Developing Transformative Therapies to Overcome Chronic Debilitating Diseases

Company Update • September 2022

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.

Highlights

- Topline results from the Phase 2 exploratory biomarker trial suggest directional and exciting signals of NE3107's potential in Alzheimer's Disease (AD)
  - The majority of patients (especially those with MMSE >=20) saw improvements in ADAS-Cog12 and the Global Rating of Change (an instrument that tracks improvements in a patient's conditions, abilities and overall sense of well-being)
  - 60%+ of patients saw reductions in TNFα levels (i.e., inflammation), which is significantly correlated with ADAS-Cog improvements
  - 60%+ of patients experienced improvements in the ratio of p-tau : $A\beta_{42}$ in CSF[1]
  - Early readings of imaging data suggest certain changes in the brain consistent with increased blood flow and reduced oxidative stress that are consistent with the mechanism for NE3107 and are unlikely to be accounted for by placebo effect
- Other trials are progressing and should readout over the next 12 months
  - The Phase 2 trial for NE3107 in Parkinson should be fully enrolled in a couple of months and readout by the end of 2022. This trial is primarily a drug-drug interaction study that we also designed to determine if the promotoric activity seen preclinically can be detected in humans
  - The Phase 3 trial for NE3107 in Alzheimer's has enrolled one-half of its targeted 316 patients and should readout in mid-2023 unless a sample size increase (up to 400 patients) is recommended by the DSMB later this year
  - The Phase 2b trial for BIV201 in refractory ascites has been slower to enroll than expected and was especially impacted by Covid-19. Patient screening has picked up in recent weeks and should readout mid-2023 if screening keeps up the current pace

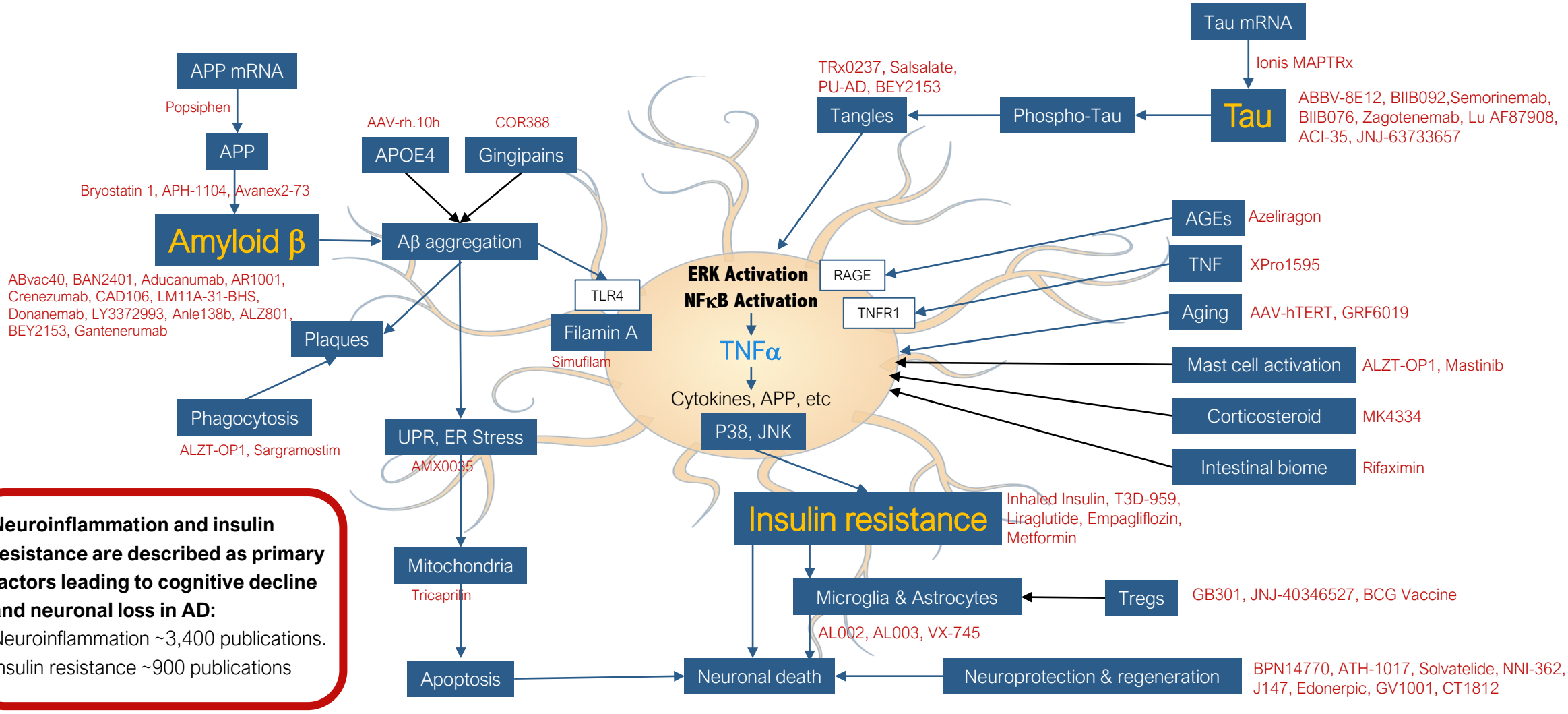
1. The ratio of p-tau : Ab42 in CSF has been to be predictive of PET amyloid status. Campbell et al. *Alzheimers Dement (Amst).* 2021; 13(1): e12190

Expected Catalysts & anticipated timelines (US)

* 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.

Background

Alzheimer's Disease cascade



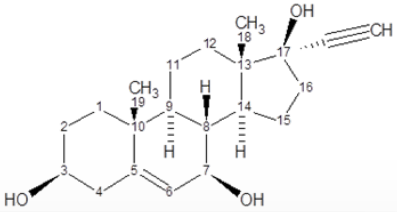
Neuroinflammation and insulin resistance are described as primary factors leading to cognitive decline and neuronal loss in AD:
Neuroinflammation ~3,400 publications.
Insulin resistance ~900 publications

NE3107 provides a novel mechanism of action in AD research

- AD research has largely focused on Amyloid Beta (Aβ) and phospho-tau (p-tau) for decades and has resulted in a large number of trials in the AD drug development pipeline in 2021 focused on these mechanisms.[1]
- More recently, however, research focus has shifted towards inhibiting neuroinflammation
  - 23 disease-modifying agents are listed in clinicaltrials.gov in 2021 as investigating inflammation or the immune system[1]
  - NE3107 is the only molecule with a Phase 3 trial underway (NCT04669028) in mild- to moderate-AD patients whereas 17 other agents are in Phase 2
  - NE3107 is the only molecule in this group that is pursuing a two-pronged approach targeting both neuroinflammation and insulin resistance. Insulin resistance is induced by neuroinflammation and can further exacerbate neuroinflammation in return
- Tumor Necrosis Factor Alpha (TNFα) is a cytokine identified as a major regulator of inflammation whereby its excessive activation is associated with chronic inflammation.[2]
  - It is considered to be the central mediator of inflammation due to its role at the top of the biochemical pathways that lead to the production of other inflammatory factors downstream
  - Patients with rheumatoid arthritis and psoriasis treated with anti-TNF agents have been shown to have lower risk of AD[3]
- NE3107's unique mechanism can modulate TNFα expression in both the CNS and periphery
  - Preclinical studies showed NE3107 is a modulator of TNFα production through its ability to modulate the activation of the Extracellular Regulated Kinase (ERK) and Nuclear Factor kappa B (NFκB).[4]
  - By down regulating the activation of ERK and NFκB, NE3107 has been shown to reduce the production of TNFα.[5]
  - Newly generated data supporting a new patent application show that NE3107 inhibits ERK activation and blocks the phosphorylation of TNF receptor 1 (TNFR1) in an Ikk-MAP3K8-MEK dependent pathway to decrease forward-feeding TNF inflammatory cascades , thereby lowering the expression of other downstream inflammatory factors.

1. Cummings et al. 2022 DOI: 10.1002/trc2.12295
2. Jang et al. Int J Mol Sci. 2021 Mar; 22(5): 2719.
3. Zhou et al. 2020 PLoS ONE 15(3): e0229819.
4. Lu 2010 *Am J Physiol Endocrinol Metab* 298 E1036; Wang 2010 *J Pharmacol Exp Ther* 333 70
5. Offner 2009 *J Pharmacol Exp Ther* 329 1100

Background on NE3107



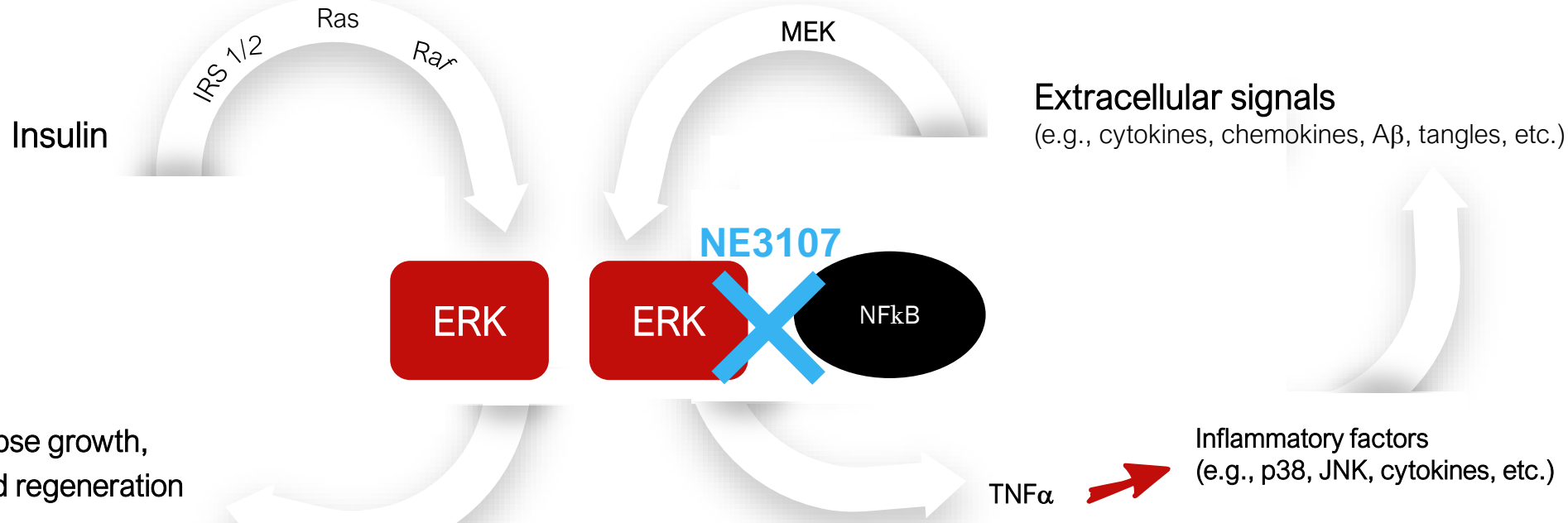
First-in-class molecule with desirable characteristics

- 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)

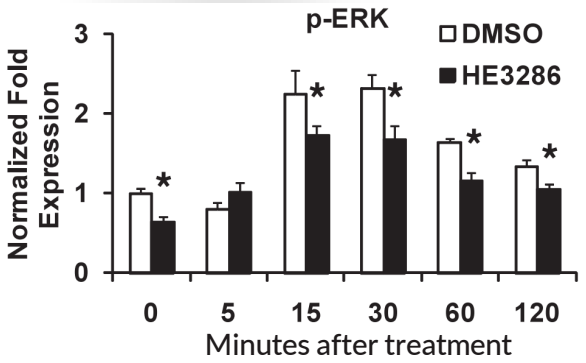
- Originated by Hollis-Eden Pharmaceuticals (renamed Harbor Therapeutics)
  - NE3107[1] is a synthetic analogue of a metabolite of the adrenal hormone DHEA
- Phase 1 and 2 trials in diabetic patients showed that NE3107:
  - Showed no differences in AEs compared to placebo
  - Decreased insulin resistance,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

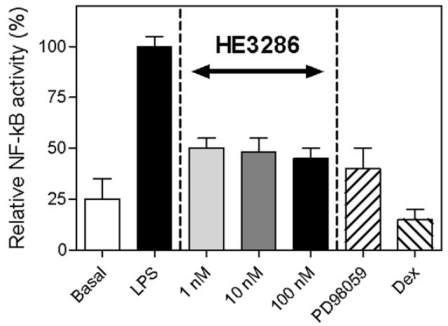


NE3107 Reduces ERK Activation



Lu 2010 Am J Physiol Endocrinol Metab 298 E1036

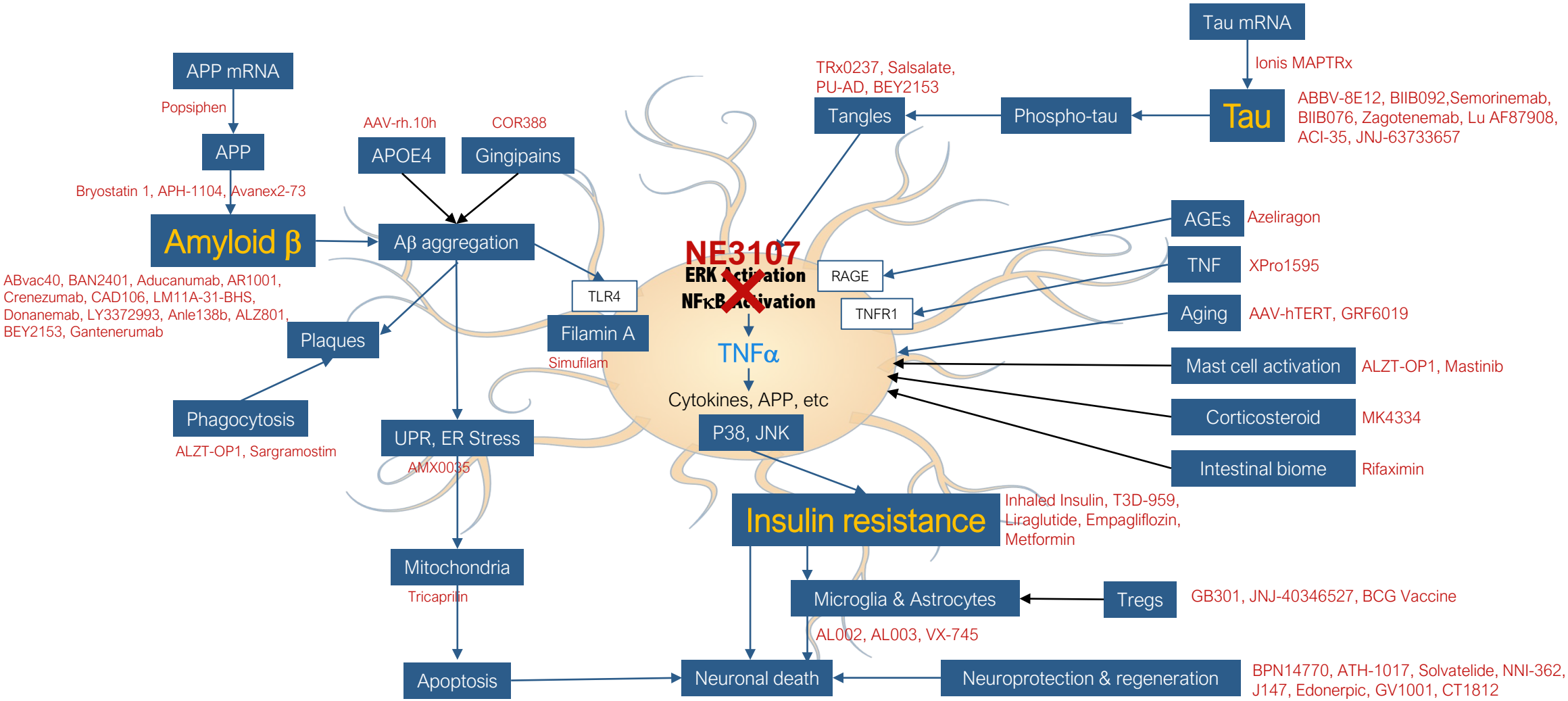
NE3107 Reduces NFkB Activity



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



Topline results from Investigator-Sponsored Phase 2 Exploratory Biomarker Trial in Alzheimer's Disease

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

An open-label, Investigator-Sponsored exploratory biomarker study in patients who have MCI to Mild AD conducted in clinician's practice

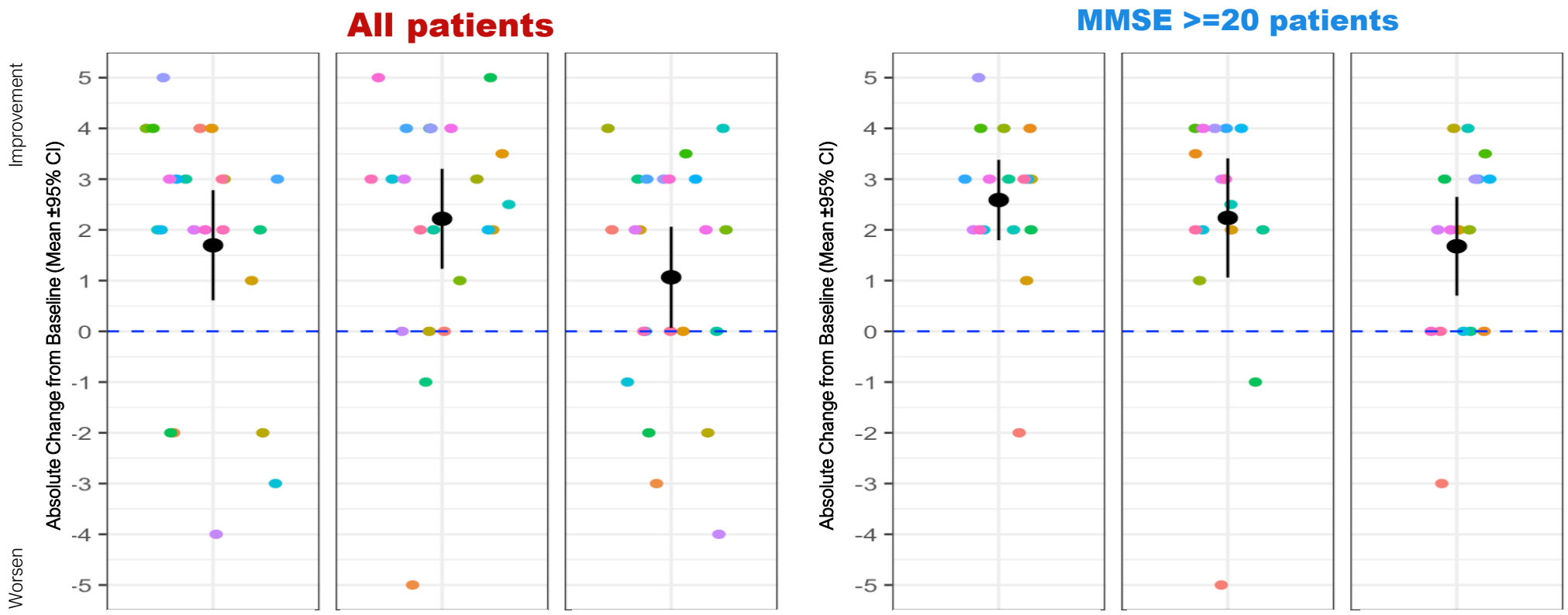
- The study enrolled 23 patients 55-89 years old, males and females experiencing cognitive decline – 17 patients with MMSE >=20 (suggesting mild cognitive impairment to mild dementia) and 6 patients with MMSE <20 (suggesting moderate dementia)
- Study seeks to measure changes in cognition through verbal 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
  - Modified Alzheimer's Disease Assessment Scale - Cognitive Subscale (ADAS-Cog12) Change
  - Mini-Mental State Examination (MMSE) Change
  - Glucose Serology/Metabolic Level Change

Key findings

- Vast majority of patients saw significant improvements with NE3107 treatment in the Global Rating of Change (overall impression of patient's daily abilities) ($p<0.0001$ to $p<0.05$)
- 82% of 17 patients with MMSE >=20 experienced a 2.6 points improvement in ADAS-Cog12 ($p=0.0046$)
- Improvements in cognition correlates with improvements in Global Rating of Change
- Reductions in TNFα (an initial factor driving inflammation) after NE3107 treatment is significantly correlated with improvements in cognition
- NE3107 treatment associated with trending improvements in the ratio of p-tau:Aβ
  - 60% 10 patients with MMSE >=20 improved 0.002 in the ratio of phospho-tau / aβ ($p=0.055$)
- Imaging data suggest patients experienced certain changes in brain after NE3107 treatment
  - 24% of patients with MMSE >=20 had increased blood flow in the brain while 6% declined. Blood flow serves as a marker for metabolism and brain activity
  - 41% to 47% of patients with MMSE >=20 had reduced the hyperactivation that results when the hippocampus (part of the brain that plays a major role in learning and memory) is stressed by insufficient blood flow while 6% declined
  - 41% of patients with MMSE >=20 had increased levels of glutathione (referred to as the master antioxidant and regulator of oxidative stress[1])
- NE3107 was well tolerated with no drug-related AEs reported

1. Teskey et al. *Advances in Clinical Chemistry* Volume 87, 2018, Pages 141-159

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

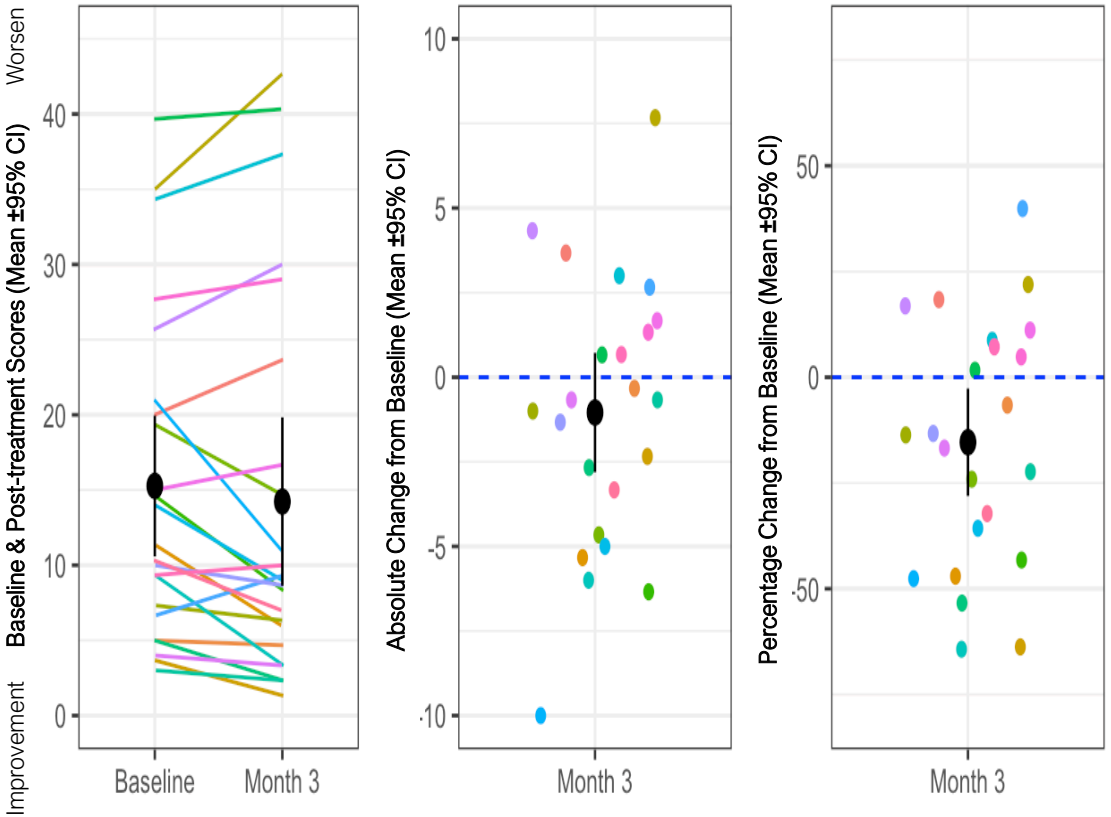


All patients			MMSE >=20 patients		
Clinician Observation	Patient Reported	Partner Reported	Clinician Observation	Patient Reported	Partner Reported
18/23 (78%)	18/23 (78%)	13/23 (57%)	16/17 (94%)	15/17 (88%)	11/17 (65%)
Mean = +1.70	Mean = +2.22	Mean = +1.07	Mean = +2.59	Mean = +2.24	Mean = +1.68
p=0.0068	p=0.0007	p=0.0464	p=0.0001	p=0.0039	p=0.0078

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

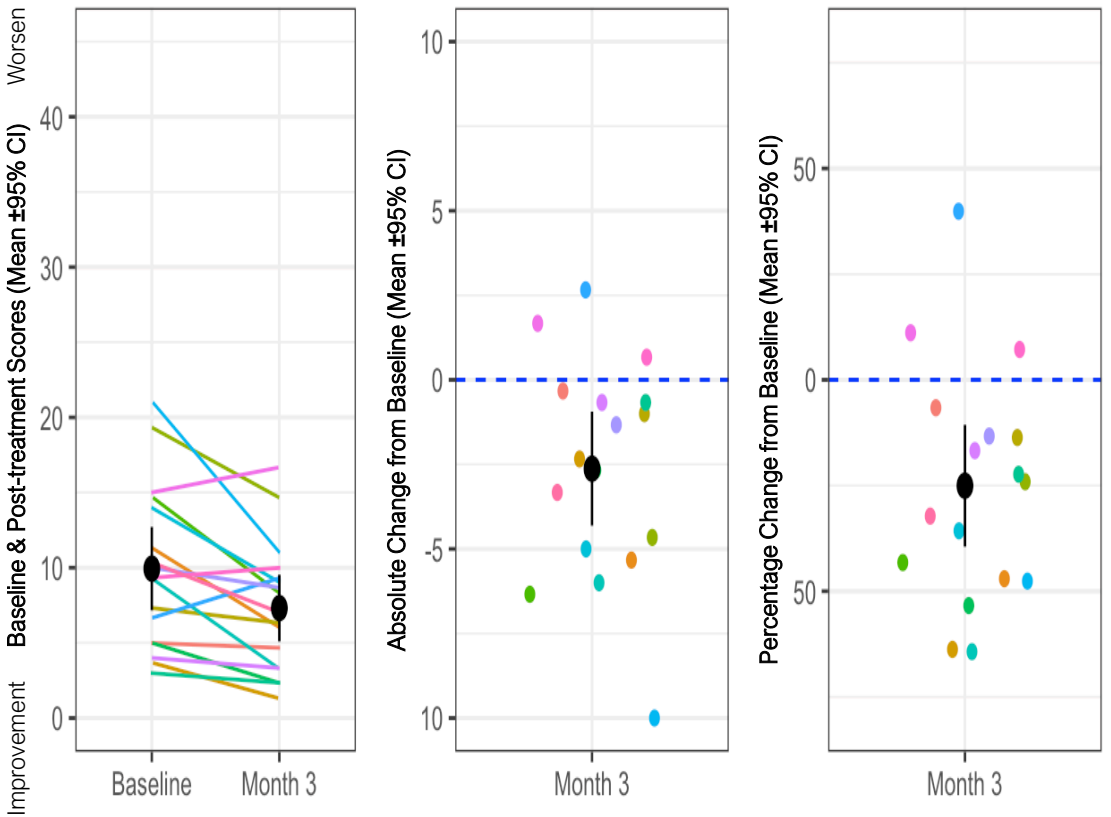
Significant improvements in ADAS-Cog12 assessment of cognition among MCI/Mild AD patients

All patients



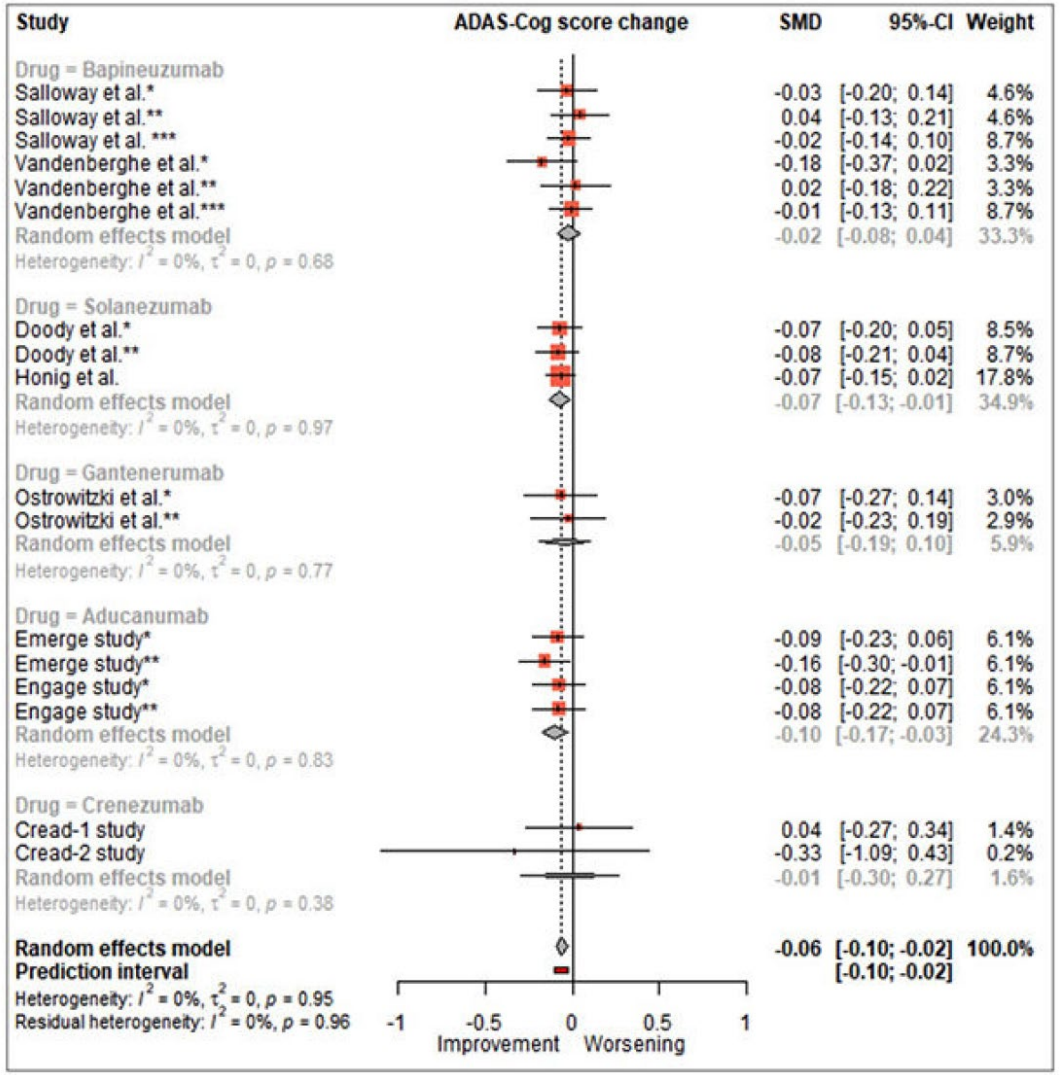
14/23 improved (61%)
Mean Absolute Change = -1.04 (p=ns)
Mean % Change = -15.4% (p=0.03)

MMSE >=20 patients



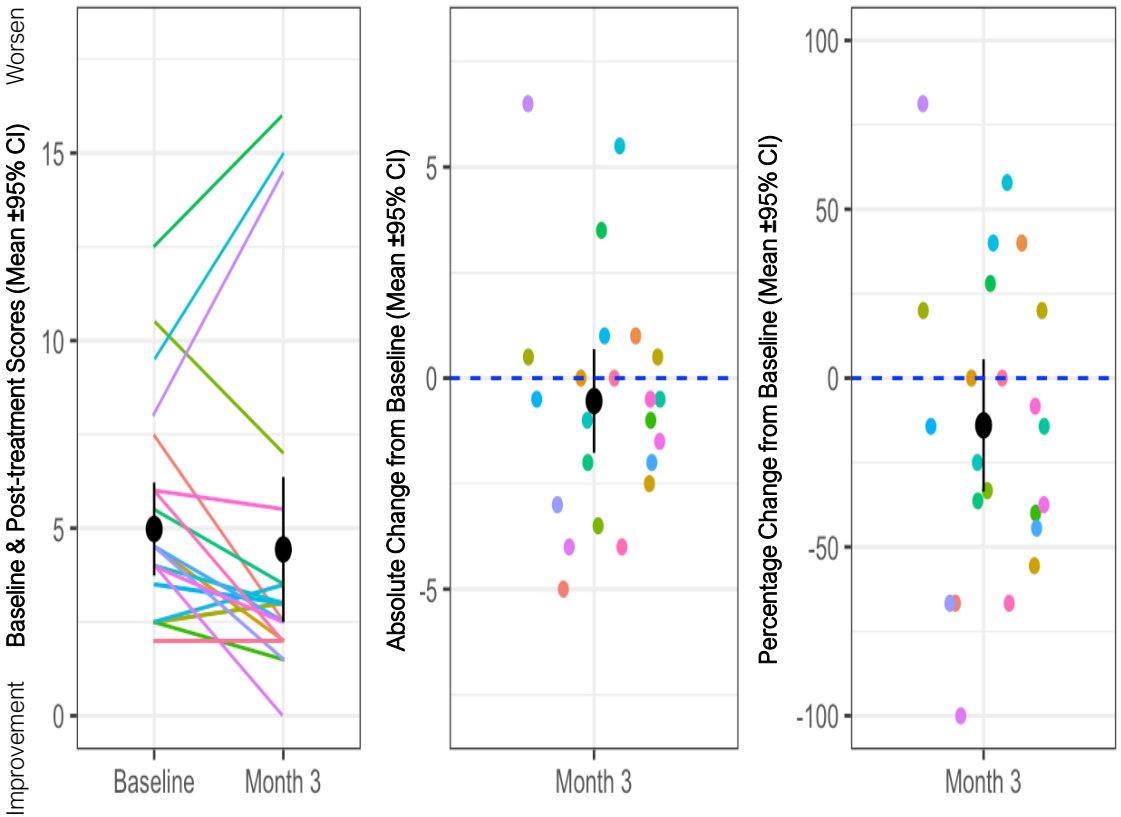
14/17 improved (82%)
Mean Absolute Change = -2.6 (p=0.0046)
Mean % Change = -25.1% (p=0.0026)

ADAS-Cog comparative change



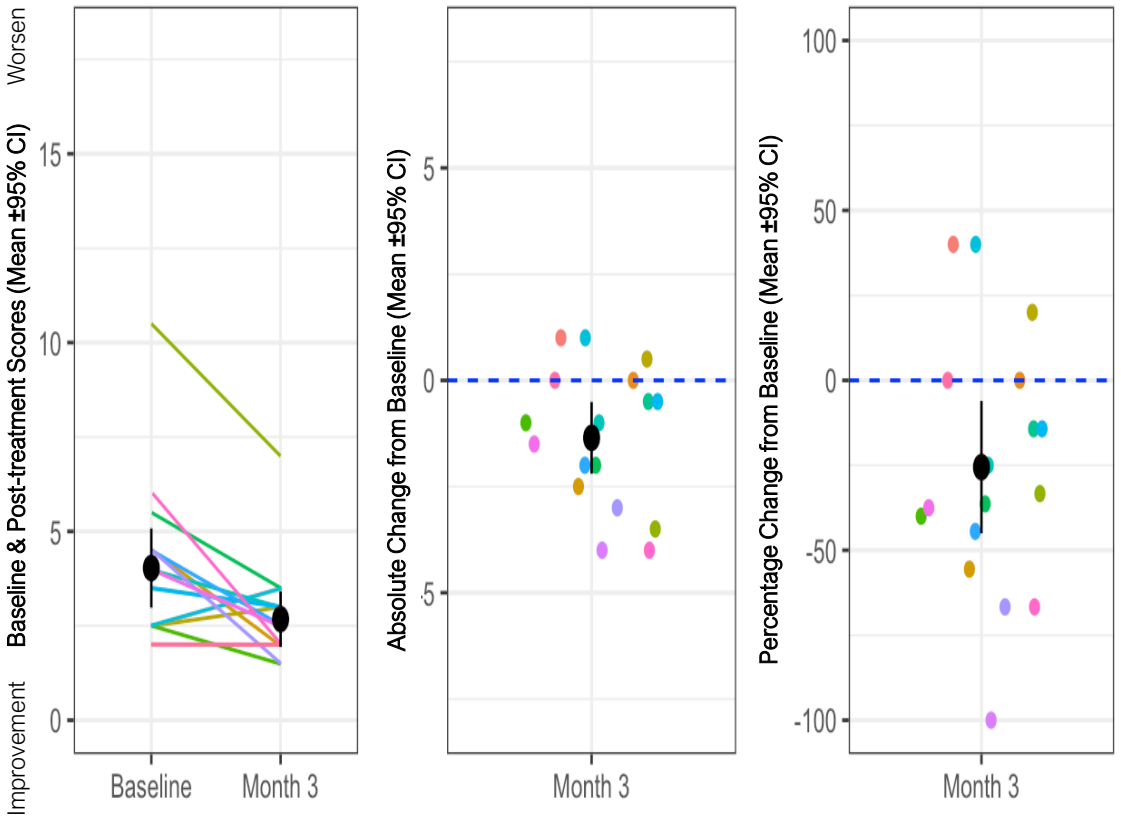
Cognition improvements also seen on the Quick Dementia Rating Scale

All patients



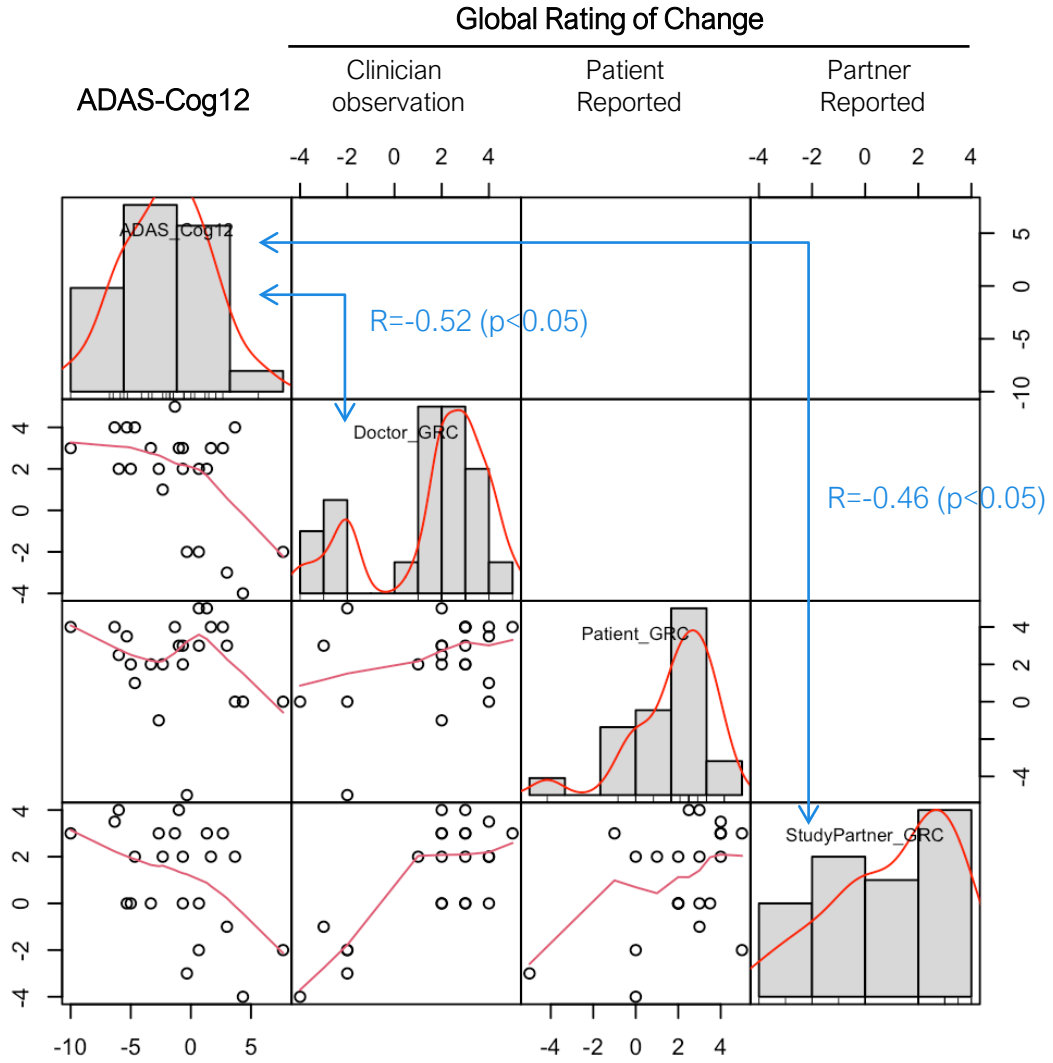
14/23 improved (61%)
Mean Absolute Change = -0.54 (p=ns)
Mean % Change = -14.0% (p=ns)

MMSE >=20 patients



14/17 improved (71%)
Mean Absolute Change = -1.35 (p=0.0052)
Mean % Change = -25.54% (p=0.025)

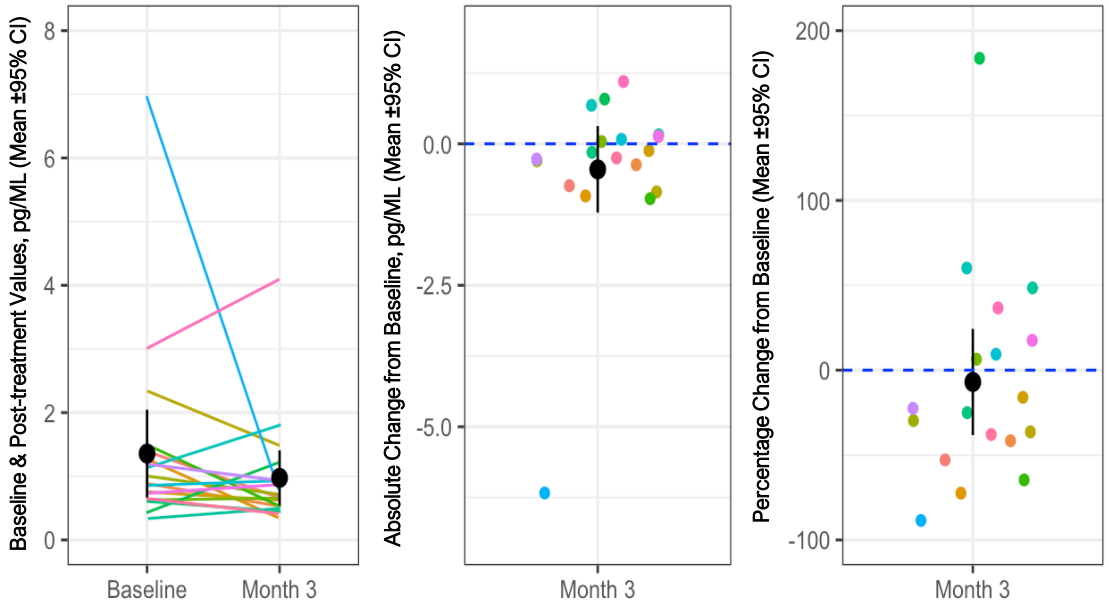
Improvements in cognition correlates with improvements in Global Rating of Change



Improvements on TNFα among MCI/Mild AD patients

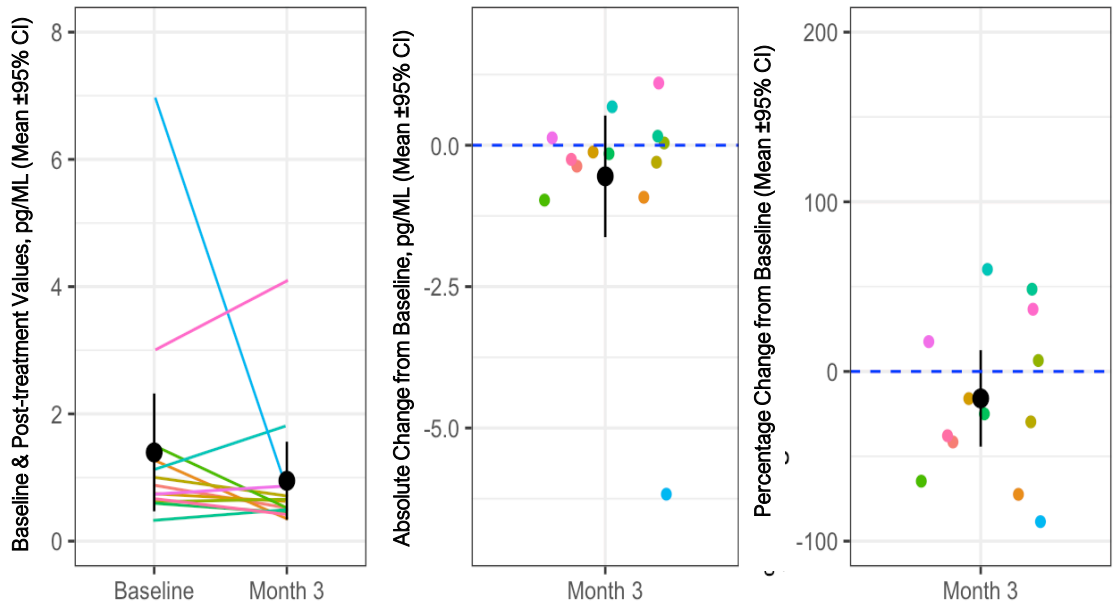
Tumor Necrosis Factor Alpha (TNFα) is a cytokine identified as a major regulator of inflammation whereby its excessive activation is associated with chronic inflammation[1]

All patients



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

MMSE >=20 patients

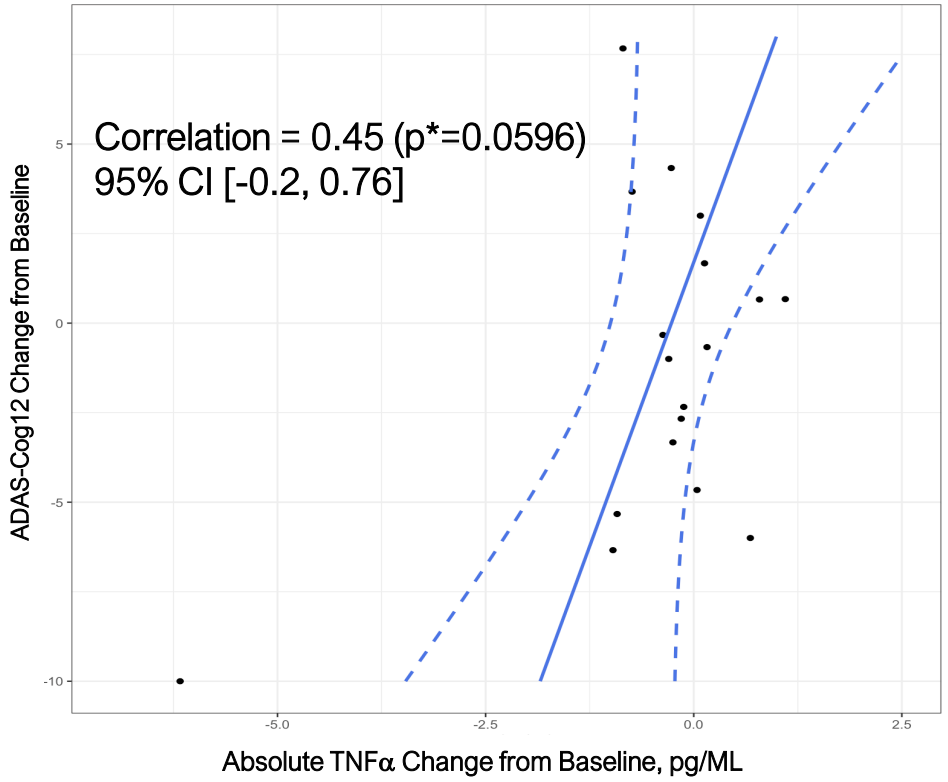


8/13 improved (62%)
Mean Absolute Change = -0.55 (p=ns)
Mean % Change = -15.9% (p=ns)

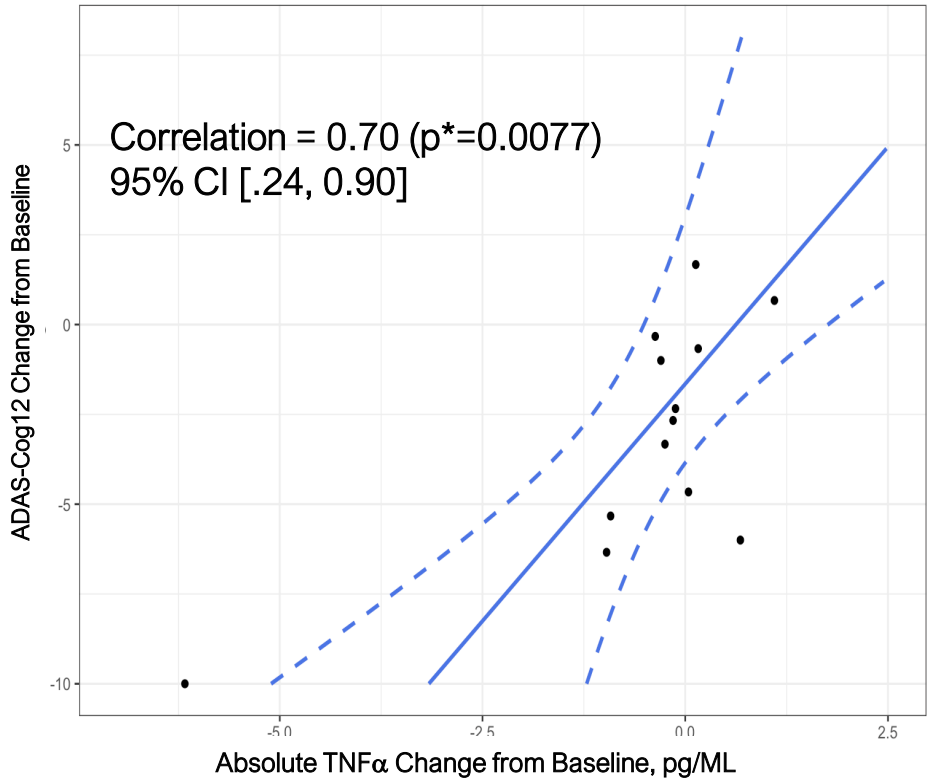
1. Jang et al. Int J Mol Sci. 2021 Mar; 22(5): 2719.

Improvements in TNFα significantly correlated to improvements in ADAS-Cog12

All patients



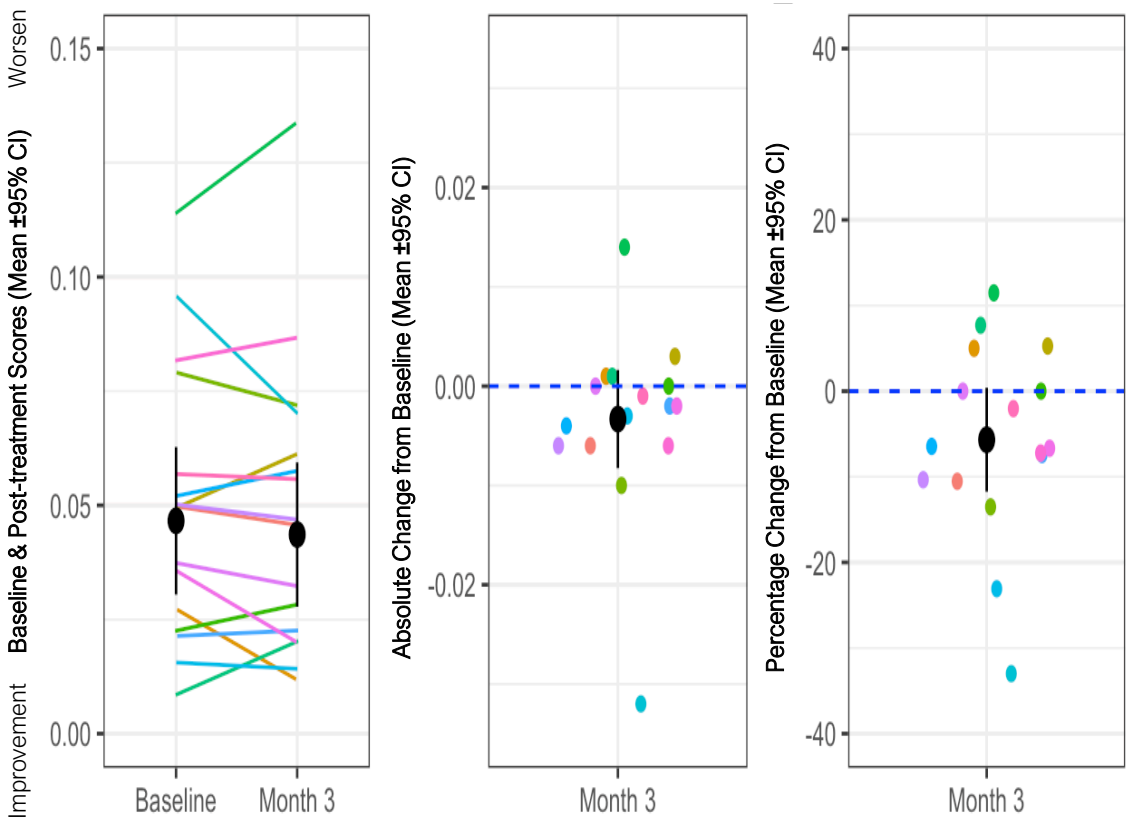
MMSE >=20 patients



* Pearson Correlation Test

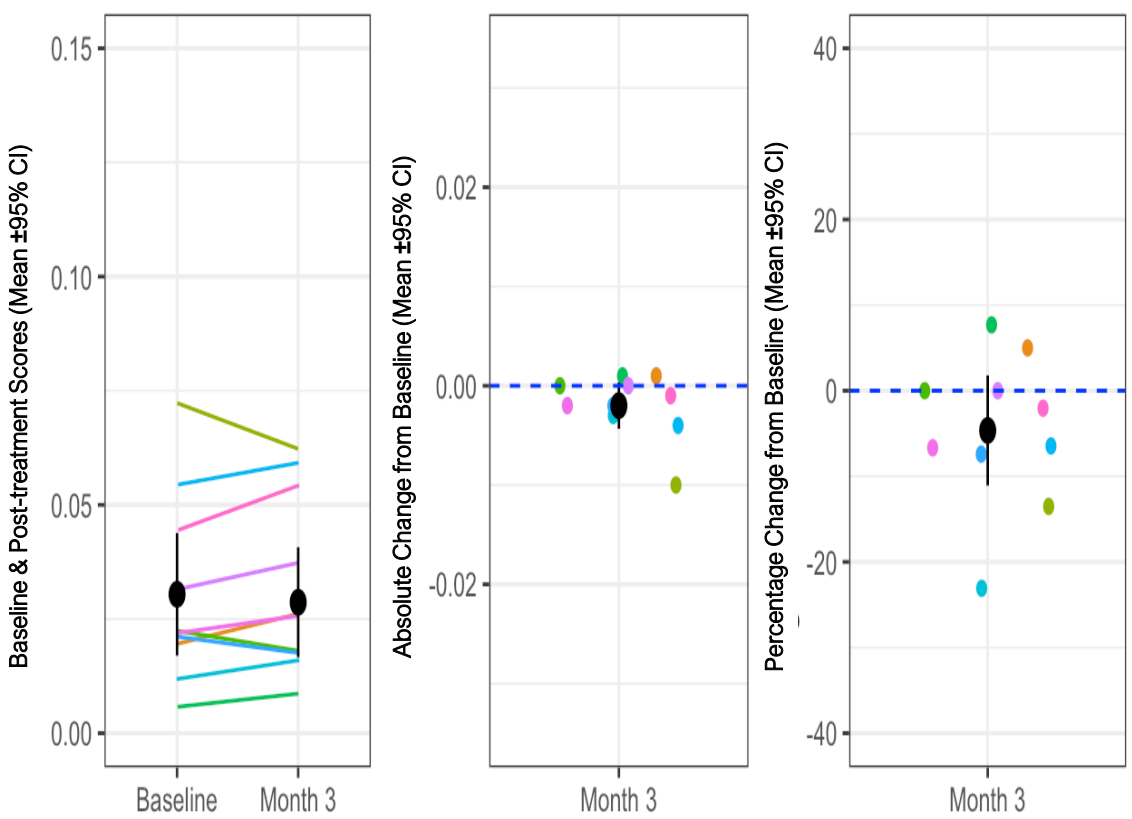
Trending improvements in the CSF p-tau : $A\beta_{42}$ Ratio, a predictive measure of PET amyloid status[1]

All patients



10/16 improved (63%)
Mean Absolute Change = -0.0033 (p=0.069)
Mean % Change = -5.67% (p=0.079)

MMSE >=20 patients

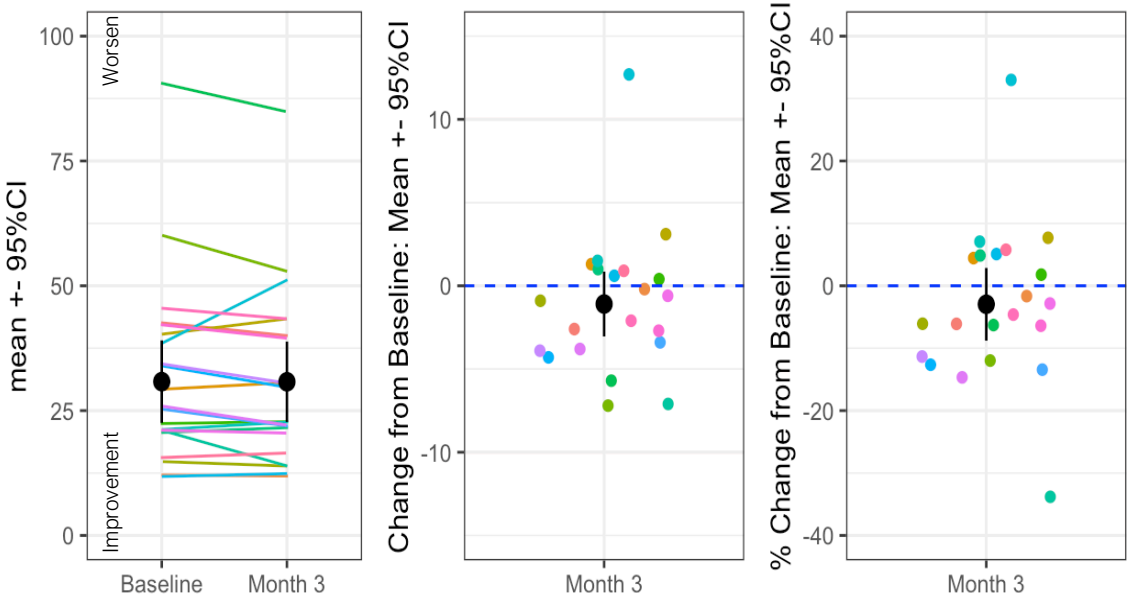


6/10 improved (60%)
Mean Absolute Change = -0.002 (p=0.055)
Mean % Change = -4.65% (p=ns)

1. Campbell et al. *Alzheimers Dement (Amst).* 2021; 13(1): e12190.

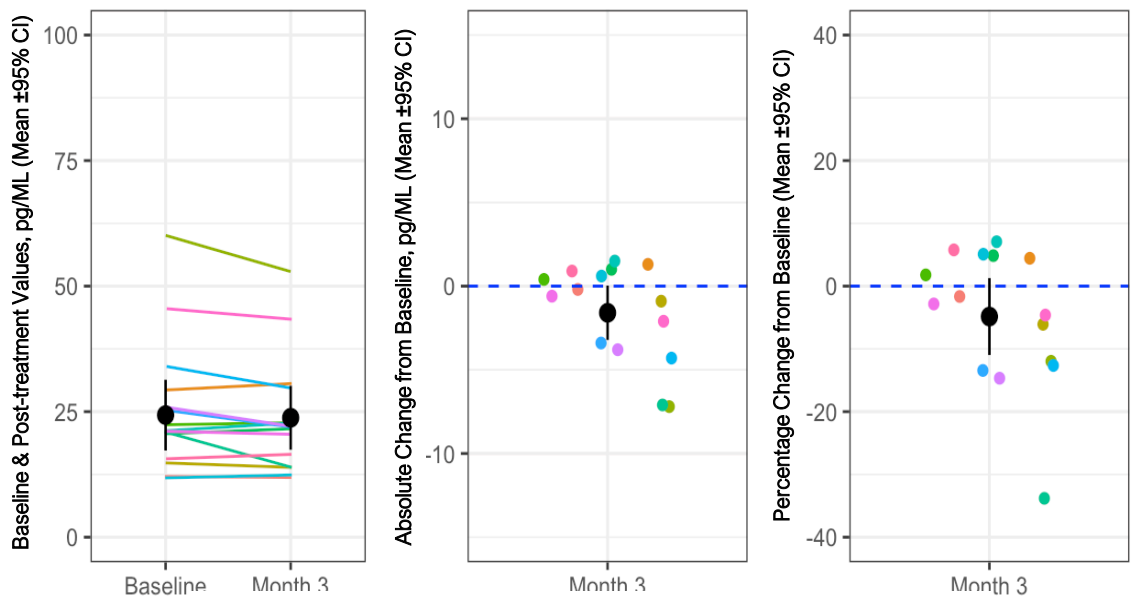
Modest improvements in CSF p-tau

All patients



13/21 improved (62%)
Mean Absolute Change = -1.10 (p=0.109)
Mean % Change = -2.95% (p=ns)

MMSE >=20 patients



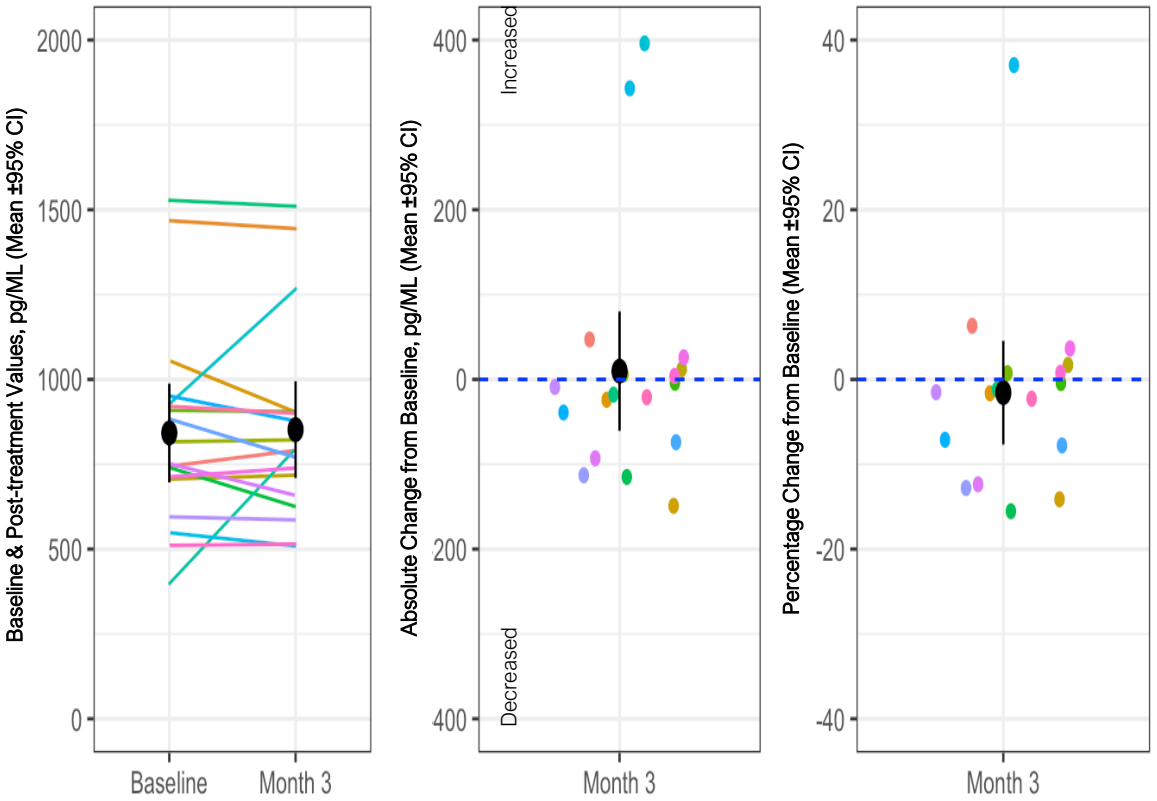
9/15 improved (60%)
Mean Absolute Change = -1.59 (p=ns)
Mean % Change = -4.97% (p=ns)

- NE3107 decreased CSF p-tau by 3% to 5% over 3 months compared to aducanumab's 13% over 12 months[1]
- Due to NE3107's mechanism, reduction in p-tau levels are expected to increase and accumulate over time

1. Hansson et al. 2021 Late-breaking roundtable, 14th Clinical Trials on Alzheimer's Disease (CTAD) Conference

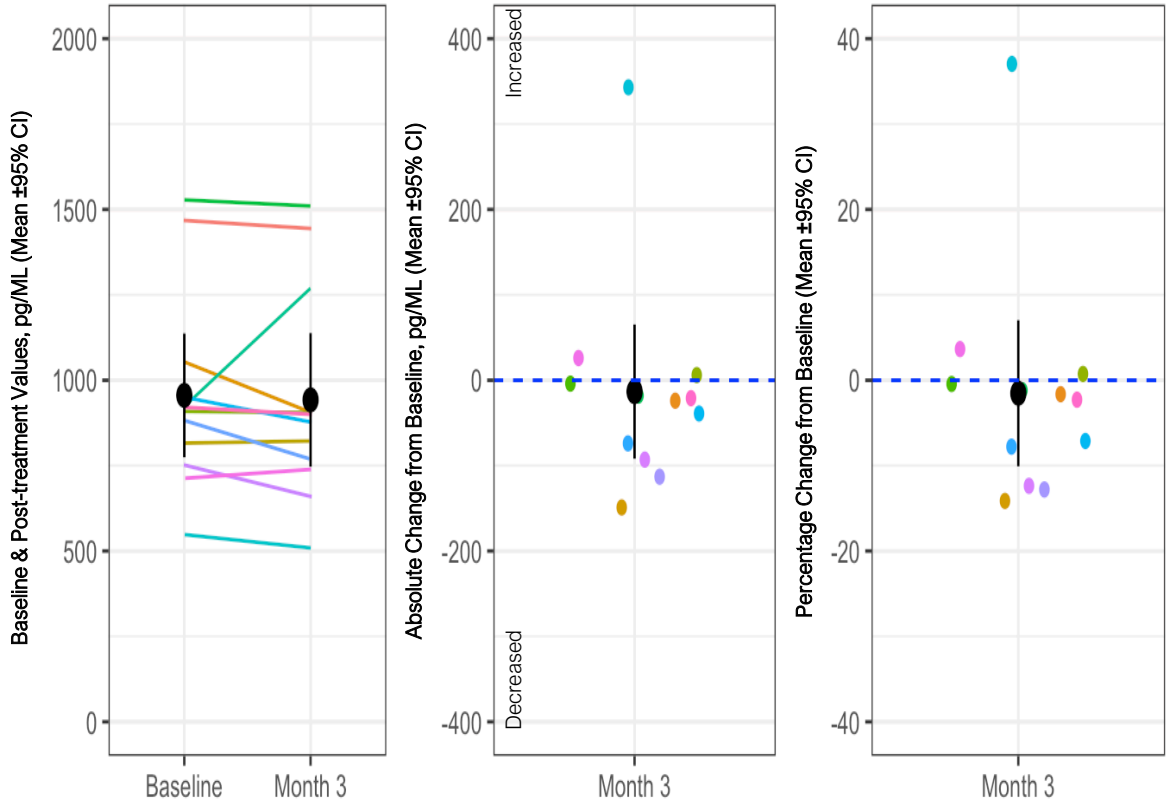
Modest improvements in CSF $A\beta_{42}$

All patients



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

MMSE >=20 patients



9/12 decreased (75%)
Mean Absolute Change = -13.3 (p=ns)
Mean % Change = -1.46% (p=ns)

Early examination of imaging data shows promising results

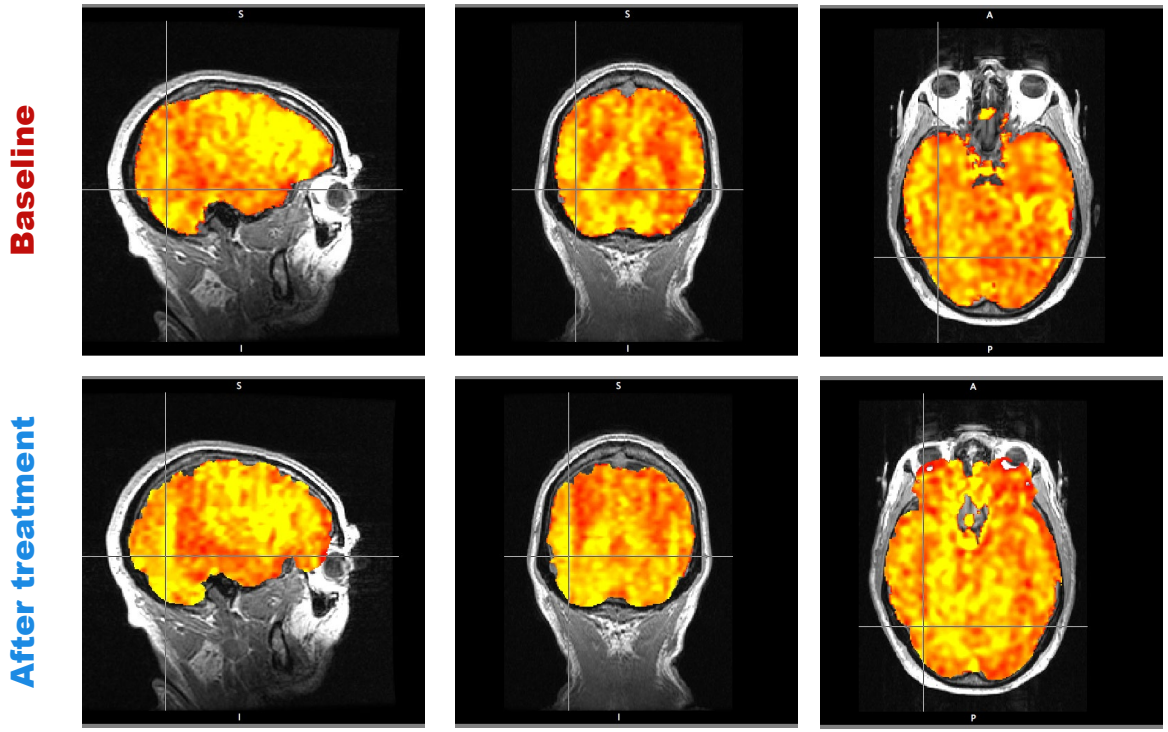
- Quantitative scoring of imaging data is currently underway
- Review of imaging scans and data by the study's Principal Investigator (who is an imaging expert) indicates that NE3107 treatment in patients with MMSE >=20 is associated with certain changes in the brain
  - 24% of patients had increase blood flow in the brain while 6% declined. Blood flow is a marker for metabolism and brain activity
  - 41% to 47% of patients had reduced the hyperactivation that results when the hippocampus (part of the brain that plays a major role in learning and memory) is stressed by insufficient blood flow while 6% declined. This is a marker for network connectivity in the brain
  - 41% of patients had increased levels of glutathione (often referred to as the master antioxidant and regulator of oxidative stress[1]) in the brain while 35% declined

1. Teskey et al. *Advances in Clinical Chemistry* Volume 87, 2018, Pages 141-159

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)

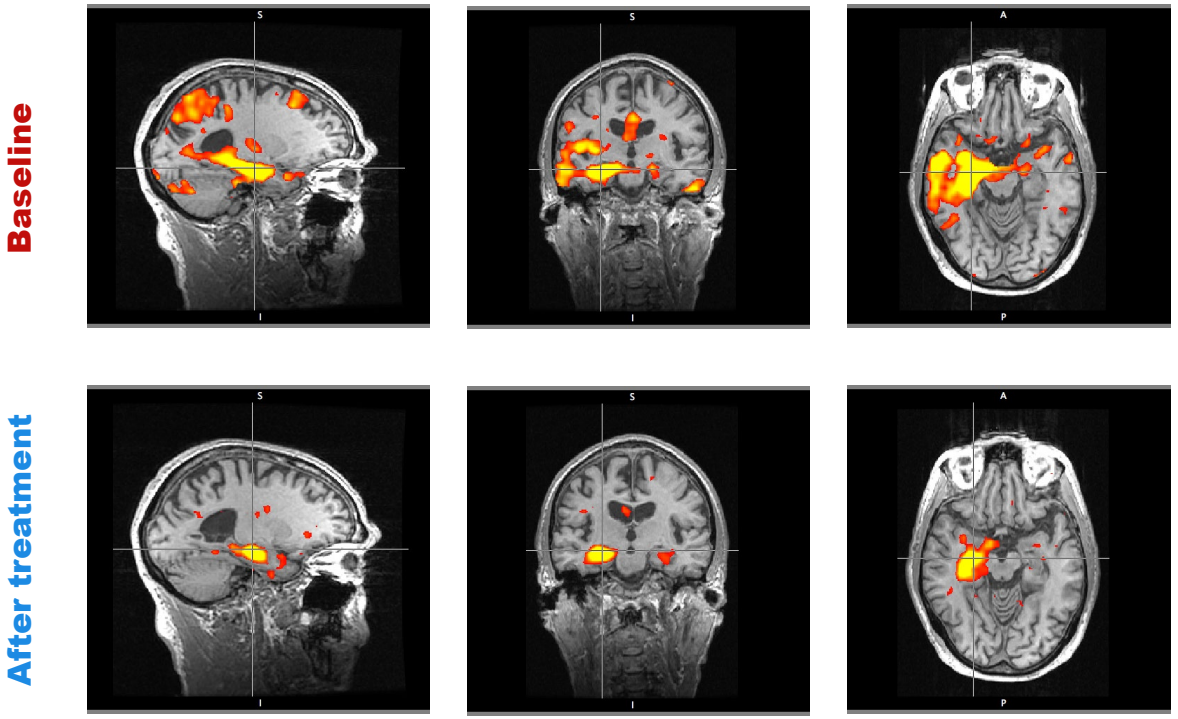


- 24% of 17 patients with MMSE >=20 had “signal” improvements in the ASL in relevant areas (temporal parietal occipital) compared to baseline. Increased blood flow serves as a marker for brain activity
- 6% of 17 patients declined

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

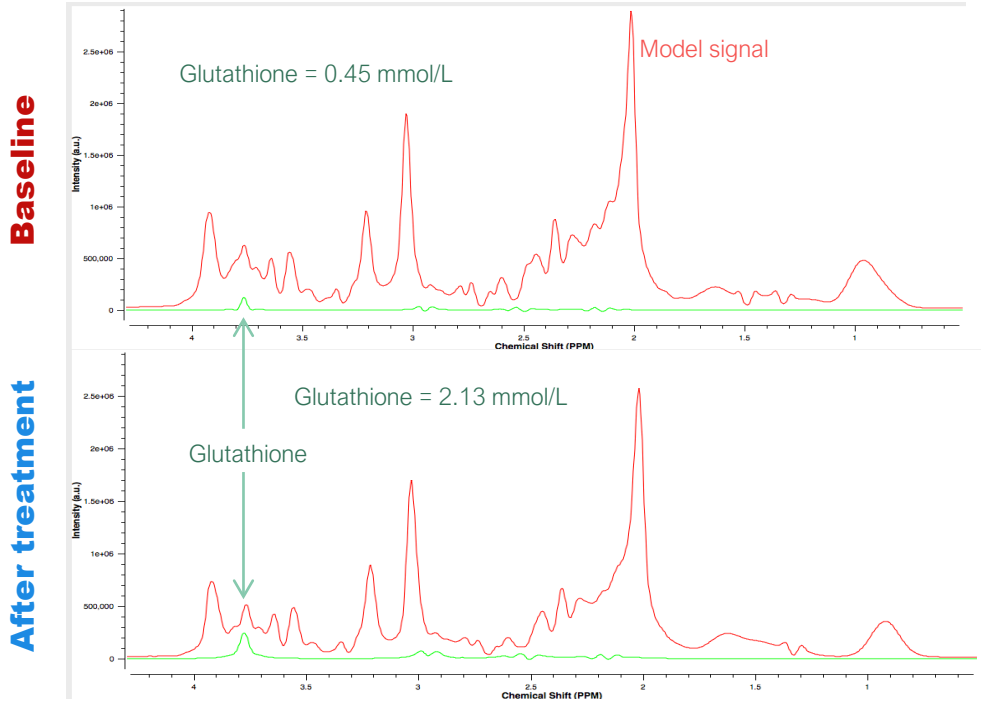
- Left lobe: 41% of 17 patients improved while 6% declined
- Right lobe: 47% of 17 patients improved while 6% declined

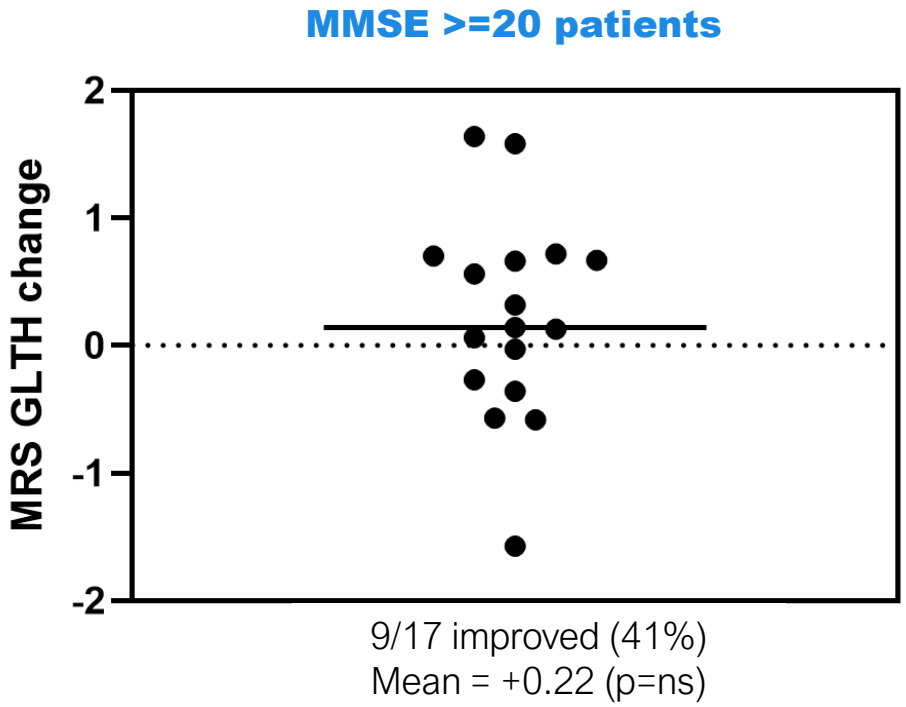
1. Aβ-induced change causes hyperactivity in cortical and hippocampal neurons as well as network hypersynchrony. Busche and Konnerth. 2016 Phil. Trans. R. Soc. B 371: 20150429

NMR spectroscopy shows that NE3107 is associated with increased glutathione in the brain

- Nuclear magnetic resonance spectroscopy is used to detect the presence of specific chemicals
- NMR spectroscopy was used to quantify the level of glutathione (referred to as the master antioxidant and regulator of oxidative stress[1])

Patient N16 – Global Rating of Change +3.0 (Partner Reported)





1. Teskey et al. *Advances in Clinical Chemistry* Volume 87, 2018, Pages 141-159

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
- Continued hypothesis testing and data analyses will be presented by the Principal Investigator and his team at the *Clinical Trial in Alzheimer's Disease (CTAD)* annual conference, to be held in San Francisco, CA November 29-December 2, 2022.

Recap

- This Investigator-Sponsored Phase 2 exploratory biomarker study was initiated to explore how NE3107's role in neuroinflammation and insulin resistance may link to the biomarkers historically used by the AD research community.
- This single-arm open-label study cannot isolate the placebo effect as a placebo-controlled double blinded trial can. However, it can provide insights that guide the design of future placebo-controlled and longer trials.
- The data in totality, however, suggest certain changes in the brain that are consistent with the mechanism for NE3107 and are unlikely to be accounted for by placebo effect, including:
  - Reduction of TNFα expression, which is significantly correlated with improvements in ADAS-Cog12
  - Reduction in the ratio of p-tau : Aβ in CSF
  - Improvements in blood flow in the brain, which serves as a marker for brain activity and metabolism
  - Reduction in the hyperactivation of the hippocampus towards a more normal state
  - Increased levels of glutathione (a regulator of oxidative stress) in the brain

Ongoing Phase 3 for Alzheimer's Disease

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 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 patients in each arm, 80% power
- Diagnosed with AD and without evidence of acute vascular pathology. 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.
  - Target engagement assessed in a small subset of active and placebo patients using PET to quantify cortical glucose utilization

Trial update

- Trial has enrolled one-half of targeted patients
- In blinded data, no drug-related adverse events have been seen in daily medical reviews
- DSMB will review data later this year to recommend whether the company should increase study size beyond targeted 316 patients

Phase 2 Drug-Drug Interaction and early Efficacy Trial in Parkinson's Disease

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

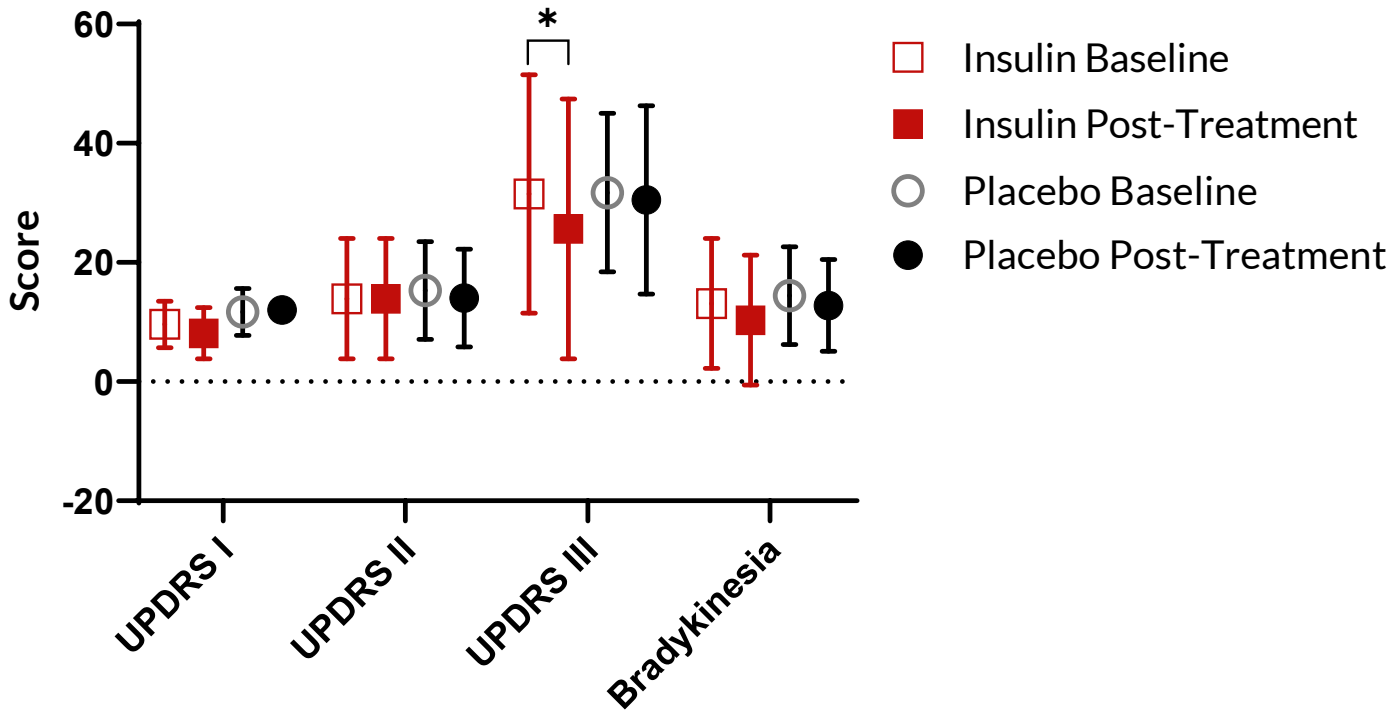
Remarkable parallels exist between AD and PD

- 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

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

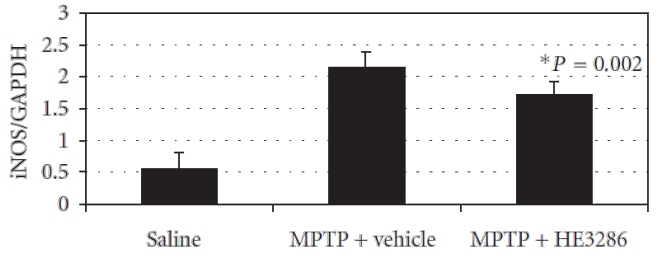


UPDRS: Unified Parkinson's Disease Rating Scale; Part I (mentation, behavior and mood); Part II (activities of daily living); Part III (Motor)
From: Novak 2019 *J Pone 0214364*

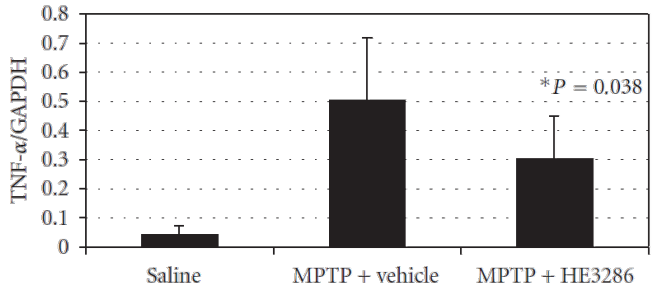
NE3107 Impact in Parkinson's Mice Models

Leads to a decrease in pro-inflammatory factors and preserves dopaminergic neurons

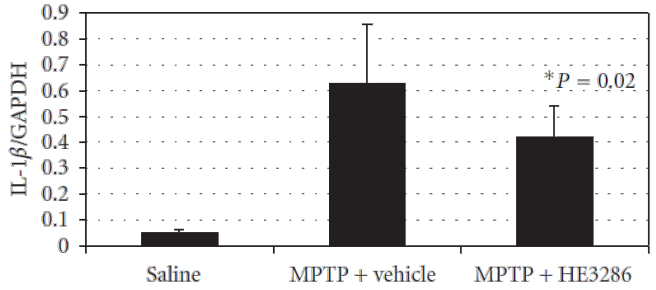
Reduced iNOS, TNF-α, and IL1β



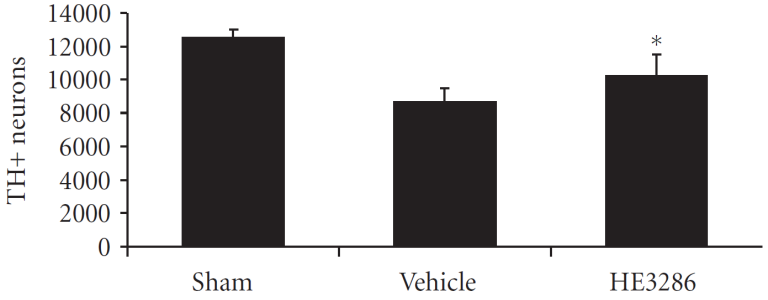
(a)



(b)



Preservation of Dopaminergic Neurons



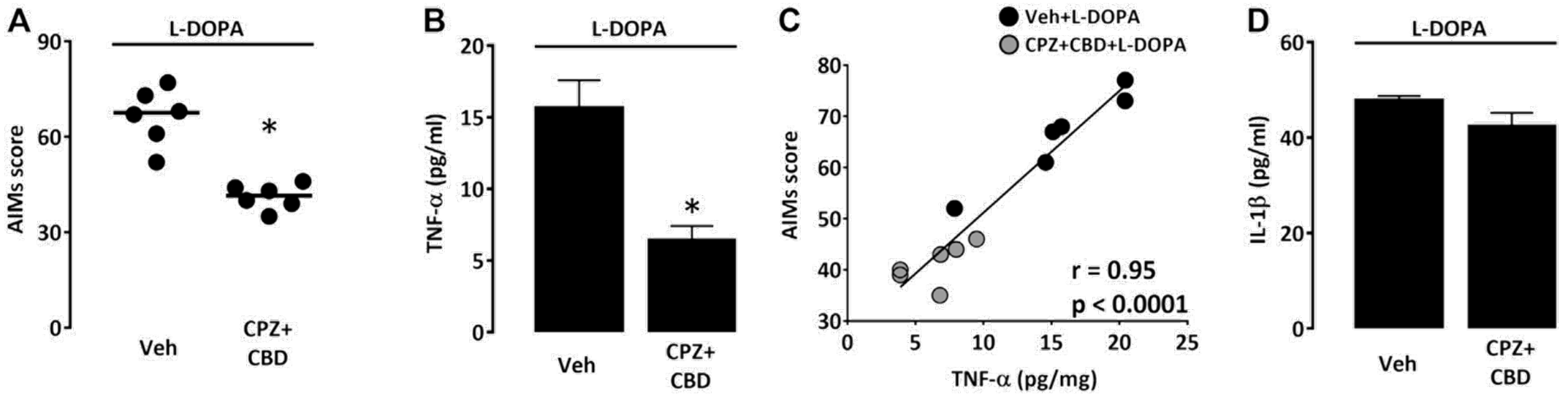
HE3286 attenuated TH-positive neuron loss in mice after MPTP injections.

Nicoletti 2012 Parkinson's Dis 969418

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

Inflammation's Role in LID

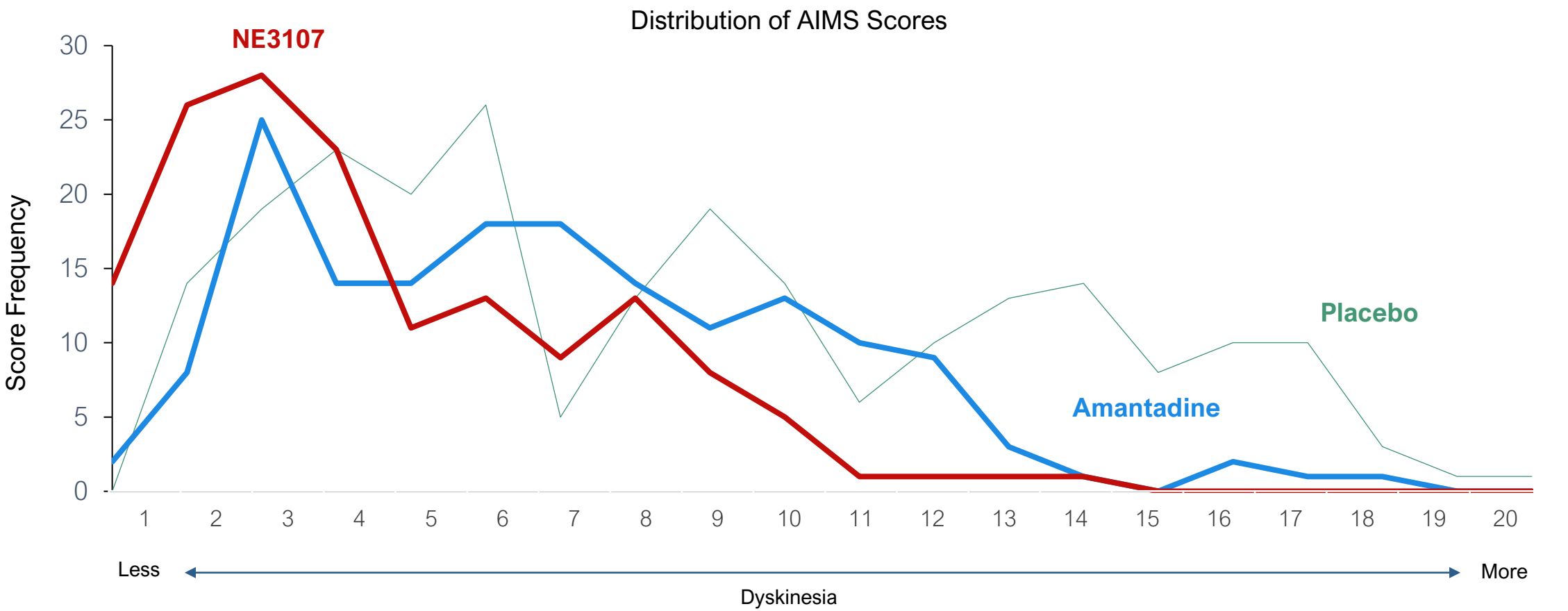
Reduction of TNF-induced inflammation reduces LID



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

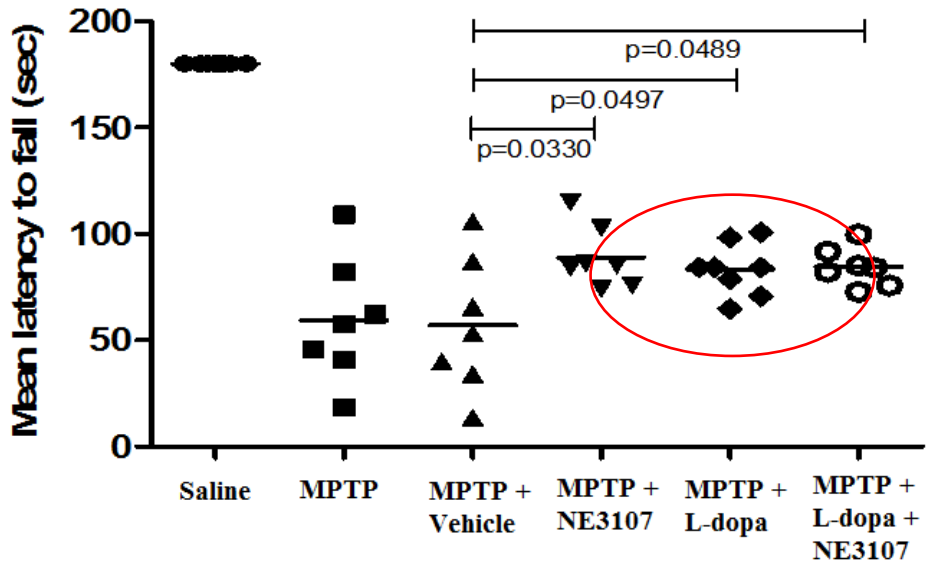
NE3107 decreases L-Dopa-induced dyskinesia (LID) in marmosets



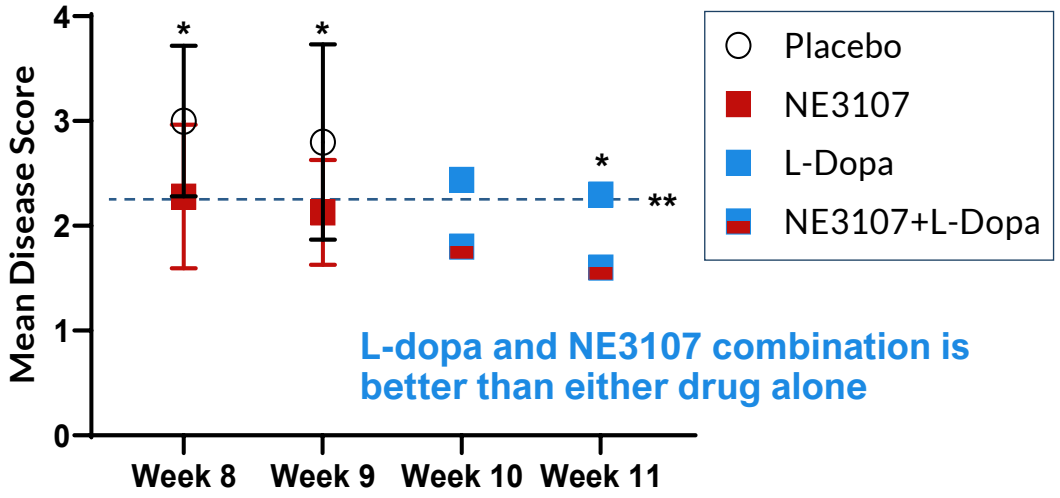
Abnormal Involuntary Movement Scale (AIMS)

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

MPTP Mouse



MPTP Marmoset treated at Week 8

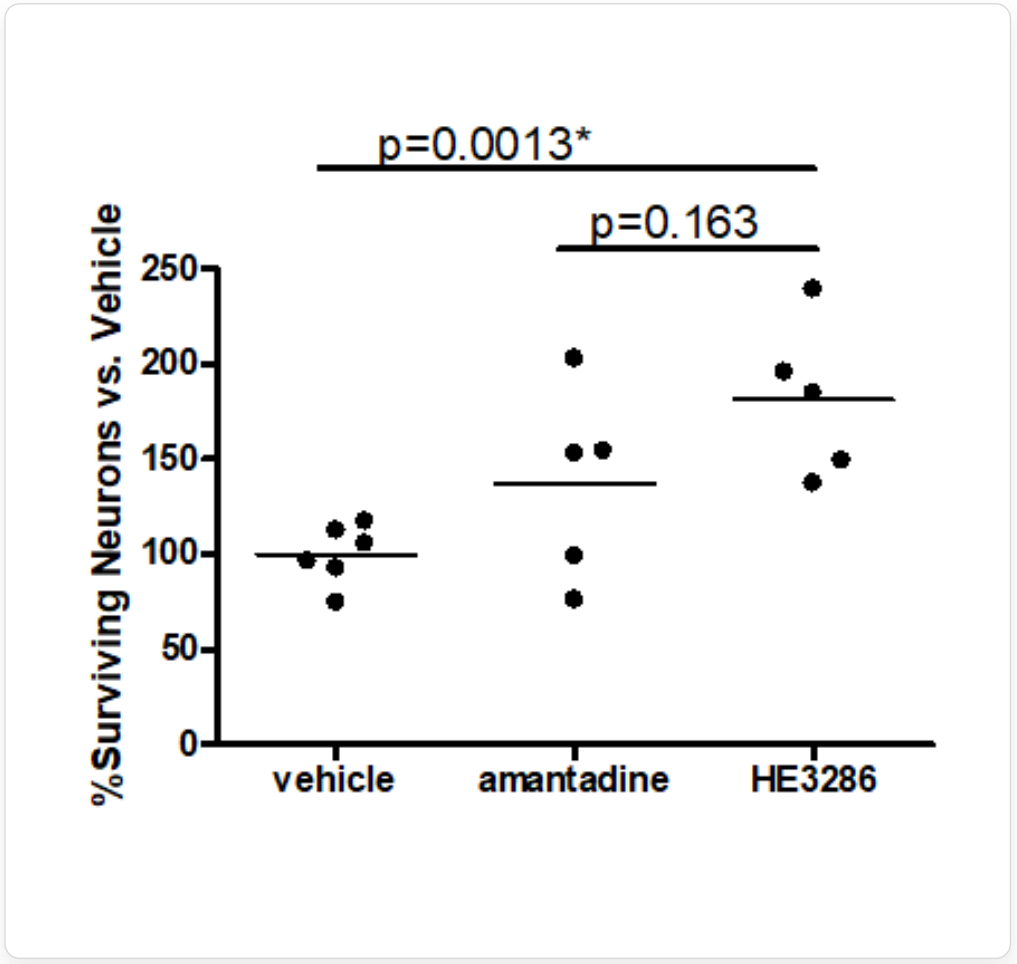


*p<0.001 compared to other treatment arm in time period
** L-Dopa at week 11 not statistically different from NE3107 at weeks 8 and 9

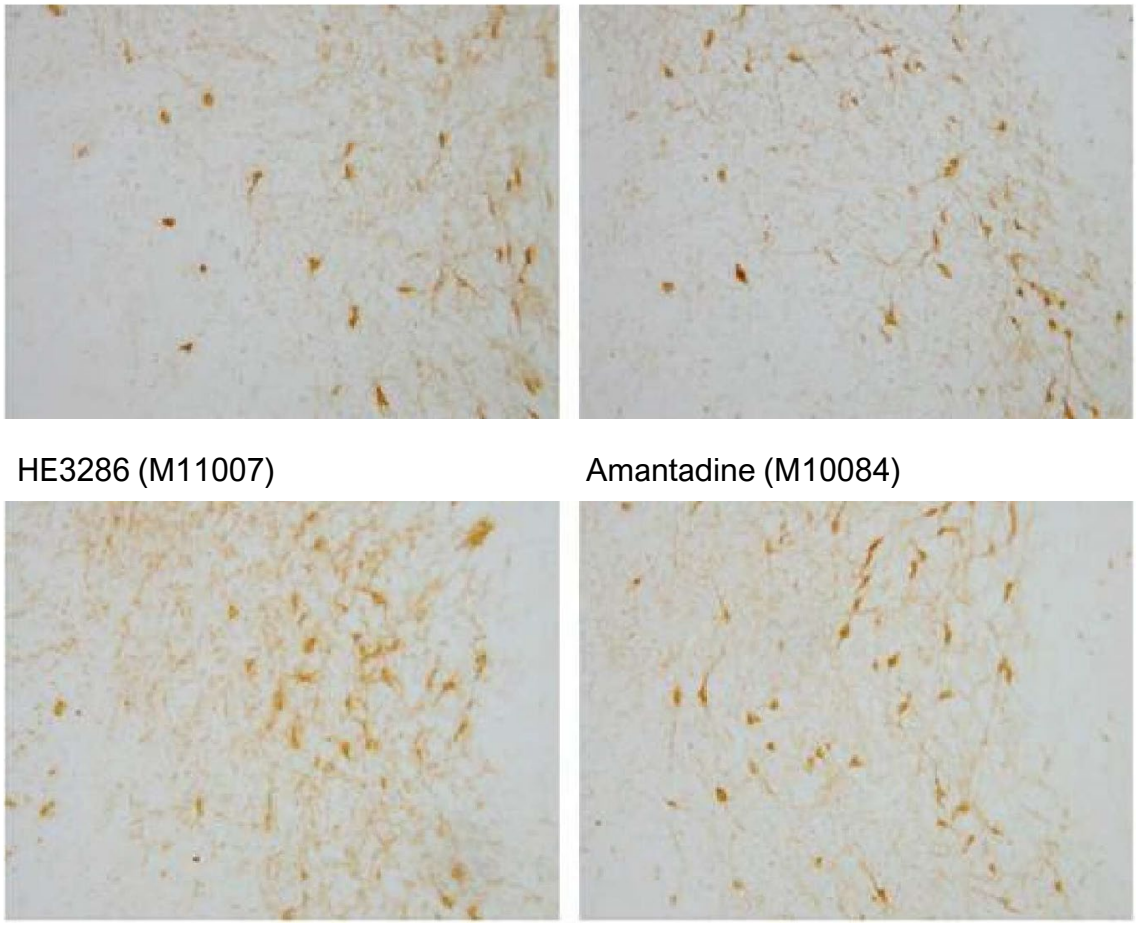
NE3107's promotoric effects observed within 4 days of treatment

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

Proceeding to Clinical Development for Parkinson's Disease

FDA requires a drug-drug interaction study before proceeding to later phase trials in patients that might use or need levodopa

- No indications of DDI in animal studies have been observed

Our Phase 2 is a combined DDI and efficacy program to see if we can detect an efficacy signal in man as we saw in marmosets

Decreased inflammation > increased insulin sensitivity > disease amelioration

- Promotoric activity
- Reduction in LID

Parkinson's Disease Clinical Development Program

NM201 Phase 2

- Assess NE3107 pro-motoric activity while satisfying FDA requirement for drug-drug interaction study with L-dopa

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

NM202 Phase 3

- Possibly pivotal design
- Evaluate NE3107 promotoric activity in 200 patients needing to start L-dopa or (depending on Phase 2 results) 200 patients on L-dopa therapy for more than 3 years
- Primary endpoint MDS-UPDRS parts 1-4

* Motor Disease Society Unified Parkinson's Disease Rating

Trial Update

- Trial should be fully enrolled over the next couple of months and read out by the end of 2022
- No drug-related adverse events have seen in daily medical reviews
- Seeing signal resembling results previously seen in marmosets
  - Detecting a signal from patients who have completed 28 days of treatment
  - Need all patients to complete study before we can quantify magnitude of impact
- Starting to plan follow-on Phase 2b/3 studies

Phase 2b Trial for BIV201 in Refractory 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

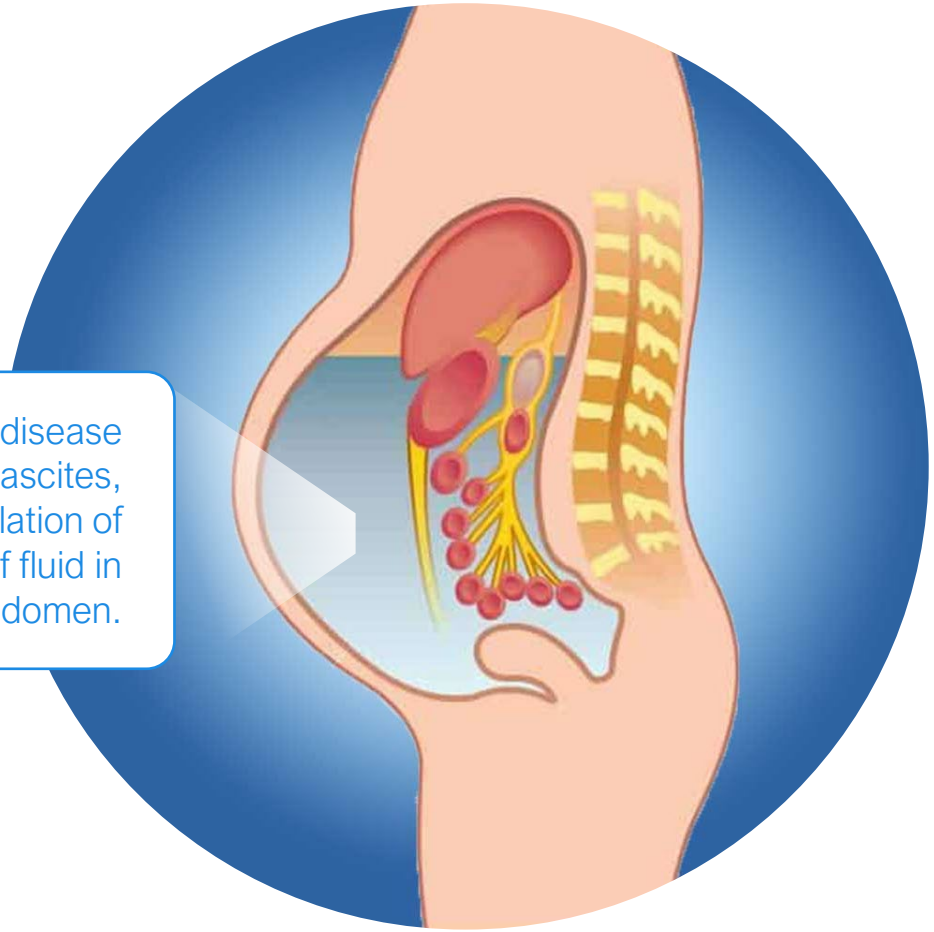
The kidneys are “burning out” by retaining massive quantities of salt and water

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



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. Seeking 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

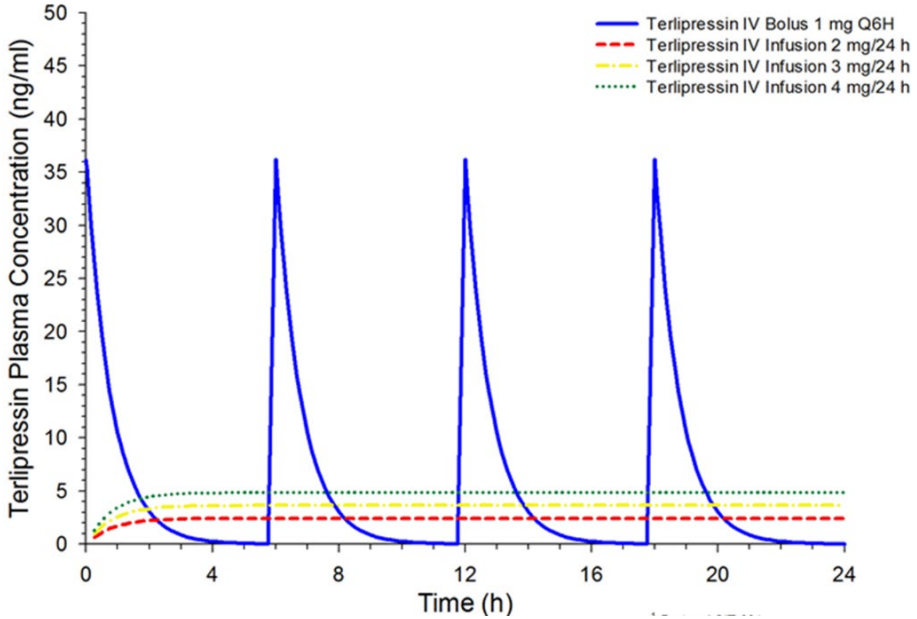
Portable pump
Carried in small satchel

*In clinical trials; not approved by FDA

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

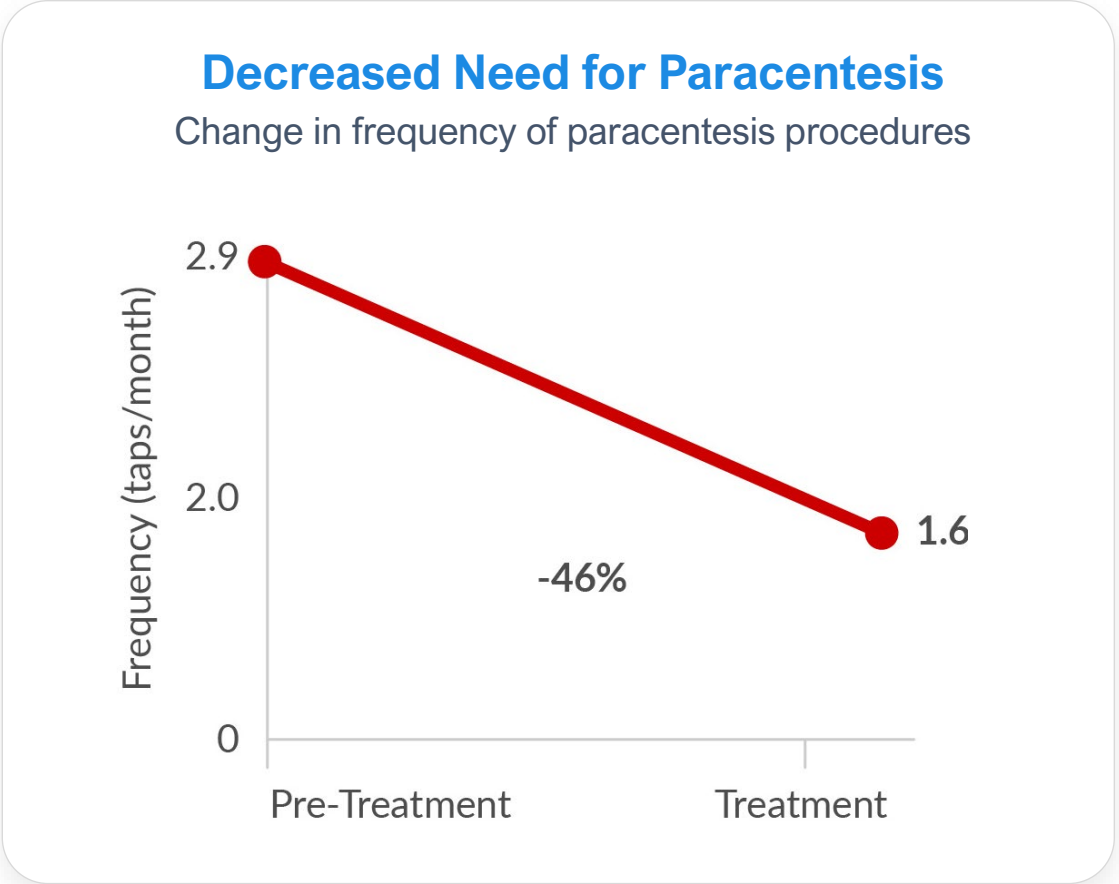
Continuous Infusion Terlipressin in 19 Refractory Ascites/HRS Patients

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)

Terlipressin Efficacy in Outpatients CIT-001

- Terlipressin (2- 4 mg/day) administered for 28 days by continuous infusion in 6 patients with refractory ascites
- Data from 28d treatment period compared to 28d pre-treatment period
  - Mean ascites volume removed decreased by 66% on average
  - 4/6 patients experienced ≥ 50% increase in the interval between LVPs after the start of treatment with terlipressin
  - 2/6 patients experienced extended control of ascites beyond the 28 days of infusion for a total of 72 and 63 days.
  - Four patients (two with SCr > 1.5 mg/dL) experienced a reduction in SCr levels during treatment.

Terlipressin is not approved in the United States

BioVie Phase 2a results - Bajaj 2020 J. Hepatol. 2020.73: S718-S719

Terlipressin Safety in Outpatients CIT-001

- Treatment generally well tolerated – no cardiac or ischemic issues
  - 3/6 patients completed 28d of treatment
- Vital signs, AEs of interest, clinical laboratory findings, and ECG monitoring unremarkable
- SAEs consistent with patient population with advanced cirrhosis
- No serious treatment-emergent adverse effects considered related to terlipressin
  - One patient discontinued treatment for treatment-related AE (asymptomatic hyponatremia), one for recurrent HE and one for leaking hernia

Terlipressin is not approved in the United States

Bajaj et al., Safety, Tolerability, Pharmacokinetics, and Efficacy of Terlipressin Delivered by Continuous Intravenous Infusion in Patients with Cirrhosis and Refractory Ascites. GastroHep published online 06 2022 https://doi.org/10.1155/2022/5065478
BioVie Phase 2a results - Bajaj 2020 J. Hepatol. 2020.73: S718-S719

BIV201 Development Plans

Ascites

- 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 patients
- Orphan drug designation; Method of use patent issued

HRS-AKI

- Planning a possible registrational Phase 3 trial in HRS-AKI*
  - For potentially better outcomes because earlier treatment is possible than for HRS Type 1
  - For potentially improved safety with continuous infusion dosing compared to Mallinckrodt's CONFIRM study of terlipressin in HRS type 1
- Mallinckrodt used bolus injections (1 or 2 mg given over approx. 5 min)
  - They achieved the efficacy objective but received two CRLs to date. NDA recently resubmitted[1]
- Agreement reached with FDA on key elements of Phase 3 trial design
  - We plan to use same PIs, same sites, and same drug product as our current ascites study
  - Orphan drug designation for HRS

biovie

- Hepatorenal syndrome – acute kidney injury

1 News Detail | Mallinckrodt Pharmaceuticals June 13, 2022

Trial update

- 11 trial sites are activated and screening/enrolling patients
- No treatment-related SAEs have been observed
- The trial is taking much longer than expected
- Patient recruitment has been hampered by Covid, which appears to have disproportionately affected this population of very sick patients
- The pace of patient screening, however, appears to be picking up in recent weeks
- At this pace, we anticipate data readout by mid-2023.

Recap

- Topline results from the Phase 2 exploratory biomarker trial suggest directional and exciting signals of NE3107's potential in Alzheimer's Disease (AD)
  - The majority of patients (especially those with MMSE >=20) saw improvements in ADAS-Cog12 and the Global Rating of Change (an instrument that tracks improvements in a patient's conditions, abilities and overall sense of well-being)
  - 60%+ of patients saw reductions in TNFα levels (i.e., inflammation), which is significantly correlated with ADAS-Cog improvements
  - 60%+ of patients experienced improvements in the ratio of p-tau : $A\beta_{42}$ in CSF[1]
  - Early readings of imaging data suggest fundamental biological improvements in blood flow and reduced oxidative stress that are consistent with the mechanism for NE3107 and are unlikely to be accounted for by placebo effect
- Other trials are progressing and should readout over the next 12 months
  - The Phase 2 trial for NE3107 in Parkinson's should be fully enrolled in a couple of months and readout by the end of 2022. This trial is primarily a drug-drug interaction study designed to determine if the promotoric activity seen preclinically can be detected in humans
  - The Phase 3 trial for NE3107 in Alzheimer's has enrolled one-half of its targeted 316 patients and should readout in mid-2023 unless a sample size increase (up to 400 patients) is recommended by the DSMB later this year
  - The Phase 2b trial for BIV201 in refractory ascites has been slower to enroll than expected and was especially impacted by Covid-19. Patient screening has picked up in recent weeks and should readout min-2023 if screening keeps up the current pace

1. The ratio of p-tau : Ab42 in CSF has been to be predictive of PET amyloid status. Campbell et al. *Alzheimers Dement (Amst).* 2021; 13(1): e12190

Thank You

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.

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

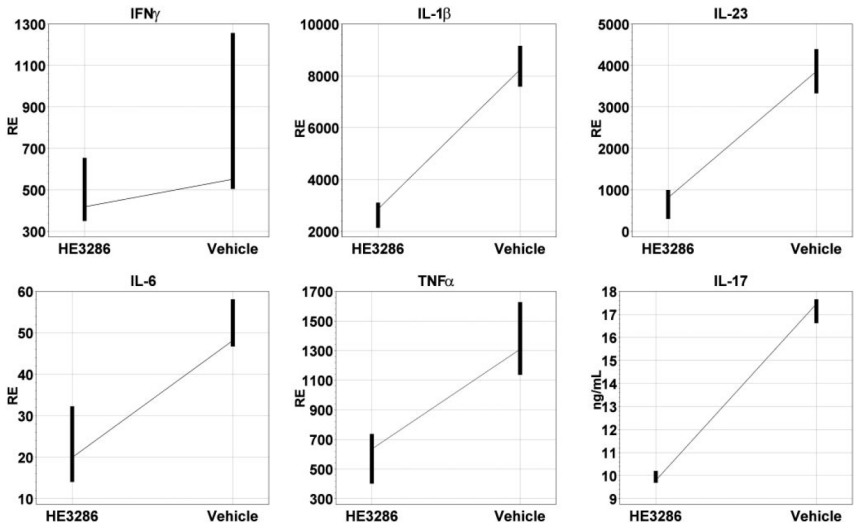
Clarence Ahlem , Neurodegenerative Disease Program
35+ years in biopharma
Vice President, Product Development Harbor Therapeutics
Director, Product Development, Hollis-Eden Pharmaceuticals
US San Diego

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

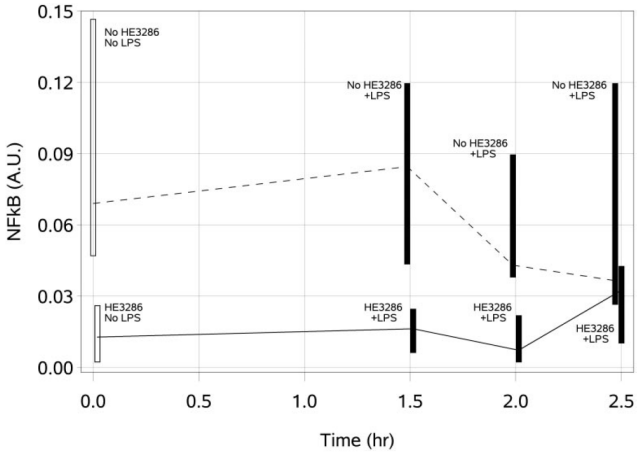
NE3107 Inhibits Systemic Inflammation

Decrease in pro-inflammatory factors across several different preclinical disease models

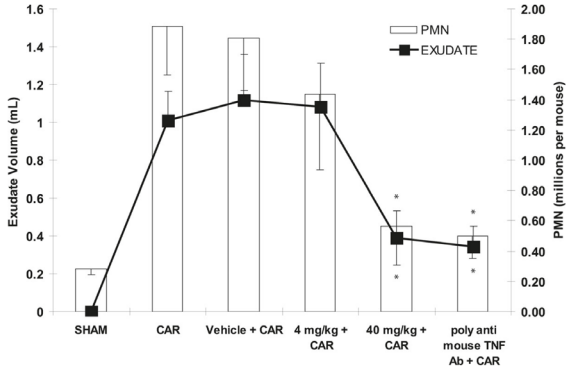
Decreased IFNγ, IL-1β, IL23, IL-4, TNFα and IL-17 Expression in an RA Model[1]

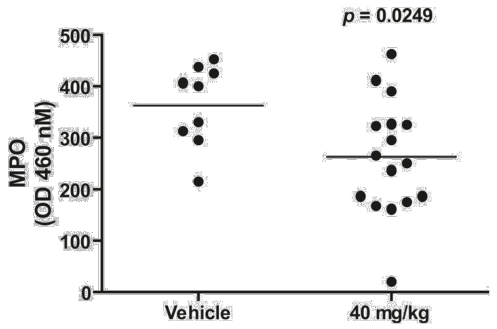


Decreased NFκB in an Endotoxin Shock Model



Decreased Inflammatory Cell Infiltrates and Myeloperoxidase in COPD Models[2]



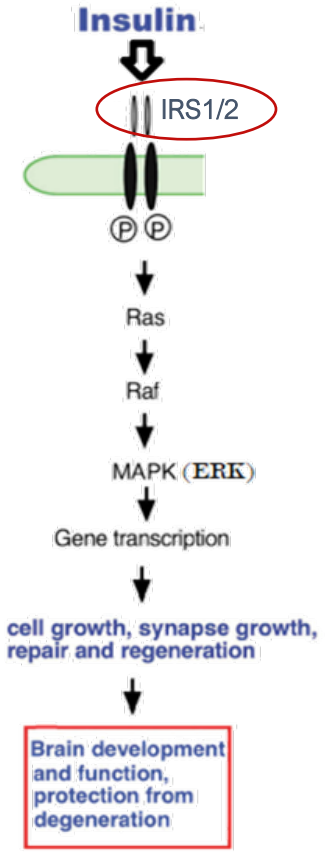


[1] Offner 2009 *J Pharmacol Exp Ther 329 1100*

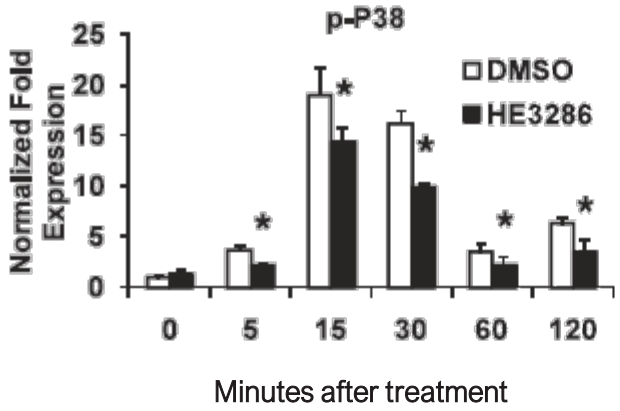
[2] Conrad 2010 *J Inflammation 7 52*

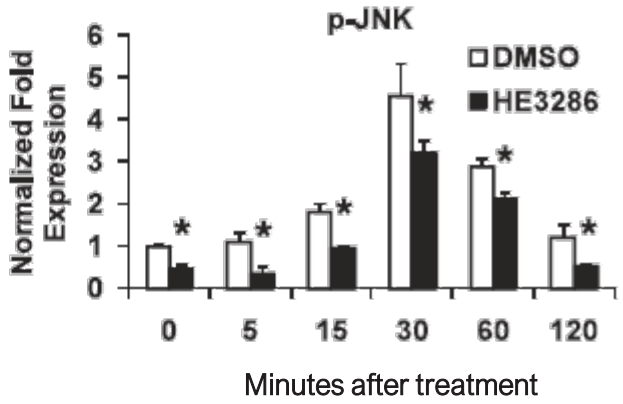
NE3107 Effect on Inflammatory ERK via IRS1/2

Targets inflammatory, but not homeostatic, ERK signaling



- Insulin Receptor Substrate (IRS) IRS1/2 can be activated by insulin *via* tyrosine phosphorylation, requires homeostatic ERK
- IRS1/2 also phosphorylated by proinflammatory serine kinases p38 and JNK, requires inflammatory ERK
  - When this happens, insulin signaling is blocked
- NE3107 blocks NFkB, p38 and JNK activation ➔ prevent IRS1/2 serine phosphorylation ➔ restores insulin signaling





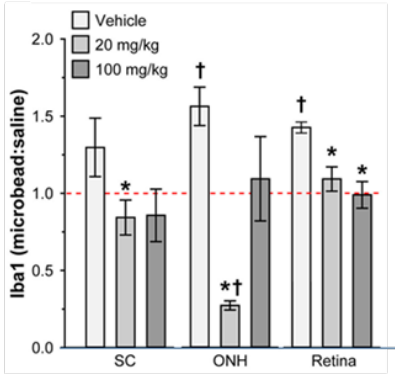
Lu 2010 *Am J Physiol Endocrinol Metab 298 E1036*

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

NE3107 Decreases Neuroinflammation

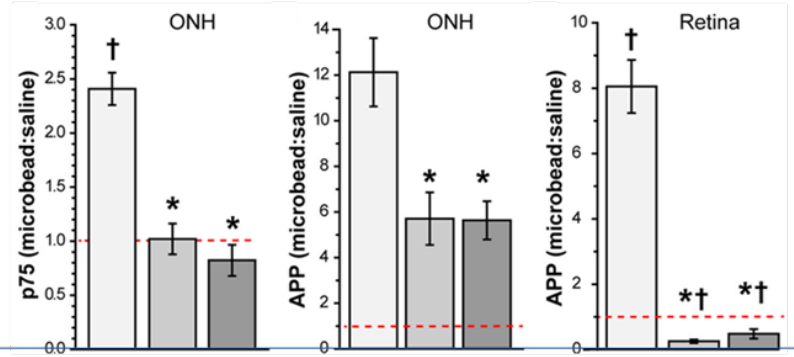
Decrease in several markers of neuroinflammation displayed in preclinical models

Decreased Activated Microglia



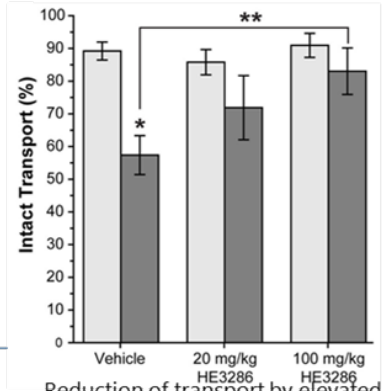
Iba1 is a microglia-specific marker.
† p<0.045 * p<0.037

Decreased Neurotrophic Receptor (p75) and Amyloid Precursor Protein (APP)



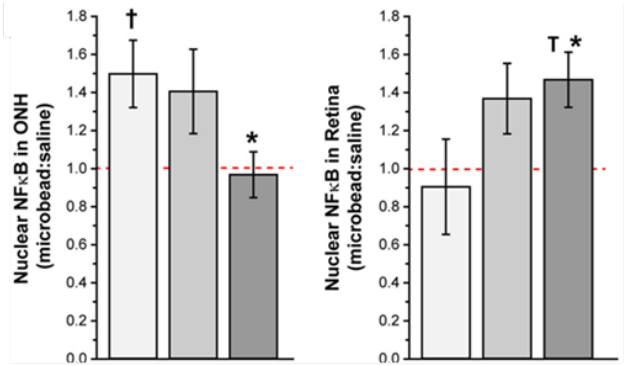
ONH: Optic nerve head. † p<0.047 * p<0.027

Rescued Axonal Transport

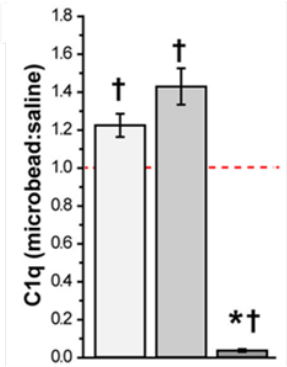


Reduction of transport by elevated intraocular pressure. * p<0.003 ** p<0.025

Decreased Inflammatory NFκB in Inflamed Optic Nerve Head; Increased Homeostatic NFκB in Retina



Decreased Inflammatory C1q



Complement component C1q involved with neuronal death. † p<0.037 * p<0.033

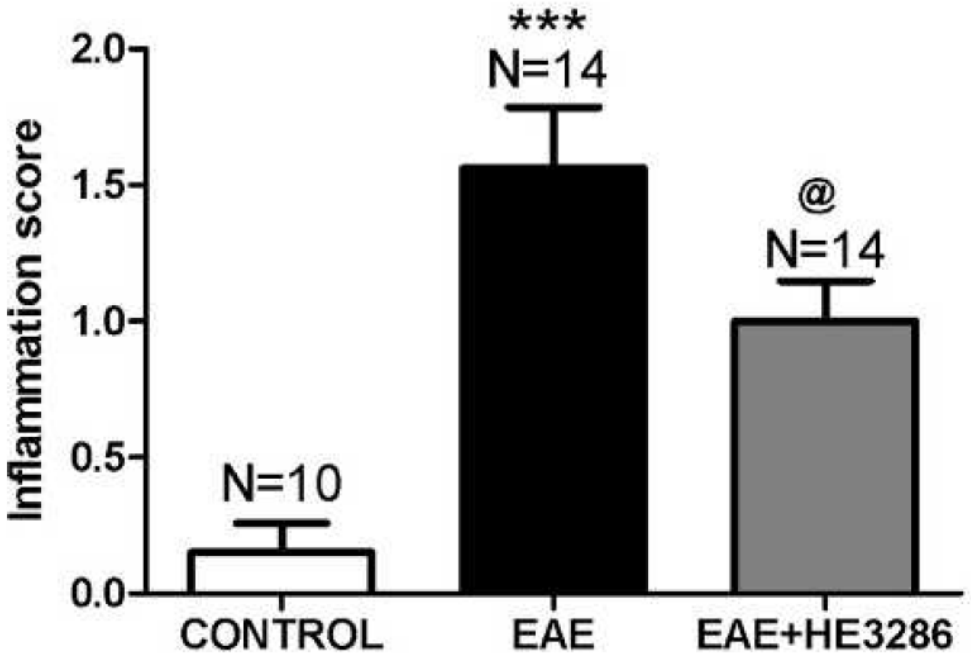
Source: Lambert 2017 *Front Neurosci 11 45*

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

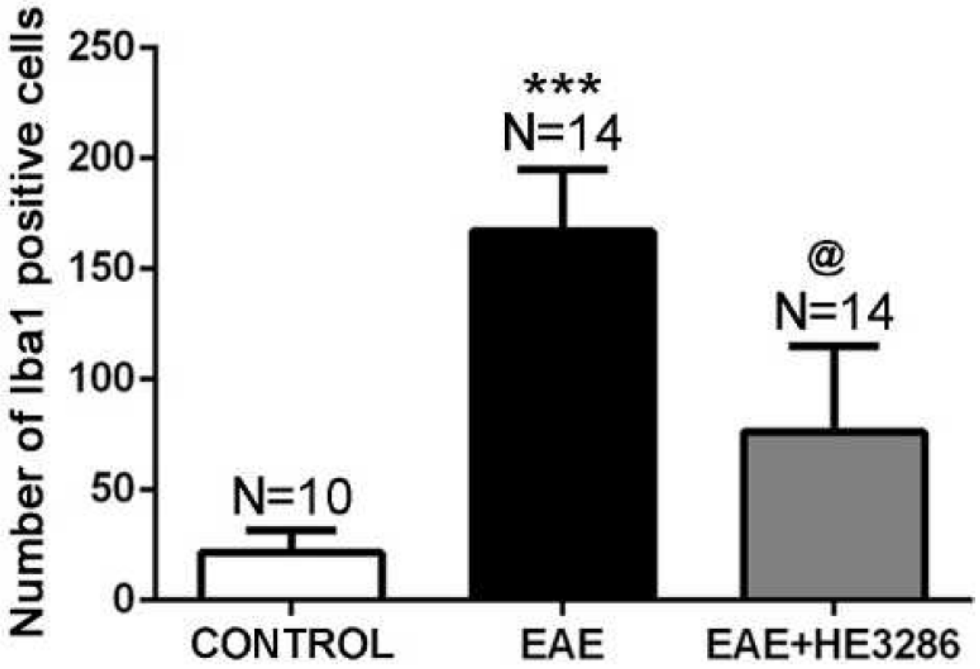
NE3107 Preserves Neuronal Function

Evidence of preserved ganglion cell function in Optic Neuritis in preclinical models

Decreases Optic Nerve Head Inflammation



Reduced Activated Microglia in Optic Nerves



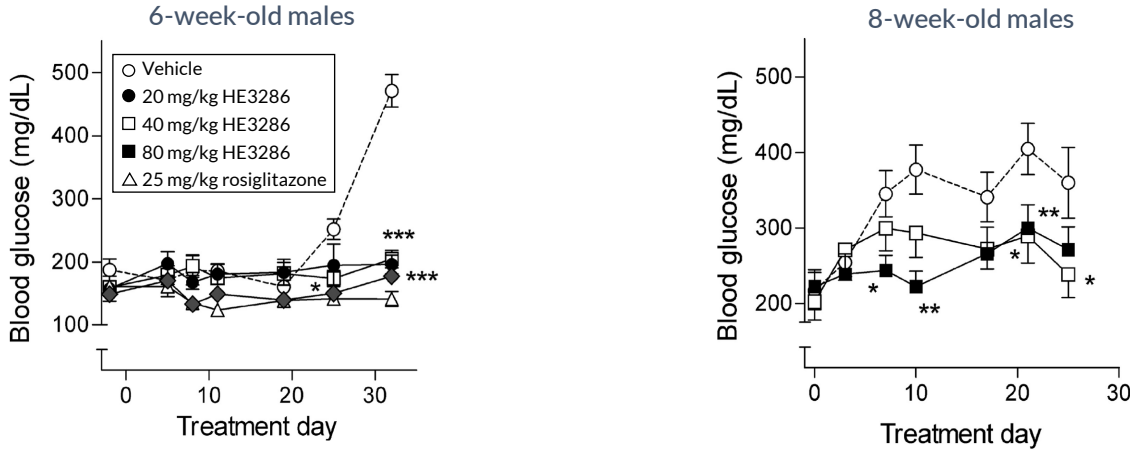
Kahn 2014 Invest Opthalmol Vis Sci 55 5744

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

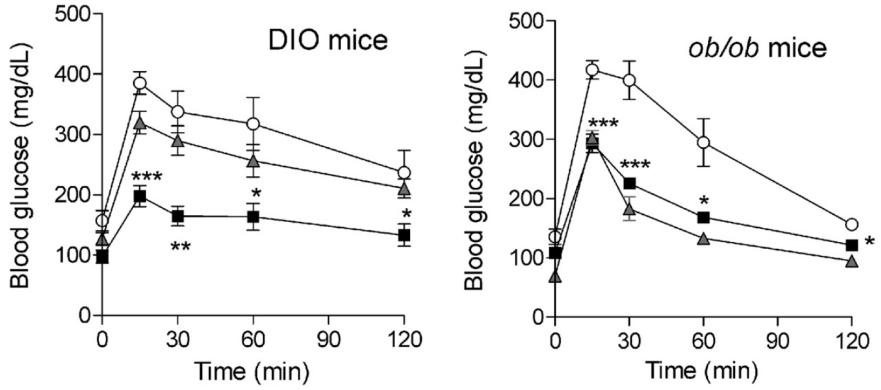
NE3107 Enhances Insulin Sensitization

Suppresses development of hyperglycemia in multiple preclinical animal models

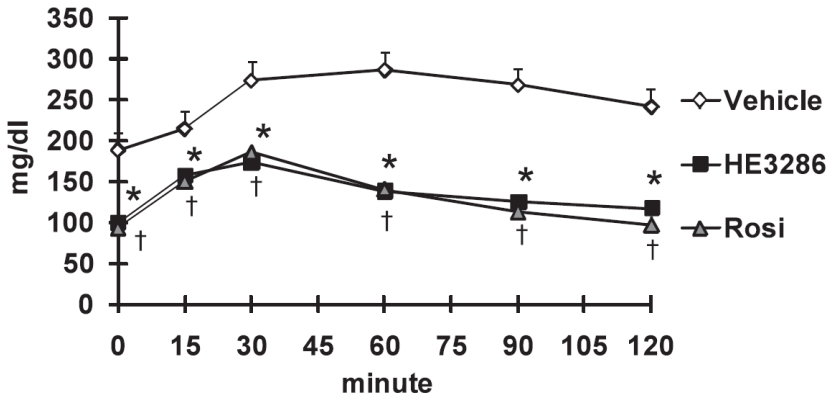
HE3286 Suppresses Development of Hyperglycemia in Diabetic db/db Mice



Decrease in Levels of Insulin and Glucose in Oral Tolerance Tests



Wang 2010 J Pharmacol Exp Ther 333 70



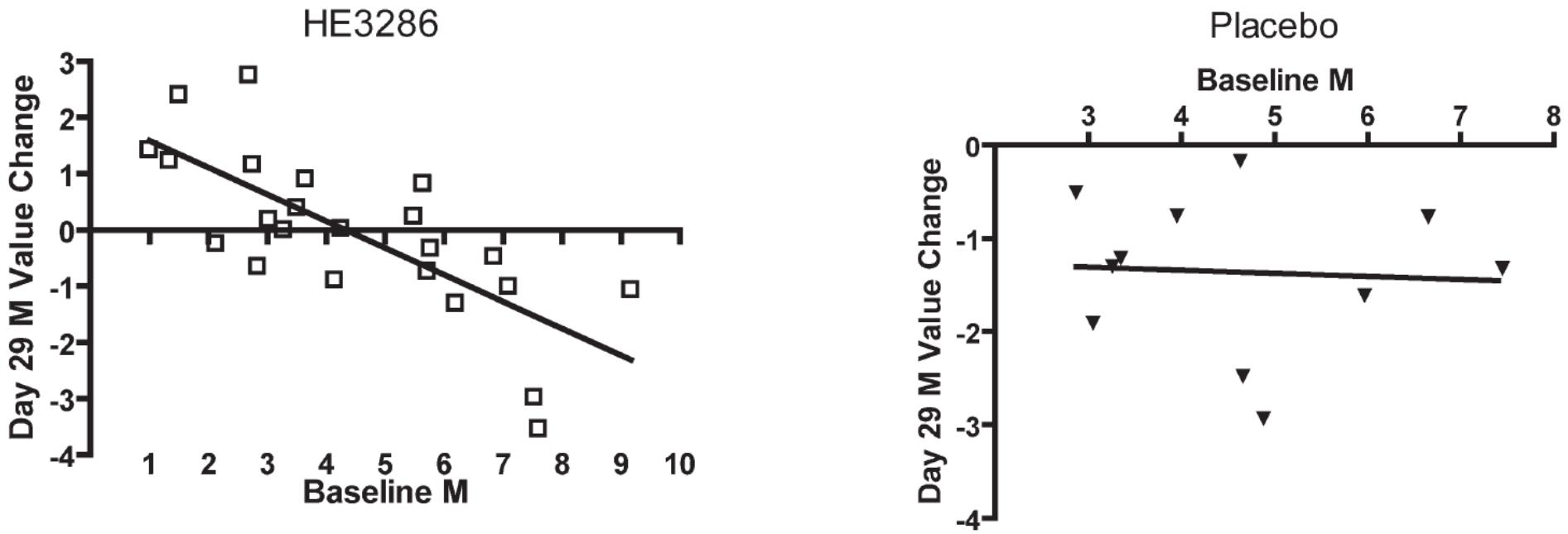
Lu 2010 Am J Physiol Endocrinol Metab 298 E1036

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

NE3107 Phase I Trial Results

Enhanced insulin sensitivity in a phase I clinical trial with impaired glucose tolerance patients

Improved Insulin-Dependent Glucose Disposal in 48 Obese, Inflamed patients



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

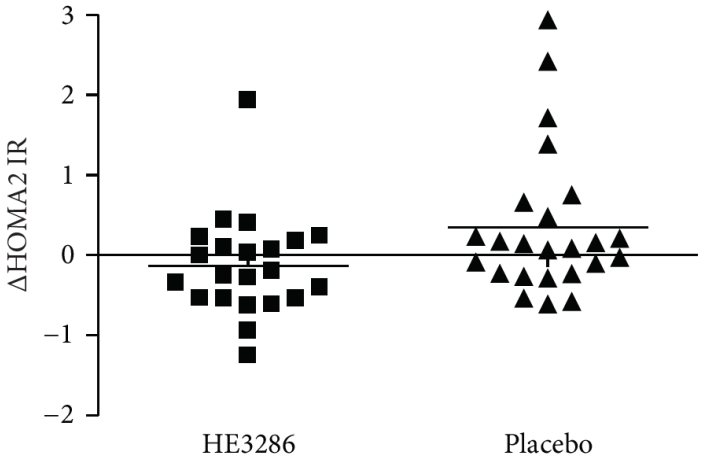
Reading 2013 Obesity 21 E343

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

NE3107 Phase 2 Trial Results

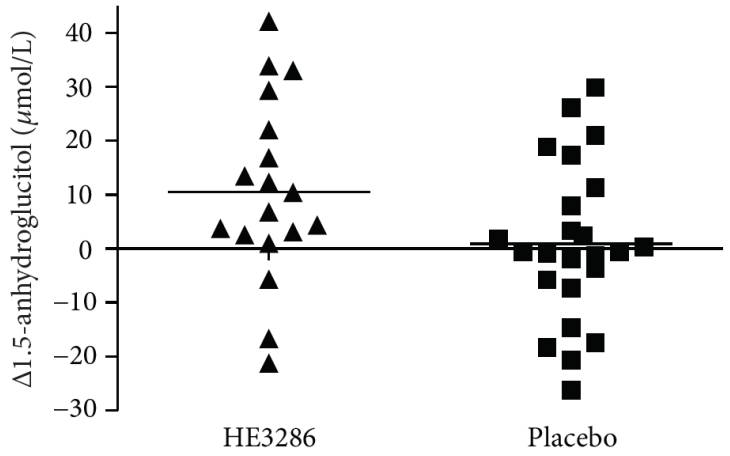
Enhanced insulin sensitivity reproduced in phase 2 trial with Type II Diabetes patients

Improvement in Homeostatic Model Assessment of Insulin



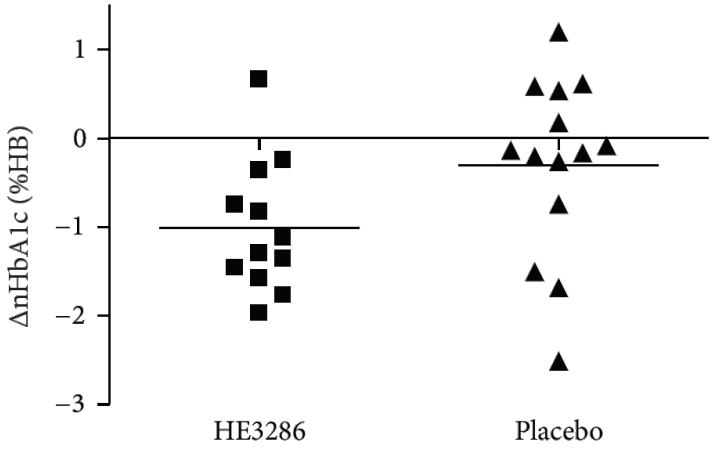
Cohort 1 baseline MCP-1 > 40 pmol/L individual subject changes in homeostatic model assessment of insulin resistance at Day 84

Decreased Postprandial Glucose



Pooled cohorts 1 and 2 changes at Day 84

Decreased HbA1c



Day 112 cohort 2 baseline BMI > 31 kg/m2

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

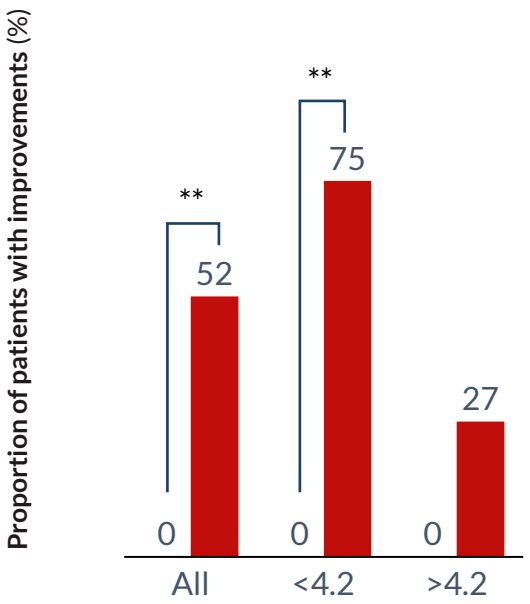
Note: NE3107 was originally known as HE3286 from Hollis Eden Pharmaceuticals

Increased Insulin Sensitization Effect on AD

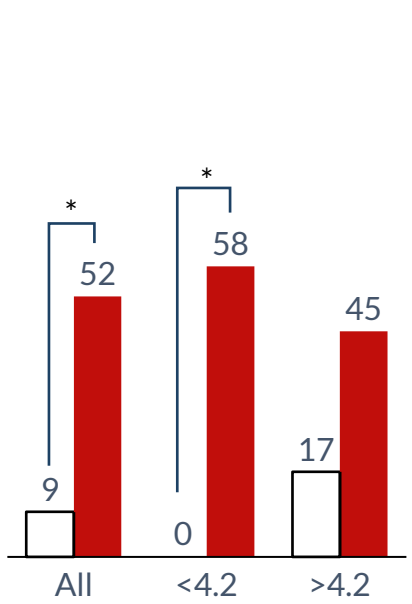
Insulin sensitizing improvement also brought improvements in AD indicators

In a Phase 2 study 48 obese patients with impaired glucose tolerance, NE3107 …

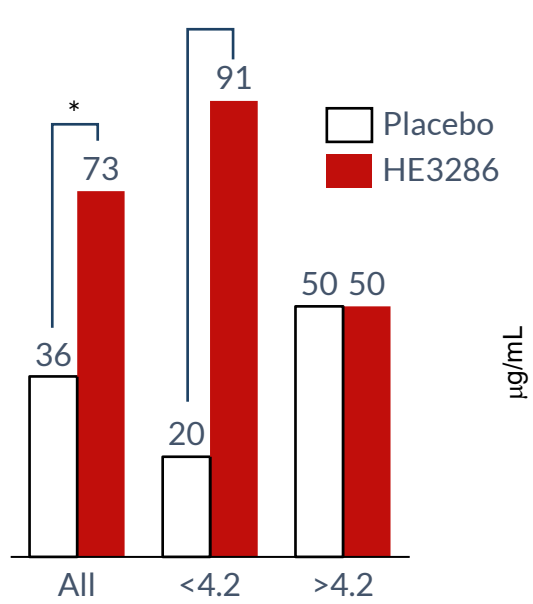
Increased Insulin Sensitivity



Increased HDL



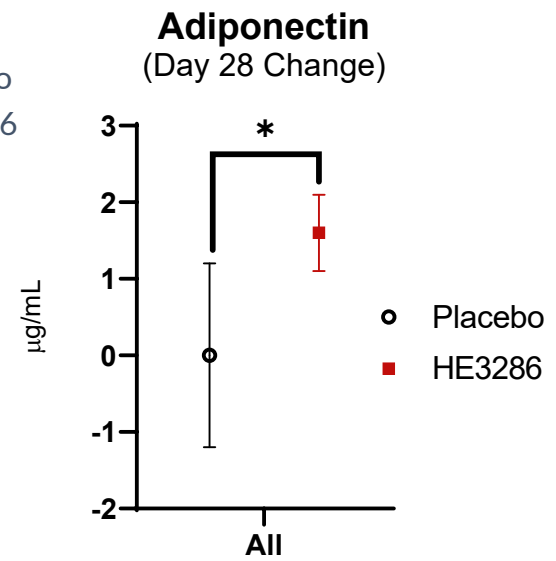
Decreased CRP



Placebo
HE3286

Baseline M***

Increased Adiponectin

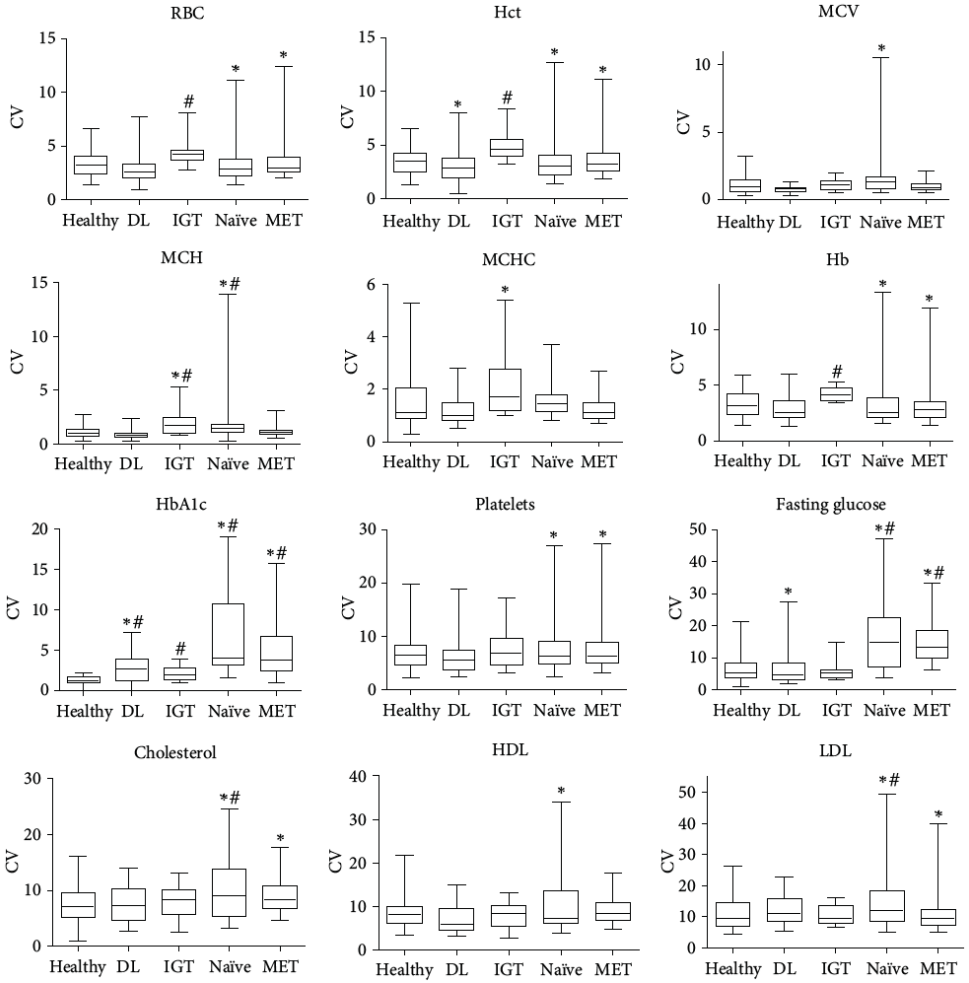


Placebo
HE3286

* 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

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 patients 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