Cardiff Oncology™

Turning the Tide on Cancer

November 2020

Forward-Looking Statements

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Investment Highlights

3rd Generation, 1st-in-class, Oral PLK1 Inhibitor

Onvansertib overcomes the shortcomings of prior PLK inhibitors:

- Highly selective for PLK1
- Orally administered
- 24-hour half-life
- Flexible dose and schedule

Specifically targets a known mechanism of cell division that is required for tumor cell viability

Preliminary clinical data demonstrate the safety, tolerability and efficacy of onvansertib in combination with SOC across multiple indications

Strong Lead Program in KRAS-mutated mCRC

Supported by compelling preliminary clinical data from a Phase 1b/2 trial showing a ten-fold improvement in ORR compared to SOC

Preclinical data support:

- MOA of synthetic lethality between KRAS mutant mCRC and PLK1 inhibition
- Synergy with irinotecan and 5-FU

First Indication: 2nd line treatment in patients who have failed 1st line treatment with FOLFOX with/without bevacizumab

Integrated Biomarker Strategy

Circulating Tumor DNA: changes in KRAS mutational burden in blood are predictive of subsequent tumor shrinkage (mCRC)

Circulating Tumor Cells: changes are predictive of overcoming anti-androgen resistance (mCRPC)

Circulating Tumor DNA: changes are predictive of decreases in leukemic bone marrow cells (AML)

Diversified Pipeline Across Numerous Cancers

Clinical data from ongoing trials support the use of onvansertib in combination regimens across numerous aggressive cancers:

- mCRC Phase 1b/2 trial
- mCRPC Phase 2 trial
- AML Phase 2 trial

Potential expansion opportunities:

- Chronic myelomonocytic leukemia
- Pancreatic cancer
- Triple negative breast cancer
- Lung cancer
- Ovarian cancer

PLK: Polo-like Kinase; SOC: Standard-of-care; ORR: Overall response rate; MOA: Mechanism of action; mCRC: metastatic colorectal cancer; mCRPC: metastatic castration resistant prostate cancer; AML: Acute myeloid leukemia

Experienced Management Team With Drug Development and Biomarker Technology Expertise

Mark Erlander, PhD
Chief Executive Officer

BIOMÉRIEUX

Johnson & Johnson PHARMACEUTICAL RESEARCH & DEVELOPMENT

Vicki Kelemen
Chief Operating Officer

Bayer HealthCare Pharmaceuticals

Agouron Pharmaceuticals, Inc.

Maxim Pharmaceuticals LTD

AMYLIN

dura PHARMACEUTICALS

Brigitte Lindsay
Vice President of Finance

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ZEISS

PHILIPS RESPIRONICS

Pipeline and Upcoming Catalysts

	Indication	Preclinical	Phase 1b	Phase 2	Next Milestone
Onvansertib Solid Tumor Programs	mCRC	Onvansertib + FOLFIRI/Avastin® in Second-Line KRAS-Mutated Metastatic Colorectal Cancer			Q1 2021 ASCO-GI
	mCRPC	Onvansertib + Zytiga® (abiraterone)/prednisone in Zytiga-Resistant Castration-Resistant Metastatic Prostate Cancer			Q1 2021 ASCO-GU
Onvansertib Hematologic Programs	AML	Onvansertib + Decitabine in Relapsed/Refractory Acute Myeloid Leukemia			Q4 2020 ASH

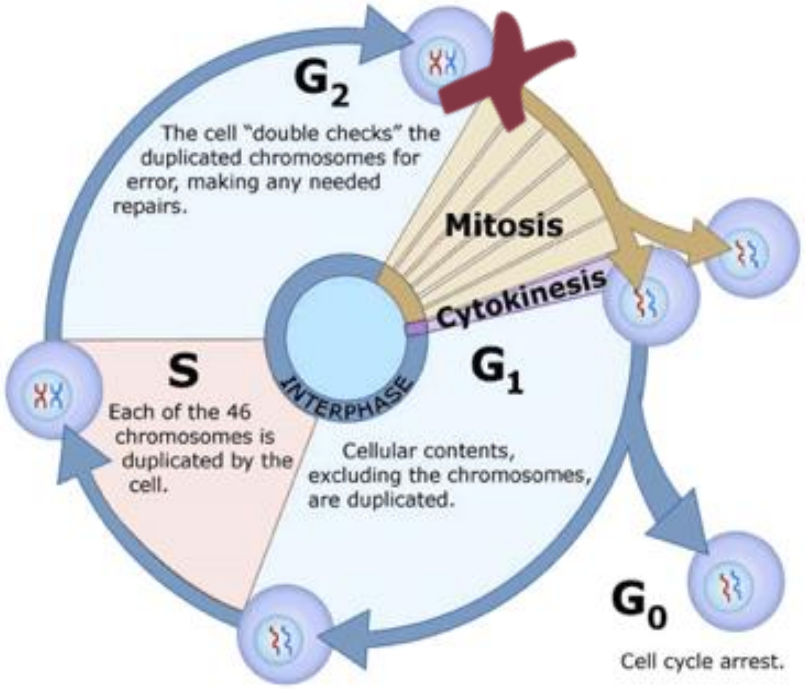
mCRC: Metastatic colorectal cancer; mCRPC: Metastatic castration resistant prostate cancer; AML: Acute myeloid leukemia

Onvansertib

3rd generation, 1st in class, oral and highly selective PLK1 inhibitor addressing unmet needs across a broad range of cancer indications

PLK1 is a Proven Therapeutic Target that is Overexpressed in Most Cancers

- PLK1 is a serine/threonine kinase and master regulator of cell-cycle progression
- PLK1 controls G2/mitosis (G2/M) checkpoint
- Inhibition of PLK1 causes mitotic arrest and subsequent cell death
- Emerging data demonstrate that PLK1 is also a key regulator of cellular functions beyond mitosis that are essential for tumor growth:
  - Biosynthesis of DNA
  - DNA Damage Response



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

[1]Zitouni et al., Nat Rev Mol Cell Biol. 2014 Jul;15(7):433-52; PLK1: Polo-like kinase 1

PLK1-Specific ATP Competitive Inhibitor[1]

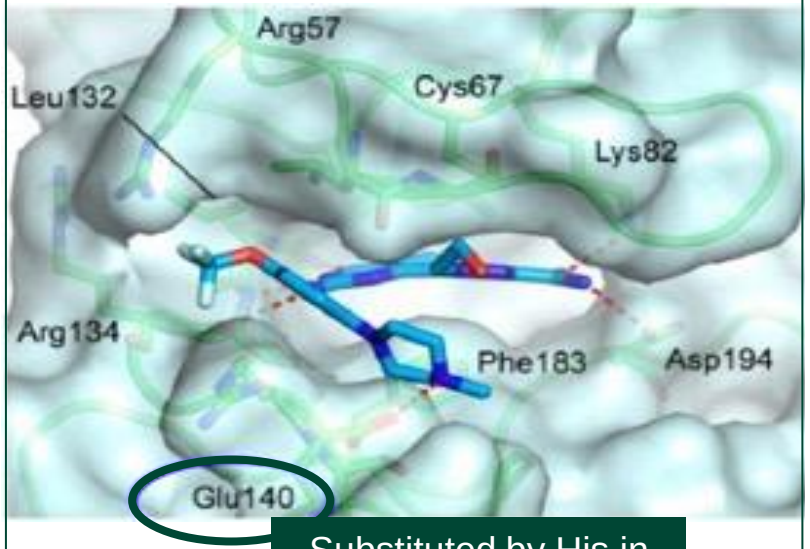
Biochemical Profile

Enzyme	IC_{50} (μM)
PLK1	**0.002**
PLK2	>10
PLK3	>10
CK2	0.4
FLT3	0.4
CDK1/CycB	3.8
42 additional kinases in house	>10
>190 additional kinases in the Millipore panel	>10

Profile Characteristics

Small Molecule	MW 648.60 Daltons
Formulation	5mg and 20mg oral gelcaps
Plasma Protein Binding	95% at 10μM and 91% at 50μM
Metabolic Overview	Moderate intrinsic clearance (9.3 mL/min/kg)[1] 2 metabolites identified in metabolic profiling in low quantities (parent drug accounted for 93% of total drug-related material)[1] No Cytochrome P450 inhibition at therapeutic concentrations[2]
Pharmacokinetics[3]	Systemic exposure of drug increased with dose, as shown by an increase in C_{max} and AUC_{0-24} T_{max} is approximatively 3h Half-life is approximately 24h

Co-crystal of Onvansertib with PLK1



Onvansertib

- A selective, ATP competitive PLK1 inhibitor
- Selectivity is driven by polar interaction with the side chain of Glu140 of PLK1
- Interaction is hampered in both PLK2 and PLK3 where Glu140 is replaced by histidine

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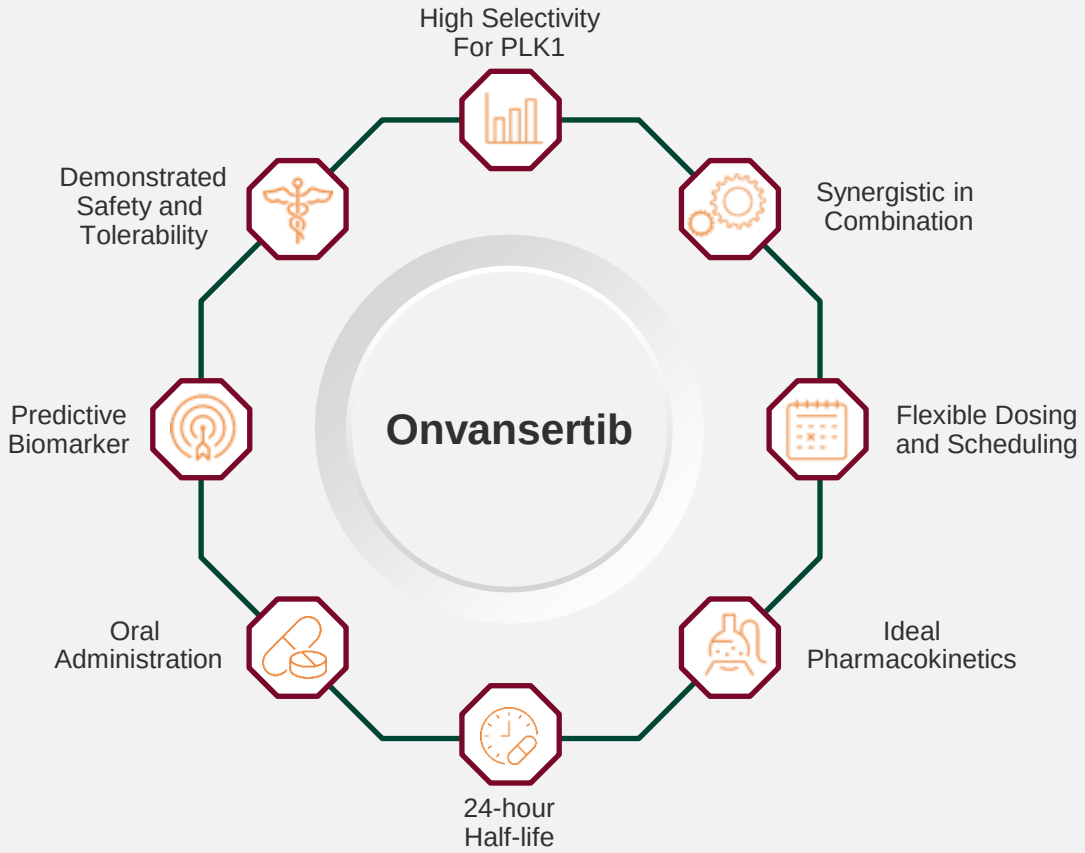
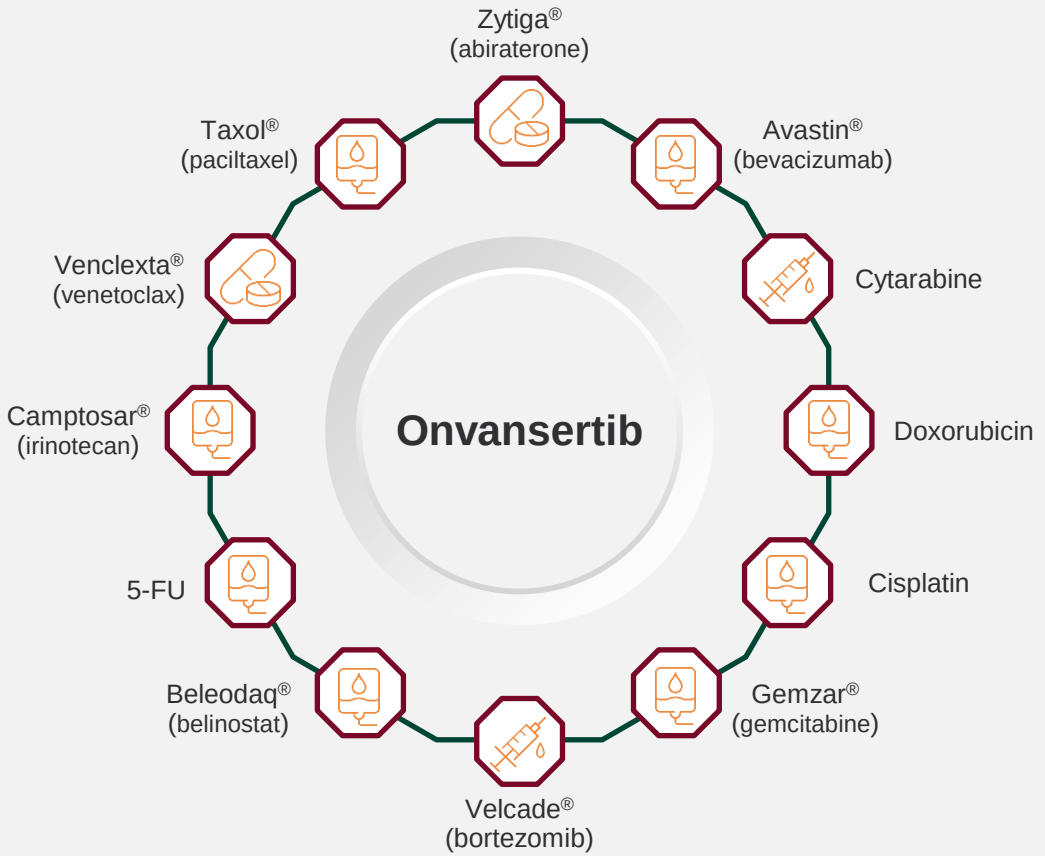
[1]Valsasina Mol Cancer Ther 2012

Onvansertib has Optimal Drug Properties and Synergistically Combines with Standard-of-Care Therapies

Optimal Drug Properties



Synergistic in Combination with Standard-of-Care Chemo and Targeted Therapies



Second-Line Treatment of KRAS-Mutated mCRC

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

Trial Sites: USC Norris Comprehensive Cancer Center; Mayo Clinic Cancer Centers

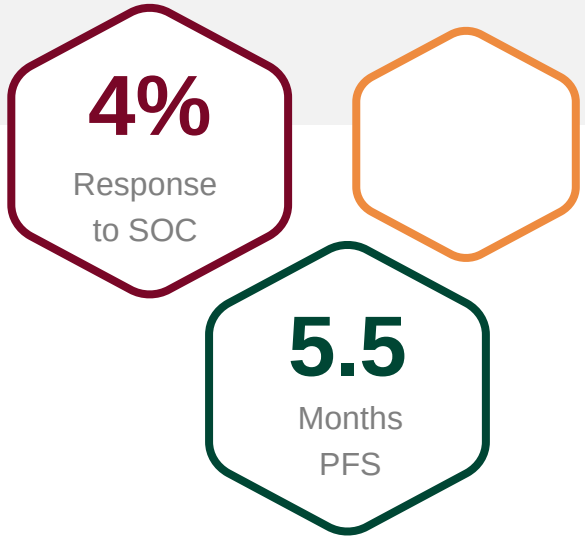
Principal Investigator: Dr. Heinz-Josef Lenz

New Second-Line Therapies are Needed to Improve Response and Increase Progression-Free Survival

50% of patients with mCRC have a KRAS mutation

Prognosis is poor with a five-year survival rate of **10%**

Other drugs currently in development do not address the most prevalent **KRAS mutations in mCRC**



Significant limitations to standard-of-care (SOC)

Current second-line standard-of-care treatment in KRAS-mutated mCRC has an overall response rate of 4% and progression-free survival (PFS) of 5.5 months[1]

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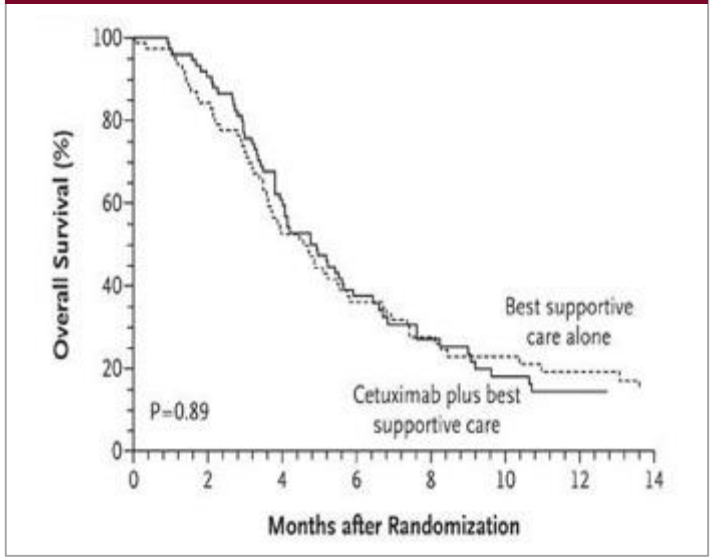
[1]Kubicka et al, Annals of Oncology 2013; 2342–2349; mCRC: Metastatic colorectal cancer

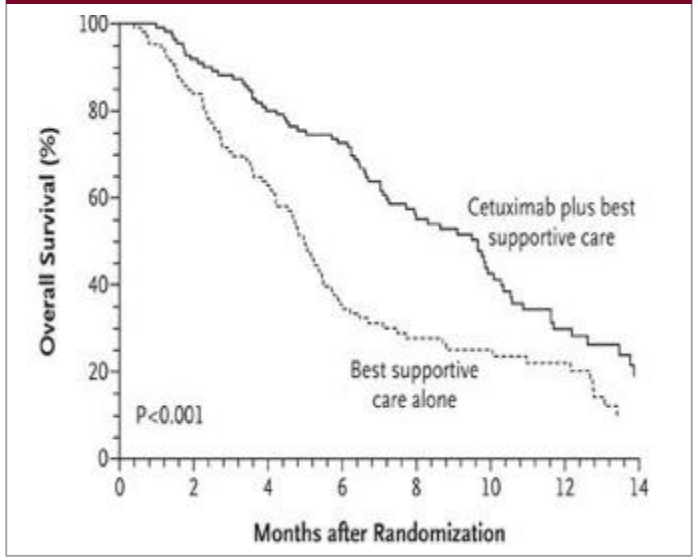
KRAS is a Pivotal Diagnostic Biomarker in the CRC Treatment Paradigm

- KRAS-mutated patients do not benefit from anti-EGFR agents:
  - No increase in OS, PFS and ORR was observed in KRAS mutant patients treated with EGFR inhibitors vs control arm[1,2]
  - The use of anti-EGFRs is therefore limited to KRAS wild-type patients
- Mutations in KRAS represent also the most frequent mechanism of resistance to anti-EGFRs (i.e. cetuximab)

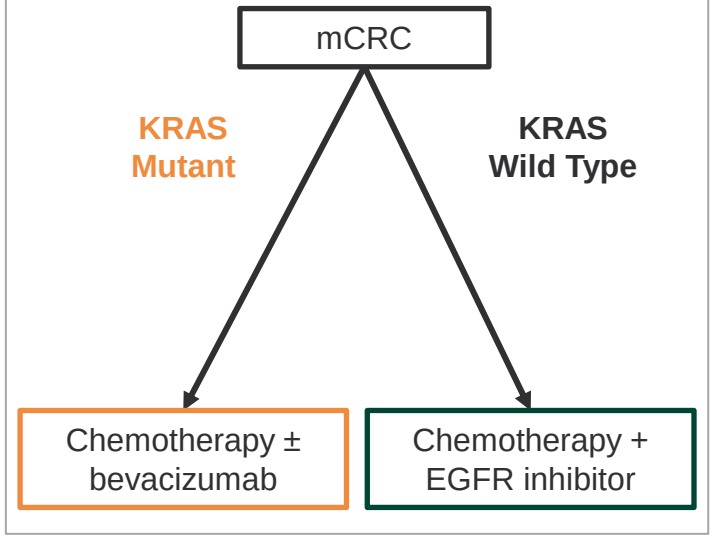
KRAS Mutant



KRAS Wild-type



Treatment Paradigm



[1]Karapetis et al., NEJM 2008;359:1757-1765; [2]Amado et al., JCO 2008, 26:1626-1634

Second-line Treatment: Real World Utilization in the US

Flatiron Health Data

255
Cancer clinics representing
1.7 million active cancer patients

14,315
Colorectal cancer patients

7,034
Colorectal cancer patients who receive second line therapy

	Number	Percent
Second-line regimens after first-line FOLFOX + bevacizumab ($N = 2470$)		
FOLFIRI + bevacizumab	1176	47.6
FOLFIRI	262	10.6
FOLFIRI + cetuximab	159	6.4
FOLFIRI + Ziv-aflibercept	114	4.6
FOLFIRI + panitumumab	91	3.7
Irinotecan + cetuximab	85	3.4
FOLFIRI + ramucirumab	74	3.0
Irinotecan + bevacizumab	53	2.1
Regorafenib	33	1.3
Irinotecan	30	1.2
Panitumumab	27	1.1
Second-line regimens after first-line FOLFOX ($N = 1249$)		
FOLFOX + bevacizumab	373	29.9
FOLFIRI	235	18.8
FOLFIRI + bevacizumab	210	16.8
Fluoropyrimidine + bevacizumab	85	6.8
FOLFIRI + cetuximab	43	3.4
FOLFOX + panitumumab	37	3.0
Irinotecan + cetuximab	24	1.9
FOLFIRI + panitumumab	23	1.8
FOLFOX + cetuximab	22	1.8
Bevacizumab	17	1.4
Irinotecan	15	1.2
Panitumumab	15	1.2
FOLFOXIRI + bevacizumab	14	1.1

Denotes combination with bevacizumab

Denotes combination with other antiangiogenics

Source: Hess, L. International Journal of Colorectal Disease; 2019. Data is limited to limited to second-line regimens used in >1% of the cohort. FOLFOX: fluoropyrimidine, leucovorin, oxaliplatin. FOLFIRI: fluoropyrimidine, leucovorin, irinotecan, FOLFOXIRI: fluoropyrimidine, leucovorin, irinotecan, oxaliplatin

New Second-line mCRC Treatment is an Unmet Need

Outcomes for patients in the 2nd line setting is poor

- Efficacy of FOLFIRI: 4% ORR and 2.5 months PFS[1]
- Addition of bevacizumab to FOLFIRI improves outcomes[2]
- However, while KRAS WT patients benefit from the addition of bevacizumab, there was no statistically significant improvement in OS for KRAS-mutant patients[3]

KRAS	Treatment	ORR	PFS (months)	HR and significance of PFS	OS (months)	HR and significance of OS
KRAS WT	FOLFIRI	5%	4.5	HR=0.61 (95 % CI 0.49-077) P <0.0001	11.1	HR=0.61 (95 % CI 0.53-0.90) P=0.0052
	FOLFIRI + Bev	7%	6.4		15.4	
KRAS MUTANT	FOLFIRI	3%	4.1	HR=0.70 (95 % CI 0.56-0.89) P = 0.0027	**10**	**HR=0.92 (95 % CI 0.71-1.18) P=0.4969**
	FOLFIRI + Bev	4%	5.5		**10.4**	

[1]Tournigand et al., JCO 2004;22(2):229-3; [2]Bennouna et al., Lancet Oncol. 2013; 14(1):29-37; [3]Kubicka, S, Annals of Oncology 2013, 24:2342-2349; CI: confidence interval, HR: hazard ration, ORR: objective response rate, PFS: progression-free survival, OS: overall survival, WT: wild-type, MUT: mutant

Magnitude of Response with Other Antiangiogenic Therapy

The anti-angiogenic agents aflibercept and ramucirumab have been approved in combination with chemotherapy in 2nd-line treatment, although they are used to a much lesser extent than bevacizumab

Trial	Agent/ARM	Patients	ORR	95% CI of ORR (%)
VELOUR Sub-group[1] (received first line therapy and bevacizumab)	FOLFIRI + aflibercept	643 (325 FOLFIRI +aflibercept)	11.8%	6.7 - **16.9**
RAISE[2]	FOLFIRI + ramucirumab	1361	13.4%*	10.7-**16.6**

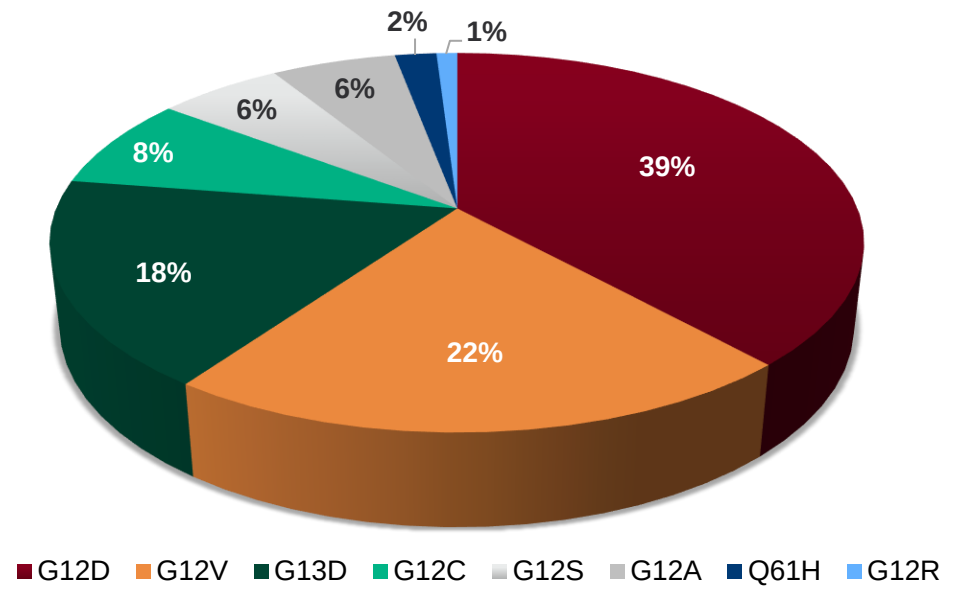
* 20% of patients were Asian, which has higher response rate

[1]Van Cutsem et al., Target Oncology 2016, 11:383-400 [2]Tabernero et al., Lancet Oncology 2015;16:499-508

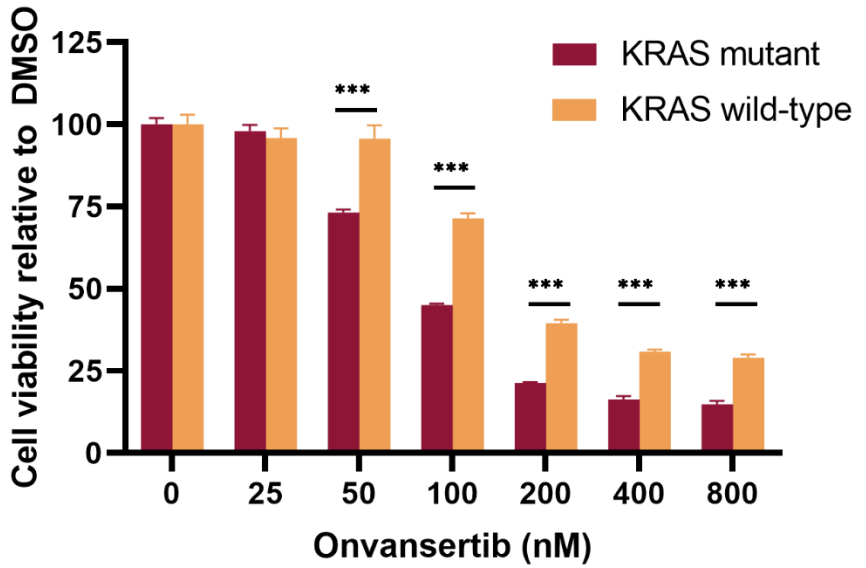
Synthetic Lethality: Cells with KRAS Mutations are Hypersensitive to Inhibition of PLK1

The output of the RAS-mutated pathway activates PLK1, which is inhibited by onvansertib

Onvansertib Addresses KRAS Mutation Subtypes in mCRC



Cell Viability in Onvansertib-Treated KRAS Mutant and Wild Type Isogenic CRC Cells



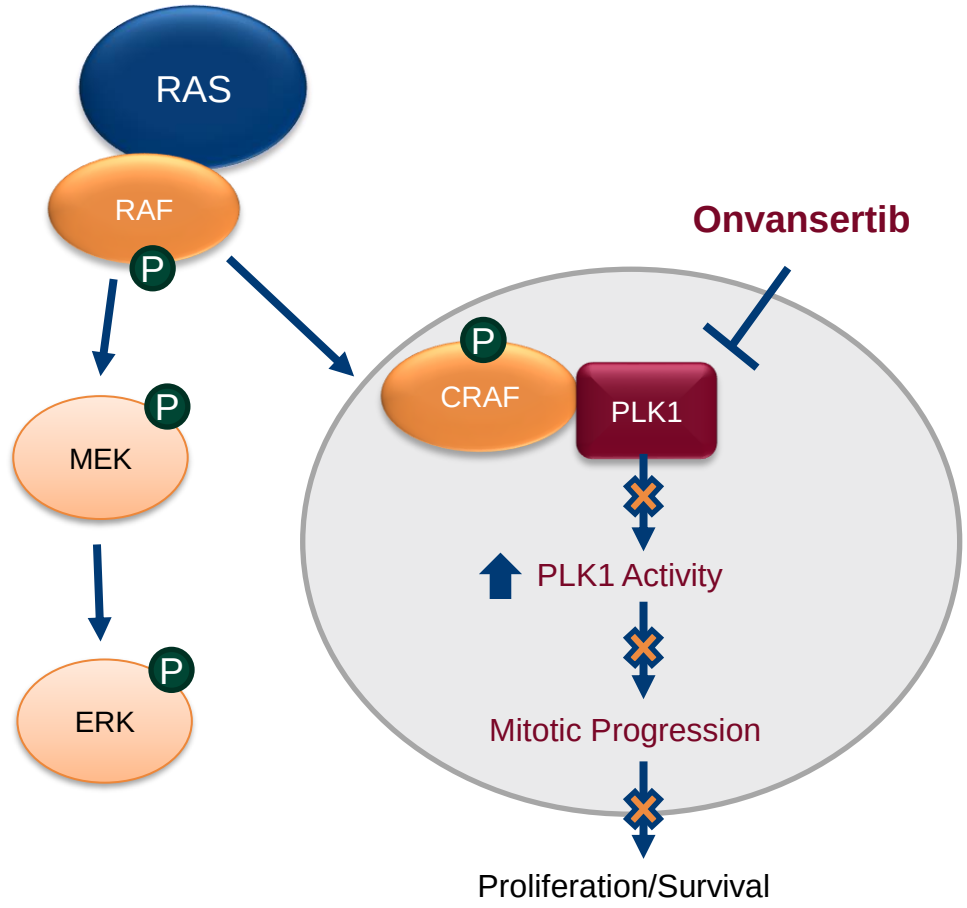
PLK1: Polo-like Kinase 1; mCRC: Metastatic colorectal cancer

PLK1 and RAS Cooperative Relationship

RAS activates PLK1 through a MEK/ERK-independent mechanism

The downstream target of KRAS, pCRAF, localizes to the mitotic spindle poles at mitosis where it interacts with PLK1 and promotes PLK1 activation, leading to mitosis and tumor progression[1]

Data suggest that KRAS-activated cells are dependent on PLK1 for their proliferation and survival and inhibition of PLK1 by onvansertib could inhibit tumor growth



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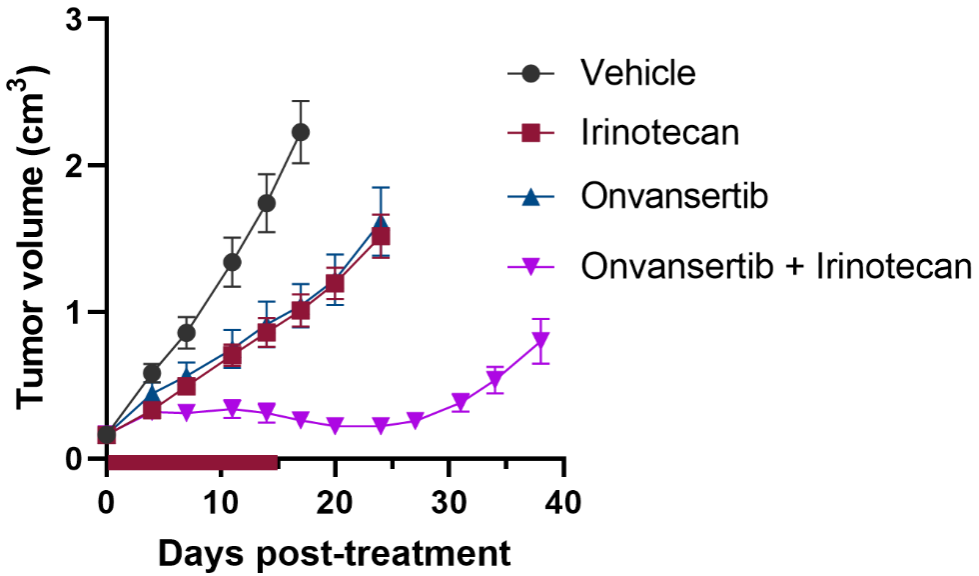
[1]Mielgo et al., Nat. Med. 2011; 17(12):1641-5

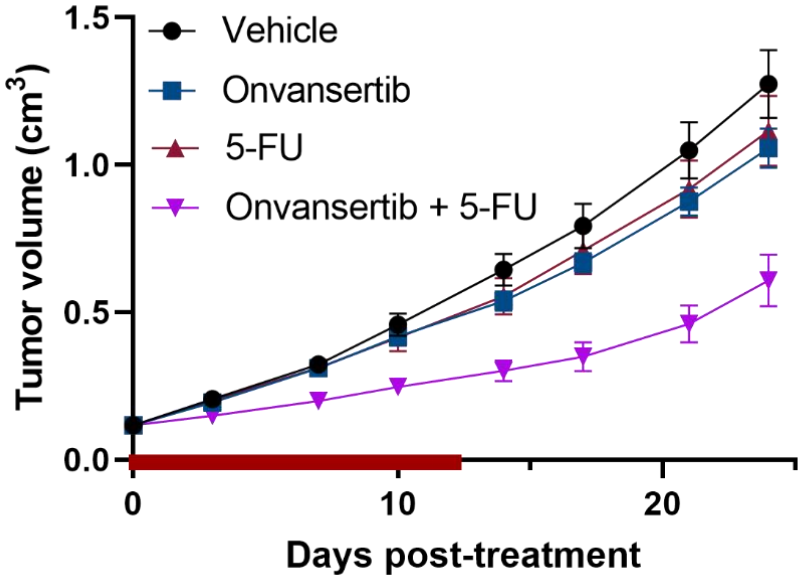
Synergy: Onvansertib in Combination with SOC Irinotecan and 5-FU

Onvansertib works synergistically in combination with standard-of-care FOLFIRI (irinotecan and 5-FU)

Synergy in Combination with Irinotecan

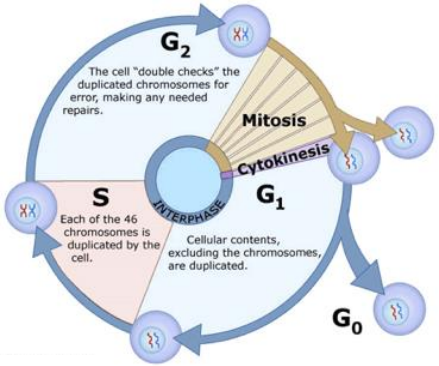


Synergy in Combination with 5-FU



PLK1 Regulates DNA Damage Response[1,2]

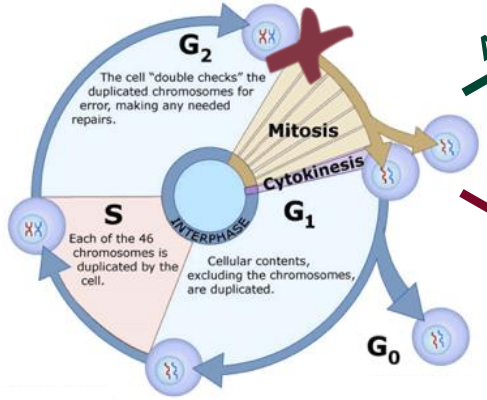
DNA Damaging Agents



- Irinotecan
- 5-FU

DNA Damage Response (DDR) arrests cells at G2/M checkpoint

G2/M Arrest



Active PLK1

PLK1 Inhibited

Mitosis

1. Checkpoint adaptation
2. PLK1 inhibits DDR, induces mitotic entry for tumor cells & cell division

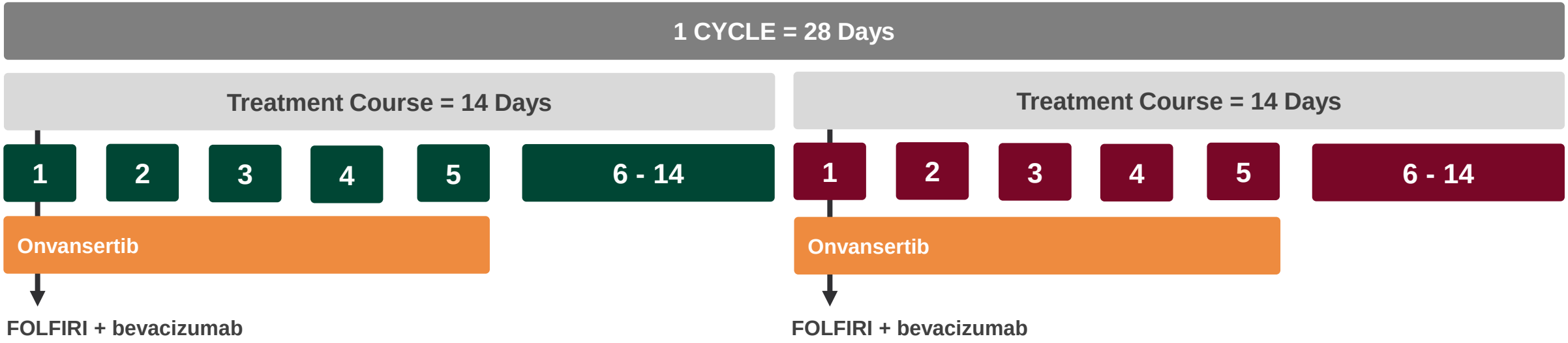
Cell Death

1. Keeps tumor cells in G2/M arrest leading to apoptosis
2. For cells that escape, mitosis is blocked, also leading to apoptosis

[1]van Vugt & Yaffe, Cell Cycle 2010 9:2097-2101; [2]van Vugt et al., 2010, PLoS 8:1-19

Phase 1b/2 Open Label Trial of Onvansertib + FOLFIRI/bevacizumab

Trial Design



Efficacy Endpoints

- Overall response in patients who receive ≥1 cycle (2 courses) of treatment
- Progression-free survival (PFS)
- Decreases in KRAS mutation burden and response to treatment

What is Clinical Trial Success

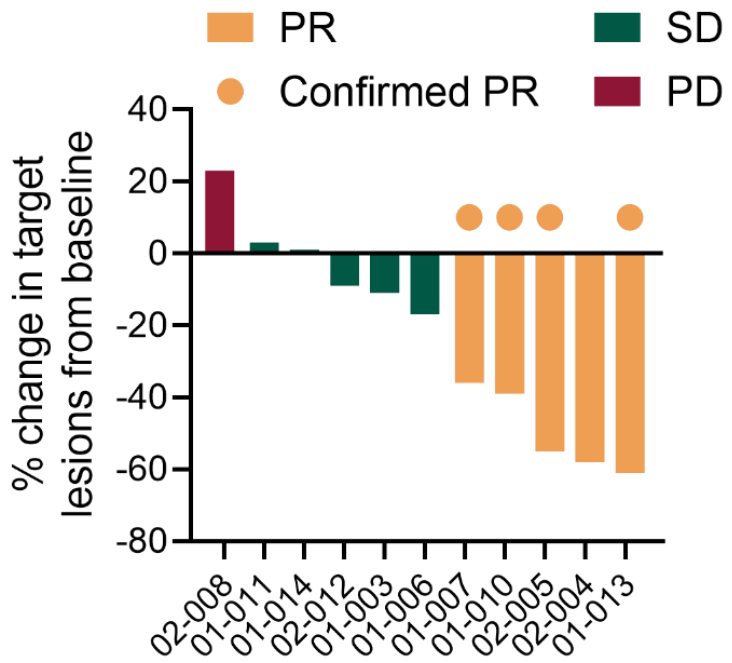
- ≥5 of 26 (~20%) patients achieve clinical response confirmed by radiographic scan
- Achieve median progression-free survival of ≥ 6 months

Response to Treatment Confirmed by Radiographic Scan

Compelling Preliminary Efficacy Data

- 10 of 11 (91%) patients had clinical benefit:
  - 5 (45%) patients achieved a partial response (PR)
  - 4 patients had a confirmed PR (≥ 30% tumor shrinkage) with 1 patient going on to curative surgery
  - 1 patient with an initial PR went off study prior to confirmatory scan due to non-treatment related event

Best Radiographic Response

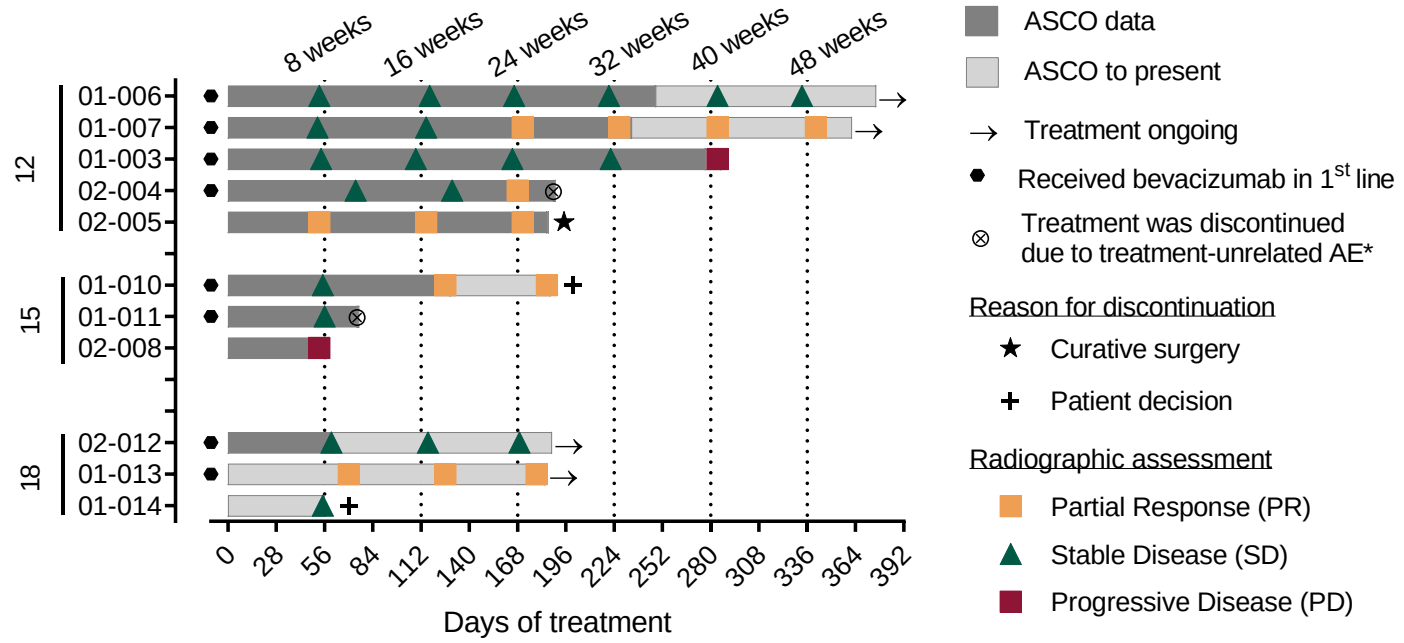


Response to Treatment Confirmed by Progression-Free Survival

Response Appears Durable

- 8 (73%) patients had durable responses of >6 months (range 6 to >12 months); 4 patients remain on treatment; median PFS has not yet been reached
- Only 1 patient progressed in <6 months while on treatment

Durability of Response



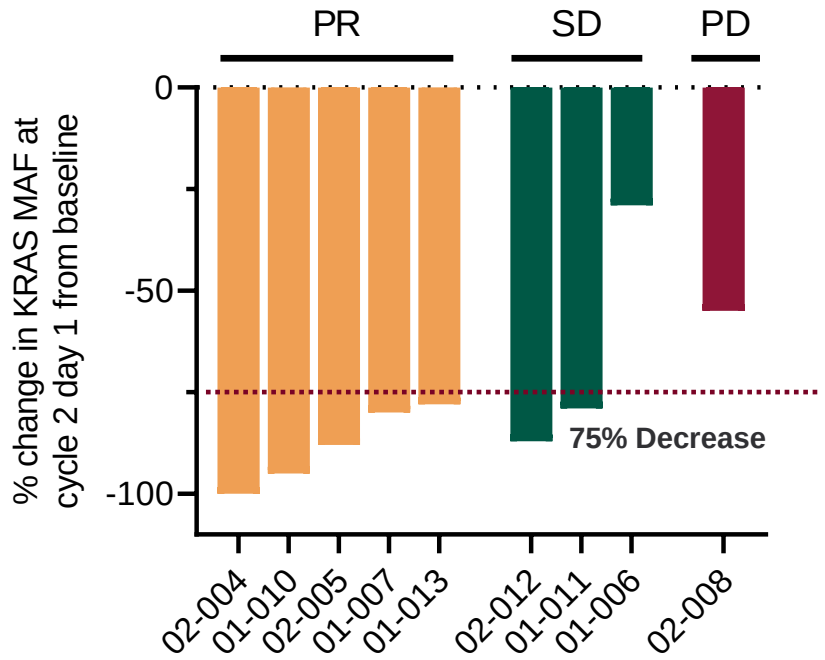
PFS: Progression-free survival

Serial Monitoring of KRAS is Predictive of Radiographic Scan Response

Monitoring KRAS mutations in plasma ctDNA may enable rapid predictions of therapeutic response

- KRAS mutant allelic frequency (MAF) was measured by digital droplet PCR (ddPCR) at baseline and at the end of Cycle 1
  - 9 of 11 patients had a KRAS variant detected by ddPCR at baseline*
  - All patients showed a decrease in KRAS MAF after the 1st cycle of treatment
- The greatest changes in KRAS were observed in patients achieving a PR (ranging from -78% to -100%)
- The patient with disease progression had only a 55% decrease in KRAS mutant allelic frequency

% KRAS MAF Changes After Cycle 1



*A KRAS mutation was detected by NGS for all 11 patients; ctDNA: Circulating tumor DNAPR: Partial response; SD: Stable disease; PD: Progressive disease

KRAS-Mutated mCRC Expanded Access Program (EAP)

- Program initiated in June 2020
- 15 sites participating across the US as of October 30
- Eligibility criteria includes:
  - Patients not meeting clinical trial inclusion criteria
  - Patients who have received 2 or more lines of prior treatment
  - Patients who have previously been treated with FOLFIRI (with or without bevacizumab)
- 19 patients treated to-date; most were progressing on treatment with FOLFIRI/bevacizumab prior to enrolling
- Changes in KRAS mutational burden is being analyzed pre-dose and at the start of each cycle of treatment

# of Sites	# of Patients Treated To-Date	# of Patients Pending Treatment
15	19	3

Catalysts and Milestones: KRAS-Mutated mCRC

Positive Phase 1b/2 results may provide an opportunity for a Phase 2b registrational trial

May 2020: Fast Track Designation

September 2020: ESMO presentation

January 2021: ASCO-GI data presentation (planned)

Q1 2021: FDA meeting to discuss regulatory path (anticipated)

mCRC: Metastatic colorectal cancer

Metastatic Castration-Resistant Prostate Cancer

Phase 2 open-label trial of onvansertib + abiraterone

Trial Sites: Beth Israel Deaconess, Dana Farber, Mass General

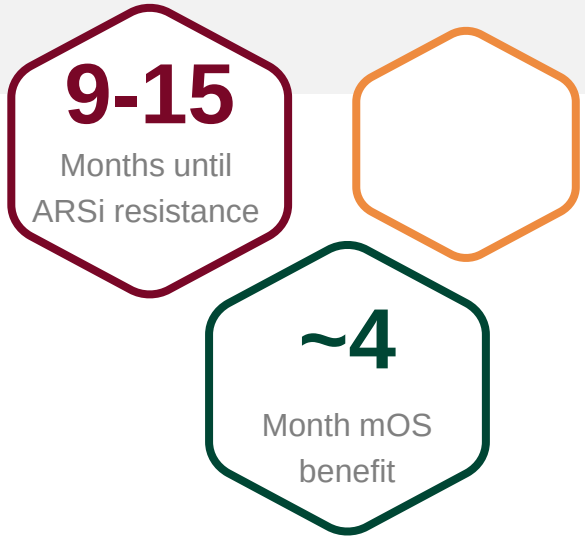
Principal Investigator: Dr. David Einstein

New Therapeutic Options are Needed to Overcome Resistance to SOC Androgen Receptor Signaling Inhibitors (ARSi)

Resistance develops to treatment with standard of care ARSi's within 9-15 months[1]

ARSi's offer a median overall survival (mOS) benefit of **only ~4 months**[1]

No effective treatment options are available for the up to 40% of mCRPC patients with an AR-V7 mutation[2]



Limited options for patients once resistant to abiraterone

New treatment options are needed to extend the duration of response to ARSi's and increase overall survival

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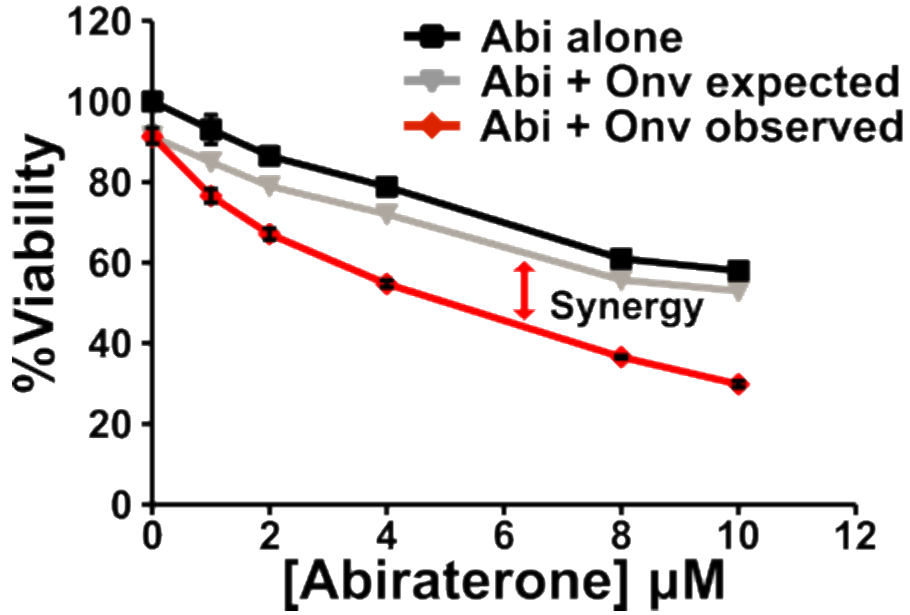
[1]Antonarakis, Emmannel – Current Understanding of Resistance to Abiraterone and Enzalutamide in Advanced Prostate Cancer; Clinical Advances in Hematology & Oncology – May 2016 – Volume 14, Issue 5; [2]Armstrong et al., 2019, JCO 37: 1120-1129; SOC: Standard-of-care; mCRPC: Metastatic castration resistant prostate cancer

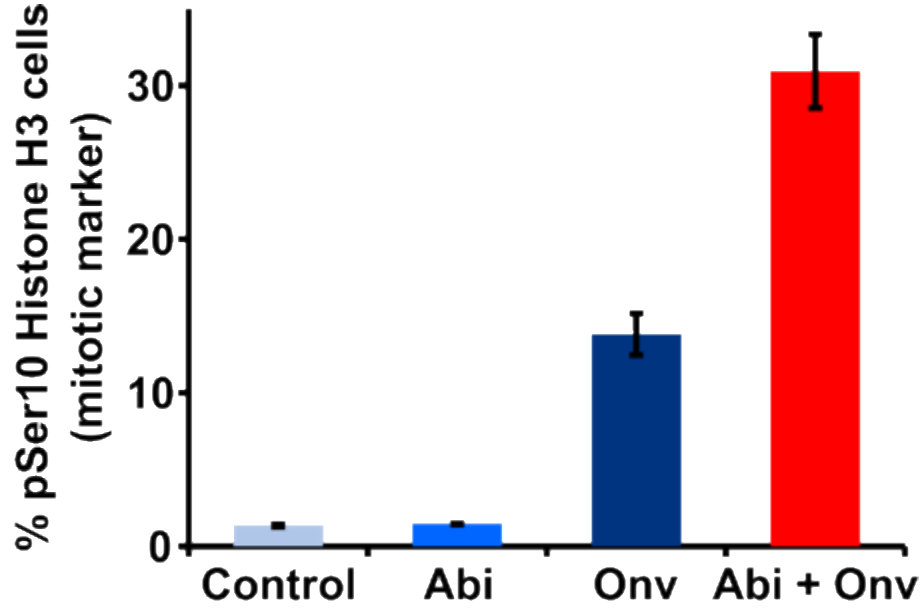
Onvansertib Extends the Response to Androgen Receptor Signaling Inhibitors

Onvansertib works synergistically in combination with abiraterone (Zytiga®) and significantly increases mitotic arrest

Onvansertib + Abiraterone (Zytiga®) Demonstrate Synergy in mCRPC model (C4-2)[1]



Onvansertib + Abiraterone (Zytiga®) Significantly Increase Mitotic Arrest[1]



[1]Patterson & Yaffe, 2019, MIT; mCRPC: Metastatic castration resistant prostate cancer

Phase 2 Open Label Trial in of Onvansertib + Abiraterone

Disease Control Assessed by PSA Stabilization

Trial Design:

	Dosing Schedule	Duration	Efficacy Endpoint
Cohort A (n = 24) Cohort Closed	Onvansertib 24mg/m^2 Days 1-5 (21-day cycle) + Zytiga® (Abiraterone)	4 Cycles = 12 Weeks	Disease Control (PSA Stabilization or Decline)
Cohort B (n = 32)	Onvansertib 18mg/m^2 Days 1-5 (14-day cycle) + Zytiga® (Abiraterone)	6 Cycles = 12 Weeks	Disease Control (PSA Stabilization or Decline)
Cohort C (n = 32)	Onvansertib 12mg/m^2 Days 1-14 (21-day cycle) + Zytiga® (Abiraterone)	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

- **Primary:** disease control evaluated as PSA decline or stabilization (PSA rise <25% over baseline)

What is Clinical Trial Success

- ~30% patients achieve primary efficacy endpoint of disease control at 12 weeks (PSA stabilization or decrease); confirmed by radiographic scan
- Achieve median radiographic PFS 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]; mCRPC: Metastatic castration resistant prostate cancer; PSA: Prostate specific antigen; PFS: Progression-free survival

Patient Baseline Characteristics and Enrollment Status

Patient Baseline Characteristics

Total patients N=39	Median [range] or n (%)
Age in Years	72 [54-87]
Nonwhite Ethnicity	5 (13%)
ECOG 0 1	 34 (87%) 5 (13%)
Years Since Diagnosis	5 [1-18]
Grade Groups 4 and 5	24 (62%)
De Novo Metastatic Disease	13 (33%)
Presence of Bone Metastasis	33 (85%)
Presence of Visceral Metastasis	13 (33%)
Baseline PSA, ng/mL	12.5 [0.6-224]
AR-V7^{+} at Baseline*	9 (23%)
Baseline CTC Count per mL of blood**	2.2 [0-87]

ECOG: Eastern Cooperative Oncology Group, AR-V7: androgen receptor variant 7, CTC: circulating tumor cells

*AR-V7 status was evaluated using the EPIC and Johns Hopkins University testing platforms **CTC count was performed by EPIC

Enrollment as of October 16th, 2020

Number of patients (N)	Arm A	Arm B	Arm C
Treated	24	11	4
Currently on Treatment	1	1	4
Completing 12-weeks	14	8	3
Discontinued before 12 weeks	10	2	0
Progressive Disease (PD) Adverse Event Withdrew Consent	3 5 2	1 1 0	0 0 0
Patients evaluable for efficacy (completed 12 weeks + PD)	17	9	3

Phase 2 Data Demonstrate the Safety and Efficacy of Onvansertib in mCRPC

Safety Assessment

Adverse events Total Patients N=39	Grade 1	Grade 2	Grade 3	Grade 4	All grades
Anemia	10	5	1		16
Thrombocytopenia	11	1		1	13
Fatigue	10	2			12
Neutropenia	1	1	7	3	12
Hypophosphatemia	3	3	4		10
WBC decrease	2	2	3	2	9
Back pain	2	3			5
Hypokalemia	3	1	1		5
Constipation	4	0			4
Nausea	3	1			4

- Most frequent Grade 3 and 4 adverse events (AEs) were expected, on-target, hematological associated with onvansertib mechanism of action
- Hematological AEs were reversible and effectively managed by dose delay, dose reduction and/or growth factor support

Efficacy Evaluation at 12-Weeks

Total Patients Evaluable N=29	Arm A	Arm B	Arm C
Evaluable for efficacy*	17	9	3
Completed 12-week treatment	14	8	3
Progressed within 12 weeks	3	1	0
Disease control**	5 (29%)	3 (33%)	2 (67%)
Radiographic stable disease	9 (53%)	5 (55%)	3 (100%)
Durable response (>7 months)	4 (23%)	4 (44%)	NA

* Completed 12 weeks of treatment or progressed within 12 weeks
** Defined as PSA stabilization or decline (PSA rise <25% over baseline)

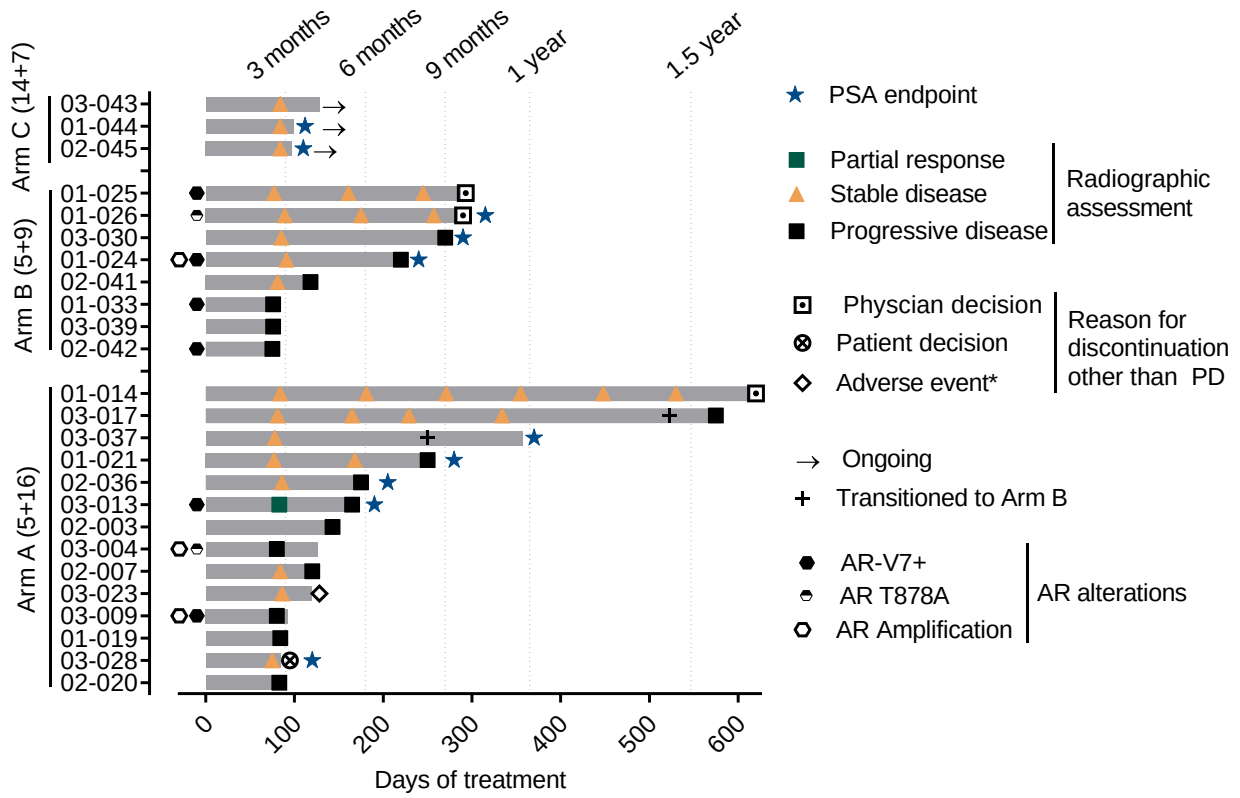
Phase 2 Data Demonstrate the Efficacy of Onvansertib and and Durability of Response Including Patients with AR Alterations

Efficacy in patients with AR alterations:

- 8 of the patients evaluable for efficacy had at least 1 AR alterations: AR-V7+ (n=6), AR T878A mutation (n=2) and/or AR amplification (n=3)
- 3 (37%) patients achieved disease control
- 4 (50%) patients had radiographic stable disease
- 3 patients had durable responses (range 7-9 months)

*AR-V7 status was evaluated using the EPIC and Johns Hopkins University testing platforms
Genomic profiling of circulating tumor DNA was performed using Gardant360® test

Treatment Response and Duration for Patients Completing 12 Weeks of Treatment



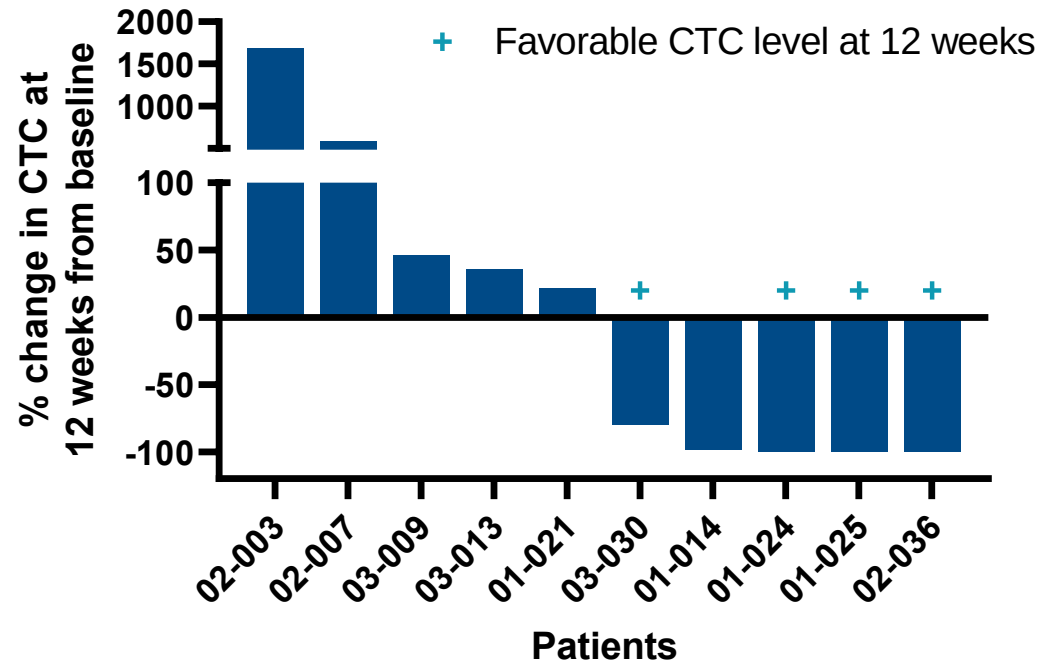
Onvansertib-Induced Circulating Tumor Cell Decrease is Associated with Progression-Free Survival

Circulating tumor cell (CTC) count, reported as favorable or unfavorable (<5 versus ≥5 CTC/7.5mL of blood, respectively) is a prognostic factor for survival in CRPC – conversion from unfavorable to favorable is associated with improved survival

At baseline, 27 (73%) of 37 patients had unfavorable CTC count; 10 were analyzed following 12 weeks of treatment:

- 5 (50%) patients had an ≥80% CTC decrease, including 2 AR-V7+ patients (01-024 and 01-025)
- 4 (40%) patients converted from unfavorable to favorable CTC level, including 3 patients with no detectable CTC
- Median time on treatment was 9.2 months for patients with CTC decrease (n=5) vs 4.9 months for patients with CTC increase (n=5)

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



CRPC: Castration resistant prostate cancer

Identifying an Onvansertib-Abiraterone Response Gene Signature

Onvansertib/Abiraterone

- Synergy study
- RNA-sequencing

Abiraterone induces expression of mitotic genes in prostate cancer cells synergistic for Onv+Abi

Identification of an Abi/Onv synergy gene signature

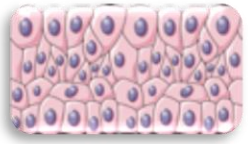
DECIPHER BIOSCIENCES

Transcriptome analysis of 32,000 prostate cancer specimens

Identified 4 molecular subtypes:
- Luminal A
- Luminal Proliferating
- Basal
- Basal Immune

Abi/Onv synergy gene signature is enriched in the Basal subtype, a subtype representing ~30% of CRPC patients and associated with lower response to androgen deprivation therapy (ADT)

Currently analyzing archived tissue from patients enrolled in the trial



Transcriptome analysis with Decipher Biosciences

Correlate clinical response with Basal molecular subtype

Catalysts and Milestones: mCRPC

Positive Phase 2 results may provide an opportunity for a Phase 2b registrational trial

October 2020: Prostate Cancer Foundation (PCF)

February 2021: ASCO-GU presentation (planned)

April 2021: AACR presentation (planned)

Q3 2021: FDA meeting to discuss regulatory pathway (anticipated)

New Clinical Programs Planned

Chronic Myelomonocytic Leukemia (CMML)

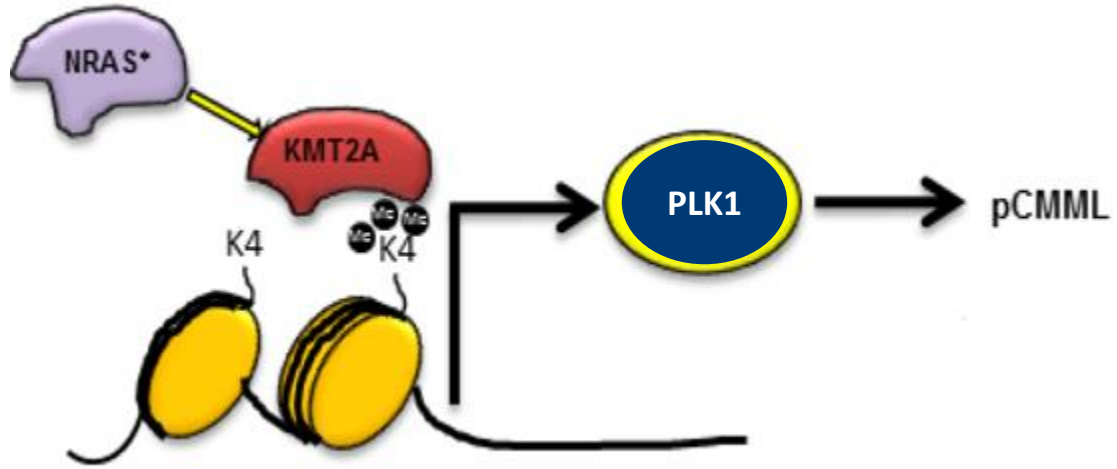
Pancreatic Ductal Adenocarcinoma (PDAC)

Phase 2 Study to Evaluate the Safety and Efficacy of Onvansertib in RAS-pathway Mutant CMML

Study Rationale

- Proliferative CMML is enriched for activating RAS pathway mutations such as NRAS, KRAS, CBL, PTPN11 and NF1, all of which have been associated with adverse outcomes
- RAS pathway mutations drive proliferative CMML via a novel RAS-KMT2A-PLK1 axis, which can be therapeutically targeted with PLK1 inhibitors
- In-vitro and in-vivo experiments with onvansertib as a single agent have shown a dose-dependent inhibition of CMML cell growth, with improved cell differentiation

Activating RAS Pathway Can Be Therapeutically Targeted with PLK1 Inhibitors



Phase 2 Two-Arm Randomized Trial of Onvansertib +/- Decitabine in RAS-Pathway Mutated CMML

Determine the safety and overall response rate (ORR) of onvansertib, a novel oral PLK1 inhibitor in RAS-pathway mutant chronic myelomonocytic leukemia

Trial Design:

	Dosing Schedule	Duration	Efficacy Endpoint
Two Arms: **Arm A (n=38) Treatment Naïve** **Arm B (n=38) Relapsed/Refractory**	Onvansertib 12mg/m^2 Days 1-14 (21-day cycle)	3 cycles monotherapy (option to add decitabine at cycle 4 if lack of efficacy with single agent)	Interim analysis of first 18 patients after 3 cycles to evaluate objective response

Eligibility Criteria:

- Newly diagnosed or relapsed/refractory to prior therapy
- RAS pathway mutant: NRAS, KRAS, PTPN11, CBL and NFI with frequency allele of ≥5%

Efficacy Endpoint:

- Overall response rate following single agent treatment with onvansertib

What is Clinical Trial Success

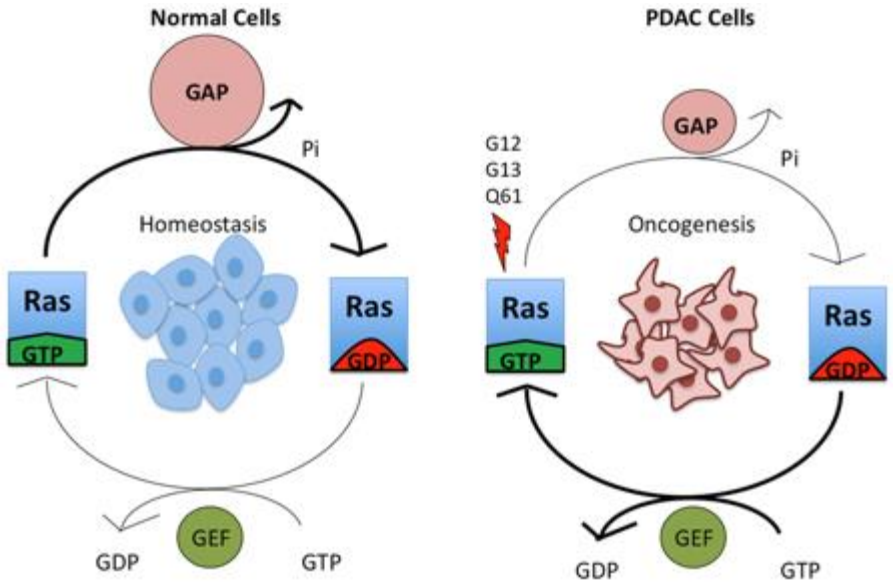
- To be established and confirmed in collaboration with investigators and statistician

Phase 2 Study of Onvansertib in Combination with 5-FU and Nal-IRI for Second Line Treatment of KRAS-Mutated Metastatic Pancreatic Ductal Adenocarcinoma (PDAC)

Study Rationale

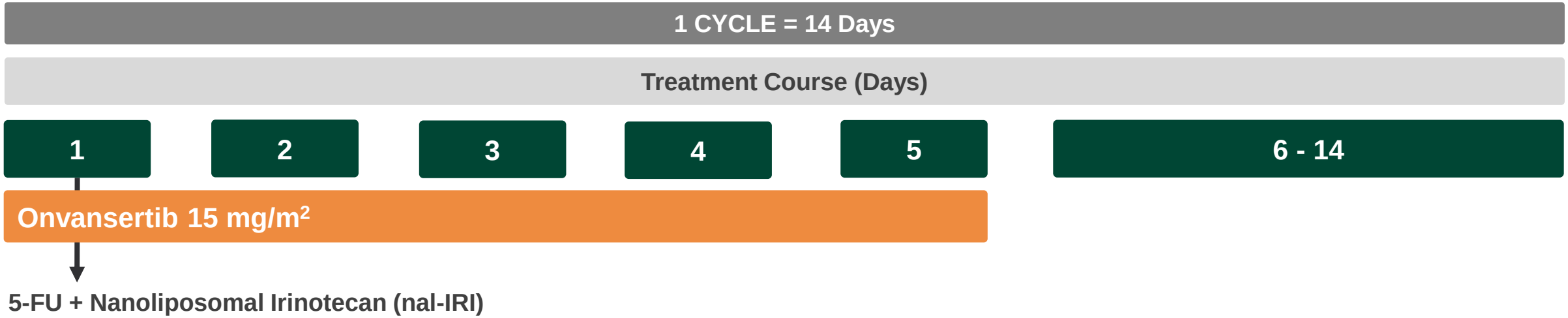
- KRAS is the most common oncogene mutated in pancreatic adenocarcinoma, which is present in ~95% of tumors
- Mutant KRAS is essential for PDAC growth, where the constitutive activated RAS proteins contribute to tumorigenesis, treatment resistance and metastases
- No effective RAS inhibitors have been approved for the treatment of KRAS-mutated pancreatic cancer
- Significant need for new effective second line treatment option

Mutant KRAS is Essential for PDAC Growth – Activated RAS Proteins Contribute to Tumorigenesis



Phase 2 Open Label Trial of Onvansertib + 5-FU and Nanoliposomal Irinotecan in KRAS-Mutated PDAC

Trial Design (~40 patients):



Eligibility Criteria

- Prior abraxane/gemcitabine and no prior irinotecan or Nal-IRI

Efficacy Endpoints

- Best overall response (complete response [CR] or partial response [PR]) and disease control rate (CR, PR or stable disease [SD])
- Progression-free survival (PFS) rate at 6 months
- Overall survival (OS)
- Reduction in KRAS allelic burden in liquid biopsies

What is Clinical Trial Success

- Achieve ≥26% overall response rate (ORR) – 9 out of 35 patients
- Achieve ≥36% progression free survival rate at 6 months – 13 out of 36 patients

Corporate

Strong Patent Portfolio

Core Technology: 3 Issued Patents to 2030 in US, Europe and Asia, with anticipated extension to 2035

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

PLK: Polo-like kinase; PSA: Prostate specific antigen

Cardiff Oncology At-A-Glance

Clinical-stage biotech company, developing **onvansertib,** an oral, highly-selective Polo-like Kinase 1 (PLK1) inhibitor, to treat cancers with the greatest medical need for new effective therapies

Exchange	Nasdaq: CRDF
Cash & Cash Equivalents (as of 10/31/20)	$131.8M
Q1 – Q3, 2020 Average Quarterly Cash Burn	$3.8M
Headquarters	San Diego, CA

Investment Highlights

3rd Generation, 1st-in-class, Oral PLK1 Inhibitor

Onvansertib overcomes the shortcomings of prior PLK inhibitors:

- Highly selective for PLK1
- Orally administered
- 24-hour half-life
- Flexible dose and schedule

Specifically targets a known mechanism of cell division that is required for tumor cell viability

Preliminary clinical data demonstrate the safety, tolerability and efficacy of onvansertib in combination with SOC across multiple indications

Strong Lead Program in KRAS-mutated mCRC

Supported by compelling preliminary clinical data from a Phase 1b/2 trial showing a ten-fold improvement in ORR compared to SOC

Preclinical data support:

- MOA of synthetic lethality between KRAS mutant mCRC and PLK1 inhibition
- Synergy with irinotecan and 5-FU

First Indication: 2nd line treatment in patients who have failed 1st line treatment with FOLFOX with/without bevacizumab

Integrated Biomarker Strategy

Circulating Tumor DNA: changes in KRAS mutational burden in blood are predictive of subsequent tumor shrinkage in mCRC

Circulating Tumor Cells: changes are predictive of overcoming anti-androgen resistance in mCRPC

Circulating Tumor DNA: changes are predictive of decreases in leukemic bone marrow cells

Diversified Pipeline Across Numerous Cancers

Clinical data from ongoing trials support the use of onvansertib in combination regimens across numerous aggressive cancers:

- mCRC Phase 1b/2 trial
- mCRPC Phase 2 trial

Potential expansion opportunities:

- Chronic myelomonocytic leukemia
- Pancreatic cancer
- Triple negative breast cancer
- Lung cancer
- Ovarian cancer

PLK: Polo-like Kinase; SOC: Standard-of-care; ORR: Overall response rate; MOA: Mechanism of action; mCRC: metastatic colorectal cancer; mCRPC: metastatic castration resistant prostate cancer; AML: Acute myeloid leukemia

Cardiff Oncology™

Thank You

for more information contact:

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