Developing Transformative Therapies to Overcome Chronic Debilitating Diseases

Corporate Presentation • June 2022

Forward-looking statements

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Highlights

NE3107 is the only inhibitor , to our knowledge, that is being studied for of inflammation and insulin resistance in Phase 3 trials for Alzheimer's Disease (AD)	• NE3107 is believed to potentially reduce neuroinflammation and insulin resistance in randomized, double-blinded, placebo-controlled Phase 2 study; potential to address the two factors described as potential key drivers of cognitive decline in AD • Phase 3 patient enrollment underway; ramping to 45 centers; data readout anticipated mid-2023 • $10+ billion annual peak sales potential
NE3107 is also being studied developed for Parkinson's disease (PD) based on similar mechanism of action	• NE3107 equally pro-motoric as levodopa, reduced severity of levodopa induced dyskinesia (LID), and preserved twice as many neurons as untreated in preclinical studies • Phase 2 trial start 1Q22; data readout anticipated mid-2022 • $3+ billion annual peak sales potential
BIV201 is the only drug to our knowledge, currently in development for refractory ascites, a condition with 50% mortality rate	• Formulation of terlipressin for continuous infusion; Method of use patent for treatment of ascites; potential to become first therapy since there are no approved drugs in the US; Orphan and Fast Track designations received • Decades of data gathered from European/Asia; No drug-related SAEs in trials thus far • Phase 2b underway with data readout anticipated early-2023 • $1+ billion global peak sales potential

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.

Portfolio Revenue Potential

		Company Estimates[2]		
	U.S. Intellectual Property	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

Roles of Inflammation and Insulin Resistance Well Established in AD Progression

At the Alzheimer's Association International Conference 2021: Hundreds of presentations on neuroinflammation as a driver of AD; Dozens of presentations on inflammation-driven brain insulin resistance

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

There are 7 current biotech/pharma anti-neuroinflammation trials

AD has been termed "Type 3 Diabetes" and "cognitive-metabolic syndrome"*

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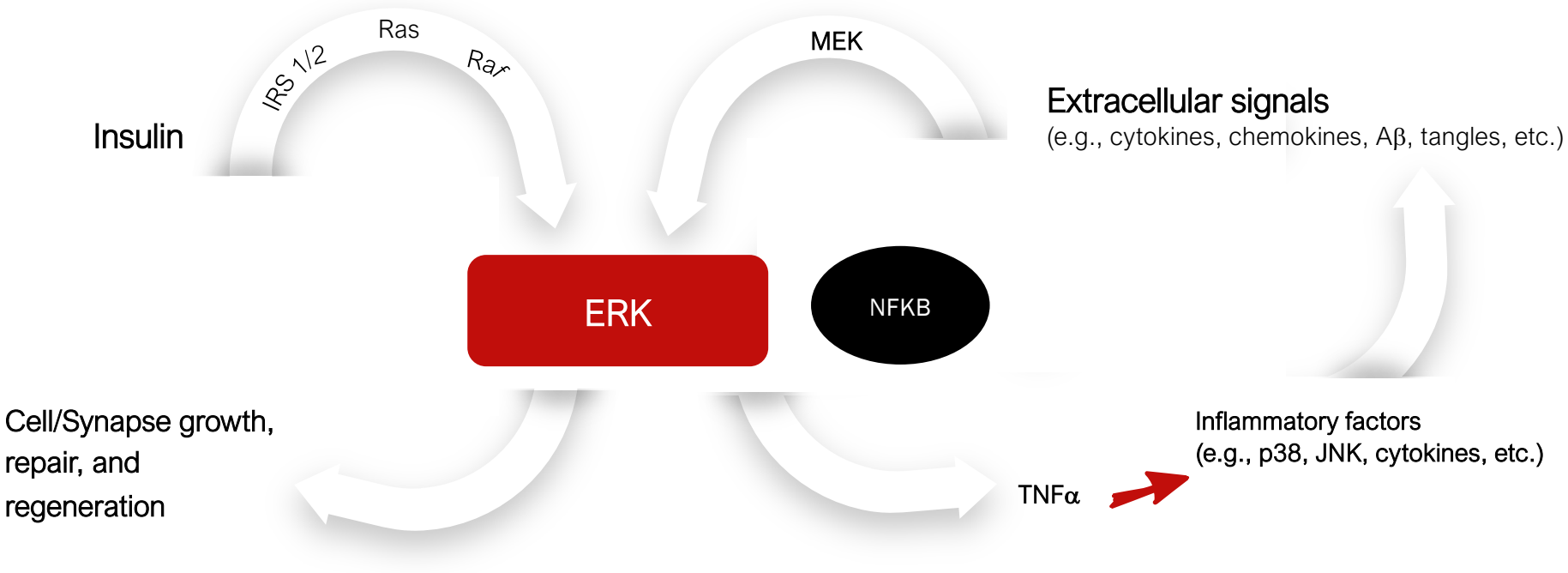
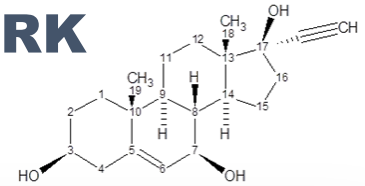
* de la Monte, S.M. and J.R. Wands, Alzheimer's disease is type 3 diabetes-evidence reviewed. J Diabetes Sci Technol, 2008. 2(6): p. 1101-13

Extracellular Signal-Regulated Kinase (ERK)

Critical player for both homeostasis and inflammation (and resulting inflammation-induced insulin resistance)

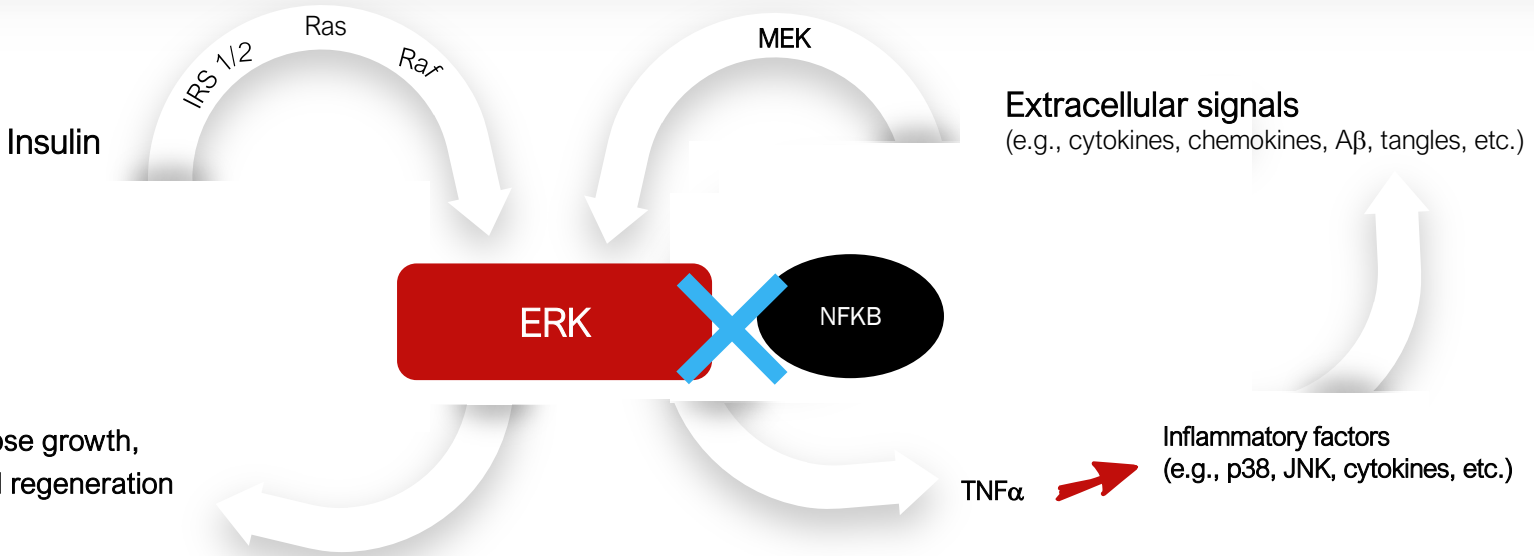


NE3107 Blocks Inflammatory, but not Homeostatic ERK Signaling



First-in-class molecule with desirable characteristics

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



Prior NE3107 Clinical Studies

Phase 1[1]

Obese, impaired glucose tolerant healthy volunteers

NE3107:

- Improved insulin-stimulated glucose disposal, assessed by hyperinsulinemic, euglycemic glucose clamp procedures
- Decreased C-reactive protein (CRP[2]) and increased HDL and adiponectin (both associated with benefit in AD)
- Showed no differences in AEs compared to placebo

Phase 2[3]

Obese and inflamed type 2 diabetes subjects

NE3107:

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

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

Ongoing Pivotal Phase 3 for Alzheimer's Disease

- >4,000 scientific publications support the role of inflammation and insulin resistance in AD[1]
- >80% of AD patients also have (or developing) Type 2 Diabetes[2]
- Data from preclinical and clinical studies show that NE3107:
  - Reduces inflammation and enhances insulin sensitivity, which may potentially reduce AD pathology
  - Blocks EPIC, upstream from amyloid beta and phospho-tau, further reducing inflammation
  - Demonstrates potential neuroprotective properties in models of PD, optic neuritis and glaucoma
  - NE3107 has been well-tolerated in clinical studies to date
  - Reduces systems deregulation (enhances homeostasis)
  - Crosses blood brain barrier (BBB)
- Phase 3 to review whether NE3107's impact on inflammation and insulin resistance can help reduce the rate of cognitive decline
  - Decrease inflammation ➔ increase insulin sensitivity ➔ decrease in rate of cognitive decline
  - Potential for enhancement of cognition due to insulin's role in the brain

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1. Pubmed search for "Alzherimer's disease" and "inflammation" or "insulin resistance"
2. Janson 2004 *Diabetes* 53 474

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 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

Investigator-Initiated 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

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

Proceeding to Clinical Development for Parkinson's Disease

NE3107 reduced inflammation and enhanced insulin sensitivity in animal models[1], both of which have been shown to reduce PD pathology

- Reduction of inflammation itself may be beneficial since both inflammation and low dopamine environment are required for Parkinsonism
- NE3107 was promotoric, reduced LID and preserved dopaminergic neurons in marmosets

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 test a clear and plausible hypothesis

Decreased inflammation > increased insulin sensitivity > disease amelioration

- Promotoric activity
- Reduction in LID

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1. Beck *Mov Disord.* 2018 May ; 33(5): 805–814; Lee *Int. J. Mol. Sci.* 2021, 22, 2098; Pang *Neuroscience.* 2016 March 24; 318: 157–165; Pereira *Front. Pharmacol.* 11:617085; Yan *Front. Aging Neurosci.* 12:625166.; Novak PLoS ONE 14(4): e0214364. https://doi.org/10.1371/journal.pone.0214364;

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

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* Motor Disease Society Unified Parkinson's Disease Rating

BIV201 Disease Target: Refractory Ascites

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

Paracentesis:

Withdrawal of 5–10L of ascites fluid (on average) from abdomen using a large bore needle

Provides a few days of symptomatic relief

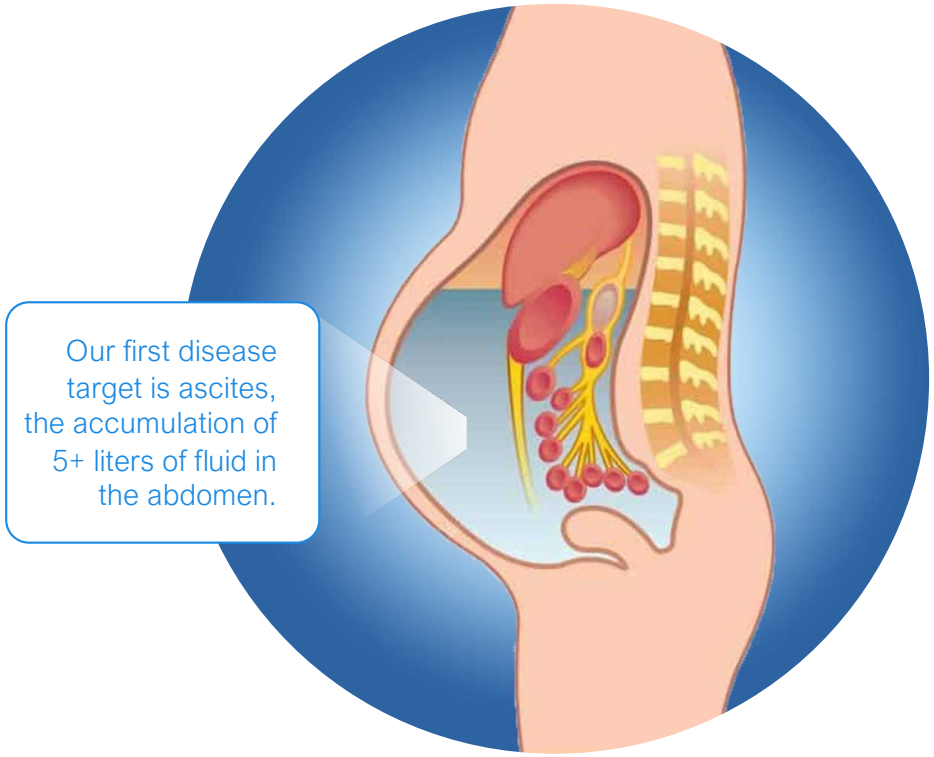
The kidneys are “burning out” by retaining massive quantities of salt and water

Patients suffer frequent life-threatening complications

No remaining options except for TIPS[1] surgery or liver transplant

Estimated $670 million addressable US market with 20,000[2] targeted patients

No drugs ever approved by FDA to treat ascites



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

Prefilled Syringe with Patent-pending Liquid Formulation

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

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

Needle or Connector

50 mL bag of saline for insertion into pump

Portable pump
Carried in small satchel

*In clinical trials; not approved by FDA

BIV201 Development Plans

Ascites

- 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

HRS-AKI

- Planning a possible registrational Phase 3 trial in HRS-AKI*
  - For better outcomes because earlier treatment is possible than for HRS Type 1
  - For 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 PI's, same sites, and same drug product as our current ascites study
  - Orphan drug designation for HRS (in addition to ascites)

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- Hepatorenal syndrome – acute kidney injury

1 News Detail | Mallinckrodt Pharmaceuticals June 13, 2022

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.

Jonathan Adams, Liver Cirrhosis Program
30+ years in biopharma
Founded LAT Pharma (BioVie predecessor)
Mission Pharmacal
Corbett and other healthcare agencies
Searle Pharmaceuticals

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

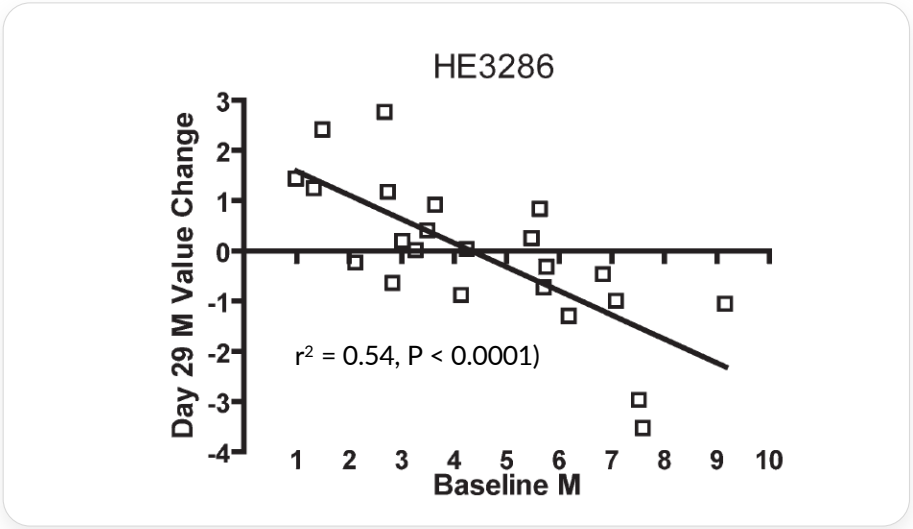
Clarence Ahlem , Neurodegenerative Disease Program
35+ years in biopharma
Vice President, Product Development Harbor Therapeutics
Director, Product Development, Hollis-Eden Pharmaceuticals
US San Diego

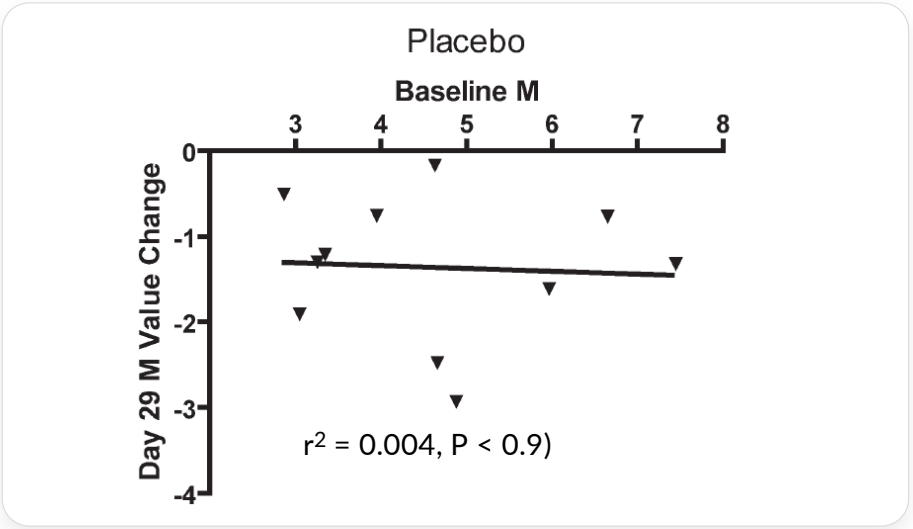
J. Wendy Kim, Chief Financial Officer
35 years in finance/ accounting
As CFO managed corporate finance and operations groups
Closed M&A transactions and secured financings
Combined 22 years at KPMG and BDO LLP

Thank You

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

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





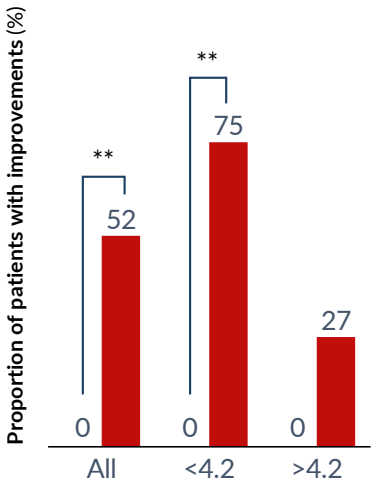
Day 29 Glucose disposal rate (M, measured in mg/kg/min in glucose clamp studies) value change as a function of baseline M

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

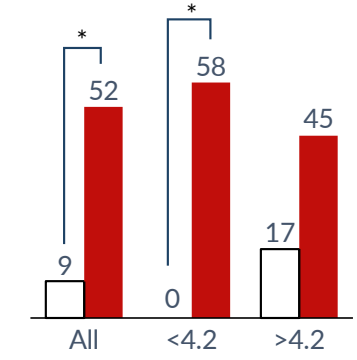
Insulin sensitizing improvement also brought improvements in AD indicators

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

Increased insulin sensitivity …

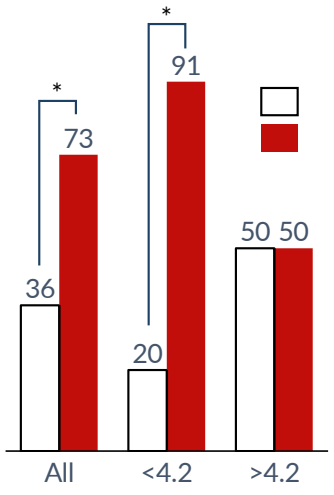


… increased HDL …

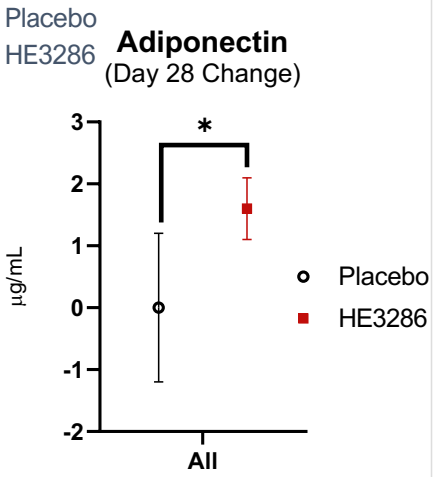


Baseline M***

… decreased CRP …



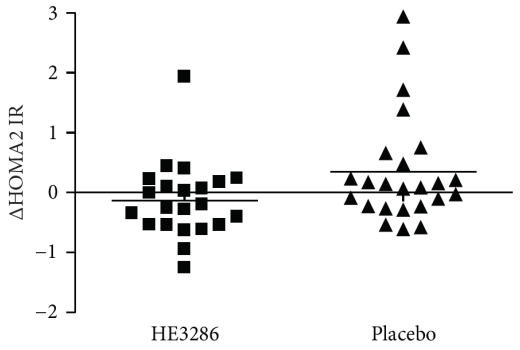
… and increased adiponectin



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

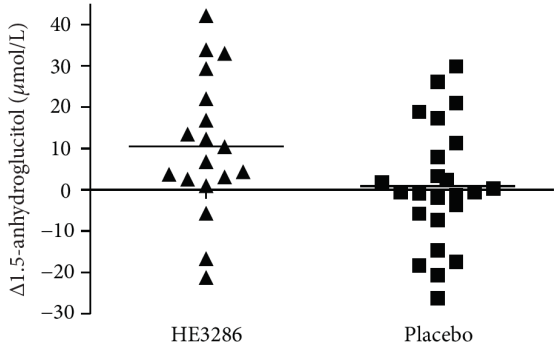
NE3107's ability to enhance insulin sensitivity reproduced in humans in Phase 2 trial with T2D patients

Improved the homeostatic model assessment of insulin resistance …



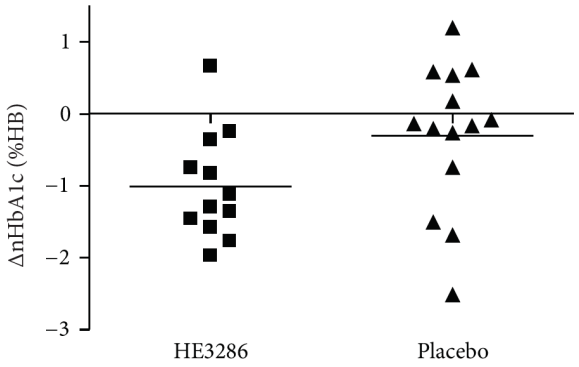
Cohort 1 baseline MCP-1 > 40 pmol/L individual subject changes in homeostatic model assessment of insulin resistance at Day 84

… decreased postprandial glucose …



Pooled cohorts 1 and 2 changes at Day 84

… decreased HbA1c …



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

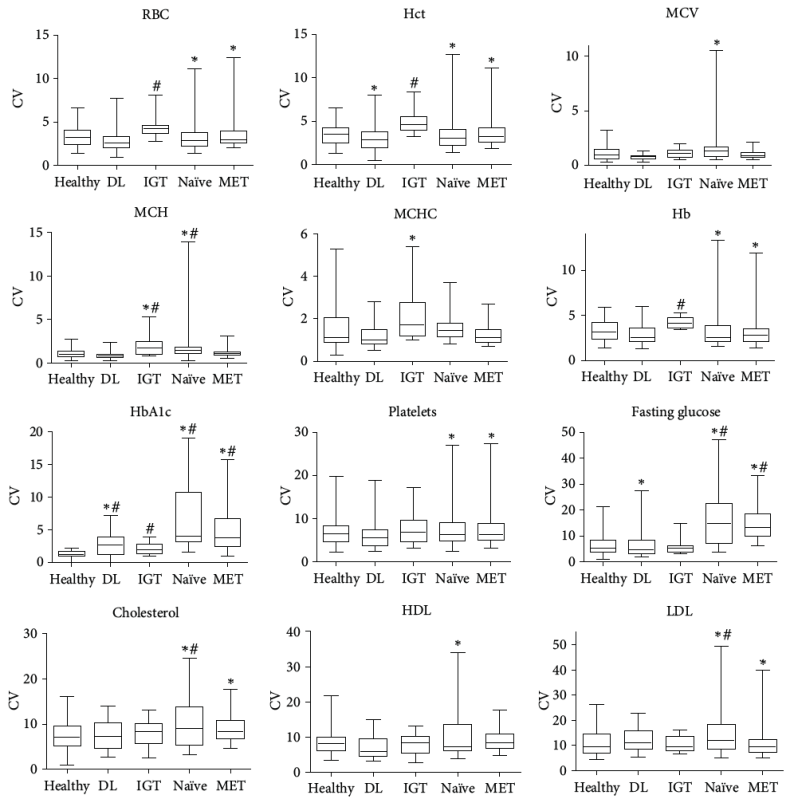
… decreased insulin resistance in inflamed T2D patients

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

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

Inflammation drives systems dysregulation

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



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

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

#Statistically Significant Welch ANOVA). ∗Statistically significant
2-sided FF test.

Reading 2013 Mediators Inflamm 814989

NE3107 decreased systems dysregulation in a Phase 2 study in advanced T2D patients

Heteroscedasticity* investigated by analyzing data distributions for normality (Shapiro-Wilks *W* test)

- Deviations from normal distribution represents dysregulation

Placebo patients had significantly abnormal data distributions after 84 days of treatment whereas NE3107 subjects did not

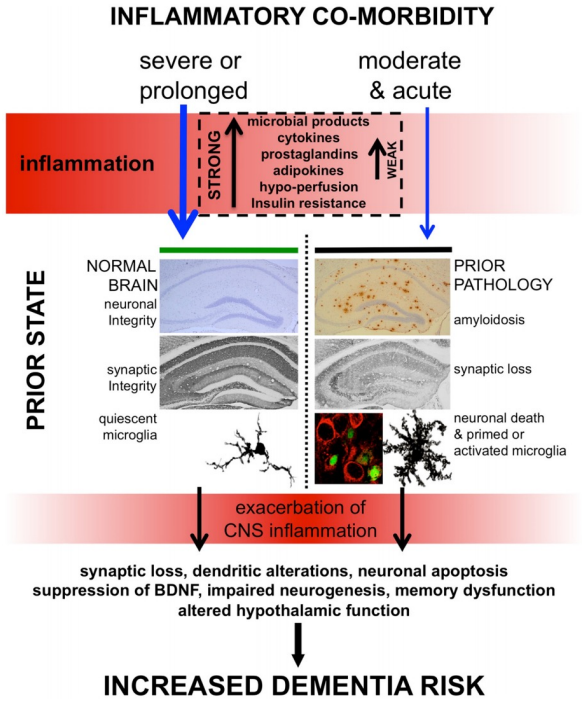
- Placebo patients had many parameters that significantly deviated from normal distribution (i.e., dysregulated)
- NE3107 treatment reduced systems dysregulation

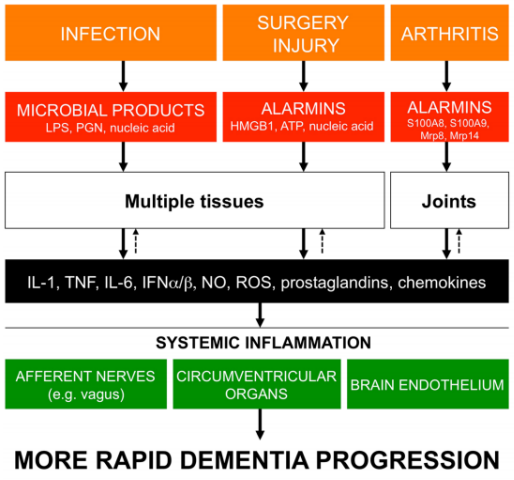
Group	Cohort 1	MCP-1 > 40	Cohort 2	BMI > 31	
HE3286	n=44	n=22		n=35	n=12
Placebo	n=51	n=25		n=34	n=15

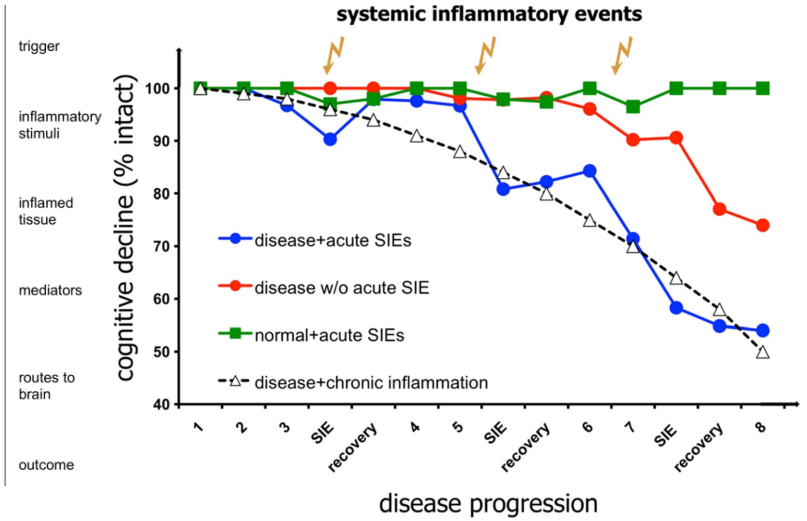
Group	Day	Parameter	HE3286 *W* test *P*	Placebo *W* test *P*
Cohort 1	84	ΔInsulin[d]	>0.1	**<0.0001**
		ΔC-peptide	>0.1	**<0.0001**
		ΔFasting glucose	>0.1	**0.02**
		ΔHOMA2 %B	>0.1	**<0.0001**
		ΔHOMA2 IR	>0.1	**0.002**
		Δleptin	>0.1	**0.005**
Cohort 1 MCP-1 > 40[b]	84	ΔHbA1c	>0.1	**0.006**
		ΔFasting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

* Heteroscedasticity describes differences in variances between groups. Reading 2013 Mediators Inflamm 814989. Note: NE3107 was originally known as HE3286 from Hollis Eden Pharmaceuticals

Systemic inflammation and co-morbidity as drivers of cognitive decline







SIEs: serious inflammatory events

Cunningham 2015 Alzheimers Res Ther 7 33

Targeting Neuroinflammation in Alzheimer's Disease

- Extensive study data supports reducing neuroinflammation as the key to slowing/reversing cognitive decline
- Other companies focused on this target:

Company	Exchange:Ticker	Drug Candidate	Clinical Status[1]	Mechanism of Action	Market Cap ($mm)[2]
BioVie Inc.	Nasdaq: BIVI	NE3107	Phase 3	Inhibits ERK/NFkB neuroinflammation Inhibits insulin resistance	$46
Cortexyme, Inc.	Nasdaq: CRTX	Atuzaginstat	Phase 2/3	Inhibits P. gingavalis toxin	$119
Cassava Sciences	Nasdaq: SAVA	Simufilam	Phase 3	Restores filamin A	$1,158
Athira Pharma	Nasdaq: ATHA	ATH-1017	Phase 3	Activates neuron regenration	$347
Vaccinex	Nasdaq: VCNX	VX15	Phase 1/2	Ab to SEMA4D neuroinflammation	$55
Annovis	NYSE: ANVS	ANVS-401	Phase 3	Decreases APP & Tau	$88
InmuneBio	Nasdaq: INMB	XPRO1595	Phase 1b	dN TNF neuroinflammation	$125
Denali Therapeutics	Nasdaq: DNLI	DNL788	preclinical	RIPK1 inhibitor, TNFR pathway	$3,087

NE3107 anticipated to have significant advantages over competition

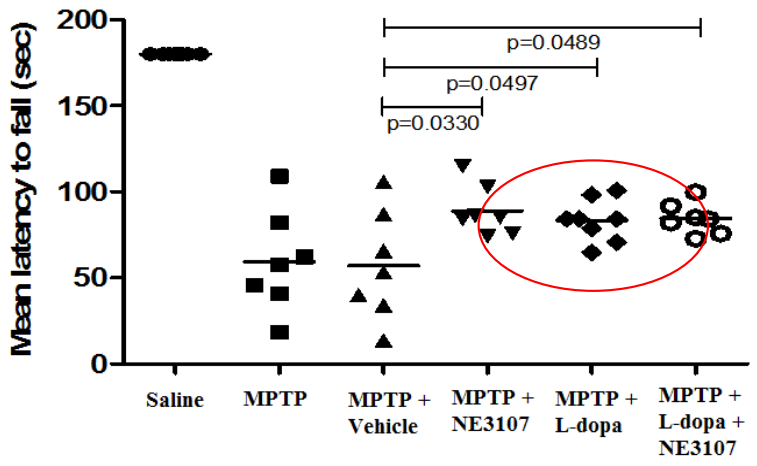
- Phase 3 clinical studies initiated with data readout targeted for mid 2023
- Potentially inhibits both overarching mechanisms of AD pathology: inflammation and insulin resistance
- Potentially inhibits proinflammatory pathways without impacting homeostasis

bioVie

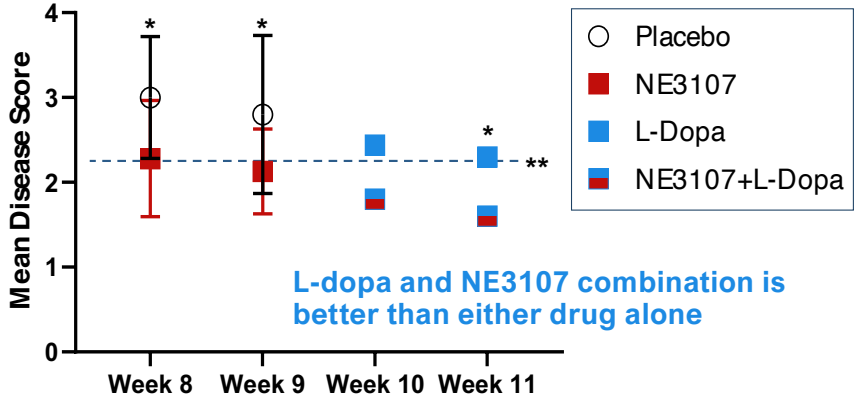
[1] As reported in Clinicaltrials.gov on 06/07/22
[2] Closing value on 06/07/22

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

MPTP Mouse



MPTP Marmoset treated at Week 8

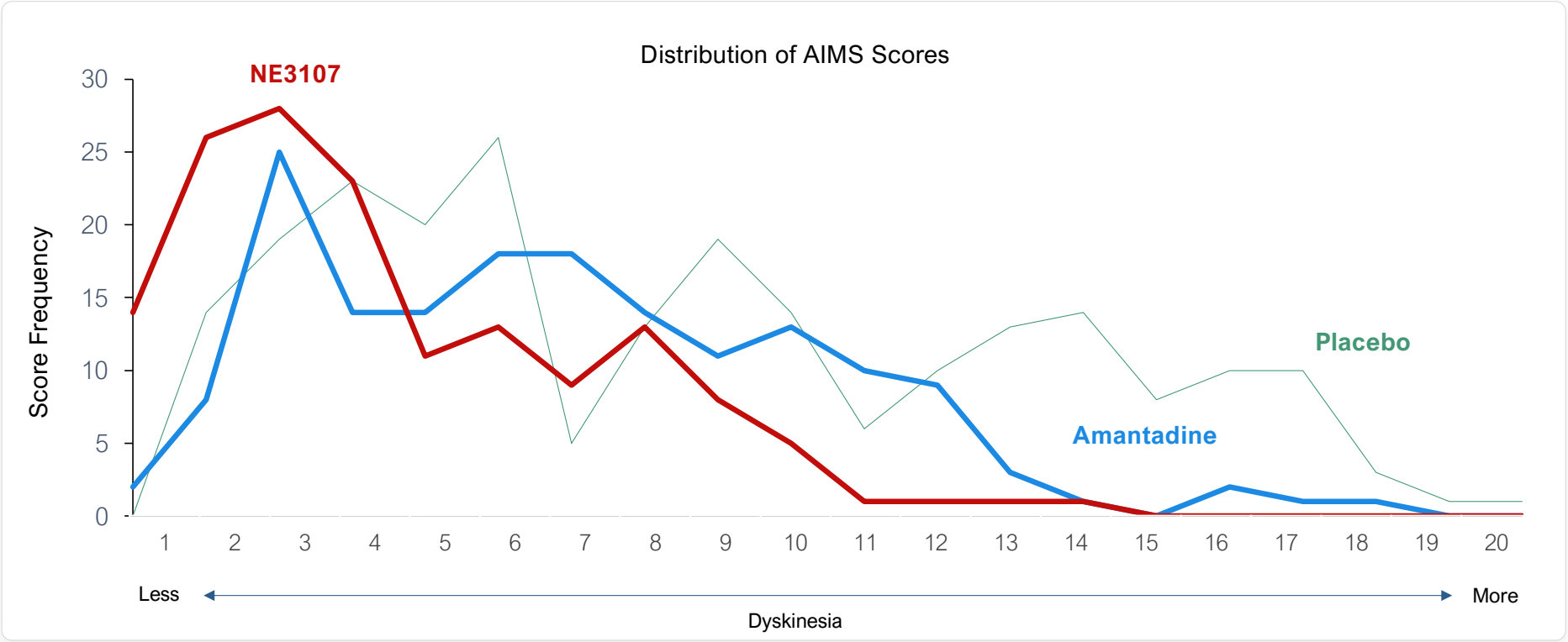


*p<0.001 compared to other treatment arm in time period
** L-Dopa at week 11 not statistically different from NE3107 at weeks 8 and 9

NE3107's promotoric effects observed within 4 days of treatment

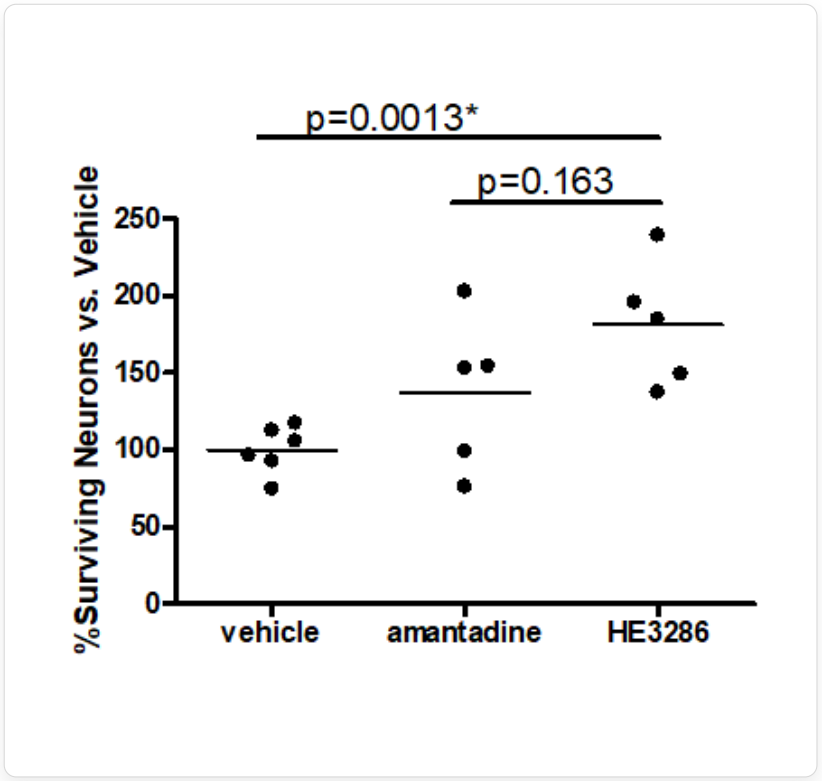
MPTP: 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine, a toxin used to mimic many aspects of PD

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

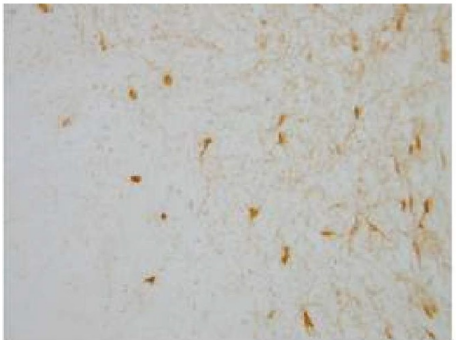


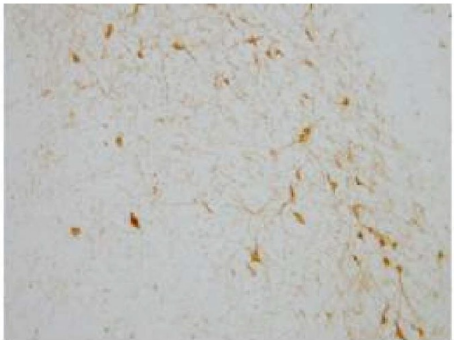
Abnormal Involuntary Movement Scale (AIMS)

NE3107 preserved TH+ neurons in MPTP marmosets

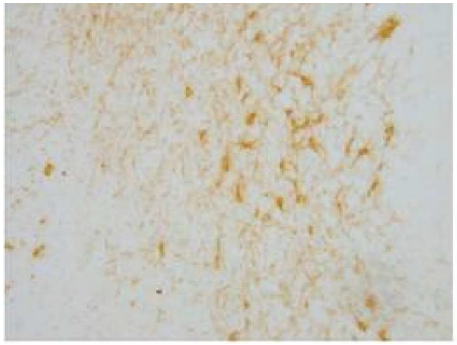


Control moneys (M09100 and M11008)

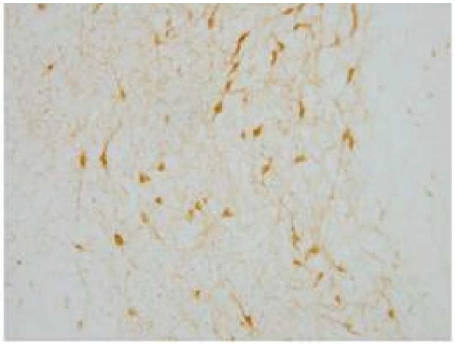




HE3286 (M11007)



Amantadine (M10084)

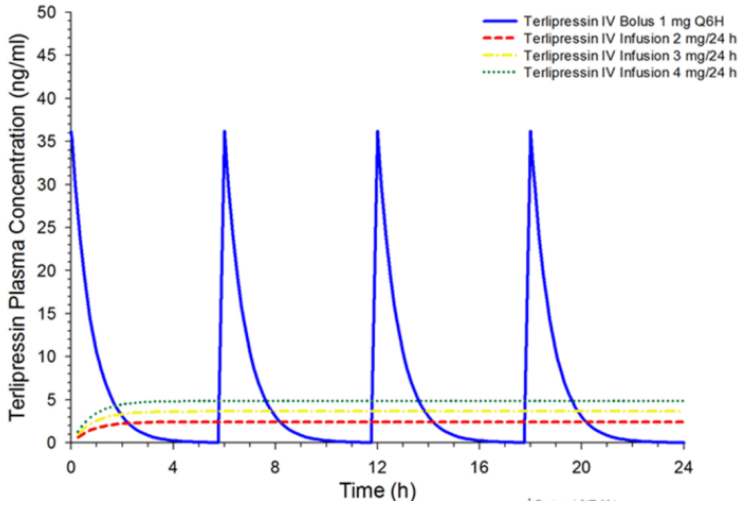


Note: NE3107 was originally known as HE3286 from Hollis Eden Pharmaceuticals

BioVie Phase 2a trial results: BIV201 Pharmacokinetics

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

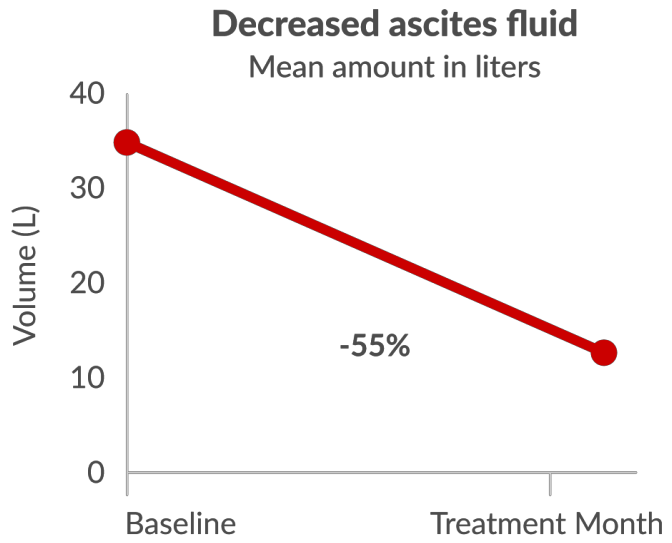
Comparison of the PK Profile of Terlipressin Administered by Continuous Infusion or Intermittent IV Boluses.

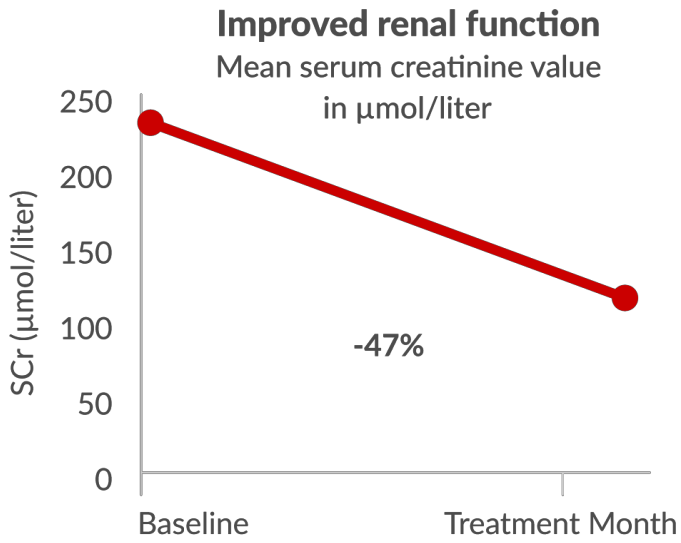


Simulated terlipressin plasma concentrations following IV bolus of 1 mg every 6 hours 1 [Lucassin, AusPAR-cer 2011] and IV continuous infusions of 2 mg, 3 mg, or 4 mg per day

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

Continuous Infusion Terlipressin in 6 Refractory Ascites/HRS Patients*





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

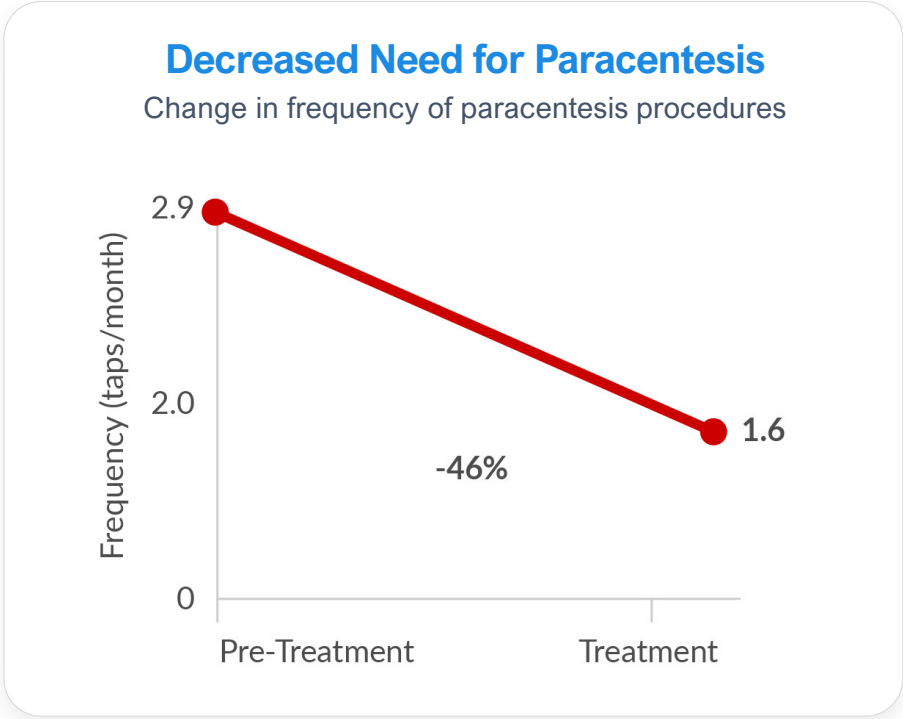
Continuous Infusion Terlipressin in 19 Refractory Ascites/HRS Patients

Pre-therapy:

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

Results:

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



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