BRIGHT MINDS

Announcement of Prader-Willi Syndrome Program

November 6, 2025

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Event speakers

Bright Minds Bio Participants

Ian McDonald

Chief Executive Officer, Director

Jan T. Pedersen, PhD, MSc

Chief Scientific Officer, Director

Stephen Collins, MD, PhD

Chief Medical Officer

Key Opinion Leaders

Jennifer Miller, MD

Dr. Miller is a Professor in the division of pediatric endocrinology at the University of Florida . She graduated with her M.D. from the University of Florida in 1998, and her M.S. in Clinical Investigation from the University of Florida in 2005. She has done all of her training in pediatrics and pediatric endocrinology at the University of Florida. She specializes in the care and treatment of individuals with Prader-Willi syndrome and other genetic causes of early-onset excessive weight gain. She has been working towards achieving an appropriate treatment for hyperphagia in patients living with Prader-Willi syndrome for the past 12 years. She currently follows over 500 patients with Prader-Willi syndrome from around the world.

Theresa V. Strong, BS, PhD

Theresa Strong, PhD, is a co-founder and Director of Research Programs at the Foundation for Prader-Willi Research (FPWR, www.fpwr.org), a nonprofit organization that supports research to advance the understanding and treatment of the rare neurodevelopmental disorder, Prader-Willi syndrome (PWS). Prior to joining FPWR full time, she had an academic career in genetics/cancer gene therapy and remains a volunteer Adjunct Professor at the University of Alabama at Birmingham. She has authored more than 75 peer-reviewed scientific publications. At FPWR, she directs the grant program, co-leads the PWS Clinical Trials Consortium and is principal investigator for the Global PWS Registry. She is also active in the broader rare disease patient advocacy community. She and her husband have four young adult children, including a son living with PWS.

Elizabeth Roof, HSP, MA

Elizabeth Roof has worked with children and teens with PWS for almost 30 years at Vanderbilt University. Elizabeth has been licensed since 1994 as a Health Service Provider in Psychology in the state of Tennessee and has been doing research with Elisabeth Dykens since 2003, focused on the psychiatric, behavioral and adaptive strengths in PWS and WS. Elizabeth has personally evaluated over 450 individuals with PWS and WS. Elizabeth has trained 27 research staff over the years, who take that expertise with them to graduate, medical, and nursing school, and out into the real world. Elizabeth also works with residential and clinical professionals and schools to provide best practices for those living with PWS and WS. Elizabeth has managed 5 NIH trials and 11 clinical trials in PWS since 2014 and works with sponsors to identify best outcome measures, study designs, and logistics that best suit PWS families. Elizabeth has spoken at PWS and WS conferences across the US, Canada, Europe, and Australia. She loves meeting new families and continuing to have lasting relationships with many families over the years.

Topic	Themes	Speaker	Timing
Introduction	• Corporate update • Announcement of new BMB-105 program	**Ian McDonald**	5 min
Overview of Prader-Willi Syndrome	• Clinical presentation (overview) • Unmet need in PWS: not only hyperphagia, but also behavioral issues. • Major neurobehavioral symptoms, e.g. aggression, impulsivity, agitation, cognitive issues	**Jennifer Miller**	10 min
Patient and family perspective	• Impact and the unmet need • Why we need better drugs targeting multiple symptoms • Severity of the disease	**Theresa Strong**	10 min
Aspects of drug development for Prader-Willi syndrome	• The need for novel drugs targeting several aspects of the disease • Assessment of neurobehavioral symptoms in clinical trials	**Elizabeth Roof**	10 min
BMB Approach to Prader-Willi syndrome	• Overview of BMB-101: 5-HT2c mechanism in Prader-Willi • Clinical trial design • Market overview	**Jan Torleif Pedersen** **Stephen Collins** **Ian McDonald**	20 min
Q&A session			15 min

Prader-Willi Syndrome

Targeting Serotonin

Ian McDonald

Chief Executive Officer
Bright Minds Biosciences

Creating New Chemical Entities That Target Serotonin Signaling

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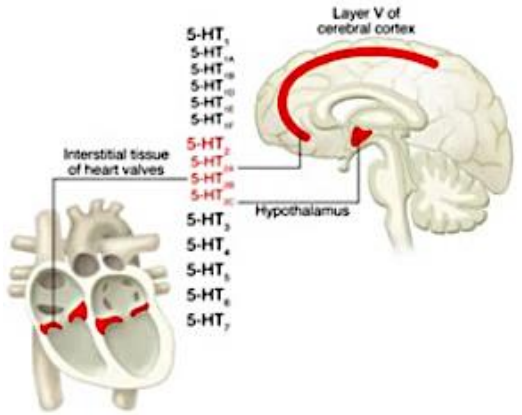
Key 5-HT$_2$ Receptors Targets

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

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

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



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

- 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

Update on BREAKTHROUGH Ph.2a study

Absence Seizures and Developmental and Epileptic Encephalopathies

BREAKTHROUGH study is ongoing in Absence Seizures and Developmental and Epileptic Encephalopathies.

- **To date, BMB-101 has been well tolerated, with no drug-related Serious Adverse Events nor any safety signals requiring any protocol adjustments.**
- **Exposure levels and tolerability achieved are consistent with expectations from Phase 1 study, supporting dose selection for future studies.**
- **Participation in the open-label extension is proceeding well, with nearly all eligible patients electing to remain on therapy under investigator supervision.**

Topline data – early January 2026

BREAKTHROUGH
Phase 2a study of BMB-101 in
- Developmental and Epileptic Encephalopathies (DEE)
- Absence Seizures

PLANNING
- Phase 2/3 Clinical study in DEE
- Phase 2/3 Clinical study in Absence seizures

These operational milestones reflect strong engagement from our investigators, families, and clinical partners, and we're grateful for their ongoing commitment to the program.

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 will initiate 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**

BMB-105 development plan in PWS

BMB-101

Clinical Studies – Phase 2a – PoPh in PWS patients

NOVA (Neuro-Obesity Validated Advancement)
A Randomized, Double-Blind, Placebo-Controlled Phase 2 Study to Assess Efficacy, Safety and Tolerability of BMB-101 Oral Solution for the Treatment of Patients with Prader-Willi Syndrome

BMB-105

Phase 1 → **Pivotal Phase 2/3**

A 3-part randomized placebo-controlled study to evaluate the safety, tolerability, pharmacokinetics and food effect of BMB-105 in Healthy Volunteers

- Singe Ascending dose (4 cohorts: 6 drug and 2 placebo)
- Food Effects (12 subjects, 6 drug and 6 placebo)
- Multiple Ascending Dose (4 cohorts: 6 drug and 2 placebo)

PWS is a complex neurodevelopmental and neurobehavioral disorder with limited and insufficient treatment options

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Energy balance

- Obesity — Glutides
- Hyperphagia — Vykat
- Feeding problems
- Growth hormone deficiency — hGrh

Neuropsychiatric symptoms

- Compulsion
- Cognitive impairment
- Sleep Disturbances
- Self-injury
- Aggression
- Epilepsy — AEDs
- Anxiety — SSRIs
- Higher Risks of death
- Psychosis and other psychiatric disorders — AAs

Dai YL, Qin YF, Chao YQ, Hu CX, Xia FL, Zou CC. The genotype and phenotype correlation of Prader-Willi syndrome. *Rare Dis Orphan Drugs J*. 2024;3:33.

Current treatments do not address the majority of PWS complex symptoms

Need for a therapy which can address the entire complex

Energy balance

- Obesity — Glutides
- [illegible] — [illegible]
- Feeding problems
- [illegible] — [illegible]

Neuropsychiatric symptoms

- Compulsion
- Cognitive impairment
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Prader-Willi Syndrome

5-HT_{2C} mechanism of action

Jan Torleif Pedersen, MSc PhD

Chief Scientific Officer
Bright Minds Biosciences

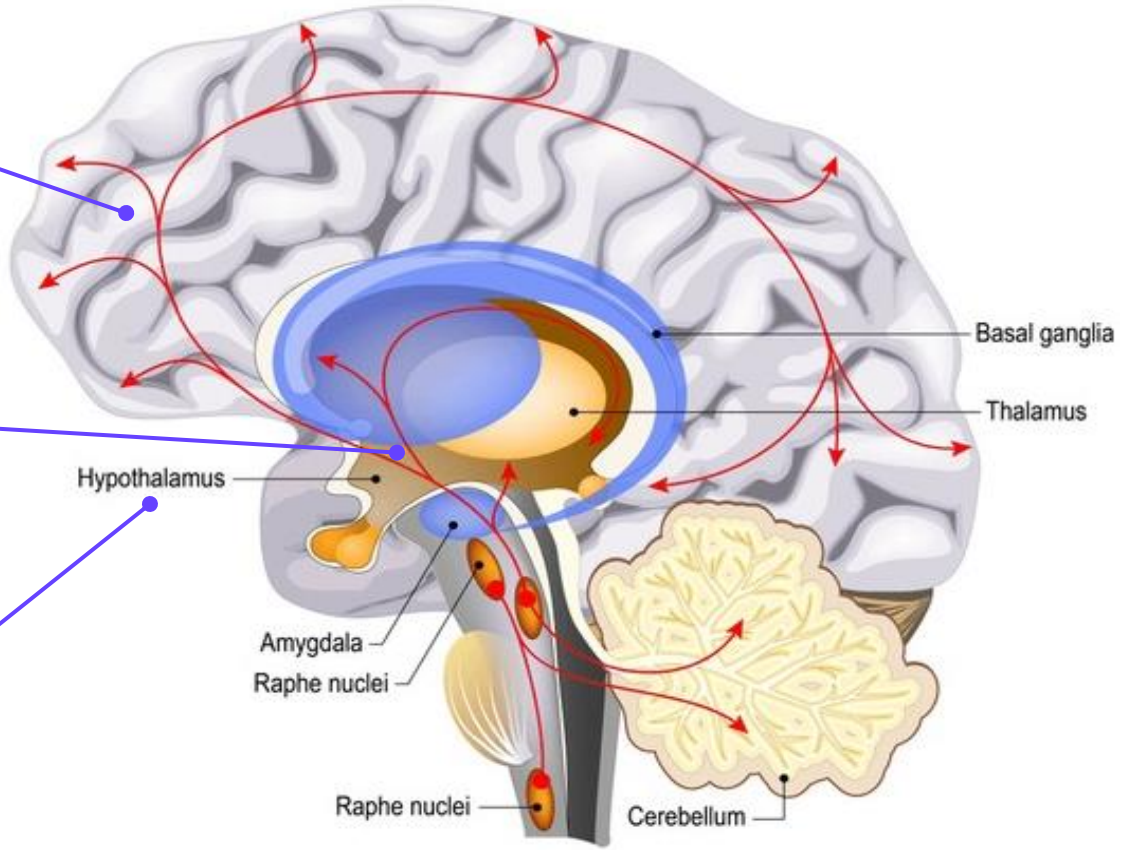
PWS is a complex neurodevelopmental and neurobehavioral disorder

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

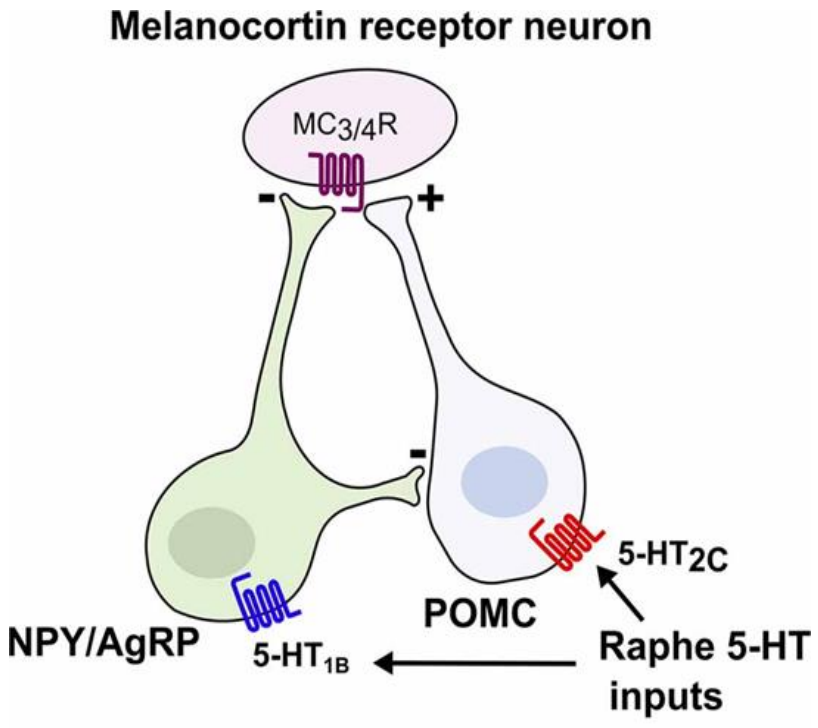
Genetics of PWS is linked to 5-HT_{2C} receptor

Genetic variants lead to heterozygous 5-HT_{2C}R loss of function

SNORD116 (RNA)

- About 50-74% of cases occur when part of the father's chromosome 15 is deleted. In 25-36% of cases, the affected person has two copies of the maternal chromosome 15 from the mother and lacks the paternal copy. 3 % of cases are caused by genetic imprinting (non-functional paternal chromosome)
- All genetic variants lead to deficient SNORD116 expression
- SNORD116 regulates the alternative splicing of 29 gene targets, among these predominantly HT_{2C}R (5-HT_{2C} receptor) exon 5b alternative splicing.
- Genetic variants lead to heterozygous 5HT_{2C}R loss of function

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

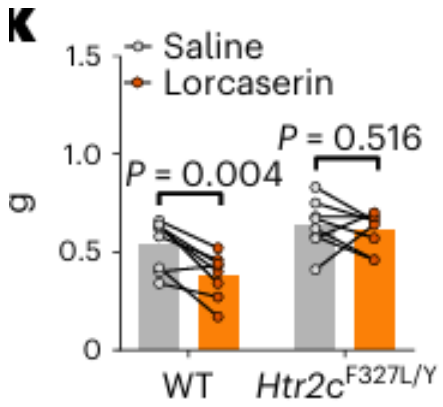


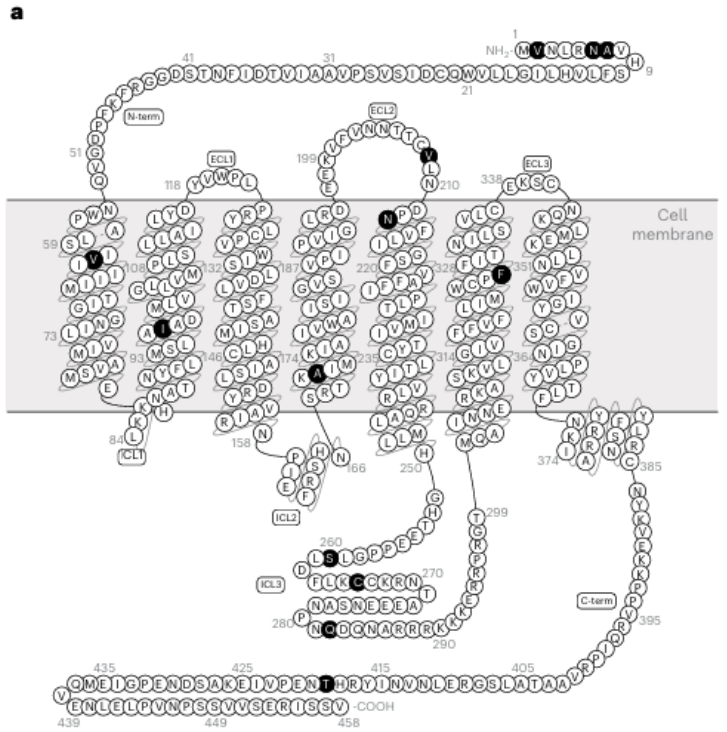
Arcuate Nucleus

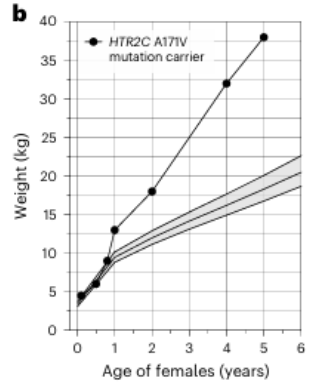
- 5-HT_{2C} receptors are Apex regulators of the neuroendocrine axis.
- 5-HT_{2C}+ neurons project to hypothalamic melanocortin neurons to suppress food intake and thereby directly regulating satiety and appetite.
- 5-HT_{2C} receptors are also involved in regulation of compulsive behavior.
- PWS patients express lower levels of mature, functional, 5-HT_{2C} receptors resulting in unabated appetite and compulsive behaviour.
- A 5-HT_{2C} agonist may compensate for loss of 5-HT tonus through the 5-HT_{2C} receptor thereby treating both hyperphagia and neurobehavioural symptoms associated with PWS

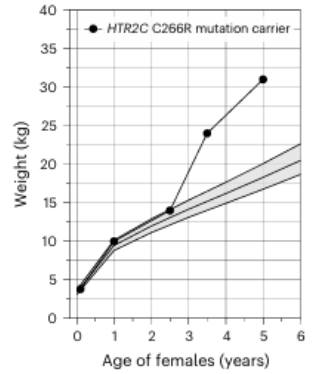
Loss of 5-HT$_{2C}$ receptor function in humans leads to obesity and maladaptive behavior

- Sporadic loss-of-function variants leads to obesity and maladaptive behaviour in humans
- Sporadic loss-of-function variants lead to partial loss of 5-HT$_{2C}$ receptor tonus.
- Phenotype is similar to that observed in PWS.





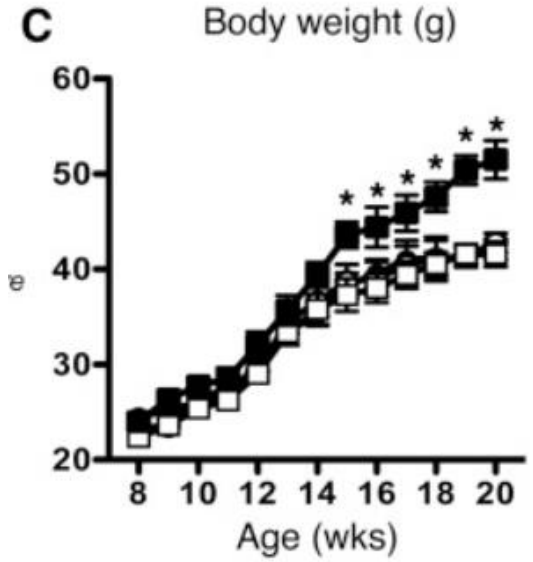




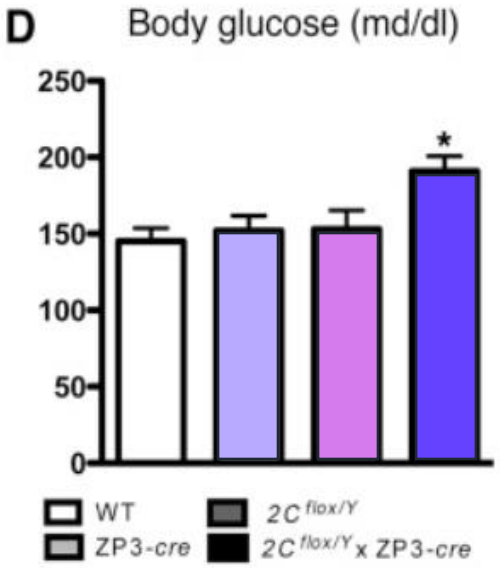
He Y, et al. Human loss-of-function variants in the serotonin 2C receptor associated with obesity and maladaptive behavior. Nat Med. 2022 Dec;28(12):2537-2546.

5-HT_{2C} receptor is a key regulator of compulsive behavior and food intake

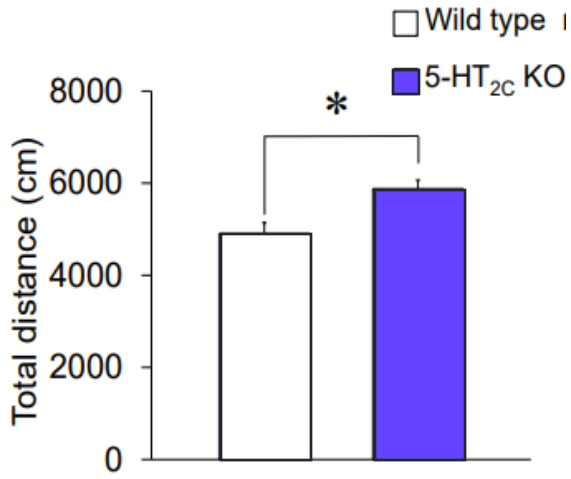
Learnings from 5-HT_{2C} KO mice



- Over-eating



- Pre-diabetic phenotype

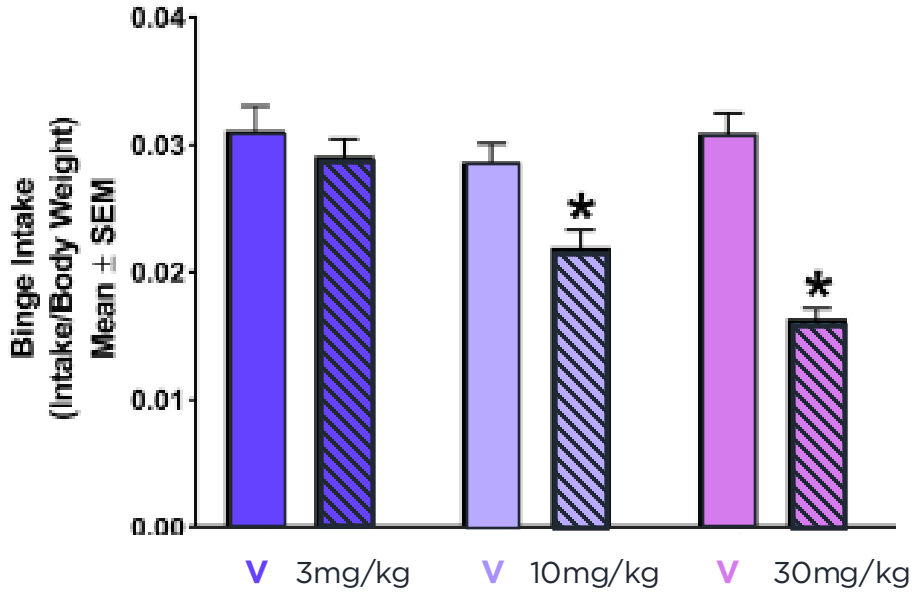


- Increased locomotor behaviour
- Attenuated contextual fear response
- Compulsive (OCD-like) behaviour

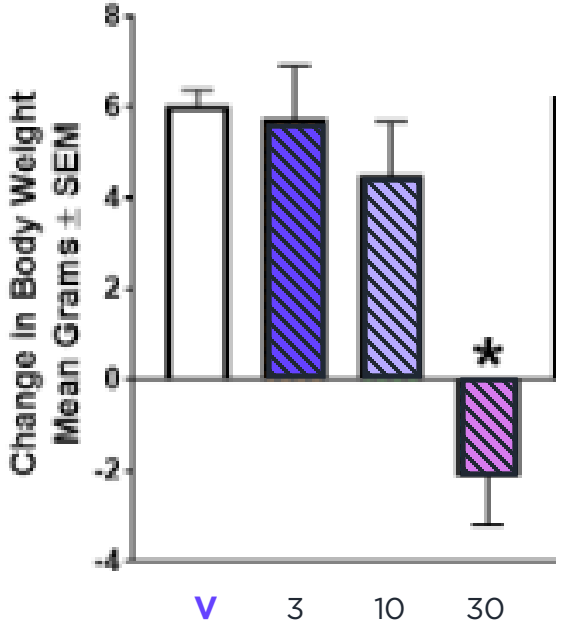
Nebuka et al, Behav Brain Res. 2020 Feb 3:379:112394.
Bouchekioua el al, Transl Psychiatry. 2022 Feb 11;12(1):58.
Chou-Green et al, Physiol Behav, 2003 Apr;78(4-5):641-9.
Berglund et al., J Clin Invest. 2013 Dec;123(12):5061-70.

BMB 5-HT$_{2C}$ agonists dose-dependently reduce binge eating episodes in rats

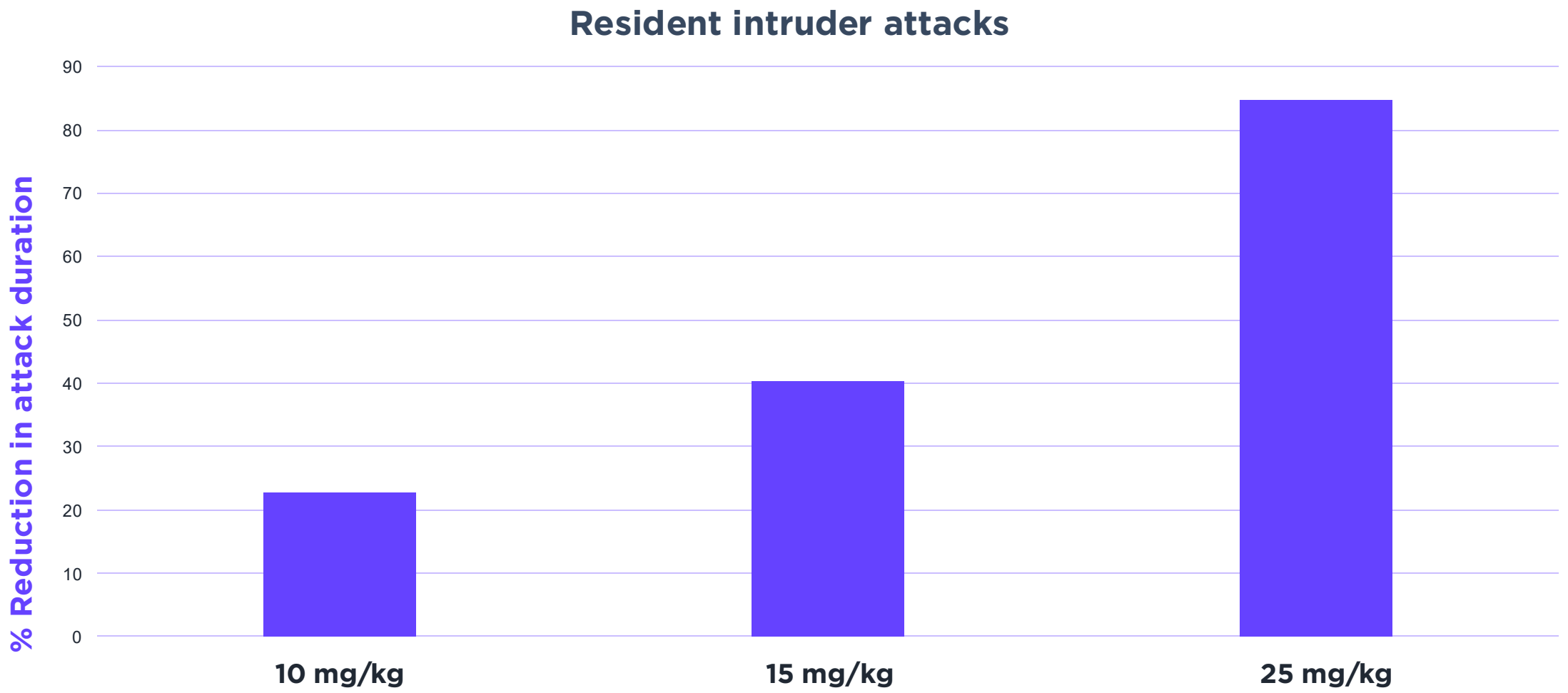
Reduced binge eating episodes in rat models



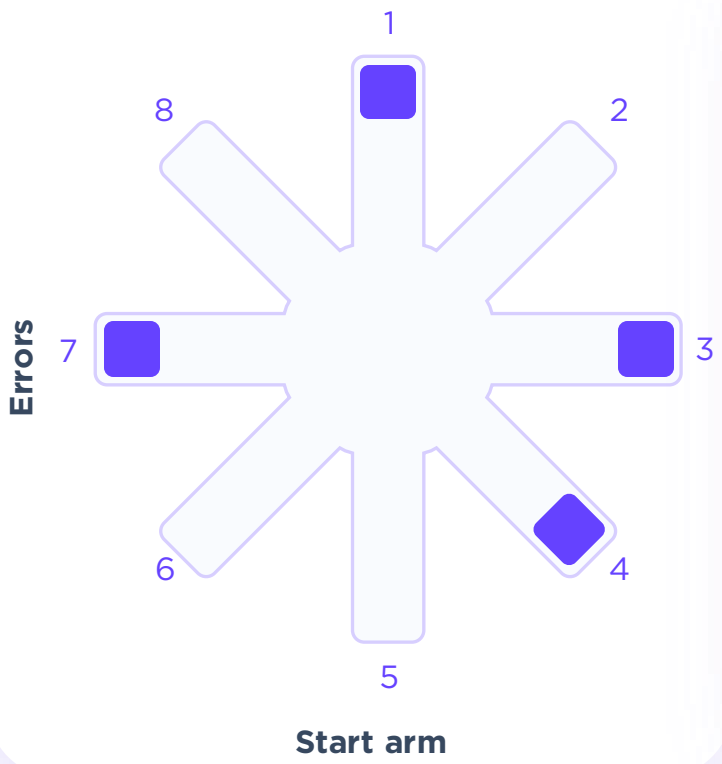
Reduced body weight in binge eating rat models



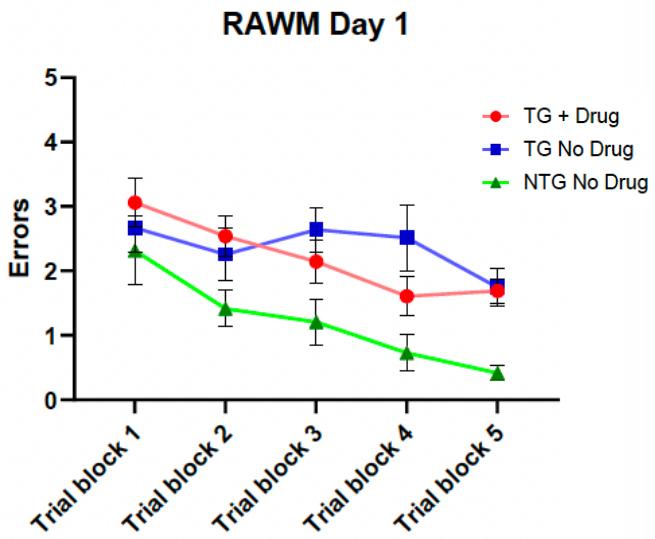
BMB 5-HT$_{2C}$ agonist reduces aggression in rat resident intruder model



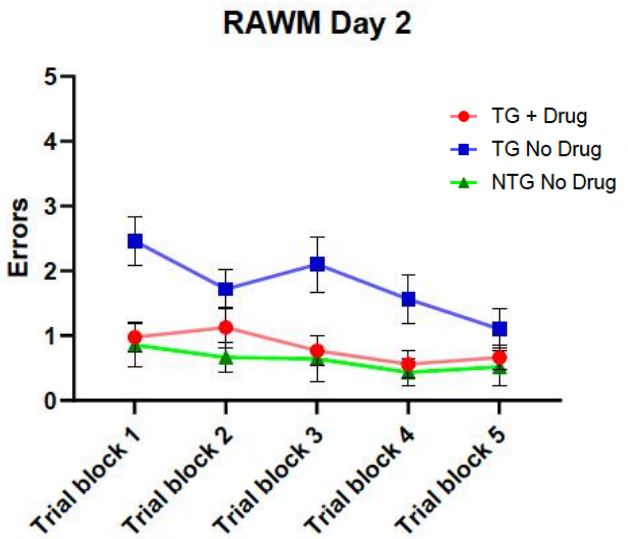
BMB 5-HT$_{2C}$ agonist improves spatial learning and memory in tau transgenic mice (Radial arm water maze)



Training day



Testing day



BMB-101 protects **PS19 P301L tau mice** mice from cognitive impairment in the radial arm water maze. Mice were tested in 5 blocks of three trials to find an escape platform in the radial arm water maze on day 1 (alternating between visible and hidden trials). Mice were then tested on day 2 for retention using 5 blocks of three trials with the platform hidden on all trials.

BMB 5-HT_{2C} agonist reverses compulsive behavior

- BMB-101 dose-dependently suppressed fentanyl self infusions.
- BMB-101 dose-dependently reduces the number of active lever presses without alterations in inactive lever presses (data not shown).
- SB242084 (5-HT_{2C} antagonist) reversed the suppression of intake induced by 20 mg/kg,
- No full reversal when BMB-101 was administered at 60 mg/kg

Fig. 1

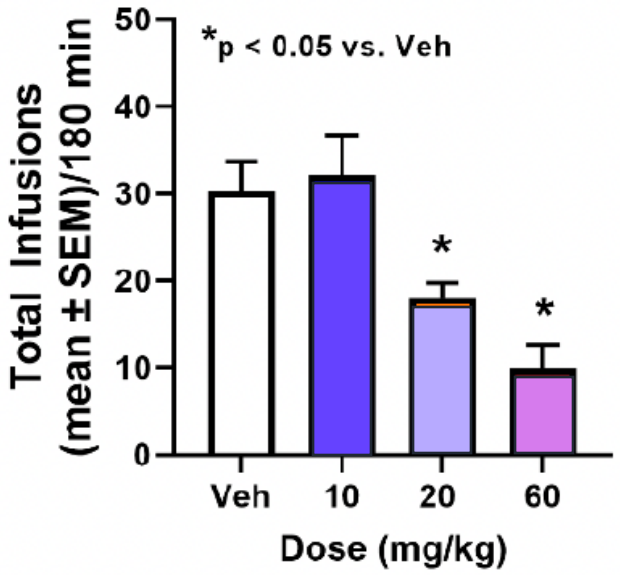
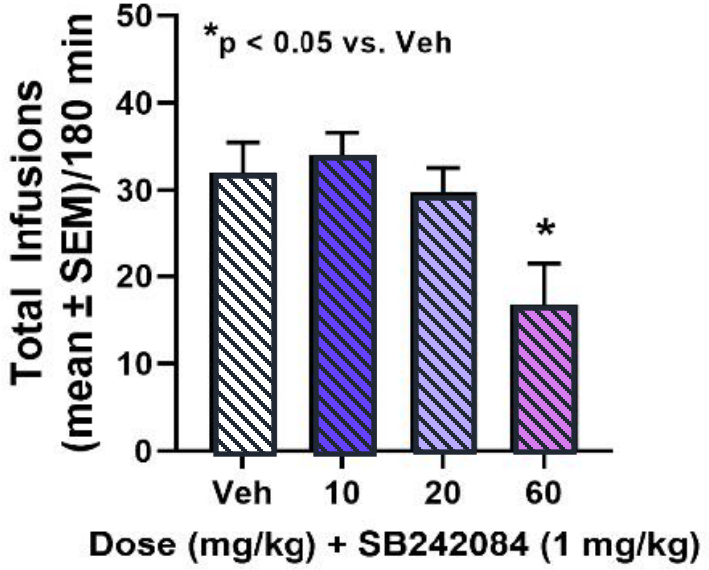


Fig. 2



BMB 5-HT$_{2C}$ agonists demonstrate preclinical efficacy across all key PWS symptom domains

Hyperphagia	Reduces binge eating and weight loss in rat model
Aggression	Reduces aggressive behaviors in resident intruder mice model
Cognitive improvements	Protects mice from cognitive impairment in the radial arm water maze
Agitation and hyperactivity	Reduces hyperactivity in mice in open field
Uncontrolled cravings Compulsive behaviors	Reduces fentanyl seeking behavior and reduces total doses of fentanyl in an opioid use rat model

Clinical POC study in PWS: Efficacy with non-selective 5-HT_{2C} agonist

Fenfluramine in Prader-Willi syndrome: a double blind, placebo controlled trial

M Selikowitz, J Sunman, A Pendergast, S Wright

Study design

- 15 patients enrolled, 8 F and 7 M
- 5 to 27 years old
- 2-week baseline period of observation
- 2 groups: 6 weeks of placebo followed by 6 weeks of fenfluramine or vice versa
- Weight, food related behaviours, aggressive behaviours and self direct behaviours were assessed

Results with fenfluramine

- **Decreased weight**
- **Decreased food related behaviour**
- **Decreased aggressive behaviours**
- Parents and **patients** frequently reported **pronounced qualitative changes**. Some parents reported that this was the first time that they had seen their children refuse food, with statements such as 'he is a different child' and **'the best time ever'**.

	Fenfluramine /Placebo (% reduction)	P value
Weight	**-2.6**	**0.02**
Food behaviour	**-5.3**	**< 0.05**
Aggressive behaviour	**-18.4**	**< 0.025**

Selikowitz M, Sunman J, Pendergast A, Wright S. Fenfluramine in Prader-Willi syndrome: a double blind, placebo controlled trial. Arch Dis Child. 1990 Jan;65(1):112-4.

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

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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

BMB-101

Novel 5-HT_{2C}
Selective Agonist

NOVA

(Neuro-Obesity Validated Advancement)

Prader-Willi Syndrome

Ph.2a clinical trial

Stephen Collins, MD, PhD

Chief Medical Officer
Bright Minds Biosciences

BMB-101 – highly selective 5-HT_{2C} agonist with excellent druggable qualities

BMB-101 – selective 5-HT_{2C} agonist, and Gq-protein biased to avoid desensitization and tolerance

Well-tolerated in Phase 1 clinical trials, no SAEs, AEs were experienced at 2.5 or above predicted therapeutic dose, so enjoys a wide therapeutic Index.

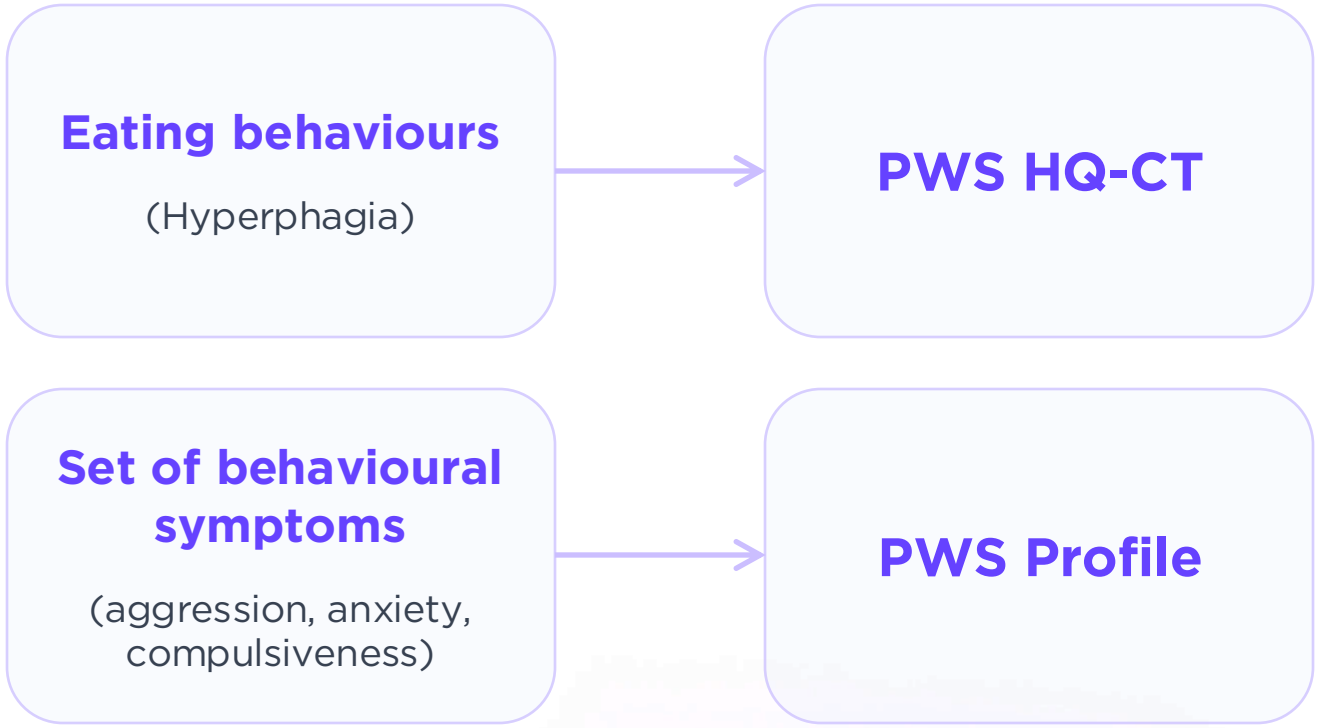
Linear PK over entire range of therapeutic dosing

Demonstrated central target engagement as measured by biomarkers and qEEG in Phase 1 study

Ph.2a clinical trial in Developmental Epilepsies and Absence Seizures ongoing

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



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

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

A Randomized, Double-Blind, Placebo-Controlled Phase 2 Study to Assess Efficacy, Safety and Tolerability of BMB-101 Oral Solution for the Treatment of Patients with Prader-Willi Syndrome

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

Lead Investigator:

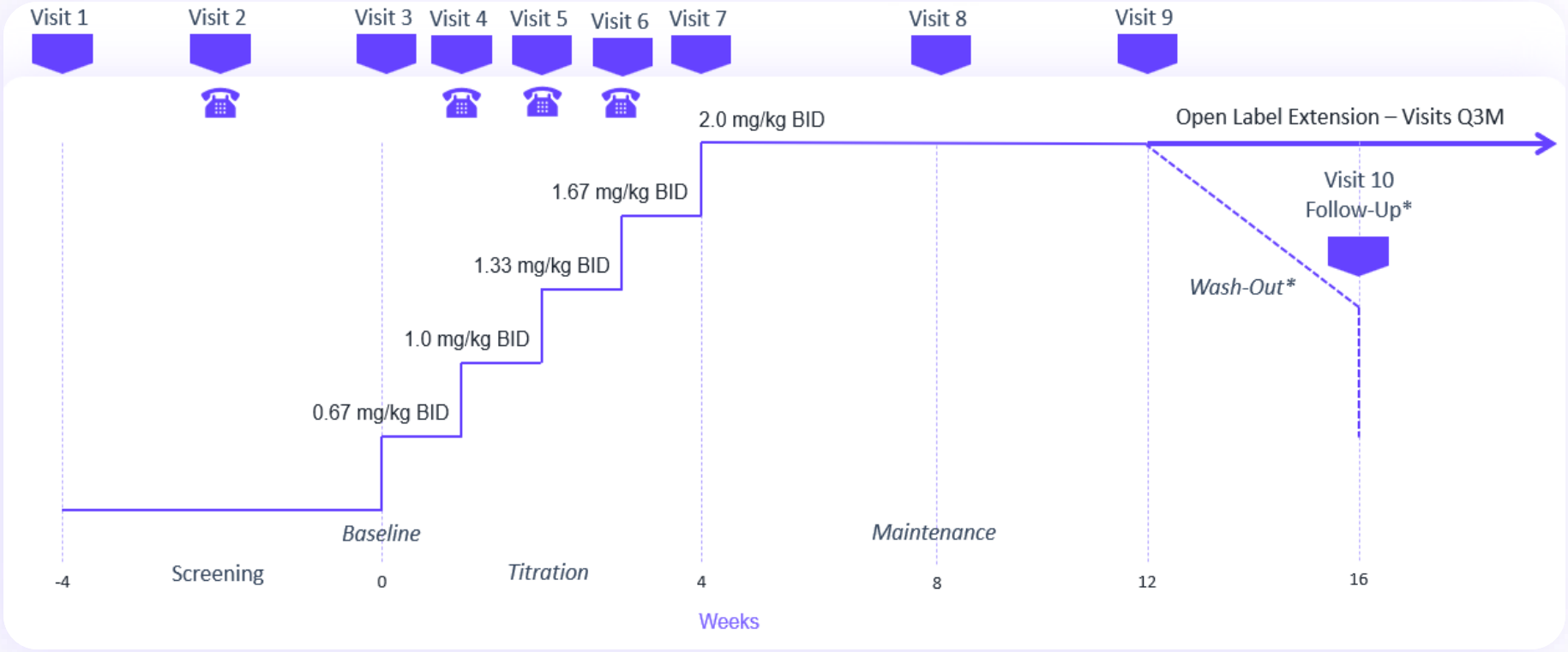
Professor Tania Markovic

An internationally known researcher and care-giver for PWS patients and families

NOVA (Neuro-Obesity Validated Advancement) PWS study design

Dose increase if drug is well tolerated

Weight-based dosing



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

BMB-105

Novel 5-HT_{2C}
Selective Agonist

Prader-Willi Syndrome

Market Potential

Ian McDonald

Chief Executive Officer, Director
Bright Minds Biosciences

Novel mechanism for Prader Willi Syndrome

	BMB-10x	Vykat (Soleno)	Carbetocin (Acadia)	ARD-101 (Aardvark)	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	Twice daily-once a week (oral or injected)
Development Stage	Clinical	Approved	Phase 3 *discontinued*	Phase 3	Pilot studies in PWS
Components of PWS	**Hyperphagia Weight loss Impulsivity Agression Congition**	Hyperphagia	Hyperphagia	Hyperphagia	Obesity

Attractive biologic rationale
Directly targeting PWS pathophysiology

- Genetic link, preclinical and clinical evidence supports potential for tageting the PWS symptom complex with 5-HT_{2C} agonism
- Demonstrated tolerability and safety in Phase 1 clinical trial
- Hyperpahgia 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)

Pipeline

Rich and diverse portfolio in neurology and psychiatry with multiple programs

Lead	Indications	Research	Phase 1	Phase 2
5-HT$_{2C}$ agonists				
BMB-101	DEE Absence seizures	Clinical Studies – Phase 2		
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		

BRIGHT MINDS

Announcement of Prader-Willi Syndrome Program

Q&A session