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Delivering effective brain therapies for rare and orphan diseases

Investor Presentation

BTI.V (TSX), BIOAF (OTCQB)
www.bioasis.us

Forward Looking Information

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 and forward-looking information within the meaning of Canadian securities legislation. This information and these statements, referred to herein as "forward-looking statements", are made as of the date of this presentation or as of the date of the effective date of information described in this presentation, as applicable. The forward-looking statements herein relate to predictions, expectations, beliefs, plans, projections, objectives, assumptions or future events or performance (often, but not always, using words or phrases such as "expects", "anticipates", "plans", "projects", "estimates", "envisages", "assumes", "intends", "strategy", "goals", "objectives" or variations thereof or stating that certain actions, events or results "may", "can", "could", "would", "might" or "will" be taken, occur or be achieved, or the negative of any of these terms and similar expressions).

All forward-looking statements are based on current beliefs as well as various assumptions made by, and information currently available to Bioasis. By their very nature, forward-looking statements involve inherent risks and uncertainties, both general and specific, and risks exist that estimates, forecasts, projections and other forward-looking statements will not be achieved or that assumptions do not reflect future experience. For a description of some of the risks that could cause our actual results to vary from those anticipated by forward-looking statements, please refer to the risk factors described in our filings with Canadian securities regulators, available at www.sedar.com. We caution any person reviewing this presentation not to place undue reliance on these forward-looking statements as a number of important factors could cause the actual outcomes to differ materially from the beliefs, plans, objectives, expectations, anticipations, estimates assumptions and intentions expressed in such forward-looking statements.

Bioasis: A Multi-Asset Rare and Orphan Disease Company

Bioasis Technologies is a multi-asset rare and orphan disease company with three Phase 2 clinical stage programs based on epidermal growth factors and a differentiated xB^3 platform for delivering therapeutics across the blood-brain barrier.

STRATEGIC PARTNERSHIPS
janssen | Pharmaceutical Companies of Johnson & Johnson Neuramedy Chiesi prothena
The extensive capabilities of the xB^3 platform continues to be validated by global life sciences companies and provide Bioasis with direct access to key opinion leaders and additional partnering opportunities.

Bioasis' Pipeline

Asset	Indication	Drug Discovery	Pre-Clinical	Phase 1	Phase 2	Phase 3
Epidermal Growth Factor Platform						
CRES101	Optic Neuritis associated with MS					
CRES101	Guillain-Barré Syndrome (GBS)					
CRES101	Chronic Inflammatory Demyelinating Polyneuritis (CIDP)					
CRES202	Alzheimer's Disease					
xB^3 Platform						
xB^3-008	Hunter Syndrome					
xB^3-007	Gaucher Disease, PD, Lewy Body Dementia					
xB^3-004	Multiple Sclerosis, Epilepsy, Autoinflammatory Diseases					
xB^3-009	CLN, Frontotemporal Dementia ALS					

Partners Utilizing xB^3 Platform

janssen | Pharmaceutical Companies of Johnson & Johnson

Neuramedy

Chiesi

prothena

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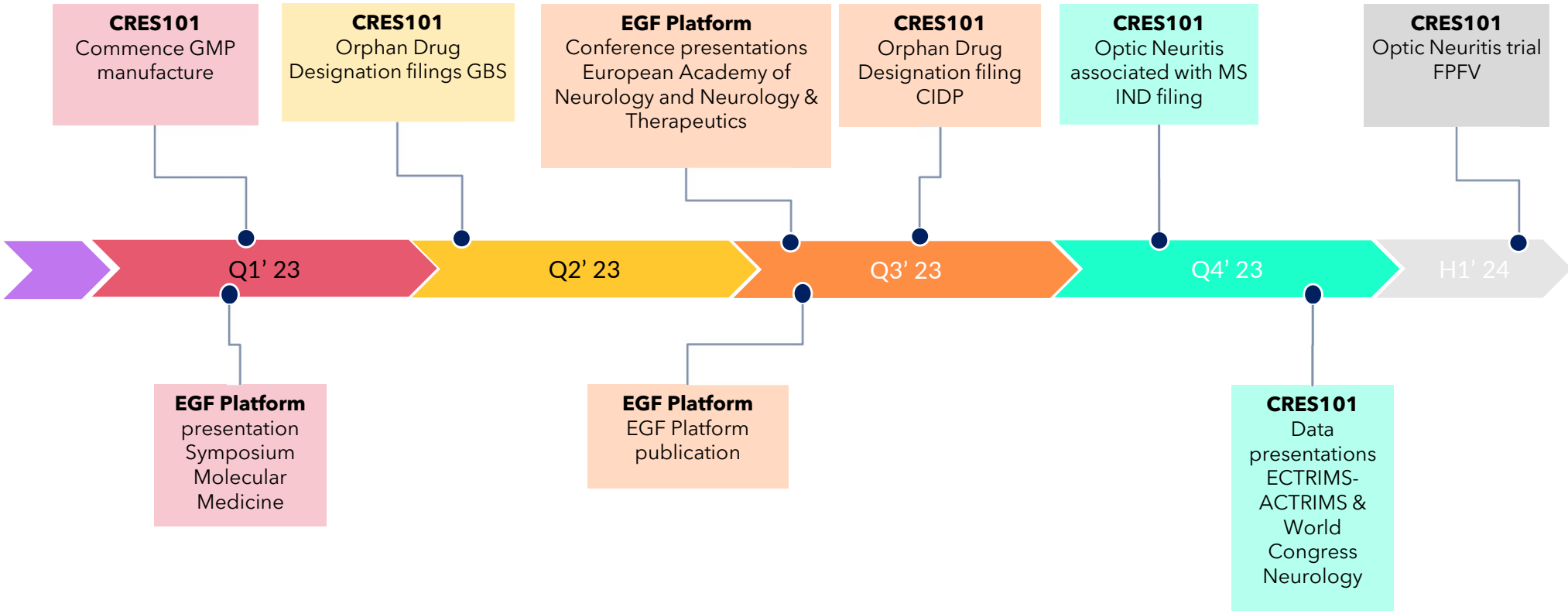
Anticipated Catalysts, subject to financing



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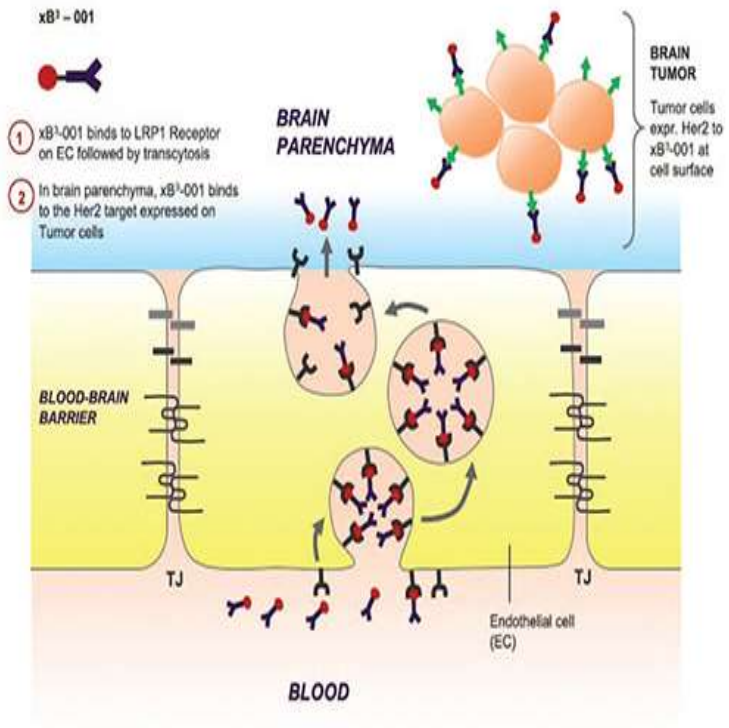
xB^3 Platform

- xB^3 platform utilizes the low-density lipoprotein-related protein 1 (LRP1) receptor
- Carries payloads across the BBB via process of endocytosis and transcytosis
- Applicable to variety of actives including monoclonal antibodies, enzymes and oligonucleotides



Partners Utilizing xB^3 Platform

Chiesi

Neuramedy

prothena

janssen | PHARMACEUTICAL COMPANIES OF Johnson & Johnson

1Thom G. et al. (2018) J Cereb Blood Flow Metab. ePub May 30, 2018.
2Eyford B.A. et al. (2021) Front Mol Biosci. Mar 26;8:611367
3Jin X. et al. (2022) Mol Ther Methods Clin Dev. Apr 19;25:370
4Singh C.S.B. (2021) Front Neurosci. V.15:596976
5Nounou M.I. et al. (2016) Pharm Res. Dec;33(12):2930-2942

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xB^3 Platform

The xB^3 Platform Technology delivers therapeutics across the blood brain barrier to treat orphan and rare genetic diseases and has been demonstrated to outperform competing blood brain barrier technologies.

Features	Bioasis' xB^3 Platform	Denali (NYSE:DNLI)	Genentech	Roche (SWX:ROG)	Armagen	Angiochem
% injected dose in brain	4-6%	1-1.5%	1-1.5%	1-1.5%	1-1.5%	1-1.5%
Mode of Action	LRP1	TfR	TfR	TfR	TfR & IR	LRP1
Payload Modalities						
Antibodies	✓	✓	✓	✓	✓	✓
Enzymes	✓	✓			✓	
siRNA	✓					
Small Molecules	✓					

Outperforms competing blood brain barrier technologies

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Bioasis vs. Denali (NYSE:DNLI)

Bioasis' xB^3 Platform Technology has been demonstrated to outperform Denali's blood brain barrier technology however, Bioasis continues to be significantly undervalued.

In USD$ millions	NYSE:DNLI	TSXV:BTI
Market Cap:	$2,628.6	$11.2
Debt:	$62.7	$1.7
LTM Product Rev:	$0.0	$0.0
Indication:	Hunter Syndrome	Hunter Syndrome
Clinical Phase:	Phase 2/3	Preclinical
Premium to BTI:	**23,470%**	
Partners:	Biogen, Takeda, SANOFI	janssen, Johnson & Johnson, Neuramedy, Chiesi, prothena

Public Comparable in Guillain-Barré Syndrome

Annexon's lead clinical candidate, ANX005, is being evaluated in Guillain-Barré Syndrome and is in a Phase 2/3 trial with data anticipated in 2023.

In $ millions	NASDAQ:ANNX	TSXV:BTI
Market Cap:	$127.3	$11.2
Debt:	$34.2	$1,7
LTM Rev:	$0.0	$0.0
Indication:	Guillain-Barré	Guillain-Barré
Clinical Phase:	Phase 2/3	Phase 2 Ready
Premium to BTI:	**1,136%**	

Targeted Rare and Orphan Disease Market

Indication	Treatment(s)	Prevalence	Key Market Players
Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)[1]	Glucocorticoids IV immunoglobulin Plasma Exchange	5-7 cases per 100,000	Grifols SA CSL Limited Shire Kedrion Takeda
Guillain-Barré Syndrome (GBS)[2]	General medical and nursing care Physiotherapy Rehabilitation IV immunoglobulin Plasma Exchange	1-2 cases per 100,000	Grifols SA CSL Limited Takeda Octapharma AG Kedrion Biopharma Annexon Biosciences
Gaucher Disease[3]	Enzyme Replacement Therapy Substrate Reduction Therapy	6,000 in the U.S. Most common genetic disorder in Ashkenazic Jewish ancestry	Sanofi Pfizer Shire Actelion
Hunters Syndrome[4]	Enzyme Replacement Therapy	1 per 100,000	Sanofi Shire GC Pharma

1 https://rarediseases.org/rare-diseases/chronic-inflammatory-demyelinating-polyneuropathy/
2 https://rarediseases.org/rare-diseases/guillain-barre-syndrome/
3 https://rarediseases.org/rare-diseases/gaucher-disease/
4 https://my.clevelandclinic.org/health/diseases/17932-hunter-syndrome

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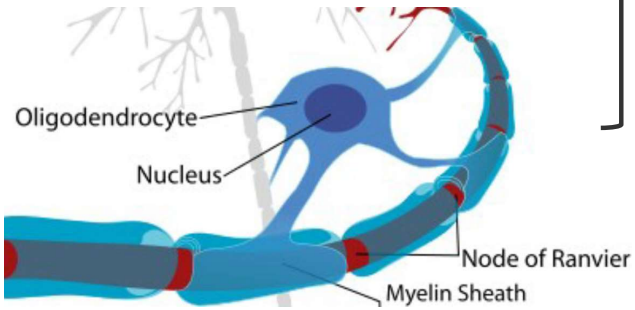
Epidermal Growth Factor (EGF) Platform
Novel Treatment Approach For Neurodegenerative Disorders

- **Epidermal Growth Factor ("EGF")**
  - Protein that stimulates cell growth and differentiation
  - Stimulates oligodendrocyte and Schwann cell differentiation and maturation for remyelination

- **Neurodegenerative Disorders**
  - EGF levels are deficient in neurodegenerative disorders



- **EGF Treatment**
  - Stimulates myelin regeneration
  - Protects nerve cells

No effective treatment exists that addresses the loss of myelin as the underlying cause of morbidity in neuroinflammatory diseases

1. Bieber et al., Proc Natl Acad Sci U.S.A, 2010;107,792-720 2. Chandran et al., Glia, 1998,24,382-9 3. Gudi et al., PLoS One, 2011;6(7):e22623
4. Knapp and Adams, Exp Cell Res, 2004,296,135-44 5. Toma et al., J Neurosci, 1992,12,2504-15 6. Scalabrino G. Cell Mol Neurobiol. 2022,42,891-916
7. Gonzalez-Perez O et al Stem Cells 2009,27:2032–2043 8. Nicoletti F et al J Neuroimmunol. 2019,332,224-232.
9. Evaluate pharma https://www.evaluate.com/vantage/articles/analysis/spotlight/remyelinating-agent-remains-distant-h

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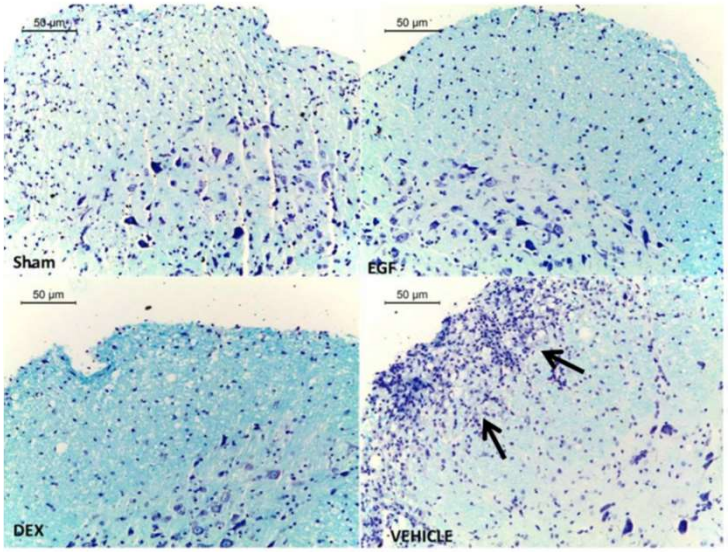
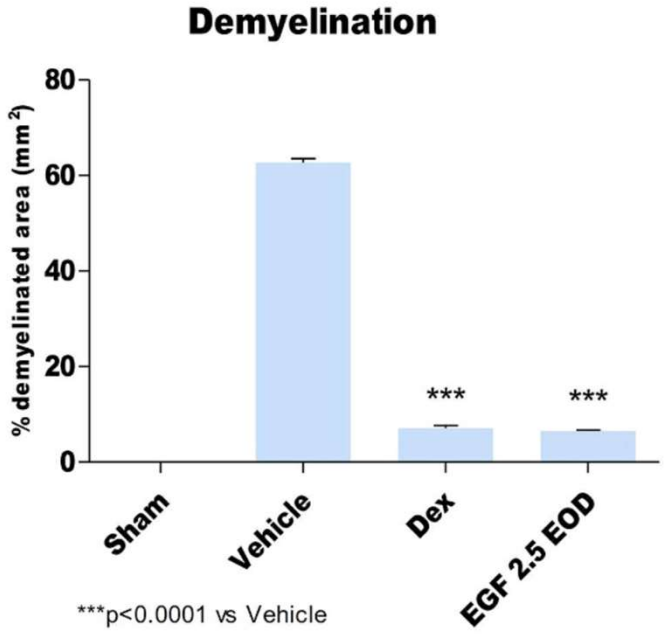
EGF Decreases Inflammation and Prevents Demyelination



- The staining with LFB shows the formation of demyelination plaques occurred in the EAE group (as shown by the arrows).

- Myelin degradation was completely attenuated in EAE mice treated with EGF.

- MOG-induced EAE (a model of MS) mouse brain demonstrated absence of any demyelination when treated with EGF (Luxol fast blue stained sections of spinal cord).



1. Nicoletti F et al J Neuroimmunol. 2019,332,224-232.

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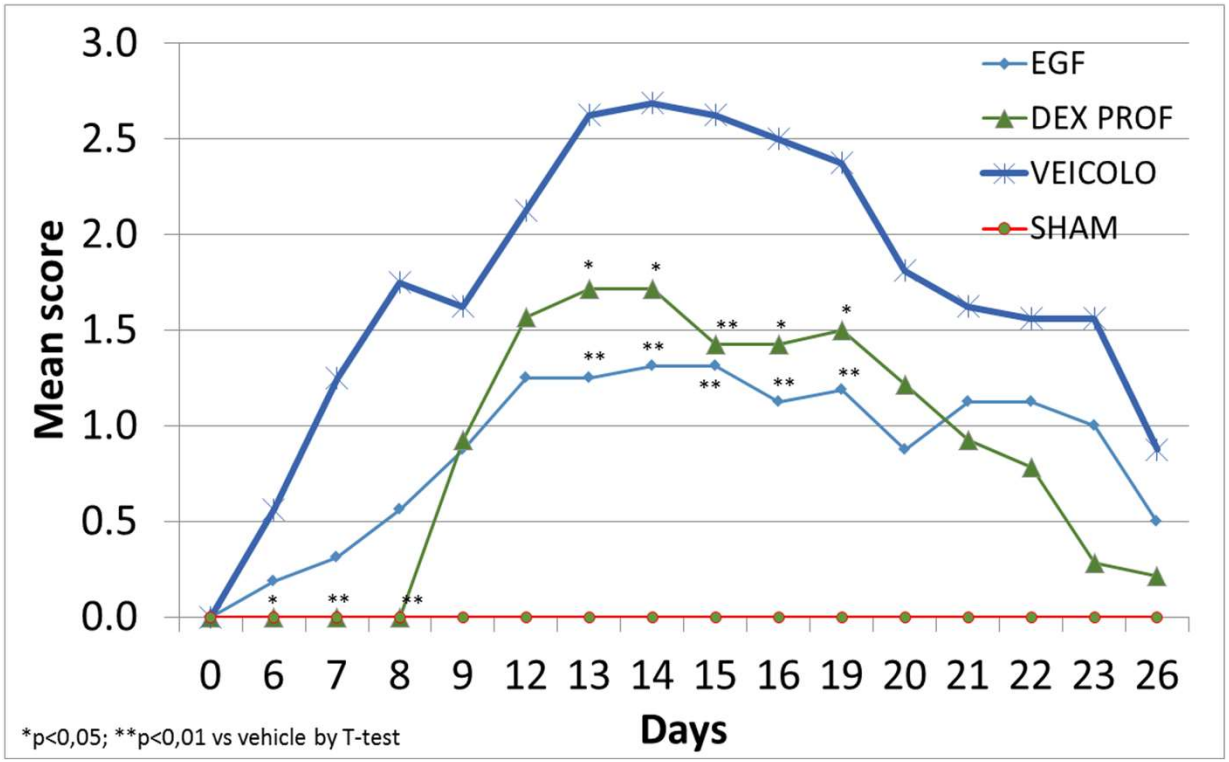
EGF Effectively Treats Neuroinflammation in EAN

- EAN is the animal model of CIDP and Guillain-Barré Syndrome



Nicoletti et al J Neuroimmunol. 2019

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CRES101
Optic Neuritis

- **CRES101 is being developed for optic neuritis associated with multiple sclerosis to reduce demyelinating inflammation of the optic nerve.**

- **Treatment Options:**
  - IV Steroids
  - IV Immunoglobulins
  - Plasma Exchanges

PREVALENCE/INCIDENCE	MARKET
US Annual Incidence estimated at **6.4** per **100,000**[1]	Optic neuritis treatment market US**$204.97M** in **2022** ↑ US**$318.31M** in **2032**[3]
Recent UK study demonstrated annual incidence of **3.7** and a prevalence of **114** per **100,000**[2]	Market expected to expand at a compound annual growth rate (CAGR) of **4.5%** through to **2032**[3]

Efficacy demonstrated in optic neuritis associated with MS would support development in chronic progressive MS

1. Percy et al; Optic Neuritis and Multiple Sclerosis An Epidemiologic Study
2. Trends in Optic Neuritis Incidence and Prevalence in the UK and Association With Systemic and Neurologic Disease
3. Future Market Insights

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Proposed Phase 2 Study Design
CRES101 – Optic Neuritis

A double-blind Phase 2 POC study of CRES 101 (EFG 1-48) in MS-related optic neuritis is planned subject to financing.

CRES101 Phase 2 POC[1-5]	
Population	▪ 40
Design:	▪ 2 groups receiving either EGF 1-48 subcutaneously on alternate days daily for 10 days or placebo as an add-on therapy to standard methylprednisolone treatment (250mg every six hours)
Primary Outcome:	▪ Change in retinal nerve fiber layer (RNFL) thickness after 16 weeks
Secondary Outcome:	▪ Optic nerve atrophy assessed by MRI ▪ Changes in visual acuity ▪ Visual field ▪ Visual Evoked Potentials (VEPs)
Duration:	▪ 1 year

1. Sullivan PB et al J Ped Surgery, 2007,42,462-469 2. Breider MA et al Vet Pathol 1996;33:184-94.
3. Reindel JF et al Toxicol Pathol 1996;24(6):669-80. 4. Henck JW et al Toxicol Sci 2001;62(1):80-91
5. Suhs KW et al Ann Neurol 2012,72,199-210

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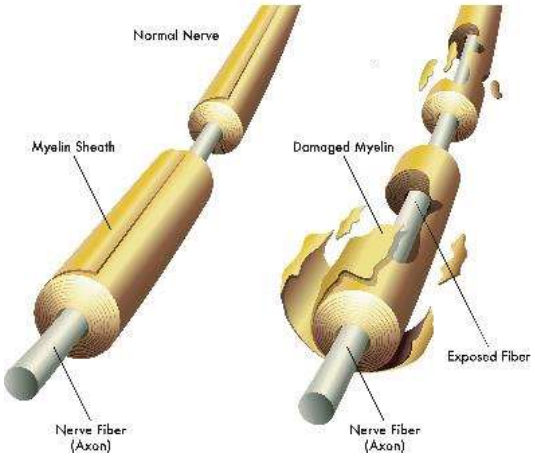
CRES101
Guillain-Barré Syndrome (GBS)

CRES101 is indicated for Guillain-Barré Syndrome, a rare and progressive disease characterized by inflammation of the nerves (polyneuritis) causing muscle weakness, sometimes progressing to complete paralysis.



PREVALENCE/INCIDENCE		MARKET
3000 to 6000 people develop GBS every year in the US[1]	**150,095** total cases of GBS worldwide[2] in 2019	Global therapy market estimated at US**$491.1M** in **2020** US**$679.6M** in **2027**
6.4% global increase in the age-standardized prevalence of GBS per **100,000** population between **1990** and **2019**[2]		**CAGR of 4.7%** over the analysis period **2020 to 2027**[3]

1. CDC
2. Journal of Neuroinflammation
3. Research and Markets

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CRES101 Proposed Phase 2 Design in Guillain-Barré Syndrome

A randomized placebo-controlled double-blind Phase 2 POC study of CRES101 in Guillain-Barré Syndrome

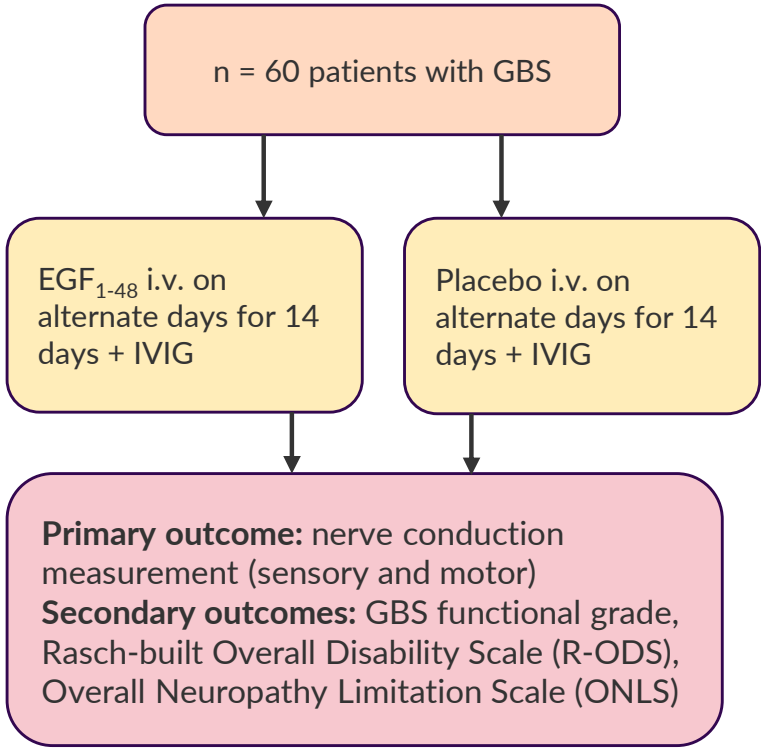
Analogy to Misawa et al study 2018 on eculizumab in GBS patients

Sixty patients with GBS randomized to 2 groups receiving either EGF_{1-48} subcutaneously on alternate days daily for 14 days or placebo as an add-on therapy to IVIG treatment.

Primary outcome:
Nerve conduction (sensory and motor at 2 weeks, 4 weeks and 8 weeks)

Secondary outcomes:
GBS functional grade,
Rasch-built Overall Disability Scale (R-ODS),
Overall Neuropathy Limitation Scale (ONLS)

Approximate duration: 1 year, subject to financing



Misawa et al Lancet Neurol 2018:17:519-29

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CRES101
Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP)

- **CRES101 is indicated for CIDP, a rare neurological disorder in which there is inflammation of nerve roots and peripheral nerves and destruction of the fatty protective covering (myelin sheath) of the nerve fibers.**

- **Treatment Options:**
  - Glucocorticoids
  - IV Immunoglobulins
  - Plasma Exchanges

PREVALENCE/INCIDENCE		MARKET
40,000 patients (approximately) affected in the United States[1]	Incidence of **0.7** to **1.6** cases per **100,000 persons per year[1]**	Global therapy market estimated to reach US**3.9billion** by the end of **2023[2]**
overall prevalence is estimated at **4.8 to 8.9** cases per	**100,000 persons[1]**	CAGR **of 6.2%[2]**

1. AJMC
2. Marketwatch

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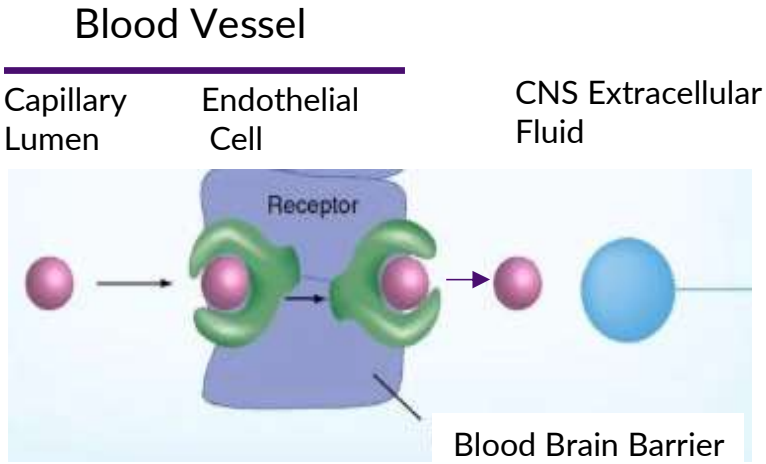
The Bioasis Platform Technology
Active Transport Across the BBB via the LRP1 Receptor



xB^3 Peptide

Derived from an iron-binding human protein found at low concentrations in the blood

- xB^3 has been optimized by Bioasis' scientists to its key constituents (12 amino acids)
- xB^3 has shown improved brain penetration over the full-length protein

Mechanism of Action (MOA)

xB^3 binds to, and moves into cells via receptor-mediated endocytosis/transcytosis involving the Low-Density Lipoprotein Receptor-related protein (LRP1) receptor

- High efficiency receptor with fast endocytosis and recycling
- LRP1 is highly expressed in critical brain regions and across multiple brain cell types
- LRP1 is overexpressed in multiple disease states including brain cancers, Alzheimer's disease and Parkinson's disease

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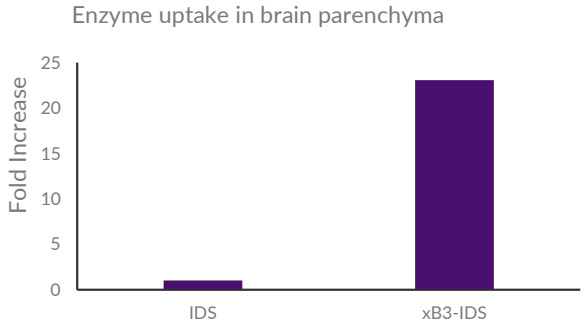
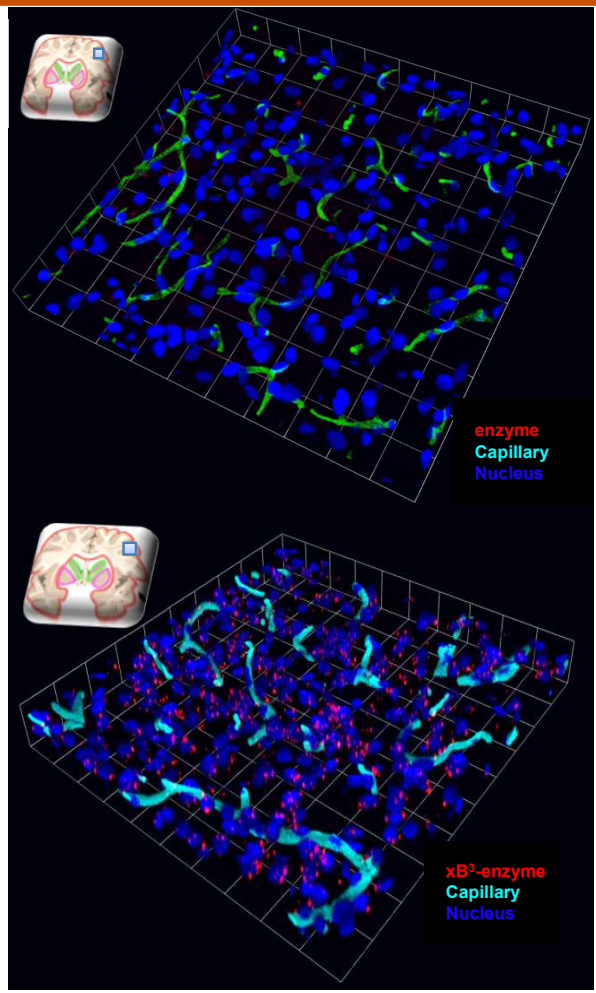
xB^3 can Effectively Deliver Enzymes to Treat Lysosomal Storage Disorders

Hunter Syndrome (MPS II)

- Lysosomal Storage disease, MPS II is caused by an iduronate 2-sulfatase (I2S) enzyme deficiency
- Currently CNS effects are untreatable

Bioasis' xB^3 peptide-I2S fusion molecule increased I2S uptake into the brain and was accompanied by cellular and biochemical changes characteristic of enzyme activity.

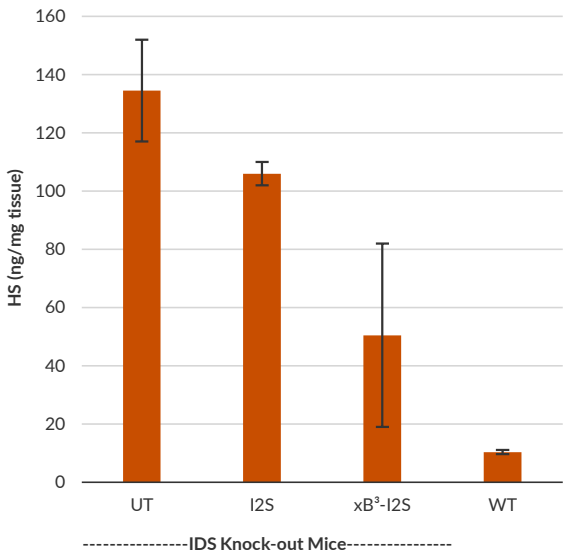




xB³-I2S Treatment Facilitated the Reduction of Heparan Sulfate Levels, Reduced Number of Storage Cell Vacuoles & Reduction in Number of Lysosome Vesicles in the Brain

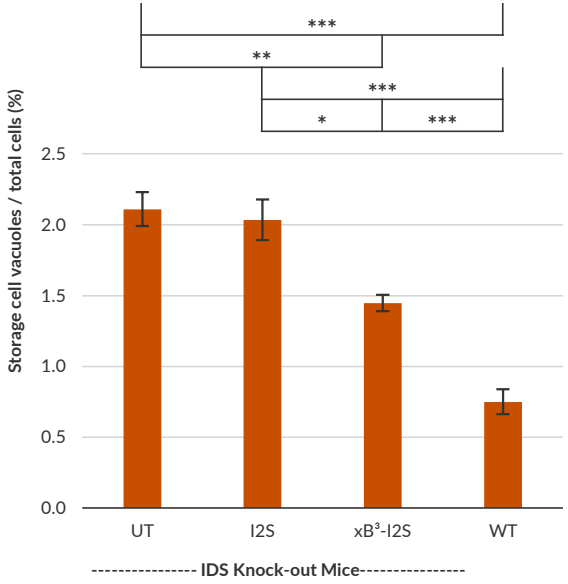
- Significant reduction in brain heparan sulfate accumulation, cell vacuolation and lysosome vesicles in a Hunter Syndrome mouse model
- Increase in brain heparan sulfate accumulation, cell vacuolation and lysosome numbers are hallmarks of Hunter Syndrome

Reduction in Heparan Sulfate Accumulation



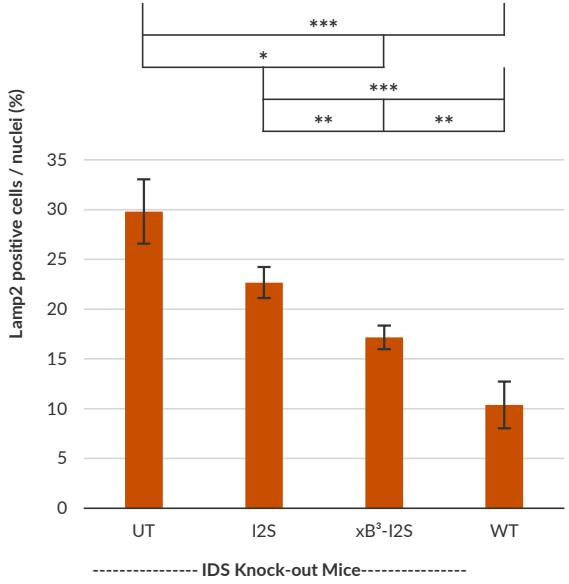
Mean ± SEM; n= 2-3

Reduction in Number of Storage Cell Vacuoles



Mean ± SEM (***P≤0.0001, **P≤0.001, *P≤0.01, One-way ANOVA); n=4-5

Reduction in Number of Lysosome Vesicles



Mean ± SEM (***P≤0.005, **P≤0.05, *P≤0.01, One-way ANOVA); n=5

Work performed at University of Padova, Italy

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Investment Highlights

- **Phase 2 ready molecule already tested in humans**
- **Rapid POC clinical trials in rare/orphan indications**
  - Guillain-Barré Syndrome (GBS)
  - Chronic Inflammatory Demyelinating Polyneuropathy (CIDP)
  - Optic Neuritis associated with MS
- **Validated xB^3 platform being utilized by global life science partners**

Partners Utilizing xB^3 Platform

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Delivering effective brain therapies for rare and orphan diseases

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