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.
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
Investment Highlights

Ovansertib
1st-in-class, 3rd-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®/Avastin®
- metastatic castrate-resistant prostate cancer (mCRPC): onvansertib + Zytiga®
- 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

## 1H2020 Key Inflection Points

<table>
<thead>
<tr>
<th>Event</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Colorectal Cancer:</strong> Phase 1b Safety and Efficacy Data</td>
<td>ASCO-GI</td>
</tr>
<tr>
<td><strong>Prostate Cancer:</strong> Phase 2 Efficacy Data</td>
<td>ASCO-GU</td>
</tr>
<tr>
<td><strong>Acute Myeloid Leukemia:</strong> Biomarker Data</td>
<td>AACR</td>
</tr>
<tr>
<td><strong>Prostate Cancer:</strong> Phase 2 and Correlative Biomarker Data</td>
<td>AACR</td>
</tr>
<tr>
<td><strong>Acute Myeloid Leukemia:</strong> Phase 2 Efficacy Data</td>
<td>EHA</td>
</tr>
</tbody>
</table>

## 2H2020 Key Inflection Points

<table>
<thead>
<tr>
<th>Event</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prostate Cancer:</strong> Phase 2 Efficacy Data</td>
<td>ESMO</td>
</tr>
<tr>
<td><strong>Colorectal Cancer:</strong> Phase 2 Efficacy Data</td>
<td>ESMO</td>
</tr>
<tr>
<td><strong>Acute Myeloid Leukemia:</strong> Phase 2 Efficacy Data</td>
<td>ESMO</td>
</tr>
<tr>
<td><strong>Prostate Cancer:</strong> Phase 2 Efficacy Data</td>
<td>EMUC</td>
</tr>
<tr>
<td><strong>Colorectal Cancer:</strong> Phase 2 Efficacy Data</td>
<td>ESMO Asia</td>
</tr>
<tr>
<td><strong>Acute Myeloid Leukemia:</strong> Phase 2 Efficacy Data</td>
<td>ASH</td>
</tr>
</tbody>
</table>
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

<table>
<thead>
<tr>
<th>Indication</th>
<th>Preclinical</th>
<th>Phase 1/1b</th>
<th>Phase 2/2b</th>
<th>Next Milestone</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Onvansertib</strong></td>
<td><strong>Solid Tumors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(2 INDs in Place)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mCRC</td>
<td>Onvansertib + FOLFIRI® /Avastin® in KRAS-mutated colorectal cancer</td>
<td></td>
<td></td>
<td>Q2 2020 AACR</td>
</tr>
<tr>
<td>mCRPC</td>
<td>Onvansertib + Zytiga® /prednisone in metastatic castrate-resistant prostate cancer</td>
<td></td>
<td></td>
<td>Q2 2020 AACR</td>
</tr>
<tr>
<td><strong>Onvansertib</strong></td>
<td><strong>Hematologic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(1 IND in Place)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AML</td>
<td>Onvansertib + decitabine in relapsed or refractory acute myeloid leukemia cancer</td>
<td></td>
<td></td>
<td>Q2 2020 AACR, EHA</td>
</tr>
</tbody>
</table>

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

- Zytiga\(^\text{®}\) (abiraterone)
- Avastin\(^\text{®}\) (bevacizumab)
- Cytarabine
- Doxorubicin
- Camptosar\(^\text{®}\) (Irinotecan)
- Beleodaq (belinostat)
- Quizartinib
- Velcade\(^\text{®}\) (bortezomib)
- Gemzar\(^\text{®}\) (gemcitabine)
- Taxol\(^\text{®}\) (paclitaxel)
- Venclexta\(^\text{®}\) (venetoclax)
Optimal Attributes for a Safe and Effective Drug
Indication: Second-Line Treatment of KRAS-Mutated Metastatic Colorectal Cancer (mCRC)

Phase 1b/2 open-label trial of onvansertib + FOLFIRI/bevacizumab

Principal Investigator
Dr. Heinz-Josef Lenz
Improving Response and Progression-Free Survival

Metastatic Colorectal Cancer (mCRC)

- Only a 4% response rate to second-line standard-of-care chemotherapy + bevacizumab\(^1\)
- Onvansertib + FOLFIRI\(^2\) significantly reduces tumor growth
- Biomarkers drive therapy decisions\(^3\)
- KRAS mutation is a biomarker for clinical response to onvansertib\(^4\)
- KRAS mutation in 50% of mCRC\(^5\)

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 assessment of KRAS mutation as an early predictor of response to treatment

Clinical Outcomes in Patients Receiving Second-Line Treatment – Chemotherapy + bevacizumab¹

<table>
<thead>
<tr>
<th>Outcome Measure</th>
<th>Chemo + Bevacizumab</th>
<th>Chemo + Bevacizumab KRAS Mutated CRC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progression-Free Survival</td>
<td>5.7 months</td>
<td>5.5 months</td>
</tr>
<tr>
<td>Overall Survival</td>
<td>11.2 months</td>
<td>10.4 months</td>
</tr>
<tr>
<td>Complete Response</td>
<td>1%</td>
<td>1%</td>
</tr>
<tr>
<td>Partial Response</td>
<td>4%</td>
<td>3%</td>
</tr>
<tr>
<td>Overall Response Rate</td>
<td>5%</td>
<td>4%</td>
</tr>
</tbody>
</table>

- In patients with KRAS-mutated colorectal cancer, the overall response rate is 4%.
- Progression-free survival is 5.5 months.

¹Kubicka et al, Annals of Oncology 2013; 2342–2349
Multiple KRAS Mutation Subtypes are Present in Each Cancer Type: Subtype Prevalence Also Differs

Onvansertib is Agnostic to KRAS Subtype

Colorectal Cancer
- G12D: 22%
- G12V: 18%
- G13D: 8%
- G12C: 6%
- G12S: 6%
- G12A: 5%
- Q61H: 5%
- G12R: 1%

Lung Cancer
- G12C: 21%
- G12V: 24%
- G12D: 3%
- G12A: 3%
- G13D: 2%
- G12S: 8%
- G12R: 39%

Pancreatic Cancer
- G12D: 48%
- G12V: 32%
- G12R: 3%
- G12C: 3%
- G12S: 3%
- G12A: 13%
- G13D: 1%

Biliary Cancer
- G12D: 55%
- G12V: 22%
- G12S: 11%
- G12C: 8%
- G12A: 4%
- G12R: 77%
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\(^1\)
  – KRAS-mutated cells are more sensitive to onvansertib than KRAS wild-type isogenic cells\(^2\)

► Synergy
  – Onvansertib + irinotecan (the “IRI” in FOLFIRI) are synergistic in CRC cell lines\(^3\)
  – 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\(^4\)

\(^1\) Luo J, Elledge SJ, Cell 2009; \(^2\) Trovagene, Investigator Brochure, 2019; \(^3\) Valsasina et al., Mol Cancer Ther 2012; \(^4\) Weiss et al, Invest New Drugs, 2017
Targeting KRAS-Mutated mCRC with Onvansertib

**Synthetic Lethality**

- Mutant KRAS is an oncogene that drive aggressive growth in many cancer types.

- Majority of KRAS mutations are undruggable; need to find “partners in crime” that if inhibited will kill tumor cells that harbor a KRAS mutation. This is done by screening every human gene (>20,000) in KRAS-mutated and wild-type cells to identify synthetic lethal partners that specifically induce cell death in KRAS-mutant, but not wild-type.

- A genome-wide RNAi screen was completed to identify what gene(s) is necessary for KRAS-mutated tumor cells to drive tumor growth. **PLK1 was identified as a synthetic lethal in KRAS mutant colorectal cancer cells**

**How Synthetic Lethality Works**

\[
\begin{align*}
\text{KRAS wild type} & \quad + \quad \text{PLK1} & = & \quad \text{Viable} \\
\text{KRAS wild type} & \quad + \quad \text{PLK1} & = & \quad \text{Viable} \\
\text{KRAS mutant} & \quad + \quad \text{PLK1} & = & \quad \text{Viable} \\
\text{KRAS mutant} & \quad + \quad \text{PLK1} & = & \quad \text{Lethal}
\end{align*}
\]


---

© trovagene ONCOLOGY
Tumor Cells Harboring KRAS Mutations are More Vulnerable to Onvansertib

- KRAS wild-type cells (normal) are minimally affected by onvansertib whereas KRAS-mutated cells are preferentially vulnerable to onvansertib at drug concentrations achieved in patients (0.1μM).

- Specifically, mitotic block which leads to cell death is observed in KRAS-mutated cells at onvansertib concentrations that do not affect wild-type cells.

¹Trovagene, Investigator Brochure, 2019
Proposed Mechanism of Lethality for KRAS-Mutants + Onvansertib

- KRAS mutants are experiencing mitotic stress and exacerbating this stress in particular ways such that interference with PLK1 leads to stress overload and tumor cell death.

- Proposed mechanism: KRAS mutant and onvansertib have dual block of the anaphase promoting complex (APC/C) - which is crucial for mitosis to occur - (metaphase → anaphase).

Luo J, Elledge SJ, Cell 2009
Anti-tumor Activity of Onvansertib as Single Agent and Synergy in Combination with Irinotecan

Anti-tumor activity of onvansertib in a KRAS-mutant CRC xenograft model (HCT116) as single agent and in combination with irinotecan

Demonstrating Clinical Benefit in KRAS-Mutated CRC as New Second-Line Treatment Option

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

1 CYCLE = 28 days

<table>
<thead>
<tr>
<th>14 days</th>
<th>14 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 2 3 4 5 6-14</td>
<td>1 2 3 4 5 6-14</td>
</tr>
</tbody>
</table>

- **Onvansertib**
- **FOLFIRI + bevacizumab**

**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

**Standard-of-Care FOLFIRI®/Avastin® Clinical Response in 2nd Line KRAS-Mutated CRC Tumors:**
overall response is 4%; median progression-free survival (PFS) is 5.5 months

What is a clinically meaningful result:
- ≥ 5 of 26 (~20%) patients achieve clinical response confirmed by radiographic scan
- Patients achieve median progression-free survival (PFS) of ≥ 6 months

1Kubicka et al, Annals of Oncology 2013; 2342–2349
Clinical Data Shows Onvansertib Effectively Targets Multiple KRAS Mutation Subtypes in CRC

To date, tumor shrinkage observed in KRAS mutations G12A, G12V, G12D, G13D which make up 85% of KRAS subtypes in CRC

Other drugs in development target only the KRAS G12C mutation, which accounts for ~8% of the KRAS mutations in CRC

Colorectal Cancer¹

- G12D: 1%
- G12V: 6%
- G13D: 6%
- G12C: 39%
- G12S: 18%
- G12A: 22%
- G12R: 8%

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
Response to Onvansertib Correlates with Decreases in KRAS Mutations to Undetectable Levels in Plasma

- Decreases in plasma KRAS mutation level has been demonstrated to be an early marker for therapeutic response (confirmed by subsequent radiographic scans)\(^1\)
- Lower limit of sensitivity for KRAS assays is 0.001%\(^2\)
- All 5 patients with undetectable levels had subsequent tumor shrinkage

\(^1\)Tie et al., 2015, Annals of Oncology 26: 1715–1722; \(^2\)BioRad Droplet Digital Assays
Radiographic and Durability of Response (as of January 23, 2020)

5 patients evaluable for response

- 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 response was confirmed at 16 weeks with further tumor shrinkage in all patients; 3 patients had a >25% decrease and 1 patient (02-005) is proceeding to curative surgery
Conclusions – Phase 1b (as of January 23, 2020)

- The 1st dose level (onvansertib 12 mg/m$^2$) was cleared for safety; the 2nd dose level (onvansertib 15 mg/m$^2$) is fully enrolled with no DLTs reported in the 2 patients treated to-date.

- Clinical benefit (SD + PR) was observed in 100% of the evaluable patients (n=5, dose level 12 mg/m$^2$):
  - 1 patient achieved PR (>30% decrease in tumor from baseline)
  - 4 patients achieved SD (11% to 28% decrease in tumor from baseline)
  - Radiographic responses were confirmed at 16 weeks with further tumor shrinkage in all patients; 3 patients had a >25% decrease
  - All patients have been on treatment >4 months and remain on treatment; 1 patient is proceeding to curative surgery

- 4 of the 5 patients evaluable for efficacy had a detectable KRAS mutant at baseline:
  - In all those patients (n=4), KRAS mutant decreased to undetectable level within the 1st cycle
  - KRAS mutant decrease to undetectable level preceded tumor shrinkage, supporting the predictive value of liquid biopsy
Indication:
metastatic Castrate-Resistant Prostate Cancer (mCRPC)

Principal Investigator
Dr. David Einstein
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

Underlying Mechanism of Action (MOA) for Onvansertib + Zytiga® in CRPC

**Onvansertib MOA:**

Inhibits tumor cell division (mitosis) by inducing G2/M arrest

---

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

Onvansertib + Zytiga® (abiraterone) significantly increase mitotic arrest

---

1Patterson & Yaffe, 2019, MIT
PLK1 Inhibition + Abiraterone Efficacy in mCRPC Model

PLK1 Inhibitor and Abiraterone Blocks Tumor Growth and PSA Increase in an AR-V7 Positive CRPC Model (LuCaP35CR)

Combination of PLK1 inhibition + abiraterone decreases tumor growth and demonstrates a decrease in PSA within an AR-V7 model

Zhang et al., 2014, Cancer Res
**Phase 2 Clinical Trial in mCRPC**
**Disease Control Assessed by PSA Stabilization**

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

<table>
<thead>
<tr>
<th>Arm</th>
<th>Dosing Regimen</th>
<th>Duration</th>
<th>Efficacy Endpoint</th>
</tr>
</thead>
<tbody>
<tr>
<td>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>
</tr>
<tr>
<td>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>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>
</tr>
</tbody>
</table>

**Eligibility Criteria:** initial resistance to Zytiga; 2 consecutive rises in PSA levels

**Efficacy Endpoint – Internationally Recognized Prostate Cancer Working Group (PCWG)**
**Primary:** disease control evaluated as PSA decline or stabilization (PSA rise <25% over baseline)

**What is a clinically meaningful result:**
- ≥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 ≥6 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.

**Evaluation of PSA and Radiographic Response At 12-Weeks**

- **Arm A**: 36% PSA Response, 60% Radiographic Response
- **Arm B**: 57% PSA Response, 80% Radiographic Response

**Days of treatment**
- 0 100 200 300 400 500

**Patients**
- Met PSA efficacy endpoint
- Radiographic assessment
- AR-V7+ alterations
- AR T878A
- Ongoing
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.

- 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)

Principal Investigator
Dr. Amer Zeidan
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%

**What is a clinically meaningful result:**
- 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)

Decitabine Arm

- 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
- 4 of the 6 patients remain on treatment and in remission
  - Duration of CR/CRi is respectively: 1.5 – 7 – 8 and 11.5 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
Conclusions
Phase 1b Study of Onvansertib in AML

► Safety: onvansertib treatment was well tolerated
  – MTD/RP2D was established at 60 mg/m² in both arms and no DLT was observed through this dose level
  – Onvansertib-related toxicities were primarily on-target hematological events, in accordance with its mechanism of action and prior Phase 1 clinical study

► Efficacy: complete response (CR/CRi) was observed in 6 patients
  – At a wide range of onvansertib doses: 27 mg/m² (2), 40 mg/m² (2), 90 mg/m² (2)
  – Primarily in combination with decitabine (n=5 in decitabine Arm vs n=1 in LDAC Arm)
  – CR/CRi rate was 31% (5/16) in patients treated with onvansertib 27-90 mg/m² in combination with decitabine

► Pharmacodynamic and biomarker analysis:
  – Onvansertib-plasma inhibitory activity was observed with all doses and positively correlated with increasing doses
  – Target engagement in circulating blasts was observed in a subset of patients and was associated with an increase in response to treatment as measured by decrease in BM blasts and rate of CR/CRi

► Phase 2: enrolling
  – is enrolling and will include 32 patients to further assess the safety, efficacy, target engagement and correlation with response of onvansertib 60 mg/m² in combination with decitabine

Zeidan A et al., ASH 2019; Abstract #230
Corporate
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 and Early 2020 Raised Capital & Clinical Research Commitment

- ~$16.0 million

Quarter Ending Cash and Cash Equivalents

- Q1’19 = $11.3M
- Q2’19 = $10.8M
- Q3’19 = $9.0M
- Q4’19 = $10.2M

Estimated Quarterly Cash Burn

- ~$4.0M
## 2020 Key Inflection Points

### 1H2020 Key Inflection Points

<table>
<thead>
<tr>
<th>Event</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colorectal Cancer: Phase 1b Safety and Efficacy Data</td>
<td>ASCO-GI</td>
</tr>
<tr>
<td>Prostate Cancer: Phase 2 Efficacy Data</td>
<td>ASCO-GU</td>
</tr>
<tr>
<td>Acute Myeloid Leukemia: Biomarker Data</td>
<td>AACR</td>
</tr>
<tr>
<td>Prostate Cancer: Phase 2 and Correlative Biomarker Data</td>
<td>AACR</td>
</tr>
<tr>
<td>Acute Myeloid Leukemia: Phase 2 Efficacy Data</td>
<td>EHA</td>
</tr>
</tbody>
</table>

### 2H2020 Key Inflection Points

<table>
<thead>
<tr>
<th>Event</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prostate Cancer: Phase 2 Efficacy Data</td>
<td>ESMO</td>
</tr>
<tr>
<td>Colorectal Cancer: Phase 2 Efficacy Data</td>
<td>ESMO</td>
</tr>
<tr>
<td>Acute Myeloid Leukemia: Phase 2 Efficacy Data</td>
<td>ESMO</td>
</tr>
<tr>
<td>Prostate Cancer: Phase 2 Efficacy Data</td>
<td>EMUC</td>
</tr>
<tr>
<td>Colorectal Cancer: Phase 2 Efficacy Data</td>
<td>ESMO Asia</td>
</tr>
<tr>
<td>Acute Myeloid Leukemia: Phase 2 Efficacy Data</td>
<td>ASH</td>
</tr>
</tbody>
</table>
Thank You

For additional information please contact: ir@trovagene.com