Certain statements in this presentation are forward-looking within the meaning of the Private Securities Litigation Reform Act of 1995. These statements may be identified by the use of words such as "anticipate," "believe," "forecast," "estimated" and "intend," or other similar terms or expressions that concern Trovagene's expectations, strategy, plans or intentions.

These forward-looking statements are based on Trovagene's current expectations and actual results could differ materially. There are a number of factors that could cause actual events to differ materially from those indicated by such forward-looking statements. While the list of factors presented in the 10-K is considered representative, no such list should be considered to be a complete statement of all potential risks and uncertainties. Unlisted factors may present significant additional obstacles to the realization of forward-looking statements. Forward-looking statements included herein are made as of the date hereof, and Trovagene does not undertake any obligation to update publicly such statements to reflect subsequent events or circumstances.
Company At-A-Glance

Clinical-stage oncology therapeutics company, developing **onvansertib**, an oral and highly-selective Polo-like Kinase 1 (PLK1) inhibitor

- Selectively targets PLK1, a proven therapeutic target; overexpressed in most cancers
- Stops division of cancer cells while limiting impact to normal cells
- Proven safety and preliminary efficacy in 3 clinical programs (mCRC, mCRPC, AML)
- Presentation of efficacy data from all 3 Phase 2 clinical trials in 2020

San Diego, CA

Nasdaq: TROV

**Clinical Development Plan:** Complete Phase 2 clinical trials of onvansertib in combination with standard-of-care therapies, in colorectal cancer, prostate cancer and acute myeloid leukemia, and advance to registrational trials
**Ovansertib**

1\textsuperscript{st}-in-class, 3\textsuperscript{rd}-generation, safe and well-tolerated, oral PLK1 inhibitor that selectively targets the PLK1 enzyme and blocks cancer cell division

**Clinical Efficacy Demonstrated**

3 ongoing clinical trials with demonstrated efficacy in patients who have developed resistance to standard-of-care or who have relapsed disease

**Predictive Biomarkers**

Assessment of response to treatment derived from a simple blood test

**Validating Combination Clinical Trials**

- KRAS-mutated metastatic colorectal cancer (mCRC): onvansertib + FOLFIRI\textsuperscript{®}/Avastin\textsuperscript{®}
- metastatic castrate-resistant prostate cancer (mCRPC): onvansertib + Zytiga\textsuperscript{®}
- acute myeloid leukemia (AML): onvansertib + decitabine

**Established Manufacturing and Drug Supply**

FDA approved, GMP facility for production of raw material and finished drug
Significant Value Creation in 2020

- Efficacy Data
- From 3 Ongoing Phase 2 Trials
- In 2020
# 2020 Key Inflection Points

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Onvansertib is a Platform for Value Creation

- **Clinical Programs Based on Scientific Rationale**: supported by preclinical and synergy data, and integration of biomarkers to rapidly assess response to treatment

- **Addressing Significant Medical Needs**:
  - overcome resistance to standard-of-care drugs
  - extend duration of response and progression-free survival

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<tr>
<th>Onvansertib Solid Tumors (2 INDs in Place)</th>
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<th>Next Milestone</th>
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<td>Onvansertib + FOLFIRI®/Avastin® in KRAS-mutated colorectal cancer</td>
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<td>mCRPC</td>
<td>Onvansertib + Zytiga®/prednisone in metastatic castrate-resistant prostate cancer</td>
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<td>AML</td>
<td>Onvansertib + decitabine in relapsed or refractory acute myeloid leukemia cancer</td>
<td>Q2 2020 AACR, EHA</td>
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IND = Investigational New Drug; mCRPC = metastatic castrate-resistant prostate cancer; mCRC = metastatic colorectal cancer; TNBC = triple-negative breast cancer; AML = acute myeloid leukemia
Onvansertib Mechanism of Action

Inhibition of PLK1 causes arrest of cell division and subsequent cell death\(^1\)


Synergistic in combination with chemotherapies and targeted therapeutics

\[\text{Taxol\textsuperscript{®} (paclitaxel)}\]
\[\text{Avastin\textsuperscript{®} (bevacizumab)}\]
\[\text{Zytiga\textsuperscript{®} (abiraterone)}\]
\[\text{Cytarabine}\]
\[\text{Doxorubicin}\]
\[\text{Gemzar\textsuperscript{®} (gemcitabine)}\]
\[\text{Velcade\textsuperscript{®} (bortezomib)}\]
\[\text{Camptosar\textsuperscript{®} (Irinotecan)}\]
\[\text{Beleodaq (belinostat)}\]
\[\text{Quizartinib}\]
\[\text{Venclexta\textsuperscript{®} (venetoclax)}\]

\[\text{Onvansertib Synergistic in Combination with SOC Therapies}\]
Optimal Attributes for a Safe and Effective Drug
Indication:
metastatic KRAS-Mutated Colorectal Cancer (mCRC)
Improving Response and Progression-Free Survival

Metastatic Colorectal Cancer (mCRC)

- Only a 5% response rate to standard-of-care FOLFOX/FOLFIRI\(^1\)
- Onvansertib + FOLFIRI\(^{®}\) significantly reduces tumor growth\(^3\)
- Biomarkers drive therapy decisions\(^2\)
- KRAS mutation is a biomarker for clinical response to onvansertib
- KRAS mutation in 50% of mCRC\(^2\)

Establishing a Successful Path Forward:

- Positive results from Phase 1b/2 trial may provide an opportunity for Phase 2b registrational trial and Fast Track Designation
- Biomarker increases likelihood of success by enabling rapid, quantitative assessment of KRAS mutation and patient response to treatment

\(^1\)King et al, Frontline Strategies for Metastatic CRC, 2016, Amer J Hem/Onc; Loree&Kopetz; \(^2\)Van Custem E, Borràs JM, Castells A et al. Improving outcomes in colorectal cancer. Where do we go from here? Eur J Cancer. 2013 Jul; 49(11): 2476–85; \(^3\)Recent Developments in treatment of mCRC, 2017, Ther Adv Med Onc; \(^4\)Investigator Brochure, Data-on-file, Trovagene
Rationale for Onvansertib + FOLFIRI®/Avastin® in KRAS-Mutated Metastatic CRC

► Synthetic Lethality
  – CRC tumor cells harboring KRAS mutation are more vulnerable to cell death with PLK1 inhibition
  – Tumor cell viability is more sensitive to onvansertib in KRAS-mutated vs KRAS wild-type isogenic cell line

► Synergy
  – Onvansertib + irinotecan (the “IRI” in FOLFIRI) are synergistic in CRC cell lines
  – Combination demonstrated significantly greater tumor growth inhibition than either drug alone

► Proof-of-Concept Clinical Response
  – Phase 1 trial in solid tumors: 3 of 5 patients with stable disease had KRAS mutation; 2 in CRC and 1 in pancreatic cancer
Demonstrating Clinical Benefit in KRAS-Mutated CRC as New Second-Line Treatment

Trial Design: Phase 1b/2, multi-center, open label trial in mCRC

Onvansertib + FOLFIRI®/Avastin® (bevacizumab) ➔ Phase 1b (n=18) ➔ Phase 2 (n=26) ➔ Onvansertib administered orally, days 1-5 every 14-days (2 courses of treatment in every 28-day cycle)

Efficacy Endpoints:
Primary: overall response in patients who receive ≥1 cycle (2 courses) of treatment
Correlative Biomarker: decreases in KRAS mutation burden and response to treatment

Current Standard-of-Care FOLFIRI®/Avastin® Clinical Response: overall response is ~5%; median progression-free survival (PFS) is ~6 months

Onvansertib: Achieving Clinical Success
• 5 of 26 (~20%) patients achieve clinical response confirmed by radiographic scan
• Patient median progression-free survival (PFS) of >6 months

King et al, Frontline Strategies for Metastatic CRC
Clinical Data Shows Onvansertib Effectively Targets KRAS Mutations in CRC

- To-date, KRAS mutation decreased in all 6 patients completing their 1st cycle of onvansertib treatment

- Mutations identified in these patients account for nearly 100% of those associated with CRC
  - G12D (39%), G12V (22%), G13D (18%), G12C (8%), G12A (6%)

- Other drugs in development target only G12C which accounts for <10% of the KRAS mutations in CRC

---

1Jones et al. Specific Mutations in KRAS Codon 12 Are Associated with Worse Overall Survival in Patients with Advanced and Recurrent Colorectal Cancer; BJC Feb. 2017
Decreases in Plasma KRAS Mutation in All Patients Treated with Onvansertib

► Changes in plasma KRAS mutation level is an early marker for therapeutic response

► Dose level 1: 4 patients had detectable KRAS mutant ctDNA at baseline; in all 4 patients KRAS was undetectable within the 1st cycle of treatment

► Dose level 2: 2 patients treated to-date had detectable KRAS mutant ctDNA at baseline; in 1 patient KRAS was undetectable within the 1st cycle of treatment

Tie et al., 2015, Annals of Oncology 26: 1715–1722
Radiographic scans performed at 8 weeks showed tumor decrease and clinical benefit in 100% (n=5) of evaluable patients treated with onvansertib 12 mg/m² (n=5); 1 patient achieved PR, 4 patients achieved SD.

Radiographic responses were confirmed at 16 weeks with further tumor shrinkage in all patients; 3 patients had a >25% decrease; 1 patient is proceeding to curative surgery.
Indication:
metastatic Castrate-Resistant Prostate Cancer (mCRPC)
Overcoming Resistance and Extending Efficacy

Metastatic Castrate-Resistant Prostate Cancer (mCRPC)

- Resistance develops to standard-of-care therapy, Zytiga® and Xtandi®, within 9-15 months
- Onvansertib + Zytiga® are synergistic in combination
- Combination significantly increase arrest of cell division
- Up to 40% AR-V7 resistance; very aggressive mutation and no effective treatment options

Establishing a Successful Path Forward:

- Positive results from Phase 2 trial may provide an opportunity for a Phase 2b registrational trial
- Proactively assessing AR-V7 enables correlation of status (+/-) with response to onvansertib treatment
- Effective treatment of AR-V7+ patients could lead to Breakthrough Designation

Rationale for Onvansertib + Zytiga® in Metastatic Castrate-Resistant Prostate Cancer (mCRPC)

- Onvansertib induces synergistic cell death and mitotic arrest in combination with abiraterone in a castration-resistant prostate cancer (CRPC) model (C4-2).

- Combination of PLKi and abiraterone blocks tumor growth and PSA increase in a CRPC xenograft model\(^3\).

Onvansertib + Zytiga® (abiraterone) demonstrates synergy in mCRPC model (C4-2)\(^1\)

Onvansertib + Zytiga® (abiraterone) significantly increases mitotic arrest\(^1\)

\(^1\)Patterson & Yaffe, 2019, MIT
Androgen-Receptor Variant 7 (AR-V7)

- AR-V7+ detection in CTCs is associated with abiraterone resistance.
- AR-V7+ has a shorter progression-free survival and overall survival in mCRPC intent-to-treat with abiraterone.
- Combination of abiraterone and PLK inhibitor (PLKi) reduces AR and AR-V7 protein expressions in CRPC cell lines.

**Figure:** PLK1 Inhibitor and Abiraterone Blocks Tumor Growth and PSA Increase in an AR-V7 Positive CRPC Model (LuCaP35CR)
Overcoming Resistance and Extending Efficacy

Trial Design: Phase 2 multi-center, open label trial in mCRPC

<table>
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<tr>
<th>Arm</th>
<th>Dosing Regimen</th>
<th>Duration</th>
<th>Efficacy Endpoint</th>
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<tr>
<td>Arm A</td>
<td>Onvansertib – 24 mg/m² Days 1-5 + Abiraterone daily</td>
<td>4 Cycles = 12 Weeks</td>
<td>Disease Control PSA Stabilization or Decline</td>
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<tr>
<td>Arm B</td>
<td>Onvansertib – 18 mg/m² Days 1-5 + Abiraterone daily</td>
<td>6 Cycles = 12 Weeks</td>
<td>Disease Control PSA Stabilization or Decline</td>
</tr>
<tr>
<td>Arm C</td>
<td>Onvansertib – 12 mg/m² Days 1-14 + Abiraterone daily</td>
<td>4 Cycles = 12 Weeks</td>
<td>Disease Control PSA Stabilization or Decline</td>
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Efficacy Endpoint – Internationally Recognized Prostate Cancer Working Group (PCWG)
Primary: disease control evaluated as PSA decline or stabilization (PSA rise <25% over baseline)
Correlative Biomarker: androgen receptor variant 7 (AR-V7) status and correlation with patient response
Current Standard-of-Care Zytiga® Clinical Response: median radiographic progression-free survival (RPFS) is ~7 months

Onvansertib: Achieving Clinical Success
• 6 of 32 (~20%) patients achieve primary efficacy endpoint of disease control at 12 weeks (PSA stabilization or decrease); confirmed by radiographic scan
• Patients achieve median RPFS of ≥7 months

Note: radiographic assessment by RECIST v1.1 [CR = disappearance of all target lesions, PR = ≥30% decrease, PD = ≥20% increase, SD = does not meet criteria for PR nor PD]
1Hussain et al., ESMO 2019
Efficacy in Abiraterone-Resistant Patients

- Overall, 63% (12 of 19) patients achieved partial response (PR) or stable disease (SD) following 12 weeks of treatment with onvansertib + abiraterone. Response to treatment was evaluated based on PSA values (primary endpoint) and radiographic scans.

- **Arm B (n=5): Onvansertib Days 1-5 in a 2-week dosing schedule**
  - 80% (4 of 5) patients had SD at 12 weeks, with 3 patients achieving the efficacy endpoint (PSA stabilization) and 3 patients remain on treatment
  - 60% (3 of 5) patients have or had progression-free survival of >7 months

- **Arm A (n=14): Onvansertib Days 1-5 in a 3-week dosing schedule**
  - 57% (8 of 14) patients had SD or PR at 12 weeks, with 5 patients achieving the efficacy endpoint (PSA stabilization) and 4 patients remain on treatment
  - 21% (3 of 14) patients have or had progression-free survival; 2 patients remain on treatment for >1 year
Onvansertib-Induced CTC Decrease is Associated with Progression-Free Survival

► CTC count, reported as favorable or unfavorable (<5 versus ≥5 CTC/7.5mL of blood, respectively) is a prognostic factor for survival in CRPC and the conversion from unfavorable to favorable is associated with improved survival.\(^7\)

► At baseline, 25 (78%) patients had unfavorable CTC count with median of 19 CTC/7.5mL

► 10 of the unfavorable patients were re-analyzed after 12 weeks of treatment
  - 5 (50%) patients had a ≥80% CTC decrease, including 2 AR-V7+ patients (01-024 and 01-025)
  - 4 (40%) patients converted from unfavorable to favorable CTC level (<5 CTC/7.5mL)
  - 3 (30%) patients had no detectable CTC
  - Median time on treatment for patients with decrease CTC (n=5) is 7 months to-date, with 4 patients remaining on treatment

► Conversely, median time on treatment for patients with increase CTC (n=5) was 5 months, and none of these patients remain on treatment

% Change in CTC at 12-weeks vs baseline in patients with unfavorable CTC level at baseline

- Favorable CTC level at 12 weeks
- Remain on treatment
Efficacy Observed in Patients with Abiraterone-Resistant AR Alterations

- AR mechanisms of resistance to abiraterone include the expression of the constitutively active AR splice variant AR-V7 and the AR gain-of-function point mutation T878A.

- Among the 19 patients who completed the 12-week treatment (Arm A + B):
  - 5 patients were AR-V7+ at baseline
  - 2 patients had AR T878A mutations at baseline

- Onvansertib showed efficacy in patients with AR alterations (N=7):
  - 6 (86%) patients had a decrease in PSA levels with the addition of onvansertib
  - 4 (57%) patients had SD or PR at 12 weeks with 3 (43%) patients achieving the primary efficacy endpoint
  - 3 patients have or had progression-free survival of >7 months, 2 patients remain on treatment

Best PSA response in AR-V7+ and AR T878A patients
Indication:
Acute Myeloid Leukemia (AML)
Addressing the Need for New Treatment Options

Relapsed Acute Myeloid Leukemia (AML)

- 5-year survival rate of only 25%\(^1\)
- Standard-of-care is venetoclax + azacytidine or decitabine; resistance develops in \(~11\) months\(^2\)
- Onvansertib induces cell death in AML model resistant to Venclexta\(^3\)

Establishing a Successful Path Forward:

- Positive results from Phase 2 trial and Orphan Drug Designation may provide an opportunity for a Phase 2b registrational trial
- Opportunity to treat patients who relapse following first-line venetoclax
- Biomarker identifies patients most likely to respond, increasing likelihood of success

\(^1\)National Cancer Institute SEER 2016; \(^2\)DiNardo et al, Blood, 2019; \(^3\)Valsasina et al., Mol Cancer Ther; 11(4) April 2012; \(^3\)Trovagene, data on file
Providing a New, Safe and Effective Treatment

**Trial Design: Phase 2 multi-center, open label trial in AML**

- **Onvansertib + Decitabine** → **Relapsed or Refractory Patients (n=32)** → **Onvansertib 60mg/m² Days 1-5 (21-28 Day Cycle)**

**Efficacy Endpoint**

**Primary:** safety and preliminary efficacy

**Correlative Biomarker:** Assess PLK1 inhibition (target engagement) by measuring changes in the PLK1 substrate pTCTP; evaluate predictive biomarkers associated with response to treatment

**Current Standard-of-Care Clinical Response:** Hypomethylating agents (decitabine and azacytidine) is 16.3% and IDH Inhibitors, ivosidenib (Agios), is 30.4%; enasidenib (Celgene) is 26.6%

**Onvansertib: Achieving Clinical Success**

- 10 of 32 (~30%) achieve complete response (CR + CRi)
- Median overall survival of >2 months for relapsed/refractory AML patients

Phase 1b Completed Trial Efficacy Summary
Patients Treated with ≥ 1 Cycle (n=36)

At the 4 higher dose levels (27 to 90 mg/m²), CR/CRi was observed in:
- 5 of 16 (31%) patients in the decitabine Arm
- Median time to achieve CR/CRi was 4 cycles (range 1-7)
- Durable responses for >7 months

1Jones et al. Specific Mutations in KRAS Codon 12 Are Associated with Worse Overall Survival in Patients with Advanced and Recurrent Colorectal Cancer; BJC Feb, 2017
Strong Patent Portfolio

► Core Technology: 3 Issued Patents to 2030 in US, Europe and Asia with extension to 2035 in US
  – Compound (onvansertib): US 8614220
  – Salt forms of onvansertib: US 8648078
  – Combinations with anti-neoplastic compounds: US 8927530

► Evergreening: Combination Therapy
  – Exclusive license from MIT for 2 US issued patents with broad method claims for combination of PLK inhibitor + anti-androgen compounds to treat any cancer
    • US 9566280, US 10155006; Expiration 2035

► Evergreening: Biomarkers
  – Method for assessing PLK1 target phosphorylation status for identifying patients to be treated with Plk1 Inhibitors
    • PCT US1948044, Expiration 2039
  – Method for treating patient with a PLK inhibitor when there is a PSA rise
    • Provisional, Expiration 2040
Business Development Strategy

Objective: Joint Development and Commercialization Partnerships

- Financial and clinical support for company-sponsored and/or investigator sponsored (IST) studies
- Maintain rights in North America in part or in whole
- Co-develop and/or out-license specific indications in Japan and Europe
- Optimize development timelines while efficiently managing resources, internal and outsourced

Current Co-Research Collaboration

- Co-research agreement with Nektar Therapeutics to evaluate onvansertib in combination with NKTR-102 in colorectal cancer

Partnering Strategy

- Successful partnership with US pharma/biotech for co-development
- Successful partnership with Japan Pharma for co-development and/or out-licensing
Financials

2019 Raised Capital & Clinical Research Commitment
• $14.5 million

Quarter Ending Cash and Cash Equivalents
• Q4'18 = $11.5M
• Q1'19 = $11.3M
• Q2'19 = $10.8M
• Q3'19 = $9.0M

Estimated Quarterly Cash Burn
• ~$4.0M
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For additional information please contact: ir@trovagene.com