BioVie Inc. (BIVI) / March 18, 2025

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Healthcare

NASDAQ: BIVI

Buy
Initiation of Coverage

Current Price
$1.01

Target Price
$6

Market Capitalization
$20.3M

Shares Outstanding
18.5M

Float
16.0M

Institutional Holdings
4.1%

12-month Low/High
$0.99 / $7.50

Average 90-day Volume
734,308.8

Fiscal Year End
June 30

Revenues ($M)			
Period	FY2024	FY2025	FY2026
Year	**$0**	**$0**	**$0**

EPS			
Period	FY2024	FY2025	FY2026
Year	**$(7.30)**	**$(1.34)**	**$(1.63)**

BioVie Inc. (NASDAQ : BIVI)

Initiate Coverage with a BUY Rating and $6 Price Target

- **Conclusion –** We initiate coverage of BioVie Inc. (BIVI) with a BUY rating and a DCF-derived price target of $6 per share. BIVI's lead asset, Bezisterim (NE3107), is currently undergoing testing for the treatment of Early Parkinson's Disease (PD) and Long-COVID. The drug also has been studied in Alzheimer's disease (AD) and theoretically implicated in longevity therapeutics. Our price target reflects the present value of future cash flows from Bezisterim in Early PD. The drug offers a new approach targeting neuroinflammation in PD patients that has been shown potentially to complement current dopamine replacement therapies and to serve as a single-agent therapy in early disease stages prior to dopaminergic intervention.
- **Bezisterim: A New Option in PD** – Bezisterim is being developed as a multi-faceted, anti-neuroinflammatory treatment for neurodegenerative diseases. The compound primarily inhibits ERK1/2 signaling, leading to downregulation of NF-κB-mediated inflammatory pathways and reduced expression of pro-inflammatory cytokines such as TNF-α and IL-6. This orally available small molecule has demonstrated a favorable safety and tolerability profile across multiple studies. Notably, Bezisterim does not interfere with traditional dopamine replacement therapies when administered adjunctively in PD patients. Preclinical and clinical observations also suggest potential as a single-agent therapy, with patients exhibiting clinical benefit outside the expected therapeutic window for dopaminergic treatments.
- **AD Trial Complications Do Not Preclude PD Benefit** – In 2023, BIVI's Phase 3 Alzheimer's disease (AD) trial faced significant setbacks after protocol and GCP violations at several sites led to the exclusion of 358 of 439 patients from the primary analysis. Third-party audits also detected Bezisterim in placebo patients and at baseline, suggesting improper dosing. Despite these issues, topline trends from the remaining patients favored Bezisterim, but failed to reach significance. The impacted sites were referred to the FDA's Office of Scientific Investigations (OSI), though the review remains pending. We anticipate these trial complications are isolated to the AD program and do not impact the integrity or opportunity of BIVI's ongoing PD development efforts.
- **Price Target and Model Assumptions** – Our $6 PT is derived from a discounted cash flow (DCF) analysis of projected revenues from Bezisterim in Early PD patients (diagnosed last 18 months). Key assumptions include a 15% discount rate to reflect clinical and regulatory risk, a $40,000 list price with a 45% gross-to-net adjustment, and gradual market penetration, reaching 10% of eligible patients by 2035. We forecast initial revenues in Early PD beginning in FY2031 at ~$126 million, with peak revenues of ~$839 million in 2035. As of December 31, 2024, BIVI reported approximately $24.4 million in CoH, providing runway through EoY 2025 and enabling completion of its ongoing Phase 2 PD trial.

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Refer to Last Page for Analyst Certification & Important Disclosures

Company Description

BioVie Inc. (BIVI) is a clinical-stage biopharmaceutical company developing therapies for neurodegenerative and liver diseases. Its lead candidate, Bezisterim (formerly NE3107, HE3286), is an orally administered ERK1/2 suppressor targeting neuroinflammation and insulin resistance, with applications in Parkinson's disease (PD), Alzheimer's disease (AD), Long-COVID, and Longevity. The company is conducting the Phase 2 SUNRISE-PD trial (NCT06757010) in early PD, a Department of Defense-funded Phase 2 Long-COVID study (NCT06847191), and plans to initiate a Phase 3 AD trial by late 2025.

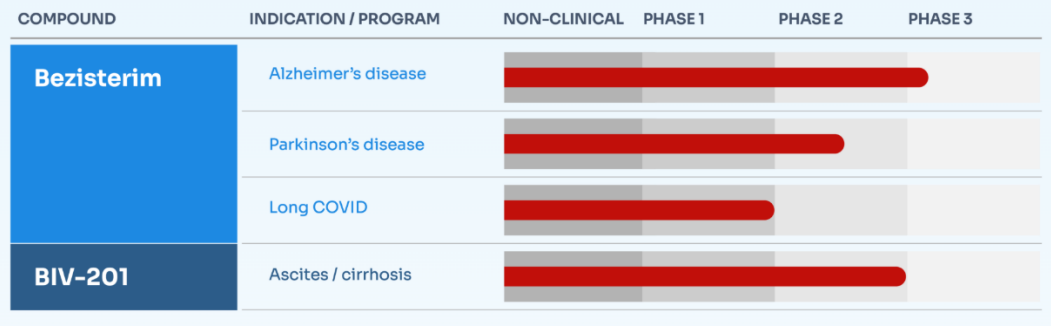
BIVI is also advancing BIV201, a continuous infusion of liquid terlipressin for ascites in liver cirrhosis patients with acute kidney injury. The company is seeking a partner for Phase 3 trials, positioning BIV201 as a potential first FDA-approved treatment for ascites. BIVI is incorporated in Nevada and headquartered in Carson City, NV.

Investment Summary

We initiate coverage of BioVie Inc. (BIVI) with a Buy rating and a DCF-derived $6 price target, reflecting projected cash flows from Bezisterim in Parkinson's disease (PD) ($5/share) and the company's current cash balance ($1/share). Unlike traditional PD therapies that focus on increasing dopamine levels, Bezisterim targets neuroinflammation, a key driver of dopaminergic neuron loss, and can be used alongside existing treatments. We apply a 15% discount rate to account for ongoing clinical development risk. We estimate BIVI will require ~$140MM to reach commercialization in 2031, with peak annual sales of ~$784MM in 2035.

Given the company's current cash position, our model does not incorporate costs or potential revenue from its Alzheimer's disease (AD) program. Initiating a Phase 3 AD trial depends on the company's ability to raise funds to complete it (est. $30-50MM). Outside of a significant partnership or out-licensing of portions of BIVI's pipeline to foreign markets, which is actively being pursued, the outcome of BIVI's ongoing SUNSHINE-PD Phase 2 study will significantly influence the company's ability to generate capital for this program. Additionally, we exclude Long-COVID, Longevity, and BIV201 from our valuation due to their early-stage development and uncertain commercialization paths, instead considering them potential upside opportunities.

BIVI Pipeline



Source: BioVie Inc. (CoA 3/1/2025)

Discounted Cash Flow Analysis Yields Fair Value of $6/share

We derive our $6 price target using a discounted cash flow analysis of Bezisterim for early Parkinson's disease (<18 months since diagnosis) in the U.S. market ($5/share), plus BIVI's current cash balance of ~$24MM ($1/share). The company has ~9.6M outstanding and exercisable warrants with a weighted average exercise price (WAEP) of $3.50, representing ~$33.6MM in potential capital if exercised. However, we exclude these warrants and proceeds from our valuation, as their exercise would push a portion of them out of the money (OOTM).

BIVI Price Target Buildup

	Rate	$MM	Per Share*
Bezisterim - Total	15%	$88	$5
Cash (Net)		$24	$1
	Total	**$113**	**$6**

*Per Share Count Assumes 18.45MM fully diluted shares
Source: Brookline Capital Markets estimates

In the US, PD incidence is expected to rise due to growth in the population of people aged 65 or older. Recent data published on the incidence of PD[1] suggests an incidence range of 108-212 per 100,000 people age 65+. We have elected to use a median incidence rate of 160 per 100,000 people age 65+. Our model also accounts for a growing number of people aged 65+ over the next 15 years by using estimates from the Congressional Budget Office (CBO), which estimates a 2% annual increase in this demographic. We view these estimates as conservative as we exclude PD patients <65.

As we do not know the treatment discontinuation rate for Bezisterim, we have assumed a five-year dropout rate for patients who receive the drug, starting with its commercialization in CY2030. Our model incorporates a somewhat aggressive 10% market penetration, supported by the potential use of Bezisterim as both a single agent and in combination with existing PD medications. This estimate is also consistent with BIVI's projections for the compound.

Parkinson's TAM for Bezistermin

Calendar Year	2030	2035	2040
PD Incidence 65+	111,170	118,930	124,122
Treated Patients	85%		
Market Penetration	10%		
Cumulative Patients Eligible for Treatment	165,642	563,860	591,332
Patients Treated with Bezisterim	2,111	45,817	50,263

Source: Brookline Capital Markets Research, CBO, BCBS.com, Willis, et al., Parkinsons Dis.,2022

For pricing, we have elected to use a list price of ~$40,000 with a gross-to-net adjustment of 55%, providing an annual revenue per patient of $18,000. This is arguably conservative, given the company estimates ~revenue per patient of $30,000. However, this gives BIVI flexibility when providing discounts and obtaining market penetration early in development. A sensitivity table below demonstrates the annual revenue/patient with different list prices and gross-to-net adjustments.

Bezisterim Annual Rev/Patient Sensitivity Table

List Price	Gross-to-Net Adjustment 60%	55%	50%	45%	40%	35%
$10,000	$4,000	$4,500	$5,000	$5,500	$6,000	$6,500
$20,000	$8,000	$9,000	$10,000	$11,000	$12,000	$13,000
$30,000	$12,000	$13,500	$15,000	$16,500	$18,000	$19,500
$40,000	$16,000	**$18,000**	$20,000	$22,000	$24,000	$26,000
$50,000	$20,000	$22,500	$25,000	$27,500	$30,000	$32,500
$60,000	$24,000	$27,000	$30,000	$33,000	$36,000	$39,000
$70,000	$28,000	$31,500	$35,000	$38,500	$42,000	$45,500
$80,000	$32,000	$36,000	$40,000	$44,000	$48,000	$52,000

Source: Brookline Capital Markets Estimates

We have incorporated a standard five-year launch curve into our model for PD commercialization. We estimate the first significant revenues of ~$126M in FY2031, with peak revenues of ~$839MM in 2035.

Launch Curve: Percentage of Peak Revenues

Year	1	2	3	4	5
% of Peak	15%	42%	68%	86%	100%

Data assume a five-year ramp: other data available

Source: Brookline Capital Markets Estimates

Gross Margin – We assume a gross margin of 85% at maturity for Bezisterim in both indications, which is comparable to other small molecules.

OpEx – For R&D, we forecast BIVI to perform two Phase 3 trials in PD. Once commercialization occurs in FY2031, we begin to taper down R&D spending to 13% of total revenues. For SG&A, we increase costs in the year preceding commercialization, assuming sales force build-up. We forecast a sales force of 70. We also assume SG&A costs of 34% of revenue when sales for a product exceed $400M (2033).

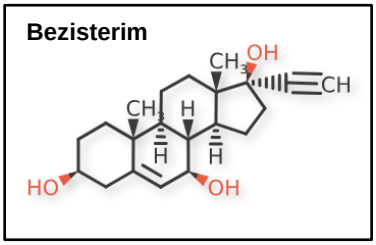
Tax Rate – 27%

Patent Expirations: BIVI currently has patents pending that would protect the use of Bezisterim in neurodegenerative conditions (US20240108636A1 and WO2024054452A1). If issued, protection would be conferred to ~2040. We anticipate these patents will be granted; therefore, we have elected not to model erosion effects for Bezisterim.

Discount Rate – We have used a 15% discount rate to reflect the risk associated with clinical trial development, FDA approval, and market acceptance of Bezisterim.

Bezisterim Overview

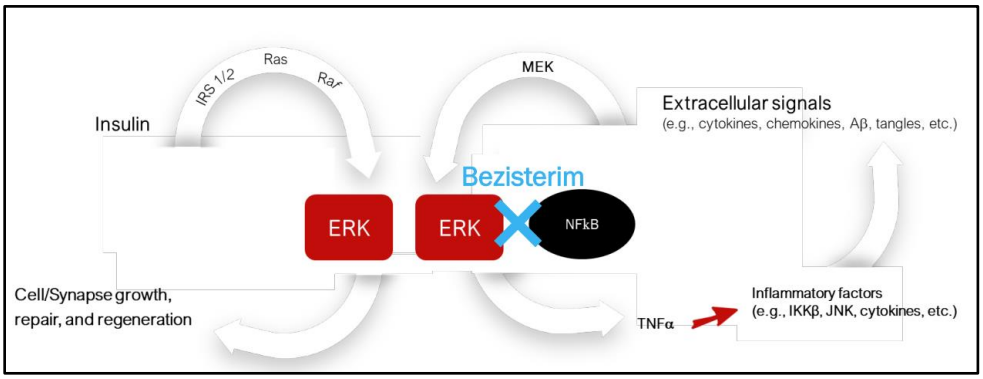
Mechanism Of Action - Bezisterim (fmr. NE3107, HE3286, and Triolex) is an orally available, blood-brain barrier-permeating small molecule with anti-inflammatory and insulin-sensitizing effects. The drug is a modified derivative of β-androstenetriol, a metabolite of Dehydroepiandrosterone (DHEA). It was modified to improve oral bioavailability and chemical stability while eliminating steroid hormone activity[1].



SOURCE: https://drugs.ncats.io/

The drug primarily inhibits inflammation by binding and selectively inhibiting extracellular signal–regulated kinases (ERK1/2). Suppressing ERK1/2 downregulates pathogenic NF-κB activity (TNF-α, IL-6, etc.), which is commonly upregulated in inflammatory or neurodegenerative indications.

Notably, Bezisterim does not activate glucocorticoid and other steroid receptors and maintains normal functions of these pathways outside of a pathogenic state[2]. The drug has also been found to partially bind to other factors associated with metabolism and inflammation including low-density lipoprotein receptor-related protein (Lrp1), protein kinases involved in inflammation signaling pathways (MAPK1,3), ribosomal s6 kinase (RSKs), and sirtuin-2[3]. Collectively, the compound acts as a partial inhibitor of NF-κB and modulator of MAPK/ERK, uncoupling inflammation and insulin resistance without broad immunosuppression.



Source: BioVie Inc.

Development History - Bezisterim originated at Hollis-Eden Pharmaceuticals in San Diego, which gave it the code HE3286. In 2010, Hollis-Eden rebranded as Harbor BioSciences and continued developing HE3286 (Triolex) as a next-generation anti-inflammatory steroid analog[4]. From 2008 to 2011, Harbor BioSciences sponsored multiple early trials of HE3286 in metabolic and inflammatory conditions – including impaired glucose tolerance (NCT00991107), type 2 diabetes (NCT00694057), rheumatoid arthritis (NCT00712114), obesity (NCT00555451), and ulcerative colitis (NCT00628433).

These Phase 1 and Phase 2 studies established a safety profile and suggested some efficacy, specifically demonstrating insulin-sensitizing and anti-inflammatory effects in humans[5]. Pharmacokinetics were dose-proportional (2–100 mg), and no serious adverse events were attributed to the drug[1]. However, Harbor Biosciences failed to generate enough funding to advance the drug into Phase 3 trials and liquidated or abandoned most of its assets in 2012.

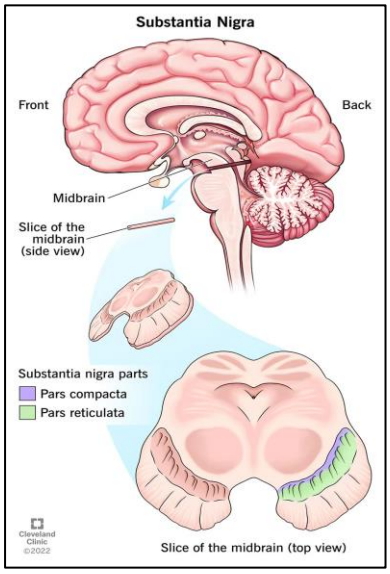
In the following years, the private company NeurMedix, Inc. acquired HE3286 and refocused the compound for neurodegenerative diseases based on emerging links between inflammation, insulin resistance, and neurodegeneration. It also renamed the compound NE3107. In February 2021, NeurMedix secured FDA authorization for a Phase 3 trial in Alzheimer's disease (AD) with NE3107[6].

In June 2021, BioVie Inc. (BIVI) announced the acquisition of NeurMedix's biopharmaceutical assets—including NE3107/HE3286 (Bezisterim)—in a stock and cash transaction[7]. BIVI has maintained the NE3107 code but also uses Bezisterim as an official generic name.

1 - https://www.alzforum.org/therapeutics/bezisterim
2 - Reading CL, Ahlem CN, Murphy MF. NM101 Phase III study of NE3107 in Alzheimer's disease: rationale, design and therapeutic modulation of neuroinflammation and insulin resistance. Neurodegener Dis Manag. 2021 Aug;11(4):289-298. doi: 10.2217/nmt-2021-0022. Epub 2021 Jul 12. PMID: 34251287.
3 - Lu M, Patsouris D, Li P, Flores-Riveros J, Frincke JM, Watkins S, Schenk S, Olefsky JM. A new antidiabetic compound attenuates inflammation and insulin resistance in Zucker diabetic fatty rats. Am J Physiol Endocrinol Metab. 2010 May;298(5):E1036-48. doi: 10.1152/ajpendo.00668.2009. Epub 2010 Feb 16. PMID: 20159859; PMCID: PMC2867370.
4 - https://www.marketscreener.com/quote/stock/HOLLIS-EDEN-PHARM-9537/news/Hollis-eden-Pharma-Hollis-Eden-Pharmaceuticals-Announces-Filing-of-IND-for-Phase-I-II-Clinical-Tri-415143/
5 - https://www.alzdiscovery.org/uploads/cognitive_vitality_media/NE3107_(HE3286).pdf
6 - https://www.globenewswire.com/news-release/2021/02/02/2168201/0/en/NeurMedix-Obtains-FDA-Authorization-for-Pivotal-Phase-3-Alzheimer-s-Trial.html
7 - https://www.globenewswire.com/news-release/2021/04/27/2218099/0/en/BioVie-Acquires-Biopharma-Assets-from-Privately-Held-NeurMedix.html

Parkinson's Disease

Parkinson's Disease (PD) is a chronic neurodegenerative disorder affecting motor coordination and cognition. Its causes are varied, with only 3-5% of cases attributed to genetic factors[1]. Though often associated with tremors, clinical diagnosis primarily relies on bradykinesia (slowness of movement or progressive halting), with patients subsequently presenting with tremors and/or rigidity.



Anatomy of the Substantia Nigra
Source: clevelandclinic.org

PD is most commonly diagnosed in individuals over 65, though a subset known as "young-onset PD" affects those under 50. The disease is more prevalent in men, but women, despite being less likely to experience cognitive decline, are more affected by bradykinesia[1]. Average life expectancy post-diagnosis ranges from 7 to 15 years.

Lewy Bodies and Alpha-Synuclein (αSyn) - PD results from both genetic and environmental influences. Approximately 15% of patients have a first-degree relative with the disease, and 5-10% carry an identifiable mutation[2]. Environmental risk factors include chemical exposures, with higher incidence observed among farmers working with herbicides.

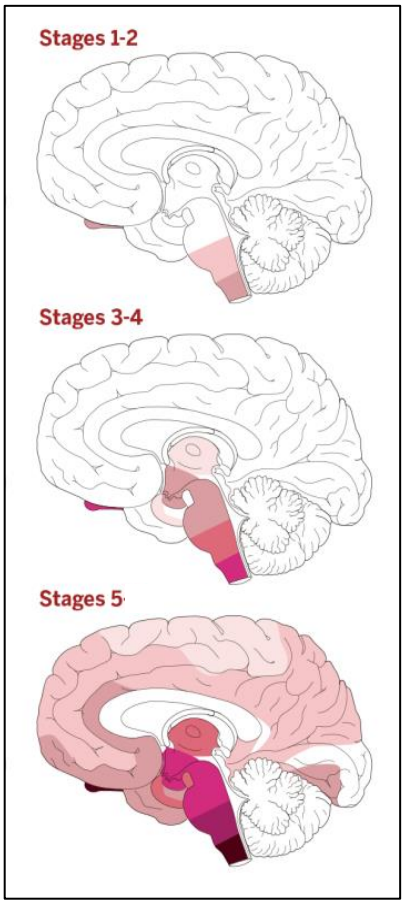


Diagram of Braak's Staging of αSyn in PD
Modified from: Goedert, *Science,* 2015

Regardless of its cause, PD's pathophysiology is characterized by the degeneration of dopamine-secreting neurons in the substantia nigra. Dopamine regulates memory, movement, motivation, mood, and attention. The neurons degenerate due to alpha-synuclein (αSyn) misfolding, leading to Lewy Neurites forming that aggregate into Lewy Bodies. PD progresses in distinct stages, clinically classified using Braak Staging[3].

Early stages often go undiagnosed due to mild or negligible symptoms. In Stage 3, Lewy Body formation and substantia nigra degeneration become evident. As the disease advances, neuronal loss extends to the middle and outer brain regions, with Lewy Body deposits spreading from the brainstem. In late-stage PD (Stage 5), patients are typically bedridden and require full-time care. Mortality often results from secondary complications such as pneumonia or falls due to declining motor coordination and respiratory function.

1 - Bloem BR, Okun MS, Klein C. Parkinson's disease. Lancet. 2021 Jun 12;397(10291):2284-2303. doi: 10.1016/S0140-6736(21)00218-X. Epub 2021 Apr 10. PMID: 33848468.
2 - Samii A, Nutt JG, Ransom BR. Parkinson's disease. Lancet. 2004 May 29;363(9423):1783-93. doi: 10.1016/S0140-6736(04)16305-8. PMID: 15172778.
3 - Braak H, Del Tredici K, Rüb U, de Vos RA, Jansen Steur EN, Braak E. Staging of brain pathology related to sporadic Parkinson's disease. Neurobiol Aging. 2003 Mar-Apr;24(2):197-211. doi: 10.1016/s0197-4580(02)00065-9. PMID: 12498954.

Current Treatments - Current treatments for PD focus on symptomatic relief, as no cure exists. Most therapies aim to modify neurotransmitter levels in the brain or slow neuron death to extend a patient's health span.

Treatment efficacy can fluctuate in patients receiving therapy for over two years. When medications are effective, patients are in an "on period," while loss of effectiveness is referred to as an "off period." The table below summarizes the current treatments available for PD patients.

Current AntiParkinson Drugs

Category	Drug Names	Brand Names	MoA	Adverse Effects
Dopamine Precursors	Levodopa + Carbidopa	Atamet, Dhivy, Duopa, Parcopa, Rytary, SINEMET, SINEMET CR	Increases levels of precursor dopamine in the brain	Nausea, Vomiting, Delirium, Motor Dysfunction
NMDA-Receptor Agonist	Amantadine	GOCOVRI, Osmolex ER, Symmetrel	Releases stored or prevents the storage of dopamine	Anticholinergic-like (Fever, Dry Mouth, Blurred Vision, Constipation, Confusion)
Dopamine Agonists	Pramipexole, Ropinirole, Rotigotine, Apomorphine	Mirapex, Mirapex ER, Requip, Requip XL, Neupro, Apokyn, KYNMOBI	Partially or Fully Stimulate Various Dompamine Receptors	Impulse Control Issues, Sleepiness, Dyskinesia, Blood Pressure Complications
MAO-B Inhibitors	Selegiline, Rasagiline	Carbex, Eldepryl, EMSAM, Zelapar, Azilect	Decreases Dopamine Metabolism	Amphetamine-like metabolite complications
Anticholinergic Drugs	Benztropine, Trihexyphenidyl, Amitriptyline, various antihitamines	Cogentin, Artane, Elavil, Tryptanol, Vanatrip	Inhibits Acetylcholine-induced Tremor	Fever, Dry Mouth, Blurred Vision, Constipation, Confusion, possible Tauopathy/Neurodegeneration catalyst
COMT Inhibitors	Entacapone, Tolcapone, Opicapone	Comtan, Tasmar, Ongentys	Decreases Dopamine Metabolism	Back pain, diarrhea, liver toxicity (tolcapone)

Source: merckmanuals.com

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Levodopa & Carbidopa - Levodopa is the most common first-line treatment for PD. This dopamine precursor crosses the blood-brain barrier (BBB) and is decarboxylated into dopamine. Since dopamine itself cannot cross the BBB, levodopa is prescribed with carbidopa, a decarboxylation inhibitor, to prevent its conversion outside the brain and increase central availability. Multiple formulations exist, including oral and inhalation options, as well as immediate-release and extended-release variations to modulate treatment response.

Short-term side effects of levodopa include nausea and vomiting, while long-term use can lead to complications such as dyskinesia (motor dysfunction), psychosis, and delirium. Different formulations are also being developed to improve treatment efficacy during on/off periods[1].

Amantadine (NMDA-Receptor Antagonist) - Amantadine (GOCOVRI, Osmolex ER, Symmetrel) is used as monotherapy or alongside levodopa/carbidopa. This oral agent is a weak, uncompetitive NMDA receptor antagonist and is thought to influence neurotransmitter storage, increasing dopamine availability. While less effective than levodopa, it is commonly used to manage levodopa-induced dyskinesia or as an adjunct during off periods[1].

Dopamine Receptor Agonists - Dopamine receptor agonists mimic dopamine by binding to its receptors. These agents have varying affinities for D1, D2, and D3 receptors, with D3 being a primary target for improving PD symptoms. D4 and D5 receptors are generally not targeted, except in apomorphine treatments, which act on a broader receptor spectrum, including D1-D5[2].

Pramipexole (Mirapex) and ropinirole (Requip) are oral agonists, while rotigotine (Neupro) is available as a transdermal patch. Apomorphine, used for severe off periods, is administered via injection (Apokyn) or sublingually (Kynmobi). Side effects of dopamine agonists include impulse control disorders, predisposing patients to compulsive gambling, shopping, hypersexuality, or overeating[1].

Monoamine Oxidase MAO-B Inhibitors - MAO-B enzymes degrade dopamine in the brain. Inhibitors like selegiline (Carbex, Eldepryl, Emsam, Zelapar) and rasagiline (Azilect) selectively block MAO-B, increasing dopamine levels. These drugs are often used in early PD to delay the need for levodopa, extending treatment efficacy. While generally well tolerated, dual treatment with levodopa can exacerbate dyskinesia, requiring careful dose management[3]. Additionally, selegiline metabolism produces amphetamine-like derivatives, whereas rasagiline does not, reducing the risk of stimulant-related side effects[4].

Anticholinergic Drugs - Anticholinergic agents are prescribed to PD patients with severe tremors to inhibit acetylcholine, a neurotransmitter involved in muscle contractions. Muscarinic acetylcholine receptors are believed to contribute to PD tremor symptoms. Common side effects include dry mouth, blurred vision, constipation, and confusion. Due to potential cognitive risks, their use is limited in older populations[5].

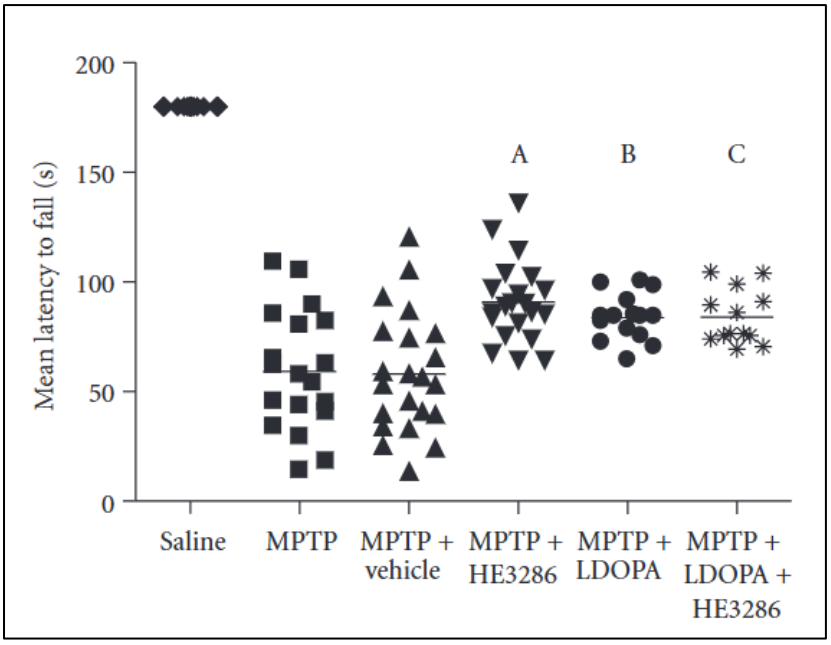
Catechol O-methyltransferase (COMT) inhibitors - COMT enzymes degrade dopamine and its metabolites. Inhibiting this process prolongs dopamine's effects, making COMT inhibitors valuable adjuncts to levodopa in patients experiencing worsening off periods. Entacapone (Comtan), tolcapone (Tasmar), and opicapone (Ongentys) are orally available COMT inhibitors administered alongside levodopa.

Tolcapone more effectively crosses the BBB than entacapone but has been associated with liver toxicity, limiting its use. Opicapone, a newer COMT inhibitor, requires once-daily dosing and does not share tolcapone's hepatic risks[6].

1 - https://www.merckmanuals.com/professional/neurologic-disorders/movement-and-cerebellar-disorders/parkinson-disease#Treatment_v1043652
2 - https://go.drugbank.com/drugs/DB00714
3 - https://www.parkinson.org/living-with-parkinsons/treatment/prescription-medications/mao-b-inhibitors#:~:text=MAO%2DB%20enzymes%20naturally%20break,%2Don)%20to%20other%20medications.
4 - Rivest J, Barclay CL, Suchowersky O. COMT inhibitors in Parkinson's disease. Can J Neurol Sci. 1999 Aug;26 Suppl 2:S34-8. doi: 10.1017/s031716710000007x. PMID: 10451758.
5 - https://www.parkinson.org/living-with-parkinsons/treatment/prescription-medications/anticholinergic-drugs
6 - Lees AJ, Ferreira J, Rascol O, et al. Opicapone as Adjunct to Levodopa Therapy in Patients With Parkinson Disease and Motor Fluctuations: A Randomized Clinical Trial. JAMA Neurol. 2017;74(2):197–206. doi:10.1001/jamaneurol.2016.4703

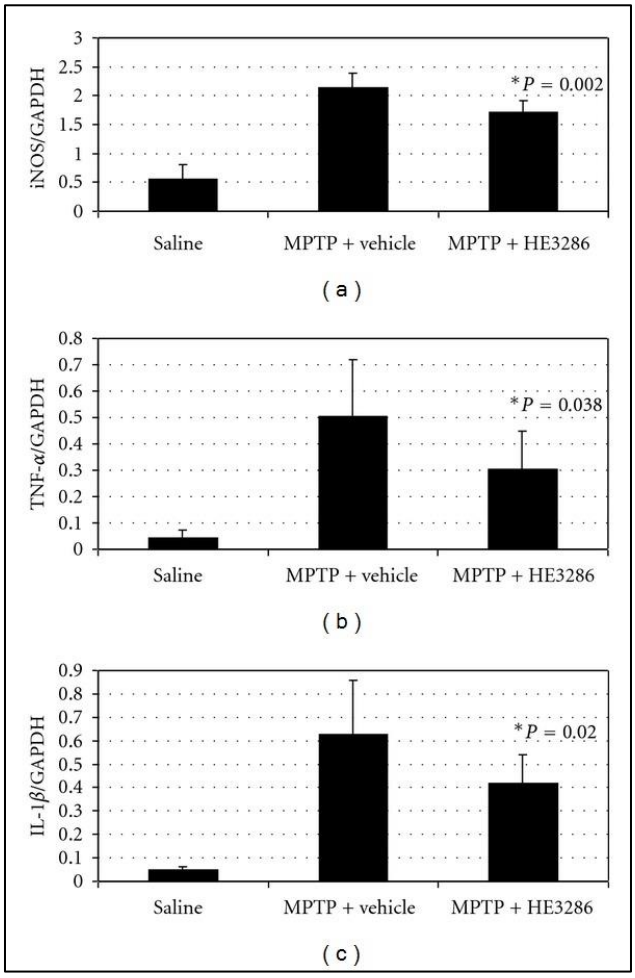
Preclinical Data Summary

MPTP PD Mouse Model - Bezisterim was first tested in a neurodegenerative mouse model in an MPTP mouse model of Parkinson's disease (PD)[1]. MPTP is a toxin that, when provided to mice, selectively damages dopaminergic neurons in the substantia nigra, mimicking PD[2]. Bezisterim treatment (40 mg/kg, oral, BID) began shortly after neurotoxin exposure and continued for 4 days. Treated mice had significantly improved motor function on rotarod tests compared to controls ($p<0.0001$) but failed to demonstrate an additive effect when paired with L-DOPA.



Rotarod test of MPTP-exposed mice treated with vehicle, Bezisterim (A; p<0.0001), L-DOPA (B; p=0.0016), and combination Bezisterim/L-DOPA (C; p=0.0031)
Source: Nicoletti F, Philippens I, Fagone P, Ahlem CN, Reading CL, Frincke JM, Auci DL. 17α-Ethynyl-androst-5-ene-3β,7β,17β-triol (HE3286) Is Neuroprotective and Reduces Motor Impairment and Neuroinflammation in a Murine MPTP Model of Parkinson's Disease. Parkinsons Dis. 2012;2012:969418. doi: 10.1155/2012/969418. Epub 2012 Sep 26. PMID: 23050197; PMCID: PMC3463191.

Neuroinflammatory markers in the brain were markedly reduced: for example, inducible nitric oxide synthase (iNOS) was 20% lower vs. control (p=0.002), TNF-α was 40% lower (p=0.038), and IL-1β was 33% lower (p=0.02). Importantly, Bezisterim-treated MPTP mice had 17% more TH+, dopaminergic neurons surviving than vehicle-treated mice (p=0.003) and 38% fewer damaged neurons (p=0.029).



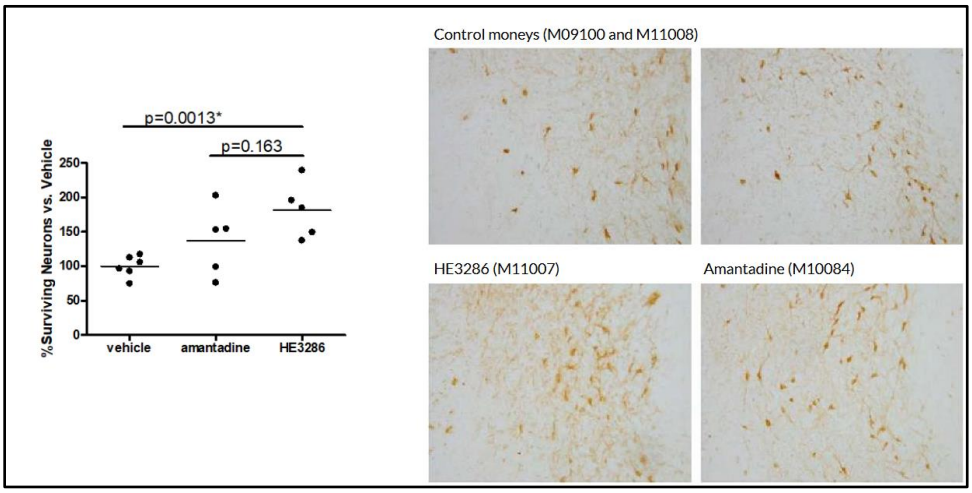
Bezisterim (HE3286) attenuated MPTP-induced expression of inflammatory cytokines by rt-PCR (GAPDH); statistics done by Mann-Whitney test

Source: Mustapha M, Mat Taib CN. MPTP-induced mouse model of Parkinson's disease: A promising direction of therapeutic strategies. Bosn J Basic Med Sci. 2021 Aug 1;21(4):422-433. doi: 10.17305/bjbms.2020.5181. PMID: 33357211; PMCID: PMC8292858.

There are limitations to the usefulness of this data, as rapidly killing dopaminergic neurons in the brain through toxin exposure is mechanistically distinct from the slowly progressing deterioration in human disease. However, despite this limitation, these data were the first to suggest that Bezisterim mitigating ERK1/2 inflammation has neuroprotective effects.

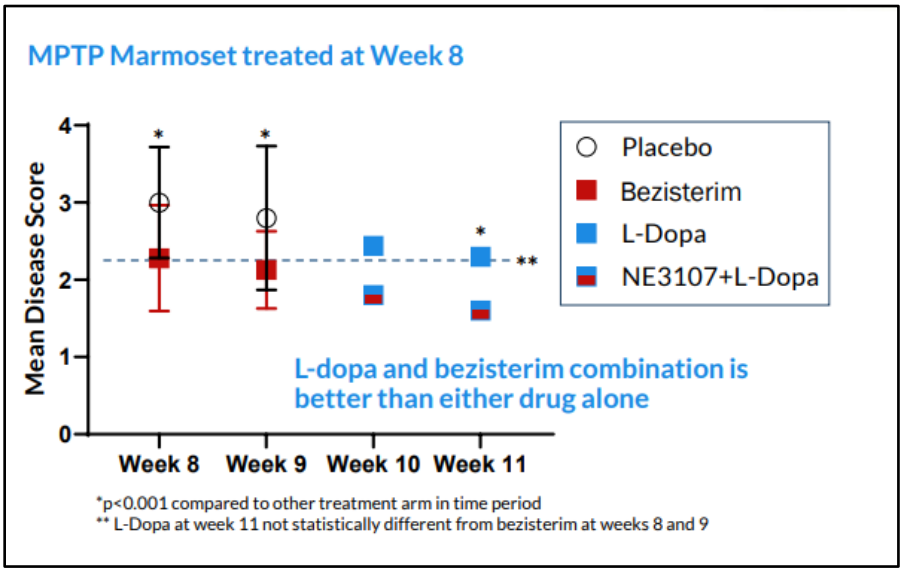
Marmoset MPTP PD Model - BIVI has run internal trials funded by the Michael J. Fox Foundation, mimicking the data found in MPTP-treated mice but using marmosets as a target species. Marmosets are considered to act as a better proxy for the human CNS, mainly due to their similar brain anatomy and neurochemistry[4].

While the data has not been formally published, released portions suggest that Bezisterim's ability to preserve dopaminergic neurons remains in this marmoset model.



Bezisterim Preservation of Dopaminergic Neurons in MPTP Marmosets / Source - BioVie Inc.

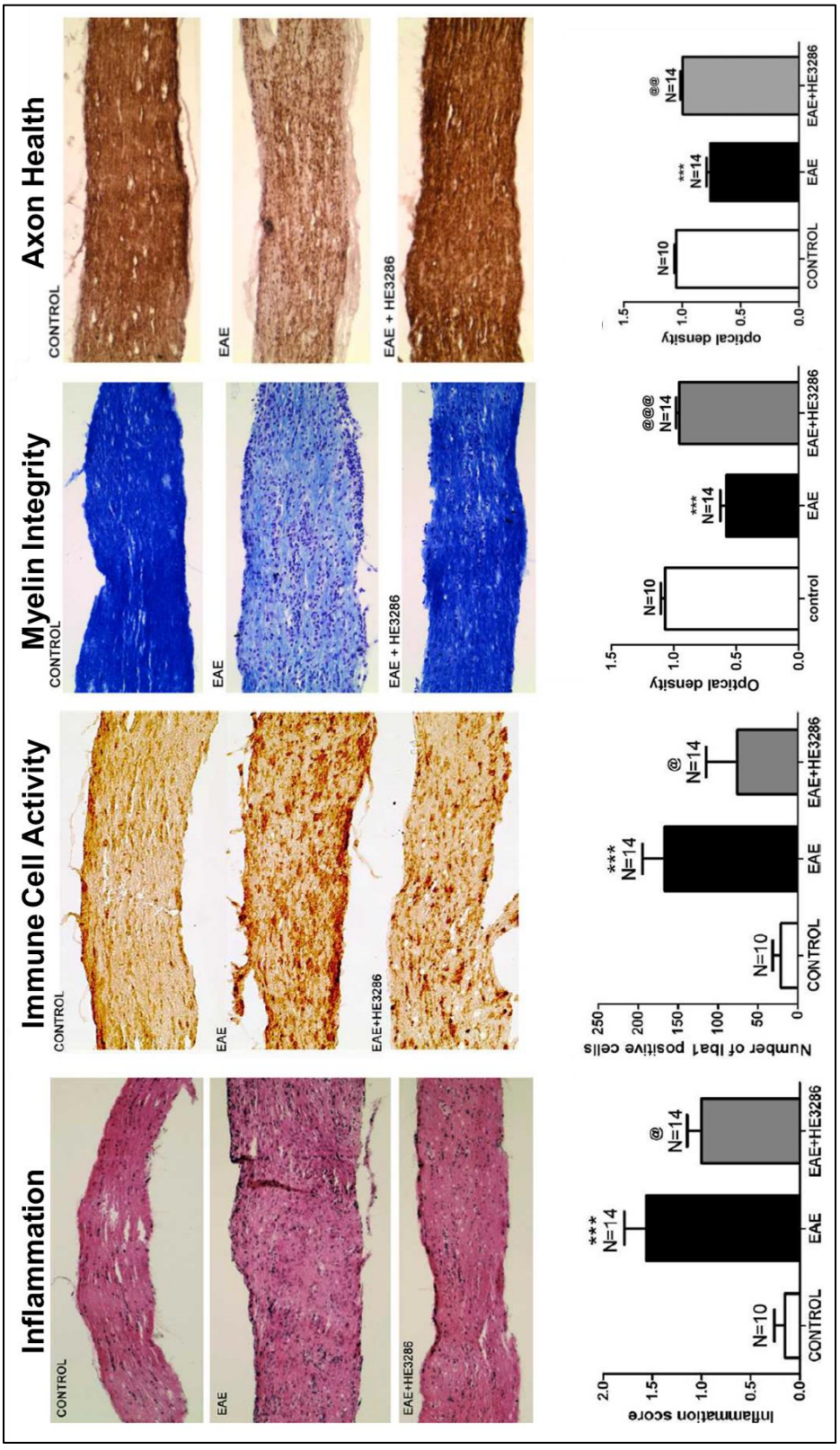
Additionally, Bezisterim treatment lowers L-Dopa-induced dyskinesia in the animals compared to placebo and monkeys treated with the anti-dyskinesia drug Amantadine. However, diverging from the mouse data, MPTP marmosets treated with Bezisterim demonstrated better coordination and balance than those treated with L-DOPA alone. This would be a key driver of acceptance into the treatment landscape for PD if Bezisterim had an additive effect on L-DOPA.



Motor Coordination Testing in MPTP Marmoset Model / Source – BioVie Inc.

Ocular Neuroinflammation – Preclinical data on Bezisterim in a neurodegenerative setting are limited outside of the MPTP PD models. However, other chronic neuroinflammation mouse models focusing on the eye have found similar benefits. In the Experimental Autoimmune Encephalomyelitis (EAE) mouse model, which is used as an analog of myelin autoimmunity (i.e., multiple sclerosis, optical neuritis), Bezisterim significantly delayed paralysis ($p<0.05$) and vision loss ($p<0.001$) compared to controls[3].

Optic nerves from Bezisterim-treated animals had significantly lower immune cell activity and corresponding inflammation. This resulted in a higher amount of myelin surrounding the optic nerve, preserving axon health and activity compared to vehicle-treated animals.



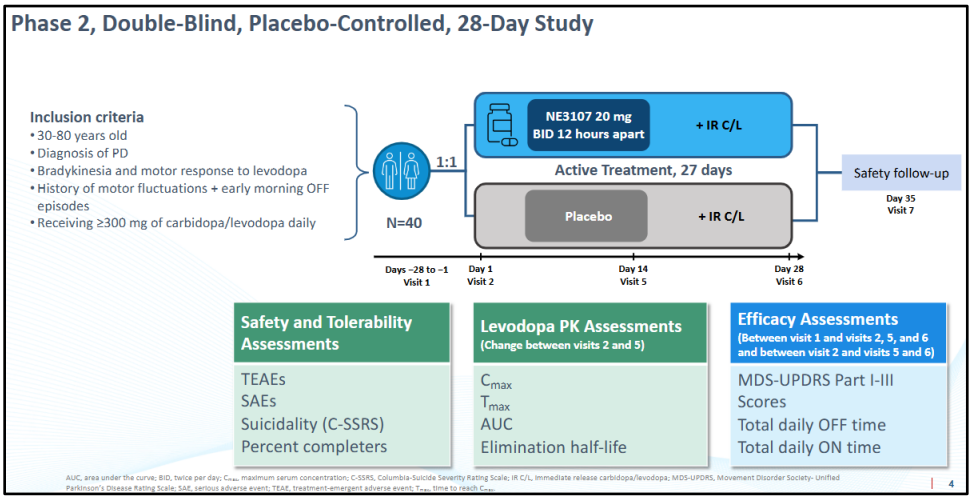
Effects of Beziserim in EAE Mouse Model of Myelin Degradation
Modified From: *Khan RS, Dine K, Luna E, Ahlem C, Shindler KS. HE3286 reduces axonal loss and preserves retinal ganglion cell function in experimental optic neuritis. Invest Ophthalmol Vis Sci. 2014 Aug 19;55(9):5744-51. doi: 10.1167/iovs.14-14672. PMID: 25139738; PMCID: PMC4161484.*

Similarly, Bezisterim selectively inhibited pro-inflammatory ERK/NK-kB signaling in a microbead glaucoma mouse model[5]. Not only did treatment rescue neuron signaling in the optic nerve, it also decreased proinflammatory signaling in supporting glial cells near the neurons. This is a notable feature for neurodegenerative conditions like PD, as surrounding supportive glial cells may amplify neurodegeneration.

1 - Nicoletti F, Philippens I, Fagone P, Ahlem CN, Reading CL, Frincke JM, Auci DL. 17α-Ethynyl-androst-5-ene-3β,7β,17β-triol (HE3286) Is Neuroprotective and Reduces Motor Impairment and Neuroinflammation in a Murine MPTP Model of Parkinson's Disease. Parkinsons Dis. 2012;2012:969418. doi: 10.1155/2012/969418. Epub 2012 Sep 26. PMID: 23050197; PMCID: PMC3463191.
2 - Mustapha M, Mat Taib CN. MPTP-induced mouse model of Parkinson's disease: A promising direction of therapeutic strategies. Bosn J Basic Med Sci. 2021 Aug 1;21(4):422-433. doi: 10.17305/bjbms.2020.5181. PMID: 33357211; PMCID: PMC8292858.
3 - Khan RS, Dine K, Luna E, Ahlem C, Shindler KS. HE3286 reduces axonal loss and preserves retinal ganglion cell function in experimental optic neuritis. Invest Ophthalmol Vis Sci. 2014 Aug 19;55(9):5744-51. doi: 10.1167/iovs.14-14672. PMID: 25139738; PMCID: PMC4161484.
4 - Philippens, I. H., A't Hart, B., & Torres, G. (2010). The MPTP marmoset model of parkinsonism: a multi-purpose non-human primate model for neurodegenerative diseases. Drug discovery today, 15(23-24), 985-990.
5 - Lambert, W. S., Carlson, B. J., Formichella, C. R., Sappington, R. M., Ahlem, C., & Calkins, D. J. (2017). Oral delivery of a synthetic sterol reduces axonopathy and inflammation in a rodent model of glaucoma. Frontiers in neuroscience, 11, 45.

Bezisterim in Parkinson's Disease

Parkinson's Phase 1/2 (NM201 - NCT05083260) - BIVI has completed a 28-day, placebo-controlled study of Bezisterim (NE3107) in Parkinson's disease (PD) patients experiencing motor fluctuations ("off" episodes) despite levodopa therapy.



Source: Ahlem, Clarence, et al. "Safety, Tolerability, and Efficacy of NE3107 From a Phase 2, Double-Blind, Placebo-Controlled Study in Levodopa/Carbidopa-Treated Patients With Parkinson's Disease." *Oral presentation at the 2023 International Conference on Alzheimer's and Parkinson's Diseases and Related Neurological Disorders*. 2023.

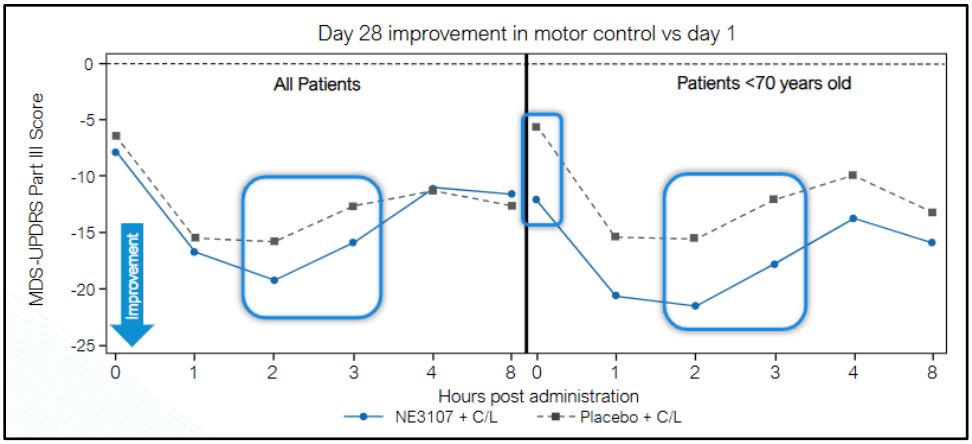
Forty-five patients on stable carbidopa/levodopa were randomized 1:1 to receive Bezisterim (20 mg oral, twice daily) or a placebo alongside their usual Parkinson's meds. The primary objectives were to evaluate safety and drug-drug interactions with levodopa (to satisfy FDA requirements), while secondary objectives examined Bezisterim's impact on Parkinsonian symptoms. Key endpoints included the UPDRS Part III motor score, daily "on/off" time, non-motor symptom scores, dyskinesia ratings, and pharmacokinetics.

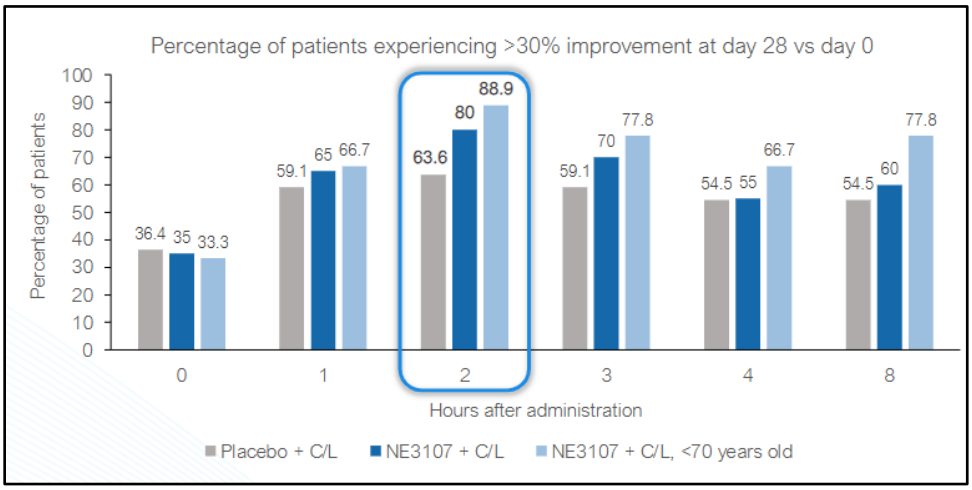
Results - The trial met its primary and secondary objectives, indicating that Bezisterim is safe with levodopa and may provide symptomatic benefits. No serious adverse interactions

were observed, and tolerability was good over the 28-day period (no significant differences in side effects vs placebo were reported in the topline release). Notably, patients receiving Bezisterim achieved better motor control than those on placebo. On Day 28, UPDRS Part III (motor) scores improved more in the Bezisterim group: the Bezisterim +levodopa patients had, on average, 3+ points greater improvement in UPDRS-III than the levodopa-only patients two to three hours after treatment. A 3-point UPDRS-III difference is clinically meaningful, so this is a notable signal of efficacy.

Furthermore, a pre-specified subgroup analysis suggested younger patients derive even more benefit. Among participants under 70 years old, those on Bezisterim improved by ~6 points more on UPDRS-III compared to placebo.

This subgroup analysis also found that before receiving Levodopa/Carbidopa, 30% (6/20) of patients receiving Bezisterim were not experiencing pathological symptoms of PD (tremors, stiffness, etc.). None of the placebo patients (0/19) demonstrated this benefit, referred to as being in an “on” state for PD patients. Overall, 88.9% of Bezisterim-treated patients achieved >30% improvement in motor scores from baseline (measured 2 hours post-dose) versus 63.6% of patients on placebo (levodopa alone).





Source: Ahlem, Clarence, et al. "Safety, Tolerability, and Efficacy of NE3107 From a Phase 2, Double-Blind, Placebo-Controlled Study in Levodopa/Carbidopa-Treated Patients With Parkinson’s Disease." *Oral presentation at the 2023 International Conference on Alzheimer’s and Parkinson’s Diseases and Related Neurological Disorders*. 2023.

Conclusion - The results from NCT05083260 predominantly indicate that Bezisterim is safe and well tolerated while not affecting traditional PD treatment efficacy and pharmacokinetics. In addition, the trial demonstrated a plausible improvement in motor coordination with Bezisterim treatment, particularly in less advanced PD. To our knowledge, no data have been published regarding behavior or mood in these patients. However, the early stage of the disease may have made identifying deficiencies difficult. The trial's small sample size precludes statistical rigor, but it justifies BIVI's current Phase 2 trial in PD.

Single Agent Bezisterim in Early PD (SUNRISE-PD; NCT06757010) – In 2025, BIVI initiated its second Phase 2 trial of Bezisterim in early Parkinson's disease (PD) patients. The SUNRISE-PD trial is enrolling 60 individuals diagnosed within the past 18 months. Patients are randomized to receive 20 mg of Bezisterim twice daily or placebo for 12 weeks. Importantly, eligible participants must be treatment-naïve, having not received prior therapy with levodopa or dopamine receptor agonists. Notably, the trial includes a 1-month run-in period before randomization to remove patients experiencing significant placebo effects from the final assessed patient pool.

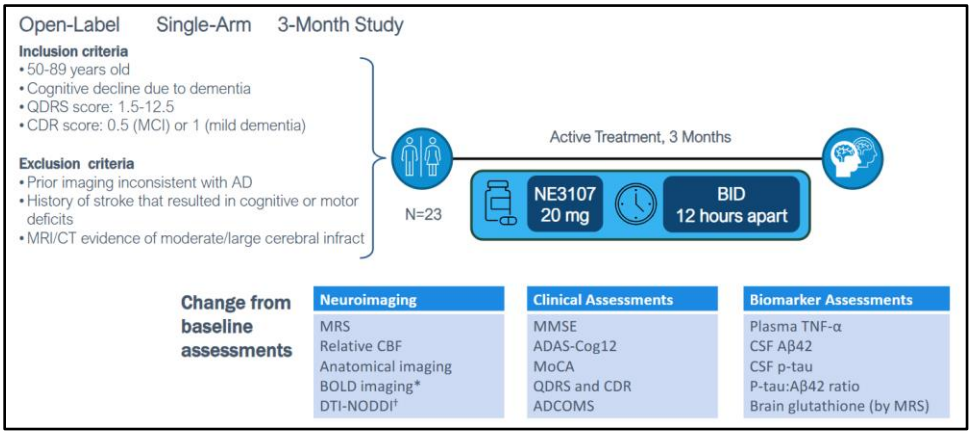
Outcomes – SUNRISE-PD's primary outcome mirrors BIVI's prior PD study, measuring the change from baseline in Movement Disorder Society-Unified Parkinson's Disease Rating Scale (MDS-UPDRS) Part III (motor examination) scores. Secondary endpoints include changes in MDS-UPDRS Parts I (non-motor experiences of daily living) and II (motor experiences of daily living), as well as clinician assessment using the Clinical Global Impression (CGI) Scale.

Rationale – BIVI's decision to explore Bezisterim as monotherapy stems from subgroup findings in its first Phase 2 trial. Prior to the morning dose of levodopa/carbidopa, 30% (6/20) of patients receiving Bezisterim exhibited an "ON" state—marked by an absence of pathological symptoms such as tremors or stiffness. By comparison, none of the placebo patients (0/19) experienced this benefit. Given the short half-life of levodopa/carbidopa (~1.5 hours, with elimination in 4–6 hours depending on formulation) and no observed impact of Bezisterim on levodopa plasma concentrations, the Hour 0 "ON" state appears attributable to Bezisterim alone.

Analysis - The 12-week treatment duration presents a double-edged sword. On one hand, if single-agent Bezisterim fails to deliver symptomatic benefit in patients nearing the threshold for dopamine-replacement therapy, the trial may face higher dropout rates than would be expected in a combination setting. On the other hand, the prior observation of an "ON" state after just four weeks suggests a pharmacodynamic effect rather than delayed disease progression. If this finding is replicated, the 12-week timeframe may allow for progressive deterioration in placebo-treated patients, potentially reducing the risk of placebo non-response.

Bezisterim in Alzheimer's Disease

Open-Label Phase 2 Biomarker Trial (NCT05227820) - In parallel with the Phase 1/2 PD study, BIVI conducted an open-label exploratory trial of Bezisterim in patients with mild cognitive impairment (MCI) or Alzheimer's disease. This study enrolled 23 patients with diagnosable cognitive decline and evidence of brain changes on MRI or PET imaging. All participants received 20 mg of Bezisterim twice daily for 12 weeks, with biomarker and imaging evaluations before and after treatment. No adverse side effects or treatment complications were noted.



Source: BioVie Inc.

Cognitive Function - After 3 months of Bezisterim, patients showed improvement in multiple cognitive scales. Improvements were seen in 4 out of 6 cognitive tests (ADAS-Cog12, MMSE, MoCA, and QDRS) compared to the patient baseline. In a subgroup of 17 patients with higher baseline cognition (MMSE >20), gains reached statistical significance, suggesting Bezisterim's cognitive benefit may be more discernible in earlier-stage patients.

Biochemical Markers - In cerebrospinal fluid (CSF), levels of phospho-tau (pTau) decreased by an average of 1.66 pg/mL ($p=0.034$) after 3 months of Bezisterim. The pTau/Aβ42 ratio, which is used to indicate AD pathology, also fell (-0.0024, $p=0.040$). The inflammatory marker, TNF-α, also declined but failed to reach significance in this sample size.

Imaging Biomarkers - MRI scans indicated improved brain physiology. Treated patients showed normalized cerebral perfusion in certain areas and increased functional connectivity in neural networks involving memory and attention. There was also a trend toward increased brain glutathione (an antioxidant marker), hinting at reduced oxidative stress. Though the glutathione rise did not reach statistical significance in the final analysis, the overall MRI findings suggest that Bezisterim may restore some neurovascular and network function in the brain.

The next page summarizes all findings from the Open-Label Phase 2 trial (NCT05227820).

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Summary of results from cognitive performance and biomarker assessments.

Endpoint	All patients					Patients with MMSE ≥ 20				
Cognitive assessment	Baseline, mean (95% CI)	Posttreatment, mean (95% CI)	Patients with improvement (%)	Mean change from baseline (95% CI)	*P* value*	Baseline, mean (95% CI)	Posttreatment, mean (95% CI)	Patients with improvement (%)	Mean change from baseline (95% CI)	*P* value*
ADAS-Cog11	15.28 (10.59, 19.96)	14.36 (8.74, 19.98)	13/23 (57)	–0.91 (–2.67, 0.84)	.29	10.50 (7.64, 13.36)	8.33 (5.51, 11.15)	13/18 (72)	–2.17 (–3.90,–0.43)	.017†
MMSE	24.00 (21.57, 26.43)	23.26 (20.39, 26.14)	8/23 (35)	–0.74 (–1.90, 0.42)	.20	26.44 (24.80, 28.09)	26.06 (24.12, 27.99)	8/18 (44)	–0.39 (–1.72, 0.95)	.55
MoCA	20.57 (17.59, 23.54)	20.52 (17.14, 23.91)	9/23 (39)	–0.04 (–0.96, 0.87)	.92	23.50 (21.44, 25.56)	24.06 (22.03, 26.08)	9/18 (50)	0.56 (–0.38, 1.49)	.23
QDRS	4.98 (3.74, 6.22)	4.44 (2.503, 6.37)	14/23 (61)	–0.54 (–1.77, 0.69)	.37	4.22 (3.16, 5.29)	2.67 (1.97, 3.36)	13/18 (72)	–1.56 (–2.46,–0.65)	.002†
CDR	0.61 (0.52, 0.70)	0.65 (0.41, 0.90)	4/23 (17)	0.04 (–0.14, 0.23)	.63	0.56 (0.48, 0.64)	0.44 (0.33, 0.56)	4/18 (22)	–0.11 (–0.22,–0.005)	.042†
ADCOMS	0.34 (0.22, 0.46)	0.35 (0.16, 0.53)	13/23 (57)	0.0049 (–0.079, 0.089)	.90	0.23 (0.16, 0.30)	0.17 (0.101, 0.21)	13/18 (72)	–0.07 (–0.12,–0.019)	.0094†

	All patients					Patients with MMSE ≥ 20				
Biomarker	Baseline, mean (95% CI)	Posttreatment, mean (95% CI)	Patients with improvement (%)	Mean change from baseline (95% CI)	*P* value*	Baseline, mean (95% CI)	Posttreatment, mean (95% CI)	Patients with improvement (%)	Mean change from baseline (95% CI)	*P* value*
Brain glutathione, mM	1.25 (0.92, 1.57)	1.35 (1.088, 1.62)	13/22 (59)	0.11 (–0.23, 0.45)	.54	1.12 (0.76, 1.48)	1.35 (1.051, 1.65)	12/18 (67)	0.23 (–0.15, 0.61)	.22
Plasma TNF-α, pg/mL	1.33 (0.73, 1.94)	0.93 (0.56, 1.304)	11/18 (61)	–0.45 (–1.22, 0.31)	.23	1.43 (0.67, 2.19)	0.91 (0.43, 1.402)	9/14 (64)	–0.56 (–1.55, 0.42)	.24
CSF P-tau:Aβ42 ratio	0.047 (0.0304, 0.063)	0.044 (0.028, 0.059)	10/16 (63)	–0.0033 (–0.008, 0.002)	.18	0.033 (0.019, 0.046)	0.0304 (0.019, 0.042)	7/11 (64)	–0.0024 (–0.005, 0.00)	.04†
CSF P-tau, pg/mL	31.87 (23.49, 40.25)	30.78 (22.72, 38.83)	13/21 (62)	–1.1 (–3.04, 0.85)	.26	26.46 (19.40, 33.52)	24.80 (18.49, 31.11)	10/16 (63)	–1.66 (–3.17,–0.14)	.033†
CSF Aβ42, pg/mL	842.3 (696.6, 987.9)	852.0 (709.2, 994.8)	11/18 (61)	9.72 (–60.72, 80.17)	.77	939.5 (770.6, 1108)	930.8 (750.8, 1111)	9/13 (69)	–8.69 (–80.95, 63.56)	.80

Aβ42 = amyloid beta peptide 42; ADAS-Cog11 = 11-item Alzheimer's Disease Assessment Scale-Cognitive Subscale; ADCOMS = Alzheimer's Disease Composite Score; CDR = Clinical Dementia Rating; CSF = cerebrospinal fluid; MMSE = Mini-Mental State Examination; MoCA = Montreal Cognitive Assessment; P-tau = phosphorylated tau protein; QDRS = Quick Dementia Rating Scale; TNF-α = tumor necrosis factor alpha.
*1-way *t*-test.
†Statistical significance ($P < .05$).

Source: Haroon, Jonathan BSa, et al., *A phase 2, open-label study of anti-inflammatory NE3107 in patients with dementias*. Medicine 103(30):p e39027, July 26, 2024. | DOI: 10.1097/MD.0000000000039027

Conclusion - This open-label study demonstrated that Bezisterim can penetrate the brain and engage its targets, leading to measurable improvements in cognition, CSF biomarkers (tau), and MRI metrics over 12 weeks. A lack of a placebo group and relatively small sample size limit the extent one can take these results, but suggest the drug has potential to positively impact AD patients, especially those early in disease progression.

Phase 3 Alzheimer's Disease Trial (NM101; NCT04669028) - BIVI launched the Phase 3 NM101 trial of Bezisterim in 2021 to evaluate its efficacy in patients with mild-to-moderate Alzheimer's disease (AD). The trial enrolled 439 patients across 30+ U.S. sites, randomizing participants to receive 20 mg of Bezisterim twice daily or placebo for 30 weeks in addition to standard-of-care AD medications. The co-primary endpoints were the change from baseline in ADAS-Cog12 and the Alzheimer's Disease Cooperative Study – Clinical Global Impression of Change (ADCS-CGIC). Secondary endpoints included additional cognitive and functional assessments and inflammatory and metabolic biomarkers.

Outcomes - The NM101 trial was designed to demonstrate Bezisterim's ability to slow cognitive decline. However, the trial failed to meet its primary endpoints. Fifteen of 39 of the clinical trial sites demonstrated significant protocol and Good Clinical Practice (GCP) deviations and presented data that was incongruent with prior effects of Bezisterim. Based on site investigations and the discovered deviations, BIVI elected to remove these sites from analysis, excluding 358/439 patients from the topline results. After site removals, only 81 patients remained in the modified intent-to-treat (mITT) population, including 57 patients with complete 30-week datasets (24 on Bezisterim, 33 on placebo). The study was therefore underpowered to detect statistically significant differences.

In those patients, Bezisterim-treated patients showed a 0.6-point advantage on the Clinical Dementia Rating – Sum of Boxes (CDR-SB), representing a 68% relative benefit compared to placebo. On ADAS-Cog12, the treatment group showed a 26% relative improvement. All cognitive and functional secondary measures numerically favored Bezisterim, with no new safety concerns.

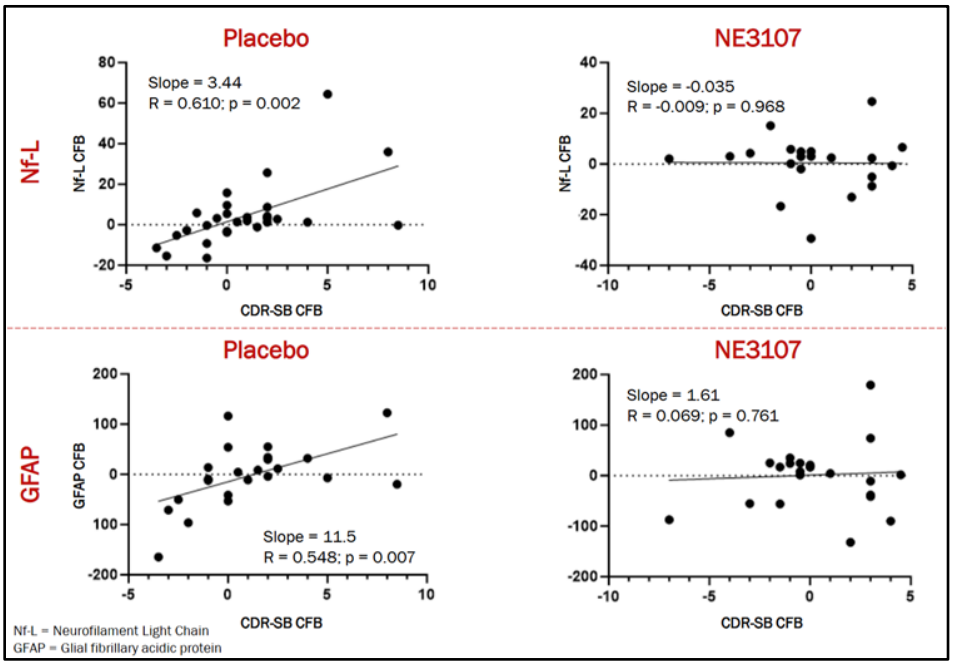
Change from baseline

	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

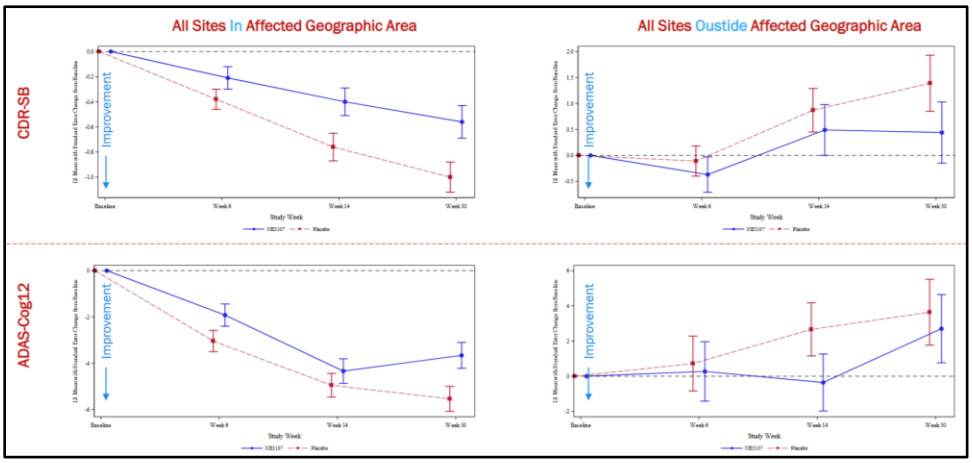
Week 30 Effects of Bezisterim and Comparable AD Data / Source: BioVie Inc.

Biomarker analysis revealed that plasma neurofilament light (NfL) and GFAP levels remained stable in the treatment group but increased in placebo, suggesting a potential neuroprotective effect.



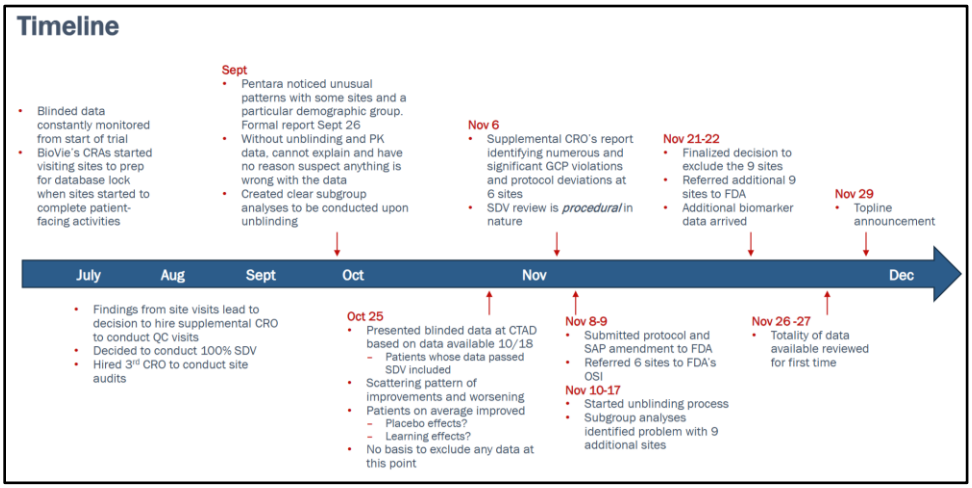
Biomarker Analysis of NM101 / Source: BioVie Inc.

Data Tampering Discussion - The NM101 trial encountered significant data integrity challenges that materially impacted the evaluable patient population. BIVI reported identifying duplicated MRI reports, identical cognitive assessments across multiple patients, and placebo group responses inconsistent with the expected progression of Alzheimer's disease (AD). Additionally, Suzanne Hendrix, CEO of Pentara Corporation, an independent firm engaged to analyze the trial, reported that Bezisterim was detected in certain placebo patients as well as in some patients at baseline.



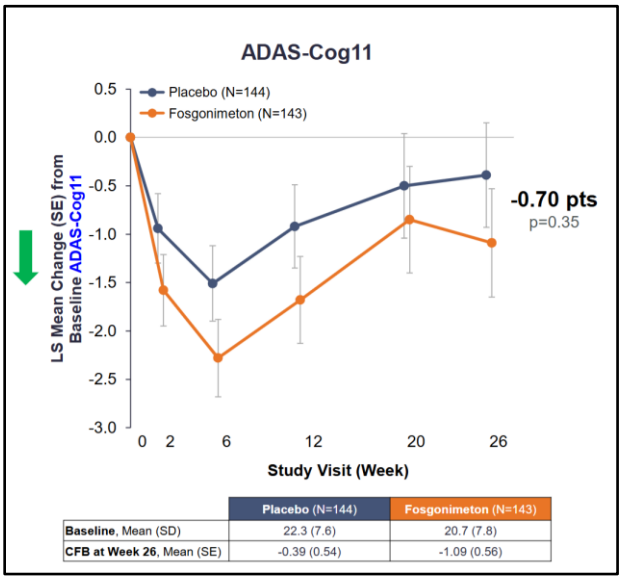
Comparison of Cognitive Outcomes from Different Geographic Areas / Source: BioVie Inc.

Following a multi-month review involving independent biostatistical consultants and two contract research organizations (CROs), six trial sites were excluded due to substantial protocol and Good Clinical Practice (GCP) deviations. Nine additional sites were removed after subgroup analyses confirmed scientifically implausible placebo improvements clustered in a specific demographic and geographic region. In total, 15 sites were referred to the FDA's Office of Scientific Investigations (OSI) for review.



Timeline of Events Surrounding Phase 3 AD Trial / Source: BioVie Inc.

While BIVI attributed the majority of the trial's failure to these protocol deviations, broader sector trends suggest this phenomenon is not unique. In 2024, multiple AD trials reported unexpected placebo performance that confounded study results. Athira Pharma's Phase 2/3 LIFT-AD trial (NCT04488419) found no difference between placebo and Fosgonimeton-treated patients, with both groups declining ~1 point on the ADAS-Cog11 scale[1]. Similarly, Annovis Bio's Phase 2/3 moderate AD trial (NCT05686044) reported that placebo outperformed its lowest Buntanetap dose, with placebo patients declining ~2 points on ADAS-Cog11[2].



ADAS-Cog11 Results from Phase 2/3 LIFT-AD Trial (NCT04488419) / Source: Athira Pharma Inc.

PRIMARY ENDPOINT IN ITT POPULATION (N=351)

ADAS-Cog11	Placebo (N=89)	7.5mg Buntanetap (N=88)	15mg Buntanetap (N=87)	30mg Buntanetap (N=87)
Baseline				
N	85	86	87	87
Mean Score (SD)	20.28 (6.79)	23.20 (7.59)	22.74 (7.57)	21.85 (7.16)
End of Trial (12 weeks)				
N	81	82	78	79
Mean Score (SD)	18.33 (6.94)	21.42 (8.42)	19.26 (7.70)	20.21 (8.94)
Difference from Baseline	-2.182	-1.452	-2.992	-2.304
P-value	0.001	0.001	0.001	0.001
Difference from Placebo		0.98 (0.75)	-0.68 (0.76)	0.09 (0.75)
P-value		0.193	0.366	0.910

ADAS-Cog11 Results from Phase 2/3 AD Moderate AD Trial (NCT05686044) / Source: Annovis Bio Inc.

Each company responded differently. Athira exited the AD program to refocus on ALS. Annovis applied post hoc biomarker analyses to refine its target population and proceeded to Phase 3 testing in early AD. These cases illustrate the inherent challenges in AD trial design, including variable placebo effects, recruitment of heterogeneous patient populations, and the possibility of insufficient drug efficacy.

Given these sector-wide examples, it remains difficult to conclusively determine whether NM101's results stem from outright data tampering or broader complexities such as placebo effects and trial design limitations. However, the geographic clustering of protocol deviations, confirmed data inconsistencies, and baseline/placebo presence of Bezisterim suggest issues that require further investigation. BIVI management noted that the FDA has not provided updates following the OSI submission, which the company interprets as a sign of an ongoing review process. At this time, the validity of the trial data remains uncertain, pending further regulatory clarity.

1 - https://investors.athira.com/static-files/0751d535-eb7c-4f79-bad8-36731b5c6236#page=7.00
2 - *Annovis Bio Web Cast 6/11/2024* (2024) *YouTube*. Available at: https://www.youtube.com/watch?v=Q54elNTgfPo&t=1s (Accessed: 17 March 2025).

Anti-inflammatory Competition in Neurodegeneration

Several drug candidates in development share Bezisterim's strategy of targeting neuroinflammation as a potential disease-modifying approach in neurodegenerative disorders. Below, we highlight key programs advancing through clinical development that modulate inflammatory pathways relevant to Alzheimer's disease (AD), dementia with Lewy bodies (DLB), and Parkinson's disease (PD).

CervoMed (CRVO; $9.34; Buy $15 PT) Neflamapimod – Neflamapimod is an oral small molecule that selectively inhibits p38α MAP kinase, reducing pro-inflammatory signaling in microglia and neurons upstream of NF-κB and cytokine production. The drug has been found to improve cognition in DLB patients. Similar to Bezisterim, Neflamapimod has demonstrated stronger benefits in earlier stages of DLB, specifically without Alzheimer's co-pathology[1].

Currently, the company does not have active plans to pursue PD, and recently announced new trials in frontotemporal dementia (FTD) and acute ischemic stroke recovery. We view the competitive potential of Neflamapimod's current development trajectory as low. Still, the

pathway overlap could result in future competition if CRVO decides to pursue PD or AD indications.

INmune Bio Inc. (INMB; $7.19; Non-Rated) XPro1595 – XPro1595 is a selective, brain-penetrant biologic designed to neutralize soluble TNFα while preserving membrane-bound TNF functions. This dominant-negative TNF variant blocks chronic inflammatory signaling through TNF receptor 1 while sparing protective TNF receptor 2 pathways. In a Phase 1b trial, 20 Alzheimer's patients received weekly subcutaneous XPro1595 (0.3–1.0 mg/kg) for three months, leading to reductions in CSF inflammatory cytokines and a 5% decline in MRI-measured white-matter free water in the high-dose group. Cognitive trajectories remained stable, and no serious adverse events were reported[2].

By targeting TNFα, a master pro-inflammatory cytokine, XPro1595 represents a parallel approach to Beziterim's – both seek to break the cycle of chronic neuroinflammation (one via cytokine neutralization, the other via kinase signaling modulation). Topline data from a larger Phase 2 trial in AD is expected in June 2025. However, the company has made no reference to pursuing PD as a target indication.

NodThera (private) Multiple NLRP3 Inflammasome Inhibitors (NT-0796) – The NLRP3 inflammasome is a protein complex in microglia and macrophages that, when activated, releases pro-inflammatory cytokines (IL-1β, IL-18) and drives chronic inflammation in neurodegenerative diseases. NodThera's NT-0796 is an orally-available brain-penetrant NLRP3 inhibitor currently being assessed in PD patients.

In a recent Phase Ib/IIa trial in Parkinson's disease patients, 28 days of NT-0796 led to a marked reduction of inflammatory biomarkers in CSF. Treated PD patients saw CSF levels of IL-1β, IL-6, and chemokines drop to levels comparable to healthy controls – effectively normalizing these inflammation markers in just one month. Indicators of neurodegeneration, like neurofilament light (NfL) and soluble TREM2, also trended downward with NT-0796[3]. These results demonstrate a potent reversal of NLRP3-mediated neuroinflammation in humans. Beziterim also intersects this pathway (NF-κB/TNF/IL-1 are downstream of NLRP3), but NLRP3 inhibitors act even further upstream by preventing cytokine release in the first place.

AB Science (ABSCF; $1.57; Non-Rated) Masitinib – Masitinib is an oral tyrosine kinase inhibitor that blocks mast cell and microglial activation (through targets like CSF1R, c-Kit, Lyn). It is being investigated in Alzheimer's (Phase 3) and ALS to reduce neuroinflammation by inhibiting microglial proliferation and mast cell-mediated inflammation. A Phase 2/3 trial in AD reported that masitinib (plus cholinesterase inhibitor) slowed cognitive decline in a subset of patients with mild AD. However, results have been mixed, and safety (risk of neutropenia and ischemic heart disease) needs monitoring. Masitinib's approach to regulating the innate immune cells in the CNS is another variant of targeting neuroinflammation. It's not directly in the ERK/NF-κB pathway, but it converges on attenuating chronically activated glia to modify the disease course[4].

1 - https://www.alzforum.org/therapeutics/neflamapimod
2 - https://www.alzforum.org/therapeutics/xpro1595
3 - https://www.nodthera.com/news/nodtheras-nlrp3-inhibitor-nt-0796-reverses-neuroinflammation-in-parkinsons-disease-phase-ib-iia-trial
4 - https://www.alzforum.org/therapeutics/masitinib

Other Bezisterim Indications & BIV201 in Ascites

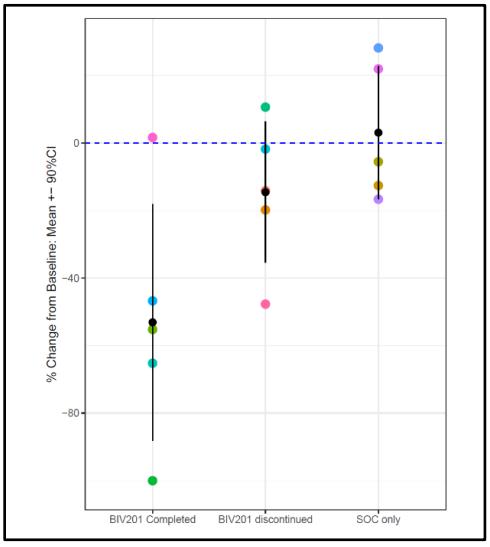
Outside BIVI's neurodegeneration programs, the company also pursues other indications for Bezisterim. We do not currently include these indications or compounds in our revenue

projections for the company, either due to the developmental stage and/or lack of clarity on commercialization. However, we acknowledge that each presents potential upside for BIVI outside of our DCF analysis.

As the company will require additional capital to rerun/initiate a Phase 3 trial in AD and/or PD, out-licensing or partnerships for these indications and compounds may serve to help alleviate dilutive fundraising.

BIV201 for Ascites - BIVI is developing BIV201, a continuous infusion of low-dose terlipressin, for the treatment of refractory ascites in patients with advanced liver cirrhosis. Refractory ascites, characterized by fluid accumulation in the abdomen unresponsive to diuretics, is a significant unmet medical need with poor prognosis and limited therapeutic options.

The company initiated an NIH-sponsored Phase 2a clinical trial, demonstrating promising results in reducing ascites fluid accumulation and improving patient outcomes. Enrollment was concluded after 15 patients of the targeted 30 were enrolled, as the reduction was biologically significant enough to achieve statistical significance (53% reduction; p<0.001). 5 out of the 10 patients initially enrolled discontinued treatment after Cycle 1 of the 28-day trial, however, these patients also demonstrated a 15% reduction in fluid volume. This positively compared to standard of care (SOC) patients, who increased ascites volume by 3.1% over the 28-day trial.



BIV201 Phase 2b Trial Results / Source: BioVie Inc

Subsequently, the FDA granted BIV201 Orphan Drug Designation for the treatment of ascites due to liver cirrhosis. In April 2022, BIVI submitted a pre-IND briefing package to the FDA for a pivotal Phase 2b/3 clinical trial. The agency provided feedback on trial design, patient population, and clinical endpoints.

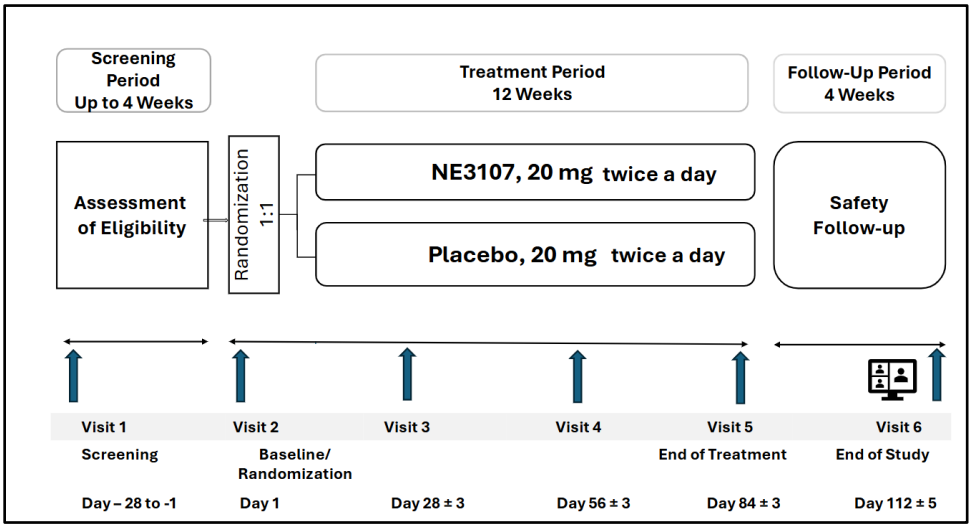
BIVI intends to initiate the pivotal trial pending a strategic partnership. The search for a partner is ongoing. If successful, BIV201 could be the first drug therapy approved for the treatment of refractory ascites in the United States, offering a novel therapeutic option to address this serious complication.

Bezisterim for Long COVID – Outside of neurodegenerative indications, BIVI is also developing Bezisterim as a novel treatment for the neurocognitive symptoms associated

with long COVID. Chronic inflammation, neuroinflammation, and blood-brain barrier dysfunction are believed to underlie persistent symptoms such as fatigue, cognitive dysfunction, and sleep disturbances in long COVID patients.

In April 2024, BIVI secured a $13.1 million clinical trial award from the U.S. Department of Defense through its Peer Reviewed Medical Research Program. The award provides non-dilutive funding to support a two-year Phase 2b clinical trial evaluating Bezisterim for neurological symptoms in long COVID. The trial was cleared by the FDA in August 2024, following Investigational New Drug (IND) approval, and received final scientific review approval from the U.S. Army Office of Human Research Oversight.

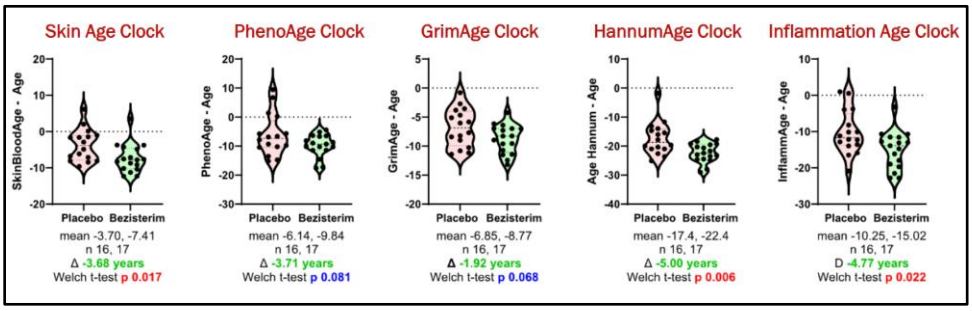
Code named ADdRESs-LC, the 1:1 placebo-controlled trial aims to enroll 208 patients suffering from Long-COVID. Diagnosis of the condition is somewhat opaque, but patients will undergo cognitive testing over a 12-week treatment with Bezisterim or placebo. BIVI is using the Cogstate Brief Battery (CBB), a collection of four computerized cognitive tests designed to assess different aspects of cognition, as the primary outcome. ADdRESs-LCl is published on Clinicaltrials.gov (NCT06847191), but is not yet recruiting.



Trial Design for Bezisterim in Long-COVID / Source: BioVie Inc.

Bezisterim for Longevity - BIVI is evaluating Bezisterim as a potential longevity therapeutic based on its impact on DNA methylation, a biomarker often associated with biological aging. In the company's Phase 3 Alzheimer's disease trial, DNA methylation testing was included as an exploratory endpoint to assess age deceleration, measuring the difference between biological and chronological age.

Preliminary analysis of 33 patients indicated trending reductions in DNA methylation across several epigenetic "clocks," genomic regions where methylation patterns correlate with aging. These early findings suggest a potential role for Bezisterim in modulating biological age, although the causative relationship between DNA methylation changes and aging remains under scientific debate.



Impact of Bezisterim on DNA Methylation "Clocks" in Phase 3 AD Trial / Source: BioVie Inc.

While aging is not currently classified as a disease by the FDA, limiting traditional pharmaceutical pathways, interest in biological aging and epigenetic interventions is growing. This area could represent a future opportunity for BIVI as the longevity field continues to expand.

Intellectual Property

As of August 15th, 2024, BIVI has 12 issued U.S. patents, 6 pending U.S. patent applications, 3 pending U.S. PCT applications, 6 issued foreign patents, and 6 pending foreign patent applications directed to protecting Bezisterim. The original composition of matter patent covering Bezisterim is set to expire on 6/2/2026. However, the company's pending patents, *Compositions for treatment of neurodegenerative conditions* (US20240108636A1) and *Methods for the treatment of mild cognitive impairment* (WO2024054452A1), should protect through ~2043, excluding patent term extensions.

Outside of Bezisterim, BIVI also holds domestic and foreign patents for BIV201, its ascites compound. BIV201 was given Orphan Drug Designation by the FDA for hepatorenal syndrome in 2018 and ascites in 2016. BIVI's current patent estate can be found below.

BIVI Patent Estate Summary

Compound	Patent/Publication #	Title/Subject	Jurisdiction	Expiration Date
BIV201	WO2020/237170	Liquid terlipressin formulations	PCT (US, EP, CN, JP)	—
	IN540813	Terlipressin formulation	IN	—
	CL68965	Terlipressin formulation	CL	—
	US11,364,277	Method for treating ascites	US	—
	EP3347032	Method for treating ascites	EP	—
Bezisterim (NE3107)	US8,586,770	Unsaturated steroid compounds	US	6/2/2026
	US8,252,947	Solid-state pharmaceutical forms	US, AU, CA, EP, KR	4/18/2030
	US9,555,046 / US9,850,271 / US10,995,112 / (pending)	Crystalline anhydrate forms	US	4/3/2029
	US8,518,922	Pharmaceutical solid-state forms	US	9/24/2031
	US9,314,471	Methods of preparing solid-state forms	US	6/28/2029
	US8,486,926	Steroid tetrol solid-state forms	US	1/10/2030
	US8,354,396	Drug identification & treatment method	US	7/7/2031
	US9,163,059 / US9,994,608	Methods for preparing 3,7-dihydroxy steroids	US, EP, JP	6/5/2029
	US9,877,972	Treatment methods using solid-state forms	US	4/3/2029
	18/511,027 (pending)	Compositions for neurodegenerative conditions	US	—
	63/621,280 (pending)	Methods of treating Long COVID	US	—
	63/610,915 (pending)	Modified C19 steroids and methods of use	US	—
	63/592,364 (pending)	Compositions and methods for amyloid beta diseases	US	—
	63/561,157 (pending)	Methods for treating biological aging	US	—

Expiration dates are projections and do not reflect potential extensions or regulatory exclusivity.
Source: BIVI Filings, Brookline Capital Markets Research

Management Team

Cuong Do MBA – *President & Chief Executive Officer*

Cuong Do, MBA, is the President, Chief Executive Officer, and Director of BioVie Inc., a clinical-stage company focused on developing treatments for neurological and liver diseases. His previous roles include President of Samsung Global Strategy Group and Chief Strategy Officer at Merck & Co., Inc., where he contributed to the development of Keytruda® (pembrolizumab). Do also has held leadership positions at Lenovo Group Ltd., TE Connectivity Ltd., and served as a Senior Partner at McKinsey & Company.

Do founded several biotechnology companies, including Callidus Biopharma, M6P Therapeutics, and Perceptivus LLC. He serves on the boards of Seneca Therapeutics, Adagene Inc., Curve UK Ltd., ILiAD Biotechnologies, True Image Interactive, Autism Impact Fund, and Celebrate the Children.

Do earned his BA from Dartmouth College and his MBA from the Tuck School of Business at Dartmouth.

Joseph M. Palumbo, MD - *Chief Medical Officer*

Dr. Joseph M. Palumbo is the Chief Medical Officer at BioVie Inc., where he oversees clinical development and operations. He is also the current Chief Executive Officer of Locumtele LLC and serves on the Board of Directors at Hydreight Technologies, Inc. Previously, he held the position of Chief Medical Officer at Zynerba Pharmaceuticals and has held senior leadership roles at Mitsubishi Tanabe Pharma and Johnson & Johnson. His industry experience includes positions at Sanofi-Synthelabo, Cephalon, and the CRO Pharmanet.

Dr. Palumbo has also held academic positions at Yale University, Cornell University, and the University of Pennsylvania. He earned a BA from the University of Pennsylvania and a Doctor of Medicine from The George Washington University School of Medicine. He completed his residency at Yale University and was a fellow in the Biological Sciences Training Program at the National Institutes of Health.

Wendy Kim, CPA - *Chief Financial Officer*
Wendy Kim, CPA, is the Chief Financial Officer of BioVie Inc., where she supervises corporate finance, financial planning, and SEC reporting. She joined BioVie in 2018 and has since directed financial strategy, bank financing, M&A transactions, and risk management. Prior to her role at BioVie, she held the CFO position at several organizations, including Landmark Education Enterprises, Inc., and worked as a Director in the National Office SEC Department at BDO USA from 2008 to 2016.

She earned a BBA in Accounting and Finance from California State University.

Clarence Ahlem - *Senior Vice President, Operations*
Clarence Ahlem is the Senior Vice President of Operations at BioVie Inc. He joined BioVie and has since been responsible for overseeing operational strategy and pharmaceutical development. Prior to BioVie, he served as Vice President of Product Development at Harbor BioSciences (formerly known as Hollis-Eden Pharmaceuticals and Harbor Therapeutics). He held leadership positions at Prolinx Biochemistry and Systemix, Inc., and spent six years at Hybritech, where he developed synthetic bifunctional antibodies for cancer therapy.

Earlier, Mr. Ahlem conducted research at the University of California, San Diego. He earned his MS in microbiology from SDSU in 1981.

Penelope Markham, Ph.D. - *Senior Vice President, Liver Disease Program*
Penelope Markham, Ph.D., is the Senior Vice President of the Liver Disease Program at BioVie Inc., a position she has held since 2021. Before this, she led the development of modified terlipressin compounds at LAT Pharma LLC, the predecessor company to BioVie, for seven years prior to its acquisition. Earlier in her career, she co-founded Influx, Inc., where she served as Research Director from 1997 to 2003, concentrating on antibiotic drug discovery. Dr. Markham has expertise in immunology, infectious diseases, bacteriology, and drug discovery research. She has also served on NIH grant review panels while consulting for pharmaceutical companies, including those involved in orphan drug development.

Dr. Markham earned a BS in Biochemistry from University College Cork, an MS from Strathclyde University, and a Ph.D. from Rush University.

Chris Reading, Ph.D. - *Senior Vice President, Alzheimer's Disease Program*
Chris Reading is the Senior Vice President of the Alzheimer's Disease Program at BioVie Inc. He previously spent 13 years at MD Anderson Cancer Center and the University of Texas Graduate School of Biomedical Sciences, where he served as an Associate Professor of Medicine in the Department of Developmental Therapeutics, holding a joint appointment in the Department of Tumor Biology. He later joined Systemix/Novartis in Palo Alto as Vice President of Product and Process Development before relocating to San Diego, where he worked on the NE3107 platform at Harbor Therapeutics for over 15 years. His research has concentrated on monoclonal antibodies, cell separation technologies, stem cell transplantation, and sterol drug development.

Dr. Reading earned a Ph.D. in Biochemistry from UC Berkeley and completed postdoctoral studies in cancer biology at UC Irvine.

David Morse, Ph.D. - *Chief Regulatory Officer and Senior Vice President*
David Morse is the Chief Regulatory Officer and Senior Vice President at BioVie Inc., where he oversees regulatory strategy and pharmaceutical development. Prior to joining BioVie, he held the position of Vice President of Global Regulatory Affairs and Vice President of Regulatory Strategy at several contract research organizations. He has also worked as an independent consultant, Associate Director at the U.S. Food and Drug Administration (FDA), and Laboratory Director in translational medicine research.

His background in regulatory affairs and product development encompasses neurology, anti-infectives, oncology, gastroenterology, dermatology, and therapeutics for radiological injuries.

Dr. Morse earned his Ph.D. from the Uniformed Services University of the Health Sciences in Bethesda, Maryland.

Board of Directors

Jim Lang - ***Chairman of the Board of Directors***
Jim Lang is the Chairman of the Board of Directors at BioVie Inc. and the founder and CEO of EVERSANA, a global provider of commercialization services for the life sciences industry. He previously served as CEO of Decision Resources Group, where he led its transformation into a healthcare data and analytics firm. He was President of Strategic Decision Group, eventually selling its life sciences practice to IQVIA. He has held executive roles at IHS CERA, Cambridge Energy Research Associates, and Strategic Decisions Group. Lang has been on multiple private and public boards, including OptimizeRx Corp. and various healthcare and life sciences companies. Additionally, he has acted as an investor and advisor to early-stage healthcare companies, including Boston Heart Labs and AlphaImpactRx.

Mr. Lang earned a BS in Electrical and Computer Engineering from the University of New Hampshire and an MBA from the Tuck School of Business at Dartmouth.

Cuong Do MBA – ***President & Chief Executive Officer***
See Above

Bob Hariri, M.D., Ph.D. - ***Independent Director***
Dr. Bob Hariri is an Independent Director at BioVie Inc., as well as the chairman, founder, and chief executive officer of Celularity, Inc., a company dedicated to human cellular therapeutics. He previously founded and served as CEO of Anthrogenesis Corporation, which was later acquired by Celgene Corporation, where he led Celgene Cellular Therapeutics. Additionally, he co-founded Human Longevity, Inc., a company focused on genomic-based health intelligence.

He has served on various public and private boards, including Cryoport, Primus Therapeutics, and The Trustees of Columbia University. Dr. Hariri has been actively involved in biomedical research, particularly in stem cell therapeutics and immuno-oncology, and played a role in the discovery of pluripotent stem cells from the human placenta. Moreover, he is a trustee at Liberty Science Center and has held leadership positions at Weill Cornell Medical College and the New Jersey Commission on Cancer Research.

Dr. Hariri earned his M.D. and Ph.D. from Weill Cornell Medical College and completed his undergraduate studies at Columbia University's Fu Foundation School of Engineering & Applied Science.

Sigmund Rogich - ***Independent Director***
Sigmund Rogich is an Independent Director at BioVie Inc. and the founder and president of The Rogich Communications Group. He previously served as the U.S. Ambassador to Iceland and has acted as a senior consultant for several presidential campaigns, including those of Ronald Reagan and George H.W. Bush. He has also held leadership positions at WorldDoc, Inc. and On Stage Entertainment, Inc.

Mr. Rogich is a board member of Keep Memory Alive, a philanthropic organization that supports clinical trials at the Cleveland Clinic's Lou Ruvo Center for Brain Health, focusing on Alzheimer's, Huntington's, and Parkinson's diseases, as well as other neurological disorders. He also serves as a director at Mystic Holdings, Inc.

Mr. Rogich earned his undergraduate degree from the University of Nevada.

Michael Sherman, J.D. - ***Independent Director***
Michael Sherman serves as an Independent Director at BioVie Inc. and is the Chairman of Ontrak, Inc. He previously held the position of Managing Director at Barclays PLC from 2008 to 2018 and at Lehman Brothers, Inc. from 1998 to 2008, totaling 30 years in the investment banking sector. He has extensive healthcare finance experience, providing guidance on financial transactions for companies such as Neurocrine Biosciences, Teva Pharmaceutical Industries, Amgen, Cubist Pharmaceuticals, Merck & Co., and Cardinal Health.

Mr. Sherman earned his undergraduate degree from the University of Pennsylvania and graduated cum laude with a J.D. from Harvard Law School.

BioVie Inc. (BIVI) / March 18, 2025

BIVI Quarterly Income Statements ($MM)

	FY2024	Q1:25	Q2:25	Q3:25E	Q4:25E	FY2025E	Q1:26E	Q2:26E	Q3:26E	Q4:26E	FY2026E
Revenue	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00
D&A	*($0.23)*	*($0.06)*	*($0.06)*	*($0.06)*	*($0.06)*	*($0.23)*	*($0.04)*	*($0.04)*	*($0.04)*	*($0.04)*	*($0.18)*
COS	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00
Gross Profit	**($0.23)**	**($0.06)**	**($0.06)**	**($0.06)**	**($0.06)**	**($0.23)**	**($0.04)**	**($0.04)**	**($0.04)**	**($0.04)**	**($0.18)**
R&D	($23.10)	($1.99)	($4.70)	($3.50)	($5.00)	($15.20)	($6.95)	($7.55)	($3.50)	($2.00)	($20.00)
S,G&A	($8.85)	($2.07)	($2.53)	($2.33)	($2.13)	($9.07)	($2.22)	($2.77)	($2.54)	($2.28)	($9.81)
Total OpEx	**($31.95)**	**($4.06)**	**($7.24)**	**($5.83)**	**($7.13)**	**($24.26)**	**($9.17)**	**($10.32)**	**($6.04)**	**($4.28)**	**($29.81)**
EBIT	**($32.18)**	**($4.12)**	**($7.29)**	**($5.89)**	**($7.19)**	**($24.49)**	**($4.12)**	**($7.29)**	**($6.08)**	**($4.32)**	**($21.82)**
Other (net)	$0.06	($0.03)	$0.18	$0.00	$0.00	$0.15	$0.00	$0.00	$0.00	$0.00	$0.00
Pretax	($32.12)	($4.15)	($7.11)	($5.89)	($7.19)	($24.34)	($9.21)	($10.36)	($6.08)	($4.32)	($29.99)
Taxes	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00	$0.00
Net income	**($32.12)**	**($4.15)**	**($7.11)**	**($5.89)**	**($7.19)**	**($24.34)**	**($9.21)**	**($10.36)**	**($6.08)**	**($4.32)**	**($29.99)**
Preferred Dividends	($0.89)	($0.33)	($0.04)	$0.00	$0.00	($0.37)	$0.00	$0.00	$0.00	$0.00	$0.00
Earnings Per Share*	**($7.30)**	**($0.70)**	**($0.46)**	**($0.32)**	**($0.39)**	**($1.34)**	**($0.50)**	**($0.56)**	**($0.33)**	**($0.23)**	**($1.63)**
Shares Outstanding (MM)	4.5	6.4	15.7	18.5	18.5	18.5	18.5	18.5	18.5	18.5	18.5

** Adjusted EPS and Share Count following Aug.6th 2024 Reverse Stock Split*

Source: BIVI Company Filings, FactSet, Brookline Capital Markets Estimates

BIVI Balance Sheets ($000s)

	3/31/2024	6/30/2024	9/30/2024	12/31/2024
Current Assets:				
Cash	$30,350	$23,844	$20,023	$24,406
Prepaid Expenses and Other Current Assets	190	204	128	276
Total Current Assets:	**$30,540**	**$24,048**	**$20,151**	**$24,681**
Operating leases	422	407	391	374
Intangible assets, net	465	408	350	293
Other assets	346	346	346	346
Non-Current Assets:	**$1,233**	**$1,160**	**$1,087**	**$1,013**
Total Assets:	**$31,773**	**$25,208**	**$21,238**	**$25,694**
Current Liabilities:				
Accounts Payable & Accrued Expenses	$4,410	$3,587	$3,453	$1,383
Operating Lease Liability (Current)	57	60	64	67
Current Portion of Note(s) Payable	7,993	5,701	3,320	0
Warrant Liability	21	4	1	7
Other Current Liabilities	0	0	0	0
Total Current Liabilities:	**$12,481**	**$9,352**	**$6,838**	**$1,457**
Operating Lease Liability (Non Current)	366.43	349.894	332.73	314.915
Non-Current Liabilities:	**$366**	**$350**	**$333**	**$315**
Total Liabilities	**$12,847**	**$9,702**	**$7,171**	**$1,772**
Total Shareholders' Equity	**$18,926**	**$15,506**	**$14,067**	**$23,923**
Total Liabilities and Shareholders' Equity	**$31,773**	**$25,208**	**$21,238**	**$25,694**

Source: BIVI Company Filings

BIVI US Forecast for Bezisterim in PD ($MM)

	Year Ended June 30											
	2024	**2025**	**2026**	**2027**	**2028**	**2029**	**2030**	**2031**	**2032**	**2033**	**2034**	**2035**
Revenue	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$126.8	$287.2	$511.0	$739.6	$839.2
D&A	*(0.2)*	*(0.2)*	*(0.2)*	*(0.2)*	*(0.2)*	*(0.2)*	*(0.2)*	*(0.2)*	*(0.2)*	*(0.2)*	*(0.2)*	*(0.2)*
COS	0.0	0.0	0.0	0.0	0.0	0.0	0.0	(38.1)	(63.2)	(76.7)	(110.9)	(125.9)
Gross Profit	**($0.2)**	**($0.2)**	**($0.2)**	**($0.2)**	**($0.2)**	**($0.2)**	**($0.2)**	**$88.6**	**$223.8**	**$434.2**	**$628.4**	**$713.1**
R&D	($23.1)	($15.2)	($20.0)	($14.0)	($15.2)	($45.9)	($12.0)	($11.2)	($26.4)	($37.8)	($86.8)	($112.0)
S,G&A	(8.8)	(9.1)	(9.8)	(11.0)	(11.0)	(12.0)	(24.2)	(71.1)	(130.4)	(173.8)	(251.5)	(285.3)
Total OpEx	**($32.0)**	**($24.3)**	**($29.8)**	**($25.0)**	**($26.2)**	**($57.9)**	**($36.2)**	**($82.3)**	**($156.8)**	**($211.5)**	**($338.3)**	**($397.3)**
EBIT	**($32.2)**	**($24.5)**	**($30.0)**	**($25.2)**	**($26.4)**	**($58.1)**	**($36.4)**	**$6.3**	**$67.0**	**$222.7**	**$290.2**	**$315.8**
Other (net)	$0.1	$0.2	$0.0	$1.0	$1.0	$1.0	$1.0	$1.0	$1.0	$1.0	$1.0	$1.0
Pretax	**($32.1)**	**($24.3)**	**($30.0)**	**($24.2)**	**($25.4)**	**($57.1)**	**($35.4)**	**$7.3**	**$68.0**	**$223.7**	**$291.2**	**$316.8**
Taxes	0	0	0	0	0	0	0	0	0	0	(16)	(86)
Net income	**($32.1)**	**($24.3)**	**($30.0)**	**($24.2)**	**($25.4)**	**($57.1)**	**($35.4)**	**$7.3**	**$68.0**	**$223.7**	**$275.1**	**$231.3**
NOL	($334.2)	($358.5)	($388.5)	($412.7)	($438.1)	($495.2)	($530.6)	($523.3)	($455.3)	($231.6)	-	-
Ratios:												
Gross Margin	NM	NM	NM	NM	NM	NM	NM	70%	78%	85%	85%	85%
Optg. Expense:												
R&D	-	-	-	-	-	-	-	9%	9%	7%	12%	13%
S,G&A	-	-	-	-	-	-	-	56%	45%	34%	34%	34%
Pretax Margin	-	-	-	-	-	-	-	6%	24%	44%	39%	38%
Tax Rate	27%											

NPV:

Stage	**Rate**	**($MM)**	**Share***
Discovery	20%	$35	$2
Clinical	15%	$88	$5
Market	10%	$180	$10

***Per Share Count Assumes 18.45MM shares**

Source: Brookline Capital Markets estimates

BROOKLINE CAPITAL MARKETS
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Global Neuropharmaceutical Valuation Data

Company	Rating	Ticker	3/18/2025 Price	($MMs) Mkt Cap	($MMs) Ent Val	P/E (x) 2024	Trailing EV Multiples EV/Sales	EV/EBIT	EV/EBITDA
Rated Companies:									
Annovis Bio Inc	HOLD; NR	ANVS-US	$1.78	$35	$10	NM	NM	NM	NM
CervoMed Inc.	BUY; $15 TP	CRVO-US	$9.34	$77	$35	NM	1.75x	NM	NM
Immunic, Inc.	BUY; $13 TP	IMUX-US	$1.13	$102	$56	NM	NM	NM	NM
Cognition Therapeutics, Inc.	BUY; $8 TP	CGTX-US	$0.44	$18	($4)	NM	NM	NM	NM
BioVie Inc. Class A	BUY; $5 TP	BIVI	$1.01	$19	($8)	NM	NM	NM	NM
Not-Rated Companies:									
Eli Lilly and Company		LLY-US	$822.51	$779,730	$773,898	65.91	16.17x	42.74x	38.72x
Vertex Pharmaceuticals Incorporated		VRTX-US	$512.23	$131,536	$127,735	NM	9.06x	22.45x	21.44x
Biogen Inc.		BIIB-US	$143.09	$20,945	$25,219	13.67	2.88x	12.35x	9.38x
Neurocrine Biosciences, Inc.		NBIX-US	$109.70	$10,937	$10,795	41.47	5.76x	21.91x	20.96x
Axsome Therapeutics, Inc.		AXSM-US	$124.76	$6,084	$5,865	NM	10.22x	NM	NM
Supernus Pharmaceuticals, Inc.		SUPN-US	$32.28	$1,802	$1,387	27.39	2.42x	25.59x	11.07x
Anavex Life Sciences Corp.		AVXL-US	$9.31	$792	$669	NM	NM	NM	NM
SAGE Therapeutics, Inc.		SAGE-US	$7.86	$483	($15)	NM	-3.94x	NM	NM
INmune Bio, Inc.		INMB-US	$7.19	$191	$114	NM	1142.28x	NM	NM
AB Science SA		ABSCF-US	$1.57	$91	$73	NM	186.07x	NM	NM
Reviva Pharmaceuticals Holdings, Inc.		RVPH-US	$1.08	$50	$31	NM	NM	NM	NM
Jupiter Neurosciences, Inc.		JUNS	$0.64	$21	$23	NM	NM	NM	NM
Cyclo Therapeutics Inc		CYTH-US	$0.65	$19	$30	NM	16.49x	NM	NM
Athira Pharma, Inc.		ATHA-US	$0.33	$13	($37)	NM	NM	NM	NM
Relmada Therapeutics Inc		RLMD-US	$0.30	$9	($45)	NM	NM	NM	NM
Alzamend Neuro, Inc.		ALZN-US	$0.94	$6	$2	NM	NM	NM	NM
					Average	**37.1x**	**152.3x**	**25.0x**	**20.3x**
					Median	**34.4x**	**9.1x**	**22.4x**	**21.0x**

Source: FactSet

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Risk Factors

The Company Will Need Substantial Additional Funding

BIVI may have to delay, reduce, or eliminate R&D programs or future commercialization efforts if it cannot raise additional capital. These expenses may increase with its ongoing activities, particularly as management continues R&D for device development, continues the growth of its sales and marketing costs, and seeks marketing approval for its products. Additional fundraising efforts may divert management's attention from their day-to-day activities, which may adversely affect the company's ability to advance its product candidates or develop new ones. Additional funding may not be available on acceptable terms or at all. The inability to raise additional capital in sufficient amounts or on acceptable terms may lead management to significantly delay, scale back, or discontinue the development or commercialization of product candidates or other research and development initiatives.

Product Development is Lengthy and Expensive and Has an Uncertain Outcome

The company may incur unexpected costs or experience delays in completing, or ultimately be unable to complete, the development and commercialization of its products. Management is unable to predict when or if any of its product candidates will prove effective or safe in humans or will receive marketing approval. Marketing approval from regulatory authorities for the sale of any product candidate requires extensive clinical trials to demonstrate the safety and efficacy of product candidates in humans. The timely completion and outcomes of product development, clinical trials, and regulatory approvals are uncertain. Clinical testing is expensive, difficult to design and implement, can take many years to complete, and is uncertain as to the outcome. There is no guarantee that these research programs will succeed or result in further commercialization.

Trading Activity Limited

The company's small market cap and the stock's limited trading volume combine to make the shares highly susceptible to volatile market fluctuations. Price changes may occur abruptly and independently of the company's financial performance or fundamental developments.

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Hold	Potential for the securities to decline or appreciate less than 15%
Sell	Potential for the securities to decline more than 15% from the current market price

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Total companies covered: 41 **Data current as of January 6, 2025**	**Buy**	**Hold**	**Sell**	**Under Review**
Brookline Capital Markets Research Coverage	83%	17%	0%	0%
% of companies in each rating category that are investment banking clients	32%	29%	0%	0%