Development and Portfolio Overview
June 2019
Summary

• Lead compound: ONC201, a novel, small molecule that targets dopamine receptor D2 (DRD2), a G-Protein Coupled Receptor (GPCR)
• ONC201 has shown clinical benefit in high-grade gliomas with the H3 K27M mutation and early efficacy for additional tumor types
• Molecular structure has allowed for the development of analogs, the first of which will enter the clinic in 2019
ONC201: First Clinical Bitopic DRD2 Antagonist

- Bitopic binding to DRD2 enables unique competitive and non-competitive antagonism
- Antagonism, via orthosteric and allosteric residues, achieves selectivity and unique functional antagonism

**Source:** Prabhu et al, Society of Neuro-Oncology, 2018
Selective DRD2 Antagonism Induces Tumor Apoptosis

- ONC201 is the first DRD2 antagonist for oncology
- Effective in tumors that express DRD2 and rely on its downstream pathways

Relevant publications
- Allen et al., *Science Translational Medicine*, 2013
- Ishida et al, *Clinical Cancer Research*, 2018
- Ishizawa et al, *Cancer Cell*, 2019
DRD2 is Overexpressed in Gliomas and a Target for ONC201

DRD2 Overexpression is Critical for Tumor Growth in GBM

ONC201 Prolongs Survival in Orthotopic GBM Models

ONC201 High-Grade Glioma Clinical Development Pathway

First in Human Phase I Trial (ONC002)

Adult Glioblastoma Phase II (ONC006)

Adult GBM Phase II H3 K27M–Mutant Glioma Dedicated Arm (ONC006)

Adult H3 K27M–Mutant Glioma Phase II (ONC013)

Pediatric H3 K27M–Mutant Glioma & DIPG Phase I (ONC014)

Various Non-Glioma Phase II Trials
ONC201 Demonstrates a Favorable Safety Profile

- >350 patients have been treated with ONC201
  - >150 high grade glioma patients
  - Pediatric experience in >60 patients consistent with adult experience
- No discontinuation due to drug-related AEs with continuous therapy up to three years
- No causality established between ONC201 and Grade 3/4 AEs
- Safety profile and convenient oral regimen enables
  - Fixed dosing in adults
  - High rate of compliance
  - Multiple therapeutic settings
  - Combination therapies

### AEs in Q1W Recurrent Glioblastoma Patients (n=20)

<table>
<thead>
<tr>
<th>Adverse Events, N (%)</th>
<th>All Adverse Events</th>
<th>Possibly/Probably-Related</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All Grades</td>
<td>Grade 3-4</td>
</tr>
<tr>
<td>Nervous system disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dizziness</td>
<td>4 (20%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Metabolism and nutrition disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypophosphatemia</td>
<td>9 (45%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Anorexia</td>
<td>4 (20%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>General disorders and administration site conditions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gait disturbance</td>
<td>8 (40%)</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Injury, poisoning and procedural complications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fall</td>
<td>8 (40%)</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Gastrointestinal disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nausea</td>
<td>7 (35%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>7 (35%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>5 (25%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Investigations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Platelet count decreased</td>
<td>6 (30%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Psychiatric disorders</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Confusion</td>
<td>5 (25%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

All AEs reported in >10% of patients with at least one event attributed by investigator as a least possibly-related to study drug.
Phase II GBM Responder: Near Complete Tumor Regression

- 22 year old patient (at enrollment) with recurrent H3 K27M–mutant glioma
- Started single agent ONC201 treatment after recurrence of tumor in March 2016
- Experienced durable complete thalamic regression; de minimis remaining lesion

**H3 K27M-mutant glioma sensitive to ONC201 and associated with positive outlier response**

Source: Arrillaga et al, Oncotarget, 2017
H3 K27M-Mutant Glioma Is A Grade IV Glioma With No Effective Drugs

- Mutation mainly occurs in cancers in younger patients in the midline of brain (50-90% of midline gliomas)
- Represents a distinct disease entity in the 2016 WHO classification
- Most frequent histone mutation in pediatric glioma
- Standard of care post-radiation is experimental due to lack of effective therapies
H3 K27M-Mutant Gliomas Exhibit Enhanced ONC201 Sensitivity

H3 K27M-mutant gliomas are highly sensitive to ONC201

H3 K27M-mutant gliomas overexpress DRD2 and are highly clonal

Source: Chi et al. SNO (2017); Solomon et al. Brain Pathology (2016)
Summary of Enrollment for Patients with H3 K27M-Mutant Glioma & DIPG

Trial Site
ONC006 (Adult H3 K27M-Mutant Glioma and Midline Gliomas)
Massachusetts General Hospital
Dana Farber Cancer Institute
Miami Cancer Institute
University of California, Los Angeles

ONC013 (Adult H3 K27M-Mutant Glioma)
New York University
Levine Cancer Institute
MD Anderson Cancer Center
University of California, San Francisco
Columbia University

Comp Use
ONC006
12
ONC013
27
Comp Use
18
Comp Use
33

ONC014 (Pediatric H3 K27M-Mutant Glioma)
New York University
MD Anderson Cancer Center
Miami Cancer Institute
University of Michigan
Emory University School of Medicine
University of Cincinnati

Total
134
Adult
57
Pediatric
77

Data as of 5/27/19
Efficacy Presented for 15 Adults with Recurrent H3 K27M-Mutant Glioma

One target registration indication is adult recurrent H3 K27M-mutant glioma

*Integrated analysis criteria for Objective Response Rate Measurement

- Histone H3 K27M mutation by IHC or sequencing test in CLIA lab
- Measurable and progressive disease by RANO
- At least prior radiotherapy
- >90 days from prior radiation
- FFPE tissue for biomarker assessments
- Corticosteroid dose must be stable or decreasing for at least 3 days prior to baseline scan
- KPS ≥ 60
- Evidence of leptomeningeal spread of disease or evidence of CSF dissemination excluded
- Single agent ONC201 until disease progression (no current anti-cancer therapies)
- Primary lesion outside of the pons or spine
- Treated under Oncoceutics’ IND

Data as of 5/27/19 with enrollment cut-off date 12/1/18
ORR by RANO in 15 Adults with Recurrent H3 K27M-Mutant Gliomas

Tumor Size Δ for 15 Adult Recurrent H3 K27M Patients

- CR - Complete Response - RANO
- PR - Partial Response - RANO
- MR - Minor Response
- SD - Stable Disease - RANO
- PD - Progressive Disease - RANO

* patient remains on study

CE: Contrast-enhancing disease evaluated by RANO-HGG
(Wen et al Journal of Clinical Oncology, 2010)

NCE: Non-Contrast-Enhancing disease evaluated by RANO-LGG
(van den Bent, Lancet Oncology, 2011)

Data as of 5/6/19 with enrollment cut-off date 12/1/18
ORR by RANO in 15 Adults with Recurrent H3 K27M-Mutant Gliomas

- Blinded, independent central review was performed for contrast-enhancing disease by RANO-HGG and non-contrast-enhancing disease by RANO-LGG
- Six patients remain on treatment; ORR not final

<table>
<thead>
<tr>
<th>Best RANO Response (N=15)</th>
<th>CE</th>
<th>NCE</th>
<th>CE or NCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete Response</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Partial Response</td>
<td>3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Minor Response</td>
<td>-</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Stable Disease</td>
<td>7</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Progress Disease</td>
<td>4</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Unevaluable</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
<td>15</td>
<td>15</td>
</tr>
</tbody>
</table>

**Objective Response Rate (95% Confidence Interval)**

- Complete Response: 27% (8-55%)
- Partial Response: 36% (13-65%)
- Stable Disease: 47% (21-73%)

**CE:** Contrast-enhancing disease evaluated by RANO-HGG
(Wen et al Journal of Clinical Oncology, 2010)

**NCE:** Non-Contrast-Enhancing disease evaluated by RANO-LGG
(van den Bent, Lancet Oncology, 2011)
Regressions Remain Durable

Tumor Size Δ for 15 Adult Recurrent H3 K27M Patients (CE/RANO-HGG)

- Onset of response: median 2.6 months (range 1.3-3.4)
- Duration of response: median not reached with median follow up of 7.7 months (range 1.8-29.8)

Tumor regressions due to ONC201 are highly durable

Data as of 5/6/19 with enrollment cut-off date 12/1/18
Adult Progression-Free Survival and Overall Survival

**Proxy Historical Control**

Studies from recurrent GBM trials for patients with unmethylated MGMT status provide best available proxy historical control.

**Weighted average from the following studies**

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>mPFS</th>
<th>PFS6</th>
<th>mOS</th>
<th>OS12</th>
</tr>
</thead>
<tbody>
<tr>
<td>BELOB 2014</td>
<td>24</td>
<td>3.0</td>
<td>0.0%</td>
<td>8.0</td>
<td></td>
</tr>
<tr>
<td>DIRECTOR 2015</td>
<td>58</td>
<td>1.8</td>
<td>6.9%</td>
<td>7.9</td>
<td>22.9%</td>
</tr>
<tr>
<td>EORTC 26101</td>
<td>126</td>
<td>1.5</td>
<td>2.3%</td>
<td>8.0</td>
<td>22.2%</td>
</tr>
</tbody>
</table>

**Progression-Free Survival (CE / RANO-HGG)**

- G15 H3 K27M (n=15)
- **PFS6: 33%**

**Overall Survival**

- G15 H3 K27M (n=15)
- mOS not reached
- Median FU 7.5m

Investigator Reported Data as of 3/25/19 with enrollment cut-off date 12/1/18
Tumor Regressions Observed in Pediatric Patients

- 6 year-old thalamic H3 K27M-mutant glioma patient who initiated ONC201 one week after radiation
- Panel C represents a 50% regression relative to panel B

A. June 2018 – At Diagnosis
B. Aug 2018 – Post-radiation
C. Mar 2019– 27 weeks ONC201

50% reduction from baseline
Key FDA Communications

• FDA granted Fast Track designation to ONC201 for the indication of adult recurrent H3 K27M-mutant high-grade glioma

• Additional communications have established the following:

1. H3 K27M-mutant glioma is a serious condition and an approvable indication

2. Patient population must be homogeneous for NDA-directed programs

3. Accelerated Approval can be permitted utilizing ORR as a primary endpoint

Additional information to be clarified at upcoming FDA meetings
Anticipated Clinical Development Timeline

<table>
<thead>
<tr>
<th>Event</th>
<th>2019</th>
<th>2020</th>
<th>2021</th>
<th>2022</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continue PII Trials (Adult H3 K27M)</td>
<td>Q1</td>
<td>Q2</td>
<td>Q3</td>
<td>Q4</td>
</tr>
<tr>
<td>Seamless transition to registration trial(s)</td>
<td>Q1</td>
<td>Q2</td>
<td>Q3</td>
<td>Q4</td>
</tr>
<tr>
<td>Registration Data Read-Out</td>
<td>Q1</td>
<td>Q2</td>
<td>Q3</td>
<td>Q4</td>
</tr>
<tr>
<td>Planned NDA Filing</td>
<td>Q1</td>
<td>Q2</td>
<td>Q3</td>
<td>Q4</td>
</tr>
<tr>
<td>FDA Approval Decision</td>
<td>Q1</td>
<td>Q2</td>
<td>Q3</td>
<td>Q4</td>
</tr>
<tr>
<td>Commercial Sales</td>
<td>Q1</td>
<td>Q2</td>
<td>Q3</td>
<td>Q4</td>
</tr>
<tr>
<td>NRG / COG Trial (H3 K27M &amp; DIPG)</td>
<td>Q1</td>
<td>Q2</td>
<td>Q3</td>
<td>Q4</td>
</tr>
<tr>
<td>BIOMEDE: Newly Diagnosed DIPG</td>
<td>Q1</td>
<td>Q2</td>
<td>Q3</td>
<td>Q4</td>
</tr>
<tr>
<td>Ongoing Trials Outside H3 K27M</td>
<td>Q1</td>
<td>Q2</td>
<td>Q3</td>
<td>Q4</td>
</tr>
</tbody>
</table>

- **2019**: Continue PII Trials (Adult H3 K27M)
- **2020**: Seamless transition to registration trial(s)
- **2021**: Registration Data Read-Out, Planned NDA Filing, FDA Approval Decision, Commercial Sales
- **2022**: NRG / COG Trial (H3 K27M & DIPG), BIOMEDE: Newly Diagnosed DIPG, Ongoing Trials Outside H3 K27M

Key Events:
- Pediatric Subcommittee of the ODAC presentation
- FDA Meeting
- Expected Enrollment Complete
- Primary readout complete
- FDA Approval
H3 K27M-Mutant Gliomas: US Incidence of ~2,000 Per Year

**Estimate Using Histology/Age: 2,045 Patients**

<table>
<thead>
<tr>
<th>By histology</th>
<th># of primary BTs</th>
<th># of gliomas</th>
<th>% K27M</th>
<th># K27M</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult gliomas</td>
<td>75,260</td>
<td>19,131</td>
<td>7.5%</td>
<td>1,435</td>
</tr>
<tr>
<td>Pediatric glioma (ex DIPG)</td>
<td>4,307</td>
<td>1,868</td>
<td>20.0%</td>
<td>374</td>
</tr>
<tr>
<td>DIPG</td>
<td>303</td>
<td>303</td>
<td>78.0%</td>
<td>236</td>
</tr>
<tr>
<td><strong>All gliomas</strong></td>
<td><strong>79,870</strong></td>
<td><strong>21,303</strong></td>
<td><strong>9.6%</strong></td>
<td><strong>2,045</strong></td>
</tr>
</tbody>
</table>

**Estimate Using Location: 1,992 Patients**

<table>
<thead>
<tr>
<th>By location</th>
<th># of primary BTs</th>
<th># of gliomas</th>
<th>% K27M</th>
<th># K27M</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thalamic</td>
<td>334</td>
<td>334</td>
<td>67.8%</td>
<td>226</td>
</tr>
<tr>
<td>DIPG</td>
<td>303</td>
<td>303</td>
<td>78.0%</td>
<td>236</td>
</tr>
<tr>
<td>Other brain stem (ex DIPG)</td>
<td>883</td>
<td>441</td>
<td>53.6%</td>
<td>236</td>
</tr>
<tr>
<td>Spinal cord</td>
<td>2,344</td>
<td>764</td>
<td>67.3%</td>
<td>514</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>1,723</td>
<td>345</td>
<td>20.0%</td>
<td>69</td>
</tr>
<tr>
<td>Pineal</td>
<td>331</td>
<td>166</td>
<td>50.0%</td>
<td>83</td>
</tr>
<tr>
<td>Ventricle</td>
<td>832</td>
<td>103</td>
<td>50.0%</td>
<td>51</td>
</tr>
<tr>
<td>Other midline</td>
<td>400</td>
<td>200</td>
<td>50.0%</td>
<td>100</td>
</tr>
<tr>
<td>Non-midline locations</td>
<td>72,720</td>
<td>18,648</td>
<td>2.6%</td>
<td>476</td>
</tr>
<tr>
<td><strong>All gliomas</strong></td>
<td><strong>79,870</strong></td>
<td><strong>21,303</strong></td>
<td><strong>9.3%</strong></td>
<td><strong>1,992</strong></td>
</tr>
</tbody>
</table>

**H3 K27M Incidence Worldwide**

<table>
<thead>
<tr>
<th>Market</th>
<th>Population (mm)</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>US</td>
<td>324</td>
<td>2,000</td>
</tr>
<tr>
<td>EU</td>
<td>509</td>
<td>3,142</td>
</tr>
<tr>
<td>China</td>
<td>1,410</td>
<td>8,704</td>
</tr>
<tr>
<td>Japan</td>
<td>127</td>
<td>784</td>
</tr>
<tr>
<td>ROW</td>
<td>5,180</td>
<td>31,975</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>7,550</strong></td>
<td><strong>46,605</strong></td>
</tr>
</tbody>
</table>

H3 K27M-mutant glioma is a well-defined orphan indication with significant unmet medical need
Competitive Landscape for H3 K27M-Mutant Gliomas

- There are currently no approved drugs that show efficacy in H3 K27M-mutant or other midline gliomas
- Preclinical studies that showed promising data have not been able to translate successfully to the clinic
- Glioblastoma therapies non-competitive with ONC201 in midline gliomas

<table>
<thead>
<tr>
<th>Radiotherapy</th>
<th>Chemotherapy</th>
<th>Immunotherapy</th>
<th>Epigenetic Targets</th>
<th>Medical Devices</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Only treatment that is considered standard of care in midline glioma</td>
<td>• No positive results with TMZ or other chemotherapies</td>
<td>• Checkpoint inhibitors not proven effective in gliomas</td>
<td>• No specific H3 K27M-mutant glioma trials</td>
<td>• Optune device (Novocure) targets supratentorial tumors, not used for midline gliomas</td>
</tr>
<tr>
<td>• No results available for convection-enhanced delivery trial</td>
<td>• No results available for convection-enhanced delivery trial</td>
<td>• No definitive results available for H3 K27M-mutant glioma vaccine trials</td>
<td>• Investigational EZH2 inhibitor does not penetrate BBB</td>
<td></td>
</tr>
</tbody>
</table>
Multiple Patents Issued in US and ROW

• Extensive portfolio of issued and pending patents for ONC201, preventing generic entry and enabling life cycle management
  • 13 total patents issued for ONC201 (9 US, 1 EU, 1 Japan, 1 Singapore, 1 Australia)
    • Use patents in the US for cancer indications including: brain, breast, lung, lymphoma, colon, adenocarcinomas, leukemias, genitourinary, and spinal cord
    • US patent for midline gliomas with an H3 K27M mutation extends the ONC201 patent life through at least 2038
    • Notice of allowance issued in US for glioma with H3 K27M mutation and midline gliomas independent of H3 K27M mutation through at least 2038
    • Notice of allowance issued in Mexico for the treatment of brain cancer
    • US patent coverage includes all combinations of ONC201 with any other therapeutic
    • US composition of matter patent issued for di-salt formulation of ONC201
    • Patent term extension available due to New Chemical Entity (NCE) and orphan drug exclusivity
  • Other imipridone family IP
    • ONC206 family: US and EU Composition of Matter patent issued; Notice of Indication of Grant in Eurasia and Israel
    • ONC212 family: US Composition of Matter patent issued
    • ONC213 family: US Composition of Matter patent issued
  • Active prosecution for ONC201 and other imipridones in Europe and ROW
High DRD2 Expression Suggests Additional Opportunity for ONC201

- DRD2 expression in normal brains is highest in specific midline areas where complete regression of malignant lesions are being observed.
- Tumor micro environment has been shown to participate in response to ONC201.

DRD2 overexpression is correlated with locations common for H3 K27M mutations.
Other Ongoing Clinical Trials

- ONC201 is also being evaluated in the following tumor types:
  - Other high-grade gliomas
  - Adrenocortical carcinoma
  - Neuroendocrine Tumors
    - Pheochromocytoma / Paraganglioma
  - Endometrial cancer
  - Non-Hodgkin’s Lymphoma
  - Acute Leukemias
  - Multiple Myeloma
  - Breast cancer
  - Other solid tumors
Synergistic Combinations Identified in Preclinical Models of Specific Cancers

**Lung**
- Allen et al. 2013 (Taxanes)

**Breast**
- Ralff et al. 2017 (Taxanes)
- Allen et al. 2013 (Taxanes)
- Jhawar et al. 2018 (Radiation)
- Baumeister et al. 2017 (PARP Inhibitors)
- Baumeister et al. 2019 (DR5 antibody)

**Hepatocellular Cancer**
- Allen et al. 2016 (Sorafenib)

**Pancreatic**
- Zhang et al. 2017 (Gemcitabine)

**Multiple Myeloma**
- Tu et al. 2017 (Bortezomib or Carfilzomib)
- Prabhu et al. 2018 (Bortezomib)
- Prabhu et al. 2018 (Ixazomib)
- Prabhu et al. 2018 (Dexamethasone)

**Prostate**
- Lev et al. 2018 (Everolimus)
- Lev et al. 2018 (Enzalutamide)
- Baumeister et al. 2017 (PARP Inhibitors)

**GIoma**
- Allen et al. 2013 (Bevacizumab)
- Baumeister et al. 2017 (PARP Inhibitors)
- Karpe-Massler et al. 2015 (BCL-2 Inhibitors)
- Tarapore et al. 2019 (Radiation)
- Zhou et al. 2019 (Temozolomide, Radiation)
- Zhang et al. 2019 (Vorinostat)

**Lymphoma**
- Talekar et al. 2015 (Cytarabine)
- Prabhu et al. 2018 (Bortezomib)
- Prabhu et al. 2018 (Cytarabine)

**AML**
- Prabhu et al. 2018 (Cytarabine)
- Allen et al. 2016 (BCL-2 Inhibitors)
- Prabhu et al. 2018 (5-azacytidine)

**CRC**
- Wagner et al. 2018 (Anti-PD-1 therapy)
- Allen et al. 2016 (Bevacizumab)
- Allen et al. 2013 (Taxanes)

**Ovarian**
- Baumeister et al. 2017 (PARP Inhibitors)
- Rumman et al. 2019 (Taxol)

**Endometrial**
- Fang et al. 2018 (Taxol)
Synergistic Antitumor Activity Observed In Preclinical Models

- ONC201 combines synergistically with numerous chemotherapies and targeted agents

**Chemotherapies**
- Cytarabine
- Paclitaxel
- Docetaxel
- Gemcitabine
- Cabazitaxel

**Targeted Agents**
- Sorafenib
- Bevacizumab
- Bortezomib
- Ixazomib
- Dexamethasone
- Everolimus
- Enzalutamide
- Olaparib
- ABT-199
- ABT-263

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Allen et al., *Cancer Research*, 2015

Ishizawa et al., *Science Signaling*, 2016

Drug Candidates From the Imipridone Platform Under Development

- Members of the imipridone family share a unique tri-heterocyclic core structure
  - Oral bioavailability
  - Wide therapeutic window
  - Blood brain barrier penetrance
  - Selective GPCR engagement
- Imipridones selectively engage GPCRs
  - Common target of prescription drugs (~40%)
  - Underexploited in oncology
- ONC201 is the lead, clinical stage compound
- ONC206 clinical introduction planned for 2019
- ONC212 IND-enabling studies planned for 2020
- ONC213 late preclinical development candidate
- Structure activity relationship indicates platform potential for multiple GPCRs
Multiple Opportunities for Collaboration

• Lead asset: ONC201
  • Partnership for late-stage clinical development and commercialization
• Development candidates: ONC206, ONC212, ONC213
  • Demonstrated anti-cancer efficacy in-vivo
  • Partnership for IND and Phase I/II studies