BRIGHT MINDS

Novel Drugs for Targeted Treatment of
CNS & Neuropsychiatric Disorders

March 2026

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Bright Minds is a clinical-stage biotechnology company focused on developing next-generation serotonergic treatments for patients with neurological and psychiatric disorders

- Proprietary platform of highly selective 5-HT2A and 5-HT2C agonists
- Robust pipeline of innovative compounds to address neuropsychiatric and neurodegenerative target areas where current treatments are inadequate
- Lead asset, BMB-101, had positive Phase 2 topline results, potentially addressing a large treatment gap in adult patients with drug-resistant Absence Seizures
- Estimated $21 billion refractory market in Absence Seizures and Developmental and Encephalopathic Epilepsies

Creating New Chemical Entities That Target Serotonin Signaling

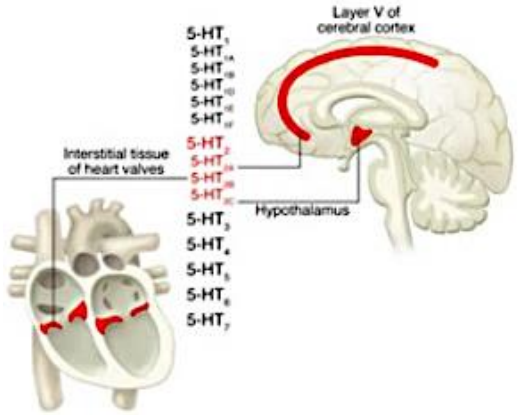
Key 5-HT$_2$ Receptors Targets

5-HT$_{2A}$ Agonists
Depression, PTSD

5-HT$_{2C}$ Agonists
Epilepsy, Impulsivity control, Hyperphagia

5-HT$_{2A/2C}$ Agonists
Depression, Anxiety, Pain, Migraine

- Based on a proprietary chemistry platform Bright Minds have developed highly selective 5-HT$_{2A}$ and 5-HT$_{2C}$ agonists without 5-HT$_{2B}$ activity
- 5-HT$_{2B}$ activation is associated with undesirable cardiac valvulopathy



Serotonin (5-HT) is the most prominent neurotransmitter in the brain and modulates many functions

Pipeline

Rich and diverse portfolio in neurology and psychiatry with multiple programs

Lead	Indications	Research	Phase 1	Phase 2	Phase 3

5-HT_{2C} agonists

BMB-101	DEE Absence seizures	Clinical Studies – Phase 2 topline announced Registrational trials in DEE Registrational trials in absence seizures
BMB-101	Prader-Willi Syndrome	Clinical Studies – Phase 2a - PoPh
BMB-105	Prader-Willi Syndrome	Clinical development

5-$HT_{2A/2C}$ agonists

BMB-201	Depression, Pain, Headache	Preclinical

5-HT_{2A} agonists

BMB-202	Depression (Fast-onset)	Preclinical

Epilepsy Program

BMB-101

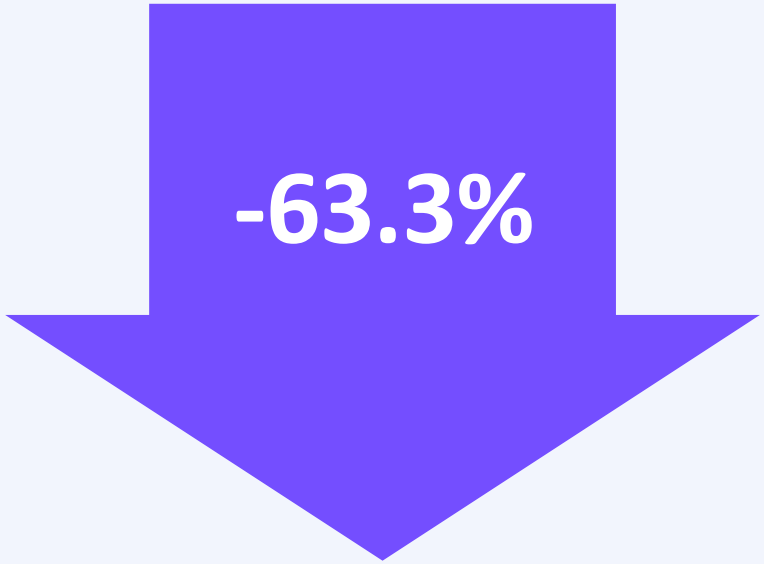
Novel 5-HT_{2C} Selective Agonist

BMB-101 demonstrates efficacy in drug resistant Absence Seizures and DEE

Absence Seizures

Median reduction of absence seizures ≥ 3s as measured by 24h EEG

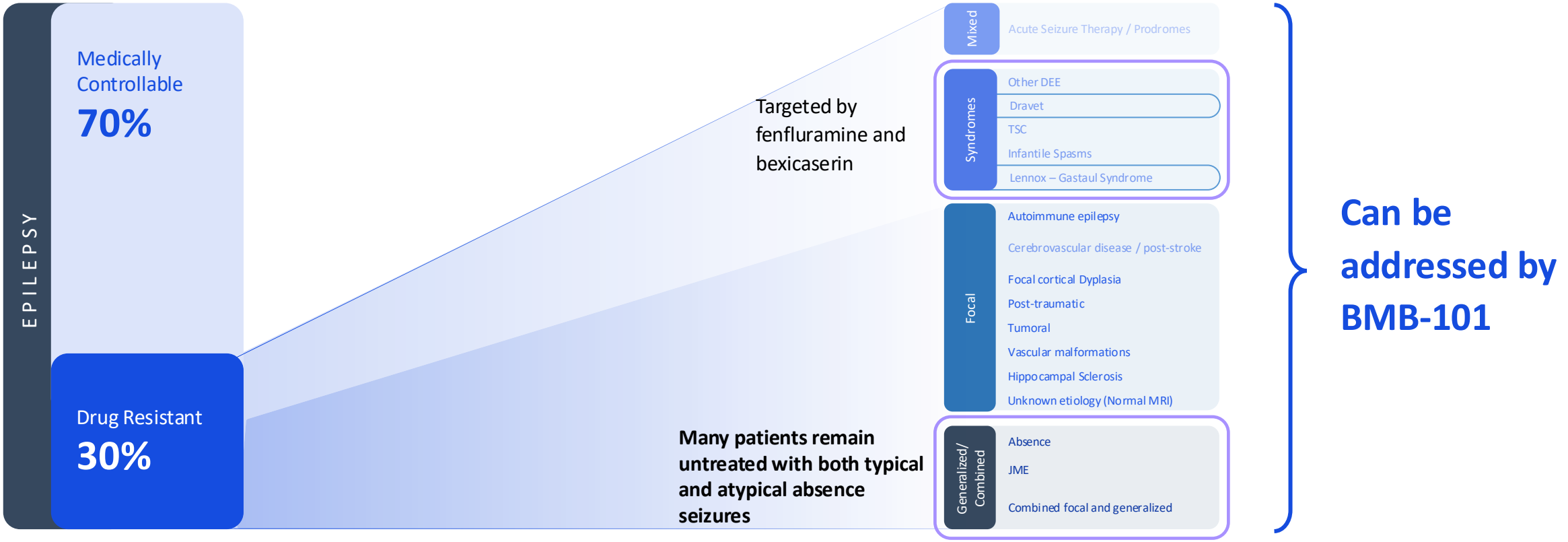
DEE



Median reduction in major motor seizures as measured by seizure diary

High unmet need beyond DEE

Addressable targets for BMB-101 range across multiple epilepsies



Today Suppression of seizures in Broad Epilepsy Population (~65 Million)

Future Focus on epilepsies without treatments

Semah F, et al. Is the underlying cause of epilepsy a major prognostic factor for recurrence? Neurology. 1998;(51):1256-1262
Brenner T et al. Prevalence of neurologic autoantibodies in cohorts of patients with new and established epilepsy. Epilepsia. 2013;54(6):1028–1035

Image credits: UCB

BMB-101 is uniquely positioned to address major unmet needs in epilepsy

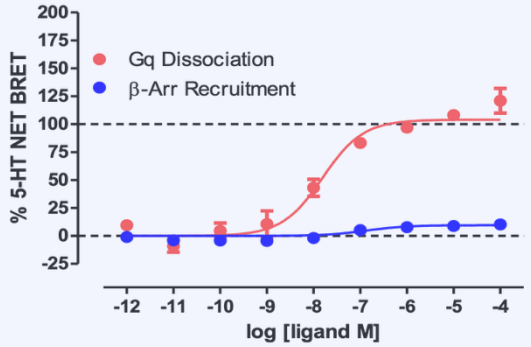
Highly selective 5-HT2C agonist

Compound	5-HT$_{2A}$	5-HT$_{2B}$	5-HT$_{2C}$
BMB-101	2280	>10000	16.2
Nor-Fenfluramine	82.8	11.6	2.5
Lorcaserin	50.1	67.4	2.4
Bexicaserin	>10000	>10000	120

Validated mechanism of action in DEEs

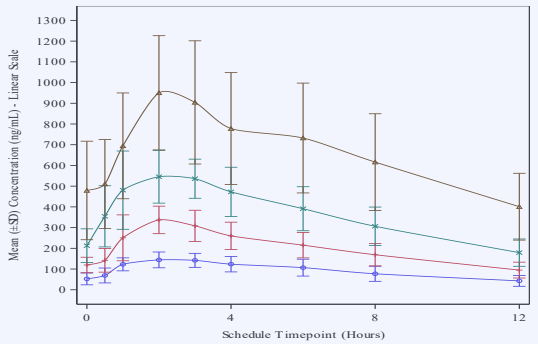
Improved safety profile

The only G protein-biased 5-HT2C agonist



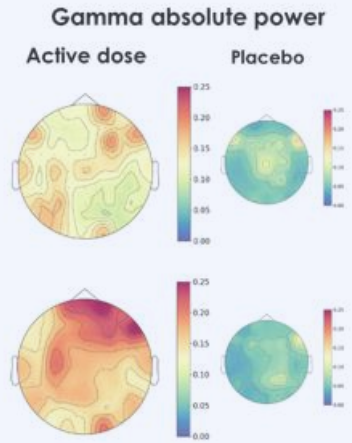
Sustained chronic effect via reduced tolerance

Safety and PK/PD properties validated in Phase 1



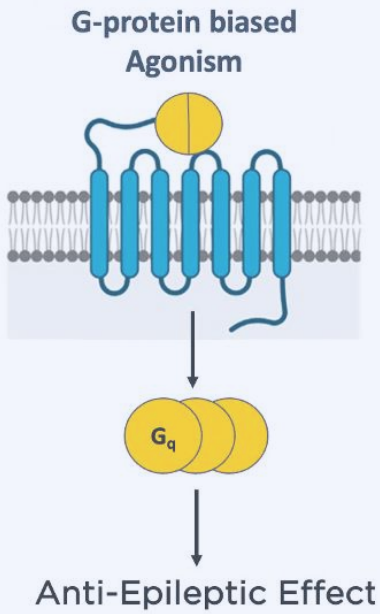
Potential for a more convenient once daily formulation

Proof of mechanism demonstrated in Ph.1 Increased gamma-power on qEEG

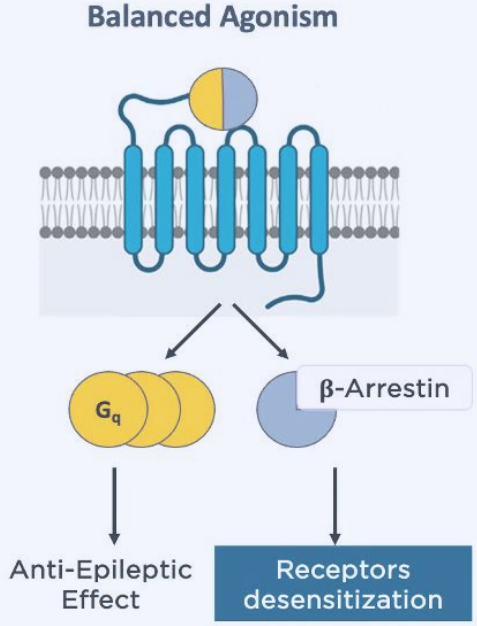


Additional behavioral/cognitive benefits

Novel 5-HT_{2C} mechanism to avoid tolerance pathways



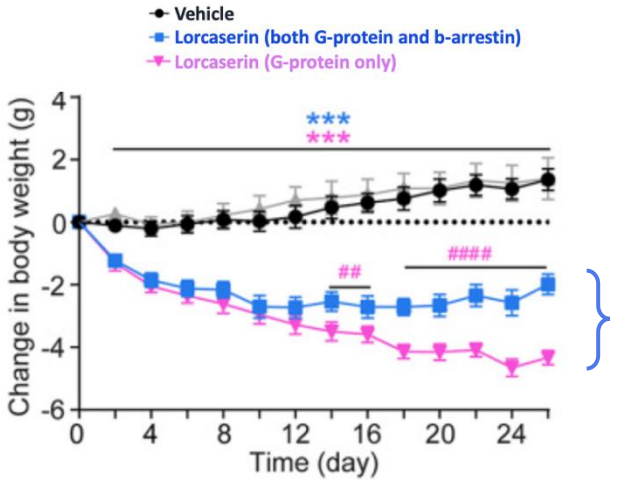
BMB-101



Fenfluramine
Lorcaserin
Bexicaserin

Beta-arrestin activation is associated with receptor desensitization and the development of tolerance.

BMB-101 is designed to avoid b-arrestin activation and produce sustained effect.



Potential for improved efficacy with a G-protein biased agonist

Deactivation of b-arrestin produced a superior and sustained effect in long-term Lorcaserin use (in vivo DIO study)

[1]He Y, et al. **Barbadin Potentiates Long-Term Effects of Lorcaserin on POMC Neurons and Weight Loss**. J Neurosci. 2021 Jun 30;41(26):5734-5746. doi: 10.1523/JNEUROSCI.3210-20.2021.

BMB-101 – Novel scaffold 5-HT_{2C} agonist

	BMB-101	Fenfluramine/ Norfenfluramine	Bexicaserin
Lack of 5-HT_{2B} liability (related to cardiac toxicity)	✓	x	✓
5-HT_{2C} Biased Agonism (Sustained efficacy)	✓	x	x
Can be Dose-optimized	✓	x	x
Increased Frontal Gamma power on qEEG	✓	Not reported	Not reported
Dosing	Once/Twice daily	Twice daily	Three times daily
Development Stage	Phase 2 completed	Approved	Phase 3
Indications	**Broad DEE** **Absence Epilepsy**	Dravet Syndrome LGS	Dravet Syndrome/LGS → Broad DEE

BMB-101 Phase 1 study

Favorable Safety & Tolerability Results Observed

Safety and tolerability

- No SAEs observed, all AEs were transient. Most common adverse effect - oral paresthesias (related to the sweet taste of the drug product)
- Most common on target AE – Headache, Nausea and photophobia
  - Common AEs for serotonergic drugs
  - Only seen at the top dose (2-3x of predicted therapeutic dose)
- Lower incidence of somnolence and GI side effects than with other 5-HT_{2C} agonists

Target engagement:

- Transient dose-dependent prolactin release
- Central target engagement by qEEG and Potential for improved cognitive performance (increase in gamma power)*

Favorable PK:

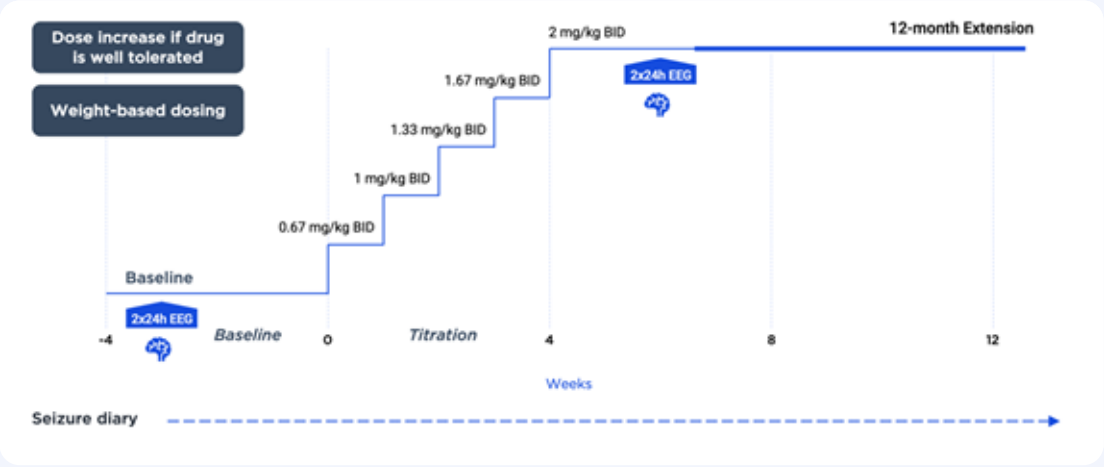
- Dose proportionality observed in SAD and MAD study. No significant food effects observed

BMB-101 Ph.2 study in adult patients with Absence Seizures or DEE

An Open-Label Phase 2 Study To Evaluate the Efficacy, Safety and Tolerability of BMB-101 in Adults

Absence Seizures Arm

Seizure frequency measured using 24h ambulatory EEG



Key entry criteria:

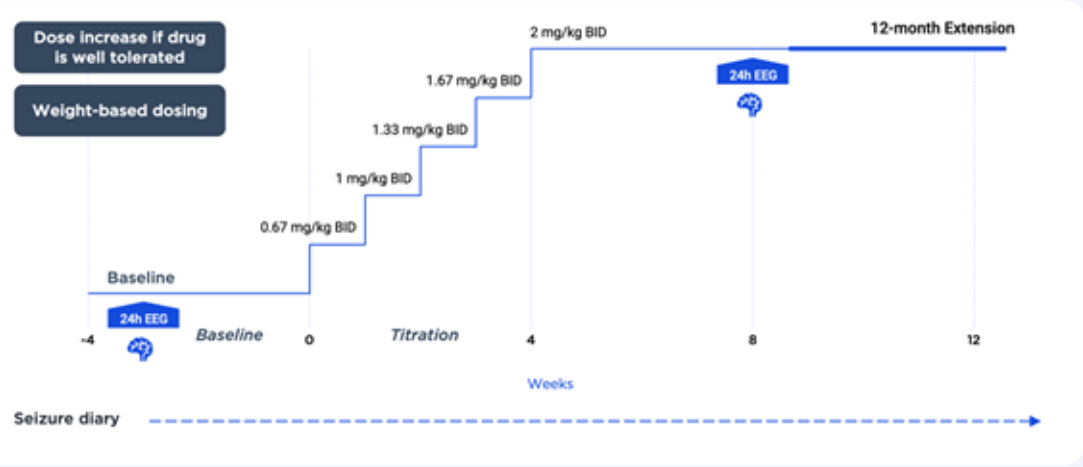
- Patients with drug-resistant absence epilepsy with an average of ≥4 absence seizures per 24 hours during baseline, as measured on 24-hour EEG

Key endpoints:

- Safety and tolerability
- Change in seizure frequency based on 24h EEG

DEE Arm

Seizure frequency measured by seizure diary



Key entry criteria:

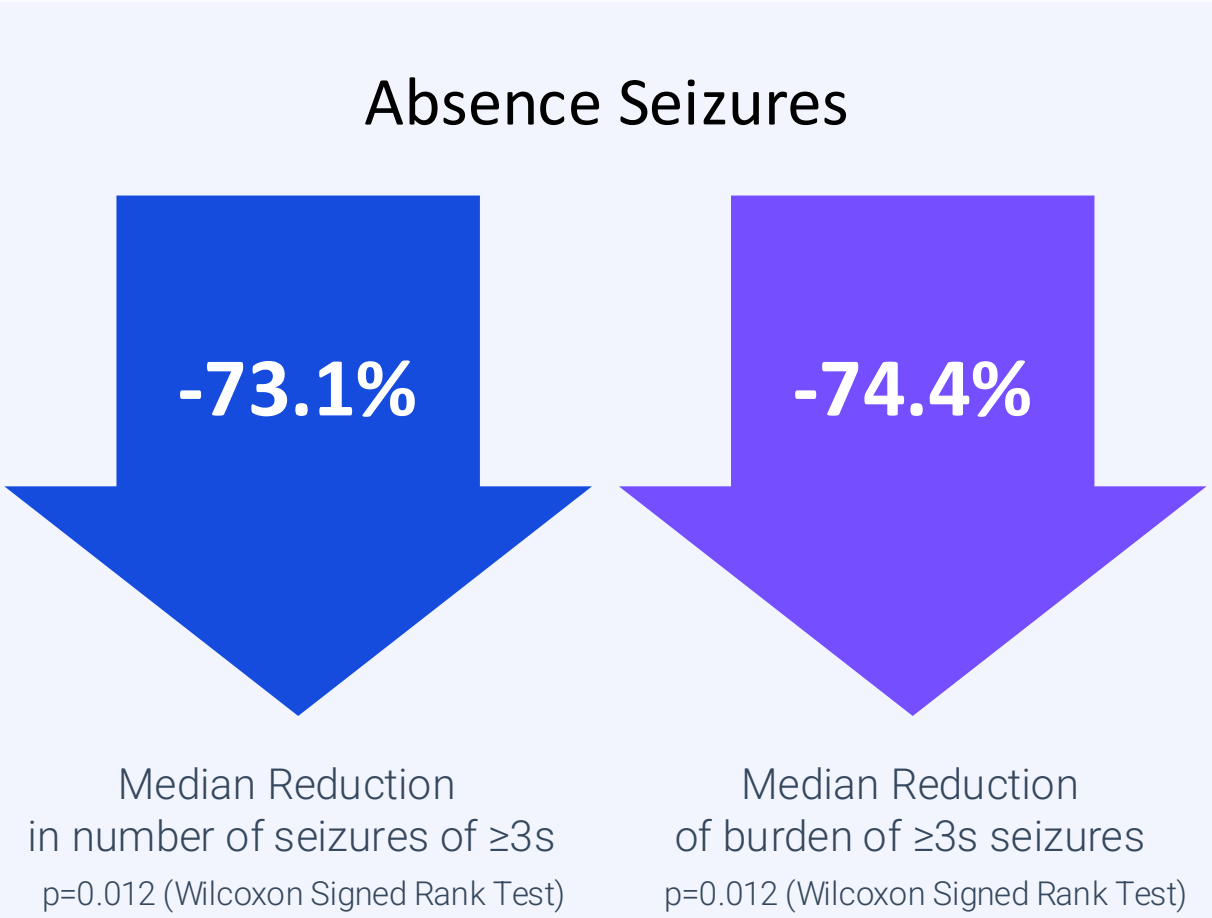
- Patients with drug resistant DEE with ≥ 4 seizures during 4-week baseline (based on seizure diary)

Key endpoints:

- Safety and tolerability
- Change in major motor seizure frequency based on seizure diary

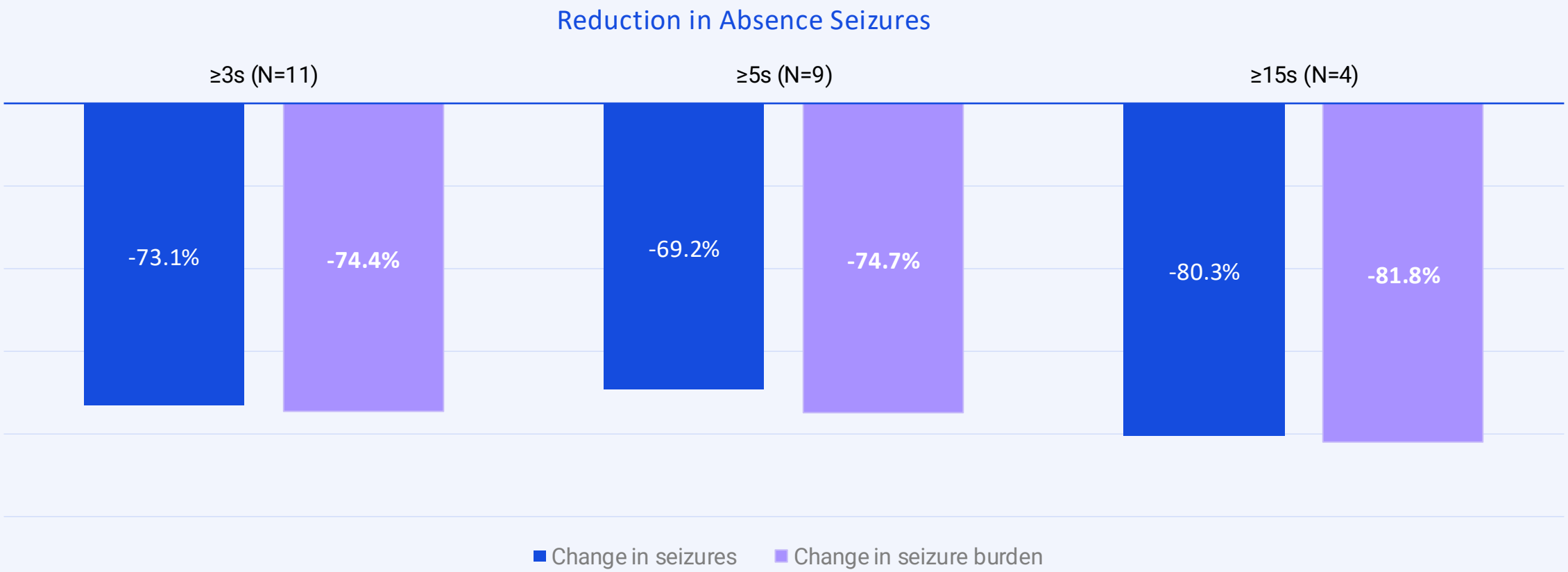
First drug to demonstrate efficacy in Absence Seizures using objective 24h EEG measures

Seizure Burden – sum of individual seizures on EEG with a duration of ≥3s



- Patients with uncontrolled absence seizures
- Seizures are counted based on 2x24h ambulatory EEG recordings done at baseline and maintenance
- EEG analysis and seizure counting is done by independent and blinded readers

Robust reduction of Absence Seizures regardless of seizure duration

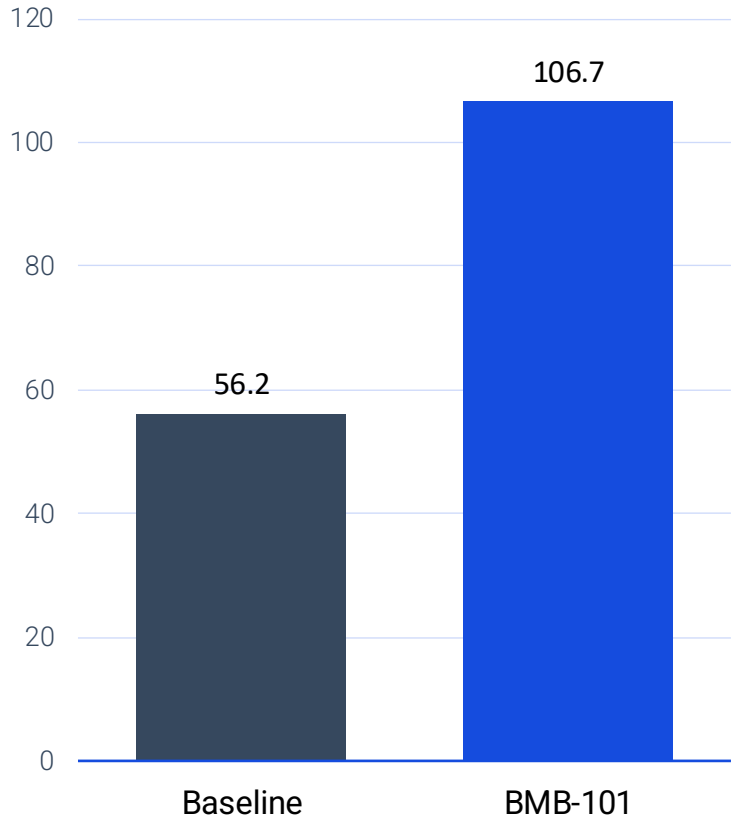


Topline data
N – number of patients with seizures of specific duration (≥3, ≥ 5, ≥ 15)
Seizure Burden – sum of individual seizures on EEG with a duration of ≥3s

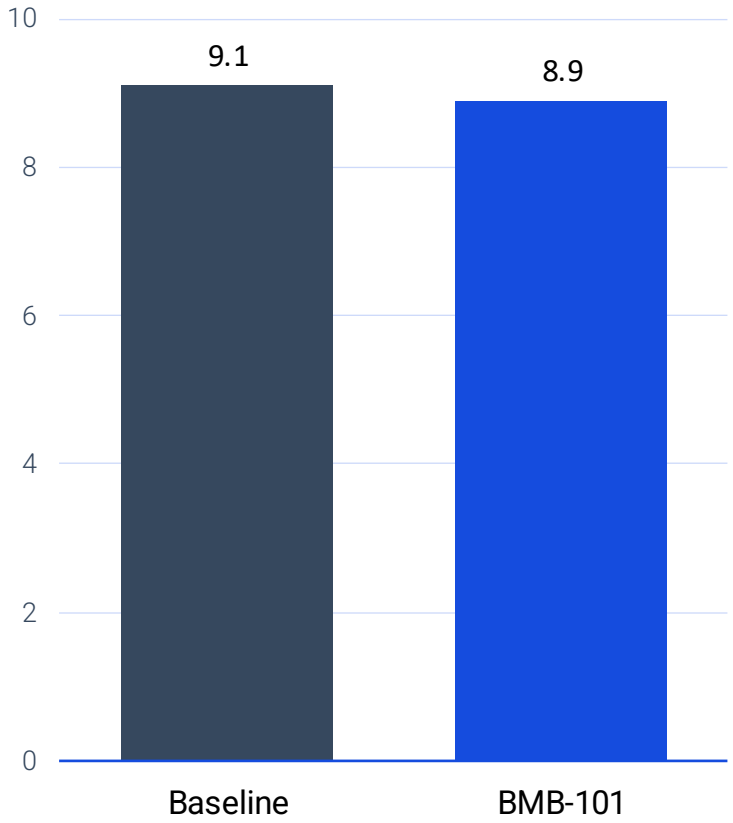
BMB-101 improves REM sleep in patients with Absence Seizures without increasing sleep duration

Increase in REM sleep duration

REM sleep duration, minutes

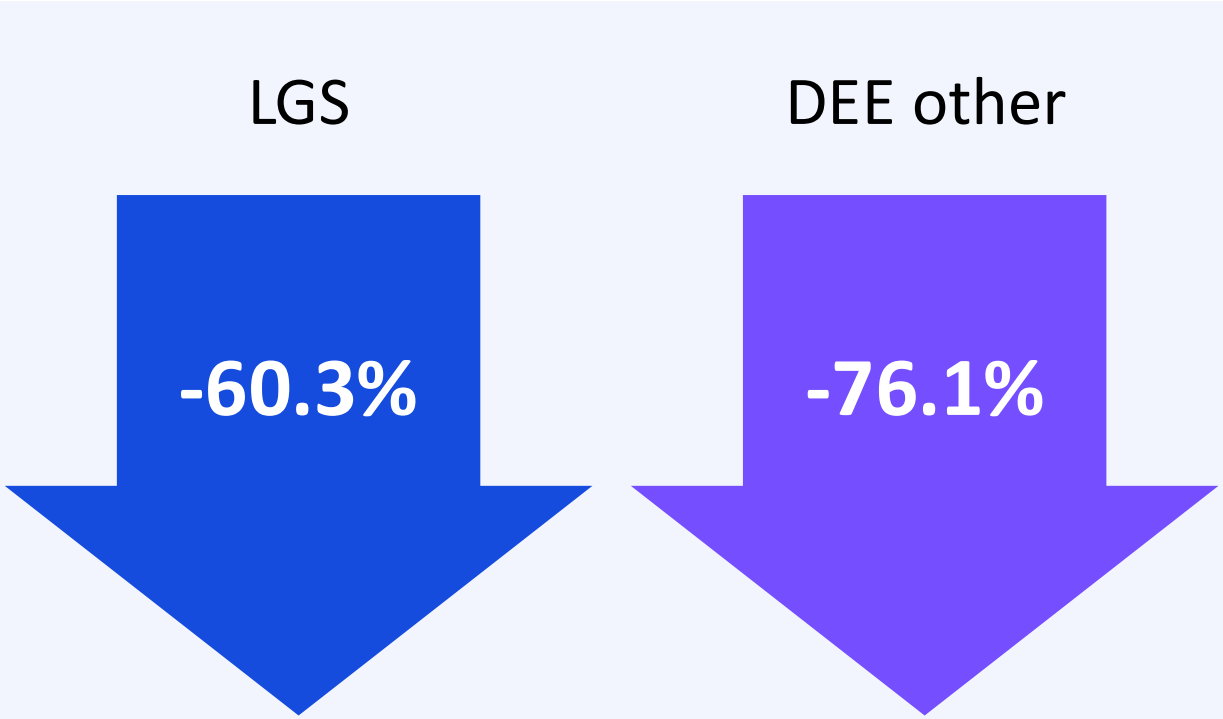


Overall sleep duration, hours

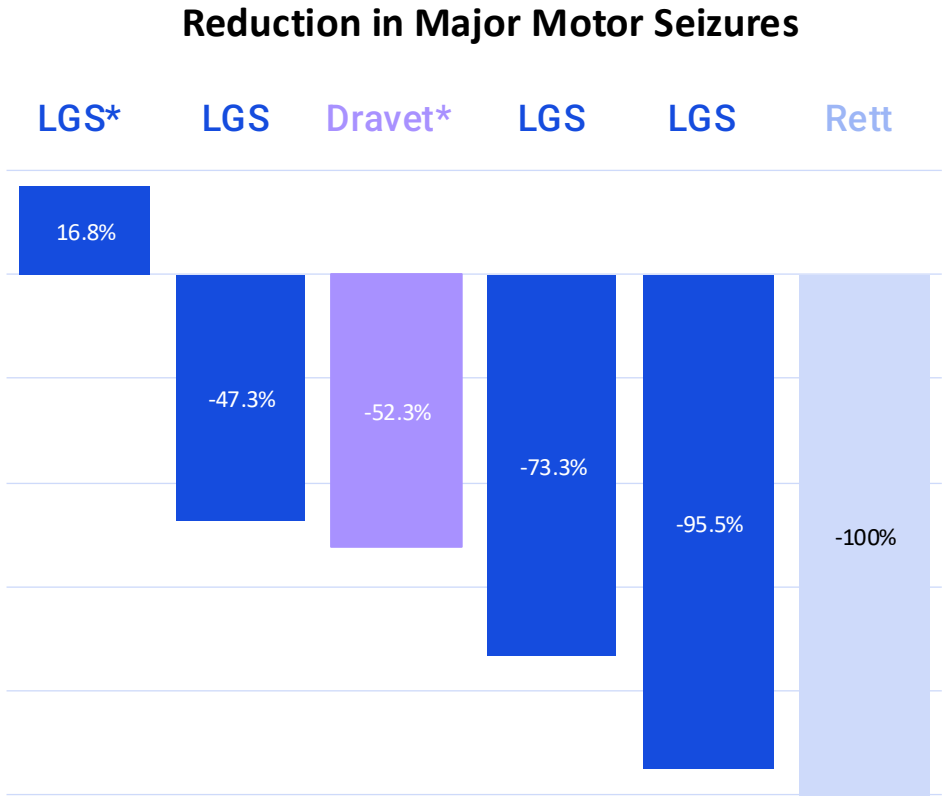


Topline data
Data available for N=11
REM – Rapid Eye Movement

Significant decrease in seizures in highly refractory DEE patients



Median reduction in Major Motor Seizures
As measured by Seizure Diary



Dravet patient previously failed fenfluramine due to efficacy

DEE – Developmental and epileptic encephalopathy, LGS -Lennox-Gastaut Syndrome, Dravet – Dravet Syndrome, Rett – Rett Syndrome
Topline data. Data through maintenance periods. *LOCF – Last observation carried forward

BMB-101 is well-tolerated and safe in patients

- Most of the TEAEs were mild (about 80%)
- No significant changes on vital signs, labs or ECG
- Three SAEs – all non drug-related: fractured shoulder with opiate-related drowsiness (2); and pneumonia (1)

Adverse Event >10%	Total (N=24)	Total %	Relation to drug PR / R, n	Relation to drug PR / R, %
Respiratory infections	5	20.8%	0 / 0	0% / 0%
Fatigue	4	16.7%	4 / 0	16.7% / 0%
Constipation	4	16.7%	3 / 0	12.5% / 0%
Headache	3	12.5%	1 / 0	4.2% / 0%
Drowsiness	3	12.5%	2 / 0	8.3% / 0%

PR = Possibly Related
R = Related

Total TEAE, n	93
Mild	74 (79.6%)
Moderate	16 (17.2%)
Severe	3 (3.2%)

Severe adverse events included:

- One patient - Dry mouth – Related (transient, no dose change, no discontinuation)
- One patient - Fractured shoulder, and drowsiness (opiates) – both not related (patient discontinued)

BRIGHT MINDS | TEAE: Treatment-Emergent Adverse Event, SAE: Serious Adverse Event
Topline data. Data through maintenance periods

Summary

Absence Seizures

DEE

- BMB-101 was safe and well tolerated in patients with DEE and Absence Seizures
- BMB-101 met both primary and secondary endpoints in DEE and Absence Seizures
- BMB-101 demonstrates a potentially best-in-class profile for patients with refractory DEE and Absence Seizures
- Favorable convenience of dosing with BID -> potentially QD administration

Prader-Willi Syndrome

5-HT_{2C} Selective Agonists

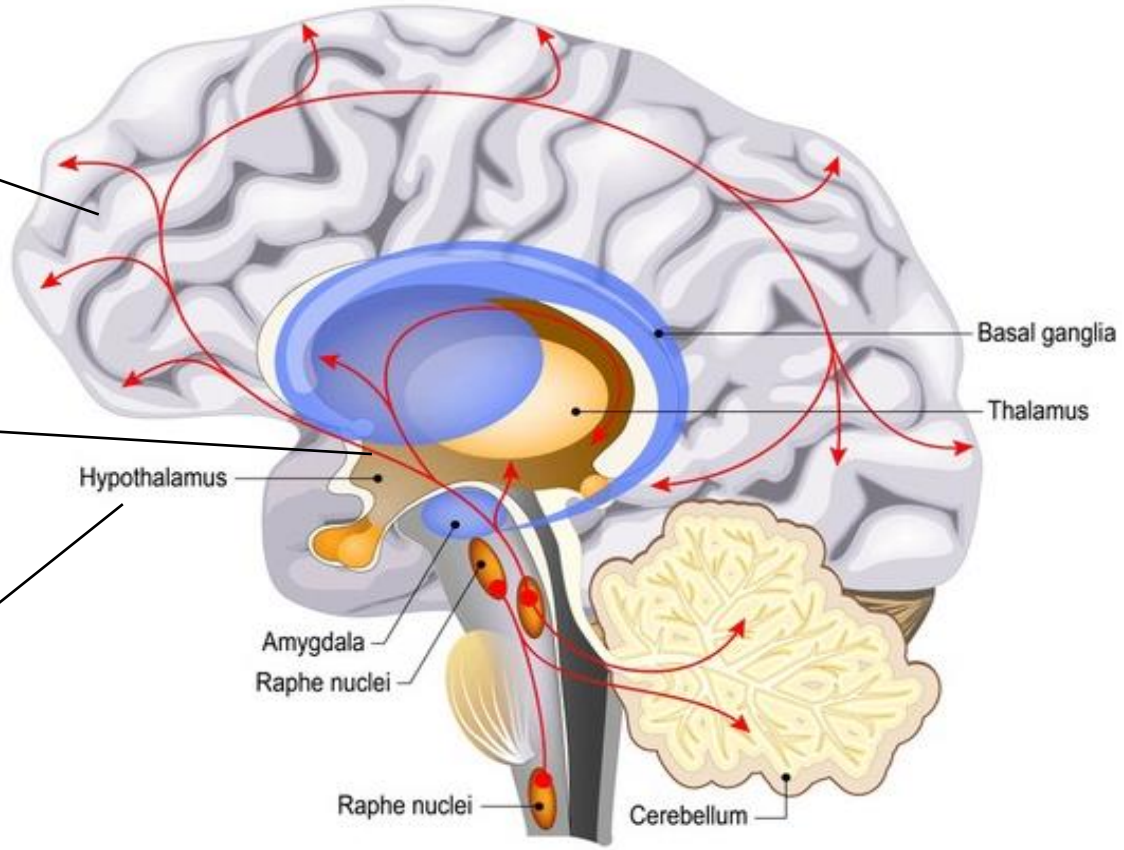
PWS is a complex neurodevelopmental and neurobehavioral disorder

Executive functions:
cognitive and behavioural issues

Reward system:
hyperphagia

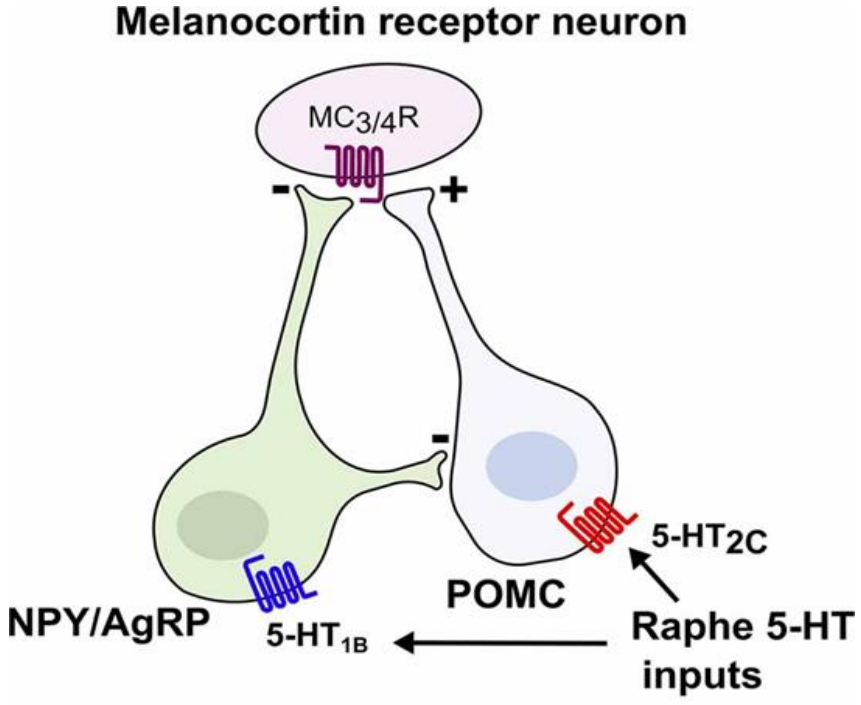
Satiety centre:
obesity

Serotonin pathway



- Hyperphagia
- Obesity
- Feeding problems
- Compulsion
- Sleep Disturbances
- Aggression
- Cognitive impairment
- Self-injury
- Anxiety
- Epilepsy
- Higher Risks of death
- Psychosis and other psychiatric disorders

5-HT_{2C} agonism directly targets underlying pathophysiology of PWS



Arcuate Nucleus

- PWS patients suffer from loss of part of the paternal chromosome 15 – leading to $5HT_{2CR}$ loss of function
- 5-HT_{2C} receptors are Apex regulators of the neuroendocrine axis.
- PWS patients express lower levels of mature, functional, 5-HT_{2C} receptors resulting in unabated appetite and compulsive behaviors.
- A 5-HT_{2C} agonist may compensate for loss of 5-HT tonus through the 5-HT_{2C} receptor thereby treating both hyperphagia and neurobehavioral symptoms associated with PWS

Initiation of Prader-Willi Clinical Program

Bright Minds has initiated clinical development of BMB-105, 5-HT_{2C} agonist specifically for Prader-Willi patients

BMB has initiated Phase 2a – Proof-of-Pharmacology program in Prader Willi Syndrome with BMB-101 to pave the way forward for a pivotal study with BMB-105

Potential for best-in-class 5-HT_{2C} agonist directly targeting pathophysiology in PWS

Clinical
5-HT_{2C} agonists

- Previously approved as weight loss drugs (lorcaserin and fenfluramine)
- Fenfluramine reduced hyperphagia and improved behaviors in a pilot clinical trial in PWS

Preclinical
BMB-101

Efficacy in multiple animal models of:

- Hyperphagia
- Weight loss
- Aggression control
- Impulsivity control
- Cognitive decline

Clinical
BMB-101

- Severely overweight (>100kg) patients in epilepsy trial lost >10% weight
- Reported improved relationship with food and overall quality of life
- 24h EEG demonstrate REM sleep improvements

NOVA: BMB-101 Phase 2a for Prader-Willi Syndrome

Proof-of-pharmacology study to demonstrate that 5-HT_{2C} agonism will address symptom complex in PWS patients

Eating behaviours
(Hyperphagia)

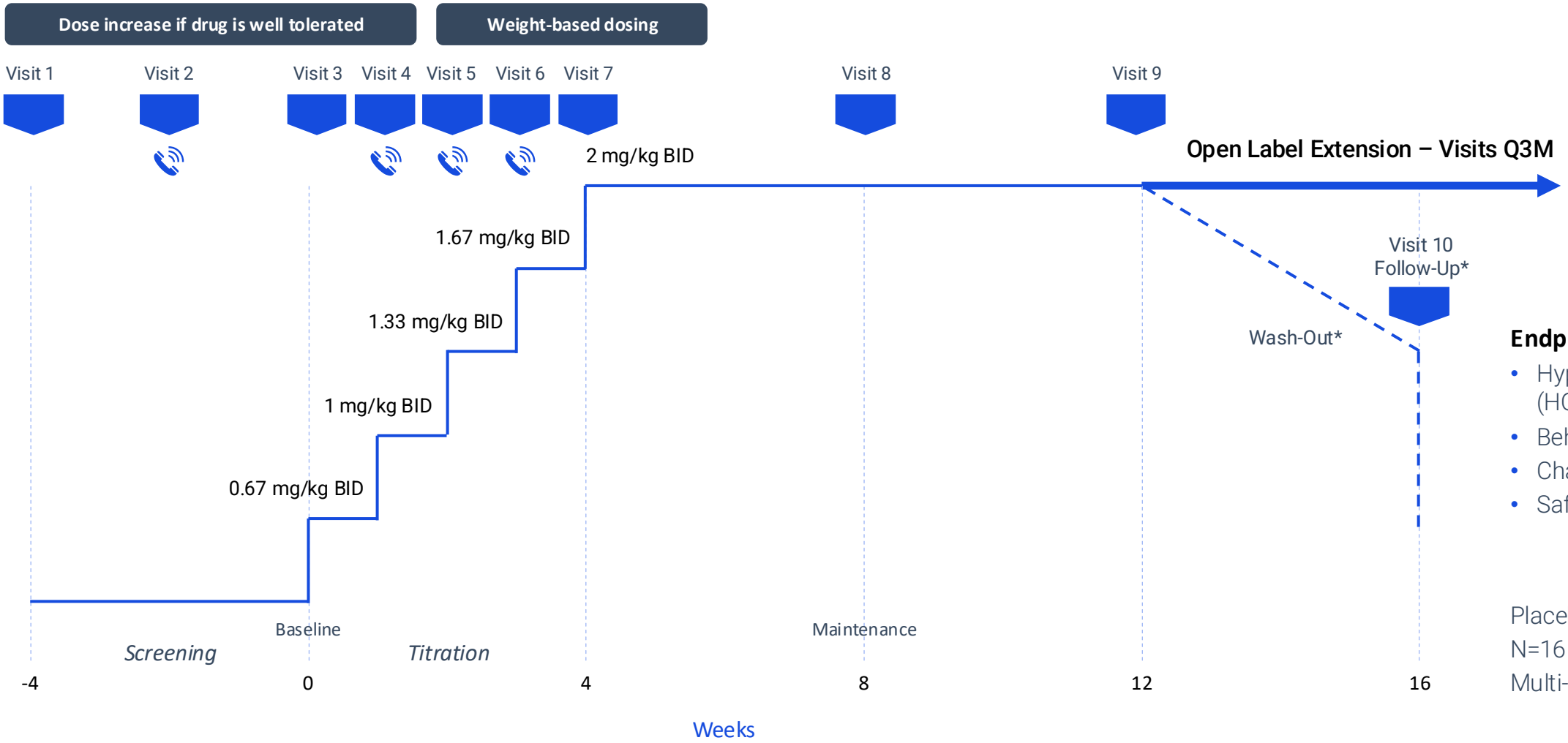
→ **PWS HQ-CT**

Set of behavioural symptoms
(aggression, anxiety, compulsiveness)

→ **PWS Profile**

As measured by instruments well accepted in the the PWS research community and regulatory bodies

NOVA (Neuro-Obesity Validated Advancement) PWS study design



Endpoints:

- Hyperphagia scores (HQ-CT, CGI-S, CaGI-S)
- Behaviors (PWS Profile)
- Change in body weight
- Safety endpoints

Placebo-controlled 1:1
N=16
Multi-center study

* Visit 10 Follow-Up visit for taper participants only who are not continuing to open label extension

Novel mechanism for Prader Willi Syndrome

	BMB-10x	Vykat (Soleno)	Carbetocin (Acadia)	ARD-101 (Aardvark)	Setmelanotide (Rhythm)	GLP-1 agonists
Targeting hyperphagia	✓	✓	✓	✓	✓	?
Targeting behavioural issues	✓	x	✓	x	?	x
Targeting cognitive issues	✓	x	x	x	?	x
Dosing	**Twice/once daily (projected)**	Once daily	Intranasal 3 times daily	Twice daily	s.q. Once Daily	Twice daily-once a week (oral or injected)
Development Stage	Clinical	Approved	Phase 3 *discontinued*	Phase 3 *paused*	Phase 2	Pilot studies in PWS
Components of PWS	**Hyperphagia Weight loss Impulsivity Agression Congition**	Hyperphagia	Hyperphagia	Hyperphagia	Hyperphagia	Obesity

Attractive biologic rationale

Directly targeting PWS pathophysiology

- Genetic link, preclinical and clinical evidence supports potential for targeting the PWS symptom complex with 5-HT_{2C} agonism

- Demonstrated tolerability and safety in Phase 1 clinical trial

- Hyperphagia and Behavioral endpoints supported by research community and regulators

With only two drugs approved there is significant market opportunity

1/15 000
births[1]

10,000 – 20,000
PWS patients in U.S.[2]

~400,000
cases worldwide

~$4.6bn

U.S. market size

Based on the annual price of approved therapy[3]

Additional research needed to assign a product-specific peak sales estimate

Reference
1. Foundation for Prader-Willi research: https://www.fpwr.org/what-is-prader-willi-syndrome#definition
2. Bohonowych 2020 PMC6770999
3. Managed Healthcare Executive, March 2025 (Vykat XR)

Opportunities for other BMB 5-HT_2 agonists

BMB-201 and 202 – potential for best-in-class 5-HT_{2A} agonists

BMB-201

Potent inducer of neuroplasticity

BMB-202

The most selective 5-HT_{2A} agonist in development*

DESCRIPTION

- ✓ Designed to have minimal or absent psychoactive effects
- ✓ Efficacy in rodent models of depression, anxiety, pain, substance use disorder
- ✓ Proprietary NCE
- ✓ ADMEPK profiling completed

- ✓ Designed to have short psychoactive effects time
- ✓ Durable effects in rodent models of depression and anxiety
- ✓ Proprietary NCE
- ✓ ADMEPK profiling completed

TREATMENT PARADIGM

Designed for chronic use and use at home

Designed for fast relief of depression and Infrequent use