Stoke Therapeutics

NASDAQ: STOK

January 2024

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This presentation contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including, but not limited to, the ability of STK-001 to treat the underlying causes of Dravet syndrome and reduce seizures or show improvements in behavior or cognition at the indicated dosing levels or at all; the timing and expected progress of clinical trials, data readouts and presentations for STK-001 and STK-002; the timing of regulatory interactions or the outcomes thereof; our future operating results, financial position and cash runway; and our expectations, plans, aspirations and goals, including those related to the goals of our collaboration with Acadia. Statements including words such as "anticipate," "plan," "will," "continue," "expect," or "ongoing" and statements in the future tense are forward-looking statements. These forward-looking statements involve risks and uncertainties, as well as assumptions, which, if they prove incorrect or do not fully materialize, could cause our results to differ materially from those expressed or implied by such forward-looking statements, including, but not limited to, risks and uncertainties related to: Stoke's ability to advance, obtain regulatory approval of, and ultimately commercialize its produce candidates; the timing of data readouts and interim and final results of preclinical and clinical trials; positive results in a clinical trial may not be replicated in subsequent trials or successes in early stage clinical trials may not be predictive of results in later stage trials; preliminary interim data readouts of ongoing trials may show results that change when such trials are completed; Stoke's ability to fund development activities and achieve development goals into 2025; Stoke's ability to protect its intellectual property; global business, political and macroeconomic conditions, including inflation, interest rate volatility, cybersecurity events, uncertainty with respect to the federal budget, instability in the global banking system and volatile market conditions, and global events, including public health crises, and ongoing geopolitical conflicts, such as the conflicts in Ukraine and the Middle East; and other risks and uncertainties described under the heading "Risk Factors" in Stoke's Annual Report on Form 10-K for the year ended December 31, 2022, its quarterly reports on Form 10-Q and the other documentation Stoke files from time to time with the Securities and Exchange Commission. These forward-looking statements speak only as of the date of this presentation, and we undertake no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.

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OUR GOAL:

Upregulate protein expression to treat the underlying cause of severe genetic diseases

Stoke's pipeline offers potential first-in-class disease modifying new medicines for diseases caused by protein insufficiency

STK-001 for Dravet syndrome

A severe and progressive genetic epilepsy

STK-002 for Autosomal Dominant Optic Atrophy (ADOA)

The most common inherited optic nerve disorder

Rett syndrome, Syngap1 syndrome

Severe and rare genetic neurodevelopmental diseases

And beyond…

~6,500 additional genes with TANGO target signatures

Advantages of Stoke's Approach vs. Other Genetic Approaches

Selectively boosts expression only in tissues where the protein is normally expressed

No observed unwanted off-target genetic effects

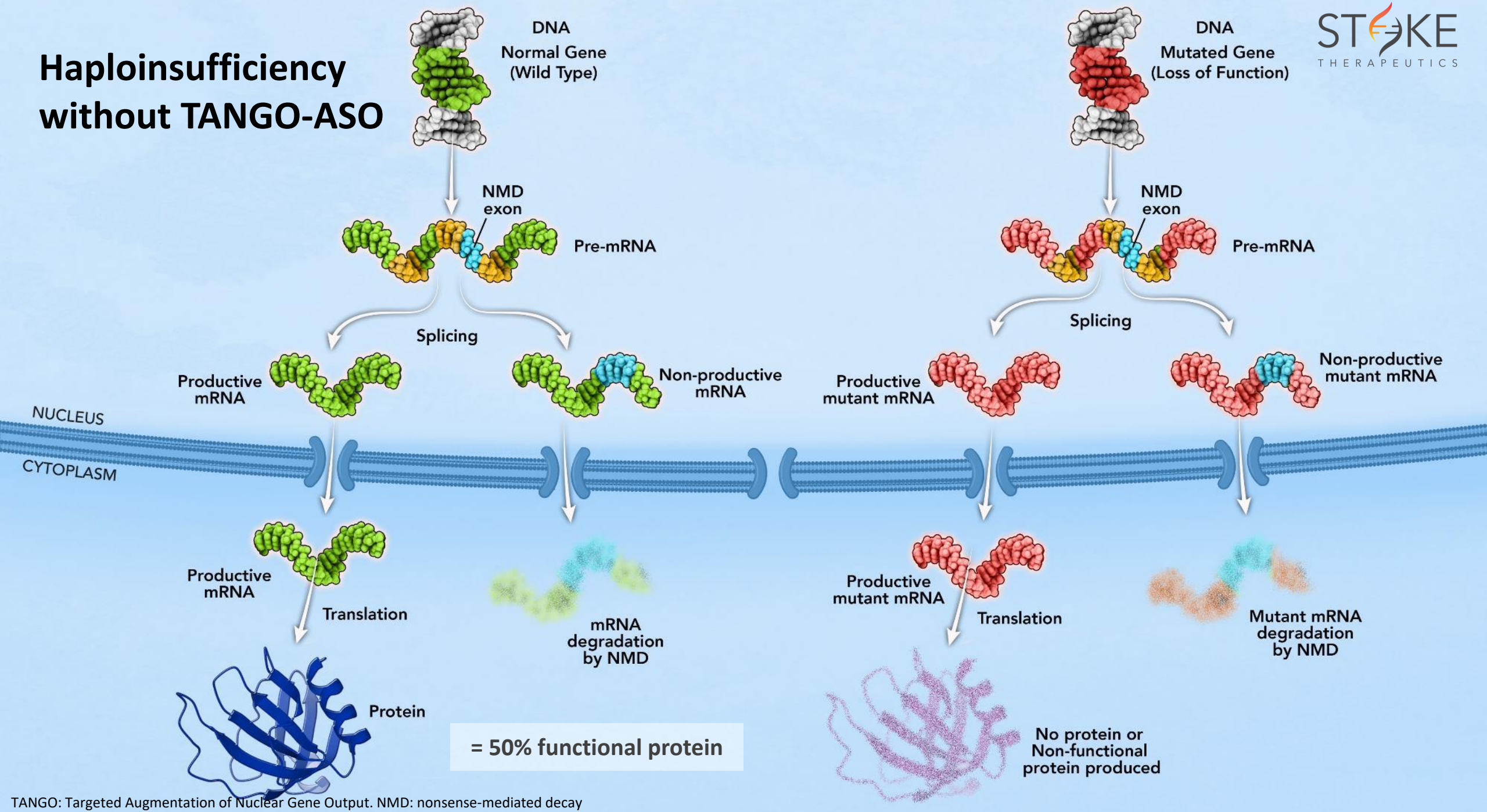
Utility across small and large gene targets and mutations

Does not alter DNA

Ability to control dose level and duration

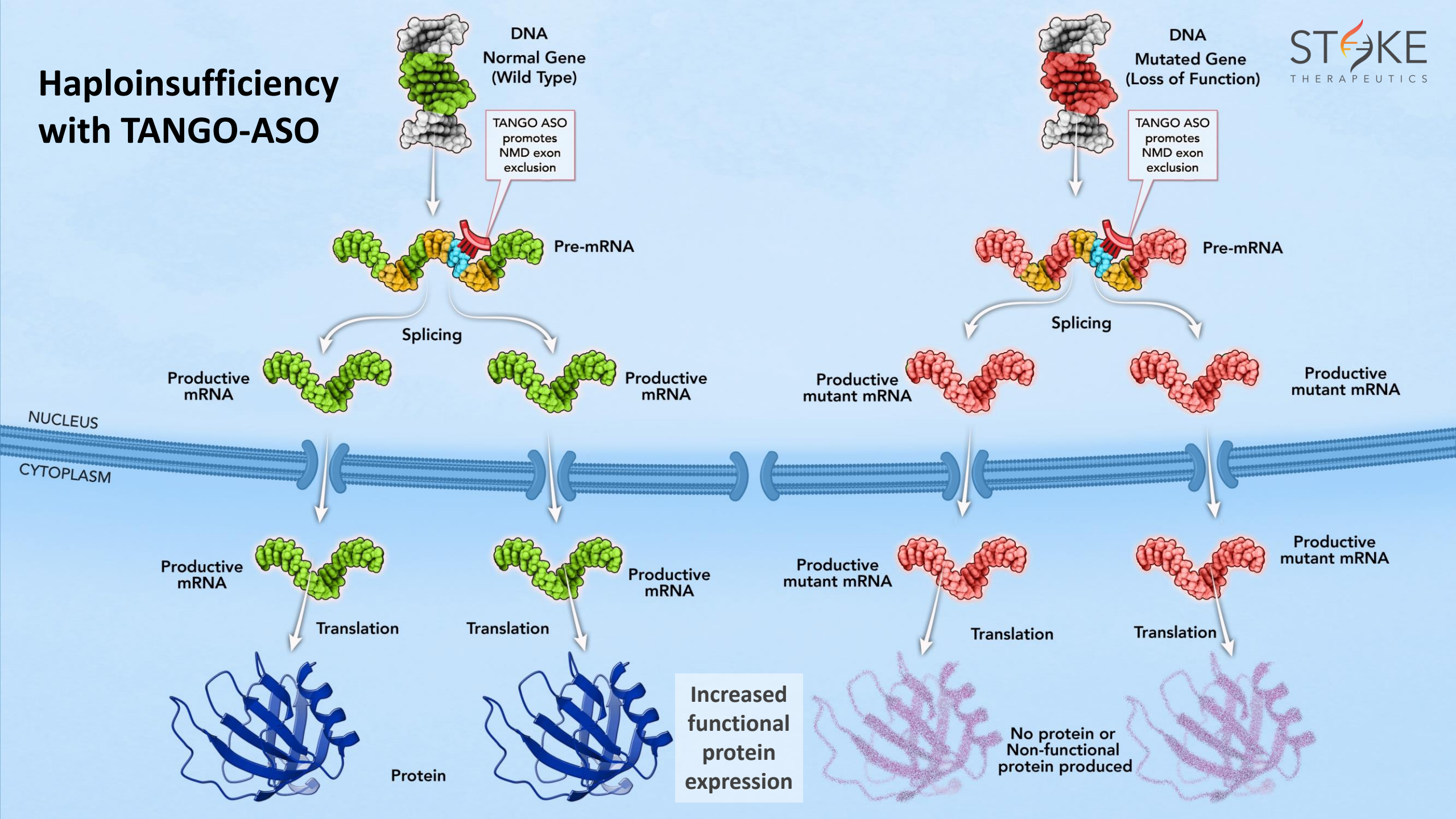
Simple and scalable manufacturing

Haploinsufficiency without TANGO-ASO



TANGO: Targeted Augmentation of Nuclear Gene Output. NMD: nonsense-mediated decay

Haploinsufficiency with TANGO-ASO



Dravet Syndrome: A Severe, Progressive Genetic Epilepsy

85%
of cases caused by a **HAPLOINSUFFICIENCY** of the *SCN1A* gene

RESULTING in

50%
$Na_V1.1$ protein expression

1 out of 16,000
babies are born with Dravet syndrome

Up to 20%
of children and adolescents with Dravet syndrome die before adulthood, due to SUDEP[1], prolonged seizures, seizure-related accidents or infections

Seizures are not adequately controlled in **90%** of people with Dravet syndrome

~35,000
people affected in the U.S., Canada, Japan, Germany, France and the UK

Dravet syndrome is not concentrated in a particular geographic area or ethnic group

[1] Sudden Unexpected Death in Epilepsy

Sources: Symonds, J. et al., *Early childhood epilepsies: epidemiology, classification, aetiology, and socio-economic determinants. Brain,* 2021. *2018 Health Advances Report*; Djémié et al., *Molecular Genetics & Genomic Medicine,* 2016; Lagae et al., *Developmental Medicine & Child Neurology,* 2017; Wu, Y. et al., *Incidence of Dravet Syndrome in a US Population. Pediatrics,* 2015. Nabbout et al., *Orphanet Journal of Rare Diseases,* 2013

The Effects of Dravet Go Beyond “Just Seizures”

Intellectual Disability & Developmental Delays

*“Over time, we have seen **slow, steady decline** in all areas, from speech, to mobility, endurance, loss of energy, tolerance for stimulation, stamina, etc.”*

Movement & Balance

*“We're disappointed when [our son’s] physical activity is limited and the short walk or visit that we plan with his grandmothers must now be changed to a longer **wheelchair ride**.”*

Language & Speech Disturbances

*“At age 19, [our son] stopped talking, seemingly **losing his capacity for speech** overnight. Most days he is silent, and though he can understand simple conversation he is largely **unable to express himself**.”*

Sleep Abnormalities

*“Every single night, he has **seizures in his sleep**. In addition to all of the other comorbidities of DS, he's **robbed of the basic human necessity** of getting a good night's sleep. This impacts our entire family, as it is hard to function on **so little sleep day after day**."*

Source: Voice of the Patient Report Published by the Dravet Syndrome Foundation, May 2022

STK-001 is on Track to be the First Disease-Modifying Treatment for Dravet Syndrome

Multiple medicines available for

Seizure management

Available medicines used to control seizures:

- Acetazolamide
- Benzodiazepines
- Brivaracetam
- Cannabidiol
- Carbamazepine
- Clobazam
- Ethosuximide
- Felbamate
- Fenfluramine
- Lamotrigine
- Levetiracetam
- Mesuximide
- Oxcarbazepine
- Phenytoin
- Rufinamide
- Stiripentol
- Topiramate
- Valproate products
- Zonisamide

Despite these treatments, seizures are not adequately controlled in 90% of patients with Dravet syndrome

No medicines available for

Syndrome management

STK-001

The first potential disease-modifying approach to address the genetic cause of Dravet syndrome

Data Support the Potential for Disease Modification with STK-001

BUTTERFLY Natural History Data

(patients 2-18 taking standard anti-seizure medicines)

- ✖ No meaningful improvement in seizure frequency

- ✖ Widening gap in cognition and behavior:
  - Expressive communication
  - Receptive communication
  - Gross motor skills

Data from Phase 1/2a and OLE Clinical Studies

- ✔ Substantial and sustained reductions in seizures with initial treatment (70mg)
  - Median reductions observed among all patients at 3 and at 6 months
  - Sustain reductions with ongoing treatment

- ✔ Substantial improvements in measures of cognition and behavior:
  - Expressive communication
  - Receptive communication
  - Gross motor skills

Safety
Generally well-tolerated in studies to date

PK data
Higher drug exposure in brain leads to greater reductions in seizure frequency

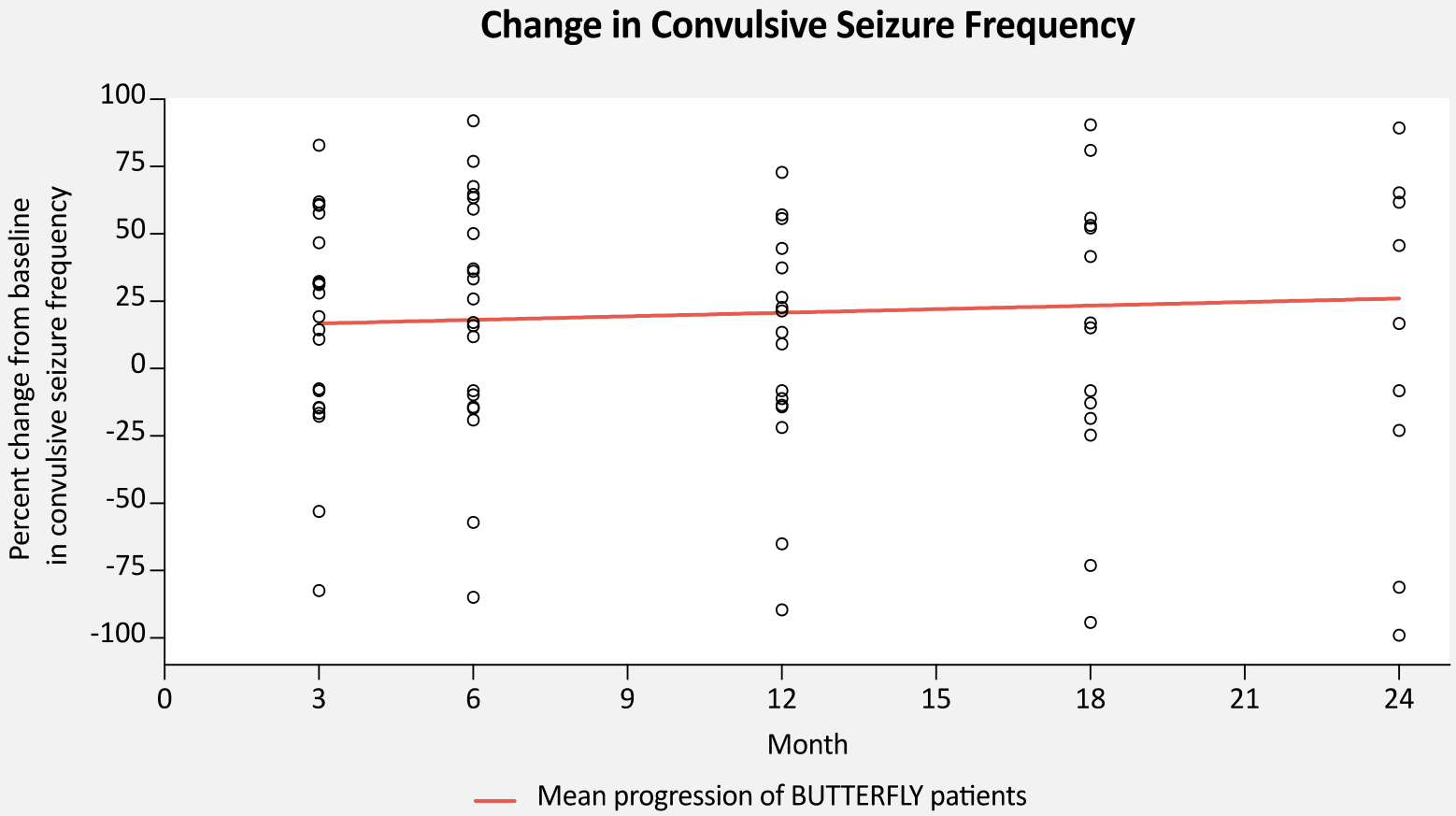
Preclinical Findings Support Clinical Development of STK-001

- Single dose restores $Na_V1.1$ to near-normal levels for >3 months in DS mice
- Significantly reduces mortality and seizure frequency in DS mice
- Achieves broad distribution and increases $Na_V1.1$ protein expression in NHPs
- NHP toxicology studies support current clinical dosing

Sources: Targeted Augmentation of Nuclear Gene Output (TANGO) of SCN1A reduces seizures and rescues parvalbumin positive interneuron firing frequency in a mouse model of Dravet syndrome (AES 2020). Wengert ER, Wagley PK, Strohm SM, Reza N, Wenker IC, Gaykema RP, Christiansen A, Liau G, Patel MK. Targeted Augmentation of Nuclear Gene Output (TANGO) of Scn1a rescues parvalbumin interneuron excitability and reduces seizures in a mouse model of Dravet Syndrome. Brain Res. 2022;1775:147743. Stoke data. TANGO oligonucleotides for the treatment of Dravet Syndrome: Safety, biodistribution and pharmacology in the non-human primate (AES 2019)

No Improvement in Convulsive Seizure Frequency Despite Treatment with Standard Anti-Seizure Medicines Over 2 Years

BUTTERFLY natural history study of 2-18 years old patients with Dravet syndrome

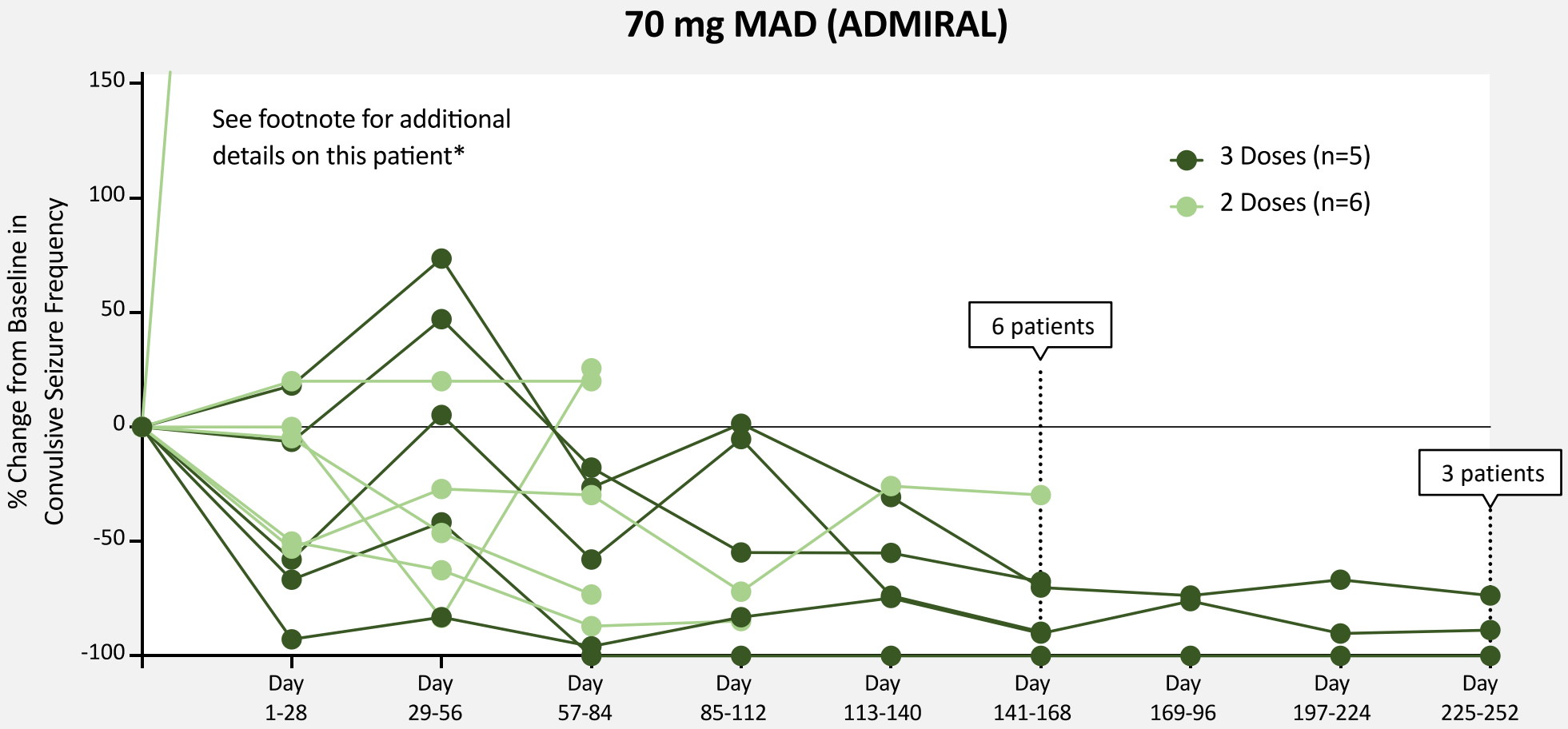


Patients were treated with the best available anti-seizure medicines	
Median baseline convulsive seizure frequency per 28 days (95% CI), n=26	
10.0 (5.50, 15.5)	
Most common ongoing anti-seizure medicines, n (%)	
Clobazam	25 (69.4%)
Fenfluramine	16 (44.4%)
Stiripentol	14 (38.9%)
Valproic Acid	14 (38.9%)
Cannabidiol	12 (33.3%)
Levetiracetam	8 (22.2%)

Source: 24-Month Analysis of BUTTERFLY: A Prospective, Observational Study to Investigate Cognition and Other Non-seizure Comorbidities in Children and Adolescents with Dravet Syndrome (DS), AES 2023.

Substantial Reductions in Seizure Frequency Observed in Patients Treated with 2 or 3 Initial Doses of STK-001 (70mg)

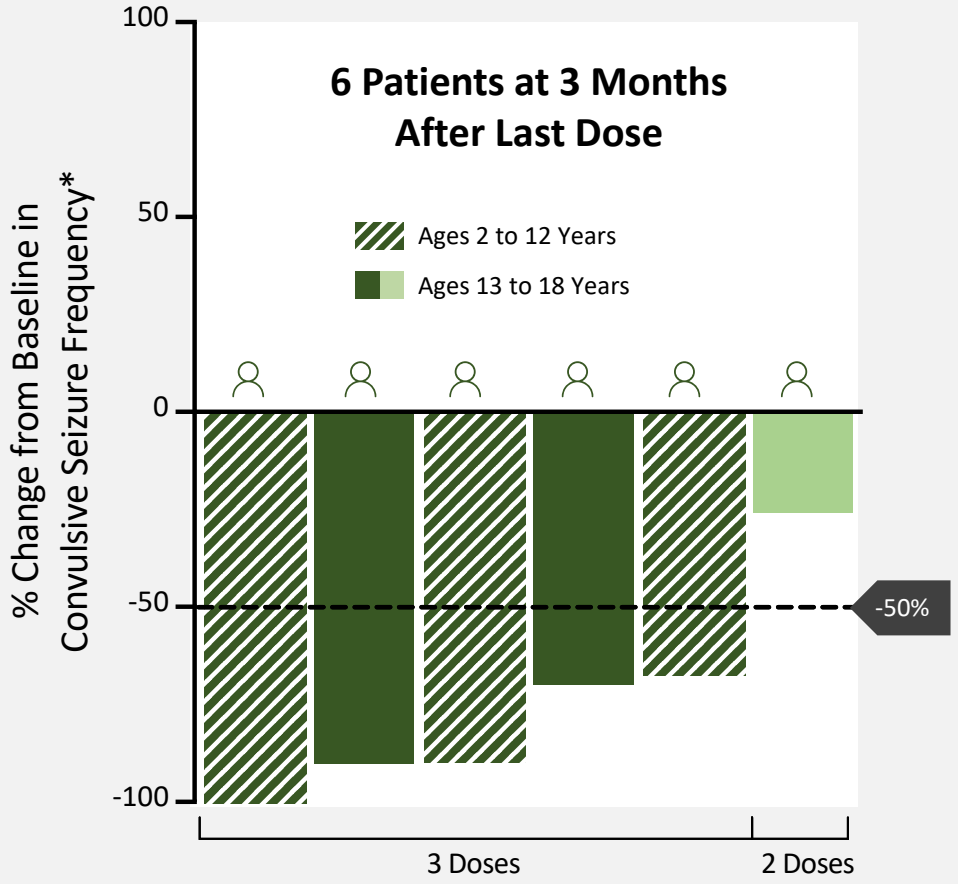
Data as of July 2023. All patients have now completed the study. End of study data anticipated 1Q 2024.

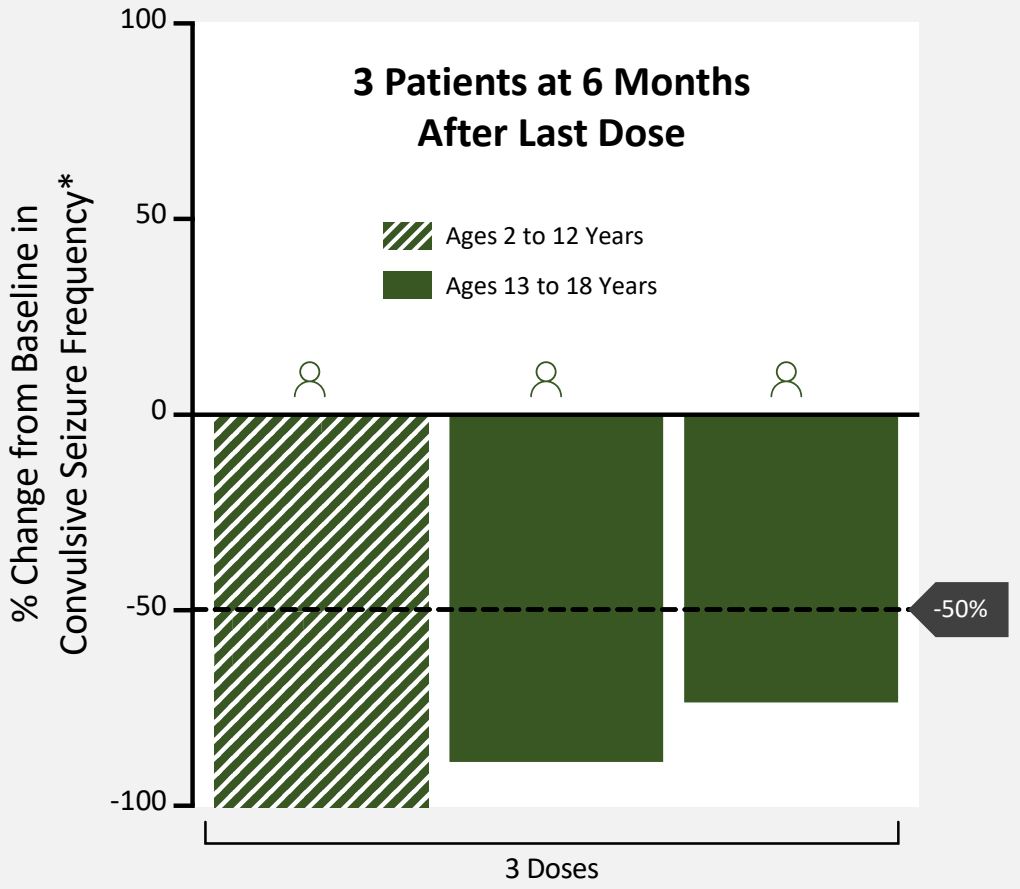


*D1-D28: +825%, D29-D56: +626%, D57-D84: +1125%, D85-D112: +717%

Source: MONARCH and ADMIRAL: Phase 1/2a Studies in US and UK Investigating Safety and Drug Exposure of STK-001, an Antisense Oligonucleotide (ASO), in Children and Adolescents with Dravet Syndrome (DS), AES 2023.

Median Reductions in Convulsive Seizure Frequency Observed Among All Patients with 2 or 3 Doses of 70mg at 3 and at 6 Months



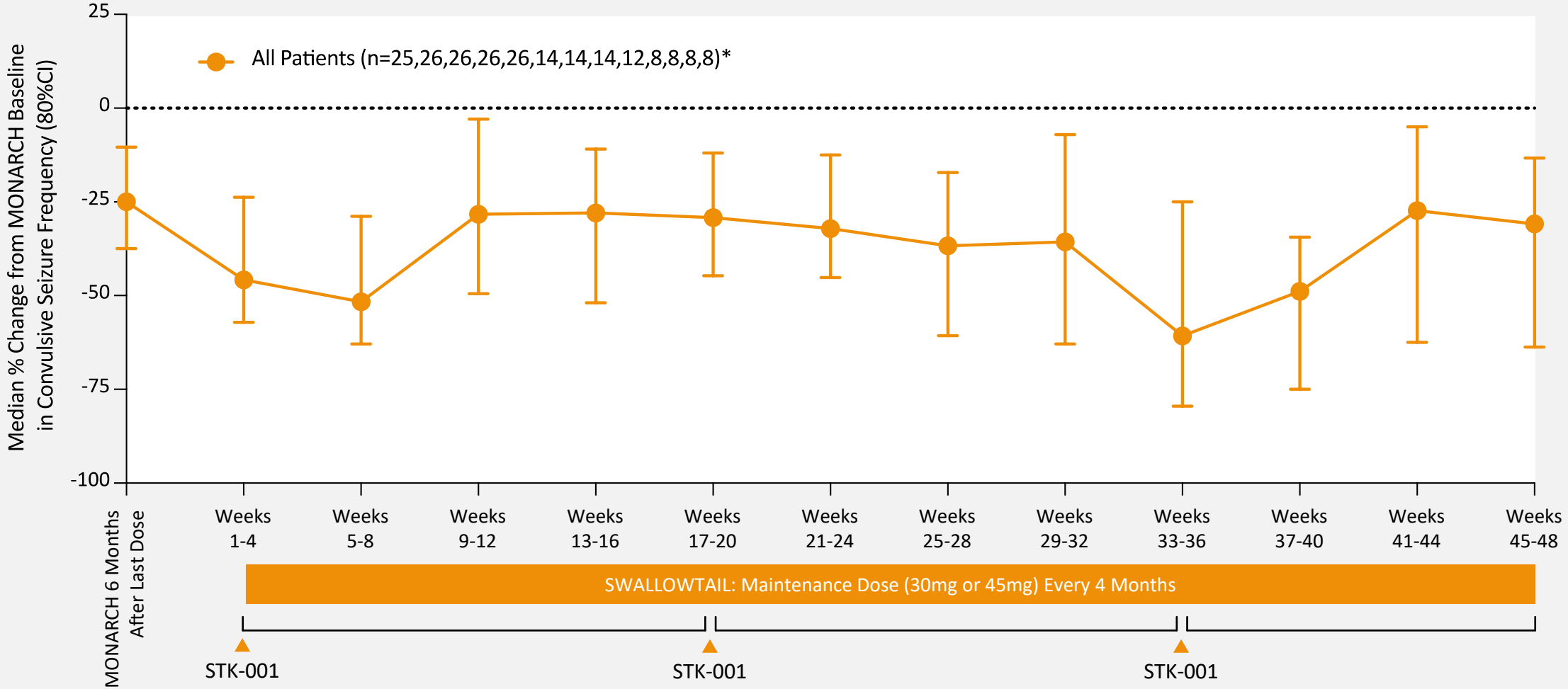


*28-day interval prior to 3 months or 6 months After Last Dose for all patients. 1 patient in 70 mg cohort received Dose 3 late; therefore, interval does not extend fully to 3 and 6 months After Last Dose for this patient. Data cutoff dates: MONARCH 13APR2023; ADMIRAL 12APR2023 and 21JUN2023

Source: MONARCH and ADMIRAL: Phase 1/2a Studies in US and UK Investigating Safety and Drug Exposure of STK-001, an Antisense Oligonucleotide (ASO), in Children and Adolescents with Dravet Syndrome (DS), AES 2023.

OLE Data: Sustained Reductions in Convulsive Seizure Frequency

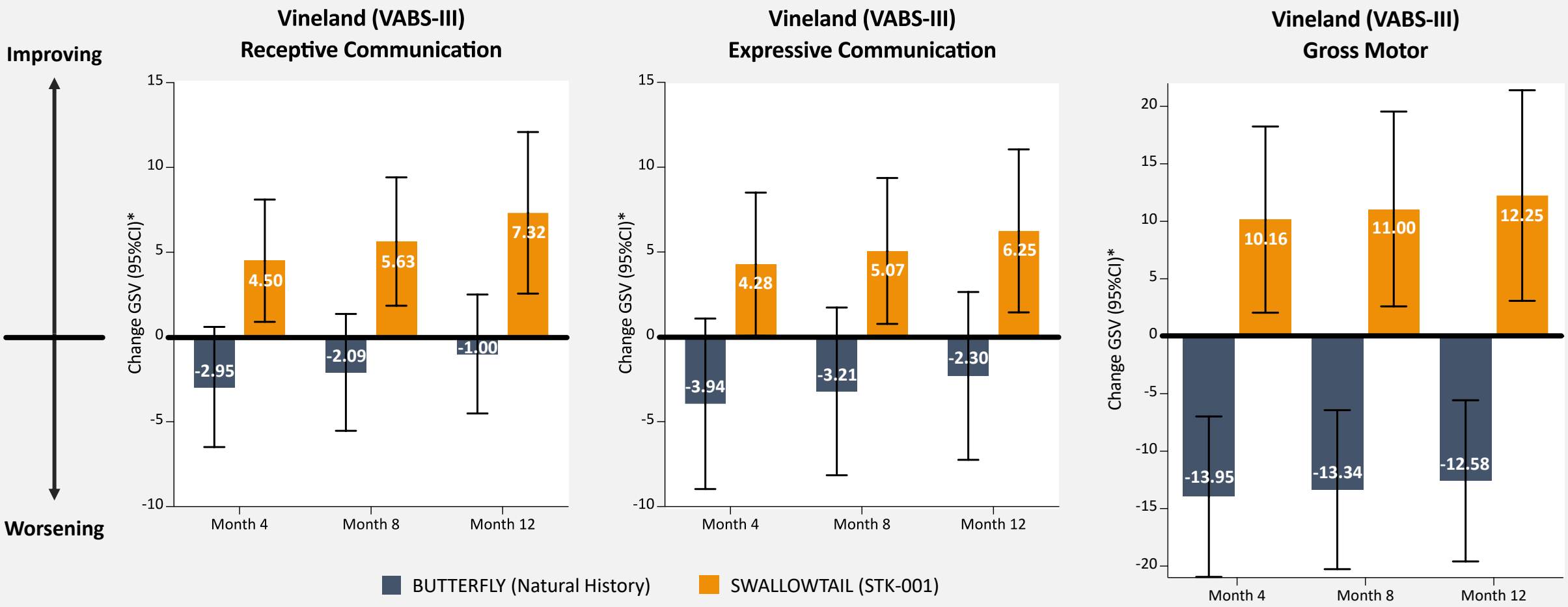
Effects observed with ongoing treatment with STK-001 at 30mg, 45mg



*No exclusion for AED modification in MONARCH or SWALLOWTAIL. Data cutoff dates: MONARCH 13APR2023; SWALLOWTAIL 24MAR2023

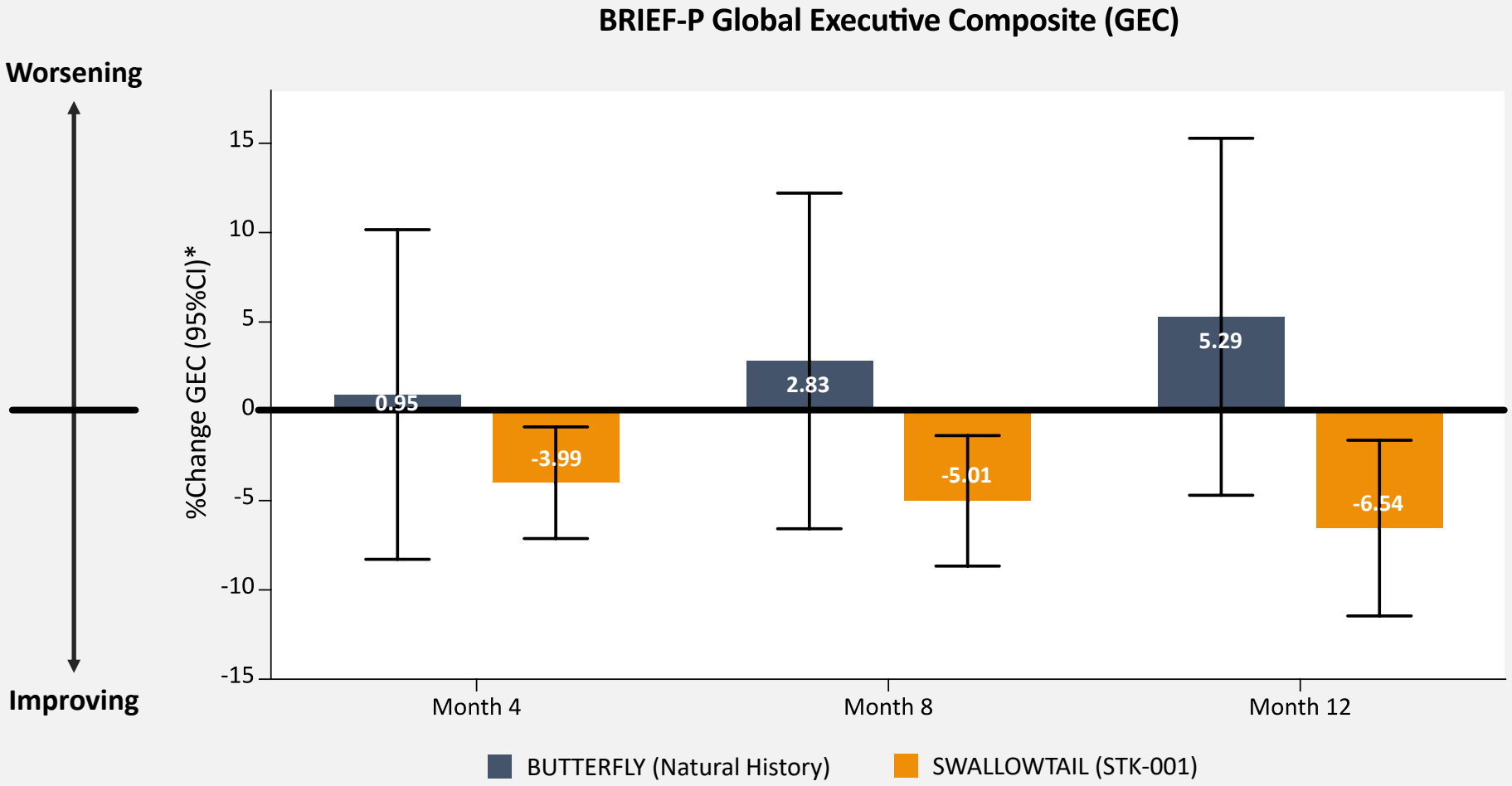
Source: SWALLOWTAIL and LONGWING: Open-Label Extension (OLE) Studies for Children and Adolescents with Dravet Syndrome (DS) who Previously Participated in a Study of Antisense Oligonucleotide (ASO) STK-001, AES 2023.

OLE Data (30mg, 45mg): Substantial Improvements in Communication and Gross Motor Skills with Ongoing Treatment



Mixed model repeated measures with AR(1) covariance structure. Baseline covariates in BUTTERFLY matched to SWALLOWTAIL. Analysis includes patients who received 30 or 45 mg for all doses in SWALLOWTAIL; BUTTERFLY sample size: n=36 at screen, n=27 at Month 12; SWALLOWTAIL sample size: n=24 at screen, n=9 at Week 48 and n=5 at Week 64. GSV = Growth Scale Value.
Source: SWALLOWTAIL and LONGWING: Open-Label Extension (OLE) Studies for Children and Adolescents with Dravet Syndrome (DS) who Previously Participated in a Study of Antisense Oligonucleotide (ASO) STK-001, AES 2023.

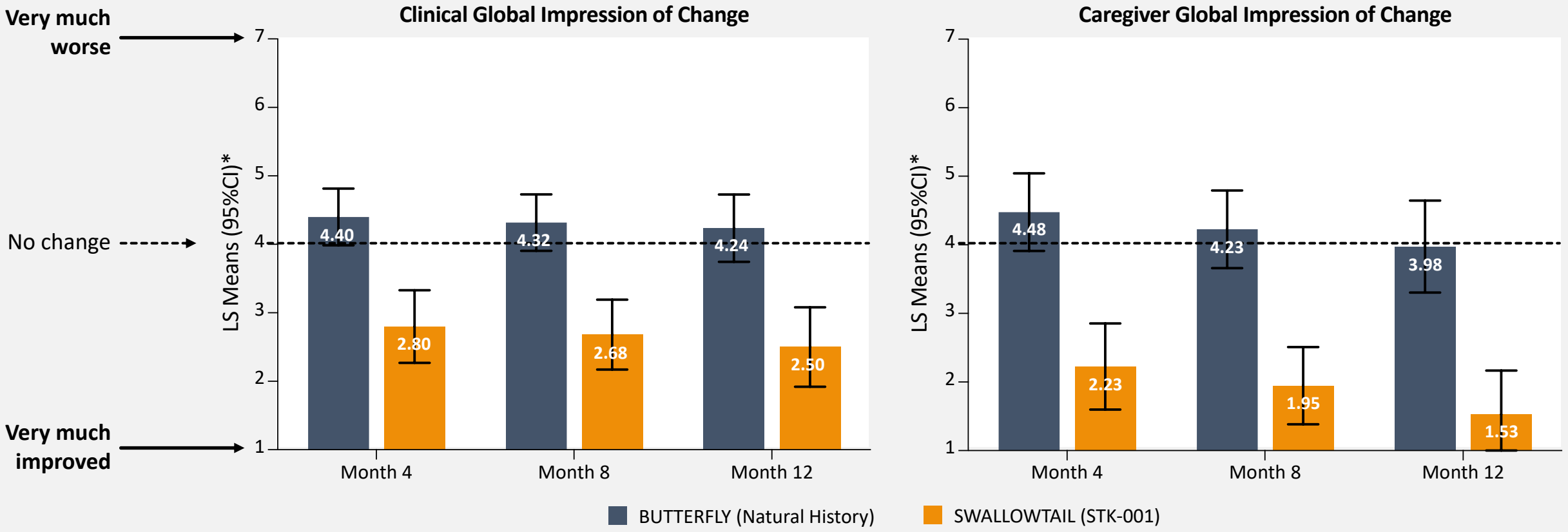
OLE Data (30mg, 45mg): Substantial Improvements in Executive Function with Ongoing Treatment



*Mixed model repeated measures with AR(1) covariance structure. Baseline covariates in BUTTERFLY matched to SWALLOWTAIL. Analysis includes all patients who received 30 or 45 mg for all doses in SWALLOWTAIL; BUTTERFLY sample size: n=36 at screen, n=30 at Month 12; SWALLOWTAIL sample size: n=25 at screen, n=9 at Week 48 and n=5 at Week 64. BRIEF-P measures executive function in children, such as the ability to organize thoughts and have working memory. Source: SWALLOWTAIL and LONGWING: Open-Label Extension (OLE) Studies for Children and Adolescents with Dravet Syndrome (DS) who Previously Participated in a Study of Antisense Oligonucleotide (ASO) STK-001, AES 2023.

OLE Data (30mg, 45mg): Substantial Improvements in Overall Condition Compared to BUTTERFLY Natural History Results

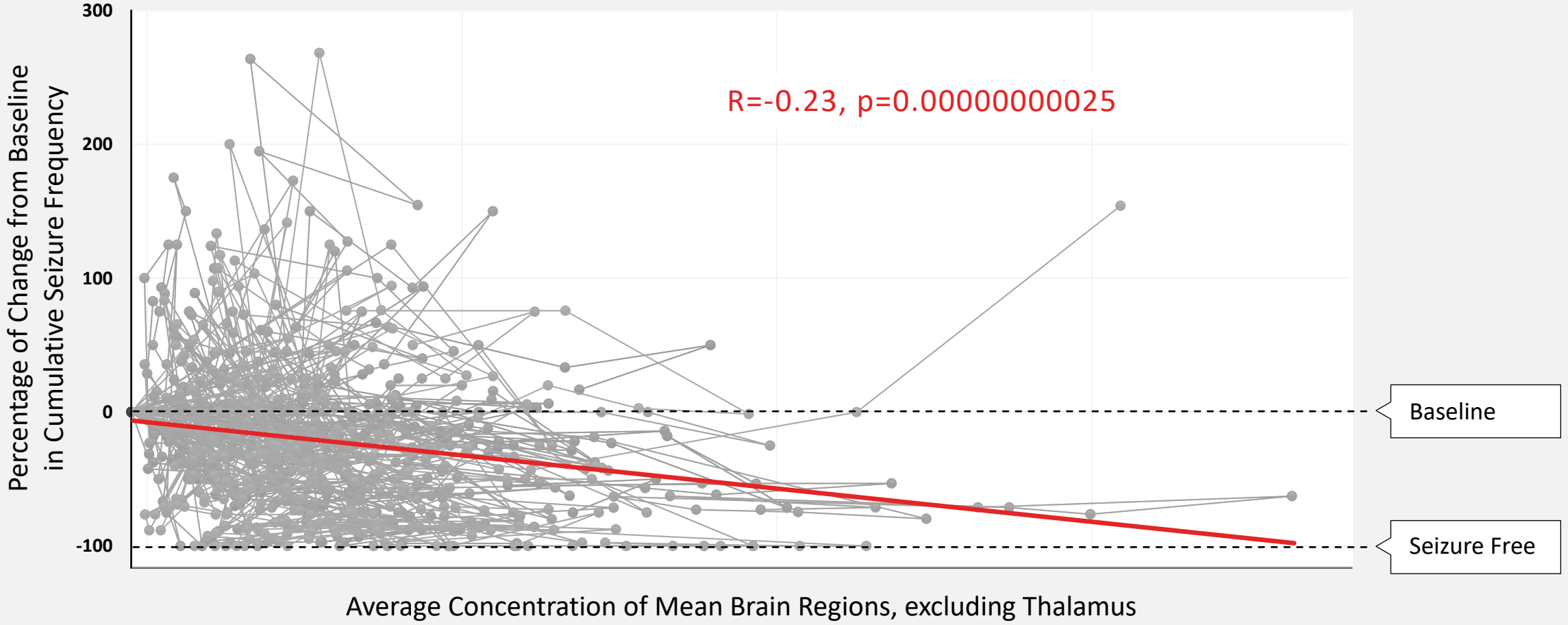
Consistent responses across caregiver and clinician ratings



Mixed model repeated measures with AR(1) covariance structure. Baseline covariates in BUTTERFLY matched to SWALLOWTAIL. Analysis includes all patients who received 30 or 45 mg for all doses in SWALLOWTAIL. For CGI-C, BUTTERFLY sample size: n=32 at Month 3, n=29 at Month 12; and for CaGI-C, BUTTERFLY sample size: n=27 at Month 3, n=24 at Month 12. For both CGI-C and CaGI-C, SWALLOWTAIL sample size: n=25 at Week 16, n=9 at Week 48 and n=5 at Week 64. CGI and CaGI in BUTTERFLY were adapted for cognition. CGI-C=Clinical Global Impression of Change and CaGI-C=Caregiver Global Impression of Change. Sources: SWALLOWTAIL and LONGWING: Open-Label Extension (OLE) Studies for Children and Adolescents with Dravet Syndrome (DS) who Previously Participated in a Study of Antisense Oligonucleotide (ASO) STK-001, AES 2023.

Higher Brain Exposure Leads to Greater Seizure Reduction

PK Modeling of exposure-seizure relationship



Source: Utilization of a Pharmacokinetic (PK) Model for STK-001 in Patients with Dravet Syndrome (DS) To Support Selection of Dosing Regimens in Clinic, AES 2023.

Single & Multiple Doses up to 70mg Were Generally Well Tolerated

MONARCH and ADMIRAL (n=74)

32% (24/74) of patients experienced a treatment-emergent adverse event (TEAE) related to study drug

Most common TEAEs related to study drug were CSF protein elevations, vomiting, and irritability

20% (15/74) had a treatment-emergent serious adverse event (TESAE). The TESAEs experienced by 14 of 15 patients were not considered related to study drug.

- 1 patient experienced Suspected Unexpected Serious Adverse Reactions (SUSARs) that the investigator attributed to STK-001. The patient completed the study.
- An amendment to the ADMIRAL study protocol allowed investigators to decide whether to administer 2 or 3 doses of STK-001 (70mg)

SWALLOWTAIL (n=44) A greater incidence of CSF protein elevations was observed compared to MONARCH & ADMIRAL

CSF protein values >50 mg/dL have been observed after dosing without any associated clinical manifestations

- 35% (26/74) of patients in MONARCH & ADMIRAL
- 64% (28/44) of patients in SWALLOWTAIL: 1 patient discontinued study treatment; 1 patient missed 1 dose

STK-001 Has Potential to Address the Genetic Cause of Dravet Syndrome

Summary of Key Clinical Data from Ongoing Studies

- ✓ Single and multiple doses of 10mg to 70mg were generally well-tolerated
- ✓ Patients treated with 2 or 3 doses of 70mg experience substantial and sustained reductions in convulsive seizures
- ✓ Reductions in seizure frequency were maintained with ongoing treatment at lower doses (30mg, 45mg)
- ✓ Improvements in assessments of cognition and behavior as measured by VABS-III* & BRIEF-P**

*Vineland Adaptive Behavior Scale (VABS-III), an assessment of adaptive behavior which refers to an individual's ability to undertake daily activities appropriate for their age group.
**Behavior Rating Inventory of Executive Function–Preschool Version, an assessment of pediatric executive function.

Autosomal Dominant Optic Atrophy (ADOA): A Severe, Progressive Optic Nerve Disorder

65-90%

of cases caused by mutations in one allele of the *OPA1* gene, most of which lead to a **HAPLOINSUFFICIENCY**

RESULTING in

50%

OPA1 protein expression and disease manifestation

1 out of 30,000

people are affected globally with a higher incidence of ~1 out of 10,000 in Denmark due to a founder effect

>400

Different *OPA1* mutations reported in ADOA patients

Up to 46%

of patients are registered legally blind

80%

of patients are symptomatic by age 10

~18,000

people affected in the U.S., Canada, Japan, Germany, France and the UK

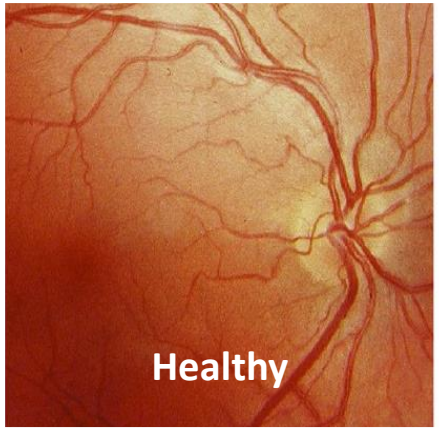
Sources: Yu-Wai-Man P et al. *Ophthalmology*, 2010; Yu-Wai-Man P, Chinnery PF. *Ophthalmology*, 2013; P. Amati-Bonneau P et al. *The International Journal of Biochemistry & Cell Biology*, 2009; Lenaers G, Hamel C, Delettre C, et al. *Orphanet J Rare Dis*, 2012; Chun BY and Rizzo JF III. *Curr Opin Ophthalmol*, 2016; Le Roux B, Lenaers G, Zanlonghi X et al. *Orphanet J Rare Dis*, 2019; "What is ADOA?" Autosomal Dominant Optic Atrophy Association. Accessed May 6, 2020, from https://www.adoaa.org/what-is-adoa;

No Approved Disease-Modifying Therapies for ADOA

Healthy Vision

Simulation of Optic Neuropathy

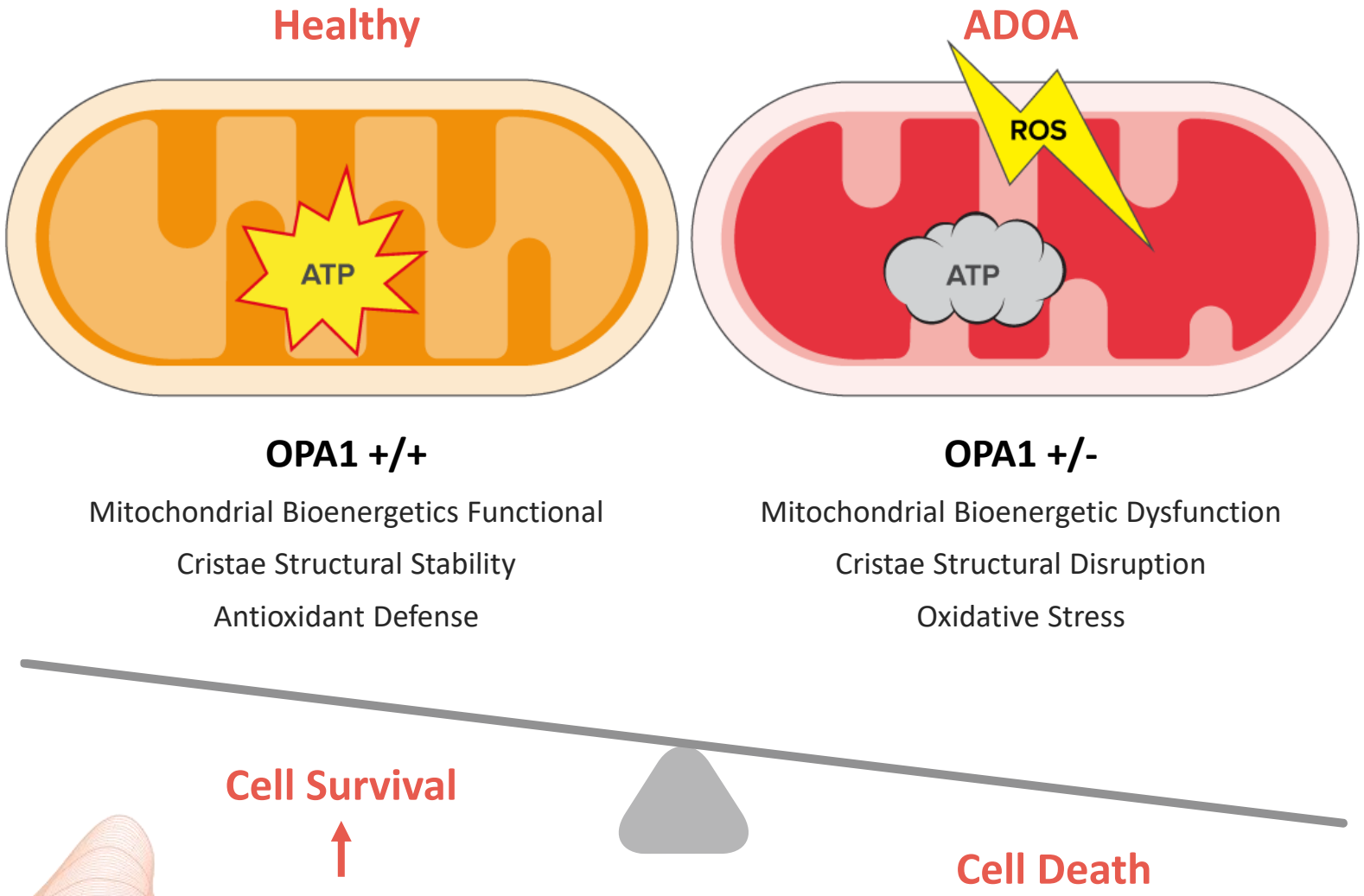
- Most common inherited optic nerve disorder
- Leads to central field defects and reduced color vision in both eyes
- Severity can vary; rate of vision loss difficult to predict
- Supportive services and low-vision aids are offered for patients



Sources: Yu-Wai-Man P et al. *Ophthalmology*, 2010; Yu-Wai-Man P, Chinnery PF. *Ophthalmology*, 2013;
Lenaers G, Hamel C, Delettre C, et al. *Orphanet J Rare Dis*, 2012; Chun BY and Rizzo JF III. *Curr Opin Ophthalmol,* 2016
Image of child sourced from ICR, Ophthalmology Center Barcelona. Accessed Jan. 8, 2021 from https://icrcat.com/en/eye-conditions/leber-hereditary-optic-neuropathy/
Credit: Lhon Eye Society Sweden. Image shown depicts Leber Hereditary Optic Neuropathy, which presents visual effects similar to ADOA.

OPA1 is Critical for Normal Mitochondrial Function and Cellular Metabolism

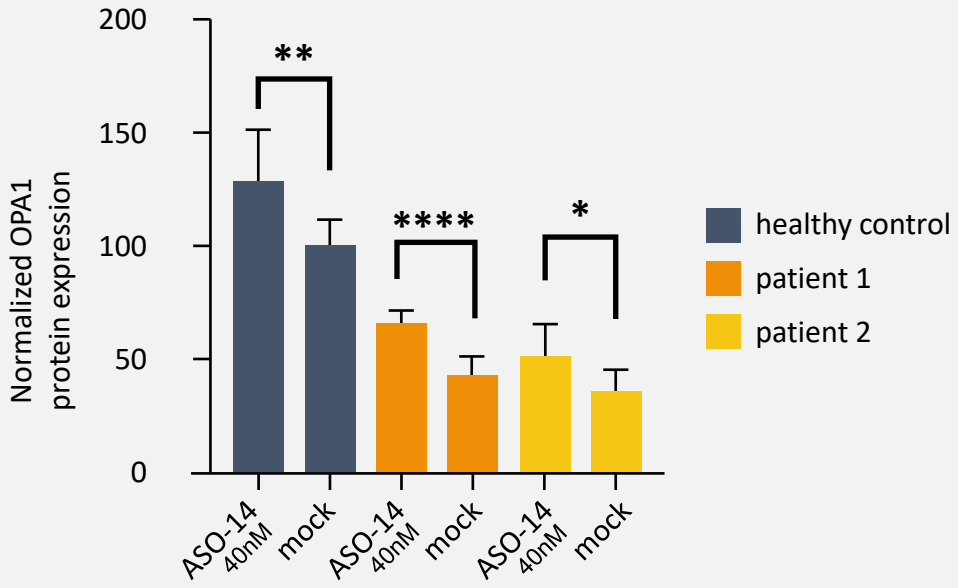
- Retinal ganglion cells have very high energy (ATP) requirements
- Impaired mitochondrial function leads to cell death
- OPA1 is critical for mitochondrial function and energy production



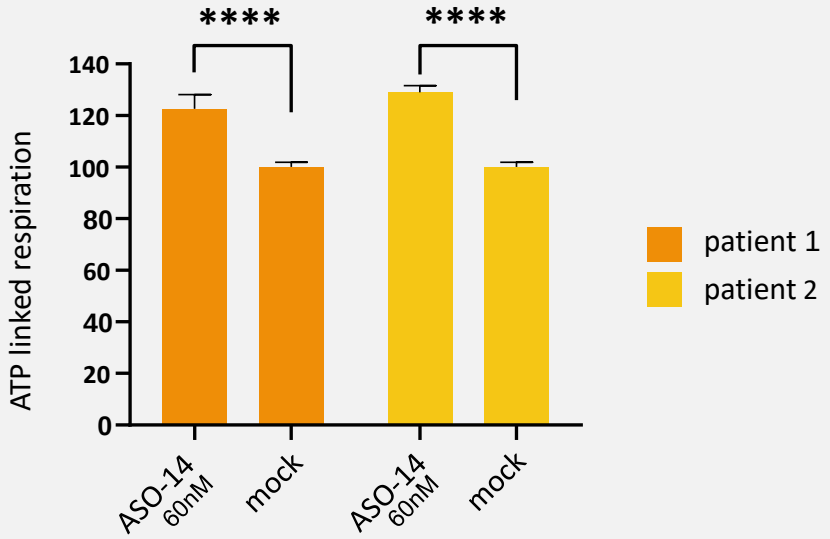
* ROS = Reactive Oxygen Species

TANGO ASO Increases OPA1 Protein and ATP Linked Mitochondrial Respiration in ADOA Patient Cells

ASO treatment increased OPA1 protein levels in OPA1 deficient ADOA patient cells



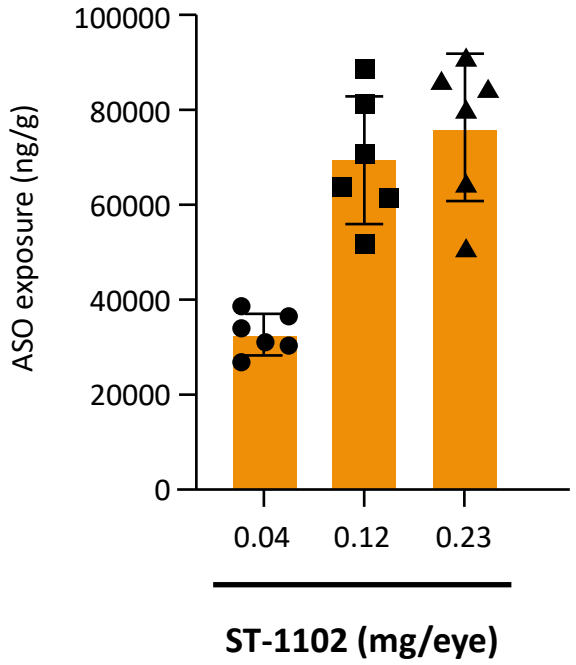
ASO treatment increased ATP linked respiration in OPA1 deficient ADOA patient cells



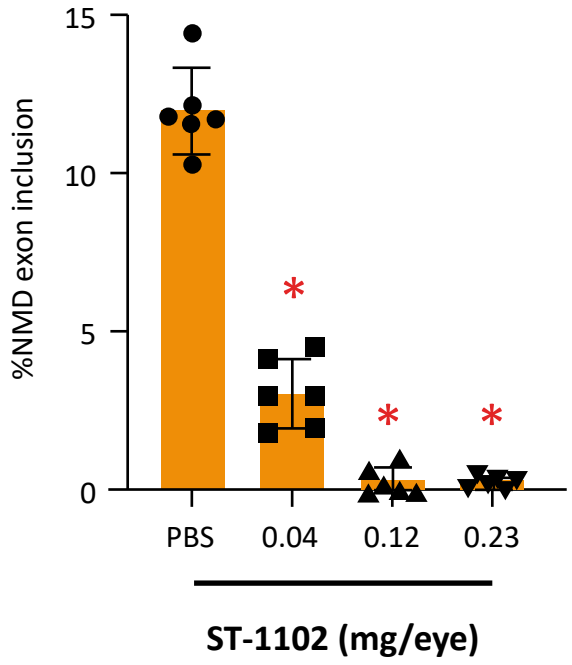
Source (left graph): Stoke data
Source (right graph): Venkatesh A, et al. Antisense oligonucleotide mediated increase in OPA1 improves mitochondrial function in fibroblasts derived from patients with autosomal dominant optic atrophy (ADOA). Presented at The Association for Research in Vision and Ophthalmology; May 1-7, 2021.

TANGO ASO Demonstrates Dose-Dependent OPA1 Protein Increases in Rabbit Retina

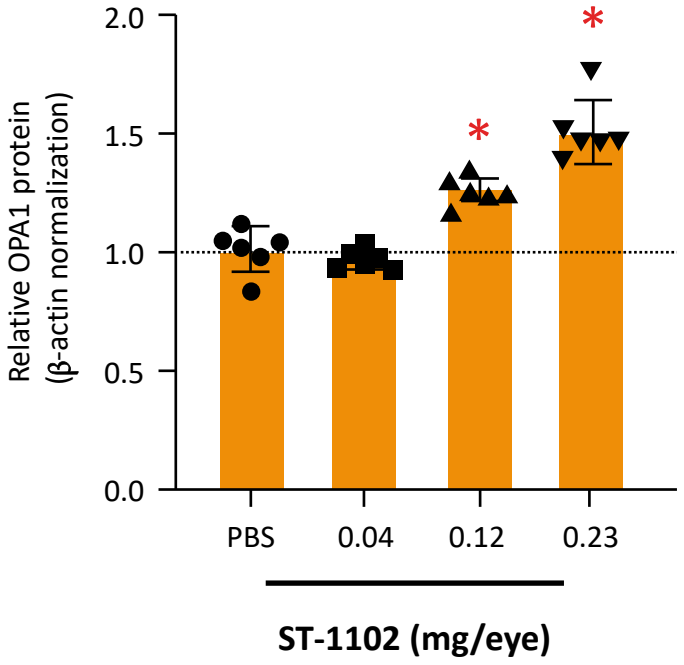
ASO exposure in retina Day 29



Target engagement Day 29



OPA1 protein Day 29

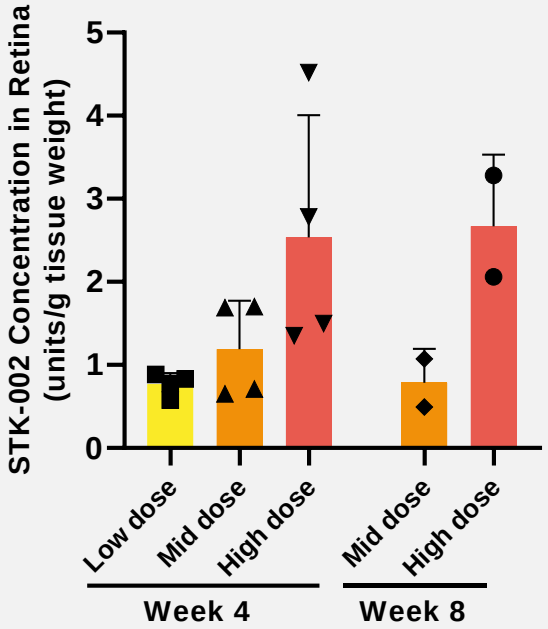


***P*<0.0005 by one-way ANOVA compared to PBS group**

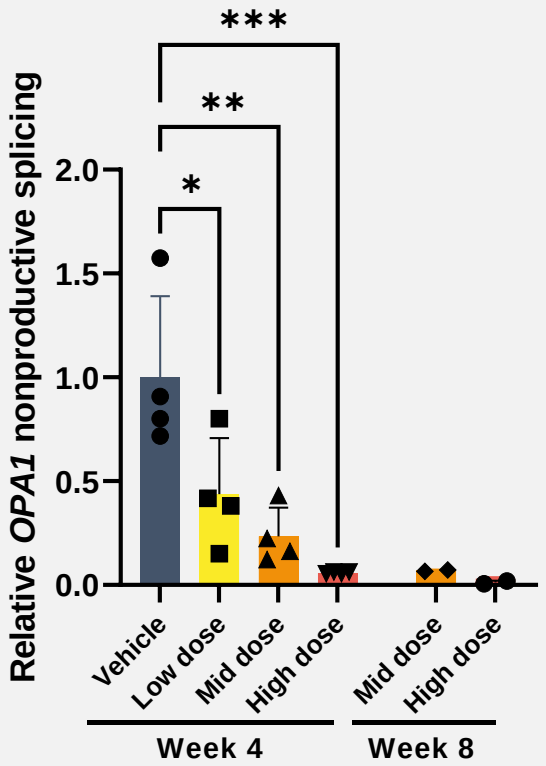
Source: Steven Gross, et al. A Prospective Natural History Study of Patients with Autosomal Dominant Optic Atrophy (ADOA) – FALCON. Presented at The North American Neuro-Ophthalmology Society; February 12-17, 2022; Austin, TX.

Dose-Related Target Engagement and OPA1 Protein Upregulation in Retinal Tissue of NHPs following IVT Administration of STK-002

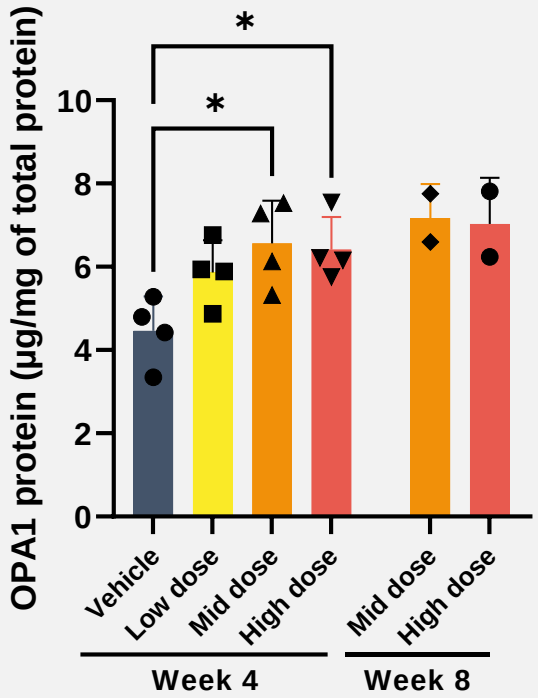
STK-002 exposure



Target engagement



OPA1 protein



NHP: Non-human primates
IVT: Intravitreal
Source: Venkatesh A, et al. STK-002, an Antisense Oligonucleotide (ASO) for the Treatment of Autosomal Dominant Optic Atrophy (ADOA), is Taken Up by Retinal Ganglion Cells (RGC) and Upregulates OPA-1 Protein Expression After Intravitreal Administration to Non-human Primates (NHPs). ASGCT; May 16-19, 2022.

Preclinical Findings Support Clinical Development of STK-002

Summary of Key Preclinical Data

- Increase OPA1 protein and ATP linked respiration in ADOA patient cells ✓
- Result in dose-dependent increases in OPA1 protein expression in rabbit retina ✓
- Were well tolerated for up to 29 days after intravitreal injection in rabbit ✓
- Dose-related increase in OPA1 protein expression was observed in NHP RGCs ✓

Phase 1 study (OSPREY) of STK-002 in the UK expected to start in 2024

OSPREY is a study of children and adults ages 6 to 55 who have an established diagnosis of ADOA and have evidence of a genetic mutation in the *OPA1* gene

Sources: Venkatesh A, et al. STK-002, an Antisense Oligonucleotide (ASO) for the Treatment of Autosomal Dominant Optic Atrophy (ADOA), is Taken Up by Retinal Ganglion Cells (RGC) and Upregulates OPA-1 Protein Expression After Intravitreal Administration to Non-human Primates (NHPs). ASGCT; May 16-19, 2022.

Our Pipeline of First-in-Class Disease Modifying Potential Medicines

PROGRAM	TARGET	DISCOVERY & PRECLINICAL	PHASE 1/2	PHASE 3	PARTNER
Central Nervous System					
Dravet Syndrome	*SCN1A*		STK-001		100% Stoke Global
SYNGAP1 Syndrome	*SYNGAP1*				Stoke : Acadia 50:50
Rett Syndrome	*MECP2*				Acadia Worldwide License
Undisclosed	*Undisclosed*				Acadia Worldwide License
Ophthalmology					
ADOA	*OPA1*	STK-002			100% Stoke Global

ADOA: Autosomal dominant optic atrophy

Rett Syndrome: A Severe, Debilitating Neurological Disorder

~33%

of cases caused by hypomorphic mutations of the *MECP2* gene[1]

RESULTING in

Partial loss of function of the MeCP2 protein

1 out of **10,000** to **15,000** females are born with Rett syndrome[2]

Period of rapid decline typically begins between

6 to **18**

months[4]

Symptoms include[3]:

- **Loss of purposeful hand use**
- **Involuntary hand movements such as handwringing**
- **Loss of speech**
- **Loss of mobility or gait disturbances**

60-80% of patients have **epilepsy**[4]

Note: All seizure types have been reported in Rett syndrome. Complex partial and generalized tonic-clonic are the most common
Sources: [1] RettBase (http://mecp2.chw.edu.au/); GnomAD (https://gnomad.broadinstitute.org); NOMAD; [2] National Institutes of Health – National Institute of Neurological Disorders and Stroke; [3] International Rett Syndrome Foundation; [4] Operta et al., Brain Behav 2019

SYNGAP1: A Severe Intellectual Disability / Developmental and Epileptic Encephalopathy (ID/DEE)

>80%

of cases caused by a **HAPLOINSUFFICIENCY** of the *SYNGAP1* gene[1]

RESULTING in

50%

SynGAP protein expression

1-2 **out of** **100,000** children are born with SYNGAP1-ID/DEE

1-2%

of all **intellectual disability** cases[2]

84%

of patients have **generalized epilepsy**[3]

100%

of patients have **developmental delay** or **intellectual disability**[3]

~50%

of patients have **autism and other behavioral abnormalities**[3]

Sources: [1] Parker et al., *American Journal of Medical Genetics*, 2015; Jimenez-Gomez et al., Journal of Neurodevelopmental Disorders, 2019; [2] SYNGAP1 Resource Guide, Second Edition; An Overview of SYNGAP1 Basic Biology and Clinical Description. Bridge the Gap SYNGAP (now SYNGAP1 Foundation); SynGAP Research Fund; [3] SYNGAP1-Related Intellectual Disability: https://www.ncbi.nlm.nih.gov/books/NBK537721/#_syngap1-id_Clinical_Characteristics_

2024 Summary of Priorities

Advance STK-001 for Dravet Syndrome to Pivotal

- Q1 Data Readout
- Pending data, request Phase 3 planning meetings with regulators

Advance STK-002 for ADOA

- Initiate Phase 1 study (OSPREY) in 2024

Develop & Expand Pipeline

- Execute on collaboration with Acadia to advance 3 neurodevelopmental programs including Rett syndrome and Syngap1 programs
- Expand TANGO ASOs as a first-in-class disease-modifying approach for additional genetic diseases

Current Liquidity Anticipated to Fund Operations to the End of 2025

$214.7M in Cash, Cash Equivalents, and Marketable Securities as of 9/30/23

ADOA: Autosomal dominant optic atrophy

STOKE THERAPEUTICS