Taking a Precision Cancer Medicine™ Approach to Develop Targeted Drugs for Cancer Indications with Significant Need for New Treatment Options

NASDAQ: TROV
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Robust, diversified pipeline with single molecule, onvansertib, addressing multiple cancer indications, each with significant medical need for new treatment options

Preclinical data demonstrating efficacy of onvansertib in combination with standard-of-care drugs, expanding therapeutic and partnership opportunities

Encouraging initial efficacy data from ongoing clinical trials with additional data readouts in 2019-2020

Precision Cancer Medicine™ approach and integration of biomarkers to target treatment for patients most likely to respond

Experienced team with proven oncology drug development track record
Experienced Management Team
Drug Development Expertise + Biomarker Technology

Thomas Adams, PhD
Chief Executive Officer and Chairman

Mark Erlander, PhD
Chief Scientific Officer

Vicki Kelemen
Vice President Clinical Development
Our Vision
Rapidly Advancing Clinical Development Programs

2H 2019
- 3 clinical-stage programs
- Safety and efficacy data readouts

1H/2H 2020
- Clinical data readouts from all 3 programs
- Phase 2 trials completed
- Registrational trials initiated

Vision 2022
- Commercialization partner(s)
- FDA approval of onvansertib in at least 1 indication
- Second compound licensed and in the clinic
Demonstrated Operational Stability
Cost-Effective and Efficient Model

Raised Capital & Clinical Research Commitment Q1-2, 2019
- $8.0 million

Cash and Cash Equivalents as of March 31, 2019
- $11.3 million

Projected Cash Ending Q2, 2019
- ~$10.8 million

Estimated Quarterly Cash Burn
- ~$4.0 million
Exclusive Global Rights to Onvansertib Licensed from Nerviano Medical Sciences (NMS) in 2017

- Largest oncology research and development company in Italy; highly regarded throughout Europe and the U.S.

- Excellent R&D Reputation and Track Record

- Completed Phase 1 Solid Tumor Trial and IND

- Completed Preclinical Data, Including Synergy

- PLK1 Proven Effective Cancer Therapeutic Target

- Established CMC (chemistry, manufacturing, controls)

- GMP / FDA Validated Manufacturing

- Licensed Drugs to Genentech, Array/Pfizer, Ignyta/Roche

- Experts in Protein Kinase Drug Development
Licensed drug with established safety and recommended Phase 2 dose

Extensive in-vitro and in-vivo data package providing rationale for combination therapy across multiple cancers

Outsourced clinical trial management to CRO, including regulatory, medical/safety monitoring, data management

Readily available supply of API and finished drug for clinical trials

3 INDs in place (1 in each oncology division of the FDA); Orphan Drug Designation in AML
We Have the Perfect Target
Onvansertib – Polo Like Kinase 1 (PLK1) Inhibitor

- Over-expressed in most cancers
- Proven target for cancer therapeutics
- Only oral, first-in-class, third-generation PLK1 in clinical trials
- Preclinical data to support clinical development across multiple tumor types

PLK1 Inhibitor
Onvansertib Targets the PLK1 Enzyme
A Proven Drug Target and Overexpressed in Most Cancers

- High selectivity for PLK1, only
- Tested against >260 kinases; PLK1 only active target (IC$_{50}$ of 2nM)
- Small molecule (MW 648.60 Daltons)
- Selectivity driven by stable interaction with carboxyl side chain of amino acid glutamate of PLK1 within PLK1’s ATP binding pocket

<table>
<thead>
<tr>
<th>PLK Member</th>
<th>Onvansertib IC$_{50}$* (μM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLK1</td>
<td>0.002</td>
</tr>
<tr>
<td>PLK2</td>
<td>&gt; 10</td>
</tr>
<tr>
<td>PLK3</td>
<td>&gt; 10</td>
</tr>
</tbody>
</table>

Onvansertib binds within PLK1 pocket

Onvansertib blocks cells from dividing by arresting them before they divide
Onvansertib
Benefiting from Class Experience

1\textsuperscript{st} and 2\textsuperscript{nd} generation PLK inhibitors demonstrated clinical activity, but were non-specific for PLK1 and had toxicity issues.

<table>
<thead>
<tr>
<th>Product Attributes</th>
<th>1\textsuperscript{st} and 2\textsuperscript{nd} Generation PLK Inhibitors</th>
<th>3\textsuperscript{rd} Generation Onvansertib</th>
</tr>
</thead>
<tbody>
<tr>
<td>Selectivity for PLK1</td>
<td>• panPLK inhibition of PLK1,2,3*</td>
<td>• Highly-selective only for PLK1</td>
</tr>
<tr>
<td>Antileukemic Activity</td>
<td>• Phase 2 &amp; 3 trial results indicate activity</td>
<td>• Clinical response in patients</td>
</tr>
<tr>
<td></td>
<td>• Improved response rates</td>
<td>• Biomarker strategy identifies patients most likely to respond</td>
</tr>
<tr>
<td>Administration</td>
<td>• Intravenous (IV)</td>
<td>• Oral</td>
</tr>
<tr>
<td>Half-Life</td>
<td>• ~135 hours (5.5 days)</td>
<td>• ~24 hours</td>
</tr>
<tr>
<td>Dosing and Schedule</td>
<td>• Fixed treatment schedule</td>
<td>• Treatment schedule flexibility</td>
</tr>
<tr>
<td></td>
<td>• Fixed dose for all patients</td>
<td>• Dose determined based on BSA</td>
</tr>
<tr>
<td>Tolerability</td>
<td>• Insufficient time between treatment cycles negatively impacted tolerability/survival</td>
<td>• Time allotted between cycles for patient recovery from on-target hematologic toxicities</td>
</tr>
<tr>
<td>Infection Prophylaxis</td>
<td>• Increased rate of fatal infections</td>
<td>• Antibiotics to proactively mitigate infections</td>
</tr>
</tbody>
</table>
Onvansertib
First-in-Class, Third-Generation PLK1 with Best-in-Class Attributes
Diversified Pipeline with 3 Clinical-Stage Programs
Opportunities in Leukemias/Lymphomas and Solid Tumors

3 Investigational New Drug (INDs) in place with the FDA

<table>
<thead>
<tr>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
</tr>
</thead>
</table>
| **Metastatic Castration-Resistant Prostate (CRPC)**  
*Phase 2 trial in combination with Zytiga® (abiraterone acetate)/prednisone - Ongoing* | | |
| **Colorectal (CRC)**  
*Phase 1b/2 trial in combination with FOLFIRI + Avastin® - Ongoing* | | |
| **Acute Myeloid Leukemia – Orphan Drug Designation in the U.S. and Europe**  
*Phase 1b/2 trial in combination with low-dose cytarabine (LDAC) or Decitabine - Ongoing* | | |
| **Myelodysplastic Syndrome**  
*Phase 1b/2 Investigator Initiated Trial (commencing Q4’19 / Q1’20)* | | |
| **Ovarian, Breast, Pancreatic, Small-Cell Lung**  
*Phase 1b/2 trial ready (Preclinical Data Available)* | | |

Leukemias and Lymphomas  
Solid Tumors
Encouraging Initial Data and Near-Term Readouts

**TROV-053**

- mCRPC Phase 2
- Initial safety and efficacy data
- Enroll 3 patients in Arm B safety lead-in
  - Identify patients with ARv7 and correlate with treatment response (Q2-3 2019)
  - Provide data from patients completing 3-months of treatment (Q4 2019)

**TROV-054**

- mCRC Phase 1b/2
- Activate clinical trial sites
- Enroll 3 patients in initial dose level cohort
  - Provide data on biomarker assessment of tumor burden change (Q3 2019)
  - Provide initial data from first cohort of 3 patients in dose escalation study (Q4 2019)
  - Initiate second dose level cohort to enroll 3 patients (Q4 2019)

**TROV-052**

- AML Phase 1b/2
- Completion of 6 dose escalation cohorts with no dose-limiting toxicities
- Initial data demonstrating efficacy – complete response (CR)
  - Determine recommended Phase 2 dose (Q4 2019)
  - Enroll patients in Phase 2 (Q1-2 2020)

mCRPC = metastatic castration-resistant prostate cancer; mCRC = metastatic colorectal cancer; AML = acute myeloid leukemia
Combination Therapy for Cancer Treatment
Two Drugs are Better Than One (1+1 = 5)

Onvansertib is uniquely synergistic (1 +1 = 5) with many FDA-approved drugs; it selectively targets the enzymatic activity of PLK1 that is fundamental for tumor growth.

Increases efficacy of the therapeutic effect, particularly when the two drugs differ in their mechanism of action and both deliver anti-tumor activity.

Decreases required dose of each drug and associated toxicity, potentially reducing side effects.

Minimizes the development of drug resistance because the two drugs block different tumor-promoting pathways for cancer growth.

Combination Therapy
The Cornerstone of Precision Cancer Medicine™

trovagene ONCOLOGY
Onvansertib
Synergy May Enhance Efficacy of Standard-of-Care Therapies

Onvansertib current clinical trials

1Data on File, Trovagene, Inc.
Phase 2 Trial: metastatic Castration-Resistant Prostate Cancer
Onvansertib Market Opportunity in mCRPC
Significant Disease Burden - Need for More Effective Treatment Options

Disease Burden

1 of 6 men will be diagnosed with prostate cancer

25,000 men die from metastatic prostate cancer annually

5-year survival rate is 37%

Treatment

Standard-of-care is Zytiga® and Xtandi®; resistance develops within 9-15 months

Tumors re-engineer androgen receptor (AR), variant 7 (ARv7); tumor growth without need for androgens

Up to 40% ARv7 resistance; very aggressive with no viable treatment options

Opportunity

PLK1 inhibition improves Zytiga® efficacy, repressing androgen signaling pathway

PLK1 inhibition destabilizes AR and ARv7

Inhibiting PLK1 blocks expression of ARv7; stopping this resistance pathway

$7.9 billion global market; $12.0 billion by 2025

4. Antonarakis, Emmannel – Current Understanding of Resistance to Abiraterone and Enzalutamide in Advanced Prostate Cancer; Clinical Advances in Hematology & Oncology – May 2016 – Volume 14, Issue 5;  
5. Armstrong et al., 2019, JCO 37: 1120-  
6. Zhang et al., 2015, Cell Cycle 14:13, 2142—2148;  
Phase 2 Clinical Trial in mCRPC  
Disease Control Assessed by PSA Stabilization or Decline

<table>
<thead>
<tr>
<th>Dosing Regimen Arm A</th>
<th>Dosing Schedule</th>
<th>Duration</th>
<th>Efficacy Endpoint</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Onvansertib – 24 mg/m² Days 1-5 (21-Day Cycle) + Abiraterone daily</td>
<td>4 Cycles = 12 Weeks</td>
<td>Disease Control PSA Stabilization or Decline</td>
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<th>Dosing Regimen Arm B</th>
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<tr>
<td></td>
<td>Onvansertib – 24 mg/m² Days 1-5 (14-Day Cycle) + Abiraterone daily</td>
<td>6 Cycles = 12 Weeks</td>
<td>Disease Control PSA Stabilization or Decline</td>
</tr>
</tbody>
</table>

**Efficacy Endpoints:** Effect of onvansertib in combination with Zytiga®/prednisone on disease control assessed by prostate-specific antigen (PSA) decline or stabilization pre- and post-treatment

**Safety Endpoint:** Safety and tolerability of onvansertib in combination with Zytiga®/prednisone

**Exploratory Endpoints:** Target inhibition of PLK1, evaluation of relevant biomarkers and correlation with patient response and genomic profile
Early PSA Response Observed
Addition of Onvansertib to Daily Zytiga®

6 patients have completed 4 cycles (3 months) of treatment with onvansertib + abiraterone
2 of 6 patients had observed declines in PSA levels after dosing with onvansertib
To-date, 1 patient in Arm A has achieved the efficacy endpoint of disease stabilization based on PSA levels (primary endpoint)

PSA trajectory in patient achieving primary efficacy endpoint changed from 100% increase (16.05 ng/ml to 34.23 ng/ml) in the 60 days prior to adding onvansertib to only an 8.4% increase during 84 days on treatment
Tumor assessed at Cycle1 Day 1 as a variant known as AR-V7, considered an aggressive tumor that is resistant to anti-androgen therapy

CT scan at end of 12 weeks (efficacy endpoint) indicates ~30% tumor shrinkage
Phase 1b/2 Trial: metastatic Colorectal Cancer
Onvansertib Market Opportunity in mCRC
Only 5% Response to Current Second-Line Therapies

**Disease Burden**

140,000 new cases of CRC in 2018

65% 5-year survival

~51,000 deaths per year from mCRC

**Treatment**

Tumor biomarkers drive therapy decisions for 1st-line mCRC therapy

~50% mCRC has RAS (KRAS) mutation

Standard-of-care is chemotherapy (FOLFOX/FOLFIRI)

2nd-line therapies have ~5% response rate in mCRC

**Opportunity**

Onvansertib + irinotecan (FOLFIRI) significantly reduces tumor growth

KRAS mutation is biomarker for onvansertib sensitivity

Research partnership with Nektar Therapeutics

$9.0 billion global market, expected to grow to $11.0 billion by 2025

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Synergy in Combination with Irinotecan
Preclinical Data Demonstrates Reduced Tumor Growth

► Combination of onvansertib with irinotecan (FOLFIRI) significantly reduces tumor growth compared to either drug alone

► In 3 independent models tested, onvansertib induced maximal tumor regression of ~84% compared to vehicle

► Kras mutation is a biomarker for onvansertib sensitivity

► KRAS mutated NIH3T3 cells showed higher sensitivity to onvansertib compared with KRAS wild-type cells

1Investigator Brochure, Data-on-file, Trovagene
Phase 1b/2 Clinical Trial in mCRC
Objective Response Rate (ORR) in Second-Line Treatment

Phase 1b: Dose escalation to assess safety and identify recommended Phase 2 dose

- Administered orally, once-daily on Days 1-5 every 14-days (2 courses per 28-day cycle)

Phase 2: Assess safety and preliminary antitumor activity

- **Efficacy Primary Endpoint:** Objective response rate (ORR) in patients who receive at least 1 cycle (2 courses) of onvansertib in combination with FOLFIRI and bevacizumab

- **Efficacy Secondary Endpoint:** Preliminary efficacy defined as complete response (CR) plus partial response (PR) plus stable disease (SD)
Phase 1b/2 Trial: Acute Myeloid Leukemia
# Onvansertib Market Opportunity in AML
Providing a New Treatment for Relapsed/Refractory Patients

## Disease Burden

<table>
<thead>
<tr>
<th>20,000 new cases annually</th>
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<tbody>
<tr>
<td>5-year survival rate of only 25%¹</td>
</tr>
</tbody>
</table>

Aggressive blood cancer that usually occurs in the elderly¹

## Treatment

Today’s standard-of-care for elderly AML patients is *Venclexta*® plus azacytidine or decitabine

Patients develop resistance to *Venclexta*® in ~11 months with no viable treatment options²

## Opportunity

Onvansertib + chemotherapy has significant activity in AML models³

Onvansertib induces cell death in AML model insensitive to *Venclexta*®⁴

Onvansertib + decitabine will be evaluated as treatment in *Venclexta*® resistant patients

$1.0 billion global market by 2023⁵

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Phase 1b/2 Clinical Trial in AML
Onvansertib + Low-Dose Cytarabine or Decitabine

**Phase 1b**: Dose escalation to assess safety and identify recommended Phase 2 dose

- Administered orally, once-daily on Days 1-5 of each cycle (21-28 days)

**Phase 2**: Assess safety and preliminary antitumor activity

- **Efficacy Endpoints**: Rate of complete response (CR + CRi) defined as morphologic leukemia-free state (MLF)
- **Exploratory Endpoints**: Evaluation of pharmacodynamic and correlative biomarkers
Patients Achieving Complete Response
Onvansertib is Safe and Well Tolerated

- Of the 26 patients evaluable for safety, 19 had an evaluable bone marrow biopsy to assess efficacy.
- Preliminary efficacy in the evaluable population includes 3 patients achieving complete response (CR) and 1 patient achieving complete response with incomplete hematologic recovery (CRi).

**Onvansertib + Decitabine**

- [Graph showing % change from baseline for Onvansertib + Decitabine]

**Onvansertib + LDAC**

- [Graph showing % change from baseline for Onvansertib + LDAC]

- **Data Labels** represent onvansertib dose (mg/m²)

- **Legend**:
  - Stable disease (SD)
  - Complete response (CR)
  - Complete response with incomplete hematological recovery (CRi)
  - Progression free survival (PFS)
  - Morphological Leukemic Free State (MLFS)
  - Complete response (CR)

- **Note**: Data Labels represent onvansertib dose (mg/m²).
Biomarker Evaluates Inhibition of PLK1 Identifies Patients Most Likely to Respond to Treatment

Blood test examines the extent that onvansertib inhibits PLK1 enzymatic activity (target engagement) by assessing the phosphorylated status of TCTP within circulating leukemic blast cells.

- Pre-dose sample
- Patient receives a single dose of onvansertib
- 3h post-dose sample
- Assess target engagement

Current method: Western-Blot

Method in development: immuno-PCR based technology

<table>
<thead>
<tr>
<th>Onvansertib</th>
<th>pTCTP</th>
<th>TCTP</th>
</tr>
</thead>
<tbody>
<tr>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

- pTCTP antibody (capture)
- pTCTP ANTIGEN
- 59-mer dsDNA
- PCR* x 30 cycles
- QUANTIFY DNA LABEL
Biomarker to Assess Inhibition of PLK1
Correlation of Biomarker\(^+\) Patients with Treatment Response

PLK1 inhibition can be monitored in patients through pTCTP status to determine target engagement of onvansertib with PLK1

- **pTCTP as a marker of PLK1 activity:**
  - PLK1 phosphorylates the translational control tumor protein (TCTP) on serine 46
  - pTCTP was identified as a specific marker for PLK1 activity in vivo in preclinical models

- The comparative change in pTCTP status between pre-dose and 3 hours post-dose is being assessed
Biomarker-Positive Patients Significantly Correlated with Treatment Response

- PLK1 inhibition by onvansertib (target engagement) is correlated with higher response to treatment
  - Patients with target-engagement had a significantly greater decrease in BM blasts compared to patients with no target-engagement
  - 6 out of the 9 patients with target-engagement had a decrease in BM blasts ≥ 50%
  - Among the 4 patients with objective responses, 3 had target engagement (≥ 50% decrease in pTCTP) and 1 had a 40% decrease in pTCTP

* Patient sample showed a 40% reduction in pTCTP
Robust, diversified pipeline with single molecule, onvansertib, addressing multiple cancer indications, each with significant medical need for new treatment options

Preclinical data demonstrating efficacy of onvansertib in combination with standard-of-care drugs, expanding therapeutic and partnership opportunities

Encouraging initial efficacy data from ongoing clinical trials with additional data readouts in 2019-2020

Precision Cancer Medicine™ approach and integration of biomarkers to target treatment for patients most likely to respond

Experienced team with proven oncology drug development track record
Thank You

For additional information please contact: ir@trovagene.com