Unlocking the science of longevity to develop transformative therapies

Corporate Presentation • December 2023

Forward-looking statements

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Overview

We believe that TNFα-mediated inflammation

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

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

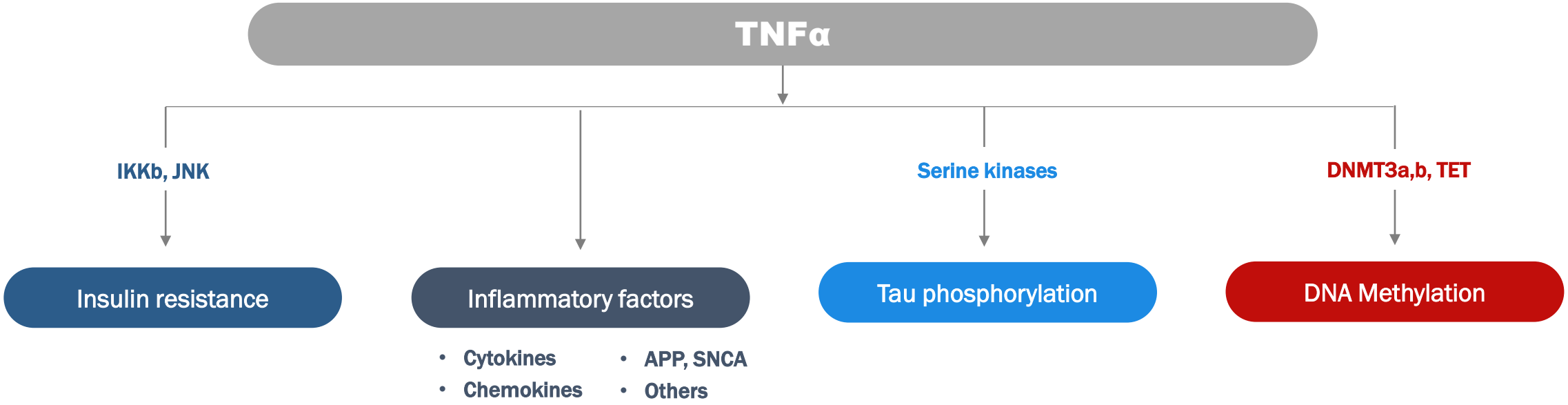
- Reduced inflammation and the associated insulin resistance
- Improved cognition and function, lowered amyloid β and p-tau levels, and improved brain imaging scans in Alzheimer’s Disease (AD)
- Improved motor control and “morning on” symptoms in Parkinson’s disease (PD)
- Lowered DNA methylation levels

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

- Provides epigenetic basis to explain improvements observed in AD and PD trials

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

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



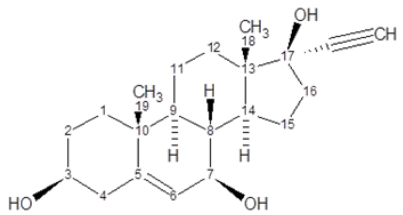
Diabetes
Metabolic disorders
Parkinson's

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

Alzheimer's

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

NE3107's mechanism of action

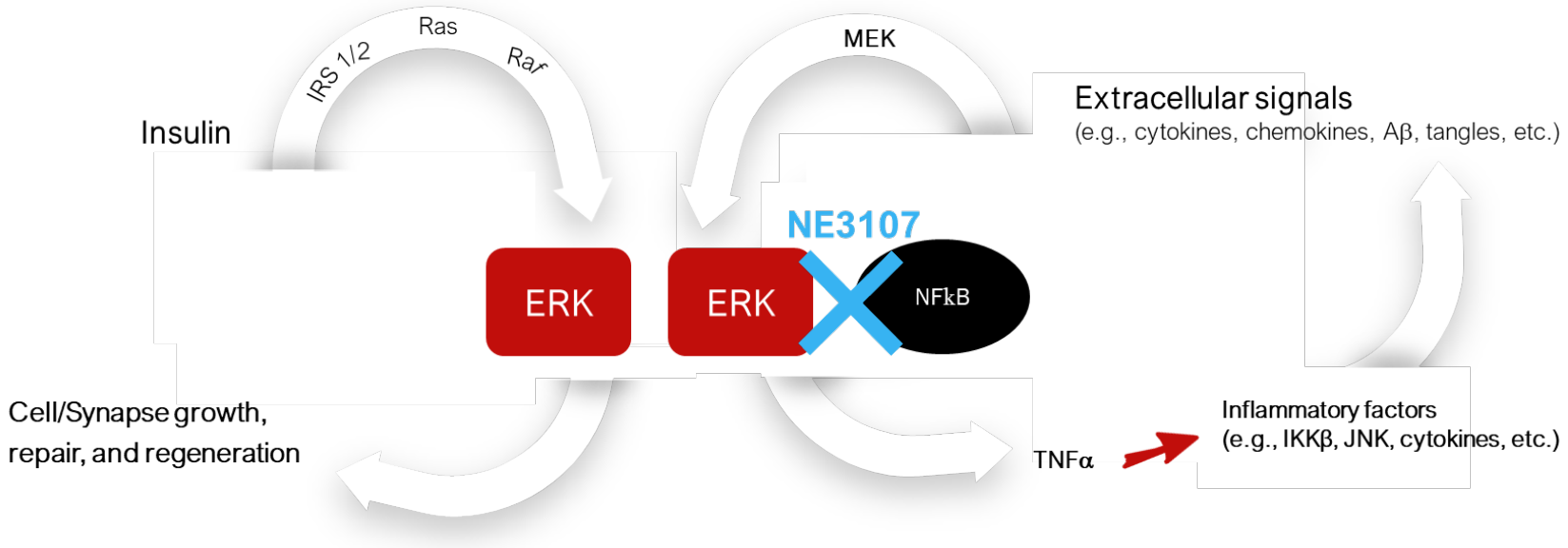


First-in-class molecule with desirable characteristics

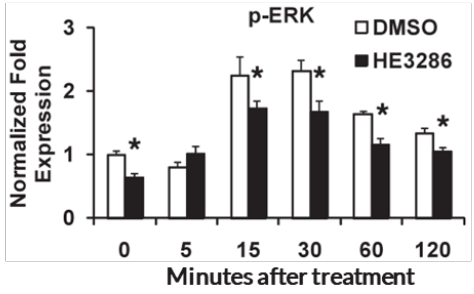
Small molecule; orally bioavailable

Crosses blood-brain barrier, thus CNS and peripheral applications

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

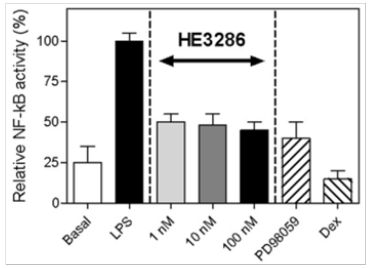


NE3107 Reduces ERK Activation



Lu 2010 Am J Physiol Endocrinol Metab 298 E1036

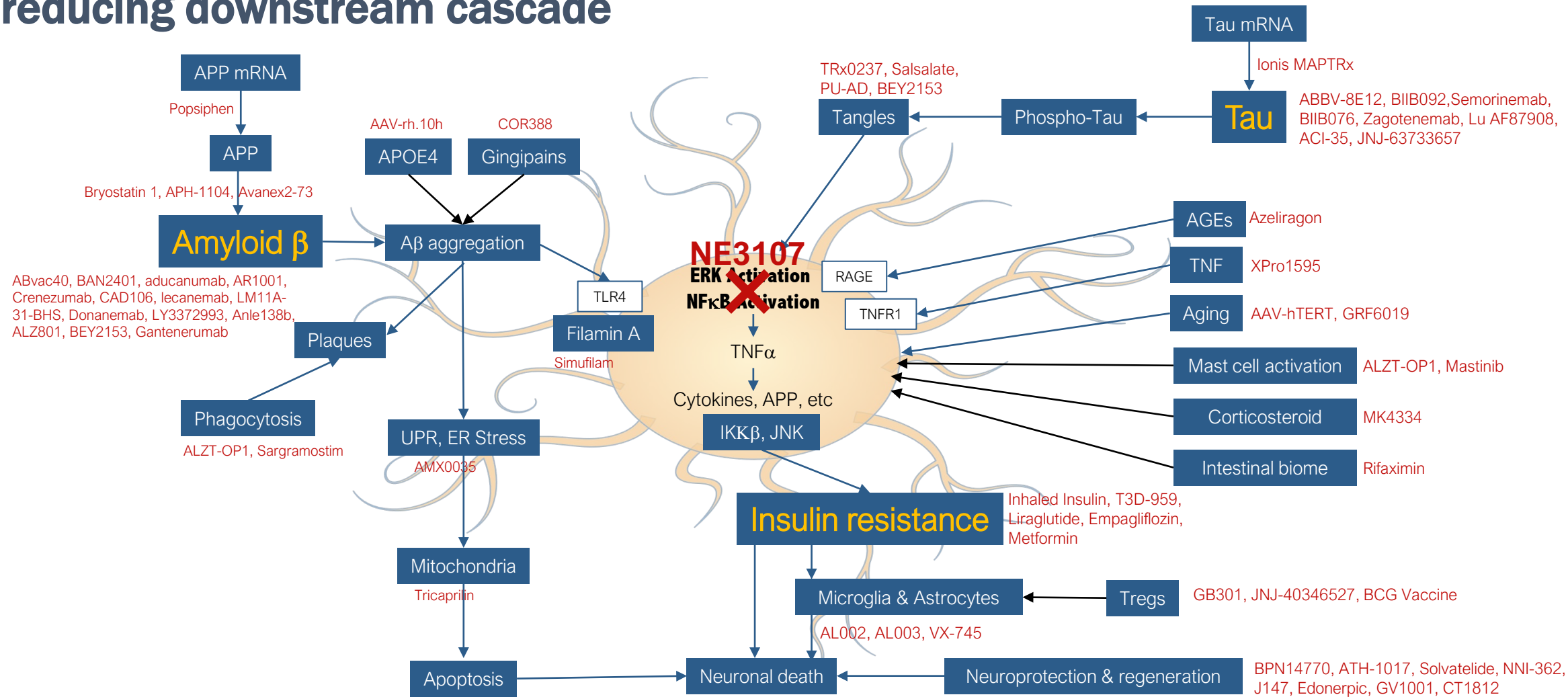
NE3107 Reduces NFkB Activity



Wang 2010 J Pharmacol Exp Ther 333 70

NE3107
in Alzheimer's Disease

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



NM101 Phase 3 trial in Mild to Moderate Alzheimer's

A Phase 3, Double-blind, Randomized, Placebo-controlled, Parallel Group, Multicenter Study of NE3107. Enrolled 439 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; 1:1 randomization; 80% power
- Diagnosed with probable 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 Dementia Rating-Sum of Boxes (CDR-SB) comparing the NE3107 group to the placebo group
  - 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
- **Secondary endpoints**
  - ADCS-ADL (functional), Alzheimer's Disease Cooperative Study-Clinical Global Impression of Change (ADCS-CGIC), 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 active and placebo subjects
  - Target engagement assessed in a small subset of active and placebo subjects using PET to quantify cortical glucose utilization

Trial Summary

- NE3107 appears to be biologically active
- Cognitive, functional, biomarker efficacy signal suggest that NE3107:
  - Has a treatment advantage equal to or greater than results reported from clinical trials from approved monoclonal antibody treatments;
  - Associated with a benign safety profile
- Unanticipated exclusion of 258 patients from 15 sites due to deviations led to study being underpowered. Adaptive feature of trial allows the Company to continue enrolling patients to reach statistical significance

Safety Profile

	NE3107	Placebo	Total
COVID-19	9.5%	17.6%	13.2%
Urinary tract infection	7.1%	8.8%	7.9%
Blood thyroid stimulating hormone increased	7.1%	2.9%	5.3%
Fall	2.4%	8.8%	5.3%
Headache	9.5%	0.0%	5.3%
Diarrhoea	4.8%	2.9%	3.9%
Dizziness	2.4%	5.9%	3.9%
Hypertension	2.4%	5.9%	3.9%
Nausea	4.8%	2.9%	3.9%
Pneumonia	4.8%	2.9%	3.9%
Vomiting	2.4%	5.9%	3.9%
Blood testosterone decreased	0.0%	5.9%	2.6%
Gastroenteritis viral	0.0%	5.9%	2.6%
Nasopharyngitis	4.8%	0.0%	2.6%
Rash	0.0%	5.9%	2.6%
Thyroxine decreased	2.4%	2.9%	2.6%
Tri-iodothyronine decreased	2.4%	2.9%	2.6%
Abdominal pain	0.0%	2.9%	1.3%
Abdominal pain upper	0.0%	2.9%	1.3%
Accelerated idioventricular rhythm	2.4%	0.0%	1.3%
Agitation	0.0%	2.9%	1.3%
Aortic valve replacement	2.4%	0.0%	1.3%

	NE3107	Placebo	Total
Atrioventricular block first degree	2.4%	0.0%	1.3%
Bile duct stone	0.0%	2.9%	1.3%
Blood lactate dehydrogenase abnormal	0.0%	2.9%	1.3%
Blood prolactin decreased	2.4%	0.0%	1.3%
Blood prolactin increased	2.4%	0.0%	1.3%
Blood sodium abnormal	0.0%	2.9%	1.3%
Blood sodium increased	0.0%	2.9%	1.3%
Blood thyroid stimulating hormone decreased	0.0%	2.9%	1.3%
Bronchitis	0.0%	2.9%	1.3%
Calculus bladder	2.4%	0.0%	1.3%
Cholelithiasis	2.4%	0.0%	1.3%
Cough	0.0%	2.9%	1.3%
Delirium	0.0%	2.9%	1.3%
Dementia Alzheimer's type	2.4%	0.0%	1.3%
Dermatitis	2.4%	0.0%	1.3%
Dysphagia	2.4%	0.0%	1.3%
Dysuria	0.0%	2.9%	1.3%
Electrocardiogram abnormal	2.4%	0.0%	1.3%
Eosinophil count increased	2.4%	0.0%	1.3%
Eustachian tube dysfunction	2.4%	0.0%	1.3%
Hordeolum	0.0%	2.9%	1.3%
Hyperkalaemia	2.4%	0.0%	1.3%

	NE3107	Placebo	Total
Hypothyroidism	2.4%	0.0%	1.3%
Hypoxia	0.0%	2.9%	1.3%
Incontinence	0.0%	2.9%	1.3%
Increased appetite	2.4%	0.0%	1.3%
Influenza	0.0%	2.9%	1.3%
Insomnia	2.4%	0.0%	1.3%
International normalised ratio increased	0.0%	2.9%	1.3%
Lethargy	2.4%	0.0%	1.3%
Lipase increased	0.0%	2.9%	1.3%
Muscle spasms	2.4%	0.0%	1.3%
Nephrolithiasis	0.0%	2.9%	1.3%
Nightmare	0.0%	2.9%	1.3%
Obsessive-compulsive disorder	2.4%	0.0%	1.3%
Oesophageal food impaction	0.0%	2.9%	1.3%
Optic ischaemic neuropathy	2.4%	0.0%	1.3%
Orthostatic hypotension	2.4%	0.0%	1.3%
Papilloedema	2.4%	0.0%	1.3%
Paranasal sinus discomfort	0.0%	2.9%	1.3%
Patient elopement	0.0%	2.9%	1.3%
Pelvic fracture	0.0%	2.9%	1.3%
Pharyngitis streptococcal	2.4%	0.0%	1.3%

Baseline Characteristics of Per-Protocol Population

	NE3107	Placebo
N	24	33
Age – Mean years (Standard Deviation)	75.1 (6.4)	74.9 (5.9)
<70 years	25.0%	24.2%
>=70	75.0%	75.8@
Gender		
Male	37.5%	51.5%
Female	62.5%	48.5%
Race		
Asian	0%	9.1%
Black/African American	8.3%	3.0%
Caucasian	91.7%	84.8%
Mot Reported	0%	3.0%
CDR-SB – Mean (SD)	6.58 (2.5)	7.15 (2.3)
ADAS-Cog12 – Mean (SD)	31.0 (7.6)	34.4 (11.8)
MMSE – Mean (SD)	20.4 (2.6)	19.5 (3.0)
ADCS-ADL – Mean (SD)	58.8 (12.4)	56.0 (14.1)
ADCOMS – Mean (SD)	0.81 (0.2)	0.88 (0.3)

Week 30 Suggest NE3107 Advantage vs. Placebo is Comparable to or Better than Results Reported from Clinical Trials by Approved Medications

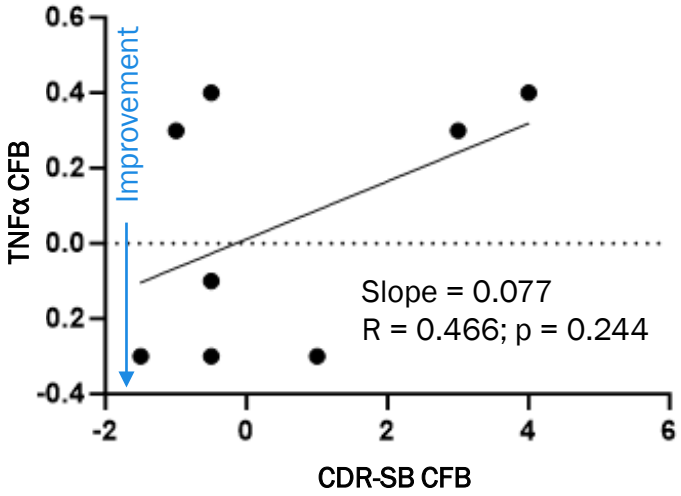
Change from baseline

	Placebo Decline	NE3107	NE3107 vs. Placebo	Comparator (18 mos)
CDR-SB (lower is improvement)	+1.39 (p=0.0125; n=26)	+0.44 (p=0.4522; n=24)	-0.95 (68%) (p=0.2278)	-0.45 (27%)[1] -0.39 (22%)[2]
ADAS-Cog12 (lower is improvement)	+3.64 (p=0.0545; n=23)	+2.70 (p=0.1618; n=24)	-0.94 (26%) (p=0.7212)	-1.44 (25%)[1] -1.40 (27%)[2]
MMSE (higher is improvement)	-2.54 (p=0.0007; n=26)	-1.52 (p=0.0547; n=24)	+1.02 (40%) (p=0.3181)	+0.6 (18%)[2]
ADCS-ADL (higher is improvement)	-6.54 (p<0.0001; n=27)	-3.46 (p=0.0435; n=24)	+3.08 (47%) (p=0.1620)	+2.0 (36%)[1]
ADCS-CGIC (lower is improvement)	+0.31 (p=0.2733; n=26)	-0.12 (p=0.6951; n=24)	-0.43 (139%) (p=0.2866)	
ADCOMS (lower is improvement)	+0.11 (p=0.0358; n=22)	+0.09 (p=0.1094; n=24)	-0.03 (27%) (p=0.7063)	-0.05 (23%)[1]

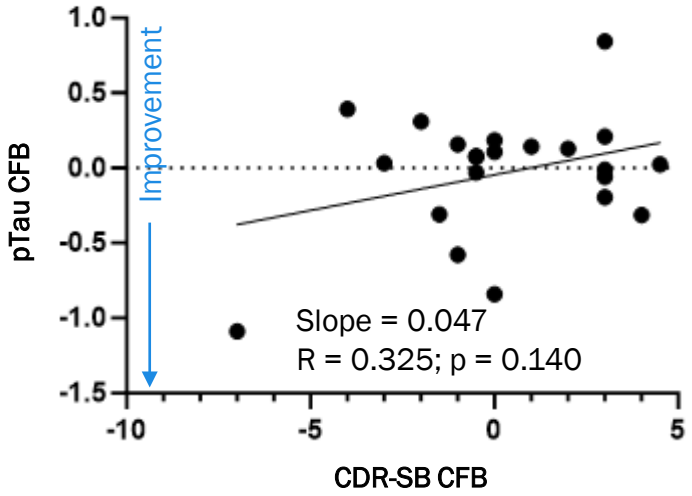
[1] Lecanamab after 18 months; van Dyck et al. *N Engl J Med* 2023;388:9-2
[2] Aducunumab after 18 months; Haeberlein et al. *J Prev Alz Dis* 2022;2(9):197-210

NE3107-treated Patients' Changes in CDR-SB Appears Correlated with Key Biomarkers

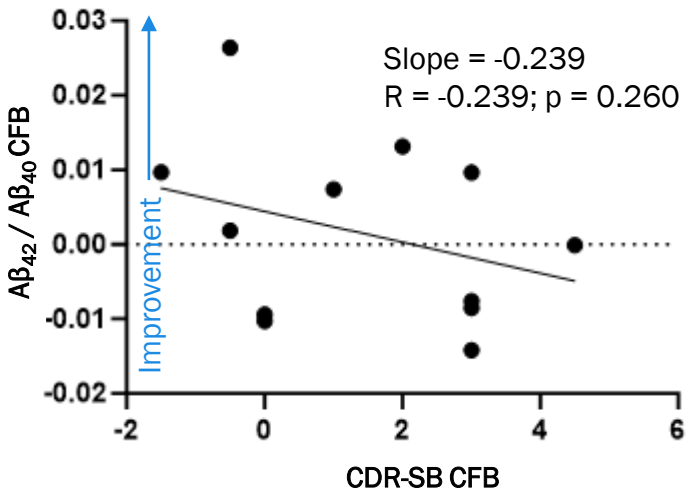
TNFα



Phospho-Tau



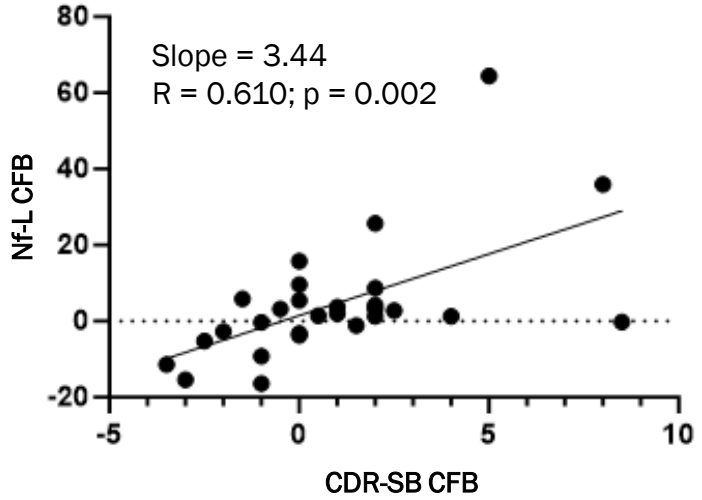
$A\beta_{42}$ / $A\beta_{40}$



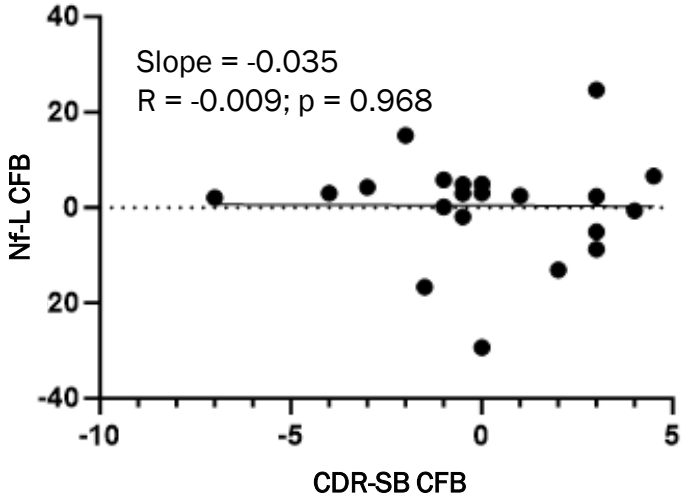
NE3107 Appears to Decrease the Neuroinflammatory Processes that Link Nf-L and GFAP to Cognitive Decline

Nf-L

Placebo

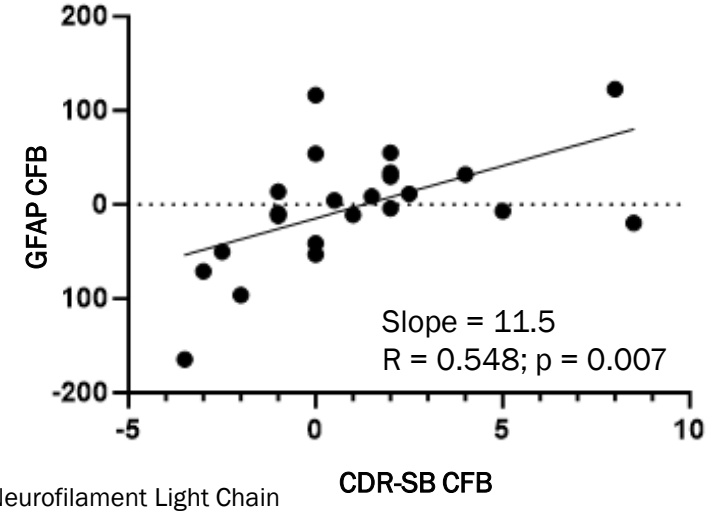


NE3107

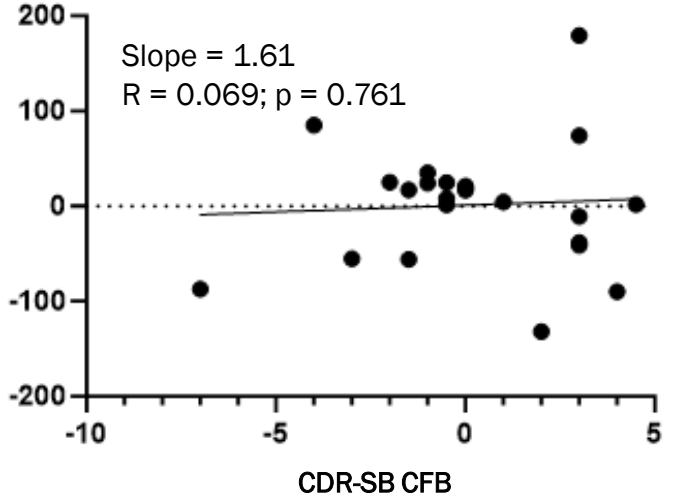


GFAP

Placebo



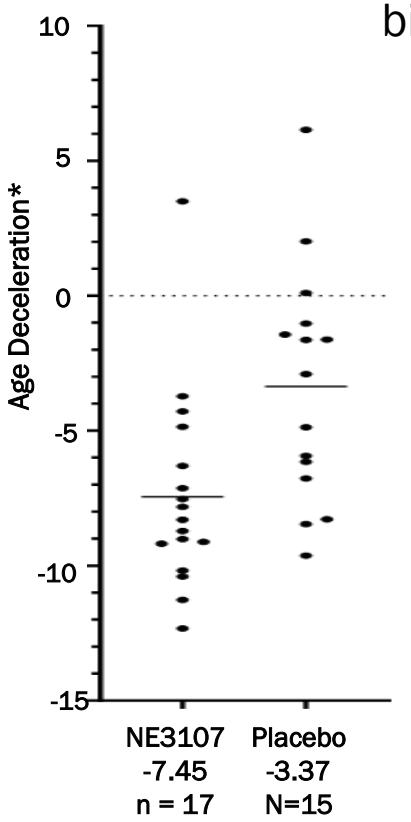
NE3107



Nf-L = Neurofilament Light Chain
GFAP = Glial fibrillary acidic protein

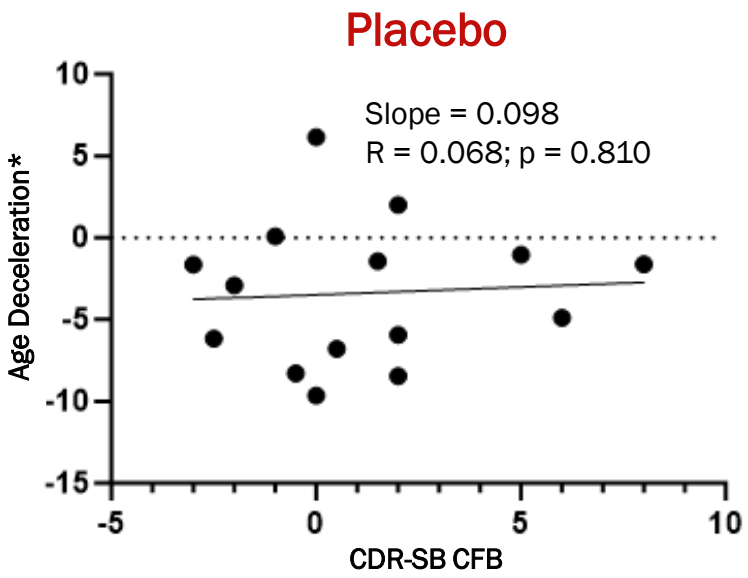
NE3107-treated patients experienced significant “age deceleration” in a manner correlated to cognitive and functional improvements

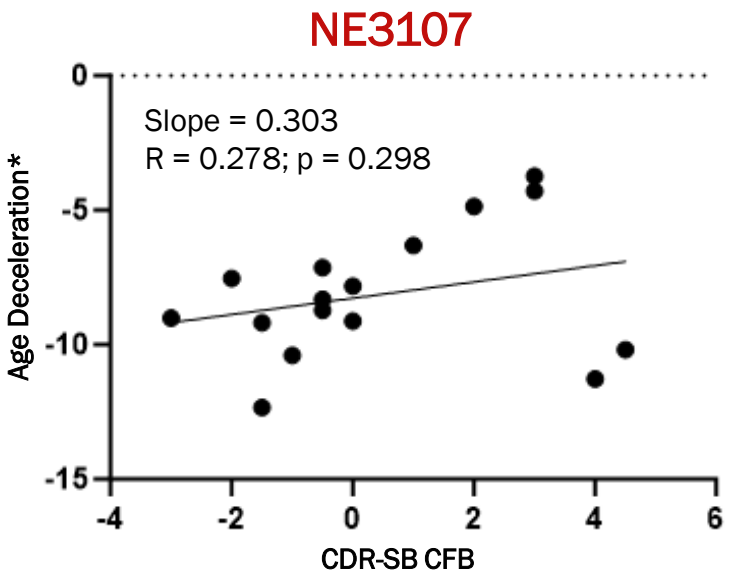
Age Deceleration[1] is used by longevity researchers[2] to measure the difference between a person’s biological age and the actual chronological age.



Difference = -4.09 years

p = 0.007





[1] DNA Methylation Skin Blood Clock Age – Chronological Age

[2] Yusupov et al. *Neuropsychopharmacology* vol 48, 1409–1417 (2023)

NE3107
in Parkinson's Disease

Parkinson's Disease Clinical Development Program

NM201 Phase 2

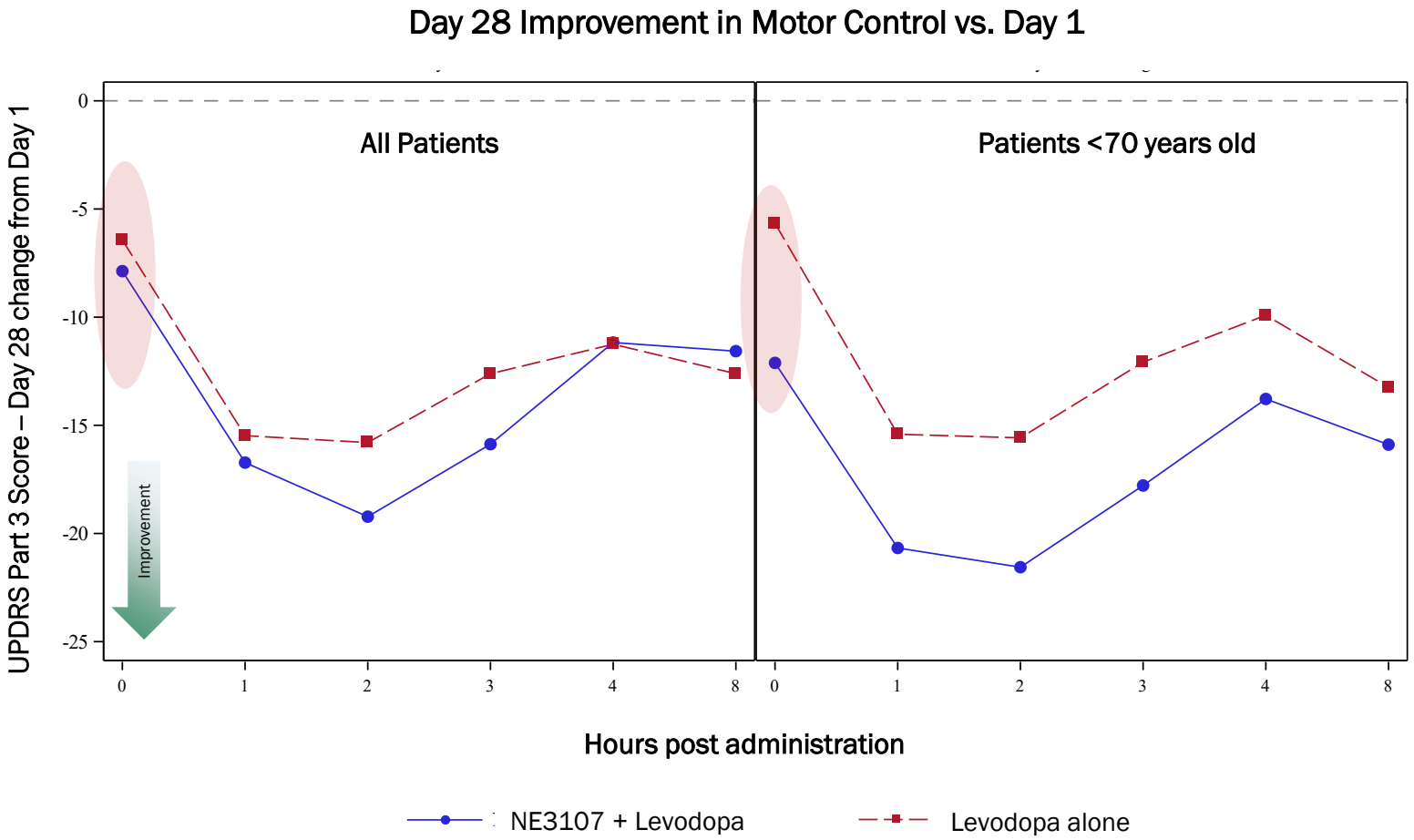
- Satisfying FDA requirement for drug-drug interaction study with L-dopa
- Detect efficacy signal for NE3107's pro-motoric activity

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

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

* Motor Disease Society Unified Parkinson's Disease Rating

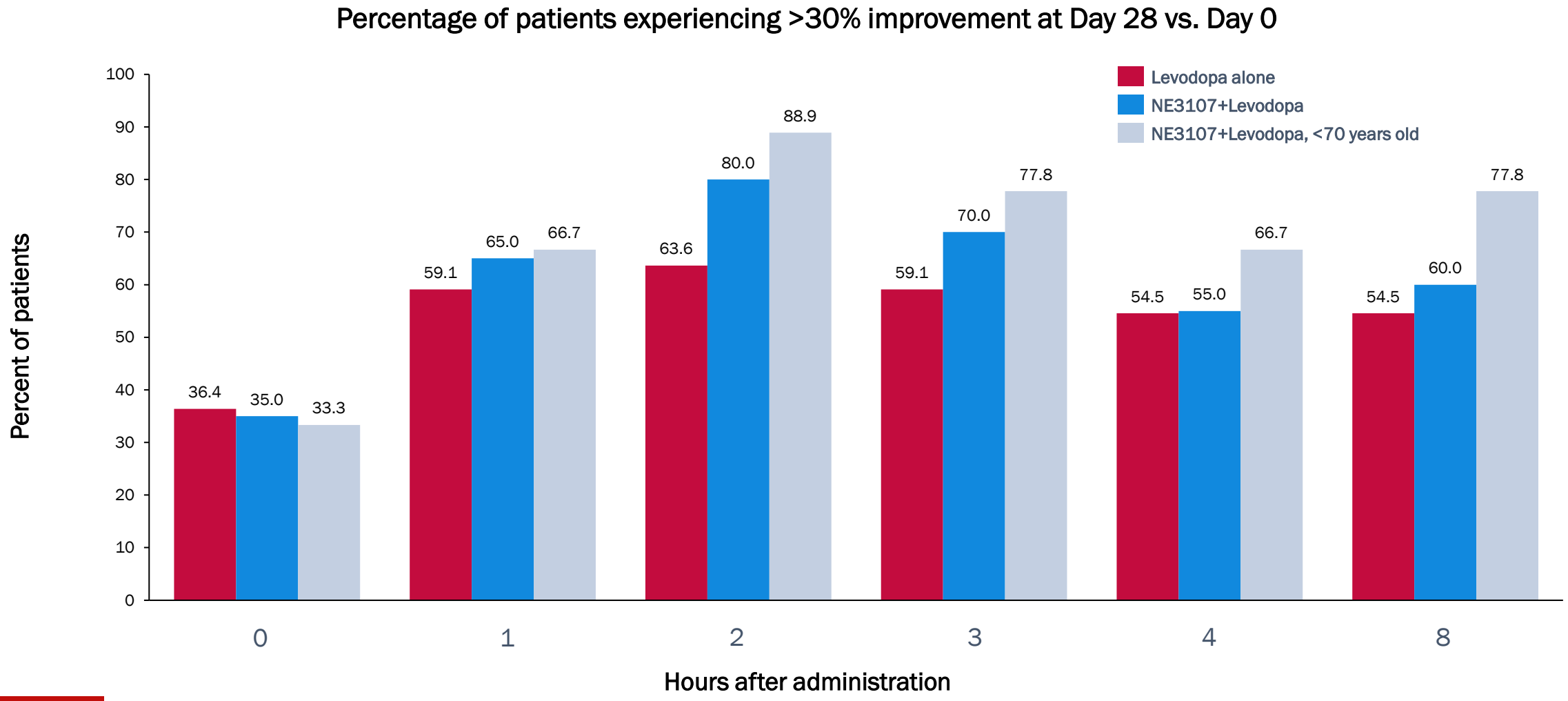
NE3107-treatment patients experienced fewer motor symptoms before morning drug administration



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

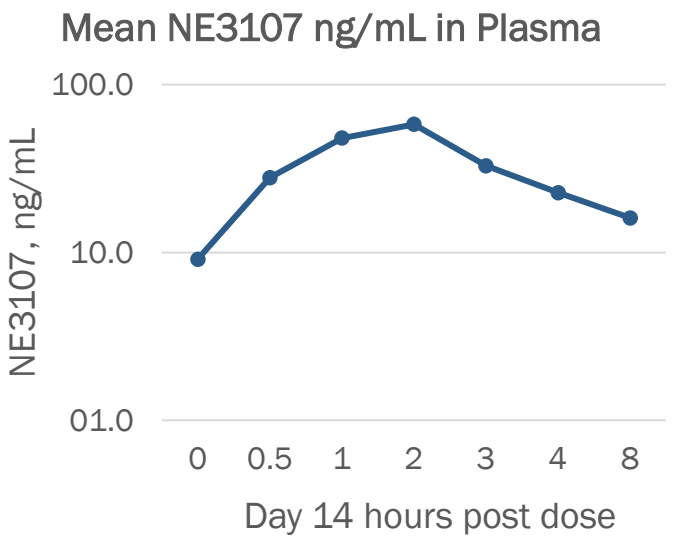
* Fisher's exact test

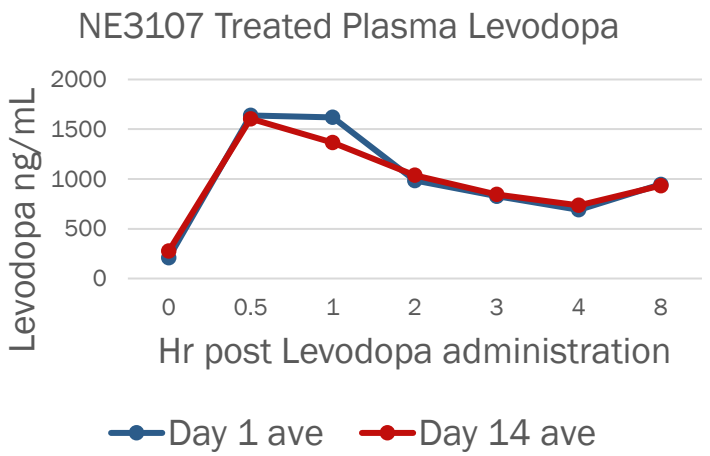
Larger proportion of patients treated with NE3107 had >30% improvements in motor control

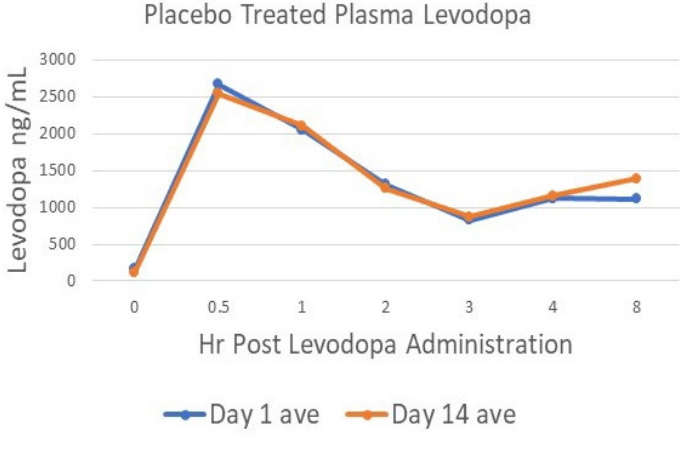


Desirable pharmacokinetics – no observed DDI

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

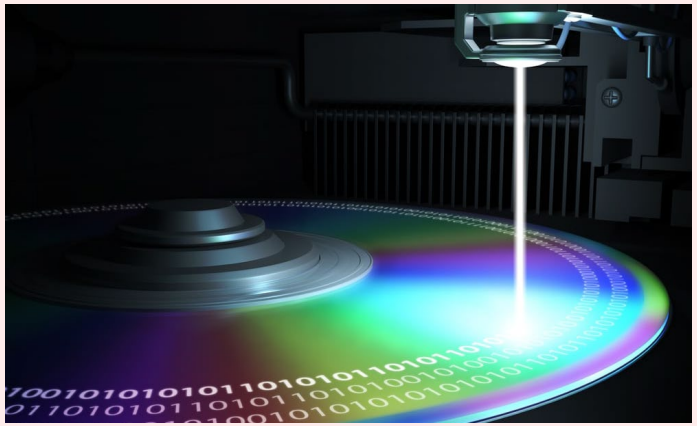






NE3107
in Longevity

Clean DVD leads to clear picture and sound



Scratches & smudges lead to skips and blurs

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

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

The same thing happens in our body

DNA methylation

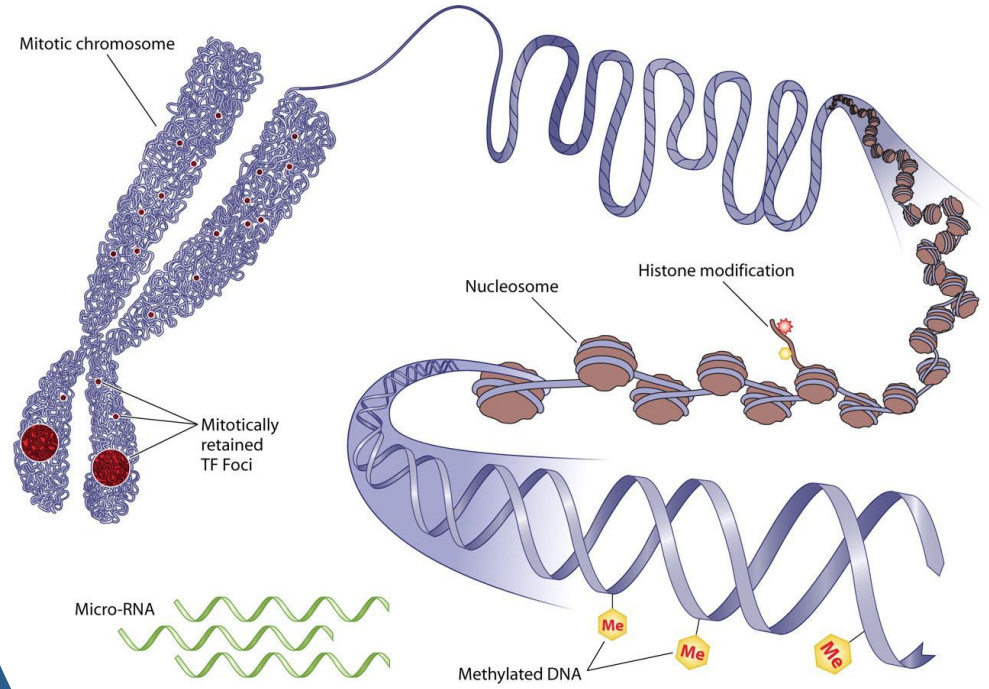
DNA methylation happens when methyl groups are added to our DNA

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

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

- 28 million CpGs in genome

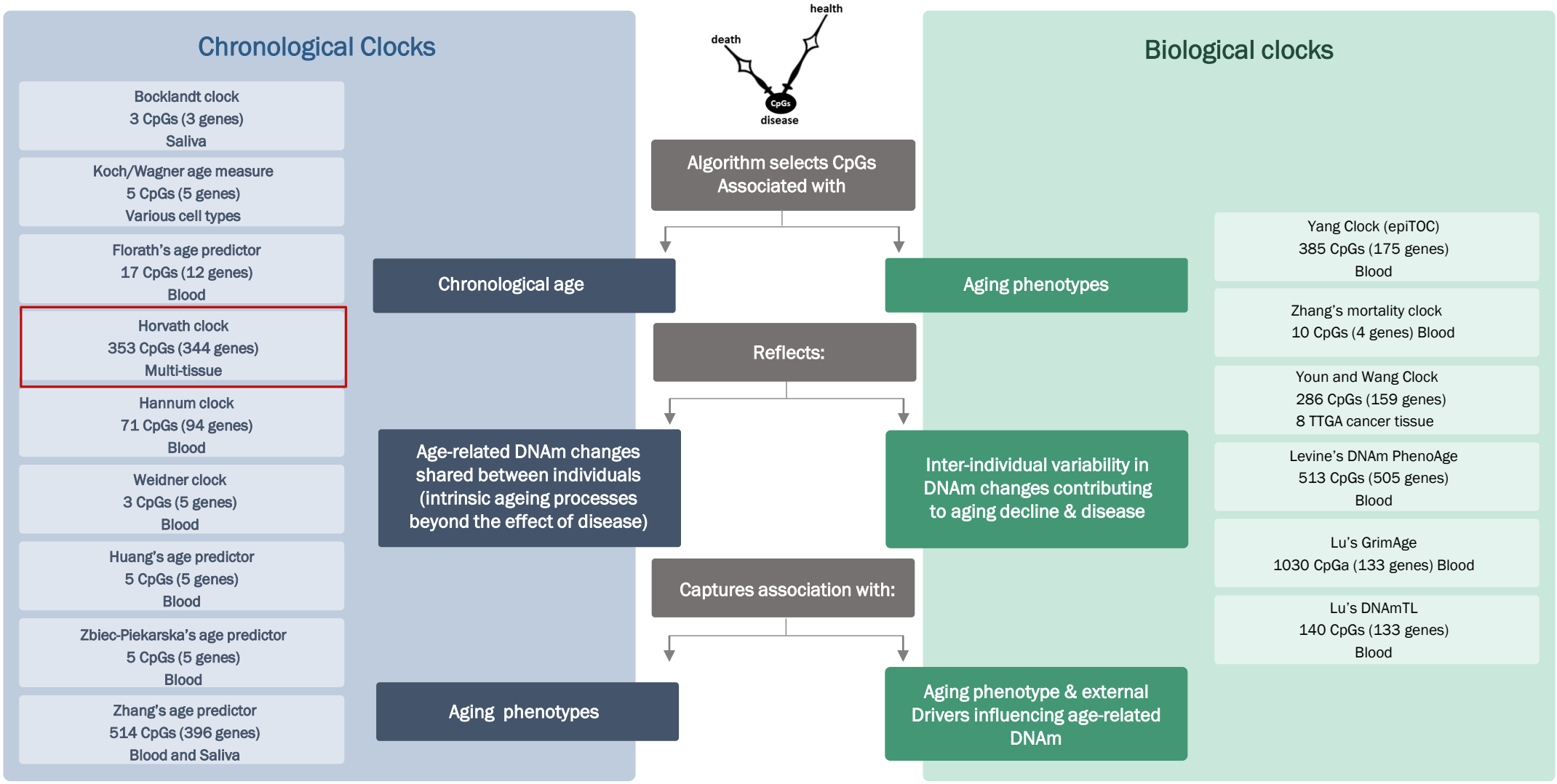
Hypermethylation of DNA is associated with many disease conditions



Observations about DNA methylation

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

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

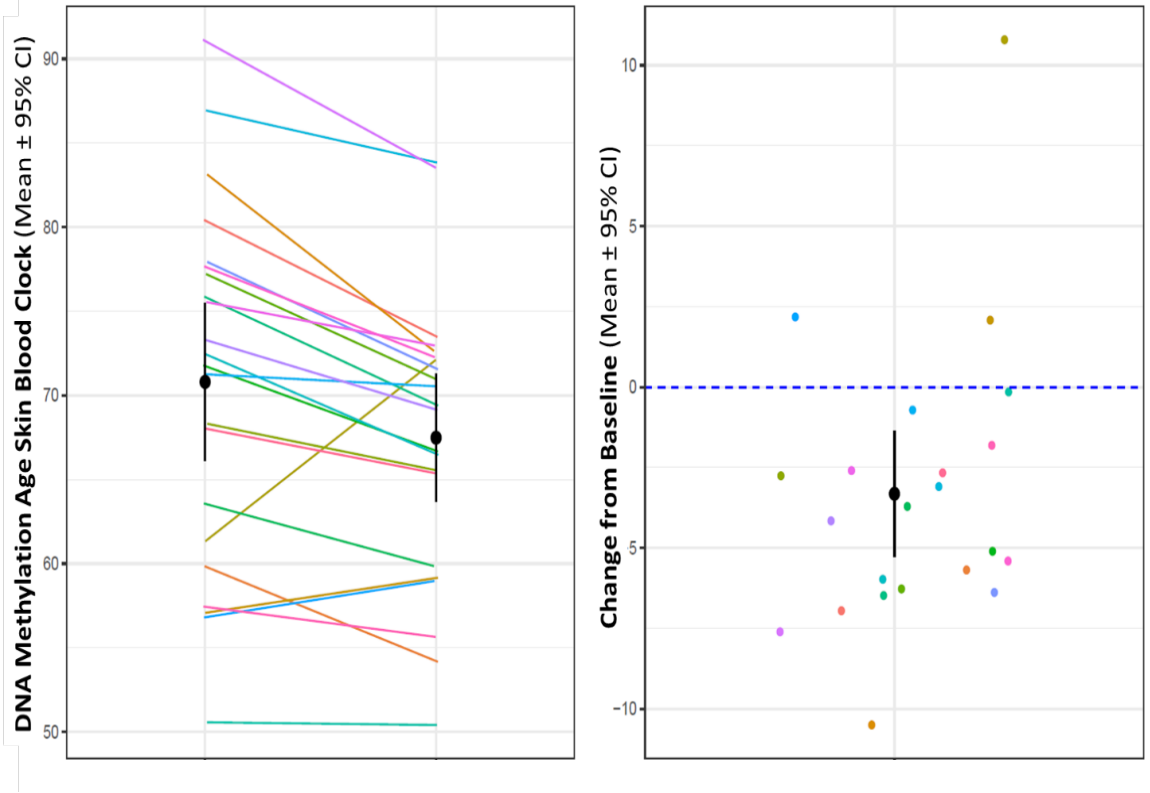


NE3107 significantly reduced DNA methylation as measured by the SkinBloodAge Clock

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

The biological clock age was in close agreement with the chronological age (72.3 vs 71.6; +0.98%) at baseline

After 3 months treatment with NE3107 there was a decrease in DNA methylation commensurate with 3.3 years reduction on the Skin Blood Clock (68.1 vs 71.6; -4.9%)

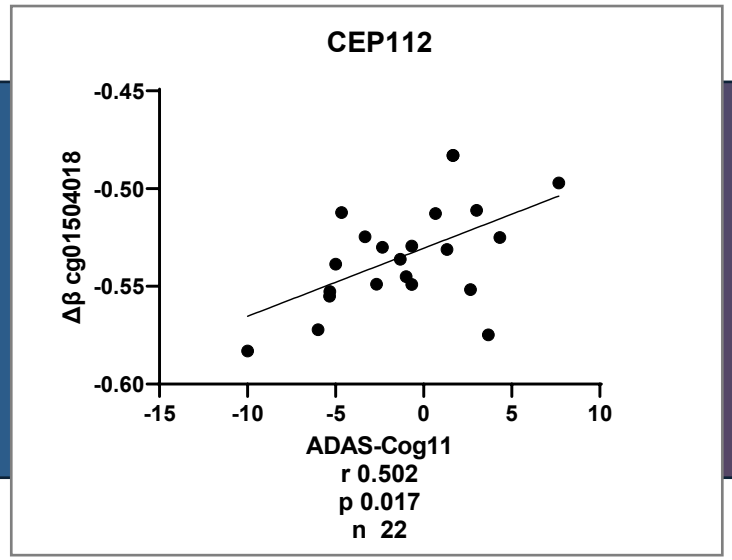


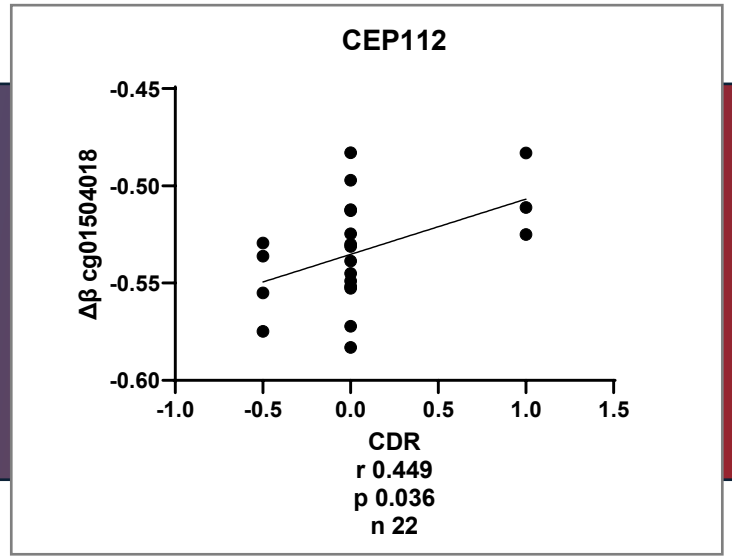
19/22 decreased (86%)

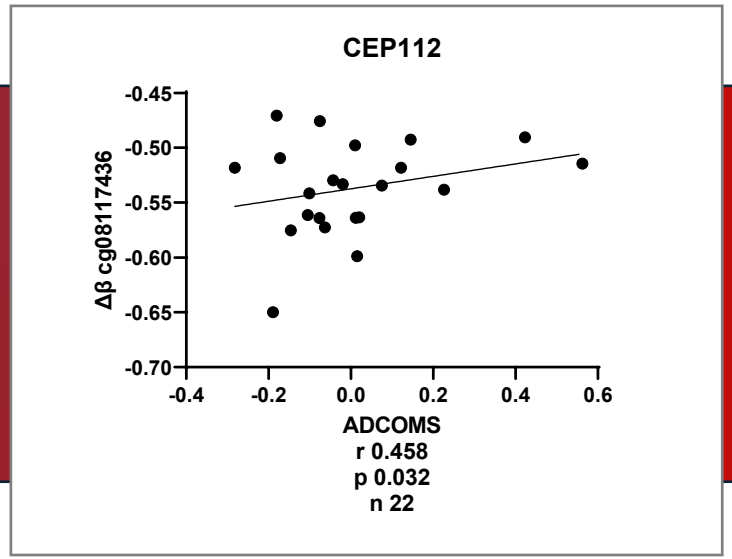
Mean Absolute Change = -3.3 years (p=0.0021)

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

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







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

* Zhang 2020 Alzheimers Res Ther 12 165

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

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

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

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

Summary of findings

01

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

02

Data show that patients treated with NE3107 experienced reduced DNA methylation

03

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

BIV201
in Ascites

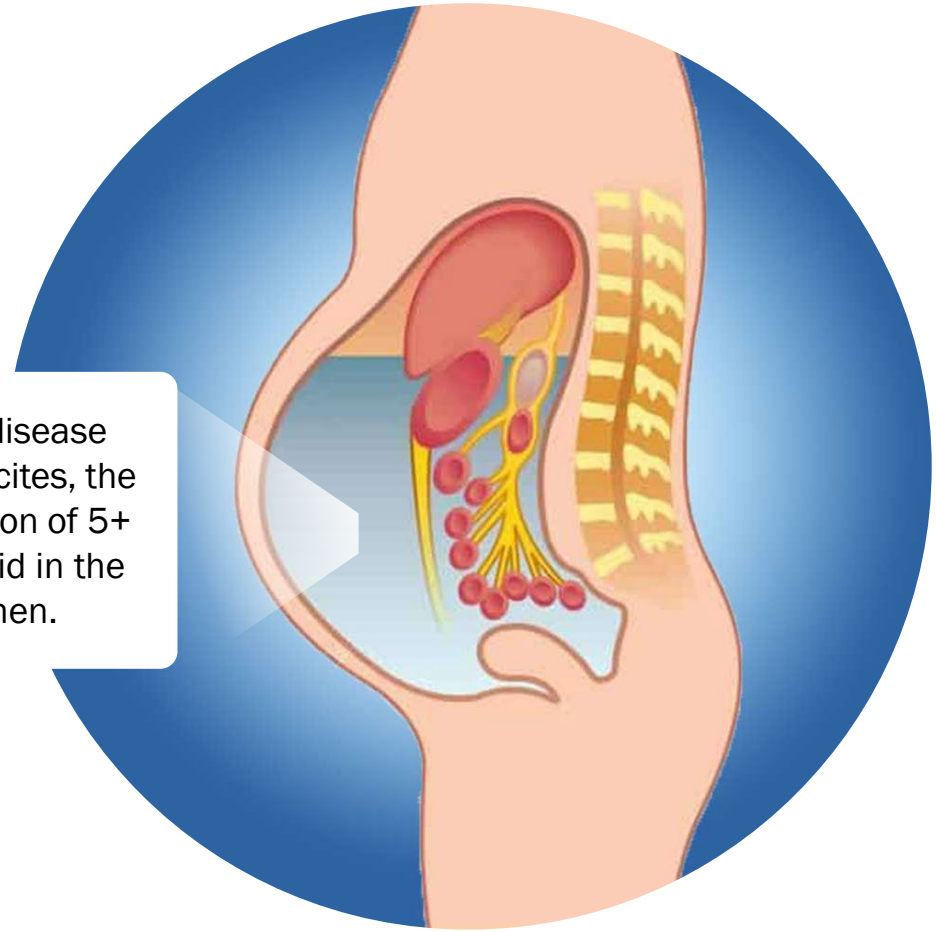
BIV201 Disease Target: Refractory Ascites

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

Paracentesis:

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

No drugs ever approved by FDA to treat ascites



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

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

Prefilled Syringe with Patent-pending Liquid Formulation

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

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

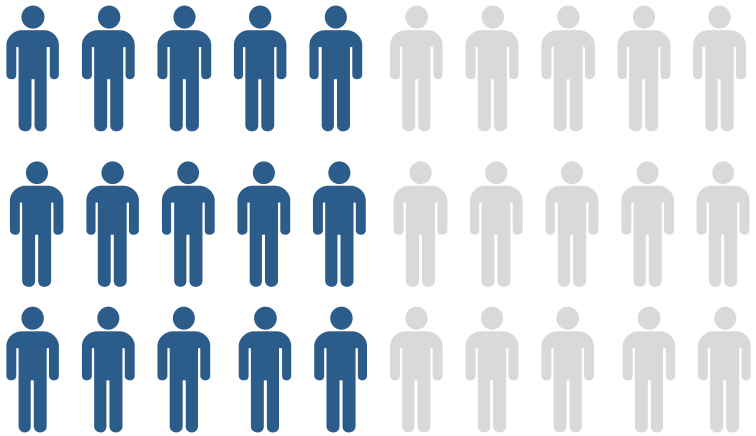
Needle or Connector

50 mL bag of saline for insertion into pump

Portable pump
Carried in small satchel

*In clinical trials; not approved by FDA

BIV201 Phase 2b trial

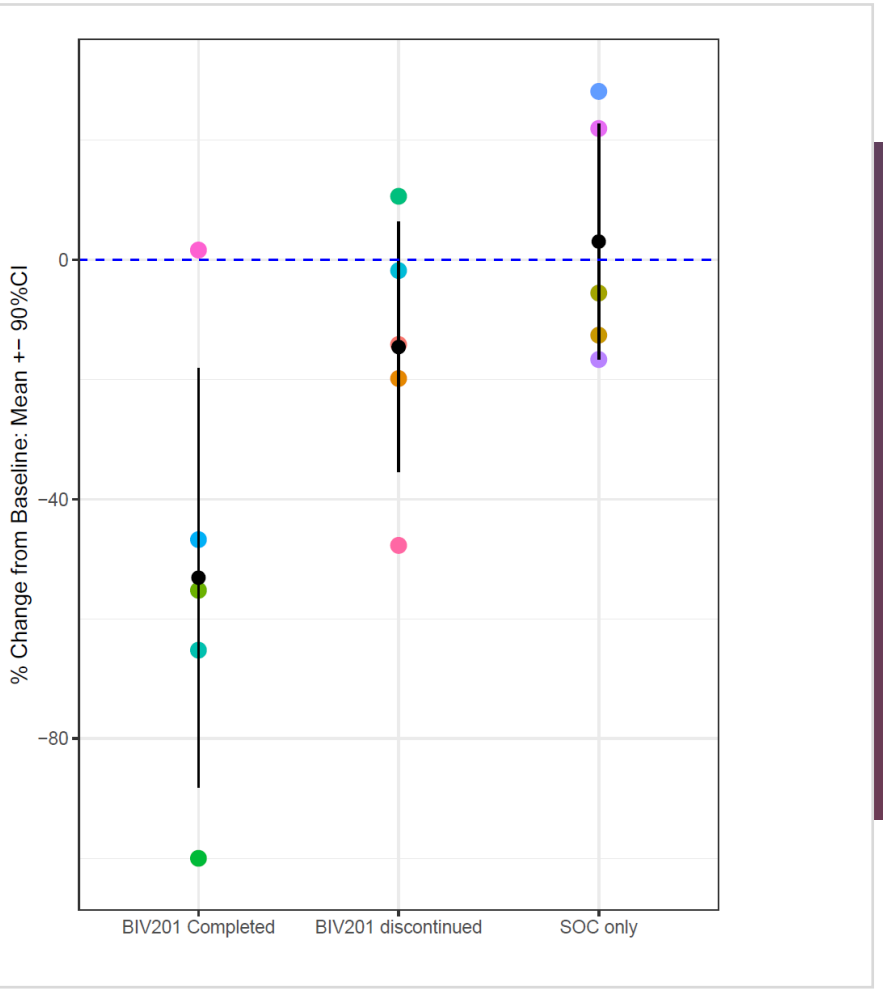


Originally targeted 30 patients randomized 2:1

Paused enrollment based on encouraging data from the first 15 patients informing next steps

- 10 randomized to BIV201; 5 randomized to standard of care
- 5 completed 2 X 28-day cycles
- 5 discontinued treatment during or at end of Cycle 1

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



53% reduction in ascites volume among patient completing BIV201 treatment

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

3.1% increase for SOC patients

p<0.001

Commercial potential in US market alone*

Alzheimer's

$30B

Annual sales for every 1 million people treated

15% market penetration

$30K/year much lower all-in cost vs. competition

Parkinson's

$3B

Annual sales for every 100,000 people treated

10% market penetration

$30K/year

Ascites

$1.6B

US peak sales

45% market penetration

$45K/year

2026 launch

2032 peak sales

Leadership Team

Deep expertise provides a strong foundation for success

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

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

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

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

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

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

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

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

Capitalization Table

As of December 15, 2023	
Common shares outstanding	39,841,080
Warrants (WAEP: $2.06)	7,770,285
Options (WAEP $ 6.38)	4,347,881
Restricted stock units	701,693
Fully diluted shares outstanding	51,959,246
Market Cap (December 15, 2023, close price $1.45)	$57,770,000

Recap

We believe that TNFα-mediated inflammation

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

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

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

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

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

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

Thank You