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

NASDAQ: TROV

trovagene
ONCOLOGY

Forward-Looking Statements

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.

Trovagene Oncology
Developing First-in-Class, Third-Generation, Oral PLK1 Inhibitor

- 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

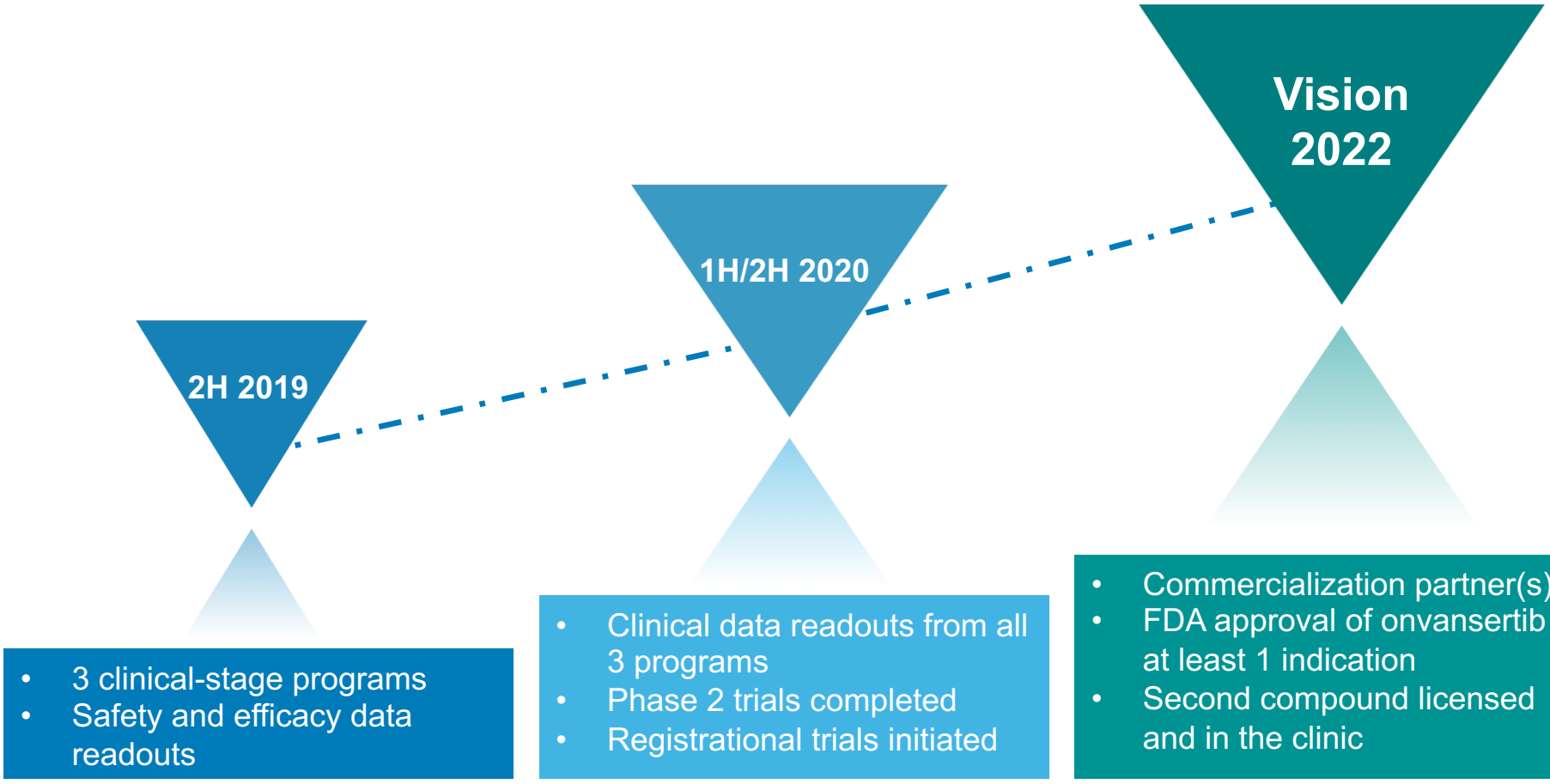
Thomas Adams, PhD
Chief Executive Officer and Chairman

Mark Erlander, PhD
Chief Scientific Officer

Vicki Kelemen
Vice President Clinical Development

Iris Genta SYNERGY PHARMACEUTICALS
GEN-PROBE CHUGAI
BIOMÉRIEUX Johnson & Johnson PHARMACEUTICAL RESEARCH & DEVELOPMENT, L.L.C.
Bayer HealthCare Pharmaceuticals Dura PHARMACEUTICALS
Agouron Pharmaceuticals, Inc. A Pfizer Company MAXIM PHARMACEUTICALS

Our Vision
Rapidly Advancing Clinical Development Programs



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

Experts in Protein Kinase Drug Development

Completed Phase 1 Solid Tumor Trial and IND

Established CMC (chemistry, manufacturing, controls)

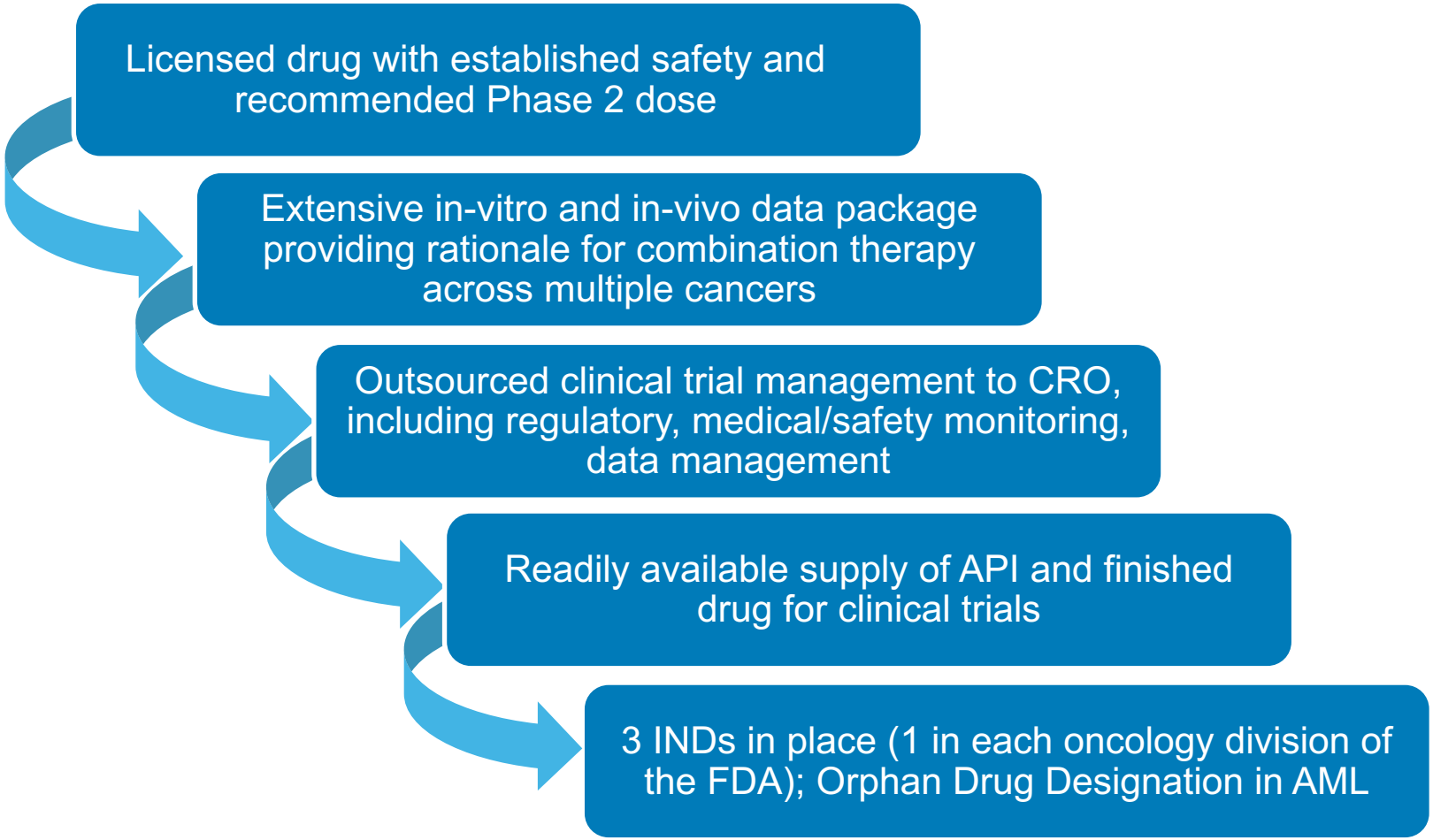
Completed Preclinical Data, Including Synergy

GMP / FDA Validated Manufacturing

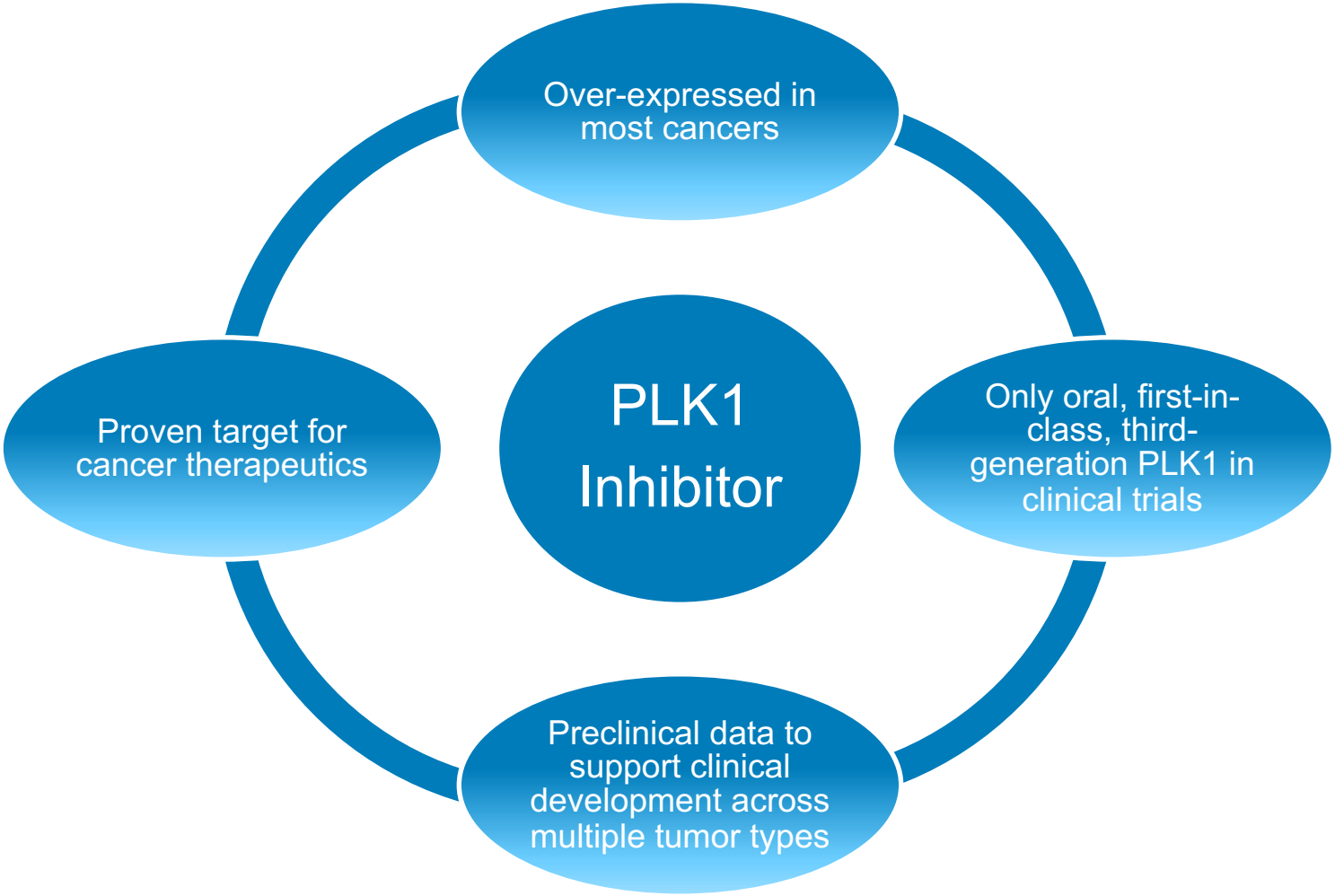
PLK1 Proven Effective Cancer Therapeutic Target

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

Optimized Operations and Clinical Development
Leveraging Internal Expertise and External Resources



We Have the Perfect Target
Onvansertib – Polo Like Kinase 1 (PLK1) Inhibitor



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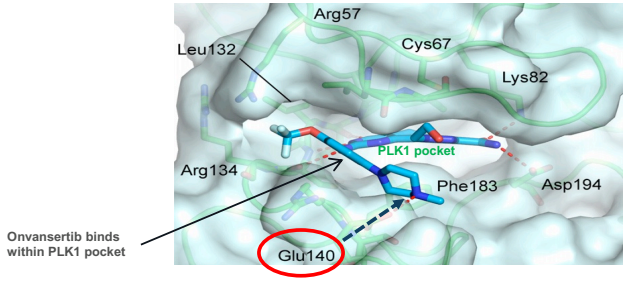
Onvansertib Targets the PLK1 Enzyme
A Proven Drug Target and Overexpressed in Most Cancers

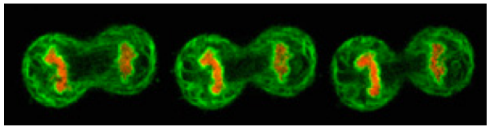
PLK Member	Onvansertib IC50* (μM)
PLK1	0.002
PLK2	> 10
PLK3	> 10

- High selectivity for PLK1, only
- Tested against >260 kinases; PLK1 only active target (IC_{50} of 2nM)

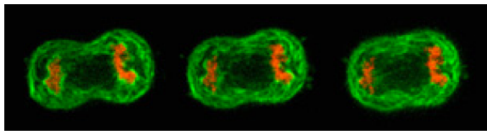
- Small molecule (MW 648.60 Daltons)
- Selectivity driven by stable interaction with carboxyl side chain of amino acid glutamate of PLK1 within PLK1's ATP binding pocket



Tumor Cell Division



Onvansertib Blocks Tumor Cell Division



- Onvansertib blocks cells from dividing by arresting them before they divide

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Onvansertib
Benefiting from Class Experience

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

Product Attributes	1st and 2nd Generation PLK Inhibitors	3rd Generation Onvansertib
Selectivity for PLK1	• panPLK inhibition of PLK1,2,3*	• Highly-selective only for PLK1
Antileukemic Activity	• Phase 2 & 3 trial results indicate activity • Improved response rates	• Clinical response in patients • Biomarker strategy identifies patients most likely to respond
Administration	• Intravenous (IV)	• Oral
Half-Life	• ~135 hours (5.5 days)	• ~24 hours
Dosing and Schedule	• Fixed treatment schedule • Fixed dose for all patients	• Treatment schedule flexibility • Dose determined based on BSA
Tolerability	• Insufficient time between treatment cycles negatively impacted tolerability/survival	• Time allotted between cycles for patient recovery from on-target hematologic toxicities
Infection Prophylaxis	• Increased rate of fatal infections	• Antibiotics to proactively mitigate infections

Onvansertib

First-in-Class, Third-Generation PLK1 with Best-in-Class Attributes

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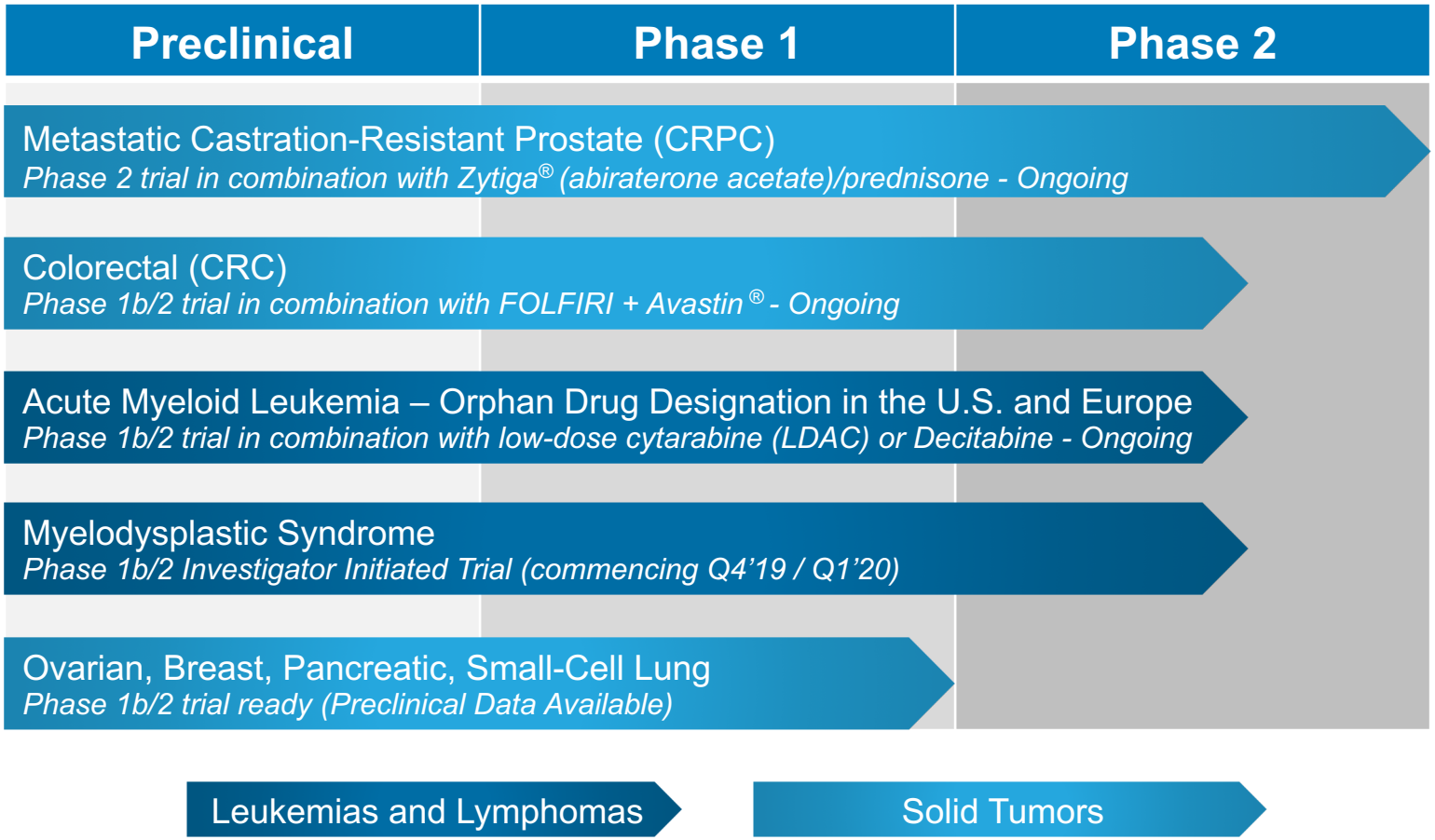
Diversified Pipeline with 3 Clinical-Stage Programs
Opportunities in Leukemias/Lymphomas and Solid Tumors

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



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

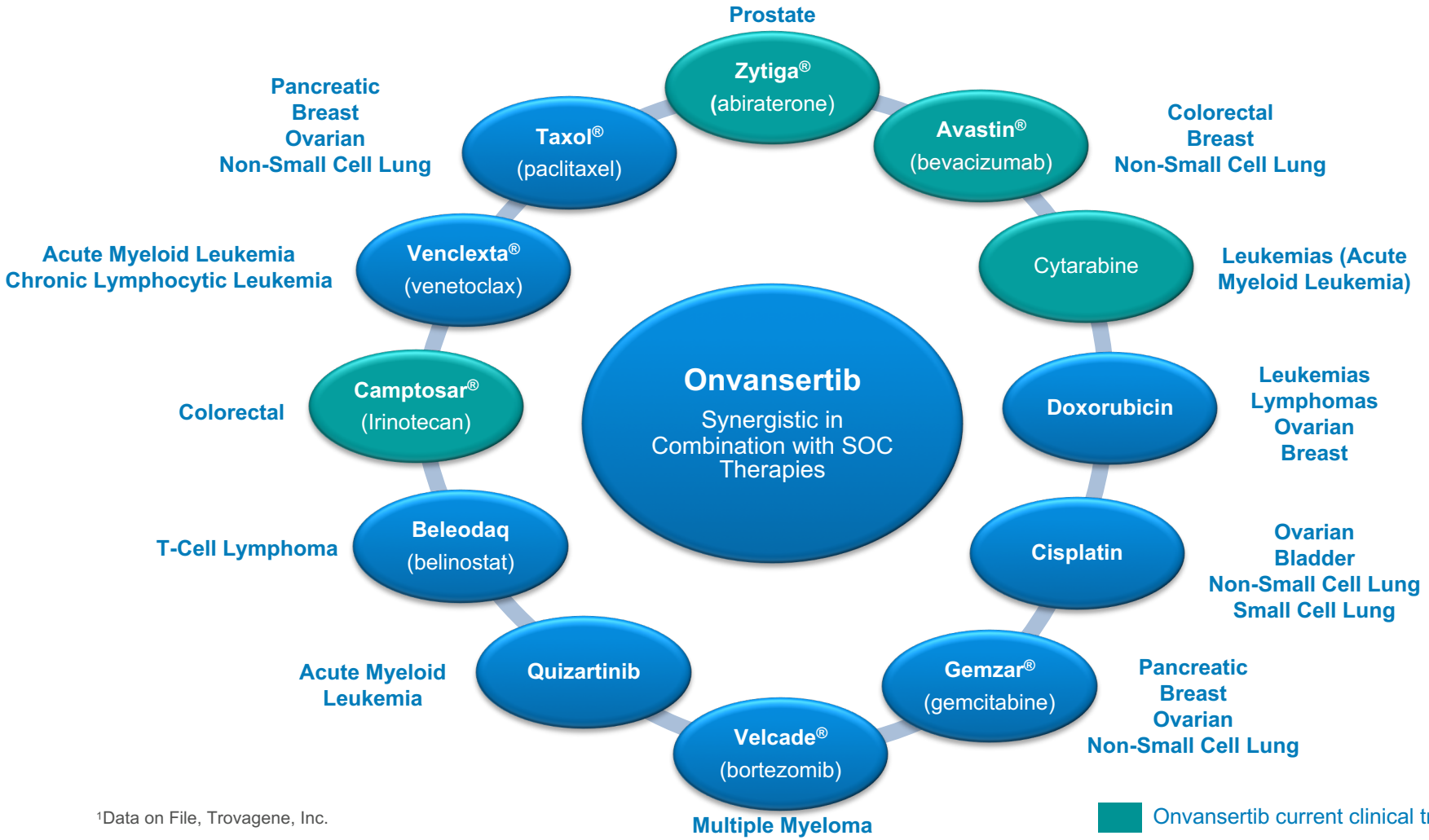
Combination Therapy
The Cornerstone of
Precision Cancer Medicine™

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

Onvansertib

Synergy May Enhance Efficacy of Standard-of-Care Therapies[1]



[1]Data on File, Trovagene, Inc.

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Phase 2 Trial: metastatic Castration-Resistant Prostate Cancer

Beth Israel Deaconess Medical Center

DANA-FARBER CANCER INSTITUTE

MASSACHUSETTS GENERAL HOSPITAL CANCER CENTER

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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***[2]

25,000 men die from metastatic prostate cancer annually[2]

5-year survival rate is ***37%***[2]

Treatment

Standard-of-care is ***Zytiga® and Xtandi®; resistance*** develops within ***9-15 months***[4]

Tumors re-engineer androgen receptor (AR), variant 7 (ARv7); tumor growth without need for androgens[4]

Up to ***40% ARv7 resistance; very aggressive*** with ***no viable treatment options***[5]

Opportunity

PLK1 inhibition improves Zytiga® efficacy, repressing androgen signaling pathway[3,4]

PLK1 inhibition ***destabilizes AR and ARv7***[6]

Inhibiting PLK1 blocks expression of ARv7; ***stopping*** this ***resistance*** pathway[6]

$7.9 billion global market; $12.0 billion by 2025[7]

[1]2017 Annual Report on Prostate Disease – Harvard Health Publications; [2]GlobalData. Prostate Cancer—Global Drug Forecast and Market Analysis to 2023. Apr, 2015; [3] National Cancer Institute Metastatic cancer. Mar, 2013. Available at: http://www.cancer.gov/about-cancer/what-is-cancer/metastatic-fact-sheet; [4]GAntonarakis, 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; [7]https://www.grandviewresearch.com/industry-analysis/prostate-cancer-therapeutics-market

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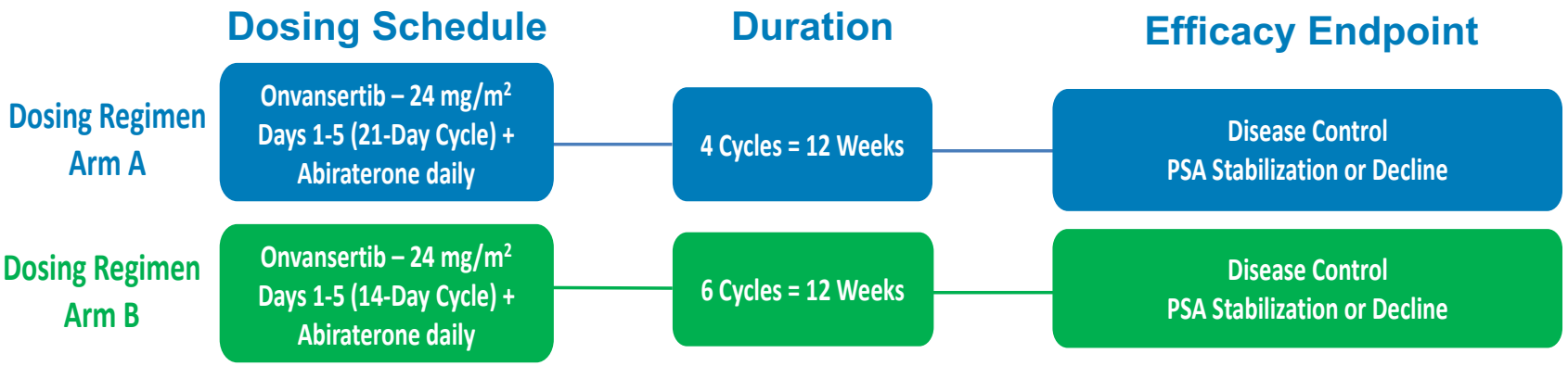
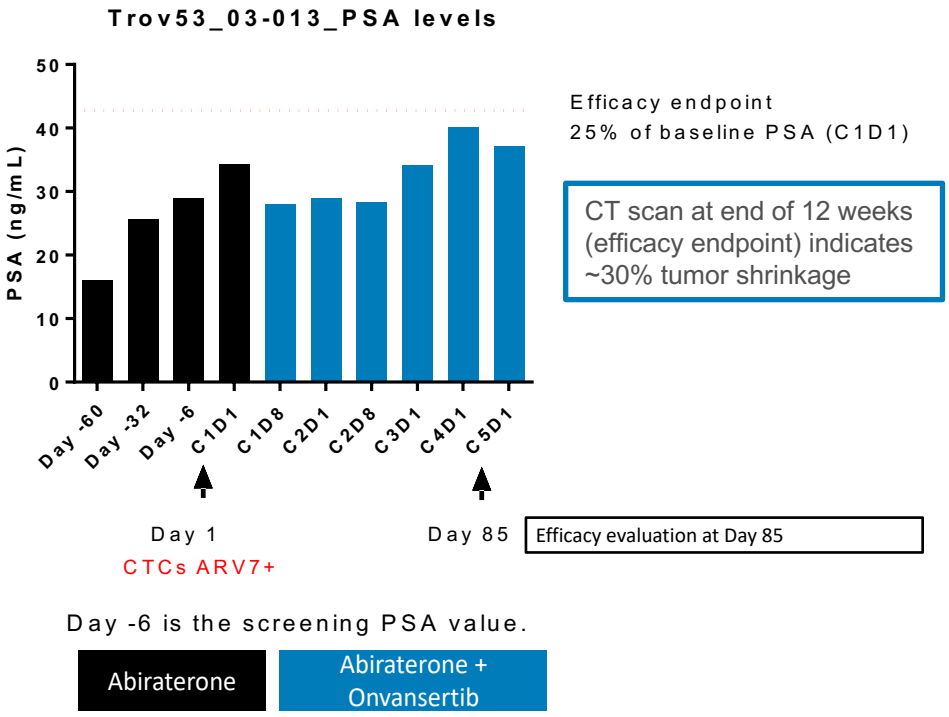
Phase 2 Clinical Trial in mCRPC
Disease Control Assessed by PSA Stabilization or Decline



- **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

Phase 1b/2 Trial: metastatic Colorectal Cancer

USC Norris Comprehensive Cancer Center
Keck Medicine of USC

hoag
Hoag Family Cancer Institute

MAYO CLINIC
Cancer Center

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Onvansertib Market Opportunity in mCRC
Only 5% Response to Current Second-Line Therapies

Disease Burden

140,000 new cases of ***CRC*** in 2018[1]

65% 5-year survival[1]

~51,000 deaths per year from ***mCRC***[1]

Treatment

Tumor ***biomarkers drive therapy decisions*** for 1st-line mCRC therapy[2]

~50% mCRC has RAS (***KRAS)*** mutation[2]

Standard-of-care is chemotherapy ***(FOLFOX/FOLFIRI)***[2]

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

Opportunity

Onvansertib + irinotecan (FOLFIRI) significantly ***reduces tumor growth***[3]

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[4]

[1]https://seer.cancer.gov/statfacts/html/colorect.html; [2]King et al, Frontline Strategies for Metastatic CRC, 2016, Amer J Hem/Onc; Loree&Kopetz, Recent Developments in treatment of mCRC, 2017, Ther Adv Med Onc; [3]Investigator Brochure, Data-on-file, Trovagene; [4]https://www.globaldata.com/store/report/gdhc141pidr--pharmapoint-colorectal-cancer-global-drug-forecast-and-market

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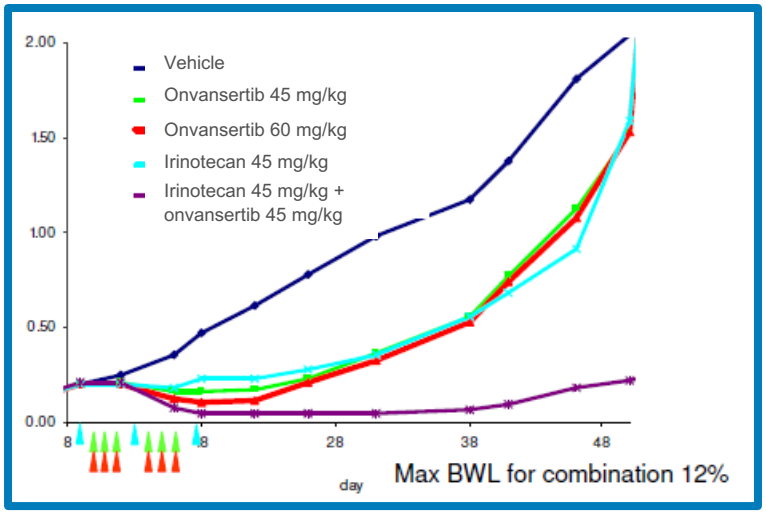
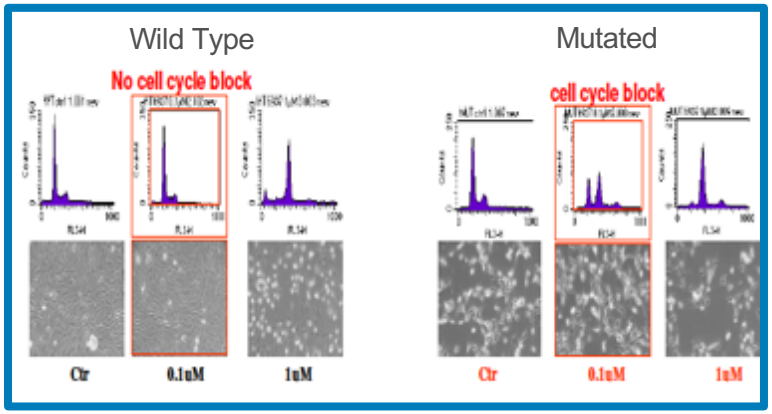
Synergy in Combination with Irinotecan
Preclinical Data Demonstrates Reduced Tumor Growth

- 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[1]



[1]Investigator Brochure, Data-on-file, Trovagene

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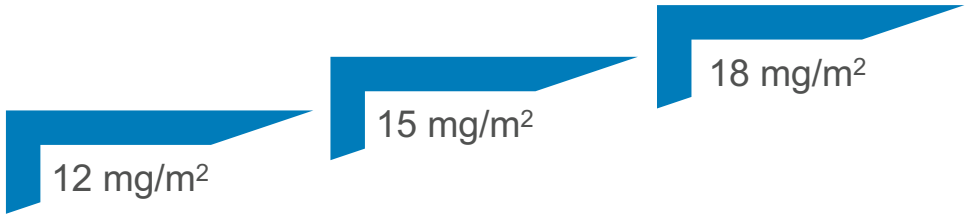
Phase 1b/2 Clinical Trial in mCRC
Objective Response Rate (ORR) in Second-Line Treatment

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

Yale CANCER CENTER

Seattle Cancer Care Alliance

UCLA Jonsson Comprehensive Cancer Center

ROSWELL PARK

UT Southwestern Harold C. Simmons Comprehensive Cancer Center

Virginia Cancer Specialists

Allina Health VIRGINIA PIPER CANCER INSTITUTE

THE UNIVERSITY OF KANSAS CANCER CENTER

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Onvansertib Market Opportunity in AML
Providing a New Treatment for Relapsed/Refractory Patients

Disease Burden

20,000 new cases ***annually***

5-year survival rate of only ***25%***[1]

Aggressive blood cancer that usually ***occurs in the elderly***[1]

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 options[2]

Opportunity

Onvansertib + chemotherapy has ***significant activity in AML*** models[3]

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 2023[5]

[1]National Cancer Institute SEER 2016; [2]DiNardo et al, Blood, 2019 [2]Valsasina et al., Mol Cancer Ther; 11(4) April 2012; [4]Trovagene, data on file;
[5]https://www.medgadget.com/2019/04/global-acute-myeloid-leukemia-treatment-market-is-expected-to-reach-usd-1-billion-with-cagr-of-5-3

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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)

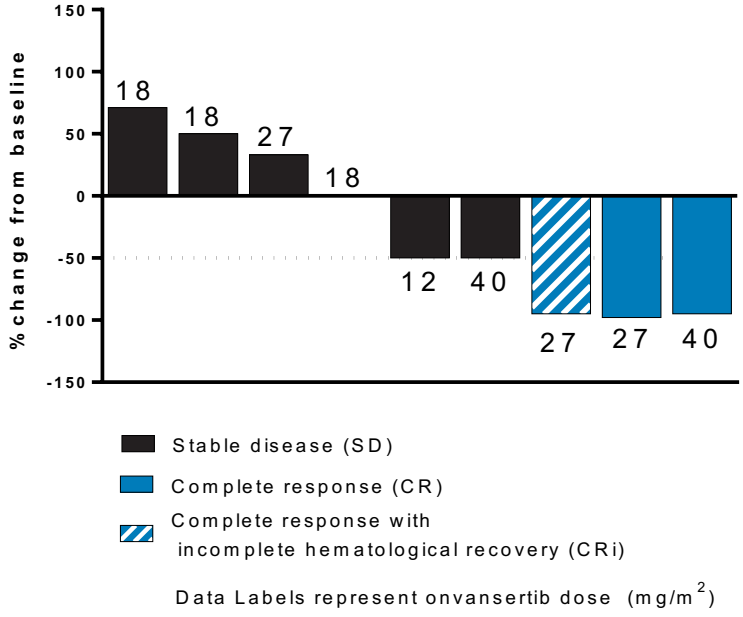
Phase 2: Assess safety and preliminary antitumor activity

- **Efficacy Endpoints:** Rate of complete response (CR + CRi) defined as morphologic leukemia-free state (MLF)
- **Exploratory Endpoints:** Evaluation of pharmacodynamic and correlative biomarkers

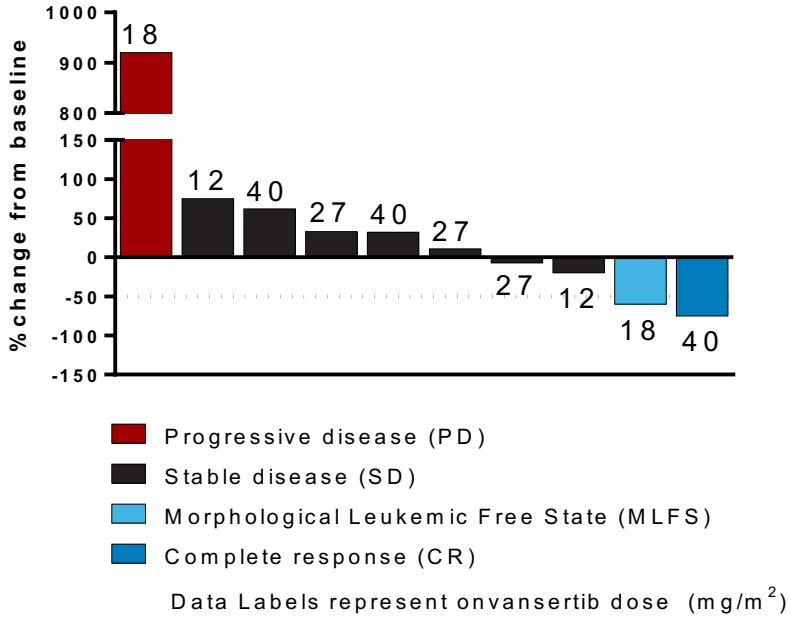
Patients Achieving Complete Response
Onvansertib is Safe and Well Tolerated

- 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



Onvansertib + LDAC



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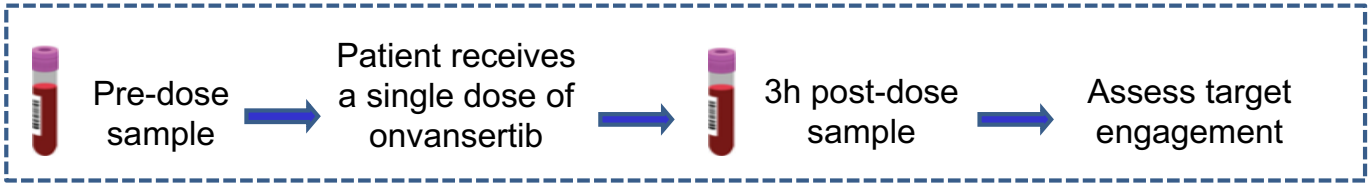
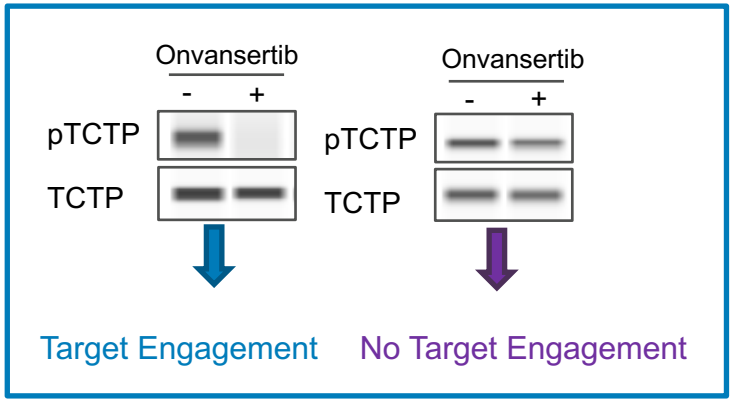
Biomarker Evaluates Inhibition of PLK1
Identifies Patients Most Likely to Respond to Treatment

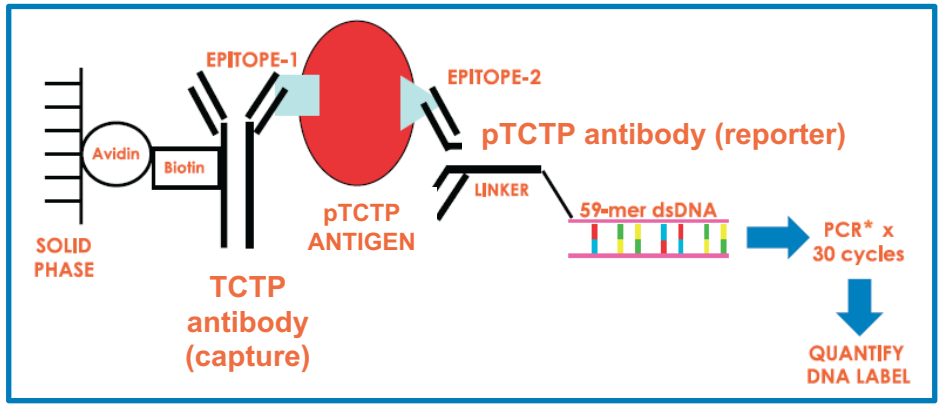
- Blood test examines the extent that onvansertib inhibits PLK1 enzymatic activity (target engagement) by assessing the phosphorylated status of TCTP within circulating leukemic blast cells



Current method: Western-Blot



Method in development: immuno-PCR based technology



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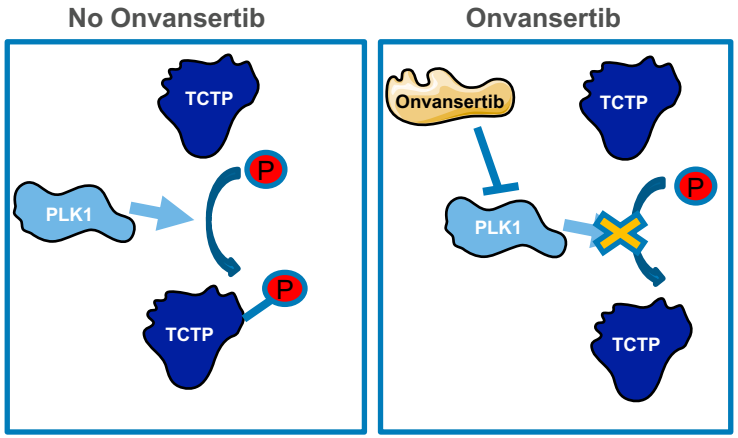
Biomarker to Assess Inhibition of PLK1
Correlation of Biomarker^{+} Patients with Treatment Response

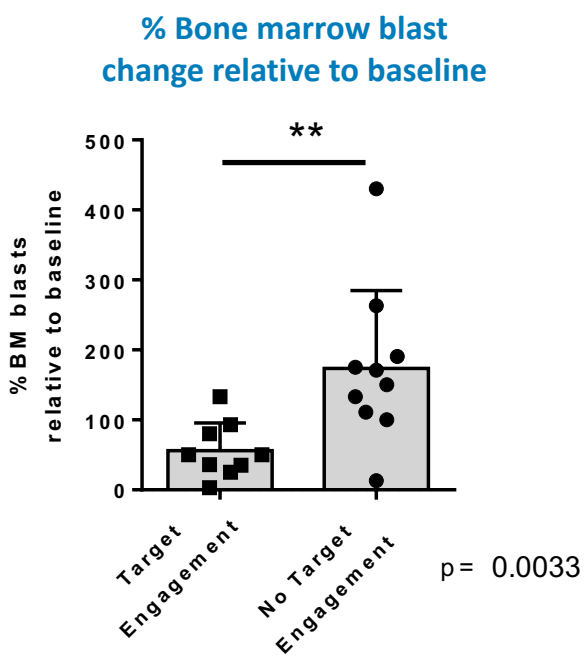
PLK1 inhibition can be monitored in patients through pTCTP status to determine target engagement of onvansertib with PLK1

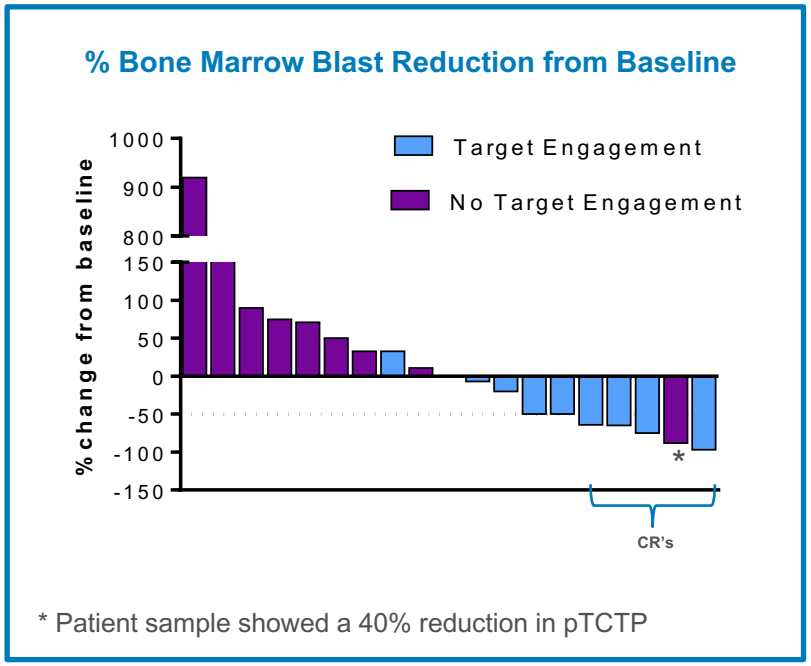
- **pTCTP as a marker of PLK1 activity:**
  - PLK1 phosphorylates the translational control tumor protein (TCTP) on serine 46
  - pTCTP was identified as a specific marker for PLK1 activity in vivo in preclinical models
- **The comparative change in pTCTP status between pre-dose and 3 hours post-dose 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





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Thank You

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For additional information please contact: ir@trovagene.com