Topline Results from SUNRISE-PD Trial

August, 2026

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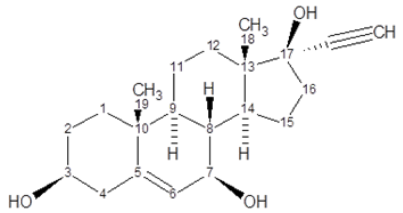
BEZISTERIM

Believed to block ERK/NFκB-mediated inflammatory signaling while preserving homeostatic function

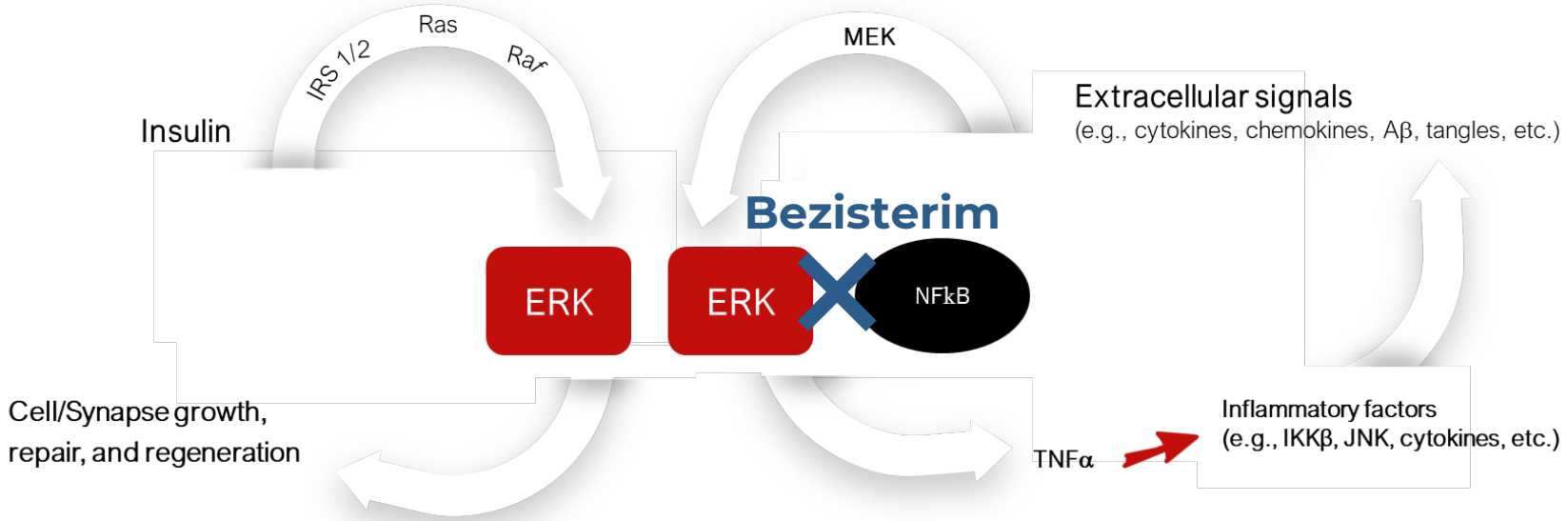


First-in-class molecule with desirable characteristics

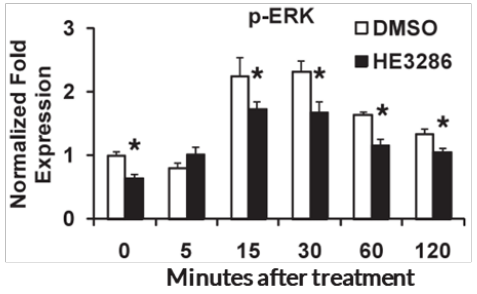
Small molecule; orally bioavailable

Crosses blood-brain barrier

No safety issues identified to date in pre-clinical and clinical trials (up to Phase 3)

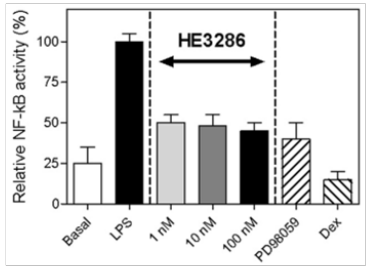


Bezisterim Reduces ERK Activation



Lu 2010 Am J Physiol Endocrinol Metab 298 E1036

Bezisterim Reduces NFkB Activation



Wang 2010 J Pharmacol Exp Ther 333 70

SUNRISE-PD PHASE 2 TRIAL

- Designed as a ***short***, signal finding trial with early-stage PD patients
  - Identify efficacy signals: endpoints with favorable treatment benefits
  - Measure the magnitude of treatment impact
  - Enable design and sizing of Phase 3 trial
- Evaluated a series of motor and non-motor endpoints
  - Early Parkinson's Neuro-Inflammation Composite 15 (EPNIC)
  - MDS-UPDRS Parts I (non-motor), II (activities of daily living), III (motor), and Total
  - PDSS-2 (sleep) and PDQ-39 (Qualify of Life)
- Evaluated a series of biomarkers, including a proteomics battery evaluating 380 different proteins
  - Neurodegenerative (NfL, GFAP, pTau-217, aβ42)
  - Neuroinflammation (CCL2, CHI3L1, VGF)
  - Peripheral inflammation (MLR, SIRI, IL-17A, IFN-γ, TNFα, others)

KEY FINDINGS

- Patients treated with bezisterim appeared to have experienced statistically significant treatment advantages compared to placebo
  - **Clinical outcomes:** composite of 15 motor and non-motor domains of PD
  - **Proteome-wide impact:** 283 / 380 proteins moving towards direction that has been shown to slow disease progression
  - **Neurodegeneration:** composite and individual biomarkers (NfL, GFAP, others)
  - **Neuroinflammation:** CCL2, CHI3L1, others
  - **Peripheral inflammation:** monocyte-to-lymphocyte ratio, SIRI, others
- Bezisterim appeared to demonstrate an effect across the trial population, and those with higher levels of inflammation at baseline experienced statistically significant advantages over placebo across all clinical measures evaluated, including UPDRS Parts I, II, and III, UPDRS Total, and the EPNIC-15 composite
- Biomarker results suggest that bezisterim may slow neurodegeneration over time – a hypothesis that we will test in the Phase 3 registrational trial
- Bezisterim was well-tolerated; similar to placebo

WHAT IS PARKINSON'S DISEASE

Progressive

A neurodegenerative disorder driven by ongoing loss of dopamine-producing neurons.

More than movement

Causes motor symptoms (tremor, slowness, stiffness) and non-motor symptoms (sleep, mood, autonomic).

No disease-modifying therapy

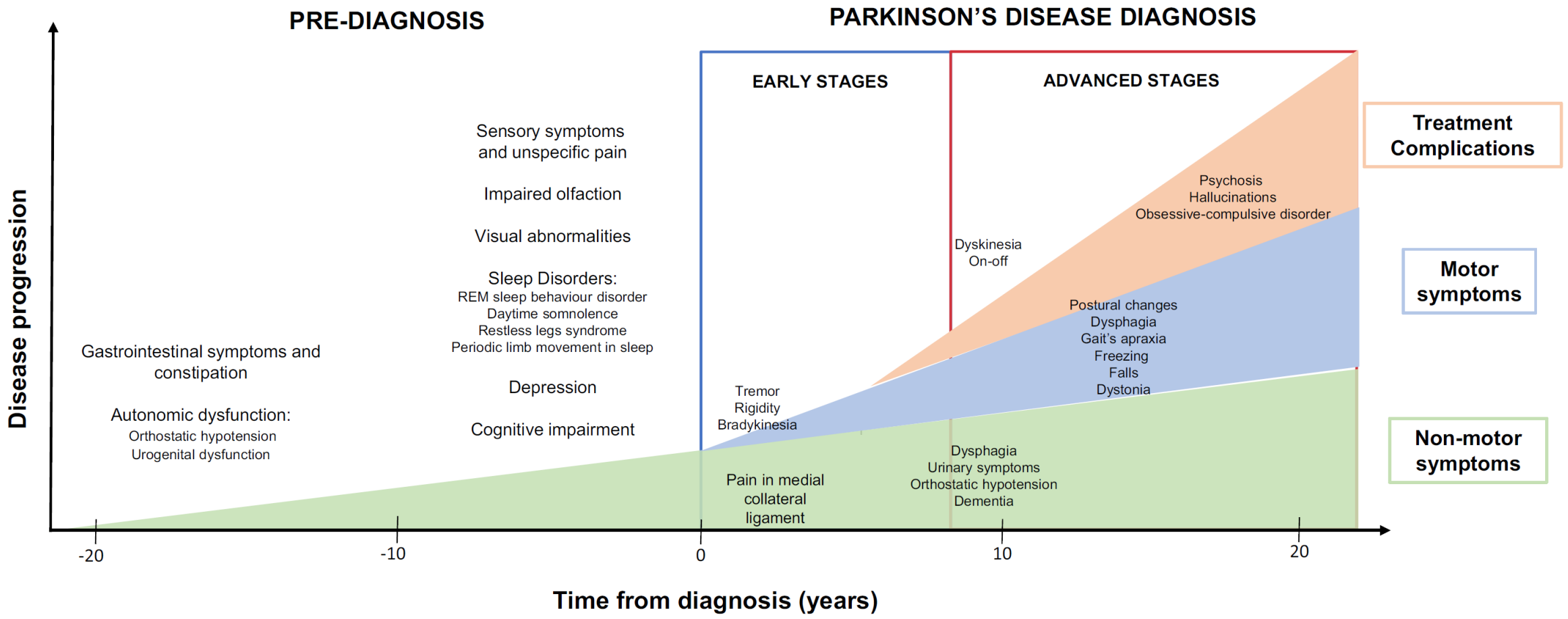
Current treatments ease symptoms; none is proven to slow the underlying disease.

The unmet need

A therapy that addresses non-motor symptoms and slows progression – not just masks symptoms – would be first-of-its-kind.

Parkinson's disease is associated with progressive loss of dopamine neurons causing both movement and non-movement symptoms. No existing therapy slows progression.

SYMPTOMATIC PROGRESSION IN PD

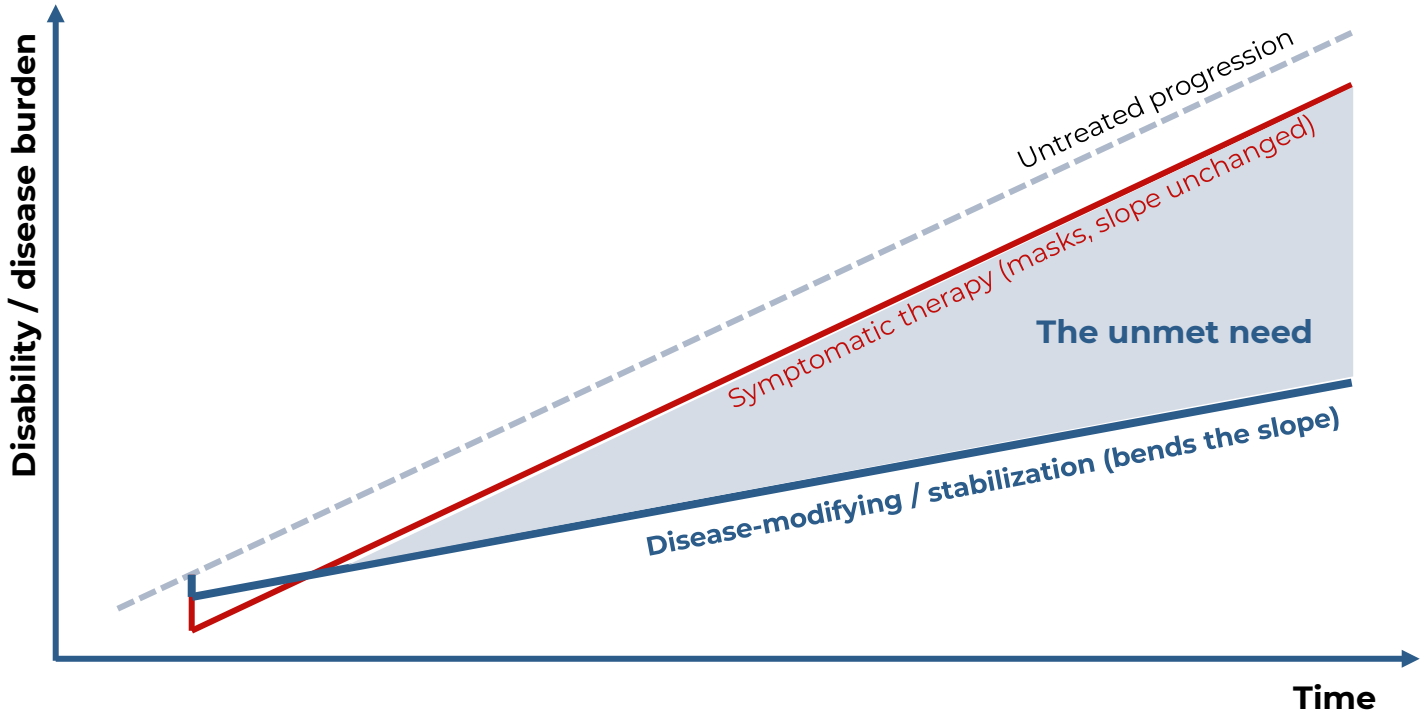


NOTE: Copied from Castilla-Cortázar et al. *J Transl Med* (2020) 18:70. https://doi.org/10.1186/s12967-020-02223-0

CONCEPTUAL DEPICTION OF THE UNMET NEED

Existing PD therapies relieve symptoms – not disease progression. A disease-modifier remains the unmet need

Illustration of the unmet need: Changing the slope to slow progression



- **Intercept shift** — symptomatic drugs mask while progression continues.
- **The real need** — to bend the slope
- **Benefit signal** — shows up as “less worsening”
- **Endpoint gap** — short-term motor endpoints poorly detect change

THE NEUROINFLAMMATION HYPOTHESIS

Why an anti-inflammatory could modify Parkinson's disease.

Peripheral inflammation

TNF–NF-κB signaling in multiple cells types, monocytes, platelets, neutrophils, visceral adipose

→

Neuroinflammation

Oxidative stress, ER stress, and insulin resistance, potentially augmented by peripheral inflammatory signaling and infiltrating cells, stresses vulnerable glia and neurons

→

Neuronal loss

Chronic inflammation contributes to progressive dopaminergic, cholinergic and noradrenergic neuron death

→

Clinical progression

Accumulating motor and non-motor decline over time

Bezisterim is believed to dampen the TNF / NF-κB step without broad immunosuppression. Reducing inflammation could slow the disease.

WHAT WE MEASURED IN THE SUNRISE-PD TRIAL

Four windows on the biology

1

Inflammation

CBC-derived ratios (MLR, SIRI, NLR...)

Systemic inflammatory tone

2

Proteome

380 plasma proteins related to inflammation and CNS disease

Coordinated molecular shift

3

Nerve injury

Neurofilament light (NfL), Glial fibrillary acidic protein (GFAP), others

Ongoing axonal damage

4

Clinical scales

MDS-UPDRS I–III, PDSS-2, PDQ-39

Symptoms & function

HOW TO READ THE NUMBERS

Two yardsticks used throughout: effect size (how big) and the p-value (how sure)

Cohen’s d — how big is the effect?

d ≈ 0.2	small
d ≈ 0.5	medium
d ≈ 0.8	large
Negative value	Treatment improvement

Most bezisterim effects sit in the “large” zone.

p-value — how sure are we?

Smaller p = less likely to be coincidental
p < 0.05 is the conventional bar for statistical significance

BASELINE CHARACTERISTICS

Treatment and placebo arms appear balanced at baseline

Characteristic	Bezisterim	Placebo	p-value
n	28	29	—
Age, years (mean ± SD)	65.8 ± 7.8	66.2 ± 7.4	0.849
Sex, Male / Female (n)	20 / 8	16 / 13	0.274
MDS-UPDRS Part I (mean ± SD)	6.2 ± 3.9	5.0 ± 3.9	0.247
MDS-UPDRS Part II (mean ± SD)	6.9 ± 3.9	7.0 ± 4.8	0.903
MDS-UPDRS Part III (mean ± SD)	25.0 ± 7.6	25.5 ± 8.8	0.993
CGI-S (mean ± SD)	2.9 ± 0.7	2.8 ± 0.6	0.716
MoCA (mean ± SD)	27.8 ± 1.4	28.1 ± 1.8	0.505
GDS-SF (mean ± SD)	1.9 ± 1.7	1.3 ± 1.0	0.098
ESS (mean ± SD)	4.6 ± 2.2	5.3 ± 2.6	0.298
Baseline Platelets, $\times 10^9$/L (mean ± SD)	235.1 ± 49.9	241.0 ± 70.7	0.731
Baseline Platelets >230, n (%)*	14 (50.0%)	14 (48.3%)	1.000
Baseline Platelets ≤230, n (%)*	11 (39.3%)	13 (44.8%)	—

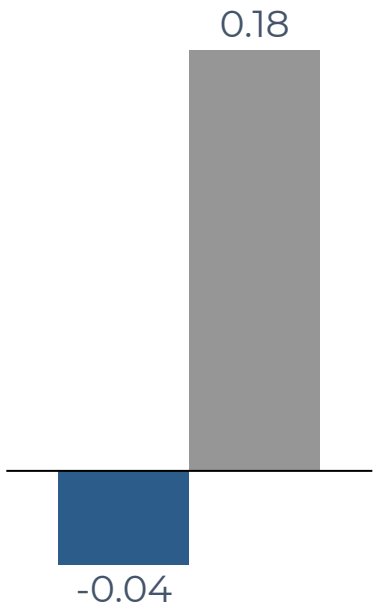
* Cutoff based on median 233 $\times 10^9$/L for study population

MDS-UPDRS = Movement Disorder Society-Unified Parkinson's Disease Rating Scale; **CGI-S** = Clinical Global Impression-Severity; **MoCA** = Montreal Cognitive Assessment; **GDS-SF** = Geriatric Depression Scale Short Form; **ESS** = Epworth Sleepiness Scale;

STABILITY OF TREATMENT EFFECT ACROSS ITT POPULATION

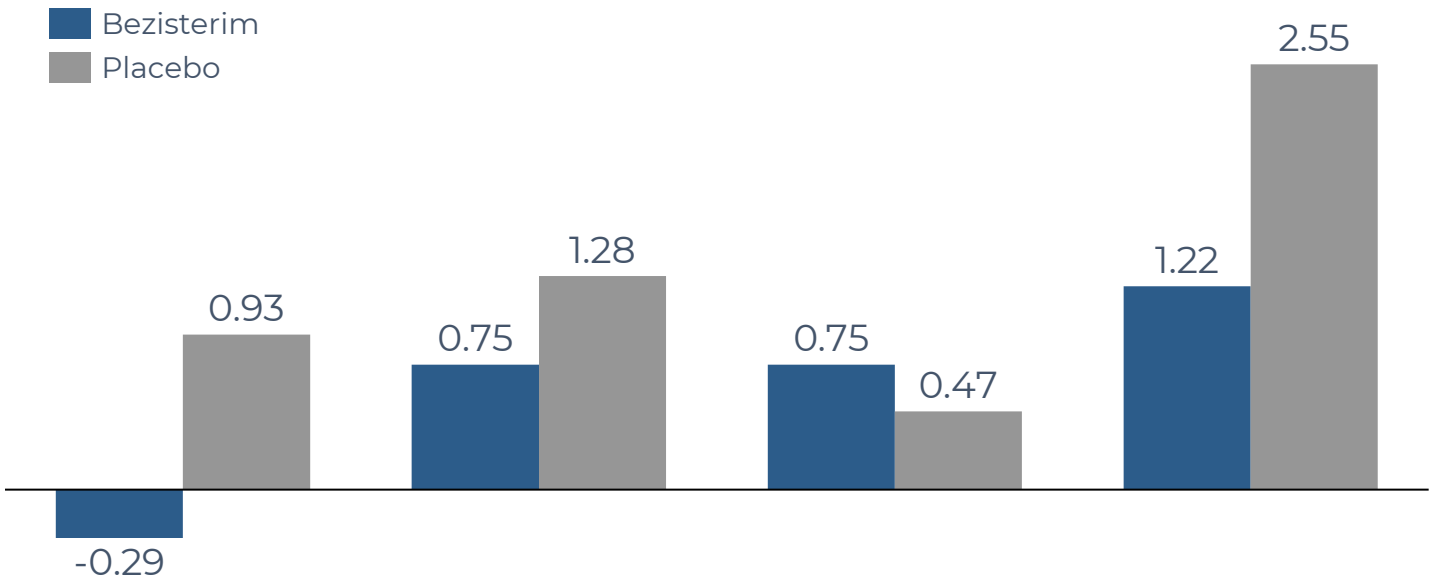
Across the Intent-to-Treat population (N=57; Bezisterim n=28, Placebo n=29), Bezisterim shows a smaller mean change from baseline than Placebo on UPDRS Part I, Part II, and the Total scores – consistent with slowing deterioration rather than immediate symptomatic improvement.

EPNIC-15



EPNIC-15
Cohen's d = -0.94
p=0.0006

MDS-UPDRS Scores



Part I
Cohen's d = -0.42
p=0.0984

Part II
Cohen's d = -0.20
p=0.2236

Part III
Cohen's d = -0.08
p=0.3926

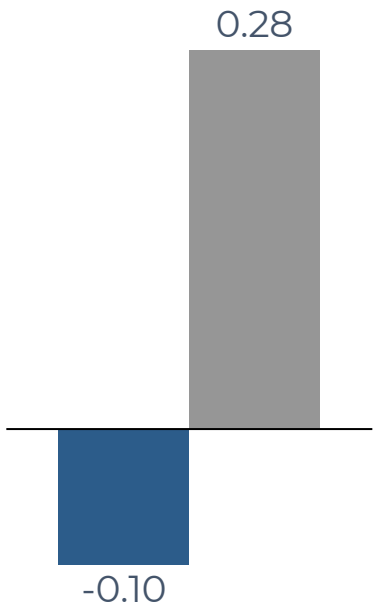
Total
Cohen's d = -0.21
p=0.2246

p-values are one-sided. Positive values indicate worsening from baseline; negative values indicate improvement.

LARGER EFFECT SIZE WITH HIGHER BASELINE INFLAMMATION

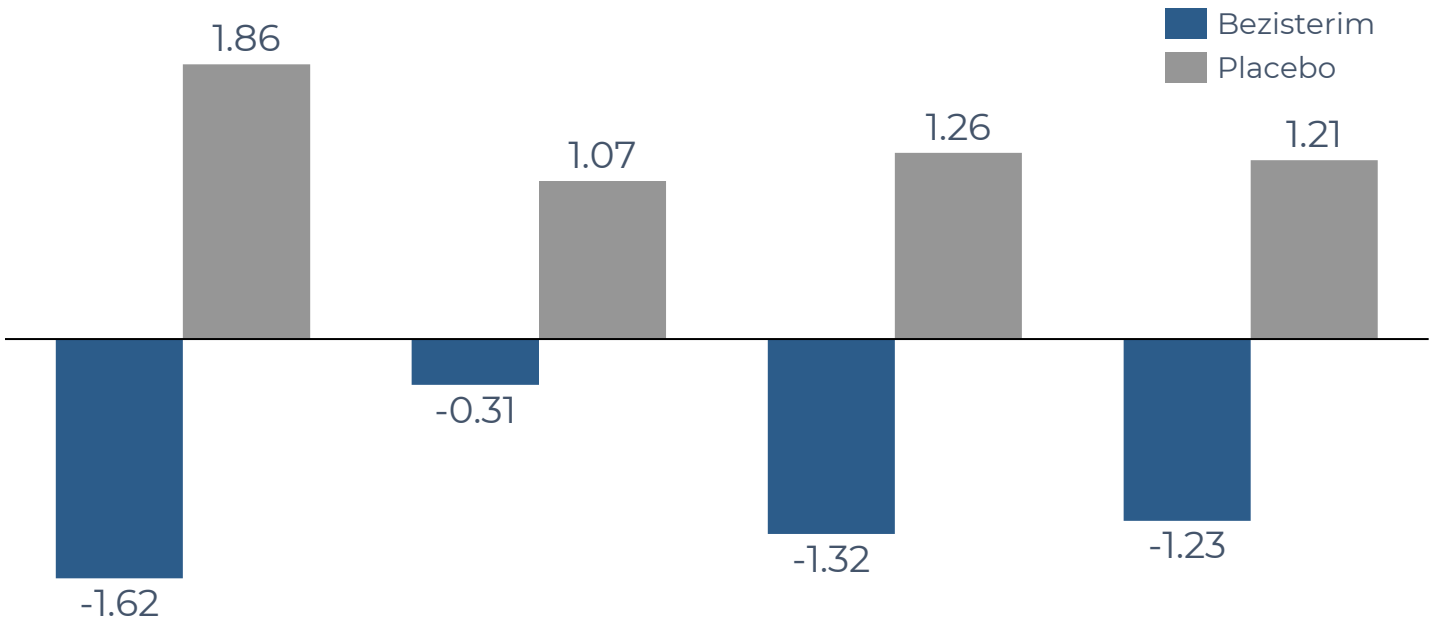
While bezisterim appeared to demonstrate an effect across the trial population, the greatest improvement was seen in those with baseline platelet counts >230 (n=27)

EPNIC-15



EPNIC-15
Cohen's d = -1.61
p=0.0002

MDS-UPDRS Scores



Part I
Cohen's d = -1.13
p=0.0062

Part II
Cohen's d = -0.71
p=0.0418

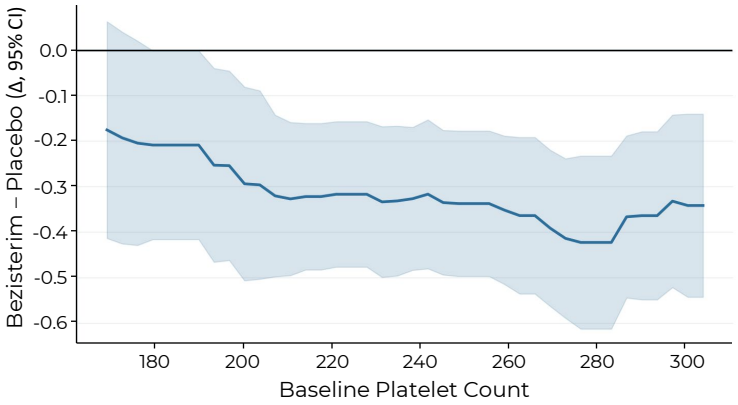
Part III
Cohen's d = -0.92
p=0.0133

Total
Cohen's d = -0.85
p=0.0198

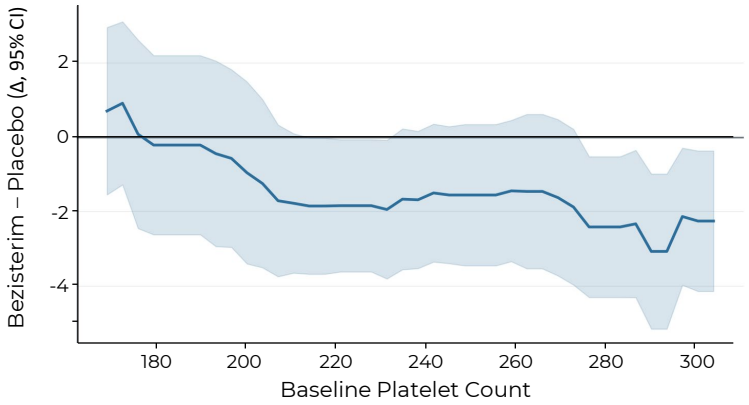
BEZISTERIM ADVANTAGE ACROSS PLATELET RANGE

Platelet levels provide an enrichment strategy – not an exclusion of subgroups with no treatment effect

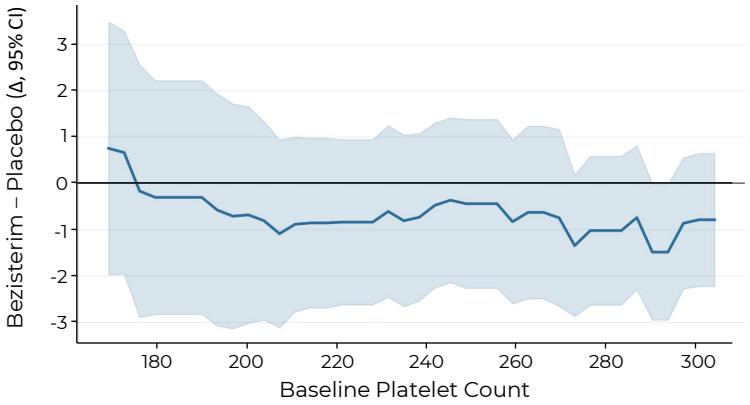
EPNIC-15



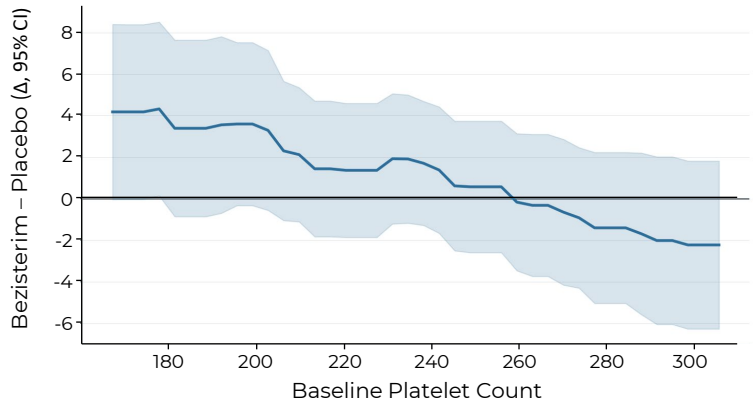
MDS-UPDRS Part I (Non-Motor)



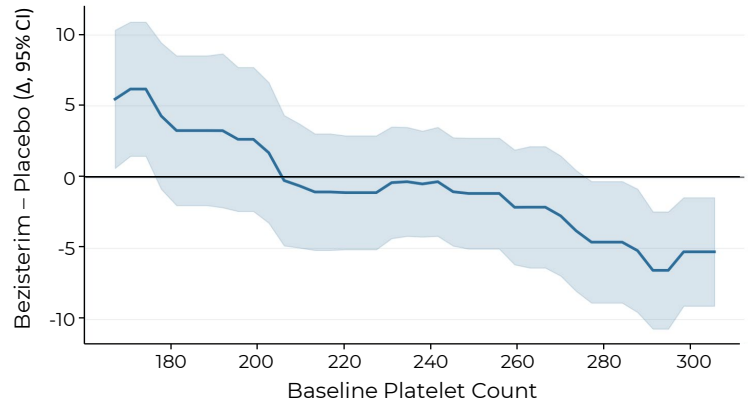
MDS-UPDRS Part II (Motor ADL)



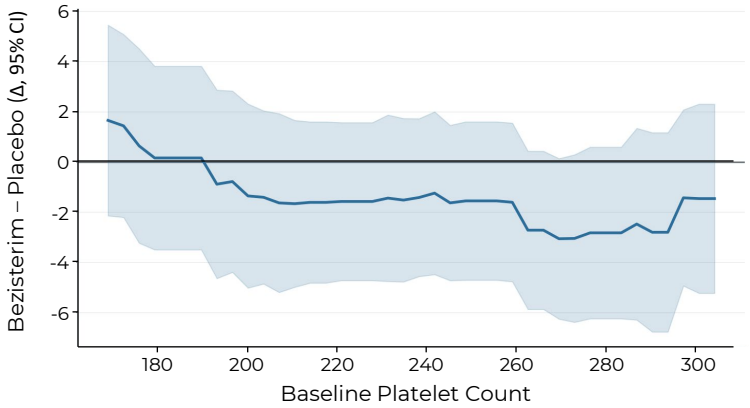
MDS-UPDRS Part III (Motor)



MDS-UPDRS Total Score



PDSS-2 (Sleep)



STEPP-style sliding window (±60 unit platelet band); shaded band = 95% CI; lower = greater bezisterim benefit

WHY EPNIC-15 COMPOSITE OF CLINICAL OUTCOMES IS NEEDED

EPNIC-15 comprises 15 subdomains from MDS-UPDRS Parts I, II, and III and PDSS-2*

- PD therapies have historically been approved based largely on the MDS-UDPRS, which are focused primarily on motor symptoms and largely for late-stage disease
  - 6 of the last 10 approvals were based primarily on daily OFF time
  - 2 of the last 10 approvals were based on UPDRS Part III scores (motor symptoms)
- Bezisterim has shown an impact on motor symptoms. But its apparent clinical impact could be broader based on a different mechanism of action
- Therefore, using motor-focused endpoints alone may undervalue the full potential of what could be the first therapy to address both the motor and non-motor symptoms of PD
- The Early Parkinson's Neuro-Inflammatory Composite-15 (EPNIC-15) comprises 15 subdomains of UPDRS Part I-III and PDSS-2
- It was constructed based on learnings from Biohaven, Broadstreet HEOR, and Pentara's efforts to create the Parkinson's Composite Scale (PARCOMS)**

EPNIC-15 item	Domain
Sleep problems	UPDRS Part I
Daytime sleepiness	UPDRS Part I
Constipation	UPDRS Part I
Hobbies/activities	UPDRS Part II
Tremor (ADL)	UPDRS Part II
Walking & balance	UPDRS Part II
Speech	UPDRS Part III
Toe tapping R	UPDRS Part III
Toe tapping L	UPDRS Part III
Leg agility L	UPDRS Part III
Posture	UPDRS Part III
Rest-tremor constancy	UPDRS Part III
Staying asleep	PDSS-2
Nocturia	PDSS-2
Morning tired	PDSS-2

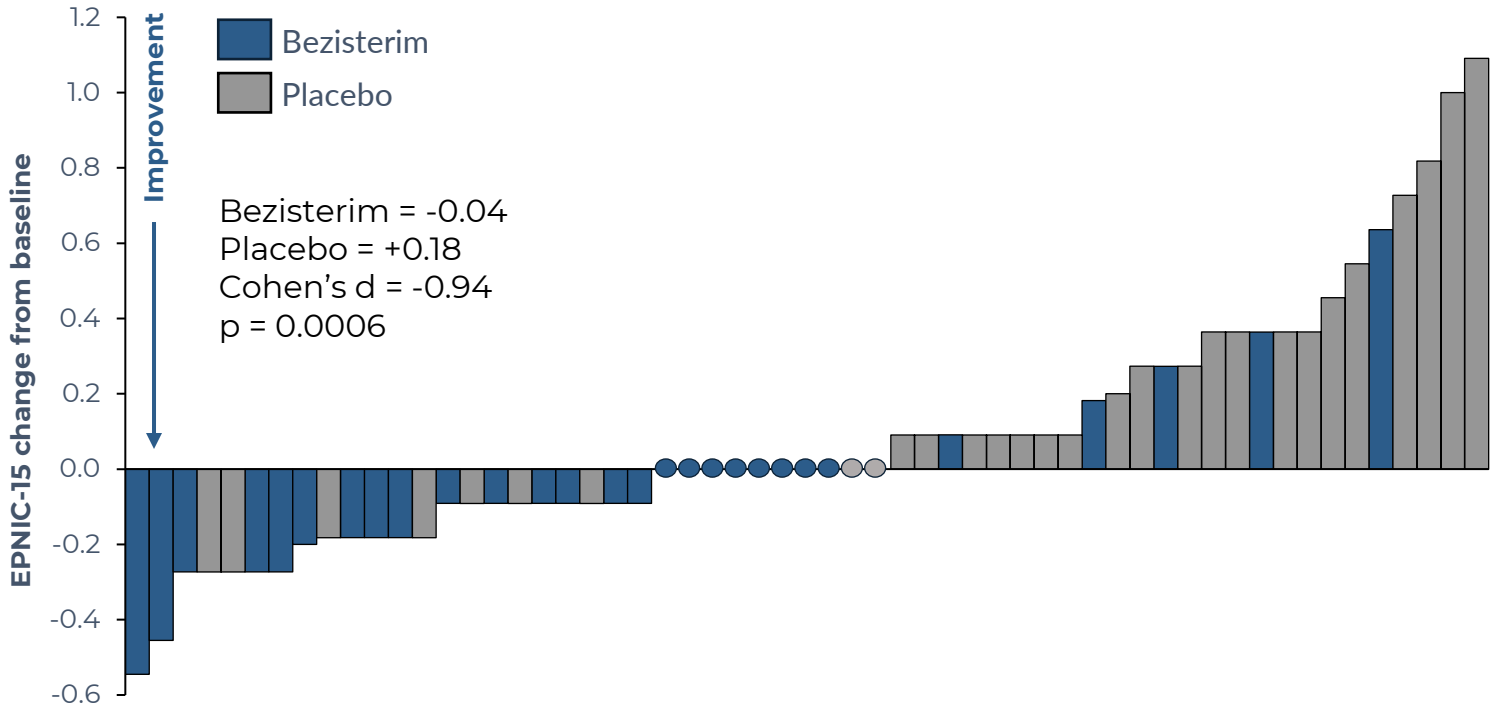
* The Movement Disorders Society's Unified Parkinson's Disease Rating Scale (MDS-UPDRS). Part I focuses non-motor symptoms; Part II on activities of daily living; and Part III on motor. Parkinson's Disease Sleep Scale (PDSS-2)

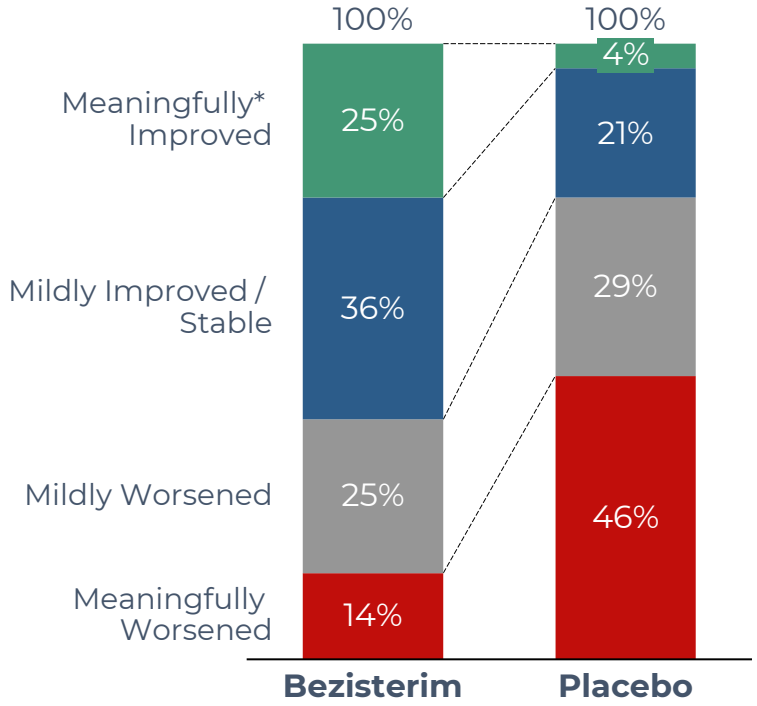
** L'Italien et al. *Neurology and Therapy* 2025;14:1609–1625; L'Italien et al. *Parkinsonism and Related Disorders* 2025;141:108077.

COMPOSITE CLINICAL OUTCOMES MEASURE

Majority of treated patients improved on EPNIC-15 while placebo moved in the opposite direction

EPNIC-15 change from baseline for individual subjects





* Meaningful is ≥ 0.5 SD from mean change

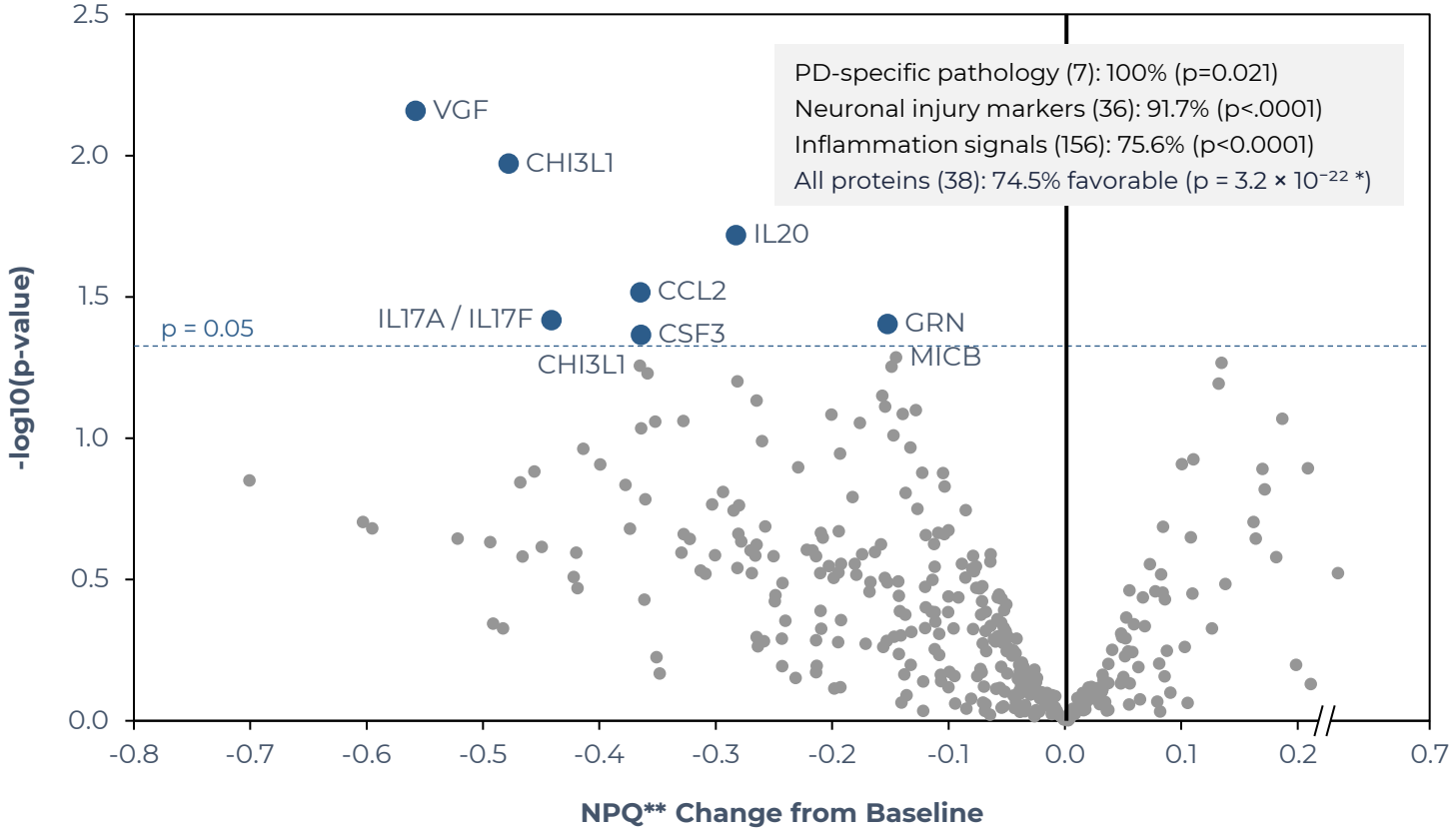
* Based on industry standard created by Norman, Sloan, and Wyrwich (2003) defining minimally important difference as a change that is 0.5 standard deviations (0.5 SD) from the mean. Thus, "Meaningful Improved" and "Meaningfully Worsened" account for those subject who improved or worsened more than 0.5 SD from the mean change

PROTEOME-WIDE DIRECTIONALITY

283 / 380 (74.5%) of proteins moved in the direction that has been shown to slow disease progression

Proteome volcano

Drug effect vs significance for every target



Top biomarker effects

Biomarker	Panel	ΔNPQ**	Cohen's d	p
VGF	CNS	-0.5580	-0.7730	0.0070
CHI3L1	CNS	-0.4782	-0.7294	0.0107
IL20	Inflam	-0.2826	-0.6629	0.0192
CCL2	CNS	-0.3649	-0.6271	0.0305
IL17A / IL17F	Inflam	-0.4413	-0.5915	0.0382
GRN	Inflam	-0.1526	-0.5805	0.0395
CSF3	Inflam	-0.3643	-0.5728	0.0432
Aβ42	CNS	-0.3519	-0.4773	0.0877
pTau-217	CNS	-0.2292	-0.4263	0.1272

* Binomial p = 3.2 × 10^{-22} assumes proteins move independently. However, they are correlated. The metric provides a strong indication of the shift in the direction of a lower inflammatory burden

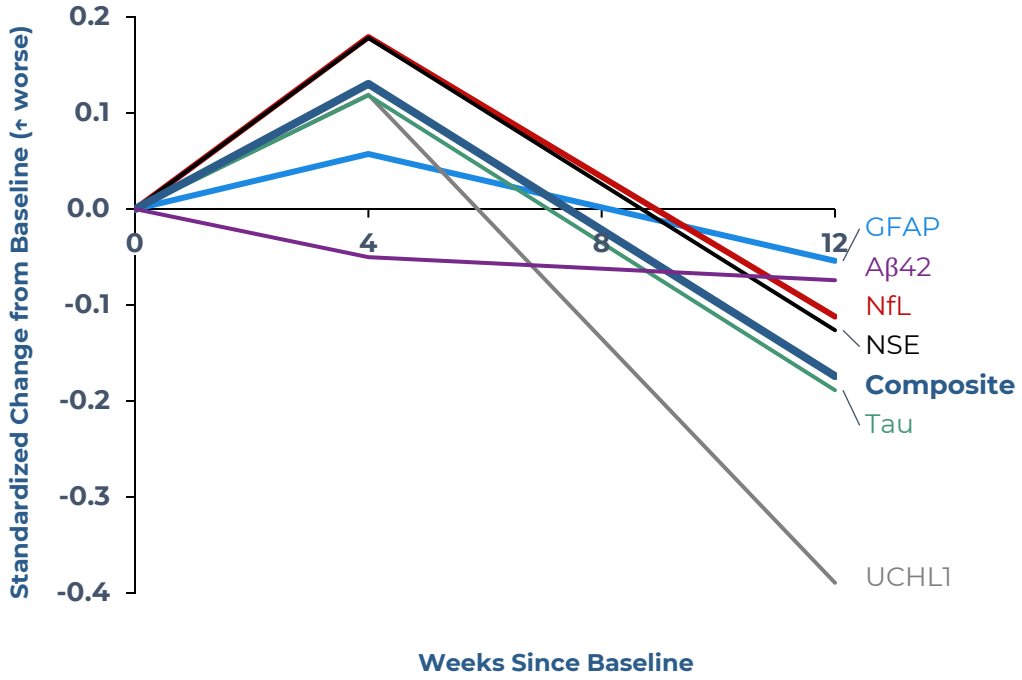
** **NPQ** = NULISA Protein Quantification, a specialized measurement unit used in proteomics data analysis from Alamar Biosciences

VGF = VGF nerve growth factor inducible protein; **CHI3L1** = chitinase-3-like protein 1 (YKL-40); **IL20** = Interleukin 20; **CCL2** = monocyte chemoattractant protein-1 (MCP-1); **IL17A/IL17F** = Interleukin 17; **GRN** = progranulin; **CSF3** = granulocyte colony-stimulating factor (G-CSF); . **Aβ42** = Beta amyloid 42; **pTau-217** = phospho-Tau 217

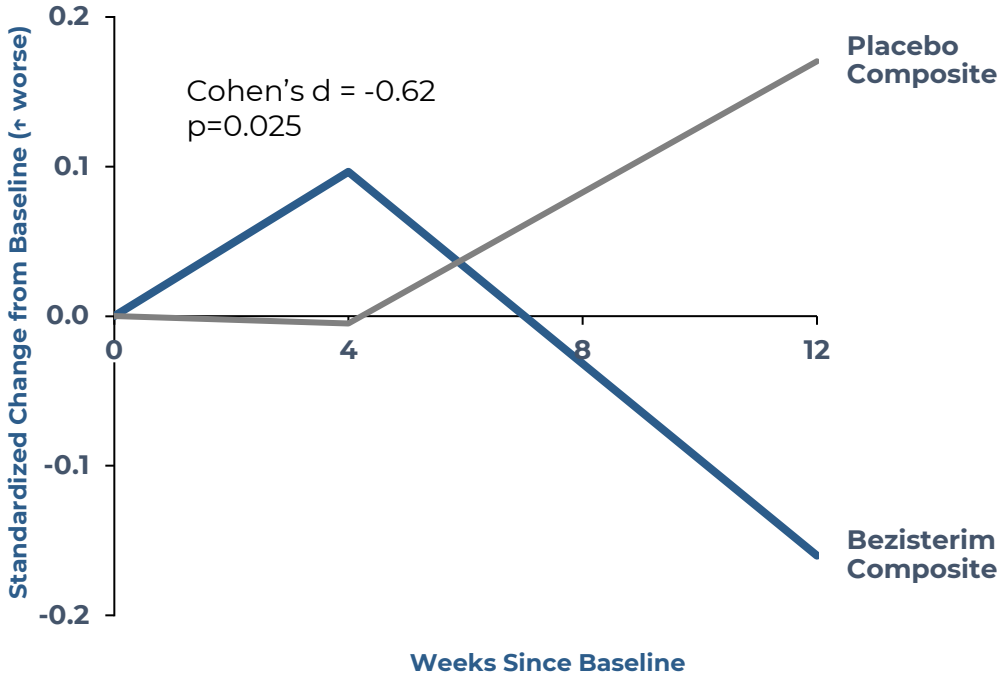
POTENTIAL IMPACT ON NEURODEGENERATION

Neuronal injury biomarkers reduced with bezisterim

Neuronal Injury Biomarkers Change from Baseline for bezisterim treated patients



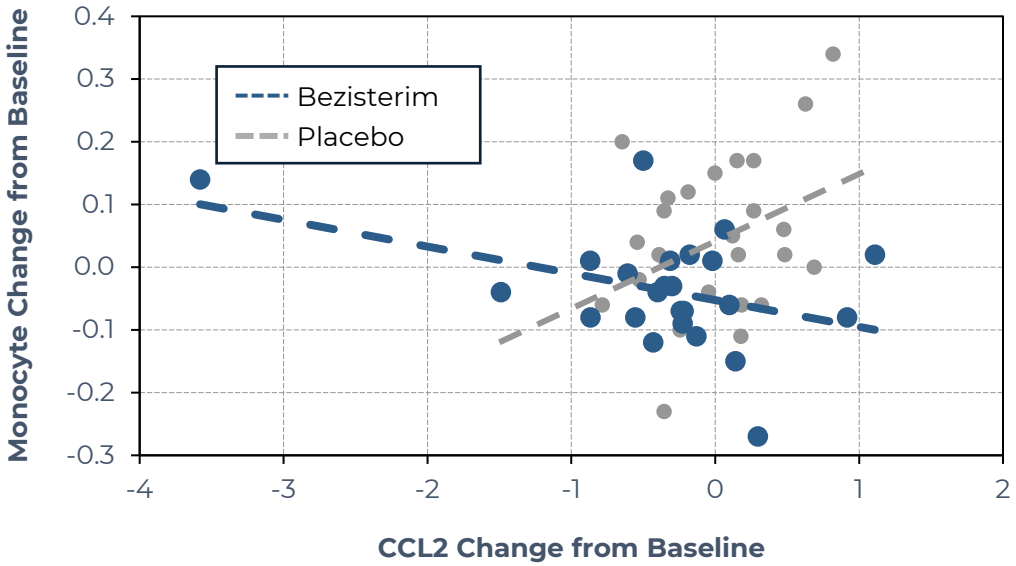
Composite Neuronal Injury Biomarkers: Bezisterim vs. Placebo



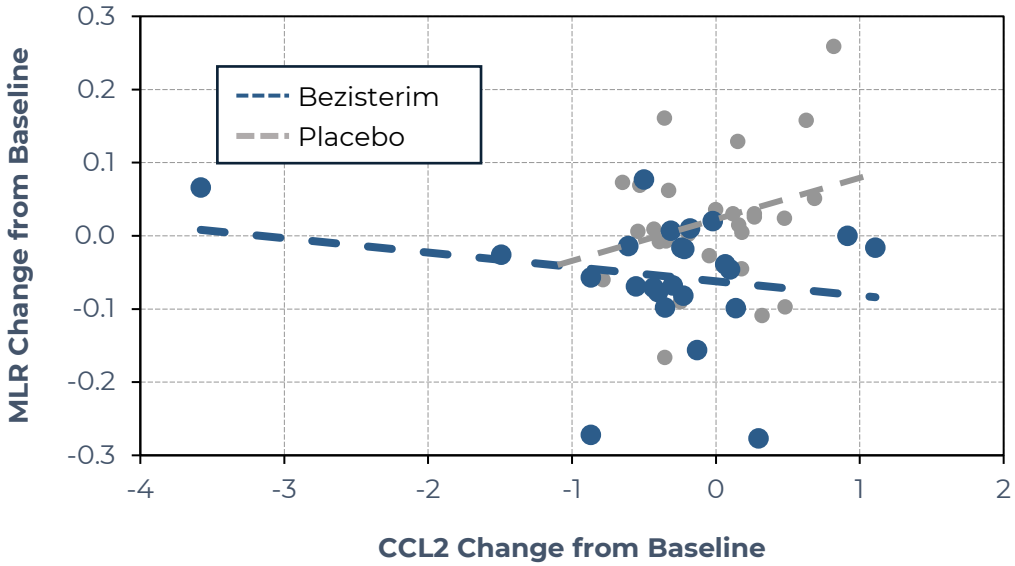
POTENTIAL MECHANISTIC EXPLANATION

Bezisterim reduces inflammatory-driven microglia activation

Bezisterim treatment → ↓ CCL2 expression → ↓ monocyte recruitment …



… resulting in lower monocyte to lymphocyte ratio (MLR)



- MLR shows the balance between the body's general inflammation (monocytes) and immune defense (lymphocytes)
- Higher MLR is associated with chronic inflammation

SAFETY & TOLERABILITY

Bezisterim safety and tolerability similar to placebo

	Bezisterim (N=28)	Placebo (N=29)
Subjects with any AE	**11 (39.3%) 19**	**15 (51.7%) 30**
Upper respiratory tract infection	5 (17.9%)	5 (17.2%)
Diarrhea	1 (3.6%)	2 (6.9%)
Headache	1 (3.6%)	1 (3.4%)
Abdominal discomfort	1 (3.6%)	0
Attention deficit hyperactivity disorder	1 (3.6%)	0
COVID-19	1 (3.6%)	0
Cough	1 (3.6%)	0
Depression	1 (3.6%)	0
Gout	1 (3.6%)	0
Musculoskeletal discomfort	1 (3.6%)	0
Uncoded	1 (3.6%)	0
Urinary tract infection	1 (3.6%)	0
Vertigo	1 (3.6%)	0
Visual impairment	1 (3.6%)	0
Back pain	0	2 (6.9%)
Fall	0	2 (6.9%)
Abdominal pain upper	0	1 (3.4%)
Arthralgia	0	1 (3.4%)
Constipation	0	1 (3.4%)
Dizziness	0	1 (3.4%)
Gastrooesophageal reflux disease	0	1 (3.4%)
Influenza	0	1 (3.4%)
Ingrowing nail	0	1 (3.4%)
Ligament sprain	0	1 (3.4%)
Menopausal symptoms	0	1 (3.4%)
Oedema peripheral	0	1 (3.4%)
Pain in extremity	0	1 (3.4%)
Presyncope	0	1 (3.4%)
Sinusitis	0	1 (3.4%)
Small airways disease	0	1 (3.4%)
Syncope	0	1 (3.4%)
Viral upper respiratory tract infection	0	1 (3.4%)

- **Overall AEs**
  - Patients with any AEs: 57.1% bezisterim vs. 58.6% placebo
  - Number of AEs: 28 bezisterim vs. 39 placebo
- **No high-severity events:** zero serious, zero severe, zero deaths.
- **Related AEs:** 1 treatment-related AE per arm; no bezisterim-related discontinuations.

* Upper respiratory tract infection

WHAT DOES IT ALL MEAN? OUR INTERPRETATION

Bezisterim has the potential to be the first of its kind treatment for Parkinson's disease

- Excellent safety profile in clinical studies to date
- Clear target engagement
  - Inflammation (CCL2, CHI3L1, MLR, SIRI, TNFα , others)
  - Proteomics: 380-target proteomic panels
  - Neurodegeneration markers (NfL, GFAP, others)
- Apparent clinical impact on both motor and non-motor symptoms
- Favorable biomarker signals for disease modification
- Clear rationale for Phase 3 trial design and endpoints
  - Both symptomatic relief and disease progression
  - Potential for enrichment strategy for Phase 3 trial

LOOKING AHEAD

- Complete and report topline data for our ADDRESS-LC Long Covid trial
- Plan for End of Phase 2 meeting with the FDA
- Plan Phase 3 potentially pivotal registrational trials
- Continue analyzing the data and preparing presentations for conferences and submitting publications to scientific journals
- Reactivate potential partnering conversations in due course

PIPELINE OVERVIEW

Multiple multi-billion-dollar US markets across the BioVie pipeline

	INDICATION	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	Annual US Sales Potential*	Catalyst
Bezisterim (formerly NE3107) Oral small molecule TNFα inhibitor	Parkinson's Disease	SUNRISE-PD · Phase 2b				$3B	August '26 Topline Data
	Long COVID	ADDRESS-LC · $13M DoW grant				$10B	3Q26 Topline Data
	Alzheimer's Disease					$30B	Phase 3 Pending
BIV201 (Continuous IV terlipressin)	Ascites					$1.6B	FDA Design Agreed

* Company estimates


biovie
Thank You