Turning the Tide on Cancer

Cardiff Oncology

May, 2020

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.

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

Investment Highlights

Ovansertib

1st-in-class, 3rd-generation, safe and well-tolerated, oral PLK1 inhibitor; selectively targets PLK1 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

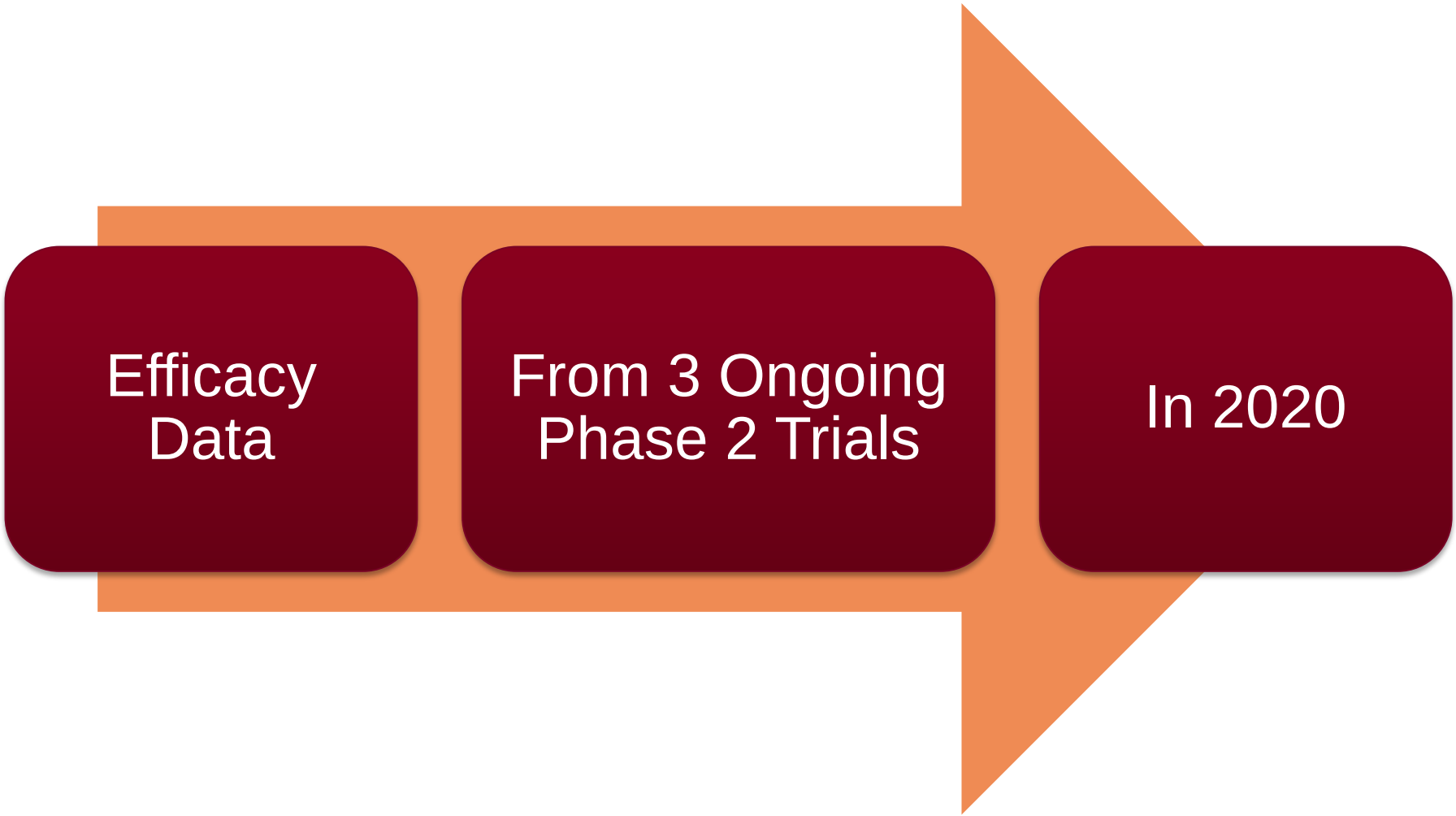
Validating Combination Clinical Trials

- 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



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2020 Key Inflection Points

1H2020 Key Inflection Points	Event	Timing
Colorectal Cancer: Phase 1b Safety and Efficacy Data	ASCO-GI	January 25th
Prostate Cancer: Phase 2 Efficacy Data	ASCO-GU	February 13th
Colorectal Cancer: Phase 1b Safety and Efficacy Data	AACR	April 27th
Colorectal Cancer: Phase 1b Safety and Efficacy Data	ASCO	May 29th – June 2nd
Acute Myeloid Leukemia: Phase 2 Efficacy Data	EHA	June 11th – 14th
Acute Myeloid Leukemia: Biomarker Data	AACR	June 22nd – 24th
2H2020 Key Inflection Points	**Event**	**Timing**
Prostate Cancer: Phase 2 Efficacy Data	ESMO	Sept. 18th – 22nd
Colorectal Cancer: Phase 2 Efficacy Data	ESMO	Sept. 18th – 22nd
Acute Myeloid Leukemia: Phase 2 Efficacy Data	ESMO	Sept. 18th – 22nd
Prostate Cancer: Phase 2 Efficacy Data	EMUC	Nov. 12th – 15th
Colorectal Cancer: Phase 2 Efficacy Data	ESMO Asia	Nov. 20th – 22nd
Acute Myeloid Leukemia: Phase 2 Efficacy Data	ASH	Dec. 5th – 8th

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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 Need for New Treatment Options:**
  - Overcome resistance to standard-of-care drugs
  - Extend response to treatment and progression-free survival (PFS)

	Indication	Preclinical	Phase 1/1b	Phase 2/2b	Next Milestone
Onvansertib Solid Tumors	mCRC	*Onvansertib + FOLFIRI/Avastin® in Second-Line KRAS-Mutated Metastatic Colorectal Cancer*			Q2 2020 ASCO
	mCRPC	*Onvansertib + Zytiga® (abiraterone)/prednisone in Zytiga-Resistant Castration-Resistant Metastatic Prostate Cancer*			Q3 2020 ESMO
Onvansertib Hematologic	AML	*Onvansertib + Decitabine in Relapsed or Refractory Acute Myeloid Leukemia*			Q2 2020 EHA

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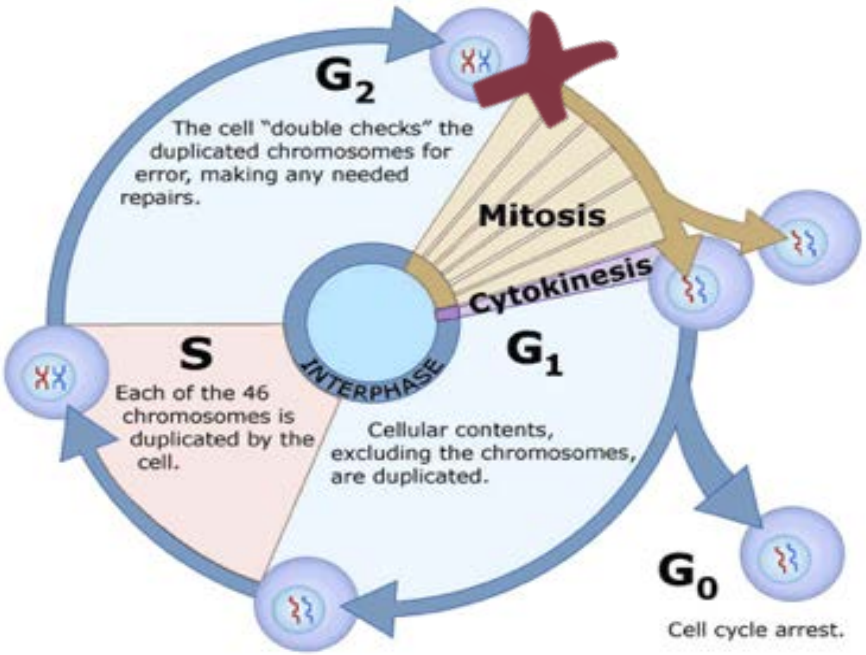
Onvansertib: Stops Cancer Cell Division and Synergistic in Combination Regimens

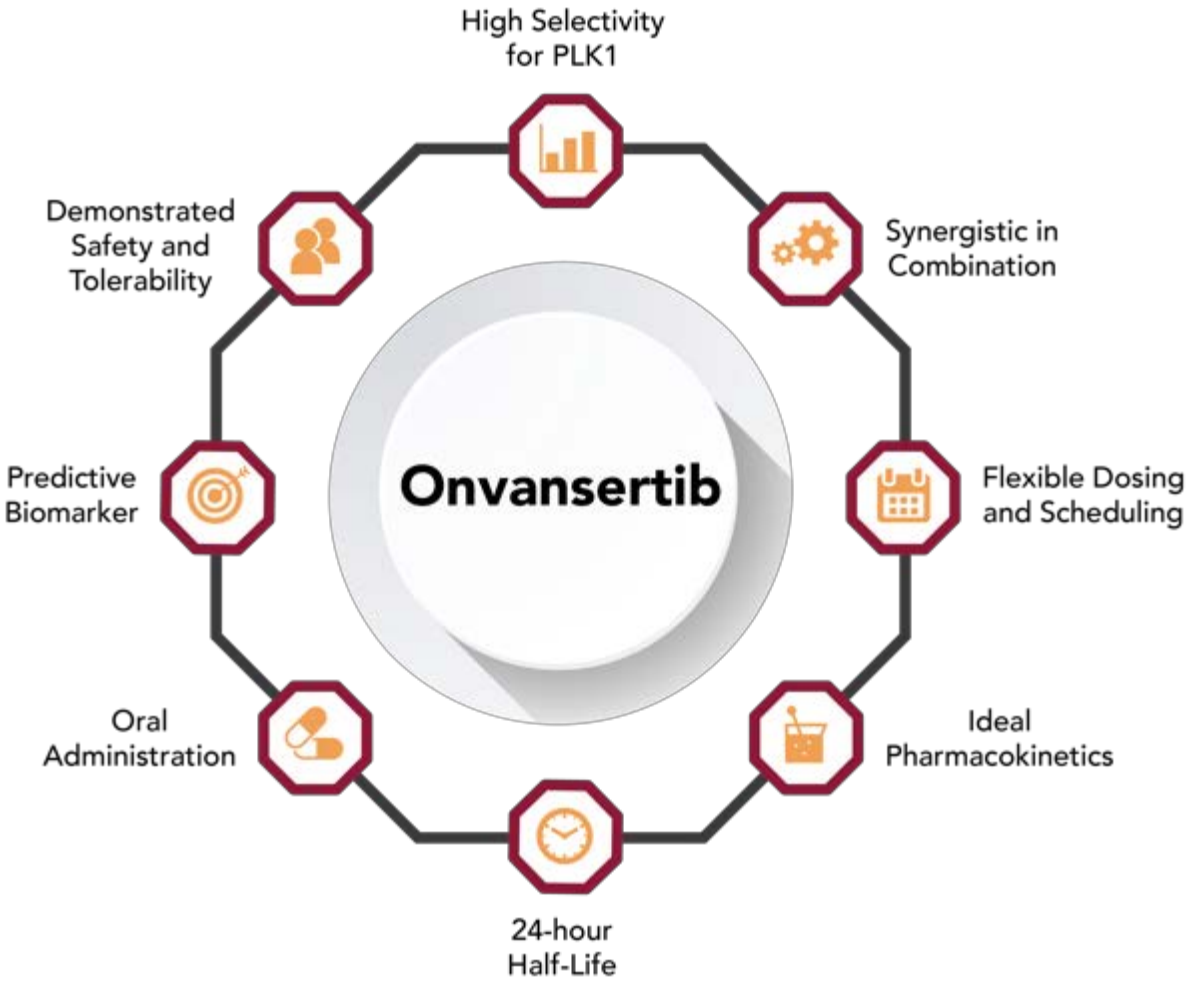
Inhibition of PLK1 causes arrest of cell division and subsequent cell death[1]



Synergistic in combination with chemotherapies and targeted therapeutics[2]

[1]Zitouni et al., Nat Rev Mol Cell Biol. 2014 Jul;15(7):433-52; [2]Data on File – Cardiff Oncology

Optimal Attributes for a Safe and Effective Drug



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Indication: Second-Line Treatment of KRAS-Mutated Metastatic Colorectal Cancer (mCRC)

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

USC Norris Comprehensive Cancer Center
Keck Medicine of USC

MAYO CLINIC
Cancer Center

Principal Investigator
Dr. Heinz-Josef Lenz

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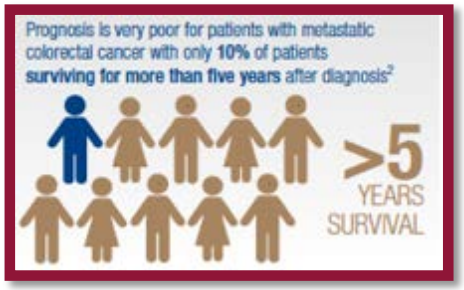
Improving Response and Progression-Free Survival

Metastatic Colorectal Cancer (mCRC)



- Only a 4% response rate to second-line standard-of-care chemotherapy + bevacizumab[1]
- Onvansertib + FOLFIRI® significantly reduces tumor growth[2]
- 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

[1]Kubicka et al, Annals of Oncology 2013; 2342–2349; [2]Investigator Brochure, Data-on-file, Trovagene; [3]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; [4]Tie et al., 2015, Annals of Oncology 26: 1715–1722; [5]Cancer Genomic Atlas Genome, Nature, 2012

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Clinical Outcomes in Patients Receiving Second-Line Treatment – Chemotherapy + bevacizumab[1]

Outcome Measure	Chemo + Bevacizumab	Chemo + Bevacizumab KRAS Mutated CRC
Progression-Free Survival	5.7 months	**5.5 months**
Overall Survival	11.2 months	**10.4 months**
Complete Response	1%	**1%**
Partial Response	4%	**3%**
Overall Response Rate	5%	**4%**

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

[1]Kubicka et al, Annals of Oncology 2013; 2342–2349

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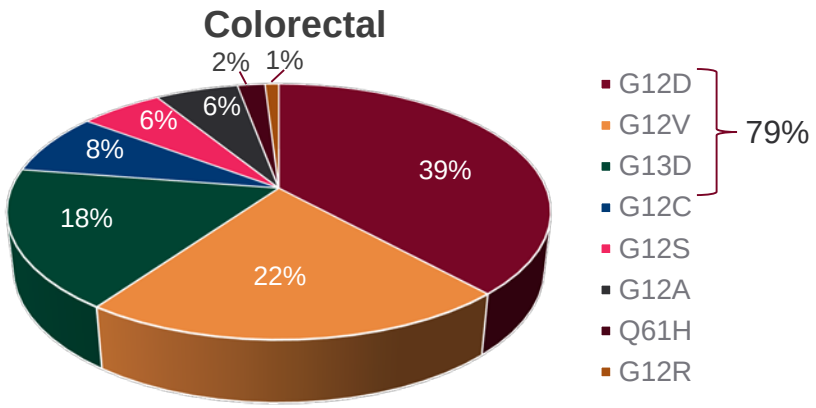
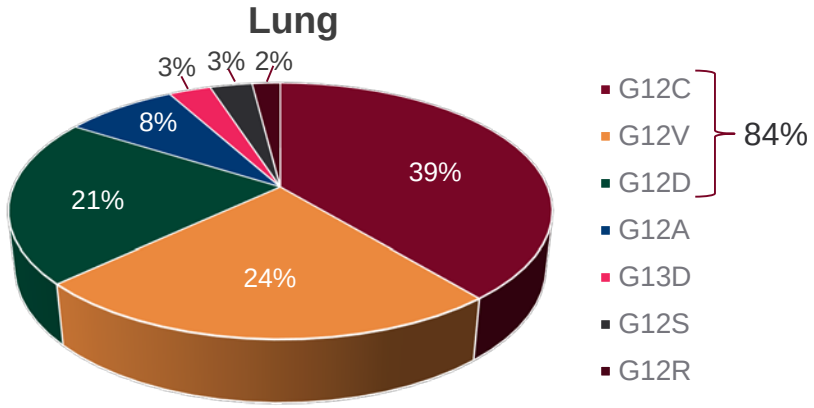
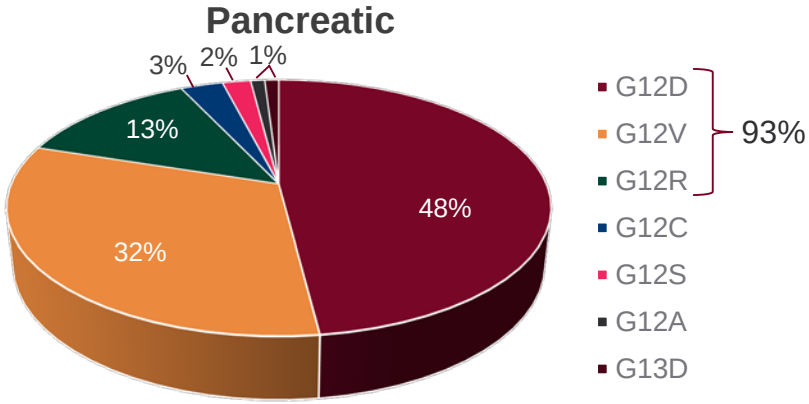
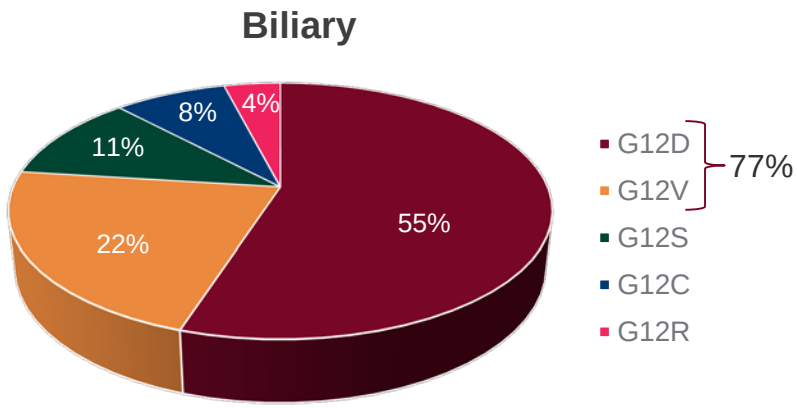
Multiple KRAS Mutation Subtypes are Present and Differ in Prevalence Across Cancer Types[1]

Onvansertib is Agnostic to KRAS Subtype









[1]COSMIC (Catalogue of Somatic Mutations in Cancer) database is the frequency of KRAS mutations across the majority of human cancers

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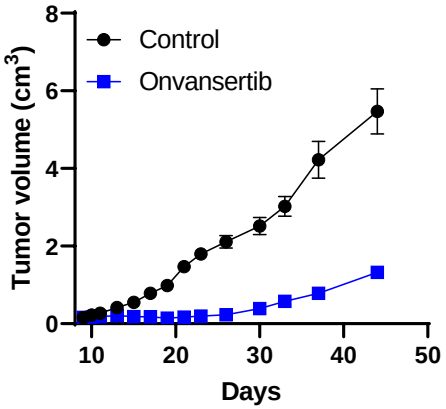
Rationale for Onvansertib + FOLFIRI® /Avastin® in KRAS-Mutated Metastatic CRC

Onvansertib Targets KRAS Mutations Through Downstream Effects on Tumor Cell Division

- **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]

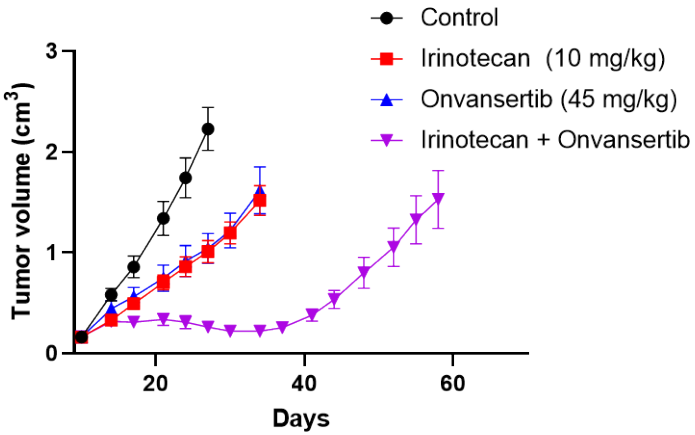
[1]Luo J, Elledge SJ, Cell 2009; [2]Trovagene, Investigator Brochure, 2019; [3]Valsasina et al., Mol Cancer Ther 2012; [4]Weiss et al, Invest New Drugs, 2017

Anti-tumor Activity of Onvansertib as Single Agent and Synergy in Combination with Irinotecan

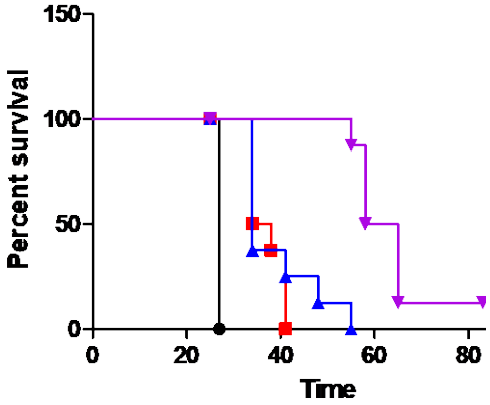


- Anti-tumor activity of onvansertib in a KRAS-mutant CRC xenograft model (HCT116) as single agent and in combination with irinotecan[1-3]

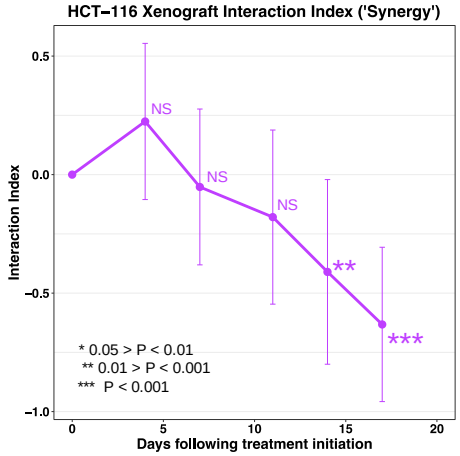
Tumor Growth



Survival



Synergy



[1]Valsasina et al., 2012, Mol Cancer Ther, 11: 1006-1016; [2]Data on file – Cardiff Oncology; [3]Method used for testing synergy: Wu et al., 2012. J Biopharm. Stat. 22(3): 535-543

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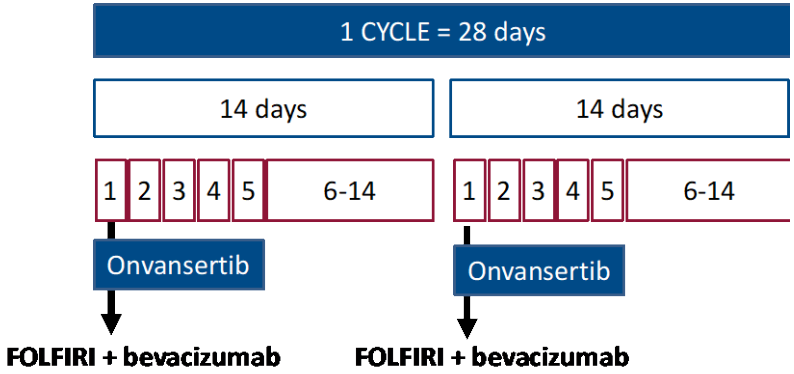
Demonstrating Clinical Benefit in KRAS-Mutated CRC as New Second-Line Treatment Option

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



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[1]

What is Clinical Trial Success:

- ≥ 5 of 26 (~20%) patients achieve clinical response confirmed by radiographic scan
- Patients achieve median progression-free survival (PFS) of ≥ 6 months

[1]Kubicka et al, Annals of Oncology 2013; 2342–2349

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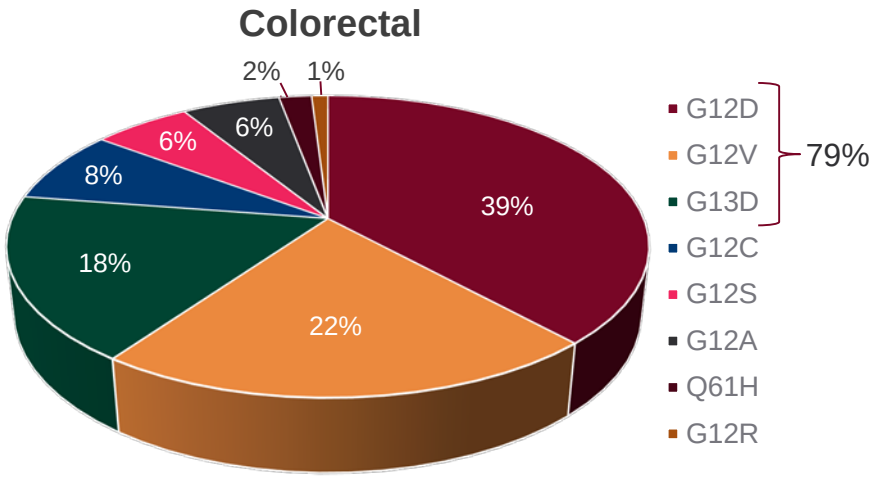
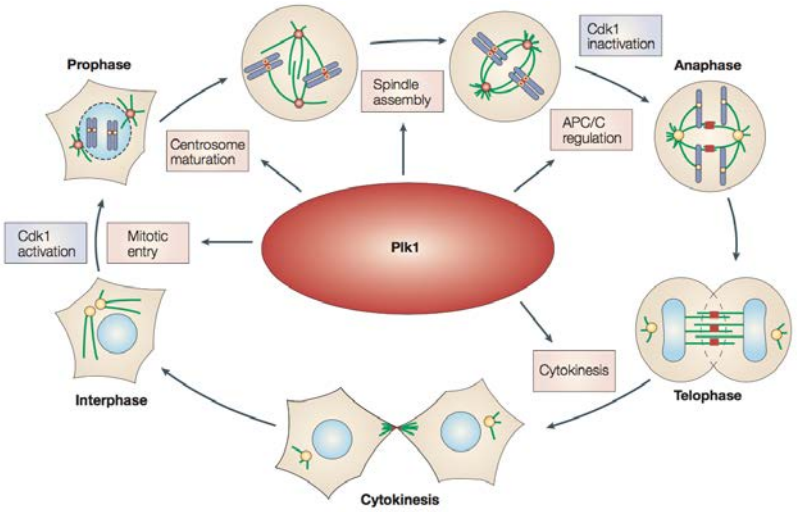
Clinical Data Shows Onvansertib Effectively Targets Multiple KRAS Mutation Subtypes in CRC



- To date, tumor shrinkage observed in KRAS mutations G12A, G12V, G12D, G13D which make up 85% of KRAS subtypes in CRC[1]
- Other drugs in development target only the KRAS G12C mutation, which accounts for ~8% of the KRAS mutations in CRC

[1]Jones 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

PLK1 Inhibition Induces Profound Arrest and Death of Tumor Cells in KRAS-Mutated CRC



- PLK1 is overexpressed in CRC and its high expression is associated with lower overall survival and poorer clinical parameters[1]
- PLK1 inhibition has been identified to have synthetic lethality with KRAS mutant in CRC cells[2]
  - PLK1 inhibition induced more profound mitotic arrest and apoptosis in KRAS mutant CRC cells than wild-type

[1]Weichert W., Worlkd J Gastroenterol 2005; [2]Luo J., Cell, 2009

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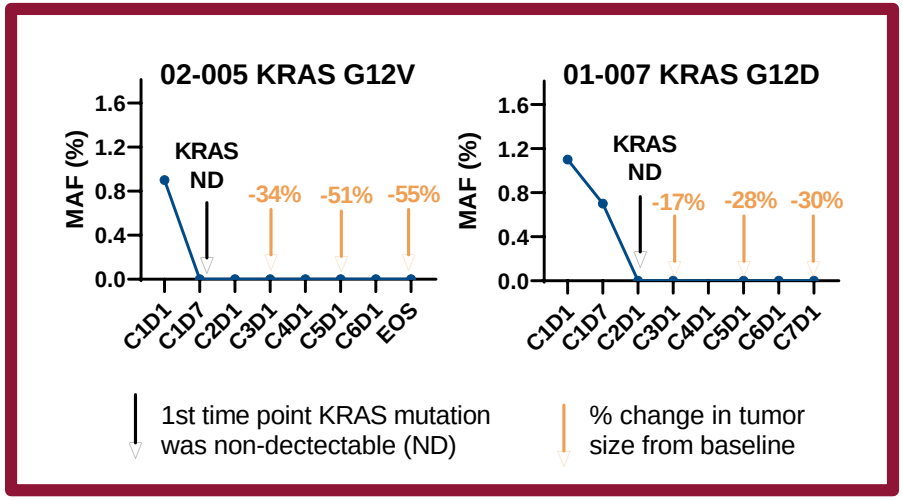
Response to Onvansertib Correlates with Decreases in KRAS Mutations to Undetectable Levels in Plasma

- Decreases in plasma KRAS mutation level has been demonstrated to be an early marker for therapeutic response[1]
- 7 of the 8 patients had a KRAS mutation detected by ctDNA analysis at baseline (ddPCR and NGS)
- Changes in KRAS mutant during cycle 1 of treatment were highly predictive of tumor regression:
  - 5 patients had a decrease in KRAS mutant to non-detectable level in cycle 1 (28 days) and subsequent tumor regression at 8 weeks (C3D1)
  - 2 patients had detectable KRAS mutant at end of cycle 1 and showed tumor growth at C3D1

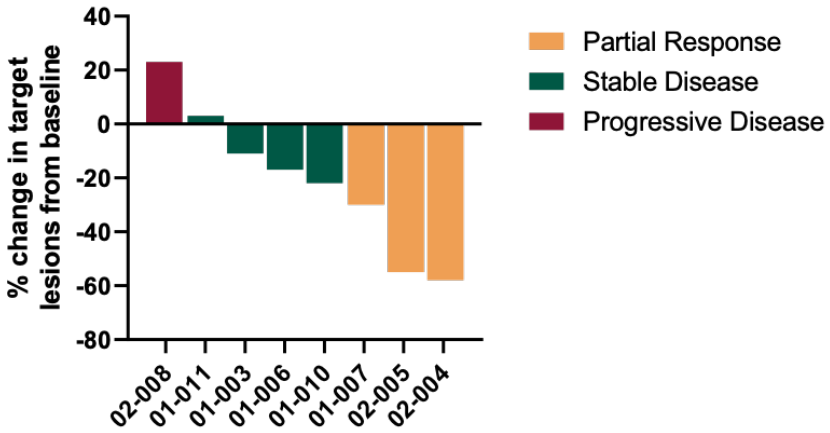


[1]Tie et al., 2015, Annals of Oncology 26: 1715–1722; [2]BioRad Droplet Digital Assays

Response to Treatment Confirmed by Radiographic Scan and Progression-Free Survival

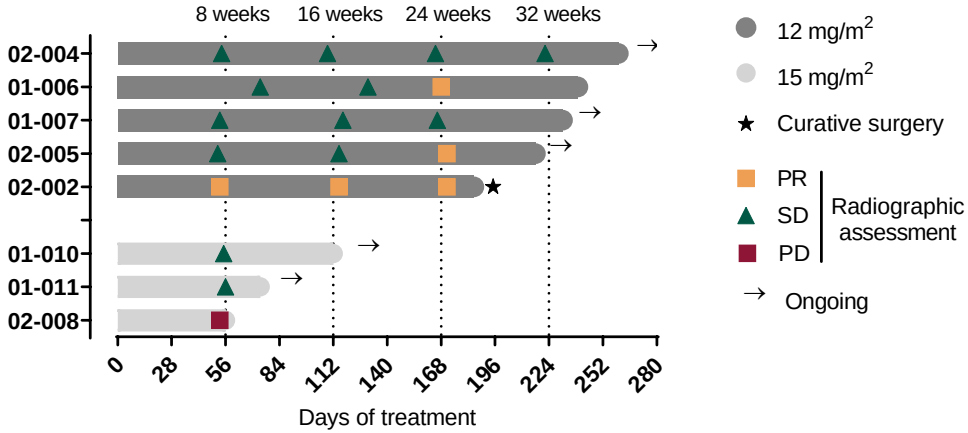
- 7 patients were evaluable for efficacy:
  - 6 of 7 (86%) had clinical benefit of partial response (PR) + stable disease (SD)
  - 3 of 7 (42%) achieved a PR; 1 patient (02-005) went on to have successful curative surgery

Radiographic Response



- Responses appear durable: progression-free survival (PFS) of >6.5 months (to-date) with patients continuing on treatment

Progression-Free Survival



Onvansertib is Showing Promise as a New Therapeutic Option for KRAS-Mutated mCRC

- The 1st two dose levels (onvansertib 12 mg/m^2 and 15 mg/m^2) were cleared for safety; the 3rd dose level (onvansertib 18 mg/m^2) is enrolling
- Clinical benefit (SD + PR) was observed in 7 (88%) of the 8 evaluable patients
- At the first two dose levels (onvansertib 12 mg/m^2 and 15 mg/m^2)
  - 4 patients had SD; 3 patients achieved PR (>30% decrease from baseline); 1 patient proceeded to successful curative surgery
  - Responses appear durable: PFS of >6.7 months to-date; 5 patients remain on treatment
- 7 of the 8 patients evaluable for efficacy had a detectable KRAS mutant at baseline:
  - In 5 patients, KRAS mutant decreased to undetectable level within the 1st cycle and KRAS mutant decrease to undetectable level preceded tumor shrinkage, supporting the predictive value of liquid biopsy
  - 2 patients had detectable KRAS mutant at end of cycle 1 and showed tumor growth at Cycle 3 Day 1

Other Drugs Currently in Development Do Not Address the Prevalent KRAS Mutations in CRC

Company and Drug Candidate	KRAS Coverage	KRAS Program(s) and Development Stage	Available Data and Status
Trovagene Oncology Onvansertib (PLK1 inhibitor)	• Pan-KRAS mutations (G12D, G12V, G12C, G13D, G12A, G12R, G12S, Exon 3 and 4)	• Phase 1b/2 trial in KRAS mutated mCRC (NCT03829410	• Preclinical activity in CRC tumors with different KRAS mutations • Phase 1b: 7 patients evaluable for response • 6 of 7 (86%) had clinical benefit (PR + SD) • Responses appear durable: PFS of ~6 months to-date with all patients remaining on treatment

Mirati Therapeutics
MRTX849
KRAS G12C Inhibitor
	• KRAS G12C+ tumors, only [occurrence: NSCLC = 14%; CRC = 4%; Panc = 2%]	• Phase 1b/2 KRYSTAL Trial (NCT03785249) in NSCLC, CRC	• ORR - SD + PR (n=10)
			  • NSCLC = 3/6 (PR)
			  • CRC = 1/4 (PR)
			• Phase 1b/2 monotherapy expansion cohorts enrolling; potential for single arm registration

Amgen
AMG510
Covalent Inhibitor
	• G12C+ tumors, only	• Phase 1/2 trial in KRAS G12C mutated solid tumors (NCT03600883)	• ORR - SD + PR (n=42)
			  • NSCLC = 7/13 (PR)
			  • CRC = 1/29 (PR)
			• FDA Fast Track Designation (NSCLC)

Boehringer BI-1701963 (SOS1:KRAS inhibitor)	• G12 and G13	• Preclinical • Phase 1 trial monotherapy and in combination with MEK-inhibitor trametinib in KRAS-mutated solid tumors (NCT04111458)	• Preclinical data shows it blocks tumor growth for G12/G13 mutations

Indication:
metastatic Castration-Resistant Prostate Cancer (mCRPC)

Principal Investigator
Dr. David Einstein

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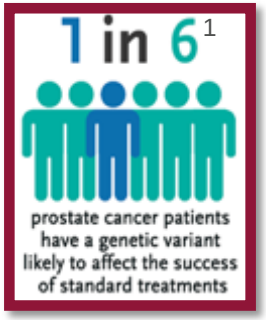
Overcoming Resistance and Extending Efficacy

Metastatic Castrate-Resistant Prostate Cancer (mCRPC)



- Resistance develops to standard-of-care therapy, Zytiga® and Xtandi®, within 9-15 months[2]
- 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[3]

Establishing a Successful Path Forward:

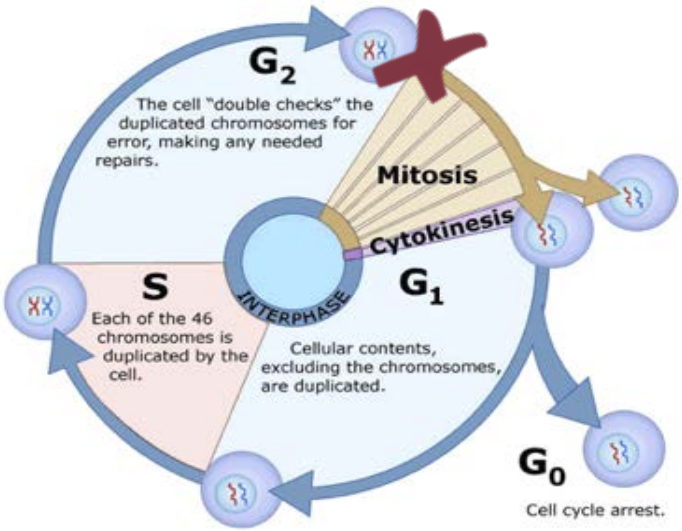
- Positive results from Phase 2 trial may provide an opportunity for a Phase 2b registrational trial
- Proactively assessing AR-V7 enables correlation of status (+/-) with response to onvansertib treatment
- Effective treatment of AR-V7+ patients could lead to Breakthrough Designation

[1]Nicolosi P, Ledet E, Yang S et al. Prevalence of germline variants in prostate cancer and implications for current genetic testing guidelines. JAMA Oncol. Published online February 7, 2019; [2]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; [3]Armstrong et al., 2019, JCO 37: 1120-1129.

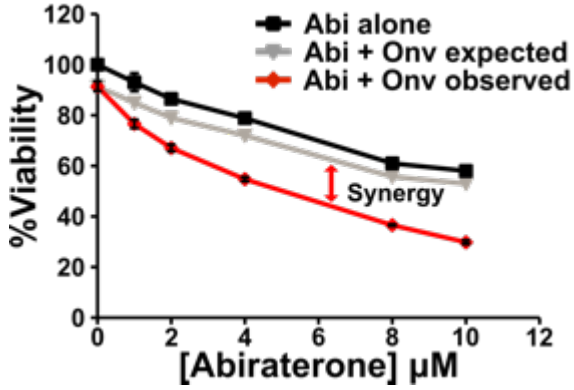
Underlying Mechanism of Action (MOA) for Onvansertib + Zytiga® in CRPC

Onvansertib Mechanism of Action

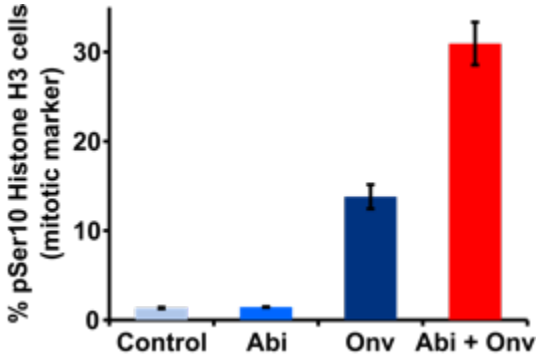
Inhibits tumor cell division (mitosis) by inducing G2/M arrest



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



Onvansertib + Zytiga® (abiraterone) significantly increase mitotic arrest[1]



[1]Patterson & Yaffe, 2019, MIT

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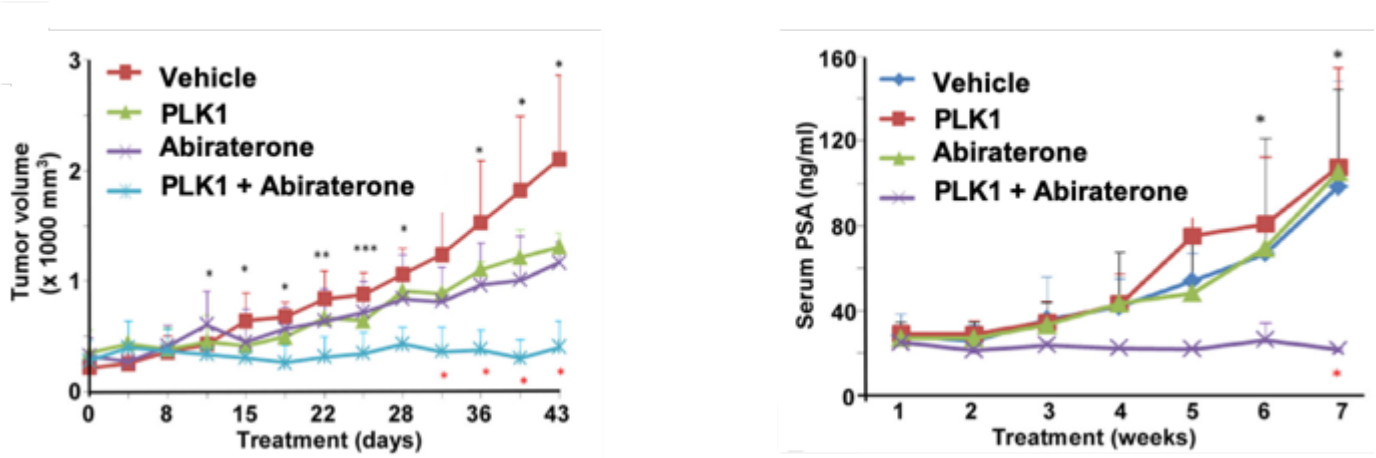
PLK1 Inhibition + Abiraterone Efficacy in mCRPC Model

PLK1 Inhibition Enhances the Efficacy of Androgen Signaling Blockade in Castration-Resistant Prostate Cancer



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

[1]Zhang et al., 2014, Cancer Res

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

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

	Dosing Schedule	Duration	Efficacy Endpoint
Cohort 1 n=32	Onvansertib 24mg/m^2 Days 1-5 (21-day cycle) + Zytiga®	4 Cycles = 12 Weeks	Disease Control PSA Stabilization or Decline
Cohort 2 n=32	Onvansertib 24mg/m^2 Days 1-5 (14-day cycle) + Zytiga®	6 Cycles = 12 Weeks	Disease Control PSA Stabilization or Decline
Cohort 3 n=32	Onvansertib 24mg/m^2 Days 1-14 (21-day cycle) + Zytiga®	4 Cycles = 12 Weeks	Disease Control PSA Stabilization or Decline

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 Clinical Trial 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 ≥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]

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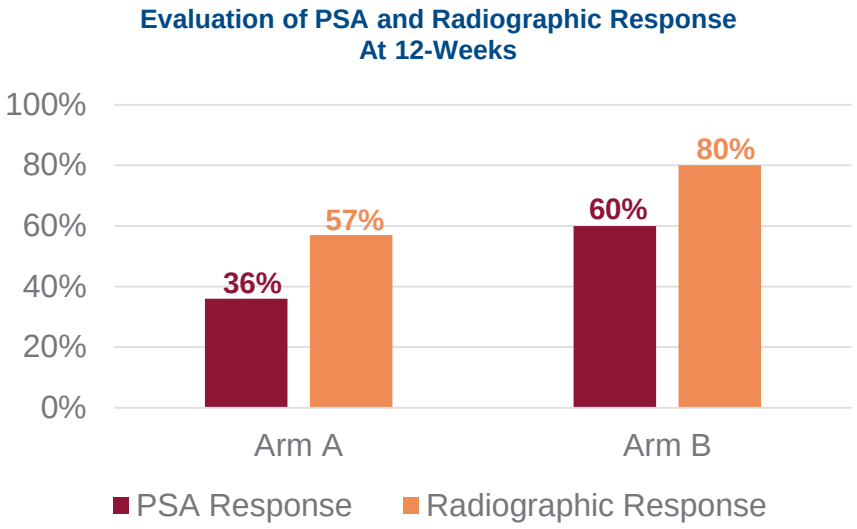
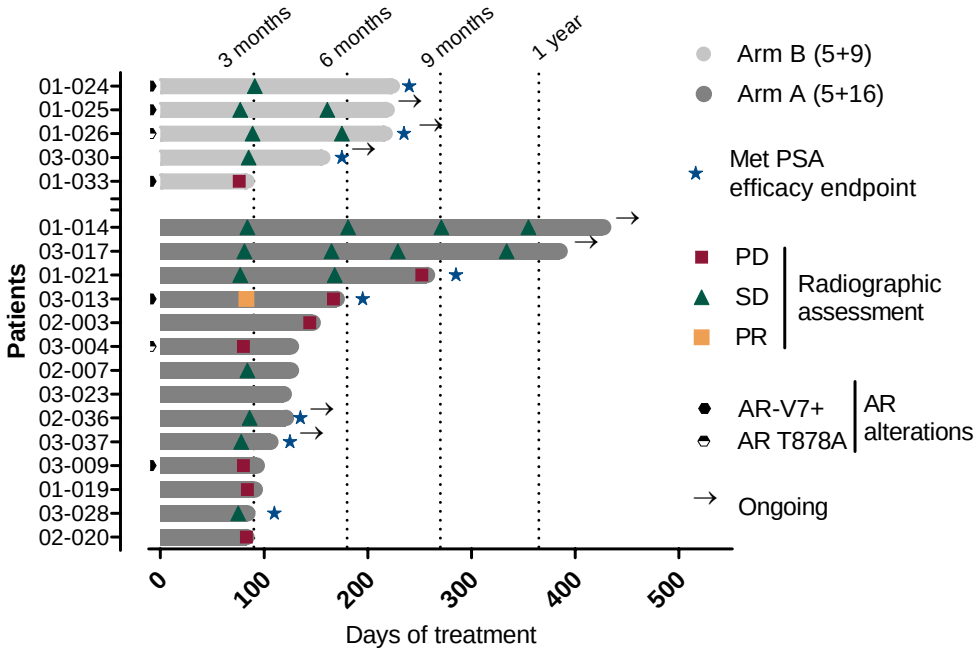
Efficacy Demonstrated in Zytiga®-Resistant Patients Treated with Onvansertib

- Overall, 63% (12 of 19) of evaluable 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





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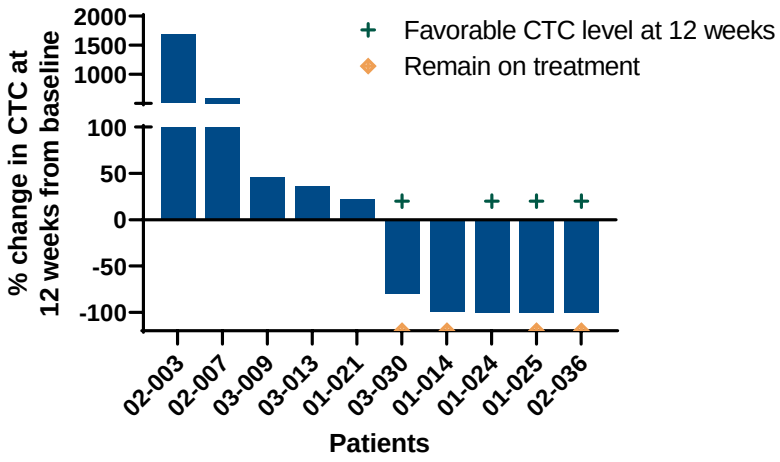
Onvansertib-Induced CTC Decrease is Associated with Progression-Free Survival

- 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 of 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: 12-Weeks vs Baseline in Patients with Unfavorable CTC Level at Baseline



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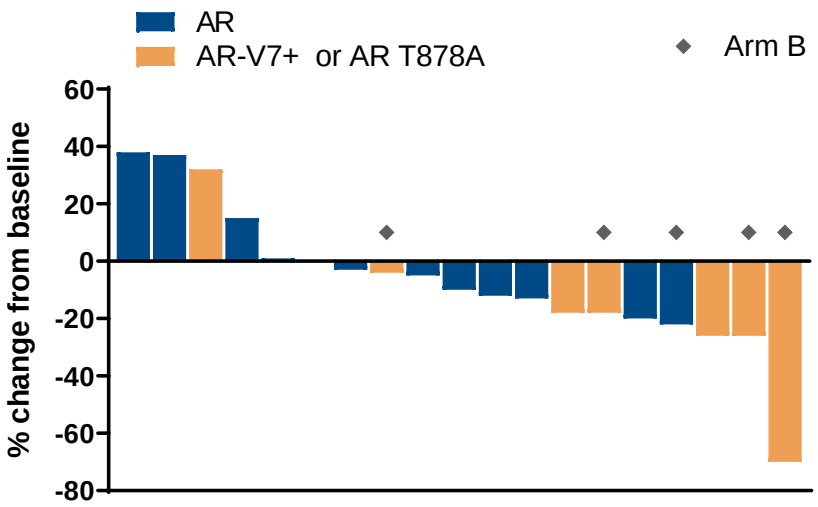
Efficacy Observed in Patients with Abiraterone-Resistant AR Alterations

- 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[6]
- 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 Positive and AR T878A Patients



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Indication:
Acute Myeloid Leukemia (AML)

Principal Investigator
Dr. Amer Zeidan

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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 plus 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 [2]Valsasina et al., Mol Cancer Ther; 11(4) April 2012; [3]Data on file – Cardiff Oncology

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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/m^2 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%[1-3]

What is Clinical Trial Success:

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

[1]Stahl et al., Blood Adv. 2018 Apr 24;2(8):923-932; [2]DiNardo et al, N Engl J Med. 2018 Jun 21;378(25):2386-2398; [3]Stein et al., Blood. 2017 Aug 10;130(6):722-731

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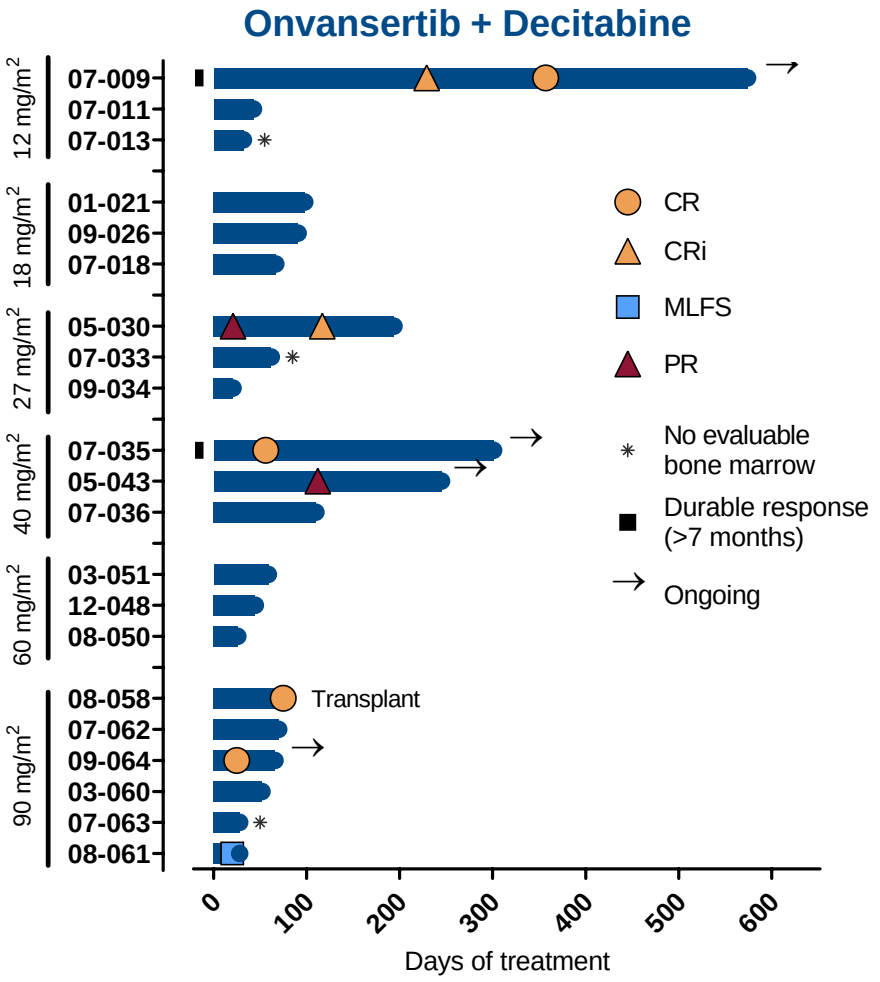
Phase 1b Completed Trial Efficacy Summary

Patients Treated with ≥ 1 Cycle (n=36)

- At the 4 higher dose levels (27 to 90 mg/m^2), 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
- 2 of the 6 responders discontinued treatment:
  - 1 patient proceeded to transplant following complete response
  - 1 patient progressed 2.5 months following CRi



Conclusions
Phase 1b Study of Onvansertib in AML[1]

- **Safety: onvansertib treatment was well tolerated**
  - MTD/RP2D was established at 60 mg/m^2 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/m^2 (2), 40 mg/m^2 (2), 90 mg/m^2 (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/m^2 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/m^2 in combination with decitabine

[1]Zeidan A et al., ASH 2019; Abstract #230

Corporate

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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

Co-Research Collaborations

- MIT to evaluate combination of Onvansertib with androgen receptor signaling inhibitors; identification of mechanism of action
- 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

- ~$18.0 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	Event	Timing
Colorectal Cancer: Phase 1b Safety and Efficacy Data	ASCO-GI	January 25th
Prostate Cancer: Phase 2 Efficacy Data	ASCO-GU	February 13th
Colorectal Cancer: Phase 1b Safety and Efficacy Data	AACR	April 27th
Colorectal Cancer: Phase 1b Safety and Efficacy Data	ASCO	May 29th – June 2nd
Acute Myeloid Leukemia: Phase 2 Efficacy Data	EHA	June 11th – 14th
Acute Myeloid Leukemia: Biomarker Data	AACR	June 22nd – 24th
2H2020 Key Inflection Points	**Event**	**Timing**
Prostate Cancer: Phase 2 Efficacy Data	ESMO	Sept. 18th – 22nd
Colorectal Cancer: Phase 2 Efficacy Data	ESMO	Sept. 18th – 22nd
Acute Myeloid Leukemia: Phase 2 Efficacy Data	ESMO	Sept. 18th – 22nd
Prostate Cancer: Phase 2 Efficacy Data	EMUC	Nov. 12th – 15th
Colorectal Cancer: Phase 2 Efficacy Data	ESMO Asia	Nov. 20th – 22nd
Acute Myeloid Leukemia: Phase 2 Efficacy Data	ASH	Dec. 5th – 8th

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Thank You

for more information contact: ir@cardiffoncology.com

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