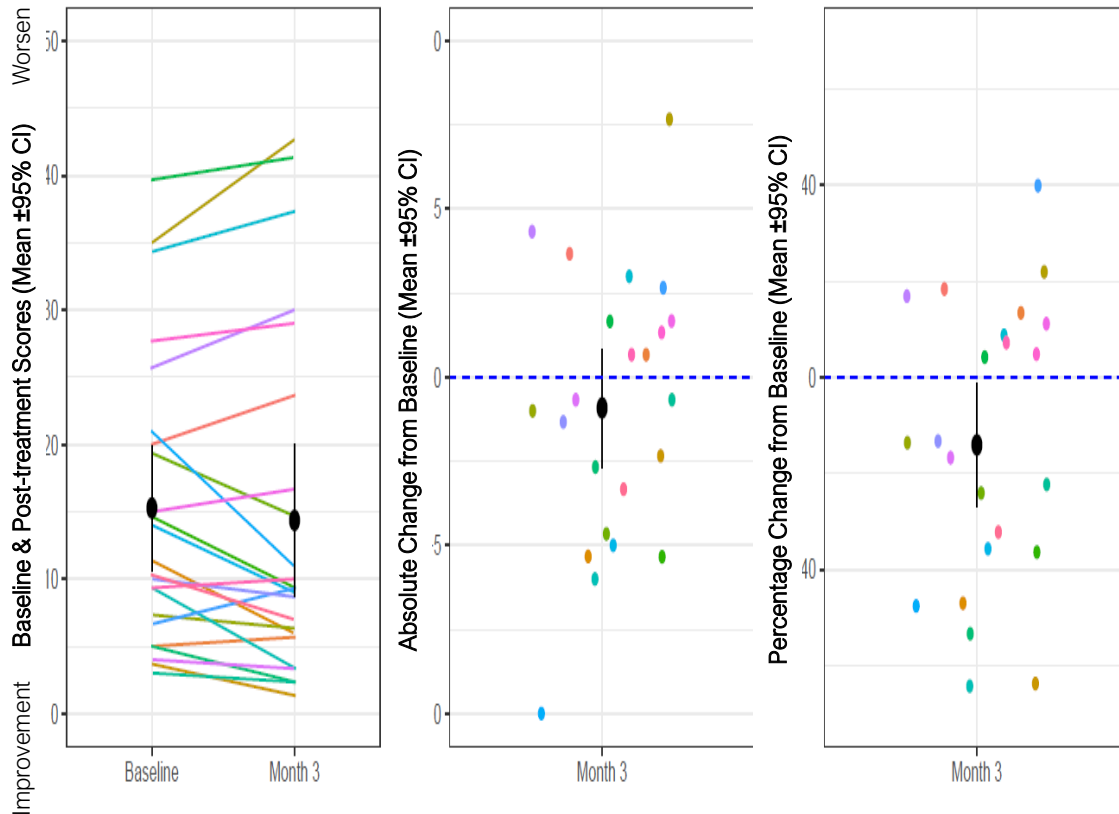
Large majority of patients improved significantly on the Global Rating of Change (overall impression)



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

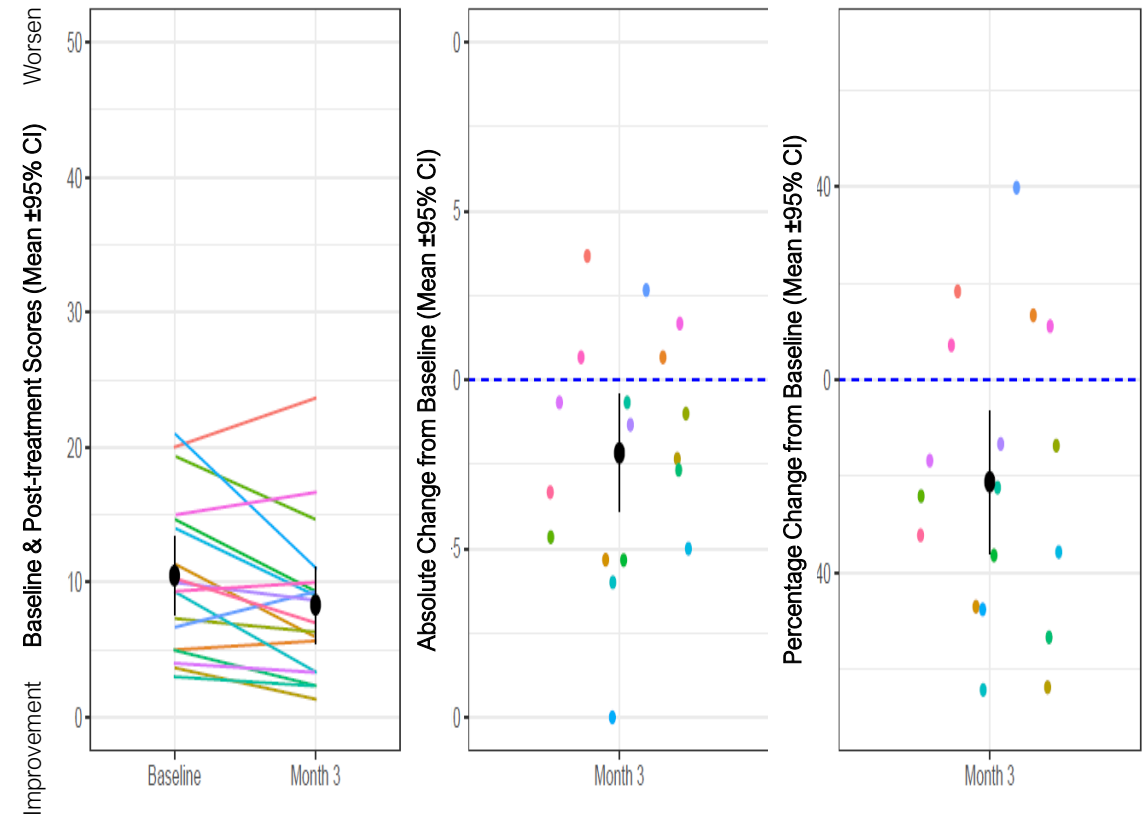
Significant improvements in ADAS-Cog12 assessment of cognition among MCI/Mild AD patients

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

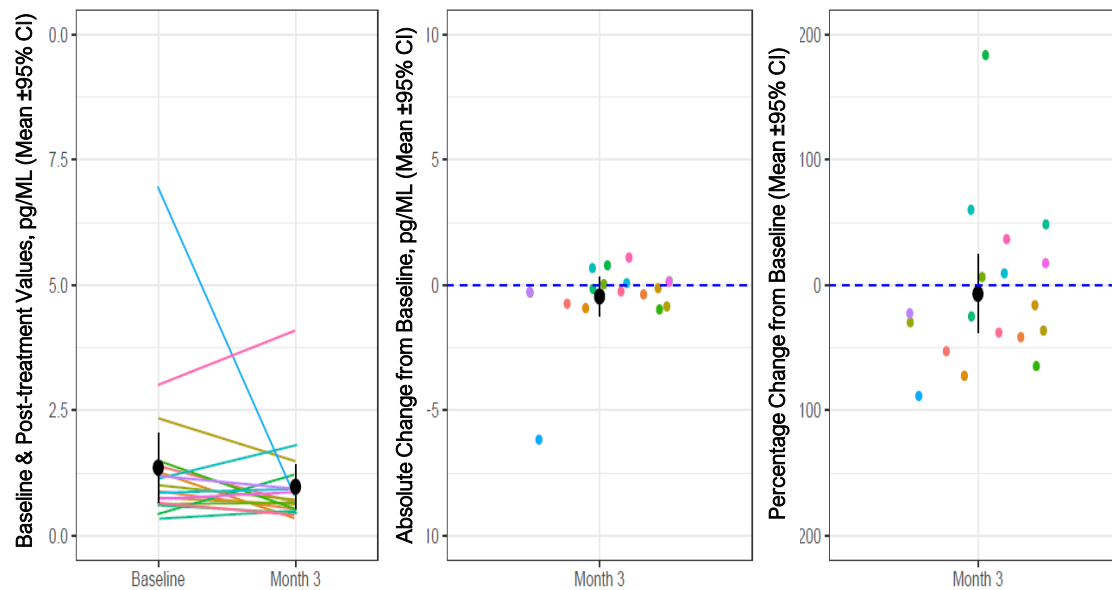
Green = Improvement

* p<0.05

Improvements on $\text{TNF}\alpha$ among MCI/Mild AD patients

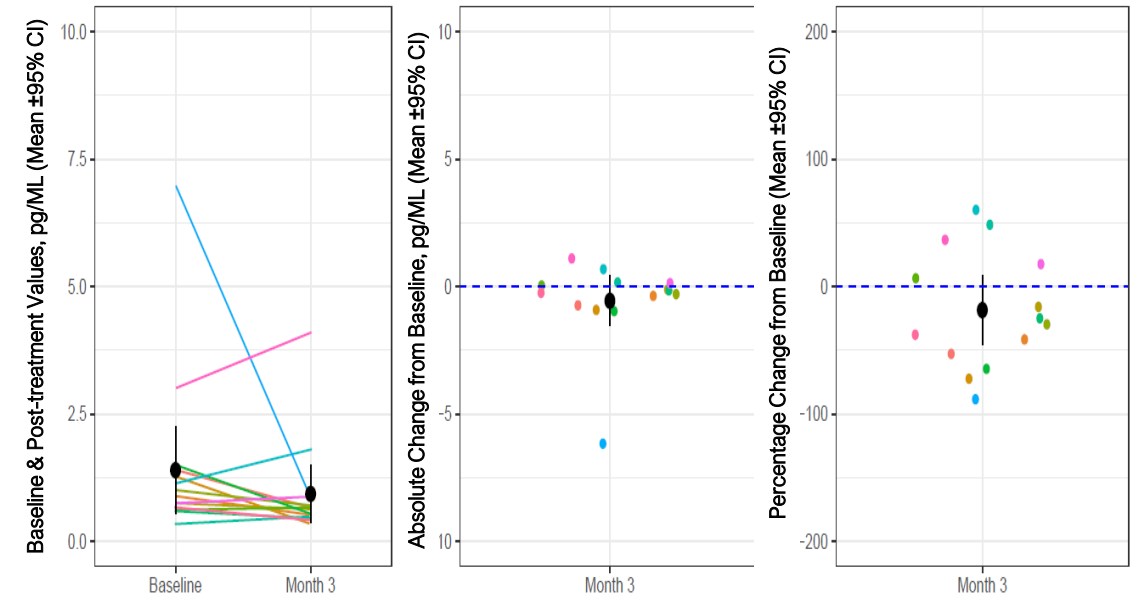
Tumor Necrosis Factor Alpha ($\text{TNF}\alpha$) is a cytokine identified as a major regulator of inflammation whereby its excessive activation is associated with chronic inflammation¹

All patients



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

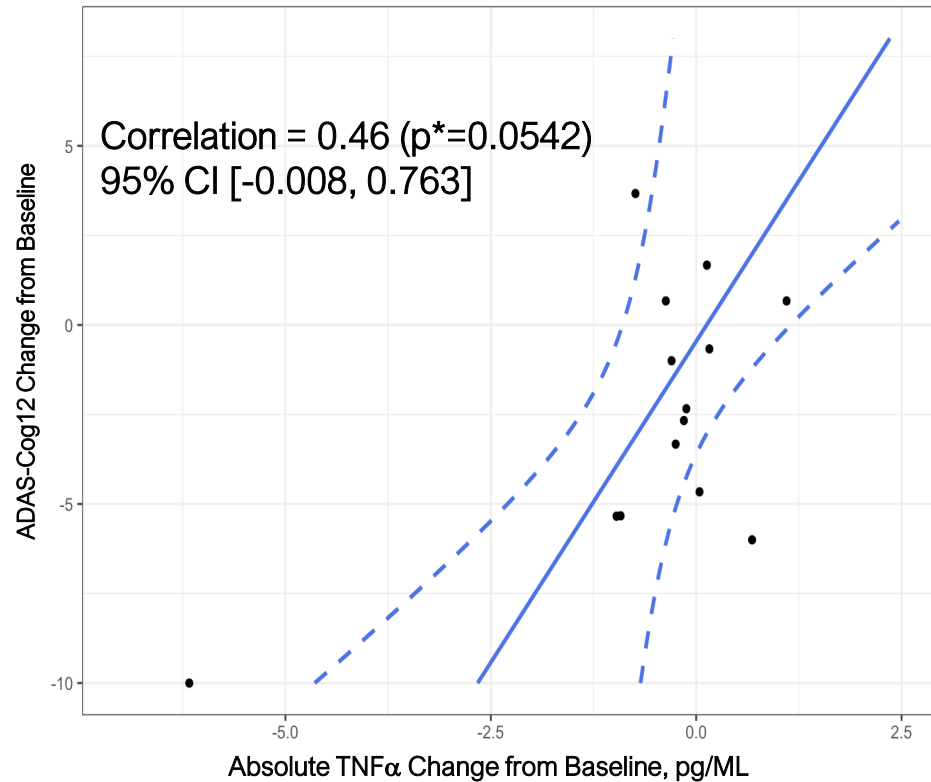
MMSE ≥ 20 patients



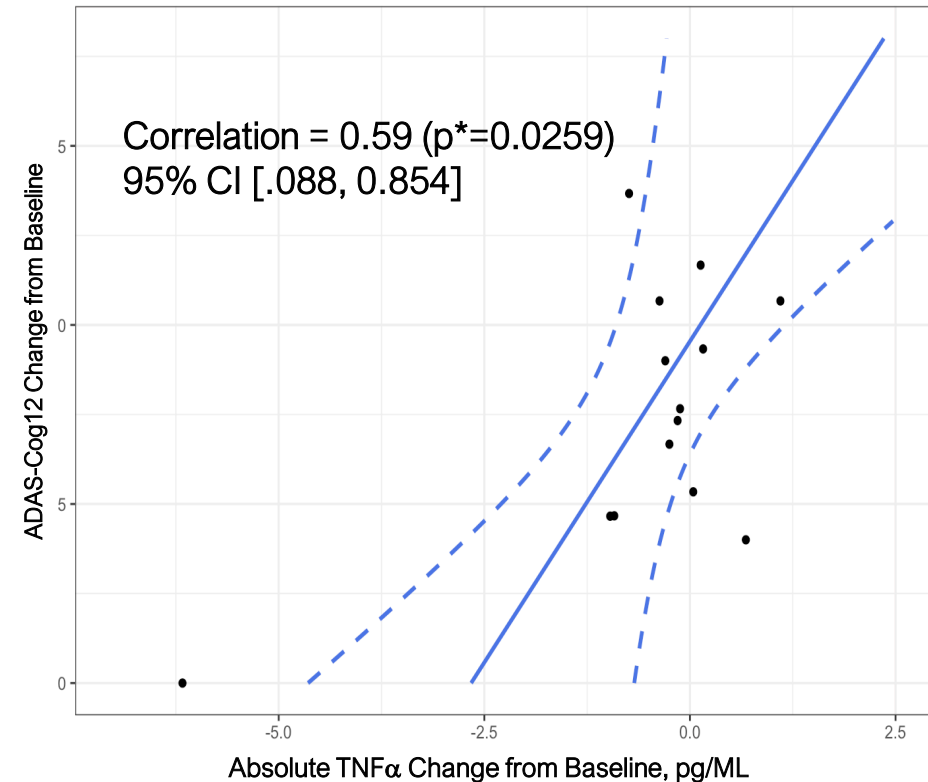
9/14 improved (64%)²
Mean Absolute Change = -0.563 (p=ns)
Mean % Change = -18.5% (p=ns)

Improvements in $\text{TNF}\alpha$ significantly correlated to improvements in ADAS-Cog12

All patients

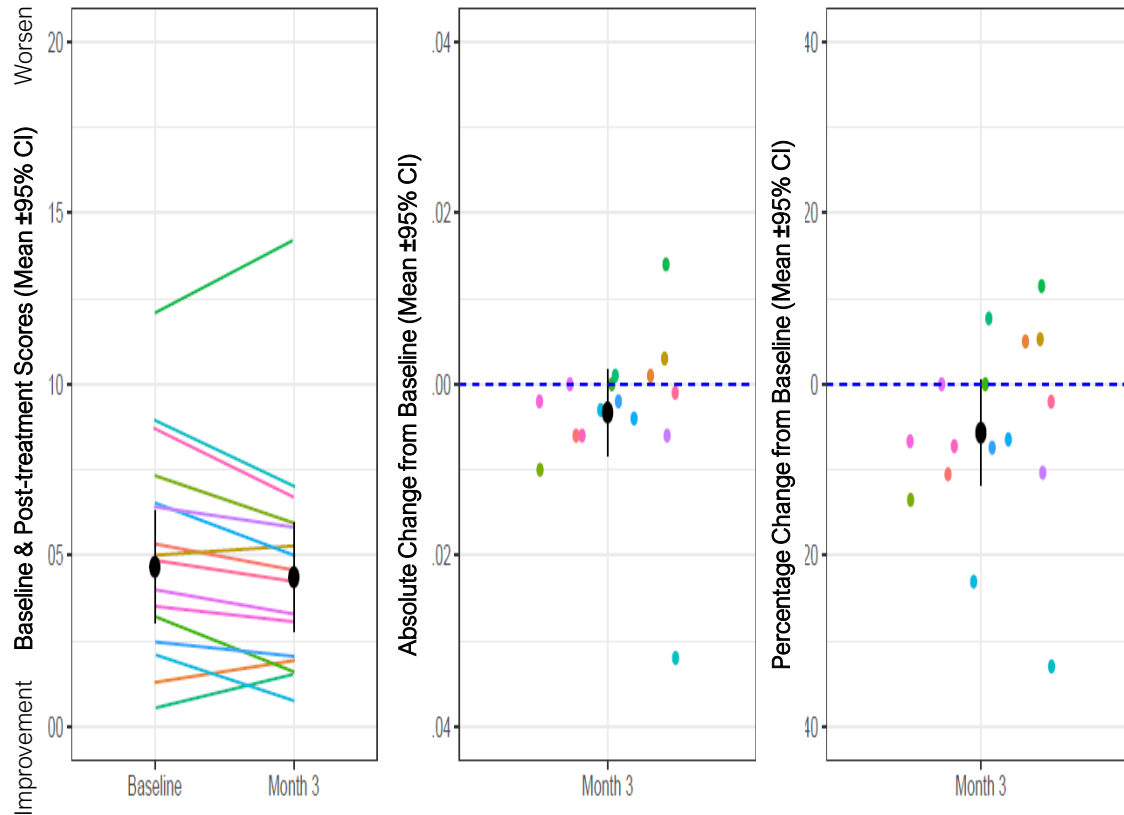


MMSE ≥ 20 patients



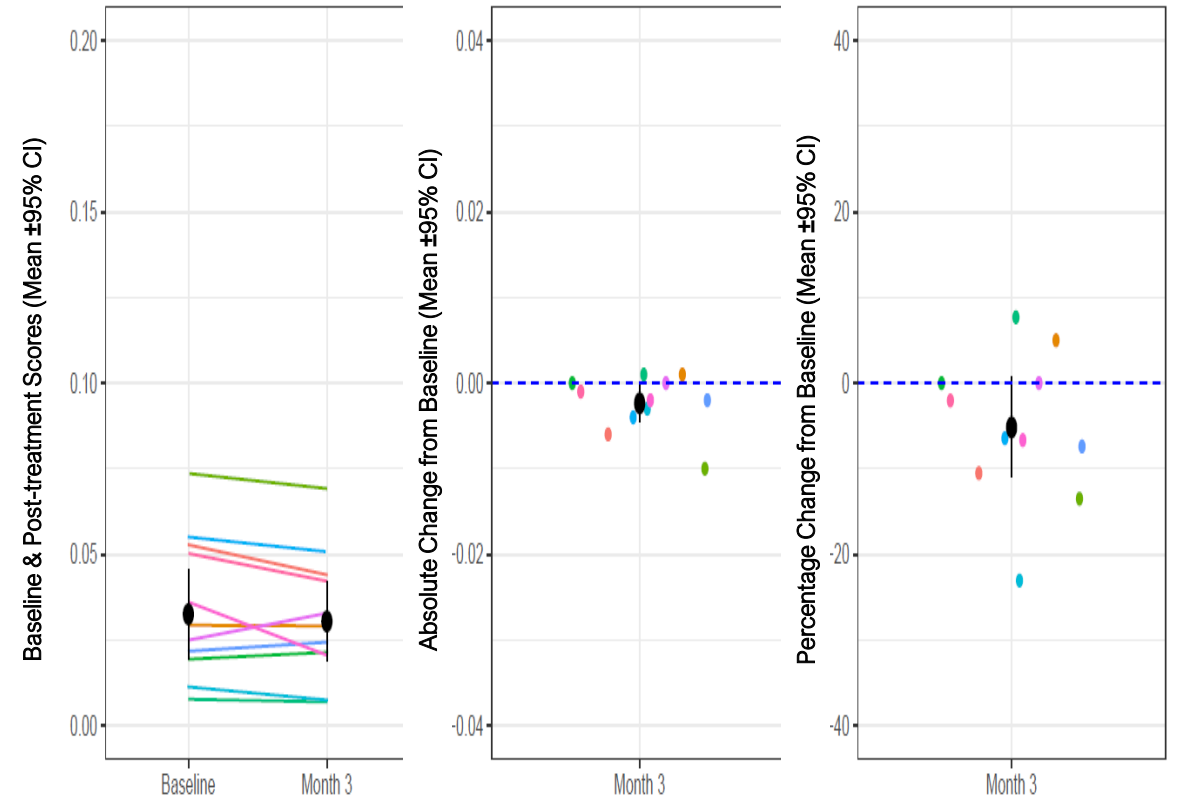
Significant improvements in the CSF p-tau : A β_{42} Ratio, a predictive measure of PET amyloid status¹...

All patients



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

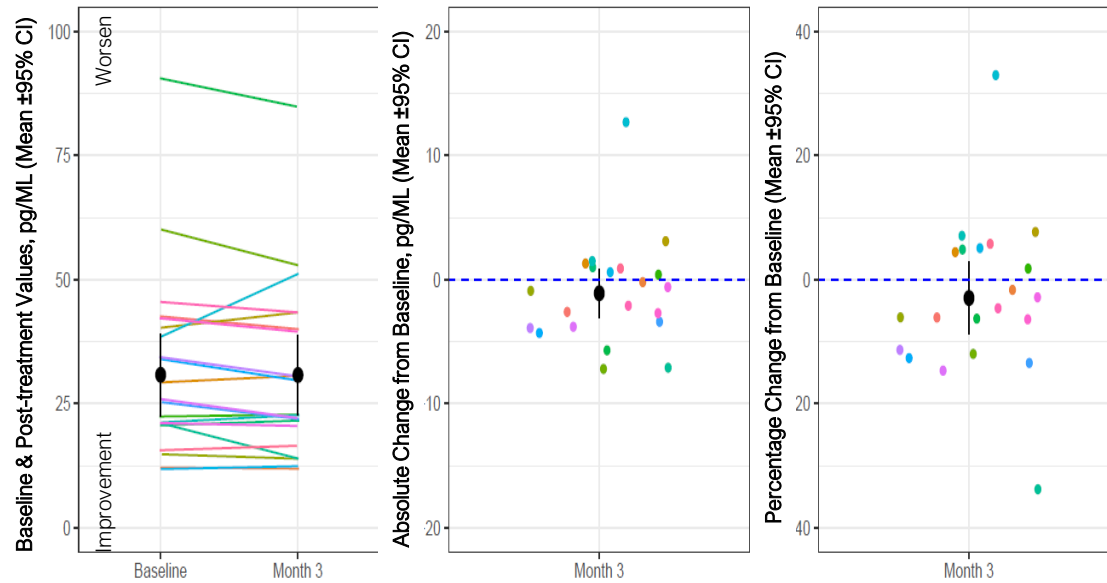
MMSE \geq 20 patients



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

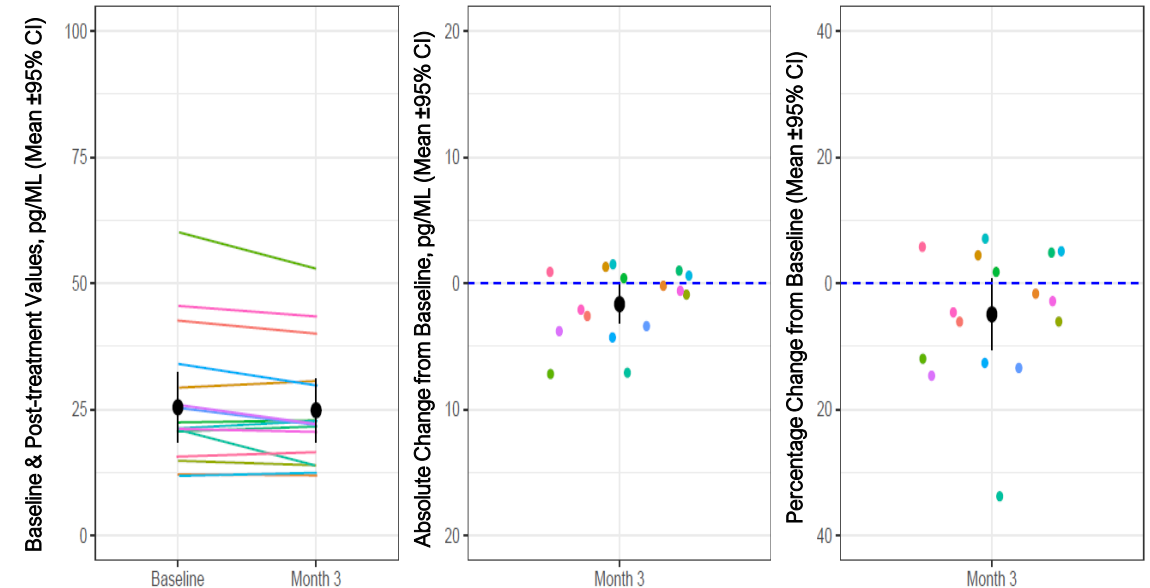
... Driven by a significant improvements in CSF p-tau

All patients



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

MMSE \geq 20 patients

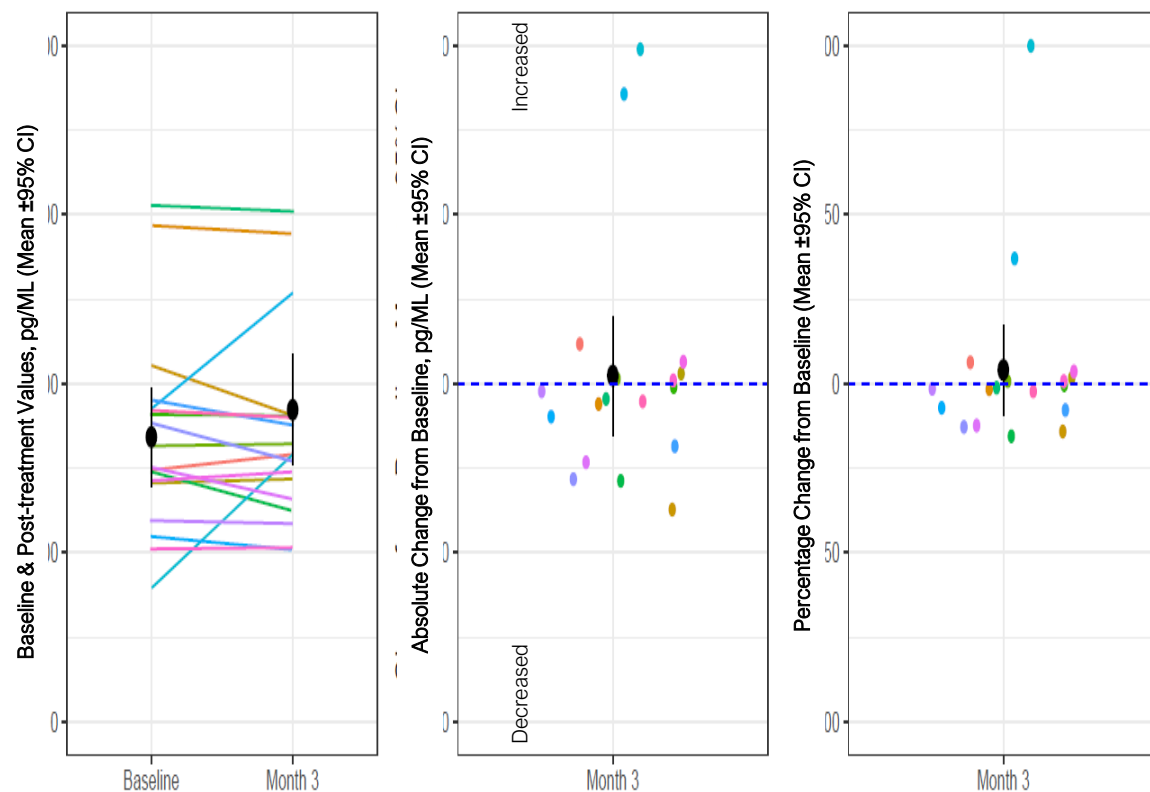


10/16 improved (63%)¹
Mean Absolute Change = -1.66 (p=0.0343)
Mean % Change = -4.93% (p=0.0852)

- 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

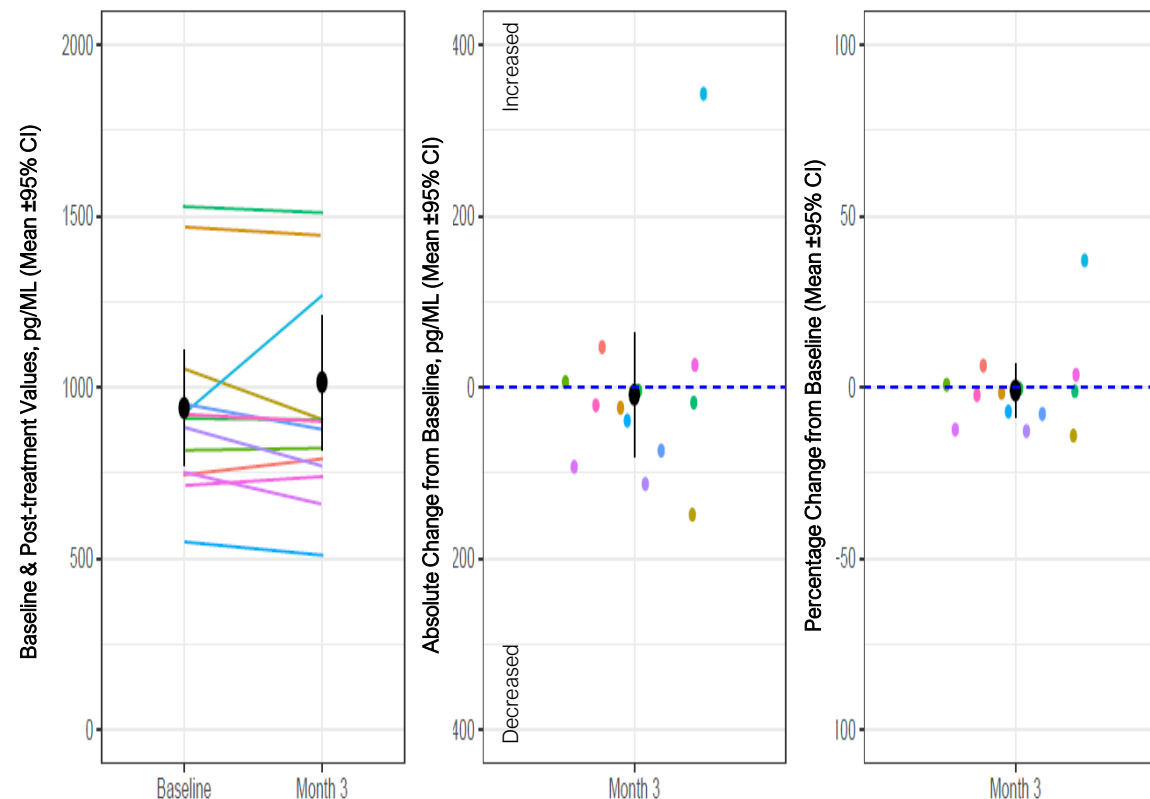
Modest improvements in CSF A β_{42}

All patients



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

MMSE \geq 20 patients

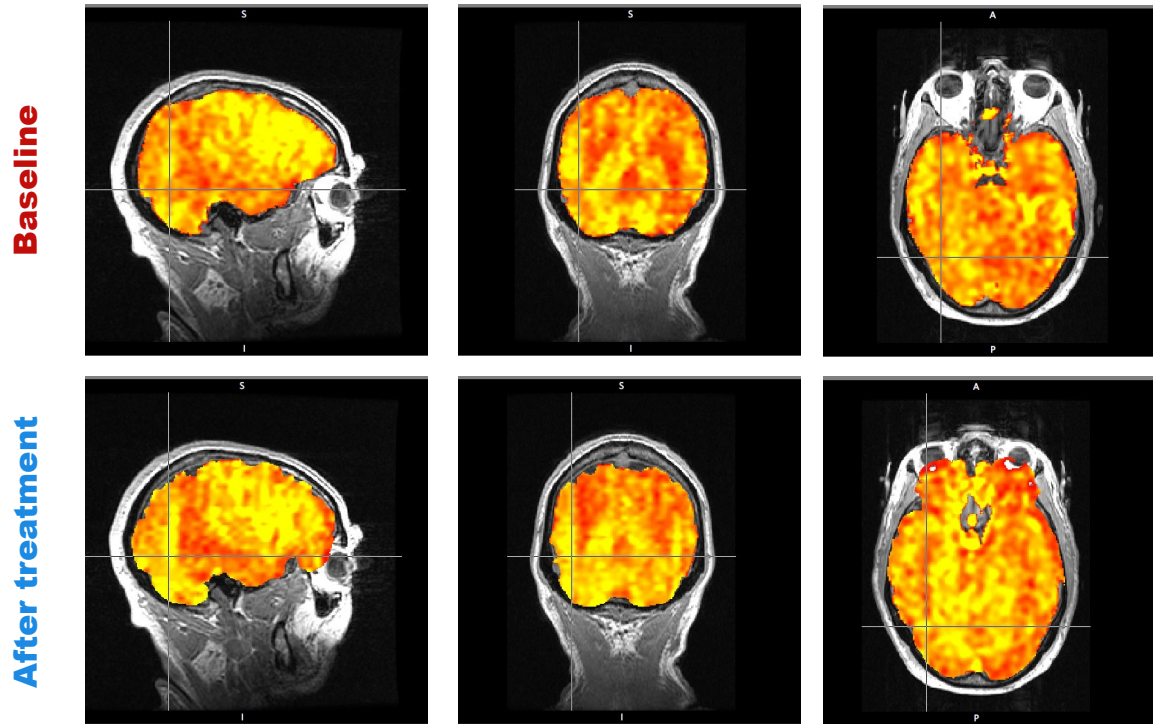


9/13 decreased (69%)
Mean Absolute Change = -8.69 (p=ns)
Mean % Change = -0.92% (p=ns)

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)



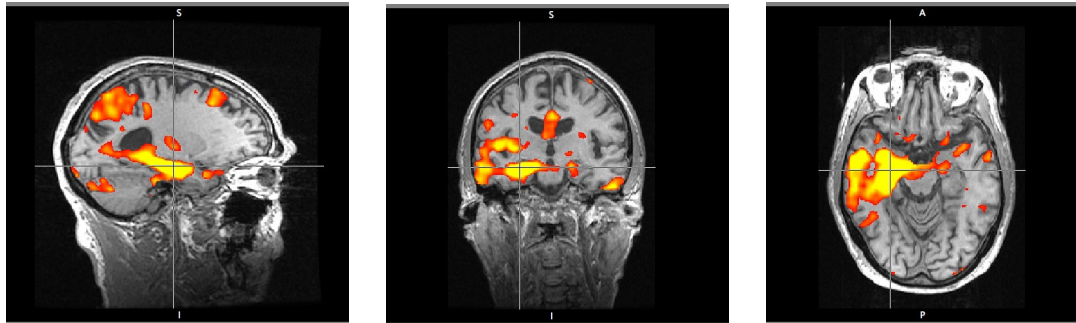
- 17% of 17 patients with MMSE ≥ 20 had “signal” improvements in the ASL in relevant areas (temporal parietal occipital) compared to baseline. Increased blood flow serves as a marker for brain activity
- 12% of 17 patients declined

Blood Oxygen Level-Dependent (BOLD) imaging shows NE3107 can reduce hyperactivation of the hippocampus¹ towards normal

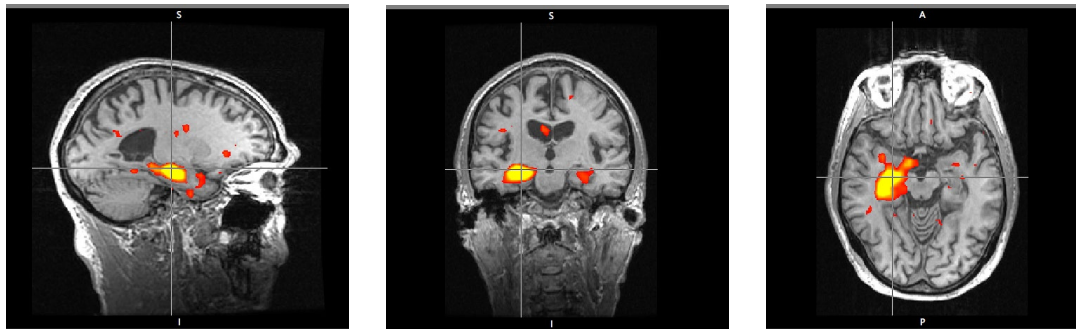
- BOLD imaging looks at the release of oxygen from blood to the tissues and characterizes network connectivity
- Image coloration indicates hyperactivation by stressed cells seeking help

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

Baseline



After treatment



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

The multifactorial nature of dementia pathology

- AD drug development at BioVie is based on the evidence that AD pathology is multifactorial in nature
 - While A β 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 TNF α (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 TNF α 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

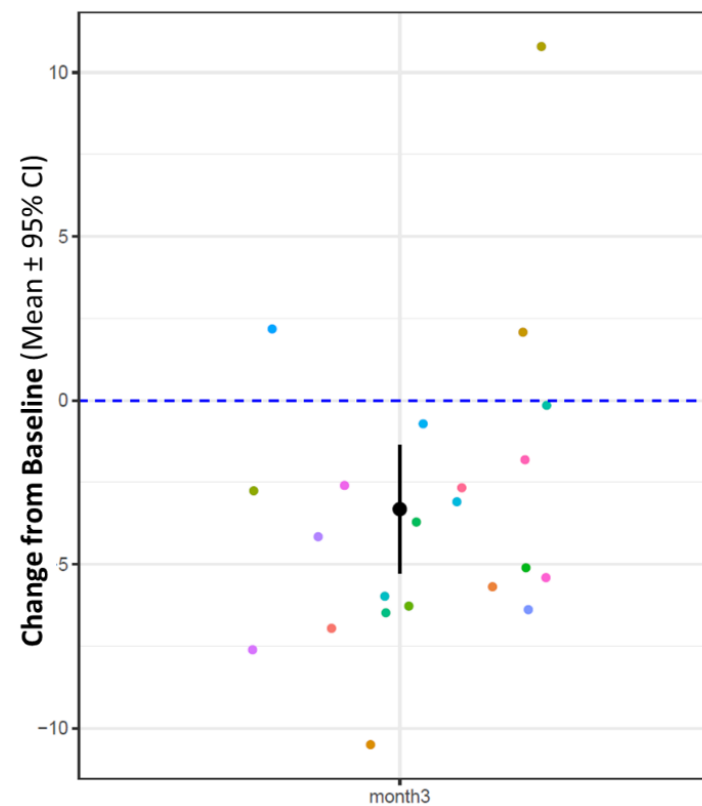
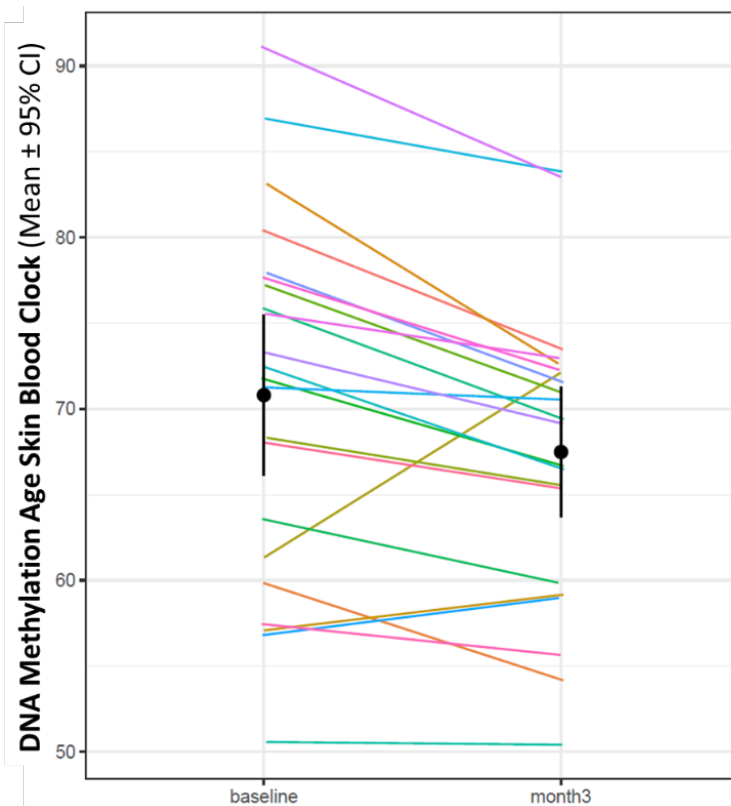
- Pivotal study for Alzheimer's disease. Two weeks each of 5 mg and 10 mg BID dose titration followed by 26 weeks of 20 mg twice daily vs. placebo, approximately 160 subjects in each arm, 80% power
- Diagnosed with AD and without evidence of a vascular contribution. Mild to moderate disease. CDR 1-2. 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

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)

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

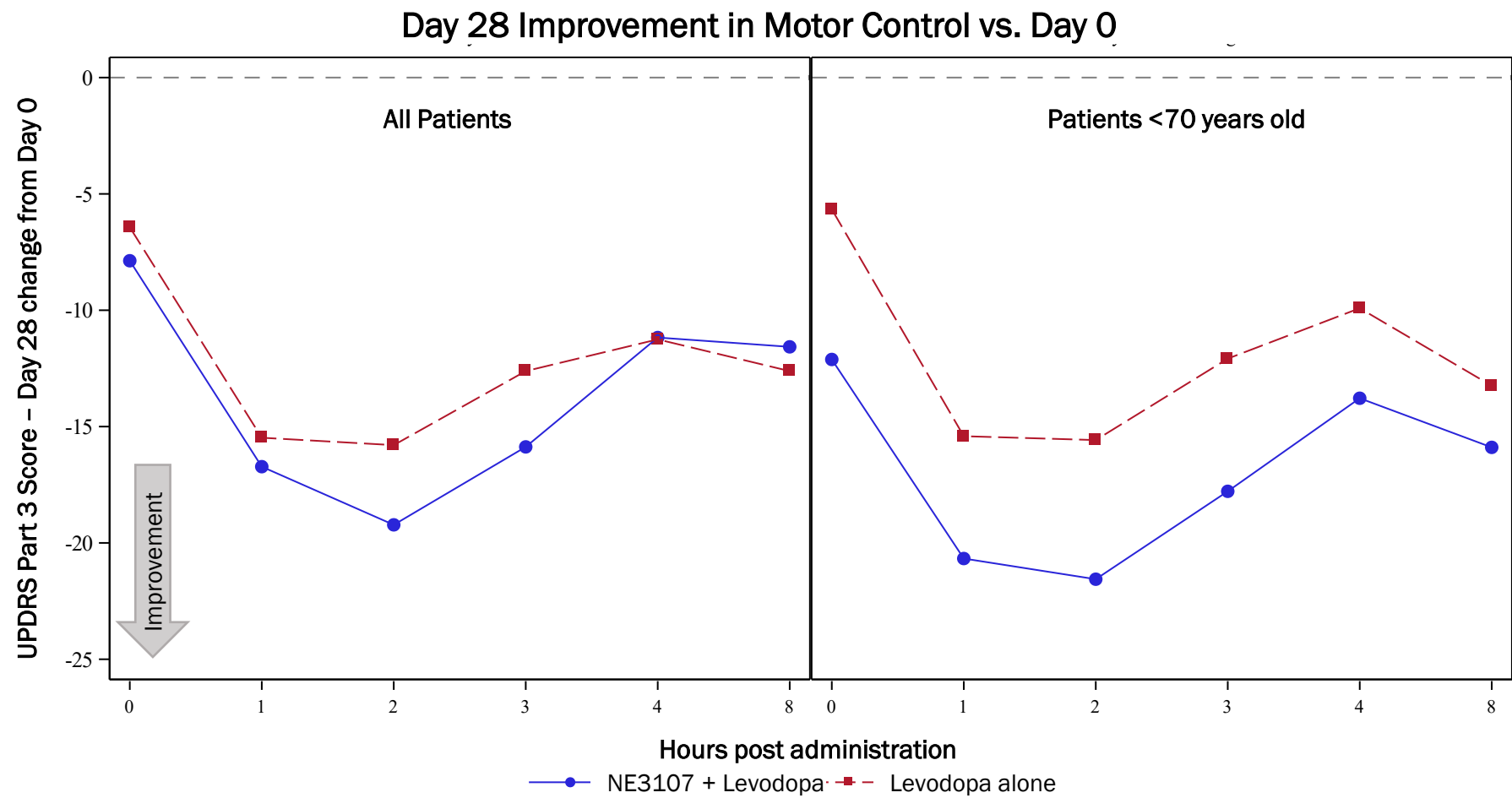
NM201 Phase 2

- 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

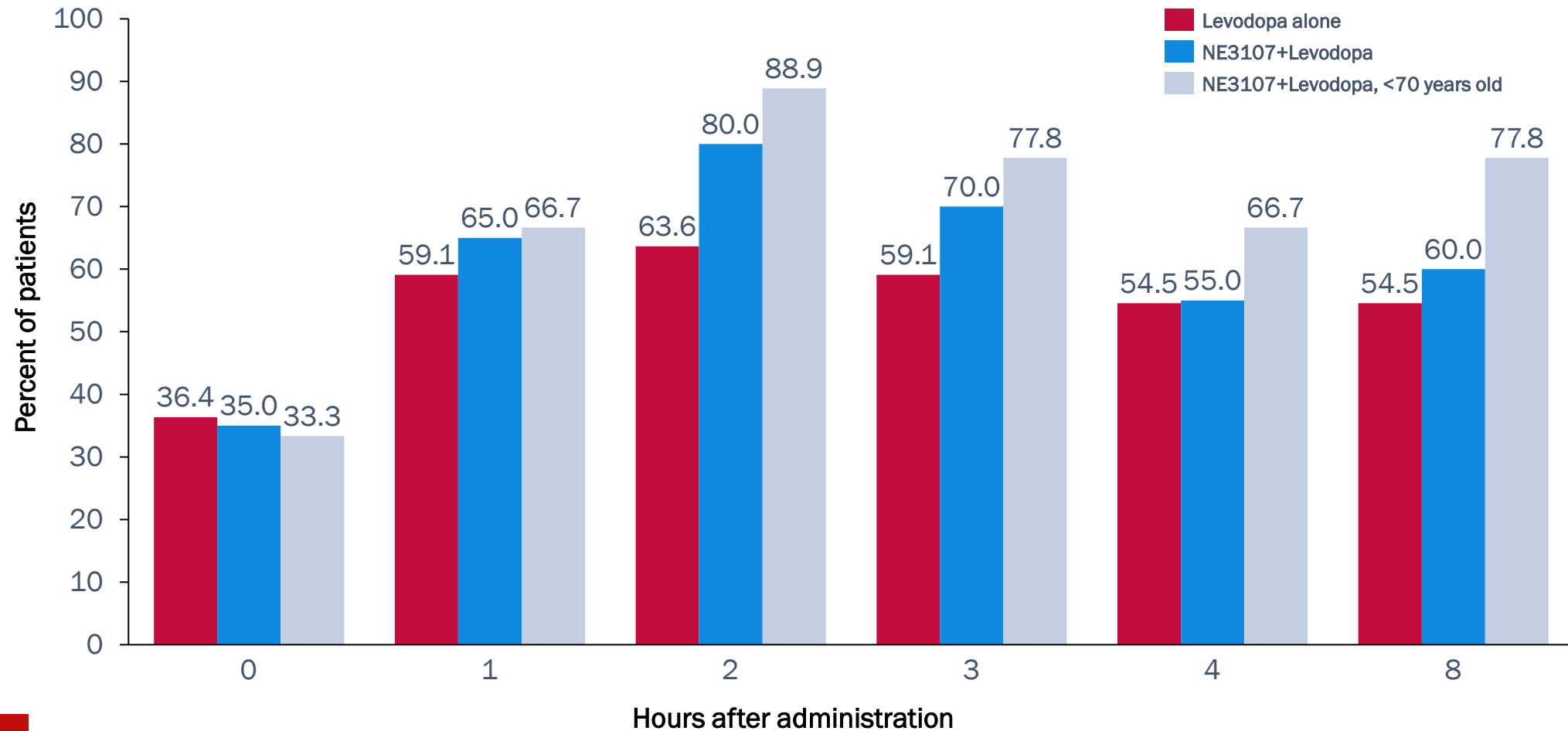
- **Safety assessments:** Standard measures of patient health, L-dopa PK and DDI
- **Efficacy assessments:** MDS-UPDRS* parts 1-3, Hauser ON/OFF Diary, Non-Motor Symptom Scale

NE3107's impact on motor control



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

Percentage of patients experiencing >30% improvement at Day 28 vs. Day 0



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

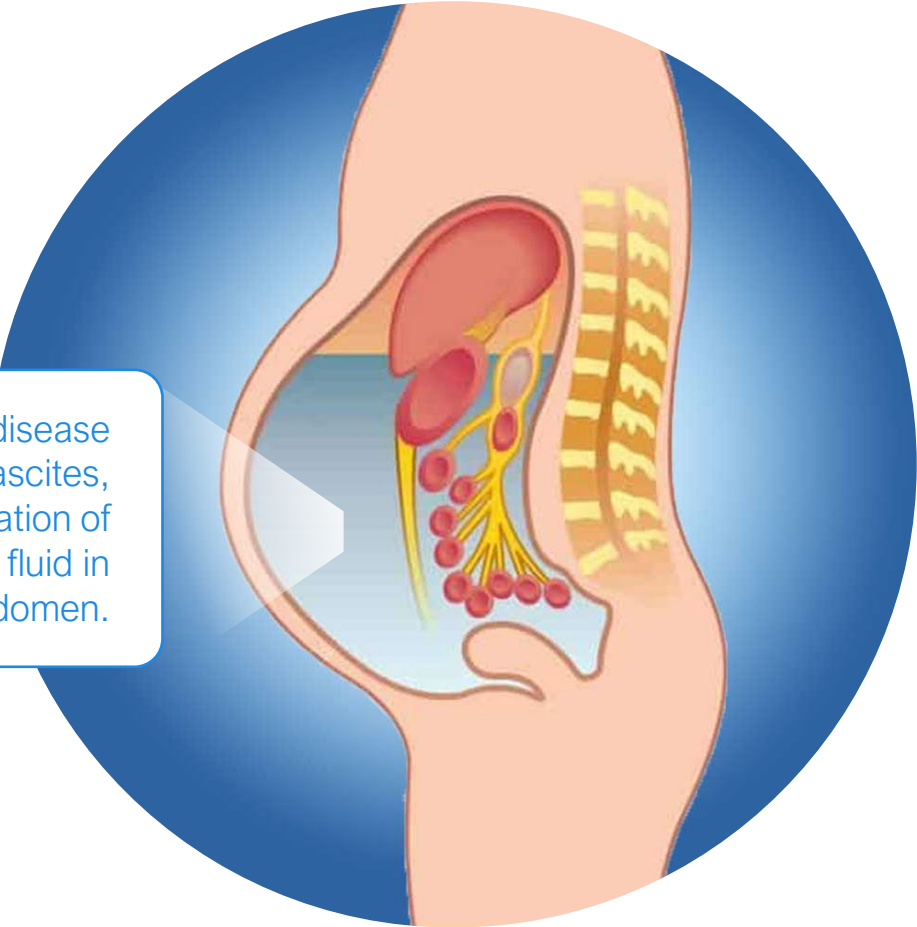
The kidneys are “burning out” by retaining massive quantities of salt and water

Patients suffer frequent life-threatening complications

No remaining options except for TIPS¹ surgery or liver transplant

Estimated \$670 million addressable US market with 20,000² 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 Development Plans

- 9 trial sites are activated and screening/enrolling patients
- 20 ascites patients to receive BIV201 therapy + standard of care (SOC); 10 to receive SOC only (control group)
 - Co-primary endpoints: Incidence of ascites-related complications over 180 days following randomization and change in ascites fluid removed over 90 days
- Timeframe: Two 28-day cycles of BIV201 therapy within 4 months, then follow patients for additional 60 days
- Similar trial design planned for a single pivotal Phase 3 planned in 2023 with ~120 subjects

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, Zynerva
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
 - 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 TNF α in manner that's correlated with cognitive improvements
 - Reduction phospho-tau production and the ratio of phosphor-tau to amyloid beta (A β)
 - 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)
- In liver disease, BIV201 is in Phase 2b for refractory ascites. In trials thus far, patients have experienced:
 - Reductions in ascites fluid build up and extension of time between paracenteses
 - No drug-related SAEs

Thank You

Portfolio Revenue Potential

	U.S. Intellectual Property	Company Estimates ²		
		US Patient Population	US Peak Sales Potential	Global Sales Potential
NE3107: Alzheimer's	Compound patent through 2031 ¹	6.1 million	\$7 billion	>\$10 billion
NE3107: Parkinson's	Compound patent through 2031 ¹	1.0 million	\$1.5 billion	>\$3 billion
BIV201: Refractory Ascites	Orphan Drug; Patent-pending formulation; Patent issued method of use	20,000	\$450 million	>\$900 million

¹ Anticipated to be extendable to 2034

² Based on internal company estimates and compilation of analyst data

Prior NE3107 Clinical Studies

Phase 1¹

Obese, impaired
glucose tolerant
healthy volunteers

NE3107:

- Improved insulin-stimulated glucose disposal, assessed by hyperinsulinemic, euglycemic glucose clamp procedures
- Decreased C-reactive protein (CRP²) and increased HDL and adiponectin (both associated with benefit in AD)
- Showed no differences in AEs compared to placebo

Phase 2³

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 placebo⁴
- Showed no differences in AEs compared to placebo

1. Reading *Mediators Inflamm* 2013 814989

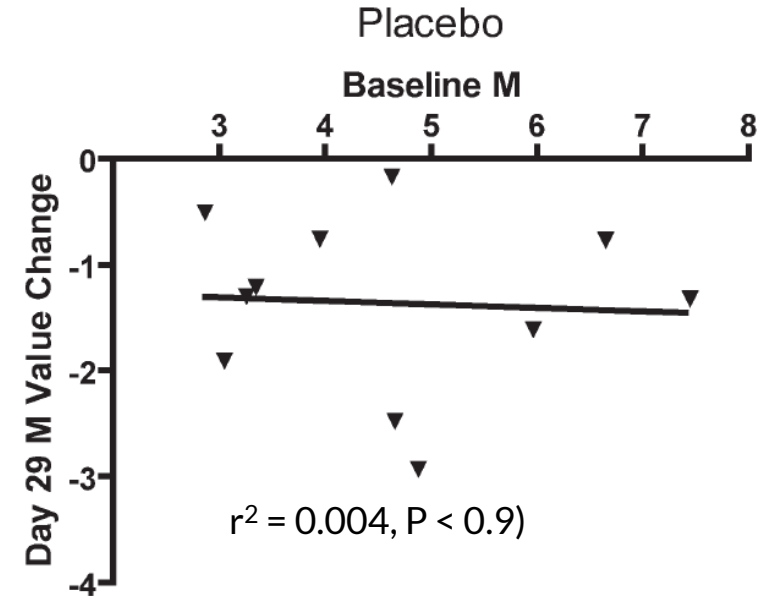
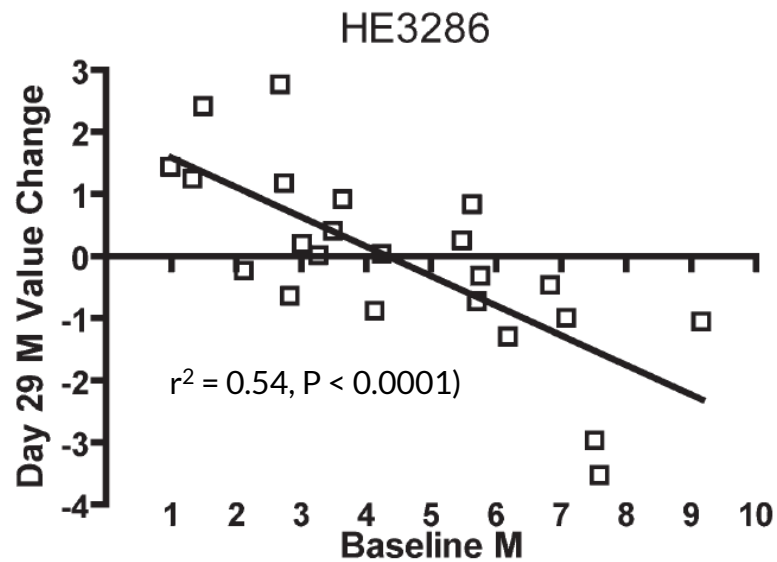
2. CRP is a nonspecific marker of inflammation that is increased in the brain and serum of patients with Alzheimer's disease (AD), and has been associated with increased risk of developing dementia

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

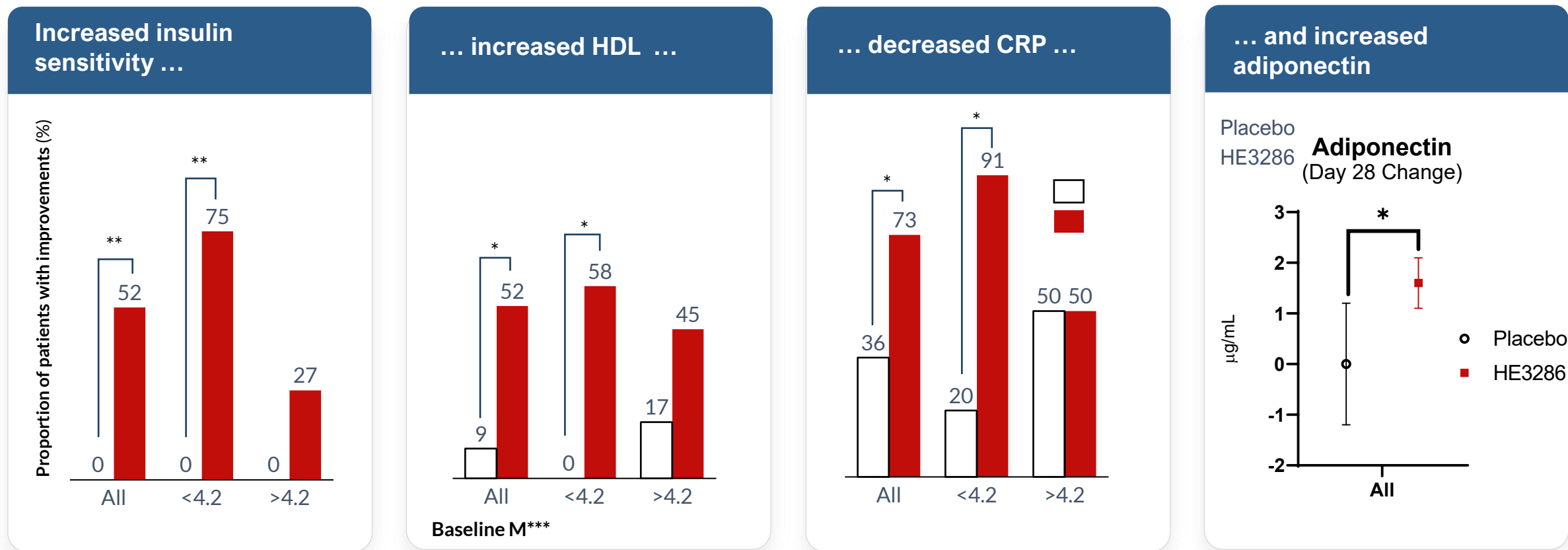
Improved insulin-dependent glucose disposal in 48 obese, inflamed subjects



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

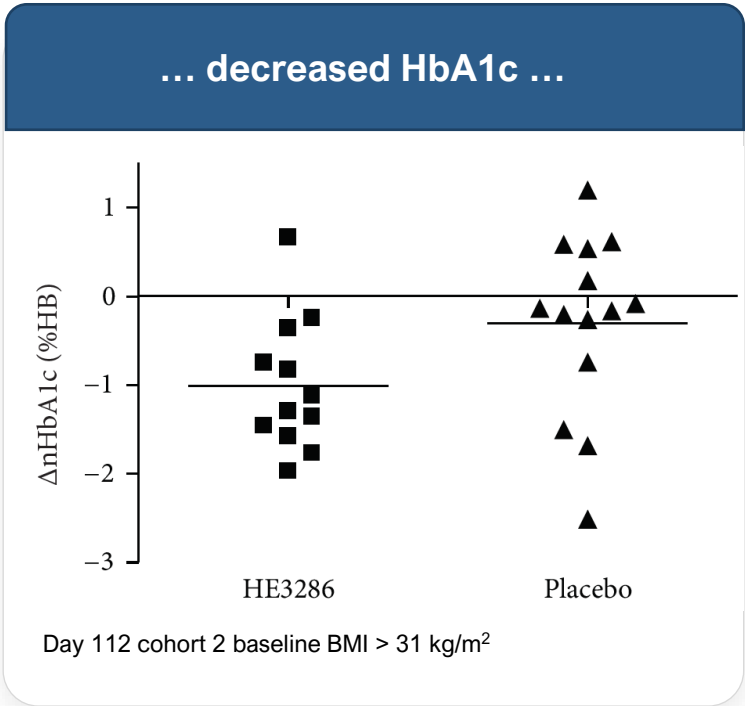
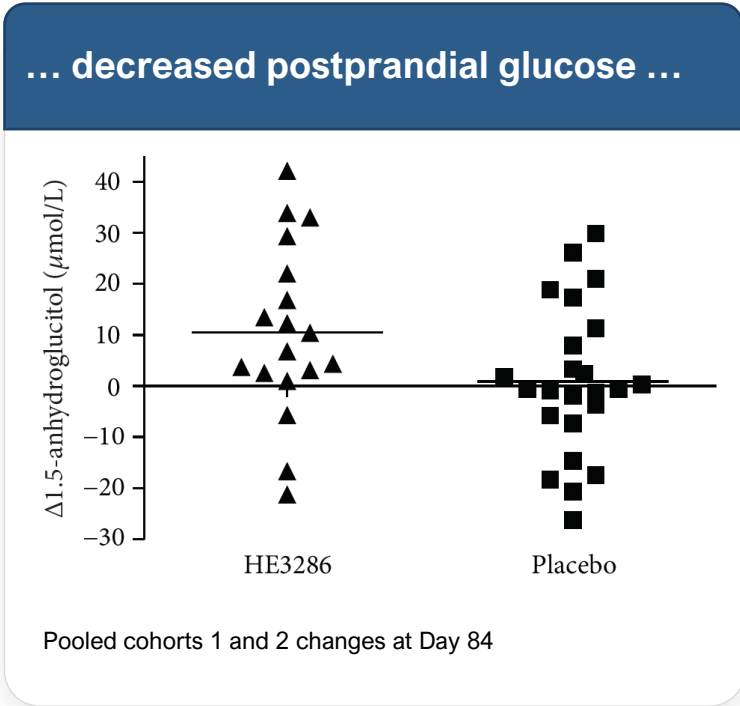
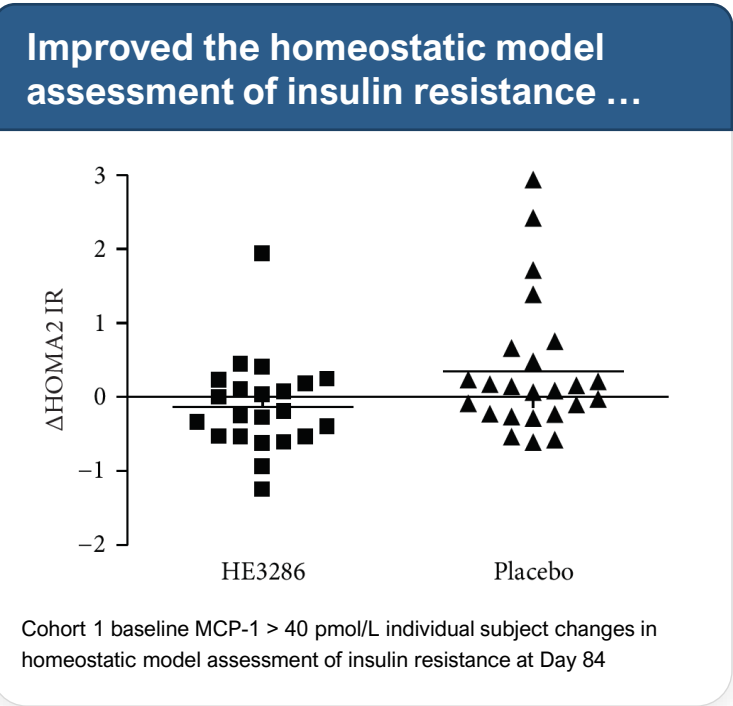
Insulin sensitizing improvement also brought improvements in AD indicators

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



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



... 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

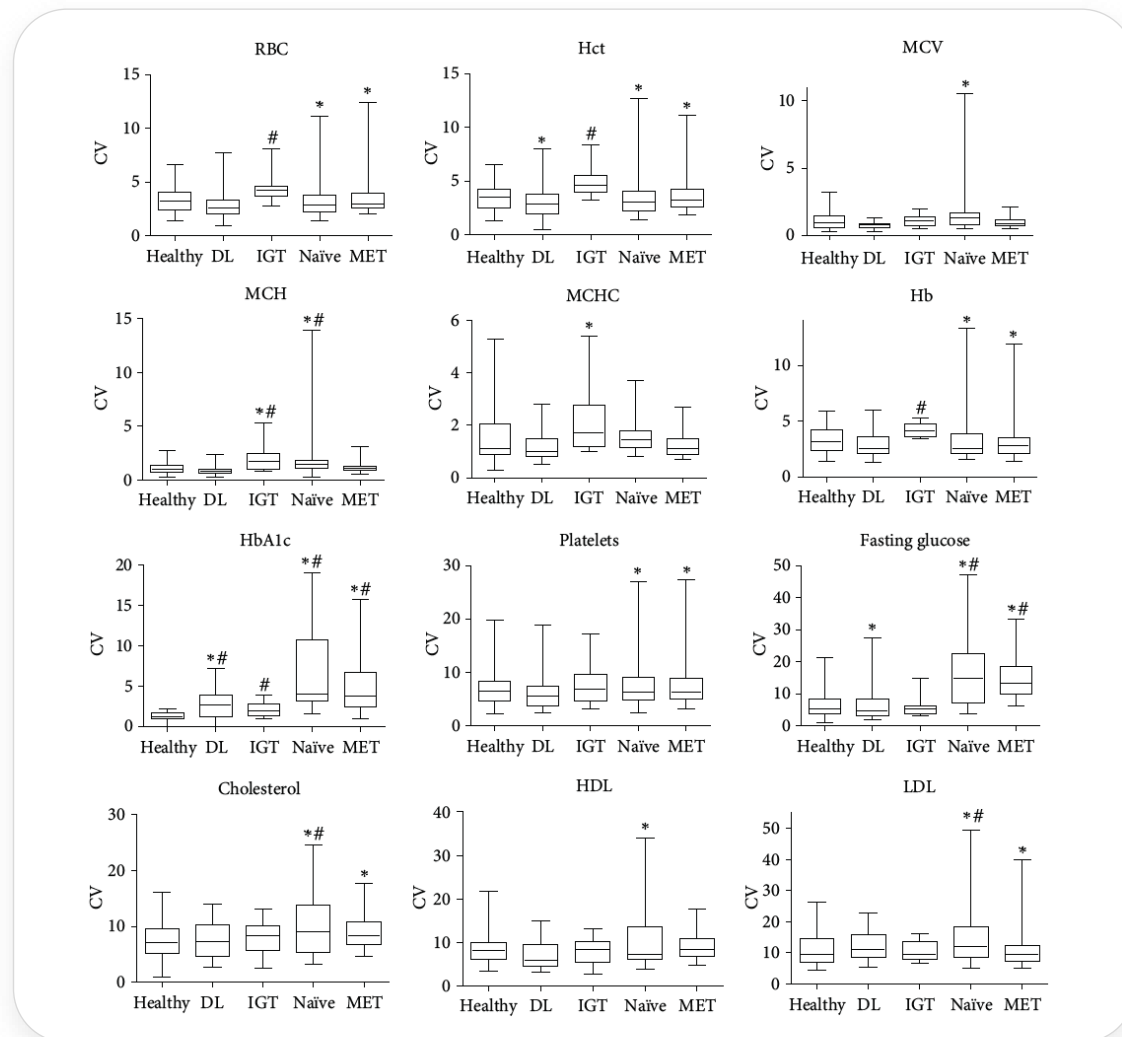
Inflammation drives systems dysregulation

- 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

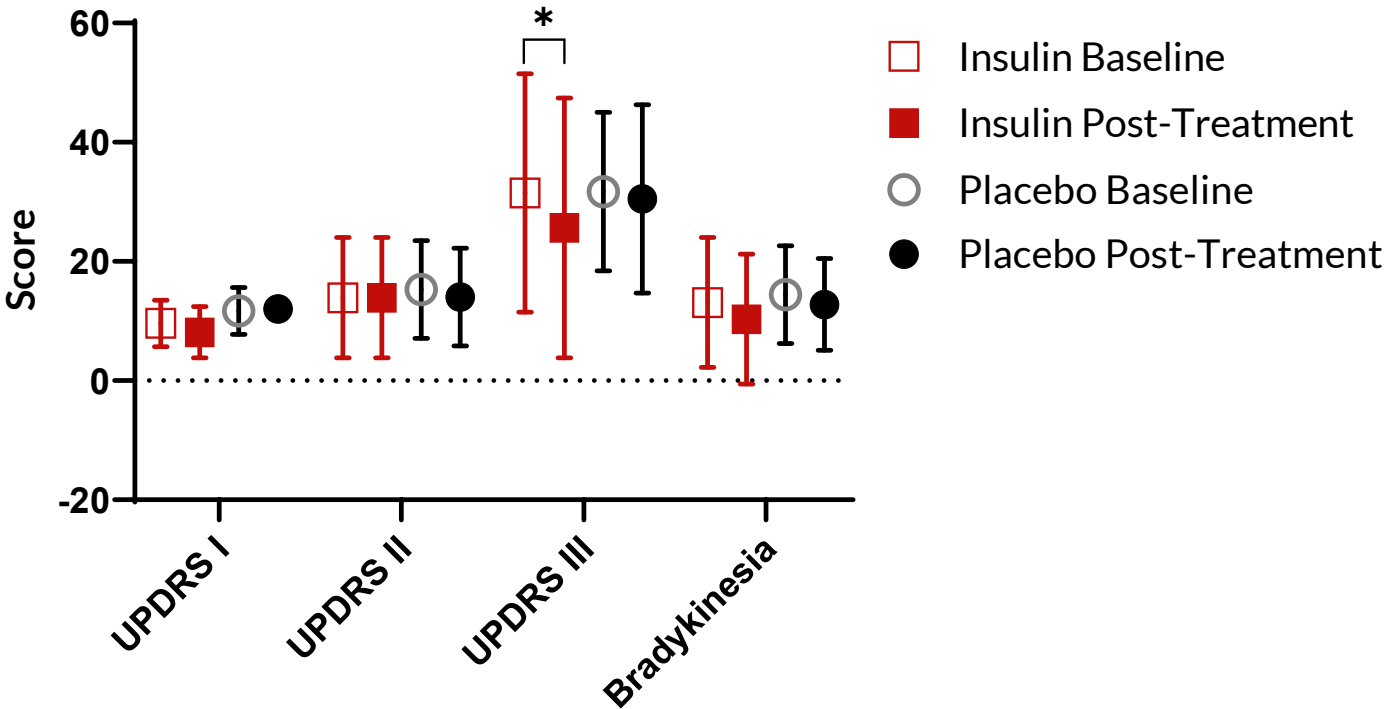
Group	Cohort 1	MCP-1 > 40	Cohort 2	BMI > 31
HE3286	n=44	n=22		n=35
Placebo	n=51	n=25		n=34

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

The Role of Reduced Insulin Signaling in Parkinson's Disease

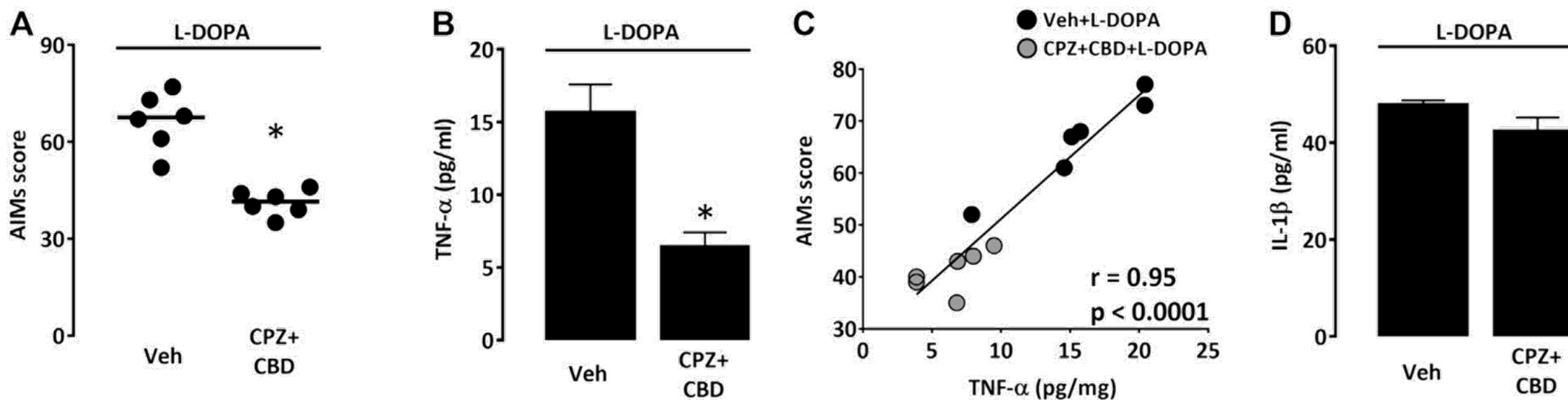
Stimulation of insulin signaling improves motor function in Parkinson's patients

Intranasal Insulin Treatment Reduced Inflammation and Improved Motor Activity



Inflammation's Role in LID

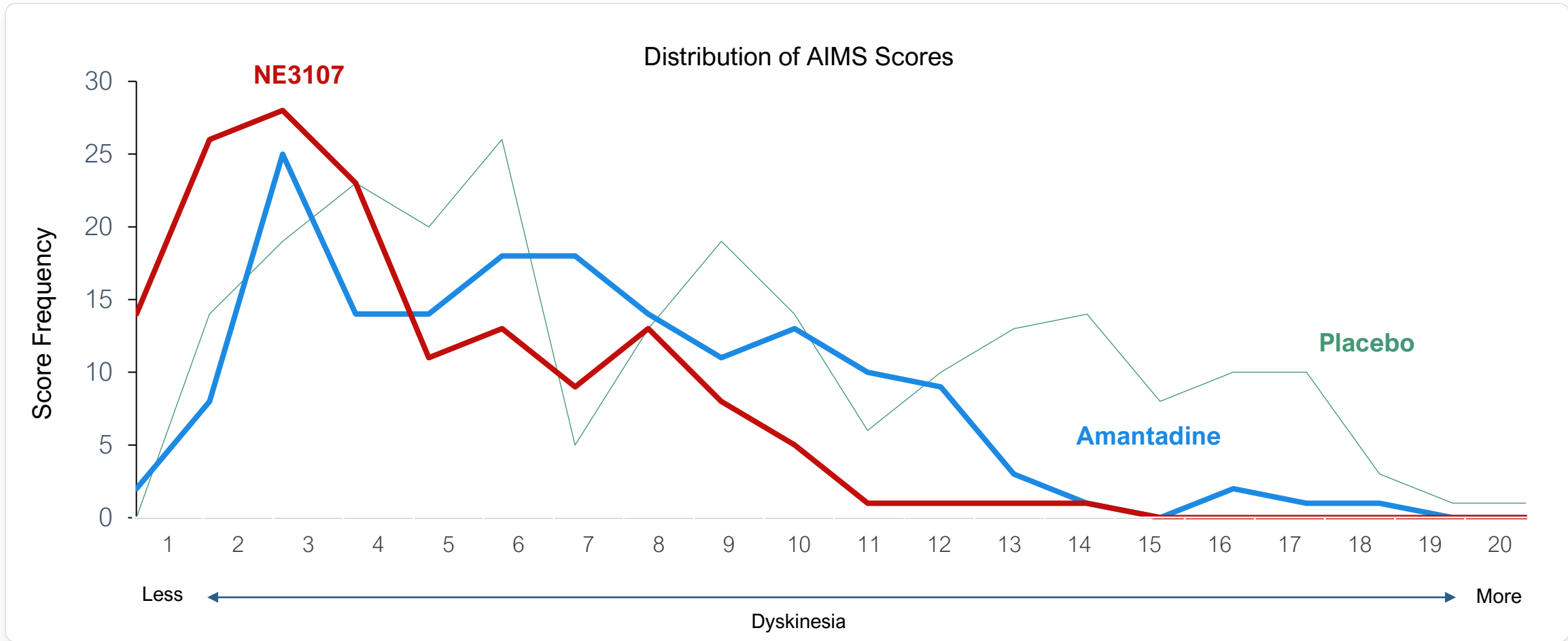
Reduction of TNF-induced inflammation reduces LID



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

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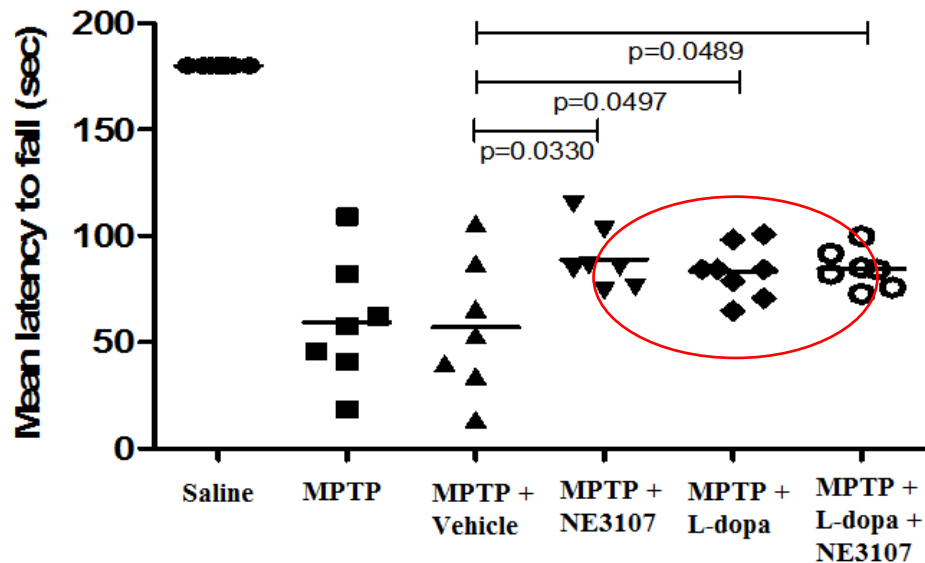
NE3107 decreases L-Dopa-induced dyskinesia (LID) in marmosets



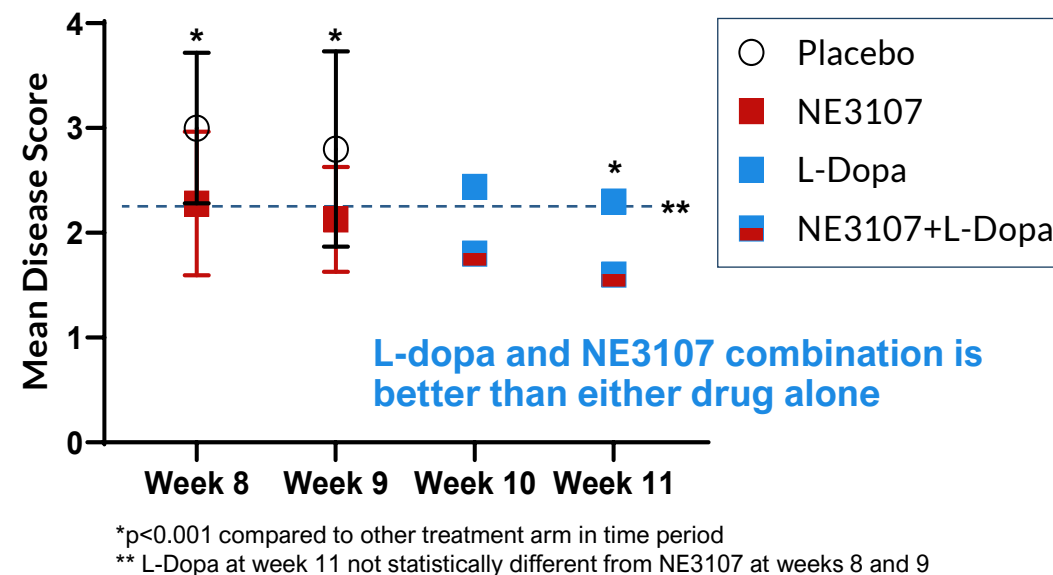
Abnormal Involuntary Movement Scale (AIMS)

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

MPTP Mouse

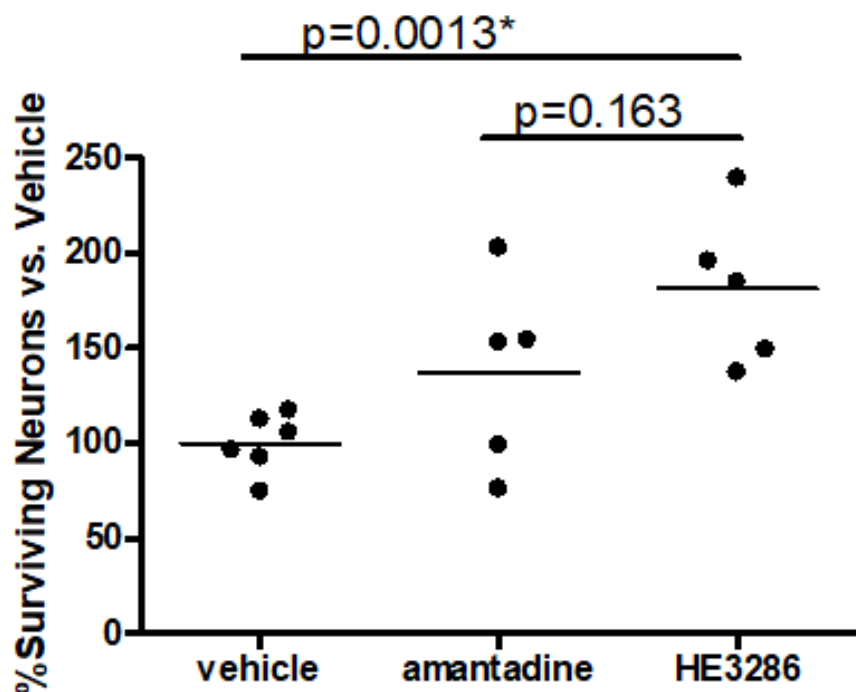


MPTP Marmoset treated at Week 8

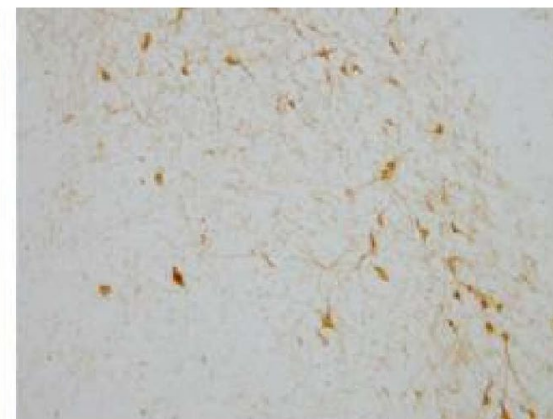
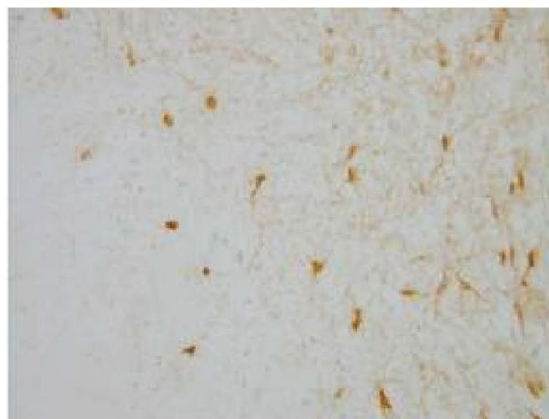


NE3107's promotoric effects observed within 4 days of treatment

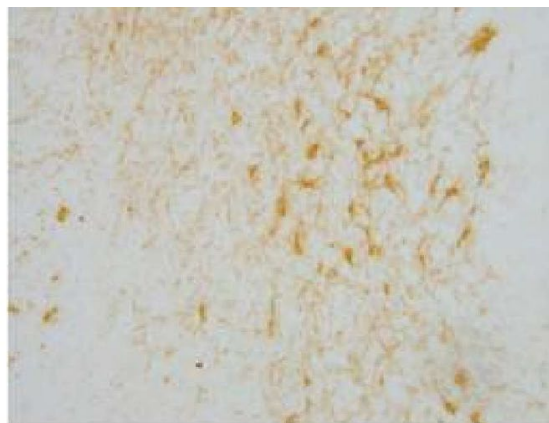
NE3107 preserved TH+ neurons in MPTP marmosets



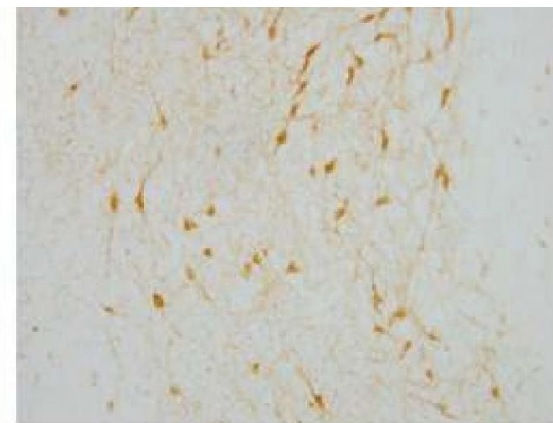
Control moneys (M09100 and M11008)



HE3286 (M11007)



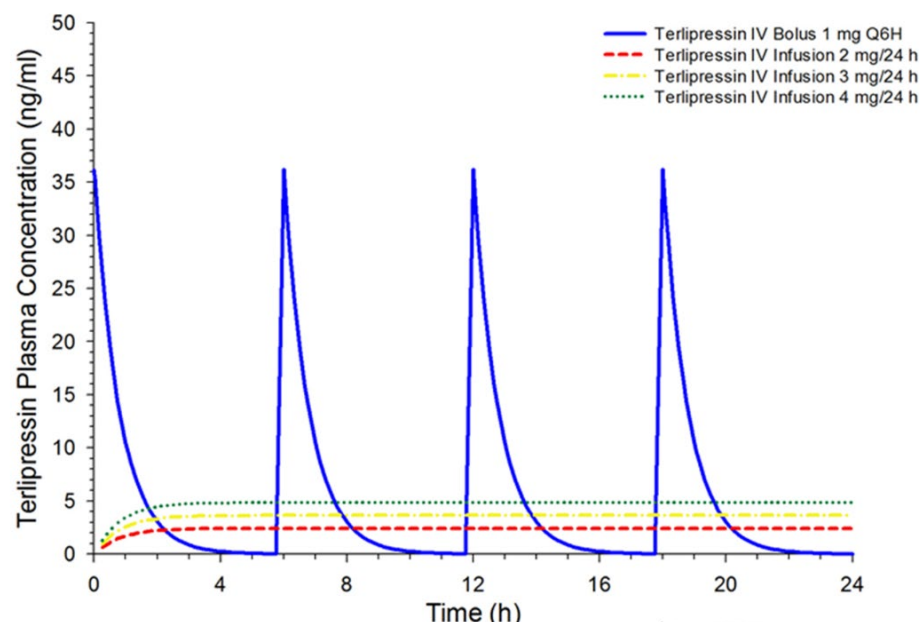
Amantadine (M10084)



BioVie Phase 2a trial results: BIV201 Pharmacokinetics

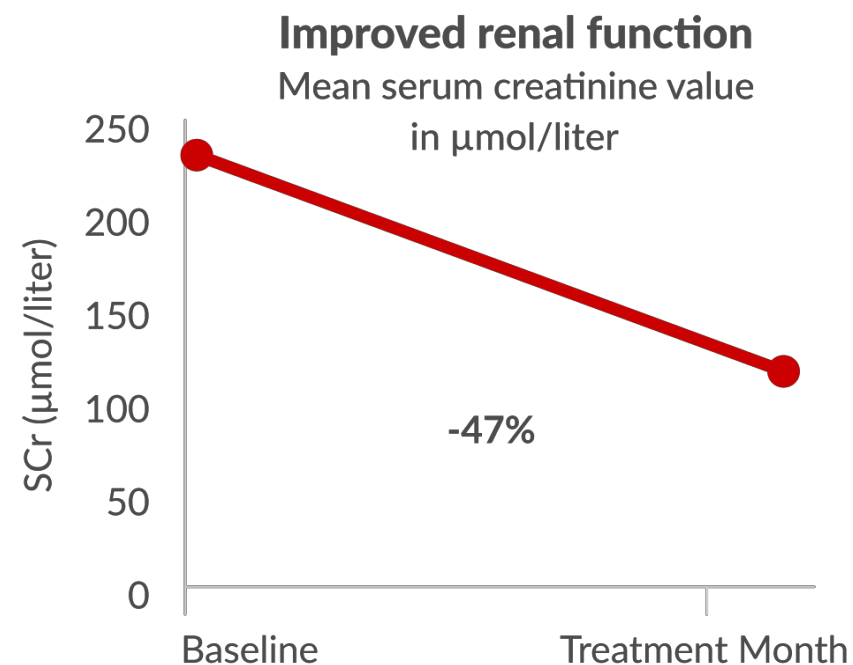
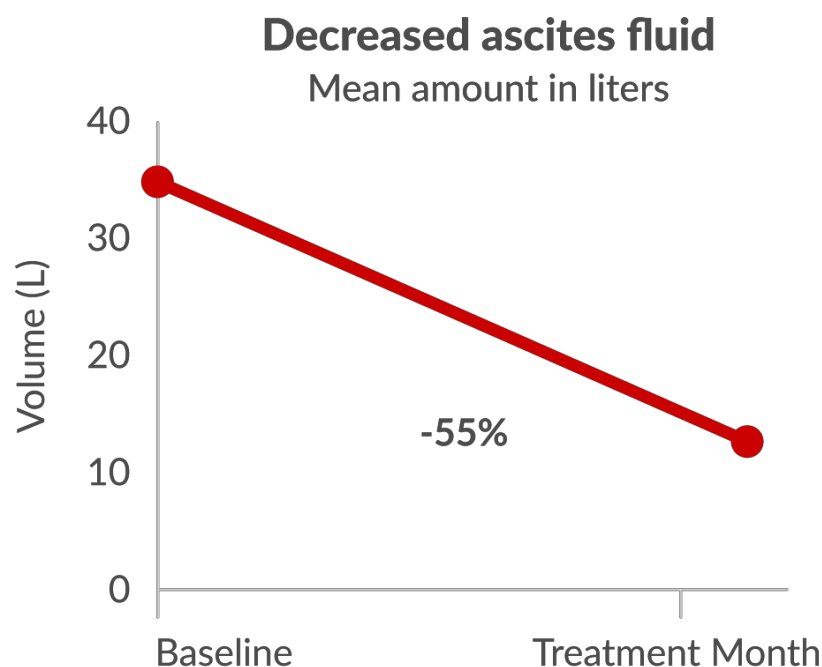
Continuous low blood levels of terlipressin versus spikes with IV-bolus dosing

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

Continuous Infusion Terlipressin in 6 Refractory Ascites/HRS Patients*



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

