Chimerix Corporate Presentation

August 3, 2023

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Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 that are subject to risks and uncertainties that could cause actual results to differ materially from those projected. Forward-looking statements include those relating to, among other things, the probability of success of the Phase 3 ACTION study, the potential filing and approval of an NDA for ONC201 and subsequent commercial opportunity, the implications of the monotherapy radiographic partial response observed during ONC206 dose escalation; the ability to reproduce clinical and pre-clinical findings, and projections regarding funding and timing of future data readouts. Among the factors and risks that could cause actual results to differ materially from those indicated in the forward-looking statements are risks related to the timing, completion and outcome of the Phase 3 ACTION study of ONC201; risks associated with repeating positive results obtained in prior preclinical or clinical studies in future studies; risks related to the clinical development of ONC206; and additional risks set forth in the Company's filings with the Securities and Exchange Commission. These forward-looking statements represent the Company's judgment as of the date of this release. The Company disclaims, however, any intent or obligation to update these forward-looking statements.

Investment highlights

High probability of success for Phase 3 ACTION study of ONC201

- Phase 2 study designed to isolate single agent activity in difficult treatment setting
- Durable responses associated with OS and other forms of clinical benefit
- Numerous independent and natural disease history studies support potential survival advantage
- Genetically selected patient population limits patient heterogeneity

Low barriers to commercial potential for ONC201

- Terminal disease with no effective therapeutic options
- High awareness for program within neuro-oncology community
- U.S. patent exclusivity through at least 2037
- Global revenue potential of ~$750m in first indication alone

Corporate capability and financial flexibility

- Leadership team successfully executed large scale studies and regulatory approvals
- Strong balance sheet fully funds ACTION study and potential ONC206 catalysts
- Opportunity for continued non-dilutive TEMBEXA milestones and royalties adds flexibility
- Track record of objectivity in creating paths to capture value

Deep pipeline across all development stages

Program	Preclinical	Phase 1	Phase 2	Registrational	FDA review	Collaborators
ONC201 (dordaviprone)						
H3 K27M-mutant glioma (orphan drug,[1] fast track[2] and rare pediatric disease designations[3])						
IITs- signal finding, multiple oncology indications/combinations						
ONC206						National Institutes of Health
CNS[4] tumors						PACIFIC PEDIATRIC NEURO-ONCOLOGY CONSORTIUM
ONC212						THE UNIVERSITY OF TEXAS MD Anderson Cancer Center
IND-enabling studies						BROWN
CMX521						
SARS-CoV-2						READDI[5]
TEMBEXA® transacted with Emergent BioSolutions						
Smallpox (orphan drug designation)				**APPROVED** June 4, 2021		BARDA, EMERGENT®

1 Malignant glioma
2 Adult recurrent H3 K27M-mutant high-grade glioma
3 H3 K27M-mutant glioma
4. Central Nervous System
5. Rapidly Emerging Antiviral Drug Development Initiative

ONC201 (dordaviprone)
Phase 2 Efficacy Analysis

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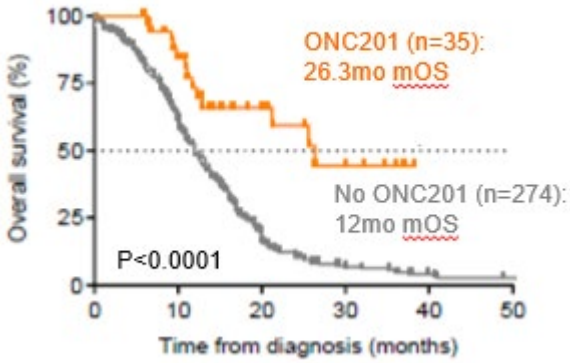
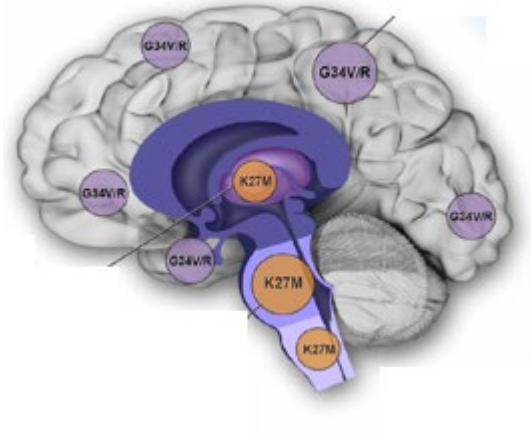
H3 K27M-mutant diffuse glioma: high unmet need

- H3 K27M mutation is predominantly found among diffuse midline gliomas (DMGs) in young adults and children
- Frontline radiotherapy remains standard of care with transient benefit; resection often not feasible
- DMGs harboring the H3 K27M mutation are WHO Grade IV; historically invariably lethal
- Studies consistently indicate longer OS of ONC201-treated glioma patients relative to diverse external controls

Frontline H3 K27M DMG External analysis reported at SNO 2022[2]



Histone H3 Mutations in CNS Tumors[1]



Company Sponsored Studies

	Natural Disease History: Recurrent H3 K27M and/or DMG[3] (n=43)	ONC201 Phase 2: Recurrent H3 K27M DMG (n=50)
Median OS,mo (95% CI)	5.1 (3.9-7.7)	13.7 (8-20.3)
OS @ 12mo (95% CI)	23.6% (11.7-37.9)	57% (41-70)
OS @ 24mo (95% CI)	11.1% (3.3-24.2)	35% (21-49)

[1] Lulla RR et al. Sci Adv. 2016;2(3):e1501354
[2] Sunjong Ji, B.S. et al, "Clinical efficacy and predictive biomarkers of ONC201 in H3 K27M-mutant diffuse midline glioma", Society of Neuro-oncology 2022
[3] The median OS was 5.1 months for the subset of patients with H3 K27M-mutant diffuse glioma excluding DIPG, CSF dissemination, spinal or leptomeningeal disease (N=12), OS at 12 mos was 25.0%, OS at 24 mos was 16.7%

Phase 2 efficacy for ONC201 in recurrent H3 K27M DMG

- ONC201 monotherapy exhibited durable, clinically meaningful efficacy in recurrent H3 K27M-mutant DMG
  - Overall Response Rate (ORR) of 30% (95% CI: 18 - 45%) by RANO HGG and/or LGG dual reader BICR
  - RANO-HGG criteria assessed by dual reader BICR
    - ORR 20% (95% CI: 10 – 34%)
    - Median Duration of Response (DOR) 11.2 months (95% CI: 3.8 – not reached)
    - Median time to response 8.3 months (range 1.9 – 15.9)
    - Disease control rate 40% (95% CI: 26 – 55%)
    - PFS at 6 months 35% (95% CI: 21 – 49%); PFS at 12 months 30% (95% CI: 17 – 44%)
  - RANO-LGG criteria assessed by dual reader BICR
    - ORR 26% (95% CI: 15 – 40%)
  - Overall survival
    - 12 months: 57% (95% CI:41 – 70%)
    - 24 months: 35% (95% CI: 21 – 49%)
- Improvements observed in performance status and reduction in corticosteroid use
- All SAEs considered not related to ONC201 by sponsor

FDA-aligned criteria for Phase 2 efficacy analysis to isolate ONC201 single agent activity

Objective

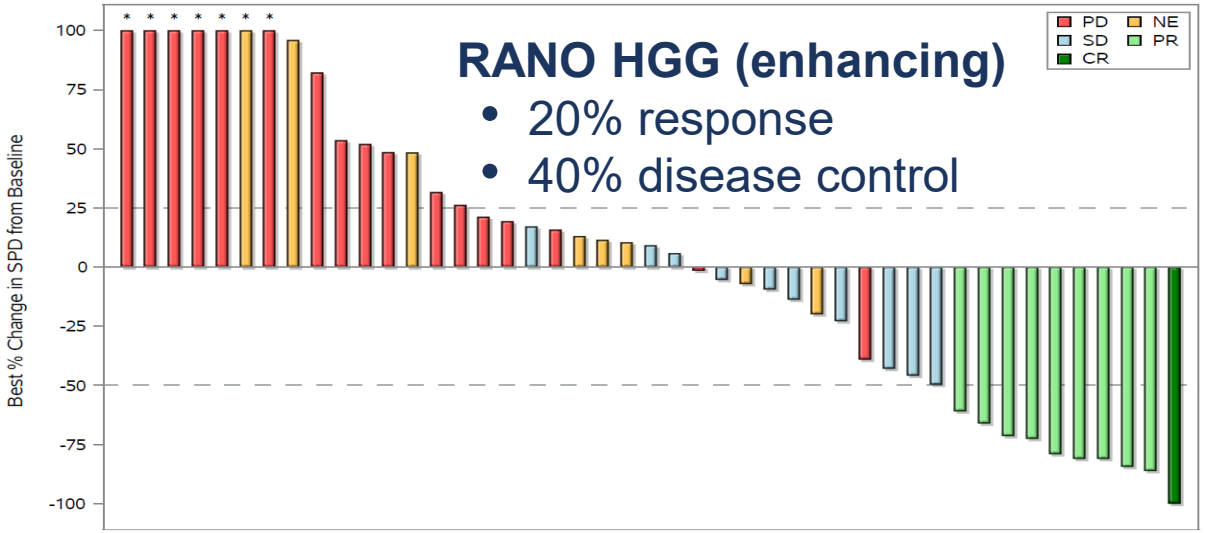
- To evaluate monotherapy efficacy of ONC201 in recurrent H3 K27M-mutant diffuse midline glioma

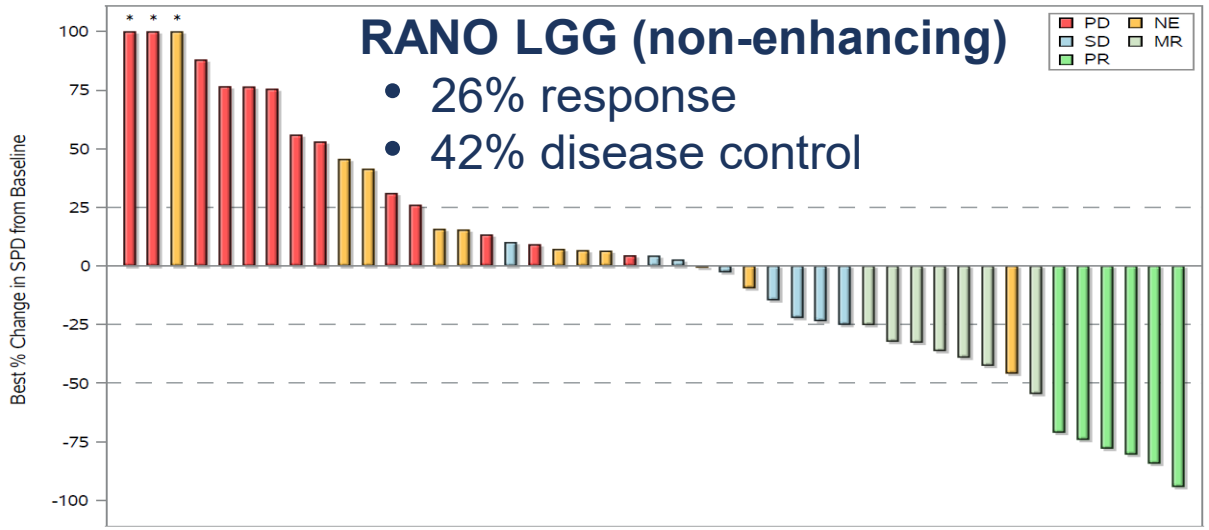
Eligibility

- Age ≥2yo and received ONC201 under studies ONC006, ONC013, ONC014, ONC016, or ONC018
- Diffuse glioma with a known H3 K27M mutation and involvement of a midline structure of the brain
- Progressive and measurable disease on contrast-enhanced brain MRI by RANO-High Grade Glioma (HGG) criteria
- Prior therapy with at least radiation
- Washouts prior to first ONC201 dose:
  - Radiation: 90 days
  - Temozolomide: 23 days / Antibodies (e.g., bevacizumab): 42 days / Other anticancer therapies: 28 days
- Baseline Performance Status ≥60
- Corticosteroids stable or decreasing for at least 3 days prior to baseline scan
- Excluded: DIPG, primary spinal tumors, atypical and non-astrocytic hist., leptomeningeal spread, CSF dissemination

ONC201 waterfall plot – 30% RANO HGG / LGG response

ONC201 Ph 2 Efficacy Analysis by BICR in Recurrent H3 K27M-mutant Diffuse Midline Glioma





- Strict selection criteria to ensure responses attributable to single agent treatment
- Responses require both imaging and clinical criteria
- Dual reader blinded independent central review (BICR)
- Growing consensus that assessment of enhancing and non-enhancing disease (RANO-HGG and RANO-LGG criteria) is needed for diffuse midline glioma

Change > 100%, PR=partial response, MR=minor response, SD=stable disease, NE=not evaluable, PD=progressive disease

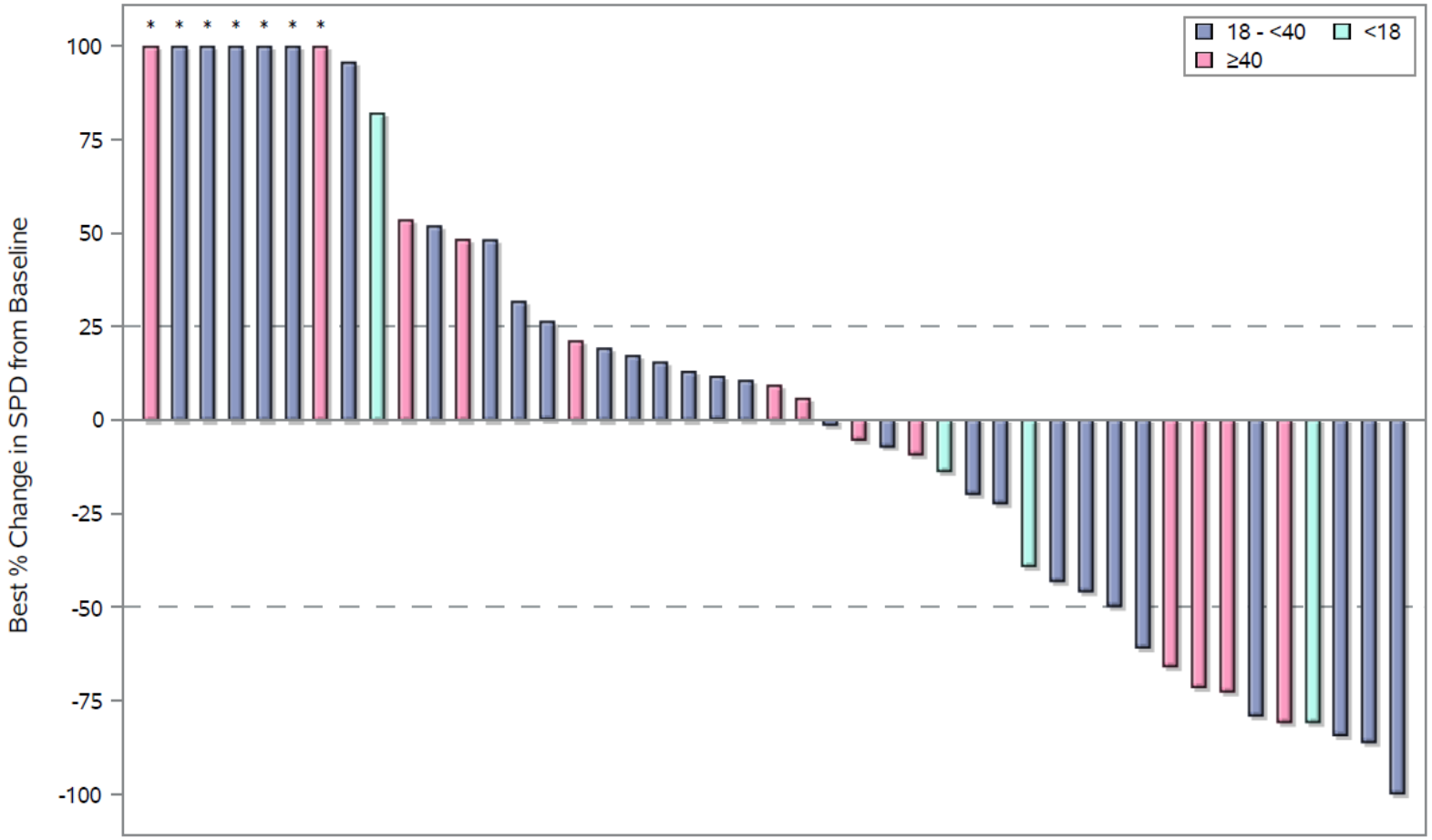
RANO-HGG responses observed across age groups

Responses by age group:

- <18 years: 1/4 (25%)
- 18-40 years: 5/32 (16%)
- ≥40 years: 4/14 (29%)

RANO-HGG response of 8-year-old subject suggests activity in this population

ONC201 Ph 2 Efficacy Analysis by BICR in Recurrent H3 K27M-mutant Diffuse Midline Glioma



* Change > 100%, SPD=sum of products of perpendicular diameters (target enhancing lesions per BICR)
Only patients with measurable target enhancing lesions at both baseline and post-baseline are included.

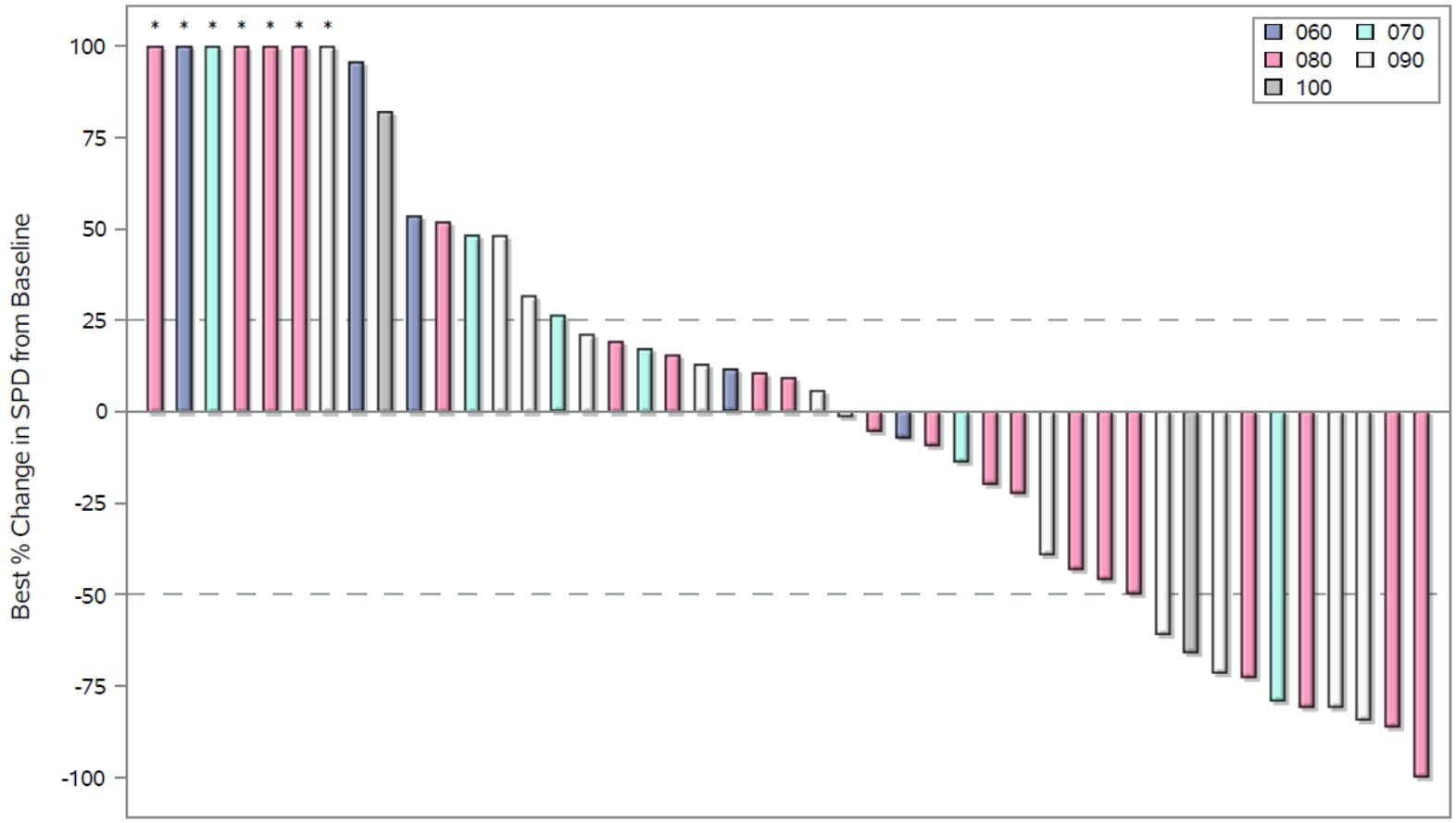
RANO HGG response correlation to performance status (PS) supports early-line trial

Predictably, patients with higher PS were more likely to respond to treatment

- 100: 1/2 (50%)
- 90: 4/14 (29%)
- 80: 4/20 (20%)
- 70: 1/7 (14%)
- 60: 0/7 (0%)

Supports hypothesis that treating earlier in disease course may enhance efficacy

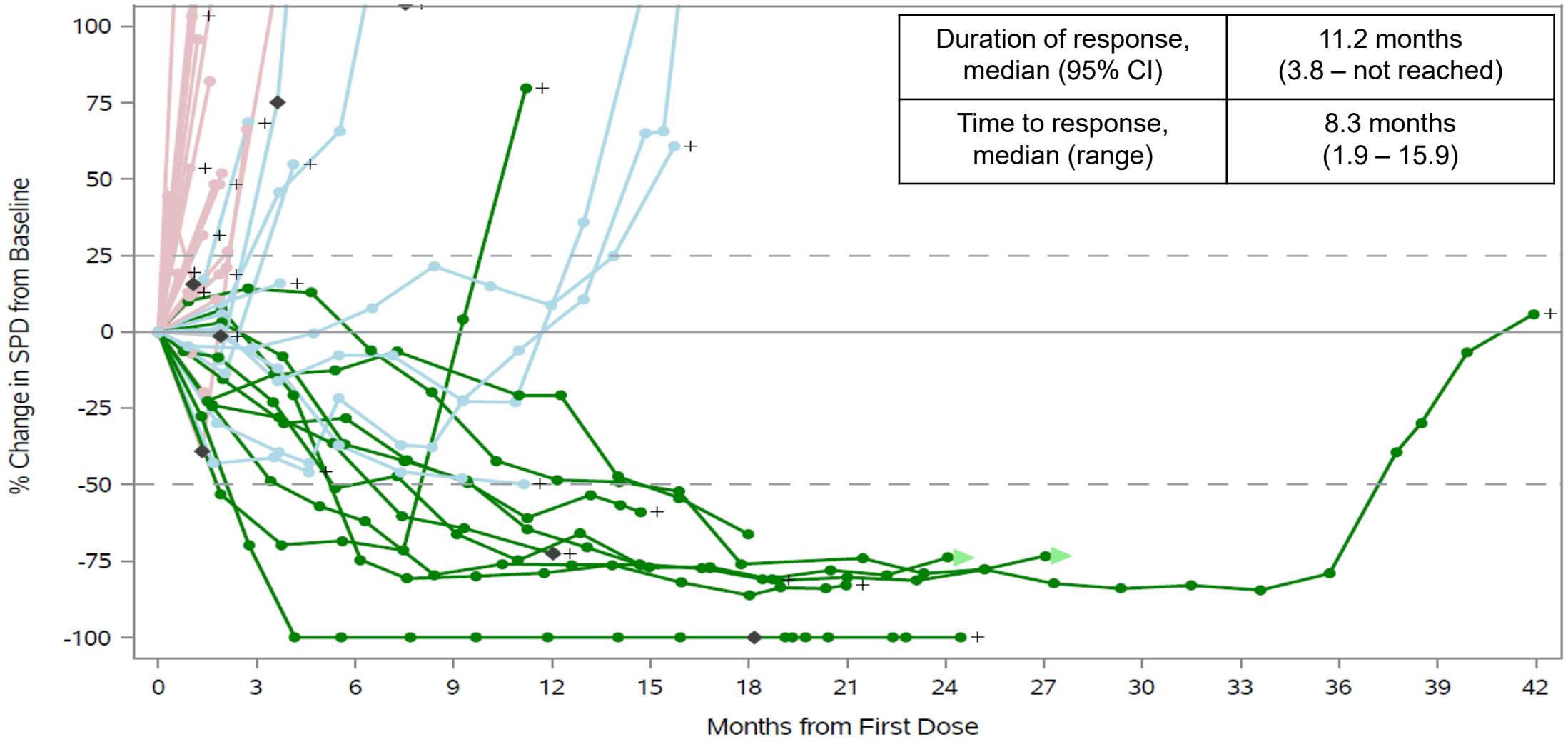
ONC201 Ph 2 Efficacy Analysis by BICR in Recurrent H3 K27M-mutant Diffuse Midline Glioma



* Change > 100%, SPD=sum of products of perpendicular diameters (target enhancing lesions per BICR)
Only patients with measurable target enhancing lesions at both baseline and post-baseline are included.

Clinically meaningful and durable RANO-HGG responses

ONC201 Phase 2 Efficacy Analysis by BICR in Recurrent H3 K27M-mutant Diffuse Midline Glioma



Duration of response, median (95% CI)	11.2 months (3.8 – not reached)
Time to response, median (range)	8.3 months (1.9 – 15.9)

SPD=sum of products of perpendicular diameters (target enhancing lesions per BICR)
Only patients with measurable target enhancing lesions by BICR at baseline and with post-baseline evaluations are included.
Three patients did not have on-treatment monotherapy MRIs available for BICR; one patient censored prior to first on-treatment MRI ; one patient did not have measurable target lesion.

ONC201 safety

Healthy Adult Dose Escalation Study[1] Incidence of ONC201-Related Adverse Events (AE)

	125 mg N=33	375 mg N=15	625 mg N=45
Any treatment-related AE	36.0%	20.0%	53.0%
Grade 1	36.0%	20.0%	53.0%
Grade 2	0	0	0
Grade 3-5	0	0	0

- In addition to healthy adult dose escalation study above, clinical pharmacology studies included: food-effect, safety pharmacology, special populations, and drug-drug interaction studies
- Treatment-related AEs were generally Grade 1 and transient across the clinical pharmacology program.
- The most commonly reported treatment-related events were mild dizziness, headache and nausea.

Treatment-related Adverse Events in ≥ 3% Glioma Patients

Treatment-related Adverse Events, Integrated Safety Data Set, (N=211 glioma patients) [2]	Related TEAEs	
	All grades	Grade > 3
Any Treatment-related AE	55.5%	11.8%
Fatigue	21.8%	2.8%
Nausea	20.4%	0
Vomiting	14.2%	0.5%
Headache	8.5%	0.5%
Lymphocyte count decreased	6.6%	0.5%
Decreased appetite	5.7%	0
White blood cell count decreased	4.7%	0.5%
ALT increased	4.3%	0.5%
Hypophosphataemia	4.3%	0
Neutrophil count decreased	3.8%	0.5%
Anaemia	3.3%	0
Diarrhea	3.3%	0

1. Based on available data as 25Apr2023 for ONC201-101
2. Reported in ONC201 Investigator Brochure

RANO responses correspond with survival & clinical benefit

- No patients who experienced a RANO-HGG response had a reported death at 24 months[2]
- RANO response strongly associated with reduction in steroid use and improvement in performance status

ONC201 Ph 2 Efficacy Analysis by BICR in Recurrent H3 K27M-mutant DMG

	All patients	RANO HGG Responders	RANO HGG and/or LGG Responders
N	50	10	15
PFS at 12 months (number of patients censored)	30%[1]	90% (0)	67% (2)
OS at 24 months (number of patients censored)[2]	35%[1]	80% (2)	53% (5)
Corticosteroids response[3] (number of patients evaluable)	47% (15)	100% (4)	100% (5)
Performance status response[4] (number of patients evaluable)	21% (34)	60% (5)	67% (9)

1. Kaplan-Meier median Progression-Free Survival or Overall Survival
2. Censored patients include those who are lost to follow-up or have withdrawn prior to the time point, as well as those who have not yet reached the time point. These patients are counted in the denominator but not counted as survivors (i.e., imputed as failure)
3. Corticosteroid response: ≥50% reduction in average daily corticosteroid dose compared to baseline with stable or improved KPS/LPS. Must be confirmed at next analysis timepoint. Corticosteroids converted into a dexamethasone equivalent dose. Baseline ≥4mg dexamethasone at baseline were evaluable.
4. Performance status response: increase in KPS/LPS compared to baseline with stable or reduced corticosteroid use. Must be confirmed at next analysis timepoint. Baseline KPS/LPS ≤80 were evaluable.

Strong rationale for phase 3 success relative to recent GBM trials

Experimental Agent	Ph2 Design	Biomarker	Response criteria	Confounded by anti-angiogenic pseudo response	ORR	Duration of response	6 month PFS	Approved?
ONC201– Ph2 rDMG	Single agent	H3 K27M	RANO BICR	No	20-30%	11.2	35%	To be determined
Temodal® temozolomide	Single agent	-	Levin	No	5%	?	21%	Yes (OS)
AVASTIN® bevacizumab	Various	-	Various	Yes	20-70%	4-6	18-50%	Yes (AA per ORR, PFS)
Cediranib	Single agent	-	MacDonald	Yes	27%	?	26%	No
Rindopepimut	Combo + Avastin	EGFRv3	RANO	Yes	30%	7.8	28%	No
Depatuxizumab mafodotin	Single agent	-	RANO	No	7%	6.7	29%	No
Enzastaurin	Combo + Avastin	-	RANO	Yes	22%	?	21%	No

Positive Neutral Negative Characteristics

WKA Yung, et al, British Journal Cancer, Aug 8, 2000; Teri Kreisl, et all, Journal Clinical Onocoloy,2009, Feb 10;27(5);740-5; Tracy Batchelor, et. all, Journal of Clinical Oncology, vol28, issue 17, Jun 10, 2010; David Reardon, et al, Clin Cancer Research Apr 2020; 26(7)1586-1594; Martin van den Bent, et al, Cancer Chemo & Pharma, 26 Oct 2017 80, 1209-1217; Yazmin Odia, et al, Journal Neuro-Oncology 127, 127-125 (2016)

ONC201 Phase 3 ACTION Study Summary

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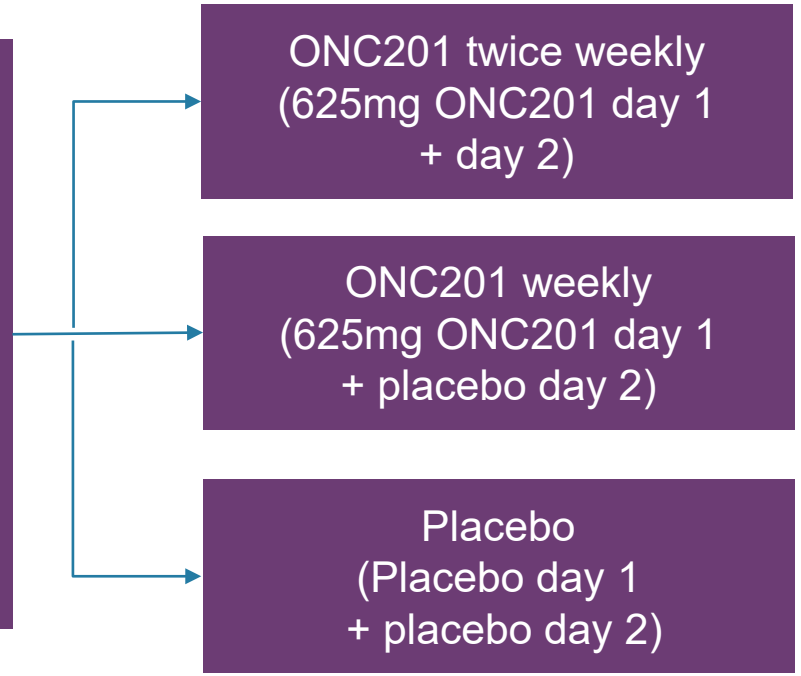
Pivotal Phase 3 ACTION trial design

Now enrolling, a randomized, double-blind, placebo-controlled, multicenter international study in 450 newly diagnosed diffuse glioma patients whose tumor harbors an H3 K27M-mutation.

Key Patient Inclusion

- H3 K27M-mutant diffuse glioma[1]
- Radiation therapy recently completed
- KPS ≥ 70 at time of randomization
- Stable steroid dose
- No prior bevacizumab
- No temozolomide within three weeks

Treatment



Endpoints

- Primary: Overall Survival
- PFS (alpha-allocated)
- Secondary: steroid response, performance status, QoL, neurologic function

1. Excludes DIPG and spinal tumors

Multiple unique aspects to ONC201 data support translation to phase 3 success

Isolated, durable single agent activity

- Responses not confounded by combination treatments
- Responses were gradual, durable, and multi-focal
- Responses observed via most stringent criteria in blinded assessment

Consistency across multiple endpoints

- Responses highly associated with other forms of clinical benefit
- PFS and OS favorable to historical benchmarks
- Multiple separate analyses suggest longer survival of patients who received ONC201

Enhanced activity not required, but likely

- Earlier setting associated with higher response rate (performance status, tumor volume)
- Addition of higher-dose study arm
- Biomarker selection supports patient homogeneity

Design provides multiple paths for success

Interim data expected in early 2025 and final data in 2026

Independent comparisons for each ONC201 arm versus control will be made at each timepoint.

First OS[1] Interim

- ~164 events
- Success at HR[3]=0.52

PFS by RANO HGG[2]

- ~286 events
- Success at HR=0.68

Second OS Interim

- ~246 events
- Success at HR=0.64

Final OS

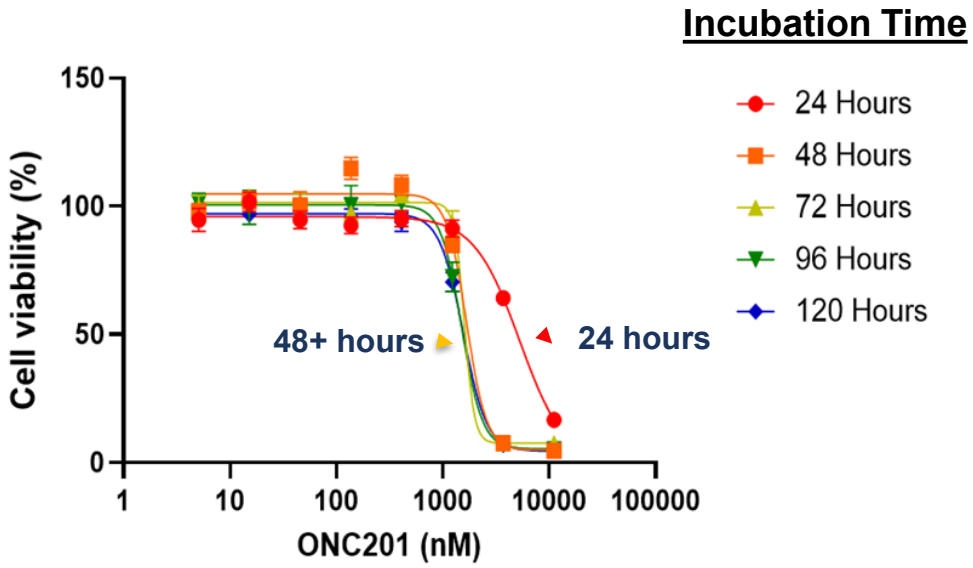
- ~327 events
- Success at HR=0.73

Powering assumptions 0.65 expected HR for OS and 0.60 expected HR for PFS

1. Overall Survival (OS)
2. Progression-free survival (PFS). PFS may provide valuable data for regulatory discussions.
3. Hazard Ratio

Potential to increase ONC201 efficacy with dose schedule

- Once per week ONC201 dosing effective as monotherapy in Phase 2 studies
- Twice per week dosing on two consecutive days expected to increase duration of therapeutic exposure
  - Increased exposure time can increase glioma sensitivity to ONC201 in vitro
  - Generally well tolerated in Phase 1 without dose limiting toxicity or AEs leading to dose modification
- Phase 3 ACTION study will evaluate once per week and twice per week dosing schedules at 625mg (or body weight equivalent)



ONC201 Market Opportunity Assessment

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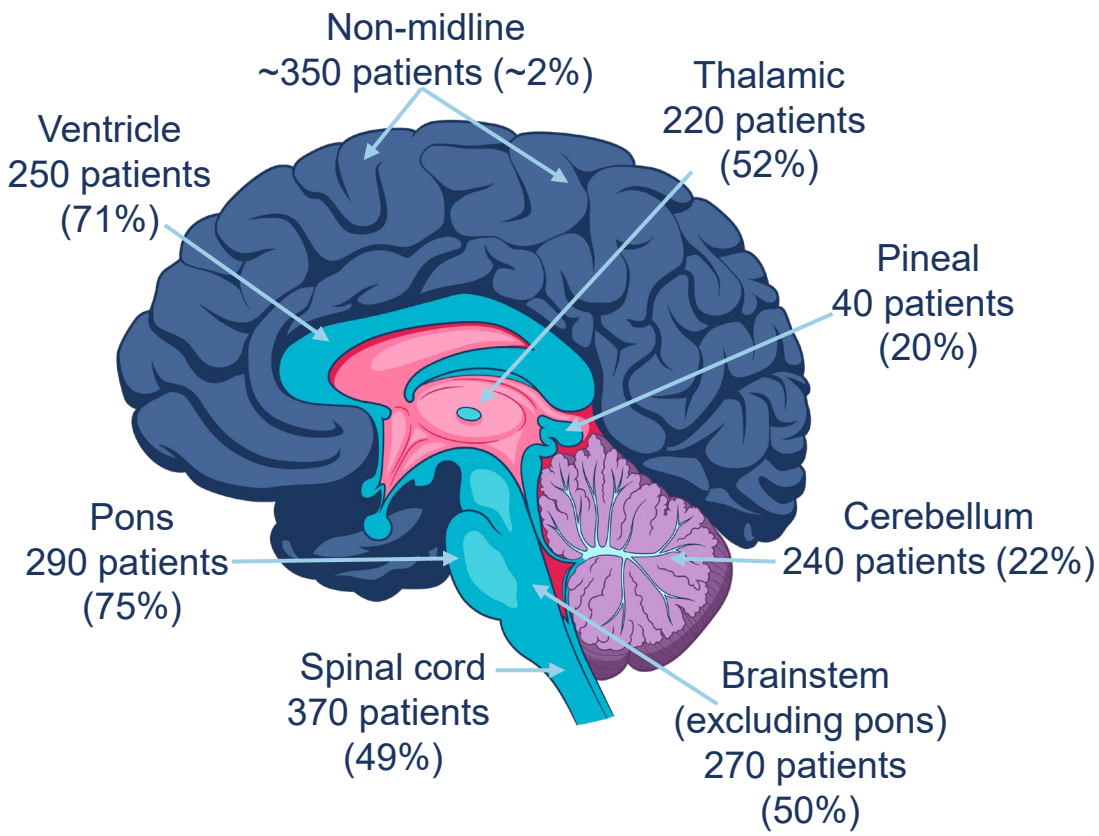
Approximately 21,000 gliomas reported in the U.S. each year, affecting all locations in the brain[1]

- **~40%** of 4,000+ ***midline gliomas*** are expected to harbor the H3 K27M mutation[2]
- **~2%** of 17,000+ ***non-midline gliomas*** are expected to harbor the H3 K27M mutation[2]
- Each year it is estimated that **~2,000** patients are affected by H3 K27M-mutant glioma in the U.S; **~5,000** patients in the top seven global markets

Estimated # of U.S. H3 K27M+ Patients by Tumor Location (rate of positivity)[2]



(1) Ostrom QT, et al. *Neuro Oncol*. 2022;24(Suppl 5):v1-v95; **(2)** Patient numbers and percentages are estimates (weighted avg per sample size) derived from a review of the literature from (2012-2023): (Aihara K, et al. *Neuro Oncol*. 2014;16(1):140-6; Feng J, et al. *Hum Pathol*. 2015;46(11):1626-32; Solomon DA, et al. *Brain Pathol*. 2016;26(5):569-80;Ryall S, et al. *Acta Neuropathol Commun*. 2016;4(1):93; Aboian MS, et al. *AJNR Am J Neuroradiol*. 2017;38(4):795-800; Wang L, et al. *Hum Pathol*. 2018;78:89-96; Castel D, et al. *Acta Neuropathol Commun*. 2018;6(1):117; Karremann M, et al. *Neuro Oncol*. 2018;20(1):123-131; Aboian MS, et al. *AJNR Am J Neuroradiol*. 2019;40(11):1804-1810; Dorfer C, et al. *Acta Neurochir (Wien)*. 2021;163(7):2025-2035; Sievers P, et al. *Neuro Oncol*. 2021;23(1):34-43; Mackay A, et al. *Cancer Cell*. 2017;32(4):520-537 e5; Huang T, et al. *Oncotarget*. 2018;9(98):37112-37124; Schreck KC, et al. *J Neurooncol*. 2019;143(1):87-93; Chiba K, et al. *World Neurosurg*. 2020;134:e530-e539; Mukasa A, et al. *Neuro Oncol*. 2014;16(Suppl 3):iii9-iii10; Castel D, et al. *Acta Neuropathol*. 2015;130(6):815-27; Khuong-Quang DA, et al. *Acta Neuropathol*. 2012;124(3):439-47; Roux A, et al. *Neuro Oncol*. 2020;22(8):1190-1202; Giagnacovo M, et al. *Childs Nerv Syst*. 2020;36(4):697-704; Wu G, et al. *Nat Genet*. 2014;46(5):444-450; Wu G, et al. *Nat Genet*. 2012;44(3):251-3; Taylor KR, et al. *Nat Genet*. 2014;46(5):457-461; Saratsis AM, et al. *Acta Neuropathol*. 2014;127(6):881-95; Erker C, et al. *Neuro Oncol*. 2022;24(1):141-152; Buczkowicz P, et al. *Acta Neuropathol*. 2014;128(4):573-81; Daoud EV, et al. *J Neuropathol Exp Neurol*. 2018;77(4):302-311; Chai RC, et al. *Acta Neuropathol Commun*. 2020;8(1):40; Yi S, et al. *Neurosurgery*. 2019;84(5):1072-1081; Gessi M, et al. *Acta Neuropathol*. 2015;130(3):435-7; Alvi MA, et al. *Mod Pathol*. 2019;32(9):1236-1243; Crotty EE, et al. *J Neurooncol*. 2020;148(3):607-617; Dono A, et al. *J Clin Neurosci*. 2020;82(Pt A):1-8; Akinduro OO, et al. *J Neurosurg Spine*. 2021;35(6):834-843; Nakata S, et al. *Brain Tumor Pathol*. 2017;34(3):113-119; Nomura M, et al. *Acta Neuropathol*. 2017;134(6):941-956; Eschbacher KL, et al. *Am J Surg Pathol*. 2021;45(8):1082-1090; D'Amico RS, et al. *J Neurooncol*. 2018;140(1):63-73; Korshunov A, et al. *Acta Neuropathol*. 2015;129(5):669-78; Aibaidula A, et al. *Neuro Oncol*. 2017;19(10):1327-1337.)

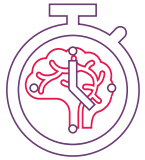
H3 K27M-mutant glioma: rapid ramp to peak revenue expected

- No approved therapies for H3 K27M mutant glioma, ONC201 is the leading program targeting this mutation globally
- Potential market opportunity ~$750 million
- Approximately 5,000 patients in top seven markets
- Ultra-orphan indication drug pricing
- H3 K27M mutations most often in children / young adults (little exposure to Medicare)
- Low barriers to adoption
  - No effective alternative therapies
  - High unaided awareness among neuro-oncologists
  - Mutation routinely identified by existing diagnostics
  - Longer-term, potentially combinable with other glioma therapies
- Patent protection for lead indication into 2037 - potential U.S. patent term extension (up to five years)

Regulatory designations

US - Orphan Drug Designation (ODD) (treatment of glioblastoma and treatment of malignant glioma)

EU - ODD for treatment of glioma



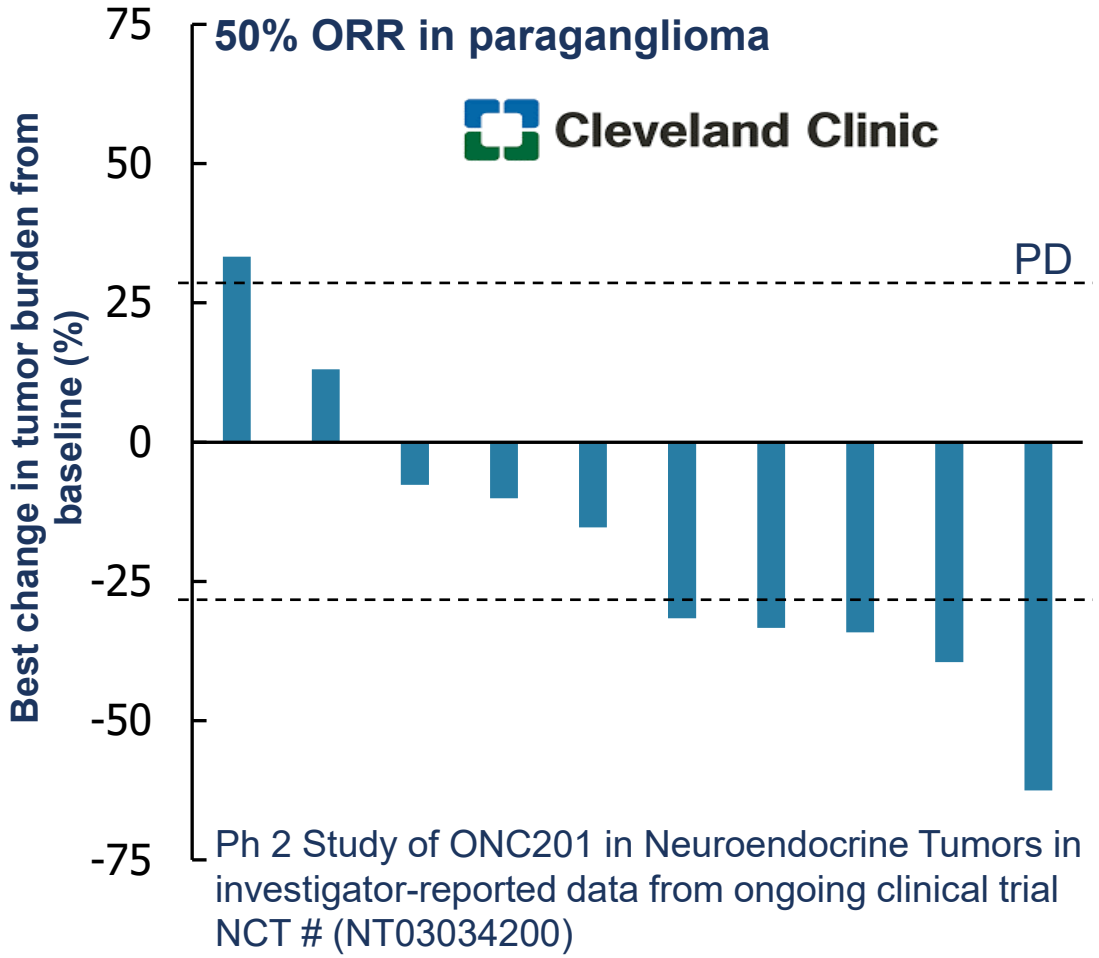
Fast Track Designation (FTD) for adult recurrent H3 K27M-mutant high-grade glioma

Rare Pediatric Disease Designation for treatment of H3 K27M mutant glioma

- Potential to receive rare pediatric voucher[1]

1. Subject to continuation of Rare Pediatric Disease Voucher Program and proceeds from voucher will be split 50/50 with legacy Oncoceutics shareholders

ONC201 interim efficacy results in dopamine-secreting tumors outside the brain



- Single agent responses in Ph 2 neuroendocrine trial of ONC201 observed in subset (paraganglioma)
- Paraganglioma are adrenal-related tumors with elevated DRD2 expression
- Five patients have been treated > 1 year
- Fewer short-term and potential long-term toxicities than other paraganglioma therapies

1. ASCO oral presentation June 2021

ONC201 Mechanism of Action

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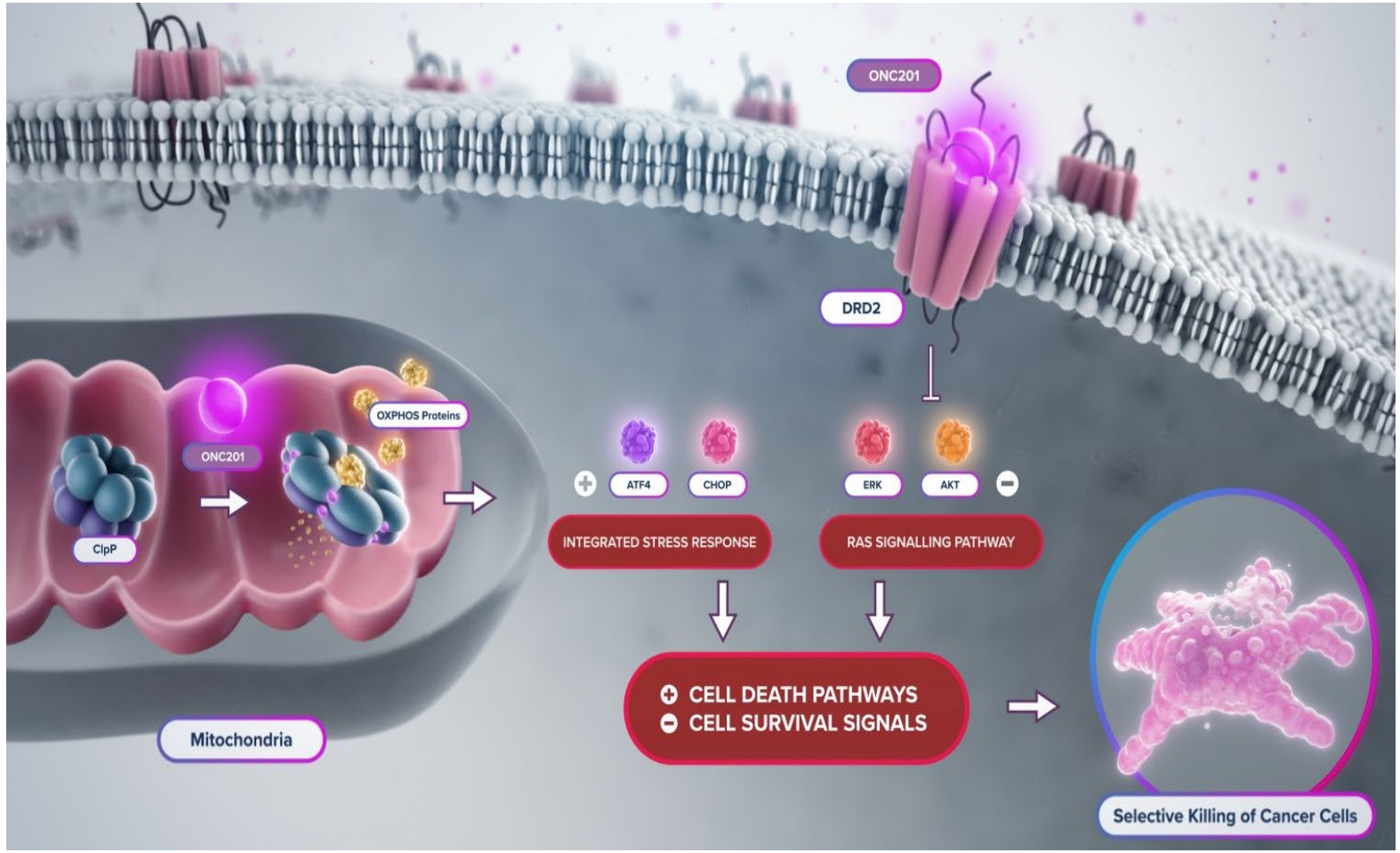
ONC201 directly engages DRD2 and ClpP

ONC201 upregulates integrated stress response, inactivates Akt/ERK, and selectively induces tumor cell death

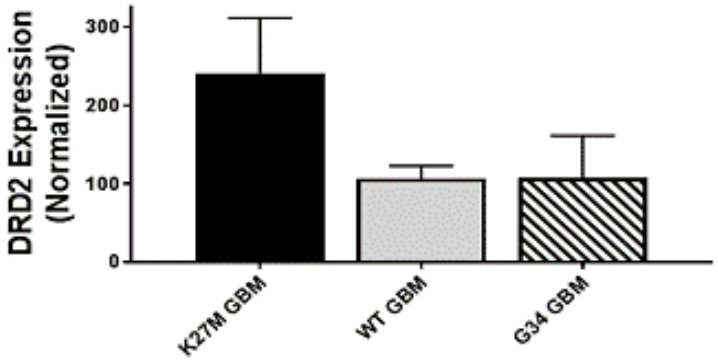


- ONC201 can selectively induce apoptosis in cancer cells by altering the activity of two protein targets
- DRD2 antagonism
  - DRD2 is a G protein-coupled neuroreceptor that regulates Ras signaling
  - ONC201 antagonizes DRD2, inhibiting Ras signaling pathways
- ClpP agonism
  - ClpP normally degrades misfolded proteins in mitochondria
  - ONC201 modifies ClpP conformation, promoting excess degradation of specific mitochondrial proteins important for cancer cell viability

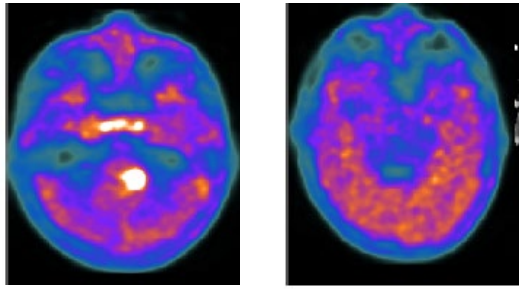
ClpP=caseinolytic protease P; OXPHOS=oxidative phosphorylation; DRD2=Dopamine receptor D2; ATF4=activating transcription factor 4; CHOP=C/EBP-homologous protein; ERK=extracellular-regulated kinase;

H3 K27M glioma primed for ONC201 sensitivity

DRD2 pathway inhibited by ONC201 is enriched in H3 K27M glioma



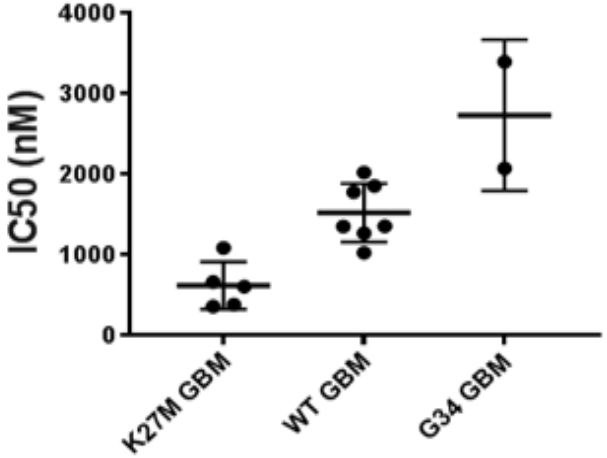
DRD2 overexpression in H3 K27M glioma



18F-DOPA PET

H3 K27M glioma often located in dopamine-rich environment

H3 K27M is hypersensitive to ONC201

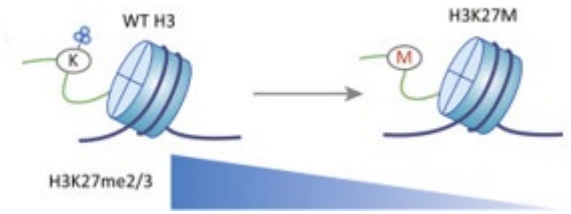


Ex vivo high grade glioma growth sensitivity to ONC201 by H3 status

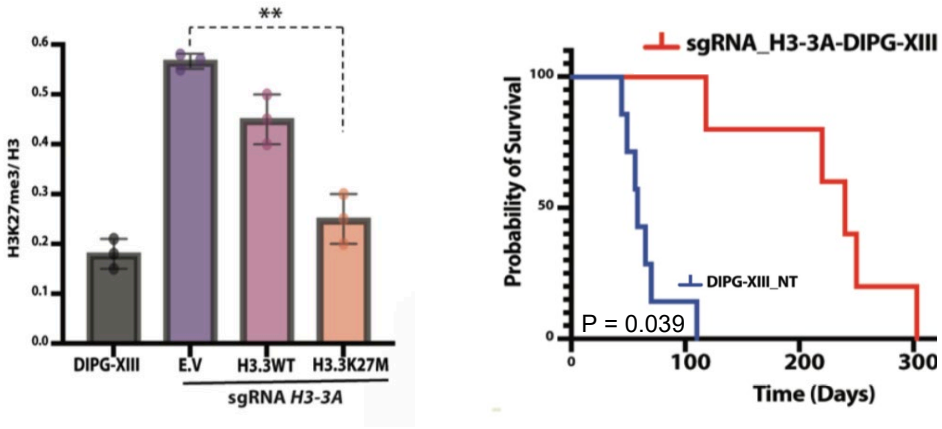
Chi et al., Society of Neuro-Oncology, 2017; Piccardo et al., Eur J. Nucl Med Mol Imaging, 2019

H3 K27M central characteristic reversed by ONC201

H3 K27M causes loss of global H3 K27 trimethylation (H3 K27me3-loss)

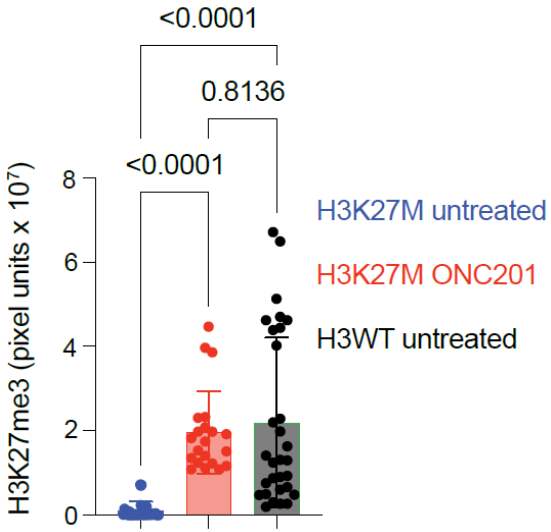


Removal of H3 K27M mutation results in reversal H3 K27me3-loss and prolonged OS in mouse models of H3 K27M glioma



CRISPR-Cas9 deletion of H3 K27M
(left) specifically increases H3 K27me3 and (right) prolongs OS

ONC201 reverses H3 K27me3-loss in H3 K27M glioma patients' tumors



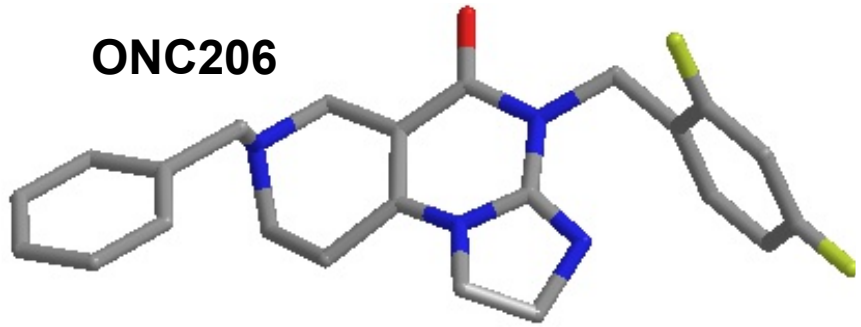
https://www.cell.com/trends/cancer/fulltext/S2405-8033%2819%2930202-X; Zhang et al, Science Translational Medicine, 2023; Koshmann et al, AACR 2023

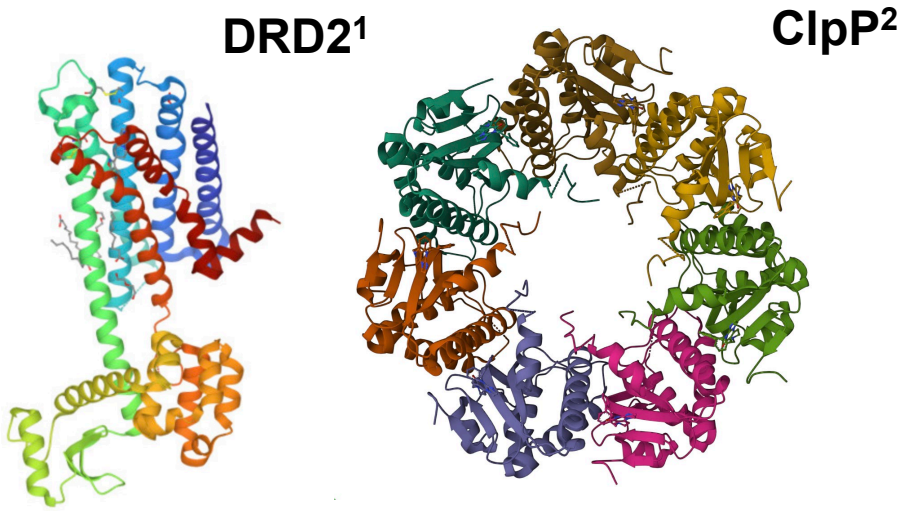
ONC206

CHIMERIX

ONC206: oral brain penetrant DRD2 antagonist + ClpP agonist

- Second generation imipridone designed to expand to new indications
- Efficacy in cell culture, xenograft and transgenic central nervous system (CNS) and other tumor models
- Oral dose escalation trials ongoing in CNS cancers
- Monotherapy response reported by investigator in early dose escalation cohort for a patient in recurrent non-H3 K27M GBM
  - Dordaviprone responses amongst CNS tumors exclusively in H3K27M gliomas
  - Dose level 2 (100mg), once weekly dosing





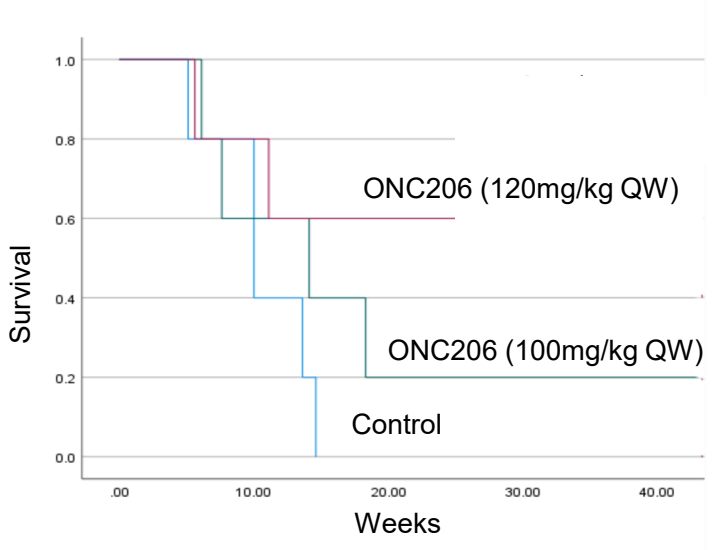
1. PDB 6CM4
2. PDB 6DL7

ONC206 monotherapy active in models of CNS and other cancers

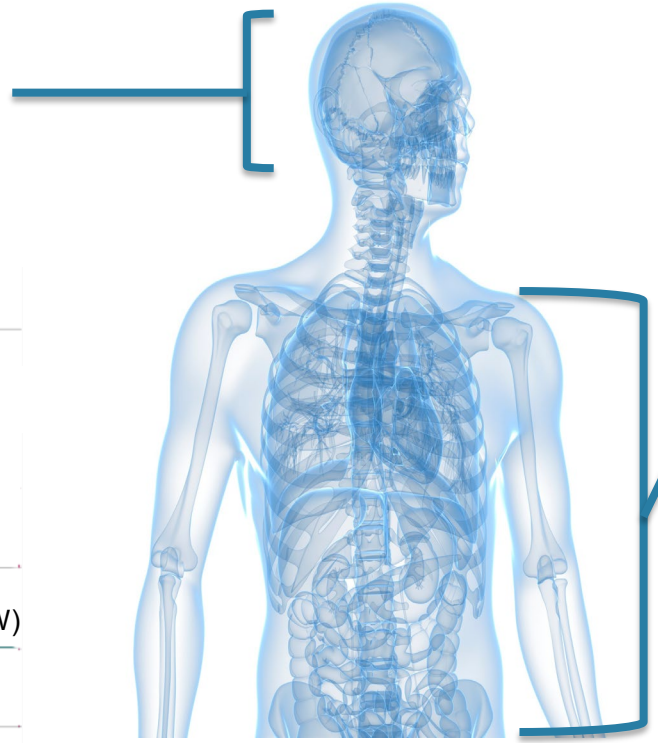
CNS Tumors

Glioblastoma[1]

Medulloblastoma[2]



Transgenic Medulloblastoma Model[2]

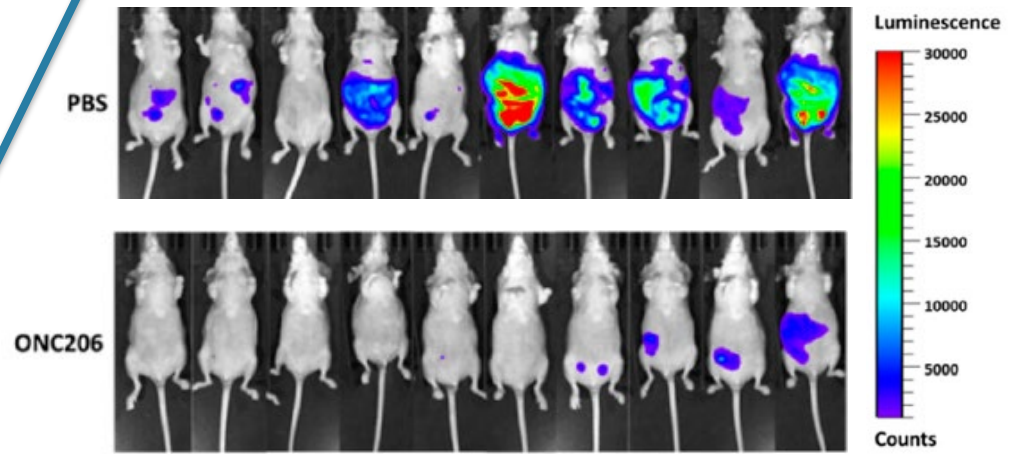


Non-CNS Solid Tumors

Endometrial cancer[3]

Ovarian cancer[4]

Cholangiocarcinoma[1]



ARK1 Human Endometrial Cancer Xenograft (100mg/kg BW; 6 wks)[3]

1. Theeler et al, SNO 2020
2. Malhotra et al, ISPNO, 2020
3. Hu et al, Cancers 2020
4. Tucker et al, American Journal of Cancer Research, 2022

ONC206 dose escalation: pediatric and adult CNS tumors

- Monotherapy dose escalation trials enrolling in parallel for adult and pediatric CNS tumors
- Response reported by investigator from early cohort (100mg QW) without H3 K27M mutation
  - 18-year-old patient with recurrent temporal lobe glioblastoma
  - Regression on MRI & metabolic reduction via PET imaging, continuing on therapy over 15 months
- Once weekly dose escalation is expected to intensify to three consecutive days per week

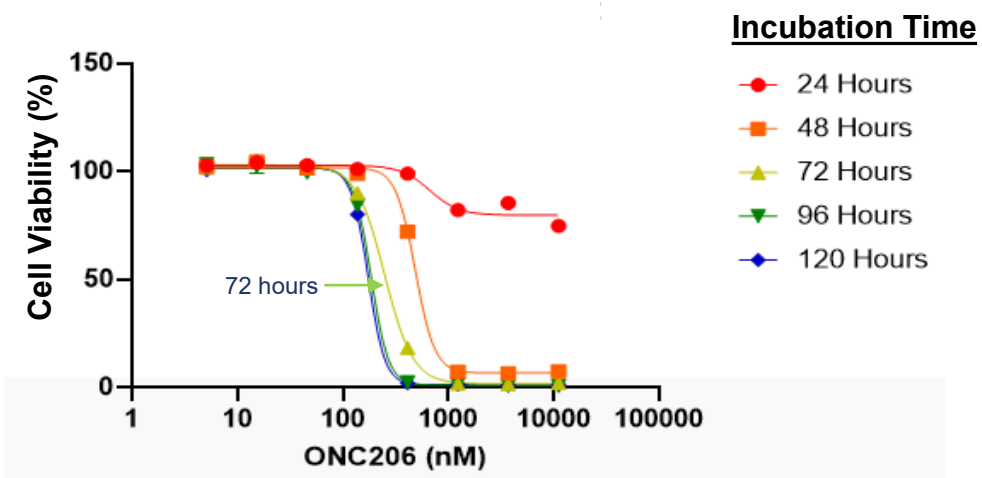
National Institutes of Health

PACIFIC PEDIATRIC NEURO-ONCOLOGY CONSORTIUM

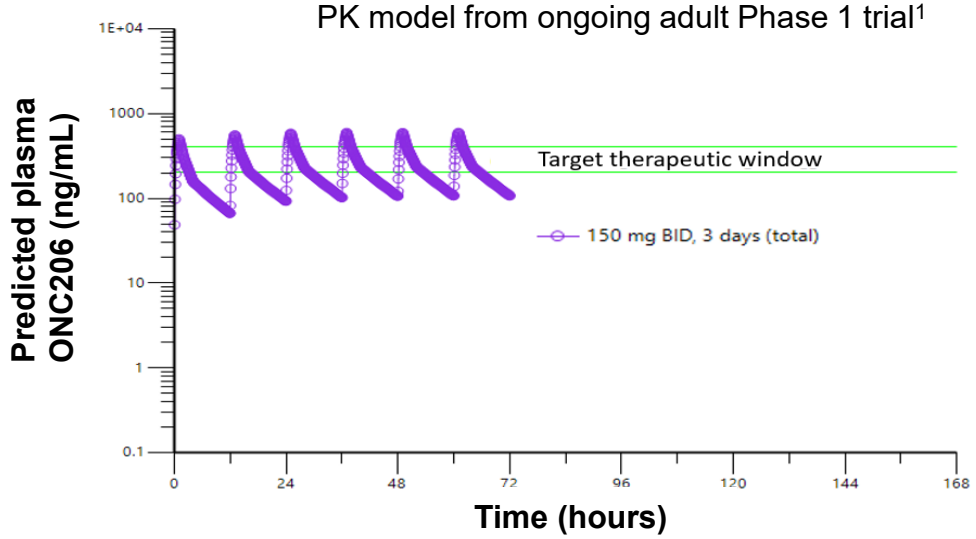
Dose intensification expected to enhance duration of therapeutic exposure

- Consecutive day dosing may increase therapeutic response
  - In vitro data demonstrates enhanced efficacy with 72 hour sustained exposure
  - Toxicology data enables safe escalation to more prolonged exposures
- Phase 1 trial data suggest a therapeutic and safe exposure possible with twice daily, three times weekly dosing

HGG in vitro response to ONC206 enhanced with exposure time



PK model from ongoing adult Phase 1 trial[1]

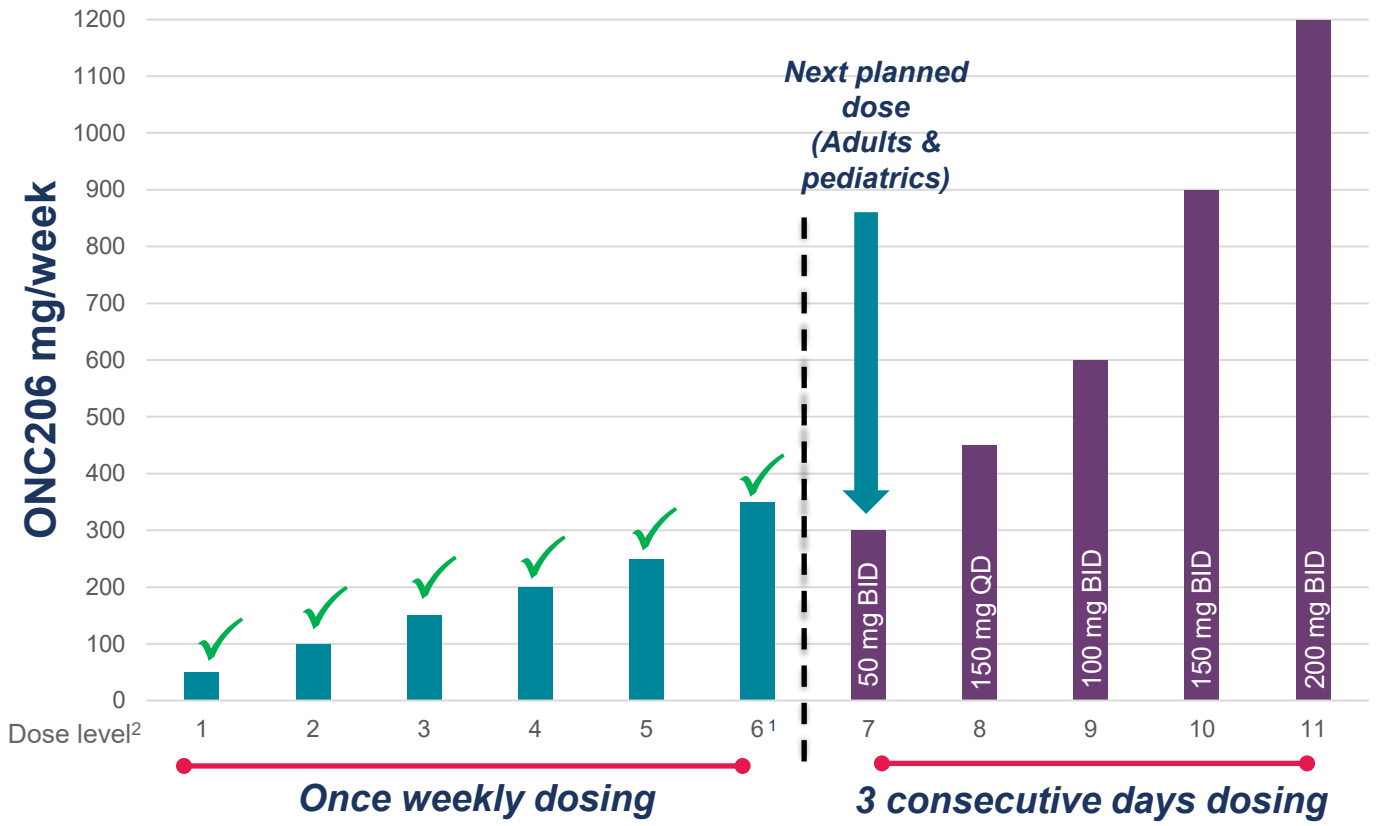


1. Internal modeling

ONC206 dose escalation increasing to more frequent dosing

Dose escalation on track for completion in 1H24

- No DLTs observed with weekly dosing
  - Similar safety profile in adults and pediatrics
  - Majority of treatment-related AEs are mild to moderate
  - Most common treatment-related events are fatigue, lymphocyte count decreased, and vomiting
  - No dose related toxicity with dose escalation – dose escalation continuing



In vitro data indicates correlation between exposure time and tumor cell viability; more frequent dosing schedule designed to increase duration of target exposure

1. Dose level 6 was conducted in adults only
2. Pediatric dose scaled by body weight

✓ Dose level complete

~30,000 new cases of GBM annually in the top 7 markets; >$2Bn market opportunity

- GBM is a rapidly progressive disease with low survival rates, few drug approvals last 25 years:
  - Temozolomide (TMZ) approved 1999
  - Bevacizumab approved 2009
- Existing therapies rarely offer durable effect
  - 3-year survival from diagnosis

1 out of 20[1]

- Chimerix retains global operational rights to ONC206[2]
- Worldwide market opportunity exceeds $2Bn
  - TMZ revenue peaked at approximately $1.4 billion in 2009, prior to going generic
    - Inflation adjusted peak: > $2.5Bn
  - New GBM therapy: 50% penetration at average price of contemporary oncology drug approvals exceeds $2Bn

1 wjso.biomedcentral.com/articles/10.1186/1477-7819-10-220
[2] Royalties and milestones are owed to legacy Oncoceutics shareholders by virtue of the 2021 merger agreement

Preclinical Development

ONC212 and CMX521

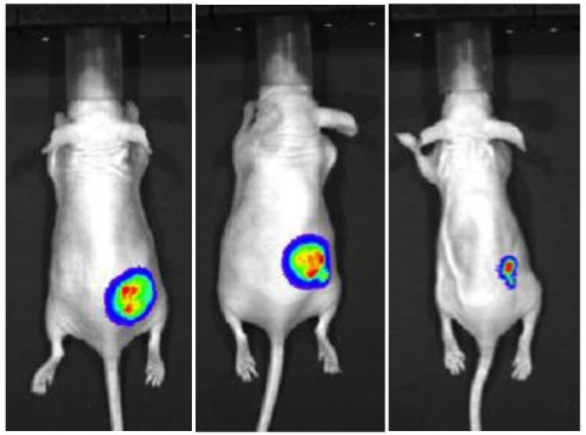
CHIMERIX

ONC212: GPR132 + ClpP Agonist

- Distinct molecular target (GPR132) and efficacy profile relative to ONC201/ONC206
- Efficacy in preclinical models of advanced cancers
- GLP-tox studies complete, potential to advance to IND
- Partnerships established for early-stage clinical trials with Brown University and MD Anderson Cancer Center
- Preclinical studies are ongoing to evaluate additional oncology indications and predictive biomarkers for ONC212 for clinical development

Pancreatic cancer model shows the potential of ONC212[1]

Vehicle ONC201 ONC212



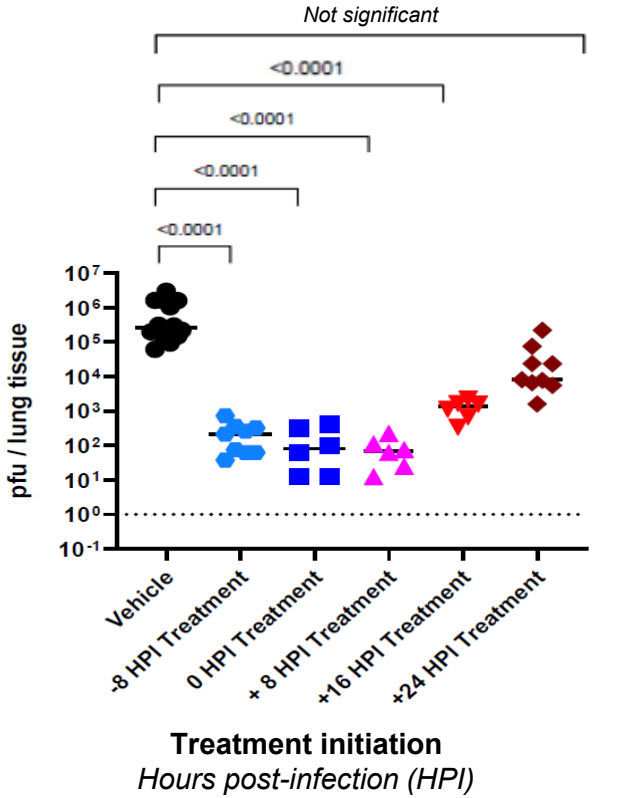
1. Lev et al, Oncotarget 2017

CMX-521: anti-SARS-CoV-2 preclinical activity

- Ribonucleoside analog that is a viral polymerase inhibitor
  - Inhaled nebulized liquid aerosol formulation; minimal systemic exposure
- Monotherapy efficacy in mouse-adapted SARS-CoV-2-MA10 model across multiple endpoints
  - Lung viral titer
  - Viral RNA parallel viral lung titer (plaque forming unit)
  - Clinical scoring (animal health)
  - Lung pathology
  - Animal weight loss

$2 million grant to fund prodrug formulations that could enable oral administration with improved lung delivery

Viral Lung Titer
Replicating SARS-COV-2-MA10[1]



Treatment initiation
Hours post-infection (HPI)

1. Replicates lung pathology of human infection 4-days post-infection. One day in mouse is 5-7 days in humans (adjusted disease course).

Corporate Update

CHIMERIX

TEMBEXA® deal term summary

Emergent BioSolutions is an experienced biodefense company collaborating with government agencies to protect public health

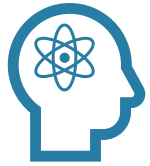
Terms summary:

- $238 million received upfront at closing in Q3 2022
- Up to an additional $124 million in potential BARDA procurement milestones
- 20% royalty on future U.S. gross profit with volumes above 1.7 million courses of therapy
- 15% royalty of all international gross profit
- Up to an additional $12.5 million in development milestones

TEMBEXA®
brincidofovir
10 mg/mL oral suspension | 100 mg tablets

Financial strength supports development through key catalysts

High probability of success for Phase 3 ACTION study of ONC201



Low barriers to commercial potential for ONC201

Corporate capability and financial flexibility

$233 million in capital to fund operations as of June 30, 2023, no debt

Fully funded Ph 3 program with multiple potential paths to approval

First-Line H3 K27M-mutant diffuse glioma – The ACTION Study

- ✓ *Trial initiated November 2022*
- ✓ *Interim OS data expected early 2025, full OS data expected 2026*

ONC206 in early dose escalation studies at NIH and PNOC

- ✓ *Investigator reported response in Non-H3 K27M recurrent glioblastoma patient*

Early-stage pipeline leverages external capital

- ✓ *Pre-clinical programs potential to advance to clinic or partner (ONC212, CMX521)*
- ✓ *Robust business development search and evaluation process*

Chimerix Corporate Presentation

CHIMERIX