CAMP4®

Corporate Overview

Pioneering a new class of RNA medicines to increase targeted gene expression.

MARCH 2026

Forward-Looking Statements

This presentation contains forward-looking statements that are based on the beliefs and assumptions and on information currently available to CAMP4's management. All statements other than statements of historical fact contained in this presentation are forward-looking statements. Forward-looking statements include information concerning the initiation, timing, progress and results of preclinical and clinical trials of CAMP4's product candidates, the timing or likelihood of regulatory filings and approvals for any of its product candidates, and estimates regarding CAMP4's expenses, future revenues, and future capital requirements. In some cases, you can identify forward-looking statements because they contain words such as "may," "might," "will," "would," "shall," "should," "expects," "plans," "anticipates," "could," "intends," "target," "projects," "contemplates," "believes," "estimates," "looks," "seeks," "predicts," "potential," "ongoing," or "continue" or the negative of these words or other similar terms or expressions that concern our expectations, strategy, plans or intentions, although not all forward-looking statements are accompanied by such words. Forward-looking statements involve known and unknown risks, uncertainties and other factors that may cause CAMP4's actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. This information was factually accurate on the date it was published. CAMP4 assumes no duty to update the information to reflect subsequent developments, except as required by law.

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CAMP4: Targeted ASO therapeutics that selectively upregulate gene expression by modulating regulatory RNA

- Developing a targeted disease modifying therapy to address dire unmet need in SYNGAP1-related disorder
  - SYNGAP1 is a haploinsufficient CNS disorder, and an optimal target for CAMP4
  - CMP-002 is designed to increase SYNGAP protein levels, restore *SYNGAP1* function and improve disease symptoms
  - >10,000 SYNGAP1 patients in the US; epi in line with rare diseases with similar unmet needs and large commercial markets

- Positioned to be first in the clinic for SYNGAP1
  - No disease modifying therapies are approved or in clinical development
  - Highly translatable preclinical models: Proof of concept data in humanized mice showed reversal of disease phenotype, primate data showed significant protein upregulation and broad ASO distribution across key brain regions believed to be critical to the disease
  - Expect to advance CMP-002 to a global Ph 1/2 study in patients as early as second half of 2026

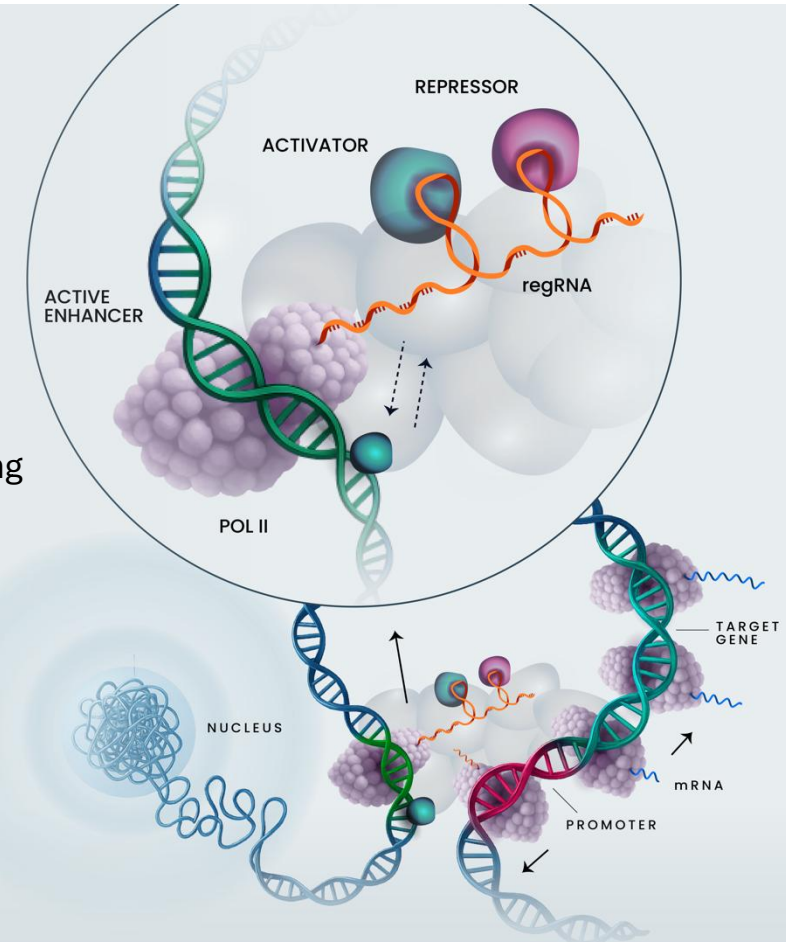
- CNS-focused pipeline, leveraging BD to derive additional value from the platform
  - Proprietary RAP Platform® was built for the discovery of novel regRNAs that regulate the expression of every protein-coding gene that can be selectively drugged using state of the art ASO chemistry
  - Additional undisclosed development epileptic encephalopathy (DEE) programs in development, similar in phenotype to SYNGAP1
  - Strategic discovery partnership with GSK unlocks additional platform value beyond CNS and validate CAMP4's novel approach to gene upregulation

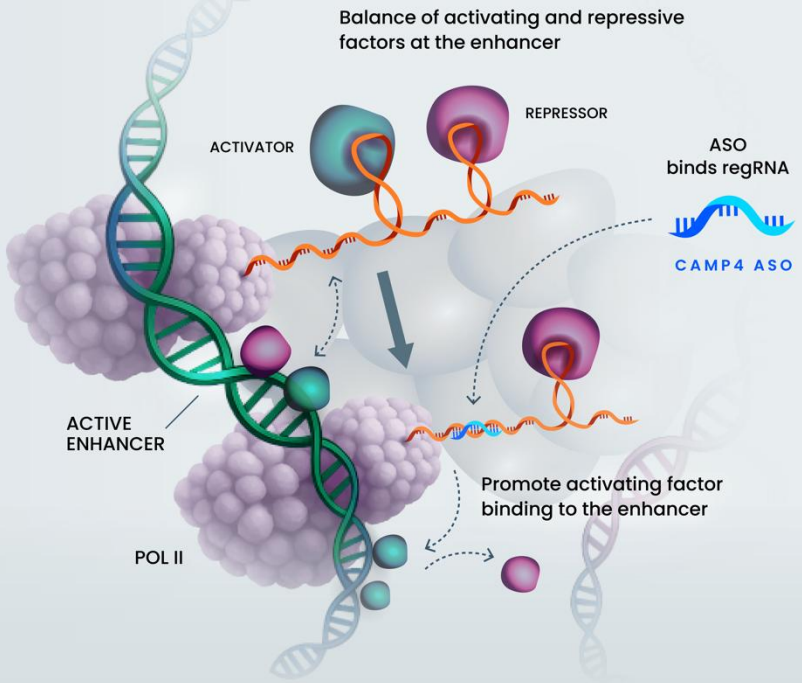
regRNAs play a central role in the regulation of every gene's expression

Increased mRNA addresses root cause of disease by returning targeted protein levels toward a healthy range

1 regRNAs originate from enhancers and promoters

2 Activators and repressors bind to regRNAs to control the expression of protein-coding genes





3 ASOs disrupt the interactions between transcription factors and regRNAs enabling increases in gene expression

Source: Sharp et al. (2022) RNA 28: 52-57; Henninger et al. (2021) Cell 184:207-225; Sartorelli & Lauberth (2020) Nat. Struct. Mol. Biol. 27: 521-8; Oksuz et al. (2023) Molecular Cell 83: 2449-63.

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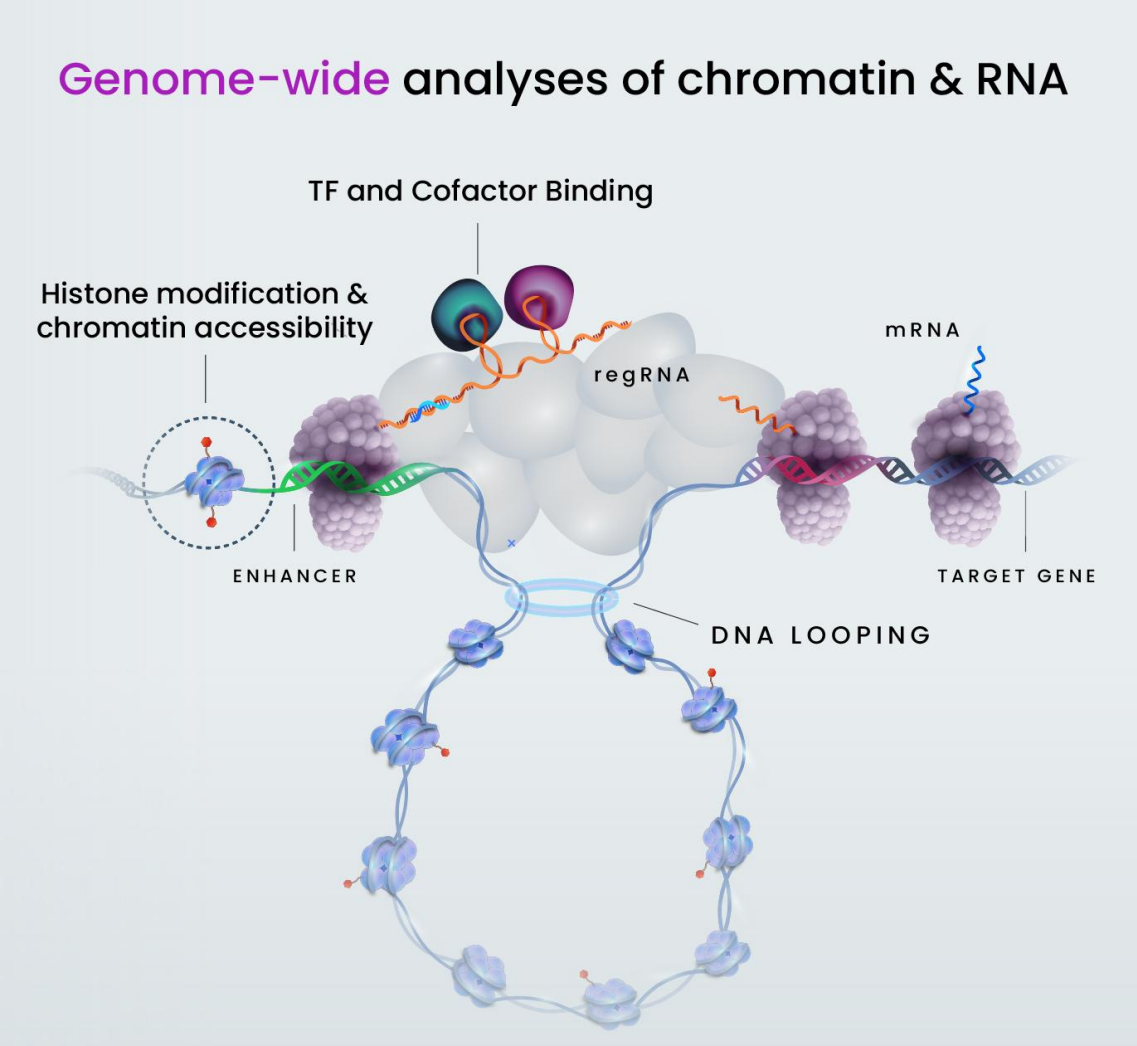
CAMP4's proprietary RAP Platform® catalogs thousands of regRNA targets and generates ASO candidates to increase gene expression

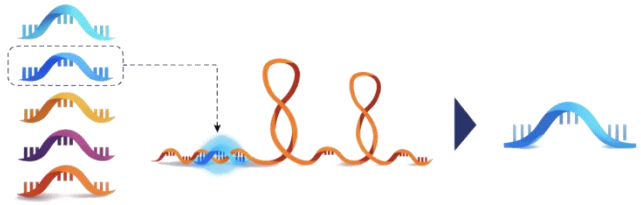


1 Map candidate regRNAs

- Generate large scale genomic datasets for cells & tissues
- Deploy proprietary ML/AI platform to identify regulatory regions
- Capture and sequence predicted regRNAs
- Create proprietary catalogs containing tens of thousands of regRNAs across diverse cell and tissue types

2 Generate ASO leads

Screen regRNAs to rapidly identify leads that upregulate target genes



3 Optimize lead candidates

Optimize chemistry and sequences for activity, pharmacology, & safety

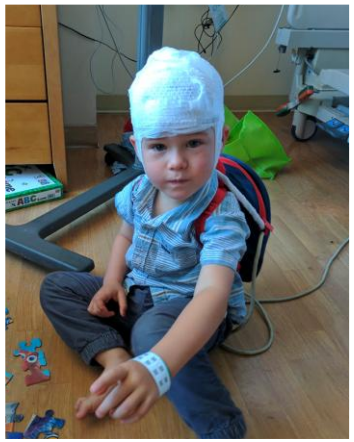
Our pipeline of CNS-focused upregulation programs

Program	Indication	Target	Discovery & Preclinical Development	Phase 1/2	Phase 3	Anticipated Milestones	Commercial Rights
CNS DISEASES							
CMP-002	SYNGAP1-related disorder	*SYNGAP1*				GLP tox studies ongoing Clinical initiation as early as H2 2026	CAMP4
New Discovery Programs	CNS	Numerous	Active discovery and development of multiple programs utilizing RAP Platform®.				CAMP4
METABOLIC DISEASES							
CMP-001	Urea Cycle Disorders	*CPS1*	Exploring potential partnership opportunities.				CAMP4
COLLABORATIONS							
Strategic research collaboration to identify and develop antisense oligonucleotide (ASO) drug candidates for multiple gene targets relevant to neurodegenerative and kidney disease indications.							GSK

SYNGAP1 patient journey: Tony and his family's experience highlights the dire unmet need for disease modifying therapy

PATIENT

Tony, 11 Years Old



TONY, 3

TONY, 8

Diagnostic Journey

- Developmental delays evident at 2, one seizure at 3
- EEG confirmed epilepsy, negative chromosomal microarray, variant confirmed by RNA Seq
- Pathogenic diagnosis at 4

CAREGIVER + FAMILY BURDEN

Immense Caregiver Burden

TONY, 8

"His spontaneous aggression leads to bruises and scary moments for family members and makes it very challenging to find childcare."

"Requires transferring to a special school. Tony is getting stronger and the future is scary."

"My life is dedicated to this cause; as a parent, it's the number one thing I strive to do for my sons: alleviate Tony's suffering to help him live the best possible life"

- Tony's Father

Patient story and images used with permission; 'Our Son Has a Rare Genetic Disorder, Life Is Risky for Us' Mike Graglia, Newsweek (January 25, 2023)

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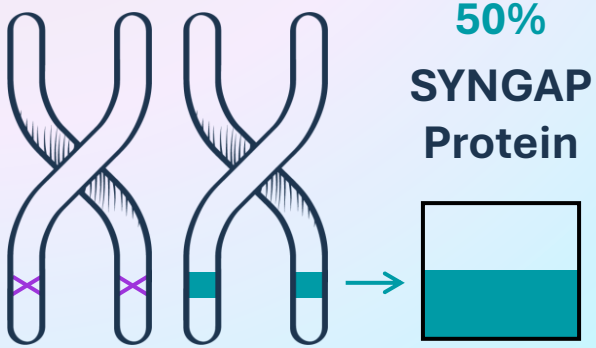
SYNGAP1 is a true haploinsufficiency with >10,000 US patients in need of therapy

Haploinsufficiency results in 50% of normal protein levels

SYNGAP1 Haploinsufficiency



50%
SYNGAP Protein

>10,000 SYNGAP1 patients in the US

6.1–10 per 100K incidence rate [1,2]

100% have intellectual disability, **~85%** have seizures, potentially experiencing **10+ per day** [3,4,5]

High unmet need for disease-modifying therapy

ICD-10 code assigned in 2021 **F78.A1**

0 approved disease modifying therapies

[1] López-Rivera et al., Brain, 2020; [2] Marotta et al., Curr Probl Pediatr Adolesc Health Care, 2024; [3] Holder et al., GeneReviews, 2019; [4] SYNGAP-Related Epilepsy, Epilepsy Foundation (Accessed May 2025); [5] Vlaskamp et al. Neurology, 2019

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Dire unmet need for a targeted disease modifying therapy to alter SYNGAP1's devasting disease course

JAELI, 16

Complex Symptoms

Developmental delay and/or intellectual disability
- ***100% of patients*** [1,2,3]

Generalized epilepsy
- ***~85% of patients*** [3,4,5]

Severe behavioral problems
- ***~70% of patients*** [1,5]

Sleep problems
- ***~60% of patients*** [2,5]

Limited communication
- ***~30% non-verbal, single words*** [4]

No Approved Therapy

Non-specific treatments have limited impact on SYNGAP1 symptoms
- Anti-seizure medications
- Cannabinoids
- Sleep medications

Polypharmacy is common – *Patient regimen* [6] *example:*
- *Epidiolex*
- *Ravicti*
- *Sodium bicarb*
- *Amantadine*

Constant patient care needed
- Caregivers vigilant at all times
  Significant lifelong cost of care

[1] Wiltrout, et al., Epilepsia, 2024; [2] Jimenez-Gomez, et al. J Neurodev Disord, 2019; [3] Holder et al., GeneReviews, 2019; [4] SYNGAP-Related Epilepsy, Epilepsy Foundation (Accessed May 2025); [5] Vlaskamp et al. Neurology, 2019; 6 SYNGAP Research Fund (SRF)

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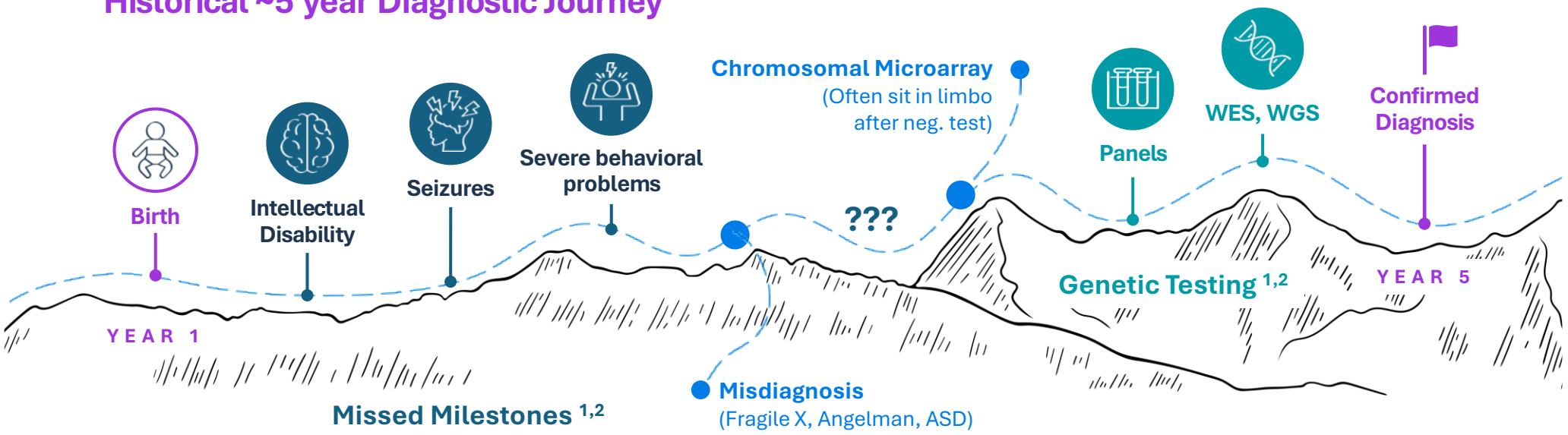
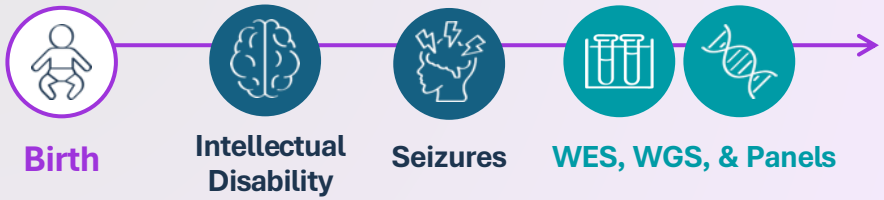
Expanding awareness and testing is enabling faster diagnosis from ~5 years to ~1 year from time of first symptom or missed milestone

Historical ~5 year Diagnostic Journey



Emerging Journey (~1 Year)



Enabled by:

- Rapid expansion of panels including SYNGAP1 [3]
- ACMG now recommends WES / WGS in pediatric pts w DD [4]
- Recent examples from Cure SYNGAP1 of pts diagnosed <1 yr

ASD = Autism Spectrum Disorders; WES = Whole Exome Sequencing, WGS = Whole Genome Sequencing, DD = Developmental Delay, ACMG = American College of Medical Genetics and Genomics
Sources: Trinity Life Sciences, Cure SYNGAP1 global census, Caregiver primary market research, [1] Vlaskamp et al. (2019), [2] Graglia et al. (2025), [3] SYNGAP1 included on gene panels provided by Invitae, Ambry Genetics, Fulgent, GeneDx, Baylor Genetics, Prevention Genetics, Revvity, [4] Manickam et al. (2021)

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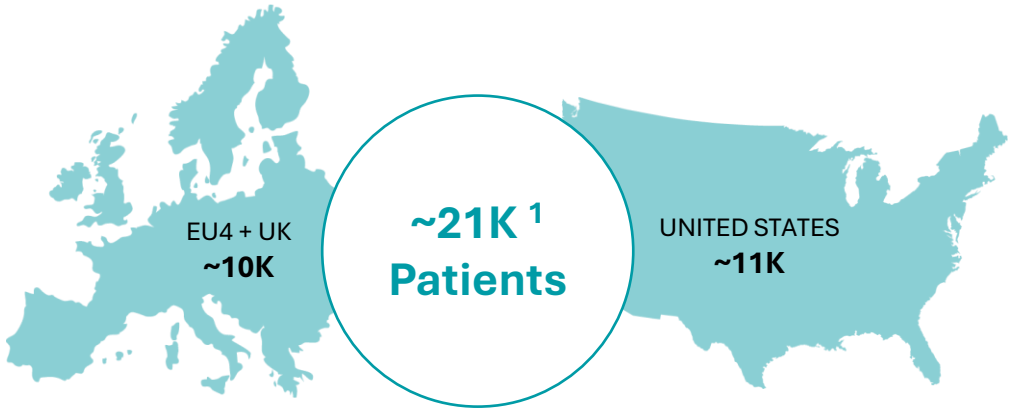
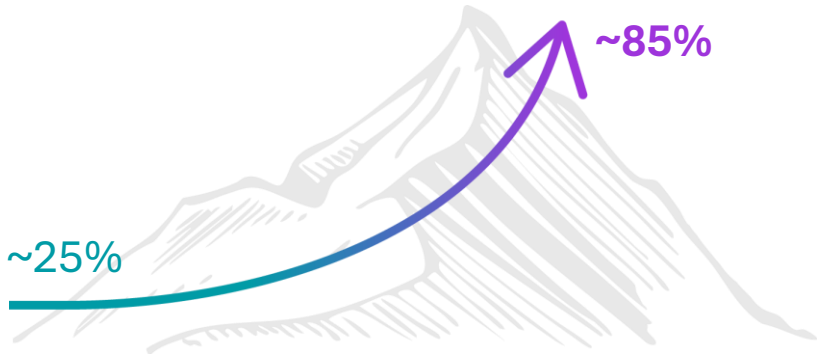
~21K patients across key global geographies (US + EU5); SYNGAP1 remains highly underdiagnosed

Major Market Prevalence



- **Scaled annual incidence to prevalence** [1]
- **Prevalence may be larger;** diagnosis rates may increase significantly with genetic testing awareness and utilization [2]
- **Third party market research** triangulated across literature, rare disease analogs, KOL interviews, Komodo claims data
- Prevalence estimates support **multi-billion $ commercial potential**

Increasing Diagnosis Rate

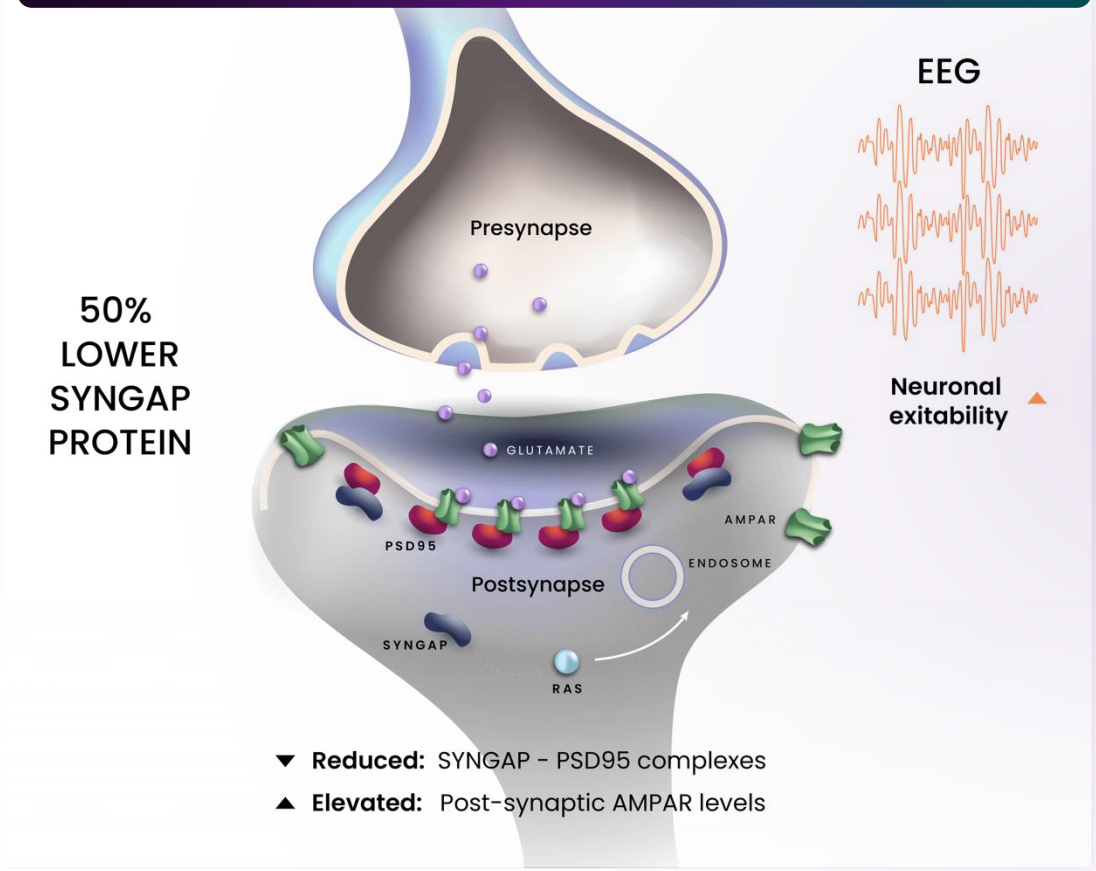


- **Predict notable increase in diagnosis:**
  - Increase in US claims, ICD-10 code added 2021 (+200 pt / yr)
  - CURE SYNGAP1 global census (+50 pt / quarter)
  - SYNGAP1 has been added to many genetic testing panels [3]
  - Increasing use of genetic testing in ASD, ID, DEE [4,5]

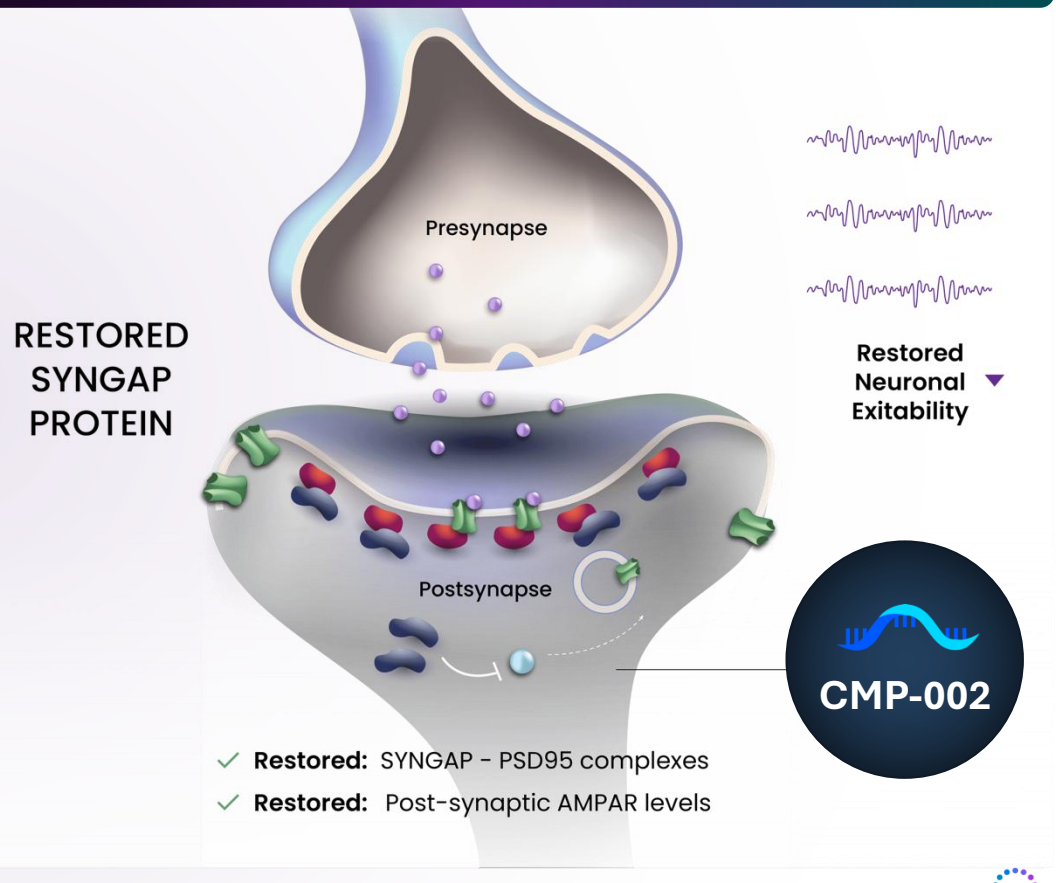
DEE = Developmental Epileptic Encephalopathies, ID = Intellectual Disability, ASD = Autism Spectrum Disorders
Sources: Trinity Life Sciences, Komodo claims data (2021-2025), Cure SYNGAP1; [1] Scaled annual incidence to prevalence using country specific live births and adjusted for mortality estimates; incidence rates based on Lopez Rivera et al. (2020) [2] Graglia et al (2025) [3] SYNGAP1 included on gene panels provided by Invitae, Ambry Genetics, Fulgent, GeneDx, Baylor Genetics, Prevention Genetics, Revvity [4] Betancur et al. (2013), (5) Sanders et al. (2018)

CAMP4 aims to increase SYNGAP protein levels, restore *SYNGAP1* function and improve disease symptoms

Mutations in *SYNGAP1* lead to decreased SYNGAP protein, causing increased synaptic firing



CMP-002 binds to a SYNGAP-specific regRNA to increase *SYNGAP1* expression, aiming to restore SYNGAP towards wild-type levels and normalize synaptic function



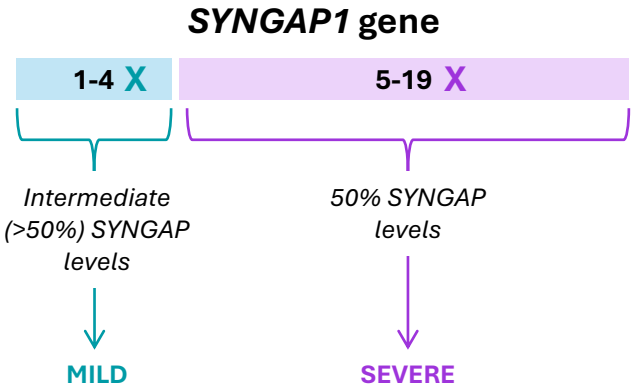
SYNGAP1 represents an ideal target for CAMP4; restoring SYNGAP protein has the potential to meaningfully improve patient outcomes

Opportunity in SYNGAP1 driven by unmet need and compelling preclinical data (presented at ASGCT May '25)

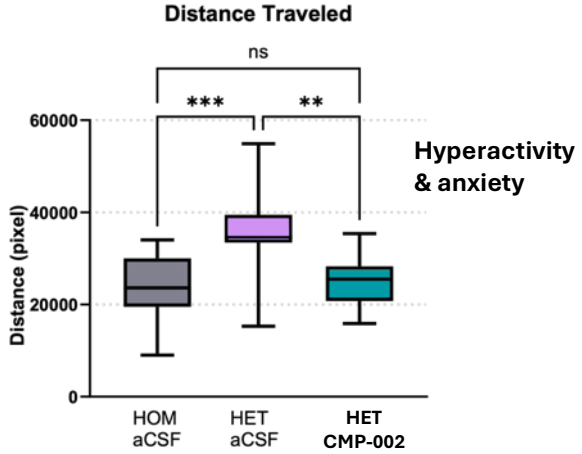
- Ultra-rare SYNGAP1 sub population with intermediate SYNGAP levels equate to milder disease state
- CMP-002 rescued functional defects in relevant human mouse model
- IT administration in NHPs well-tolerated and showed significant increase in SYNGAP in key brain regions

Precedent of ASO or siRNA activity in NHPs has translated to clinical efficacy when targeting genetic diseases

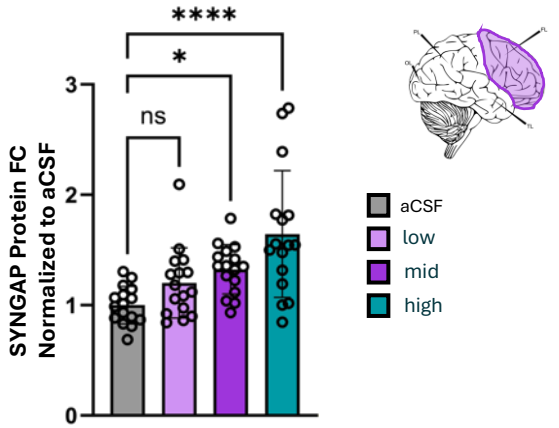
Milder disease severity (verbal responses, milder epilepsy) in minority of patients with intermediate SYNGAP levels



CMP-002 treatment rescued SYNGAP1 mouse model exhibiting disease-relevant phenotypes



CMP-002 delivered intrathecally increased SYNGAP in disease-relevant brain regions in monkeys

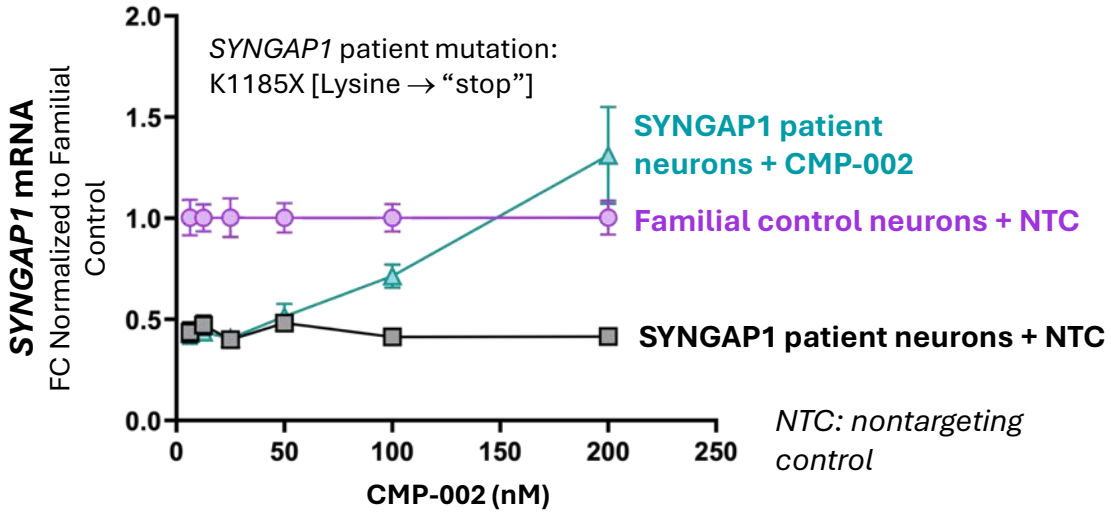


FC = Fold change

Mignot et al., 2016, Vlaskamp et al., 2019, Wiltrout et al., 2024, Kilinc et al., 2022

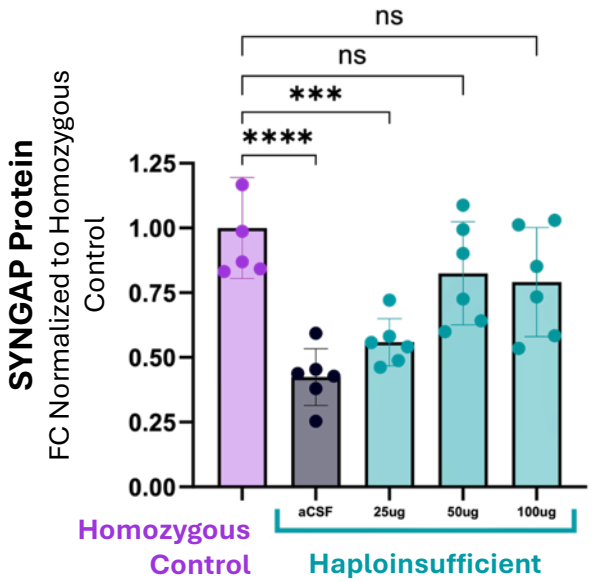
CMP-002 restores SYNGAP levels in models of haploinsufficiency

SYNGAP1 Patient iPSC-derived neurons



NTC: nontargeting control

"Humanized" *SYNGAP1* mouse



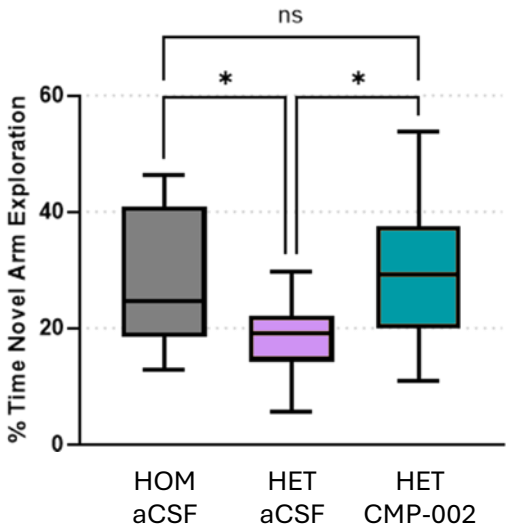
- ICV dosing in at P21
- Takedown 2-wks post-dose

Dose-dependent increase in SYNGAP approaching wild-type levels → both ASO activity and regRNA function translates from human patient-derived neurons in a dish to neurons in an intact animal brain

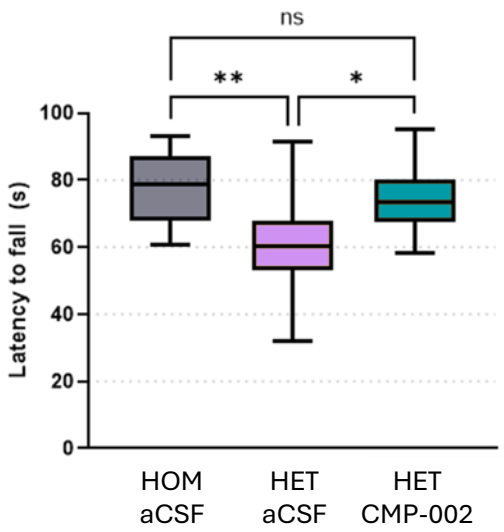
(SYNGAP-ZBTB9)[1]Bpro/Mmjax mouse strain obtained from the Mutant Mouse Resource and Research Centers (originally deposited by Benjamin Prosser, Ph.D., University of Pennsylvania).

Improved behavioral phenotypes in SYNGAP1 humanized haploinsufficient mice given CMP-002

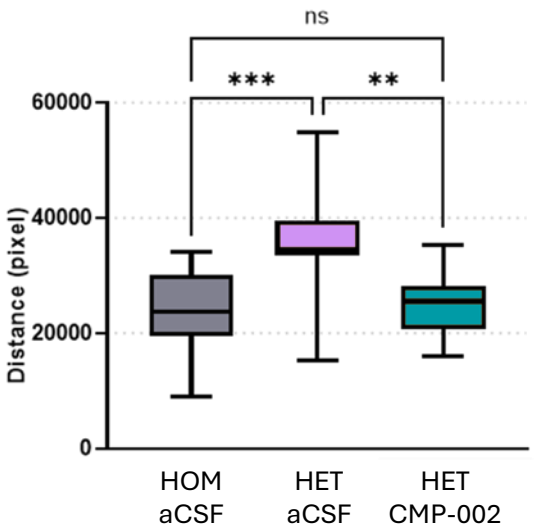
Learning & Memory



Motor Function



Hyperactivity



aCSF = artificial CSF

*, p <0.05
**, p<0.01
***, p<0.001

- HOM - aCSF (N=12)
- HET- aCSF (N=18)
- HET- CMP-002 (N=10)

Neonatal mice administered CMP-002 and assessed within 3-weeks; protein restored to near-wild type levels

Restoring SYNGAP protein to near wild-type levels ameliorates multiple behavioral phenotypes caused by *SYNGAP1* haploinsufficiency

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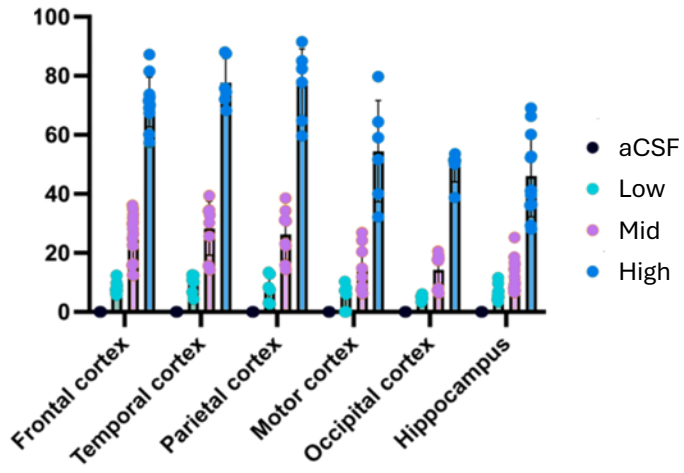
Intrathecal administration of CMP-002 to cynomolgus monkeys achieves broad brain distribution to increase SYNGAP protein levels

ASO Concentrations

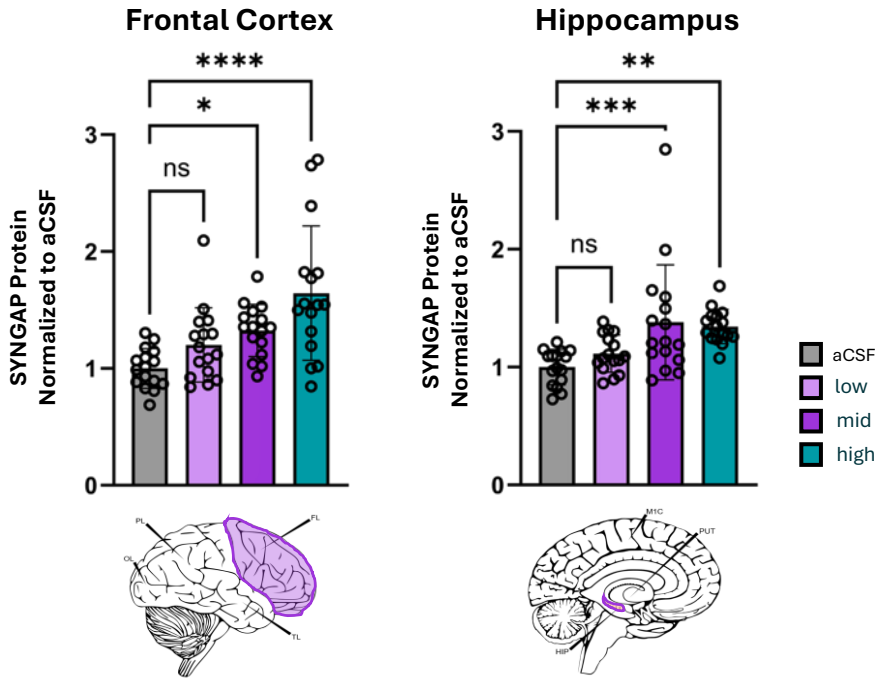
aCSF

CMP-002
(3 dose levels)

- N=4/group
- Dose Q2w x3
- Take down 2 wks post last dose



SYNGAP Protein Levels



aCSF = artificial CSF
*, p <0.05
**, p<0.01

***, p<0.001
****, p<0.0001

Summary

IT administration in NHPs was **well-tolerated**

Broad ASO distribution throughout disease-relevant brain regions

↑ SYNGAP protein throughout brain

GLP tox studies **ongoing**

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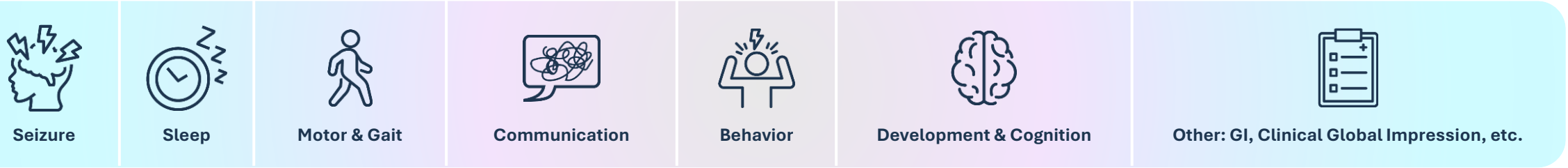
CAMP4 positioned to be first in the clinic for SYNGAP1

Standard-of-Care	• No disease modifying therapies available • Patients currently managed symptomatically, with complications of polypharmacy
Natural History Study	• Consortium-driven, funded, multi-site study* is ongoing, multiple advocacy / centers of excellence > 1 yr duration, 100 patients with 1,240 patient years of data obtainable
Center of Excellence	• Global centers of excellence, currently nodes for translational work and natural history, with readiness to expand to trial sites for rapid clinical trial conductance
Path to Clinic	• GLP toxicity studies ongoing • Potential for CMP-002 to be evaluated in a global Ph1/2 study in patients initiating as early as H2 2026
Path to Approval	• Multiple, established paths to approval for a developmental epileptic encephalopathy (DEE) • Seizure quantification + neurodevelopmental scale(s) as well as multiple clinical scales accepted by regulators

*sponsored by SYNGAP Research Foundation

Multiple regulatory precedents and validated measures to demonstrate disease-modifying effect for PoC in First-in-Human

Phase 1/2 Endpoint Categories



Regulatory Approval Paradigms

Seizure +

- Seizure as primary with secondary neurodevelopmental endpoints: fits with SYNGAP1 high seizure burden
- Multiple methodologies and regulatory-accepted scales
- Regulatory precedents across Dravet, Lennox-Gastaut, CDKL5

Combo – Composite Model (Rett Syndrome/ Angelman)

- Genetic neurodevelopmental disorders with seizures: Rett, Angelman paradigms use one-or-more endpoints to achieve PoC success
- In Rett, trofinetide approved on RSBG + CGI. FDA buy-in for 1 or more of 28 developmental milestones (DM) for Taysha
- In Angelman, Ultragenyx and Ionis: Bayley-4 and either cognition or receptive communication co-primaries

**Endpoint categories and regulatory approval paradigms are illustrative and subject to regulatory discussions*

Development path and design to maximize speed and success

Ph 1/ 2 Optimized for:

- Rapid enrollment
- Efficacy assessment across multiple domains of disease
- Optimal biological dose Selection
- Optionality around endpoint precedent and potential expedited regulatory programs participation

First-in-Human Clinical Trial Design Approach

- Straight to MAD
- Population: enriched genotype and phenotype population
- Aim to start in pediatric patients
- Comprehensive assessments that map to natural history for supportive regulatory comparison
- Open-Label Extension to demonstrate long-term disease-modifying benefit

Potential Cohort Study Design Schematic

Pending confirmation with human research ethics boards and regulators

CAMP4 team has been pioneering the field of regRNAs

Josh Mandel-Brehm
President & CEO

Biogen · polarispartners · genzyme

Kelly Gold
Chief Financial Officer

Biogen · Deutsche Bank

Dan Tardiff, PhD
Chief Scientific Officer

Pfizer · Whitehead Institute · Yumanity Therapeutics

Yuri Maricich, MD
Chief Medical Officer

corixa · Cavion · PEAR Therapeutics

Caleb Moore
Chief Business Operations Officer

genzyme · ACCELERON · CUBIST Pharmaceuticals

Michelle Gates
Chief People Officer

Akamai

Alla Sigova, PhD
SVP, Head of Platform

SAIL Biomedicines · Whitehead Institute

Satya Kuchimanchi, PhD
SVP, Technical Operations

Triplet Therapeutics · Alnylam Pharmaceuticals

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- Rick Young, PhD

* Board Chair

Building momentum and a unique value proposition

- We recently repositioned our pipeline to focus on SYNGAP1 and have funded the program to **accelerate the path to the clinic** as early as H2 2026

- SYNGAP1 represents a **major market opportunity** and is the cornerstone program for CAMP4
  - Future opportunity to build pipeline around additional developmental epileptic encephalopathies and haploinsufficient neurodevelopmental disorders

- CMP-002 poised to enter the clinic as early as second half of 2026, making it the **first potentially disease modifying medicine in the clinic** for SYNGAP1

- Leveraging RAP Platform® to **build CNS-focused pipeline** and drive value through BD
  - CNS is a target rich area for upregulation approach to address rare and prevalent diseases
  - RAP Platform® has been tested in >40 target genes associated with diseases across different tissues, generating opportunities for both pipeline expansion and high-value partnerships
  - Intend to pursue additional discovery partnerships to fully capitalize on platform's potential

Thank You