Cardiff Oncology™

Company Overview
The Onvansertib Opportunity

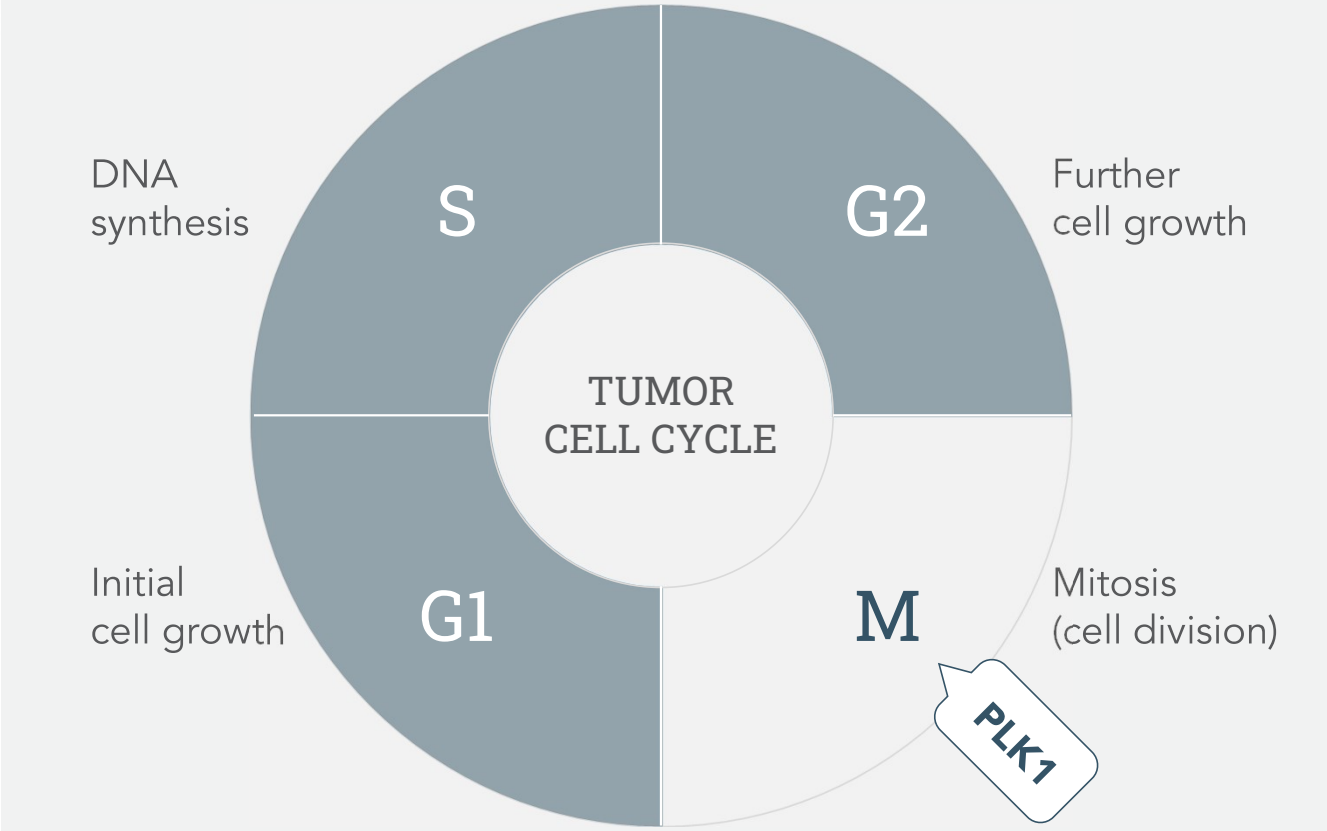
TURNING THE TIDE ON CANCER

MARCH 2022

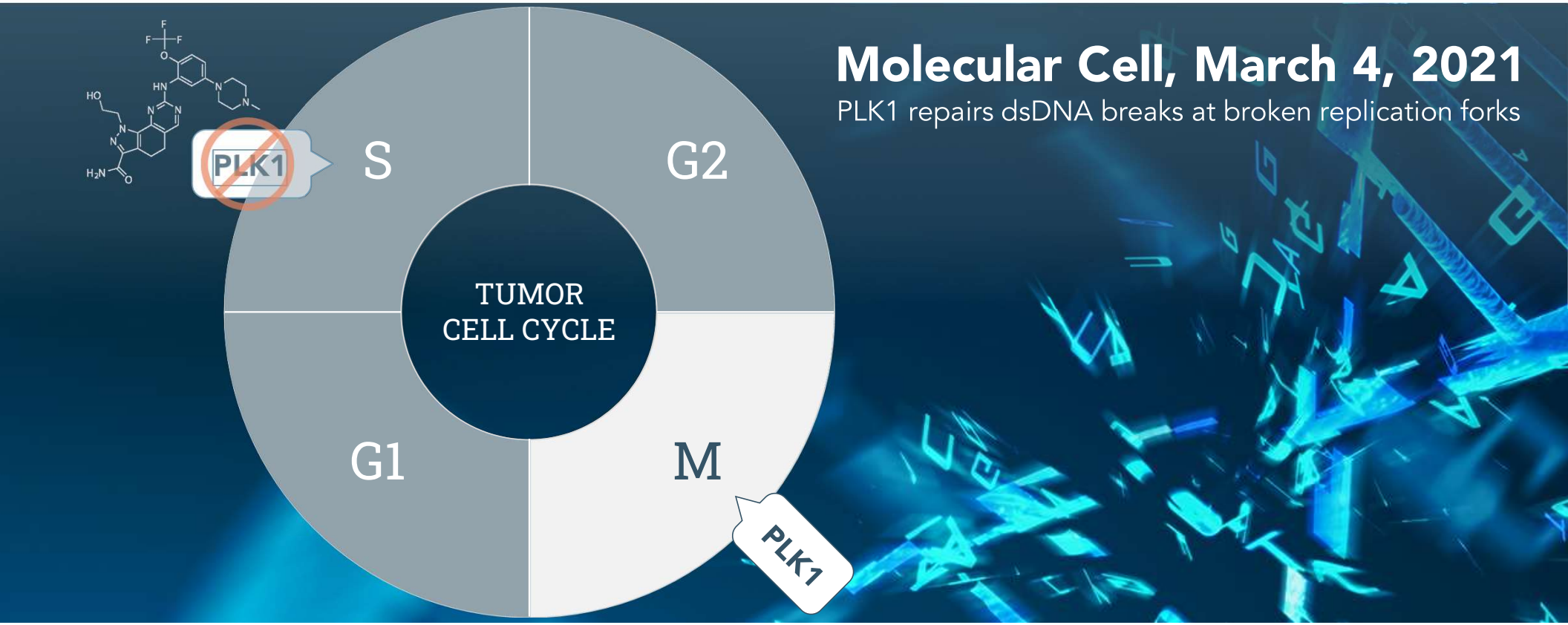
Forward-Looking Statements

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 our expectations, strategy, plans or intentions. These forward-looking statements are based on our 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. These factors include, but are not limited to, our need for additional financing; our ability to continue as a going concern; clinical trials involve a lengthy and expensive process with an uncertain outcome, and results of earlier studies and trials may not be predictive of future trial results; our clinical trials may be suspended or discontinued due to unexpected side effects or other safety risks that could preclude approval of our product candidates; risks related to business interruptions, including the outbreak of COVID-19 coronavirus, which could seriously harm our financial condition and increase our costs and expenses; uncertainties of government or third party payer reimbursement; dependence on key personnel; limited experience in marketing and sales; substantial competition; uncertainties of patent protection and litigation; dependence upon third parties; regulatory, and risks related to failure to obtain FDA clearances or approvals and noncompliance with FDA regulations. There are no guarantees that any of our technology or products will be utilized or prove to be commercially successful. Additionally, there are no guarantees that future clinical trials will be completed or successful or that any precision medicine therapeutics will receive regulatory approval for any indication or prove to be commercially successful. Investors should read the risk factors set forth in our Form 10-K for the year ended December 31, 2021, and other periodic reports filed with the Securities and Exchange Commission. While the list of factors presented here 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 we do not undertake any obligation to update publicly such statements to reflect subsequent events or circumstances.

PLK1 is hijacked by tumor cells, allowing uncontrolled growth



PLK1 repairs damaged DNA, enabling tumor cells to proliferate



Onvansertib positions Cardiff Oncology to effectively target PLK1

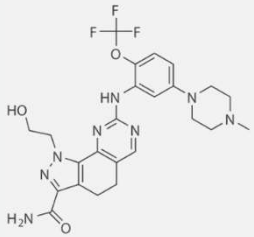
SPECIFICITY

Exquisitely specific for PLK1

ENZYME	IC_{50} (μM)
PLK1	**0.002**
PLK2	>10
PLK3	>10
CK2	0.4
FLT3	0.4
CDK1/CycB	>10
42 other kinases and >140 in the Millipore panel	>10

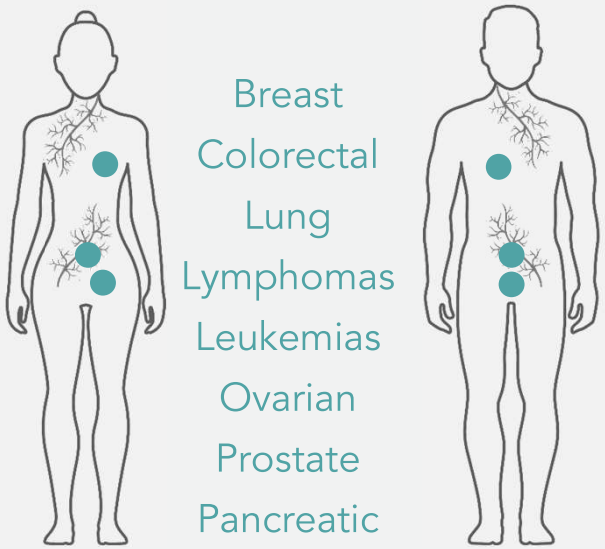
PROPERTIES

- Small molecule
- Oral dosing
- 24-hour half-life



OPPORTUNITY

PLK1 is over-expressed in many cancer types[1]



1. Renner Blood 2009; Mito Leukemia and Lymphoma 2005; 2005; Takai et al., Oncogene (2005) 24, 287–291

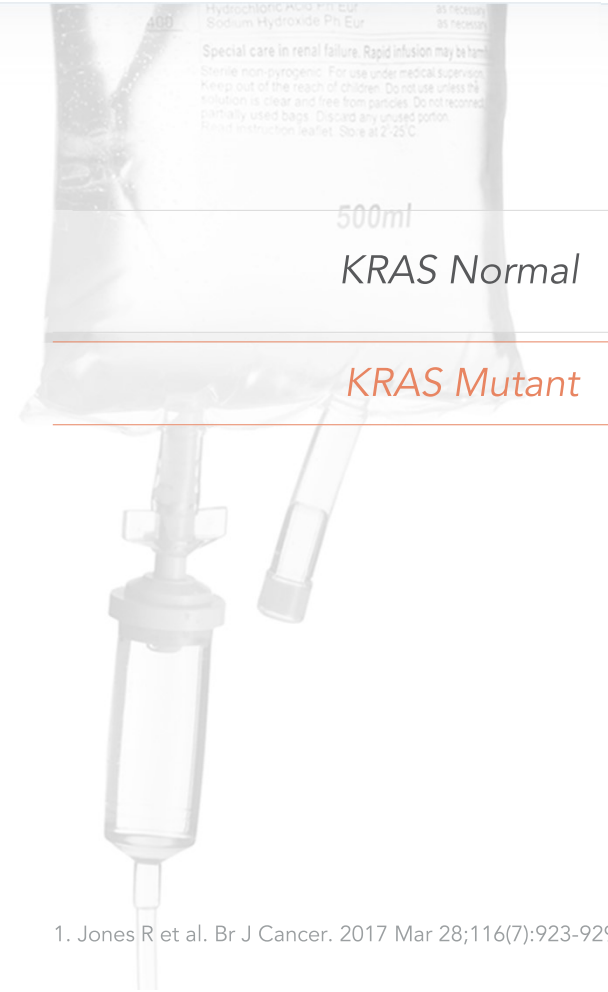
WHAT	Onvansertib has achieved
WHY	Onvansertib works
WHERE	Cardiff Oncology can go

WHAT	Onvansertib has achieved
WHY	Onvansertib works
WHERE	Cardiff Oncology can go

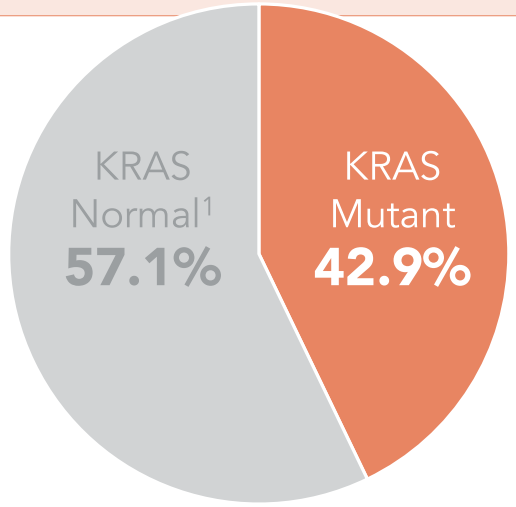
Our lead program is in KRAS-metastatic colorectal cancer (mCRC)

		Preclinical	IND En.	Ph 0/1	Ph 2	Status
mCRC	**FOLFIRI/bev**					**Enrolling**
mPDAC	Onivyde/5-FU					Enrolling
mCRPC	Abiraterone					Enrolling
PDAC	Biomarker					Target Q1 ’22
TNBC	Combo w/ Paclitaxel					Development
SCLC	Single agent					Development
CMML	Single agent					Development
Medullo-blastoma	Combo w/ radiation					Development
Ovarian	PARP inhibitors					Preclinical

Gaps in current mCRC therapies leave a significant unmet need



	1st LINE	2nd LINE
KRAS Normal	FOLFOX (chemo) + bevacizumab + EGFR inhibitor	FOLFIRI (chemo) + bevacizumab
KRAS Mutant	FOLFOX (chemo) + bevacizumab	



1. Jones R et al. Br J Cancer. 2017 Mar 28;116(7):923-929

The prognosis for second-line mCRC patients is poor

2nd LINE

FOLFIRI (chemo) + bevacizumab

5-year survival: 10%

Drugs in development do not address most prevalent KRAS mutations

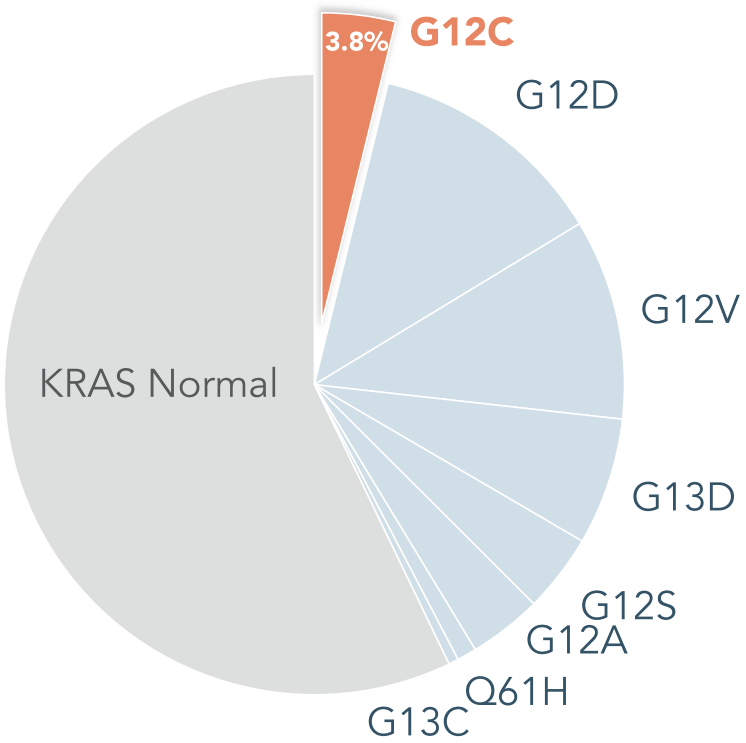
HISTORICAL ORR

5%	2006 – 2008	ML18147 Phase 3 Registrational Trial FOLFIRI + bev in second-line[1]
11.4%	2000 – 2013	Systematic Literature-Based Analysis of 23 Randomized Trials (10,800 Patients) in Second-Line mCRC[2]
13%	2015 – 2017	TRIBE2 Randomized Phase 3 Trial: SOC arm FOLFIRI + bev in Second-line following FOLFOX + bev First-line[3,4]

1. Bennouna et al., Lancet Oncol 2013; 14: 29–37; 2. Giessen et al., Acta Oncologica, 2015, 54: 187-193; 3. Cremolini et al., Lancet Oncol 2020, 21: 497–507; 4, Antoniotti et al., Correspondence Lancet Oncol June 2020. mCRC: metastatic colorectal cancer

Gaps in KRAS-mutated mCRC therapies leave a significant unmet need

KRAS Mutations in mCRC[1]

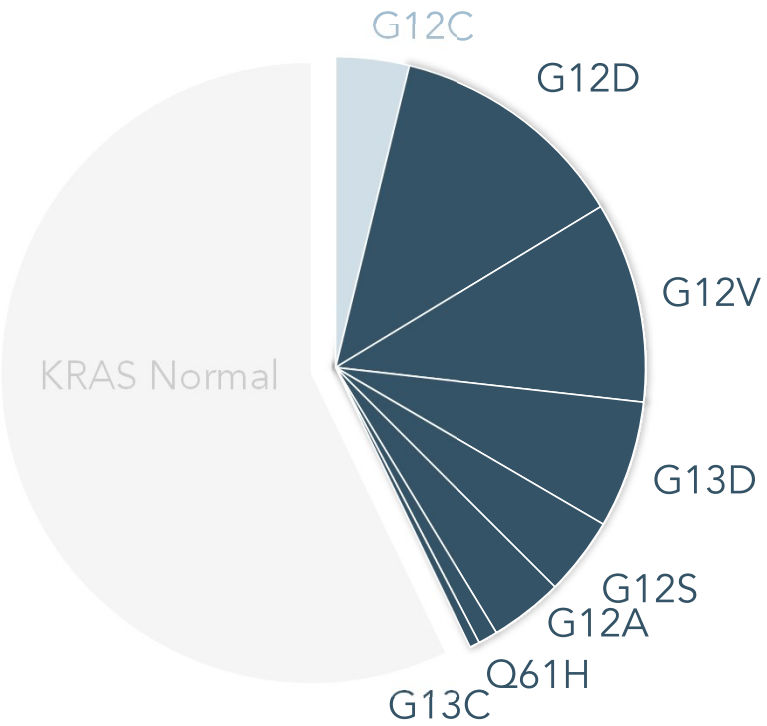


Investigational therapies (Amgen; Mirati) address the G12C KRAS mutation ***only***

1. Jones R et al. Br J Cancer. 2017 Mar 28;116(7):923-929

Gaps in KRAS-mutated mCRC therapies leave a significant unmet need

KRAS Mutations in mCRC[1]



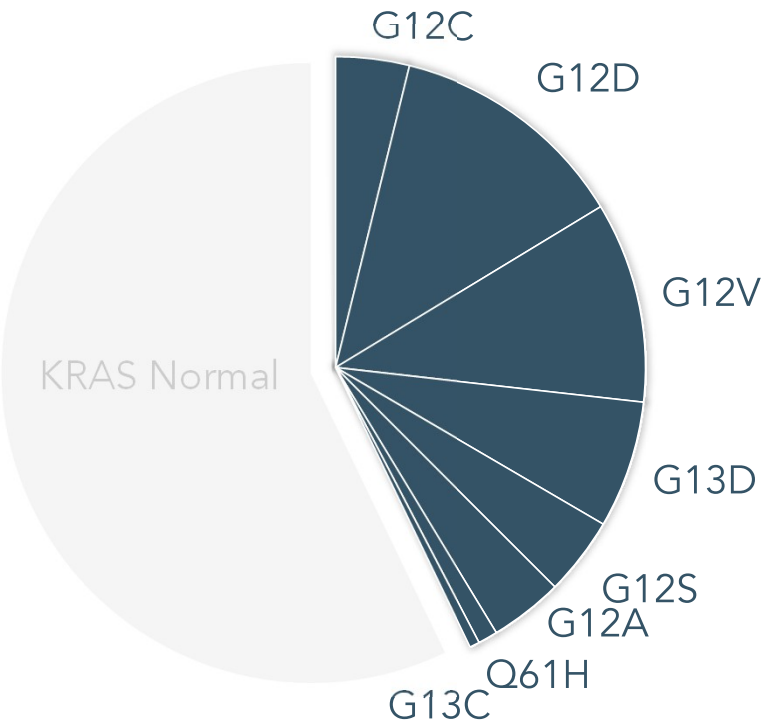
91.1%

of patients with KRAS mutations miss out on targeted therapy

1. Jones R et al. Br J Cancer. 2017 Mar 28;116(7):923-929

Gaps in KRAS-mutated mCRC therapies leave a significant unmet need

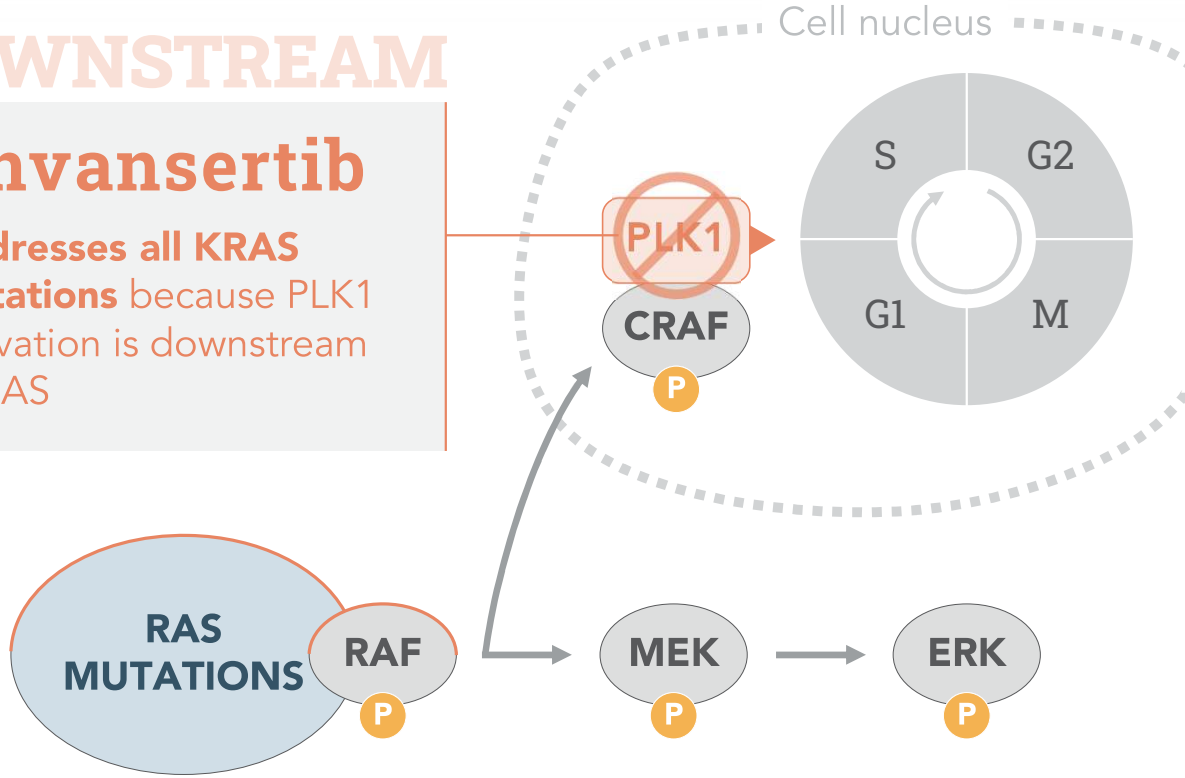
KRAS Mutations in mCRC[1]



DOWNSTREAM

Onvansertib

Addresses all KRAS mutations because PLK1 activation is downstream of RAS

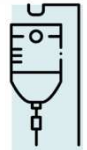


1. Jones R et al. Br J Cancer. 2017 Mar 28;116(7):923-929

Our Ph 1b/2 trial in KRAS-mutated mCRC combines onvansertib w/ SoC

One Cycle = 28 Days

WEEKS 1–2

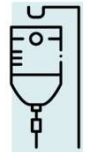


2nd-Line SoC: FOLFIRI
+ bevacizumab

6	7	8	9	10	11	12	13	14

ONVANSERTIB

WEEKS 3–4



2nd-Line SoC: FOLFIRI
+ bevacizumab

20	21	22	23	24	25	26	27	28

ONVANSERTIB

Trial endpoints measure tumor response and decrease in KRAS burden

EFFICACY ENDPOINTS

1. Primary: Objective Response Rate (ORR) per RECIST v1.1 in patients who receive ≥1 cycle of treatment
2. Secondary: Progression-Free Survival (PFS) and Duration of Response (DoR)
3. Exploratory: Decrease in KRAS mutational burden and response to treatment

Endpoints measure tumor response and decrease in KRAS burden

EVALUABLE PATIENTS AT DEC 3, 2021

	PHASE 1b	PHASE 2		
12 mg/m²	5			
15 mg/m²	6	32	RP2D[1] N=35	ALL DOSES N=48
18 mg/m²	5			

1. Three patients were excluded from the RP2D (recommended phase 2 dose) efficacy evaluation because they received onvansertib 12 mg/m² instead of the assigned per protocol dose of 15 mg/m²

Proof of concept criteria set to exceed historical ORR and mPFS

HISTORICAL ORR*

5%	2006 – 2008
11.4%	2000 – 2013
13%	2015 – 2017

HISTORICAL mPFS*

4.5–5.7 mo

PROOF OF CONCEPT CRITERIA

20% ORR

≥6 mo mPFS

* Bennouna et al., Lancet Oncol 2013; 14: 29–37; Giessen et al., Acta Oncologica, 2015, 54: 187-193; Cremol
Correspondence Lancet Oncol June 2020; ORR: Objective Response Rate; PFS: Progression-Free Survival

Results from the Ph 1b/2 trial continue to show improvement over SoC

HISTORICAL ORR

5%	2006 – 2008
11.4%	2000 – 2013
13%	2015 – 2017

HISTORICAL PFS

4.5–5.7 mo

RESULTS AT DEC 3, 2021 WITH FOLLOW UP*

20% **35%** (ALL) **34%** (RP2D) ORR

≥6 mo **9.4 mo** (ALL) mPFS

* Reflects data cutoff date of Dec 3, 2021 and includes one subsequent PR achieved by Jan 18, 2022 data release. Patient 01-046 achieved an initial PR at the 8-month scan on Dec 27, 2021

Our clinical data indicates that onvansertib + SoC is well tolerated

No major/unexpected toxicities

- Of all TEAEs, only 11% (84/788) were G3/G4
- 7 patients had a total of 11 G4 adverse events:
  - Neutropenia (n=7); Decreased WBC (n=2); Neutropenic fever (n=1); Hyperphosphatemia (n=1)
- Discontinuation of the 5-FU bolus + use of growth factors ameliorated the severity of neutropenia observed

N=50 TEAEs*	GRADE 1	2	3	4	All
Neutropenia	1	13	15	6	35
Fatigue	15	15	3	0	33
Nausea	24	7	2	0	33
Diarrhea	15	7	2	0	24
Abdominal Pain	13	7	1	0	21
Mucositis	11	6	2	0	19
Alopecia	17	2	0	0	19
WBC Decrease	6	9	2	1	18
Platelet Count Decrease	10	4	1	0	15
Hypertension	2	8	5	0	15

TEAEs*	GRADE 1	2	3	4	All
Anemia	9	4	1	0	14
Vomiting	9	3	1	0	13
Musculoskeletal Pain†	11	1	0	0	12
Infection†	3	4	4	0	11
Hemorrhage†	8	0	1	0	9
Headache	7	0	0	0	7
Neuropathy	5	2	0	0	7
GERD	7	0	0	0	7
ALT Increase	4	0	1	0	5

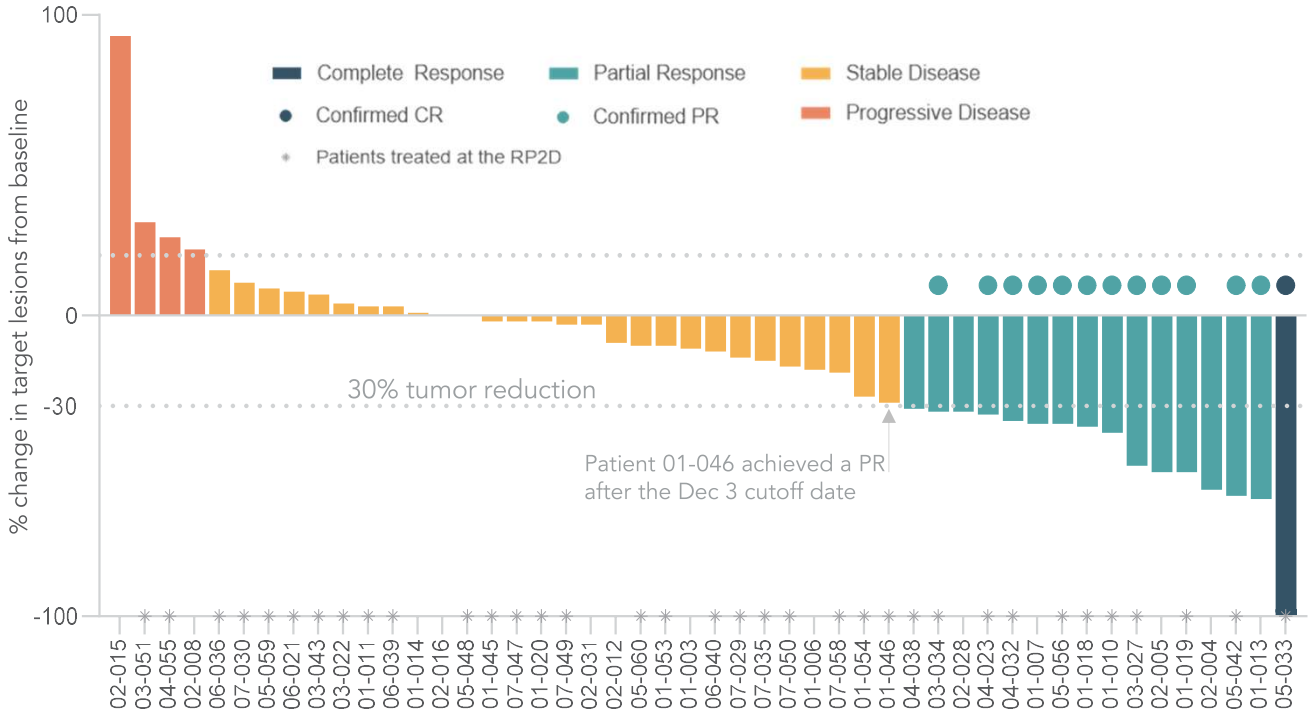
as of 03-Dec-2021

* Jan 2022 data are interim as of Dec 3, 2021 from an ongoing trial and unlocked database. N: number of patients (total N=50); events shown occurred in ≥10% of patients; numbers indicate number of patients experiencing the event, (regardless of causality); each patient is only counted once and only for the highest grade of a given event. TEAEs: Treatment Emergent Adverse Events

† Musculoskeletal pain, infection and hemorrhage are pooled terms

92% of patients achieved disease control (CR + PR + SD)

Best Radiographic Response* – all doses (as of Dec 3, 2021)



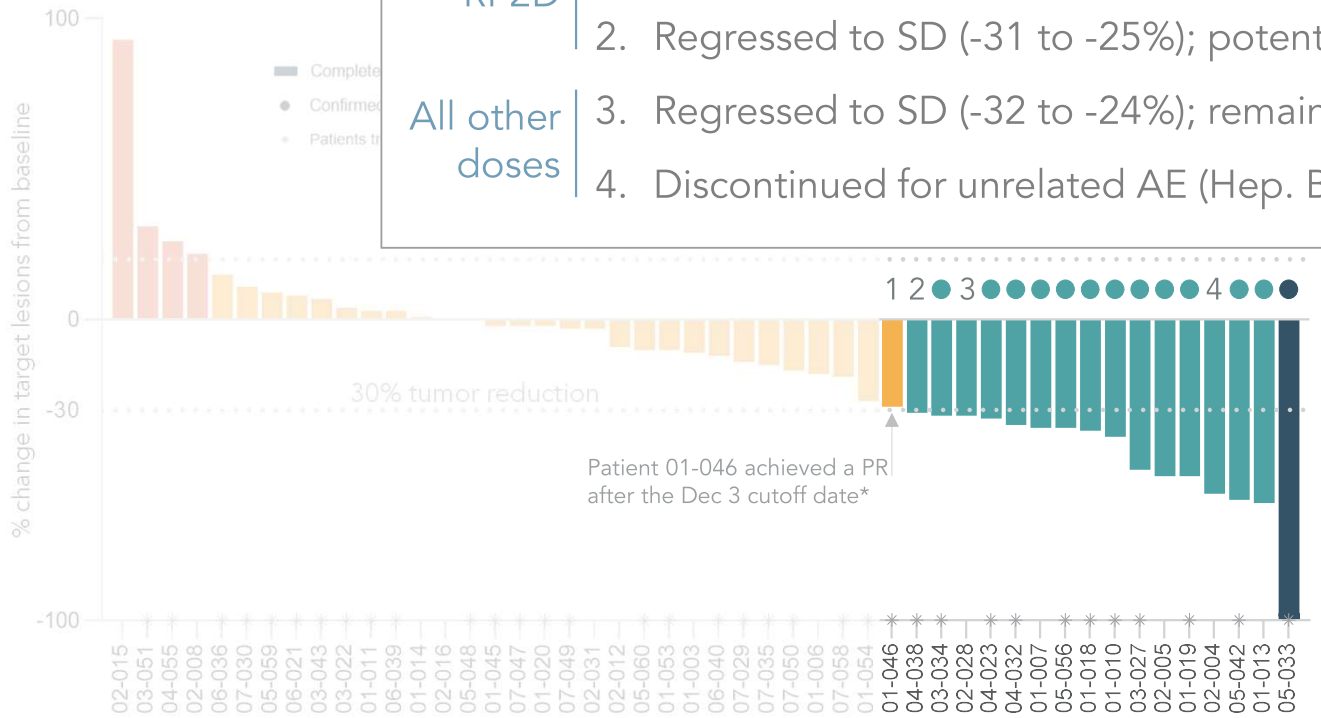
Objective Response* (CR + PR)		
– All doses	17 / 48	35%
– RP2D	12 / 35	34%
Tumor Reduction	33 / 48	69%
Disease Control Rate (CR + PR + SD)	44 / 48	92%

* Waterfall plot and table reflect interim data as of Dec 3, 2021 from an ongoing trial and unlocked database, and indicates one subsequent PR achieved on follow up through Jan 18, 2022 press release

92% of patients achieved disease control (CR + PR + SD)

PR confirmation status

RP2D	1. Waiting for confirmatory scan; remains on trial
	2. Regressed to SD (-31 to -25%); potentially curative surgery
All other doses	3. Regressed to SD (-32 to -24%); remains on trial
	4. Discontinued for unrelated AE (Hep. B)



– RP2D	17 12	48 35	35% 34%
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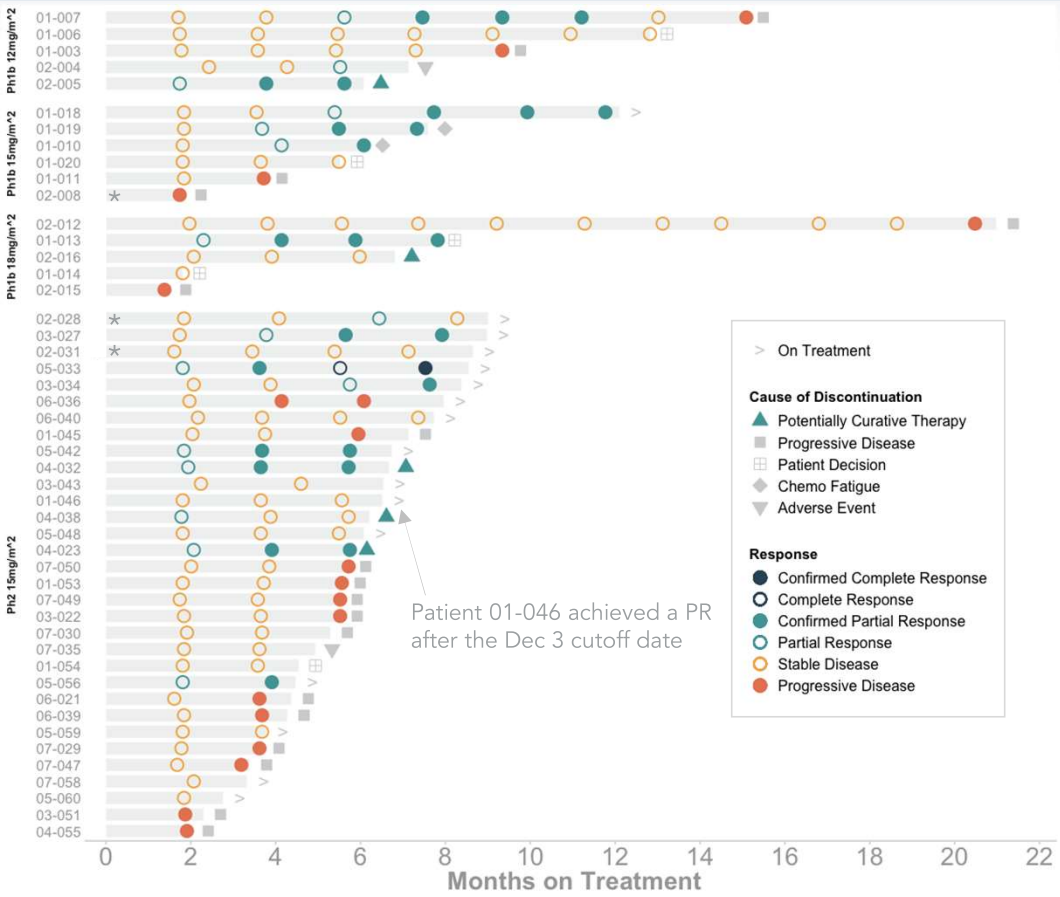
* Waterfall plot and table reflect interim data as of Dec 3, 2021 from an ongoing trial and unlocked database, and indicates one subsequent PR achieved on follow up through Jan 18, 2022 press release

Across all doses we observe initial PRs up to eight months on treatment



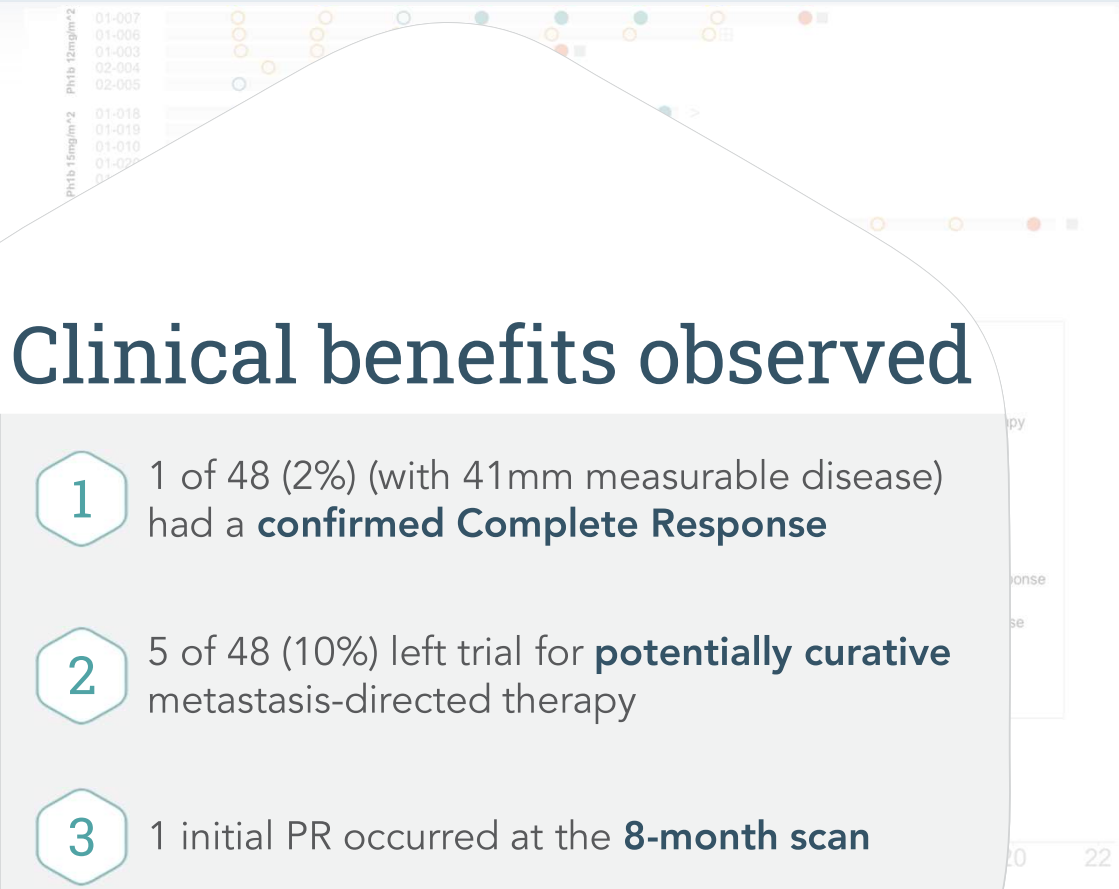
Swimmer plot† – all doses (as of Dec 3, 2021)

Evaluable Patients – all doses	48
Time of initial PR	
8-week scan	8
16-week scan	3
24-week scan	5
32-week scan†	1

⋆ Three patients were excluded from the RP2D efficacy evaluation because they received onvansertib 12 mg/m^2 instead of the assigned per protocol dose of 15 mg/m^2

† Swimmer plot and table reflect interim data as of Dec 3, 2021 from an ongoing trial and unlocked database, and indicates one subsequent PR achieved on follow up through Jan 18, 2022 press release

Clinical efficacy events of interest



Swimmer plot[†] – all doses (as of Dec 3, 2021)

Evaluable Patients – all doses	48
Time of initial PR	
8-week scan	8
16-week scan	3
24-week scan	5
32-week scan[†]	1

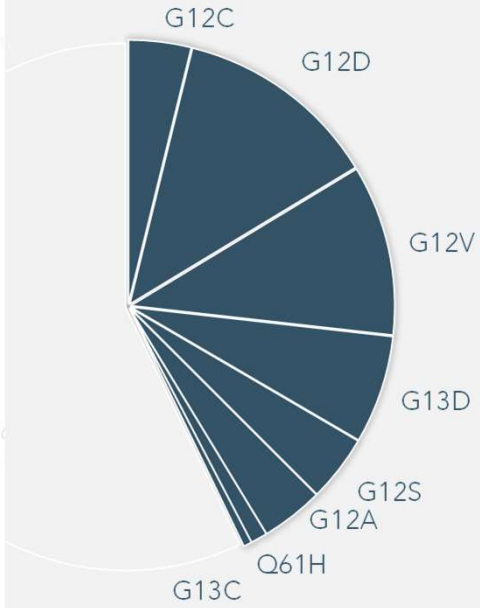
sertib 12 mg/m² instead of the assigned per protocol dose of 15 mg/m²
cked database, and indicates one subsequent PR achieved on follow up through

Clinical benefits observed

1. 1 of 48 (2%) (with 41mm measurable disease) had a **confirmed Complete Response**
2. 5 of 48 (10%) left trial for **potentially curative** metastasis-directed therapy
3. 1 initial PR occurred at the **8-month scan**

The all-dose cohort achieved responses across several KRAS mutations

Frequency of Common KRAS Mutations in mCRC[1]



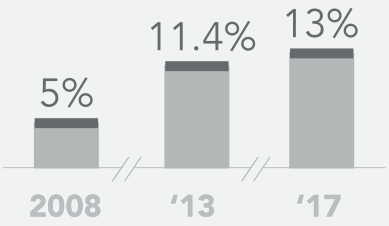
Onvansertib responses across KRAS mutations (as of Dec 3, 2021)

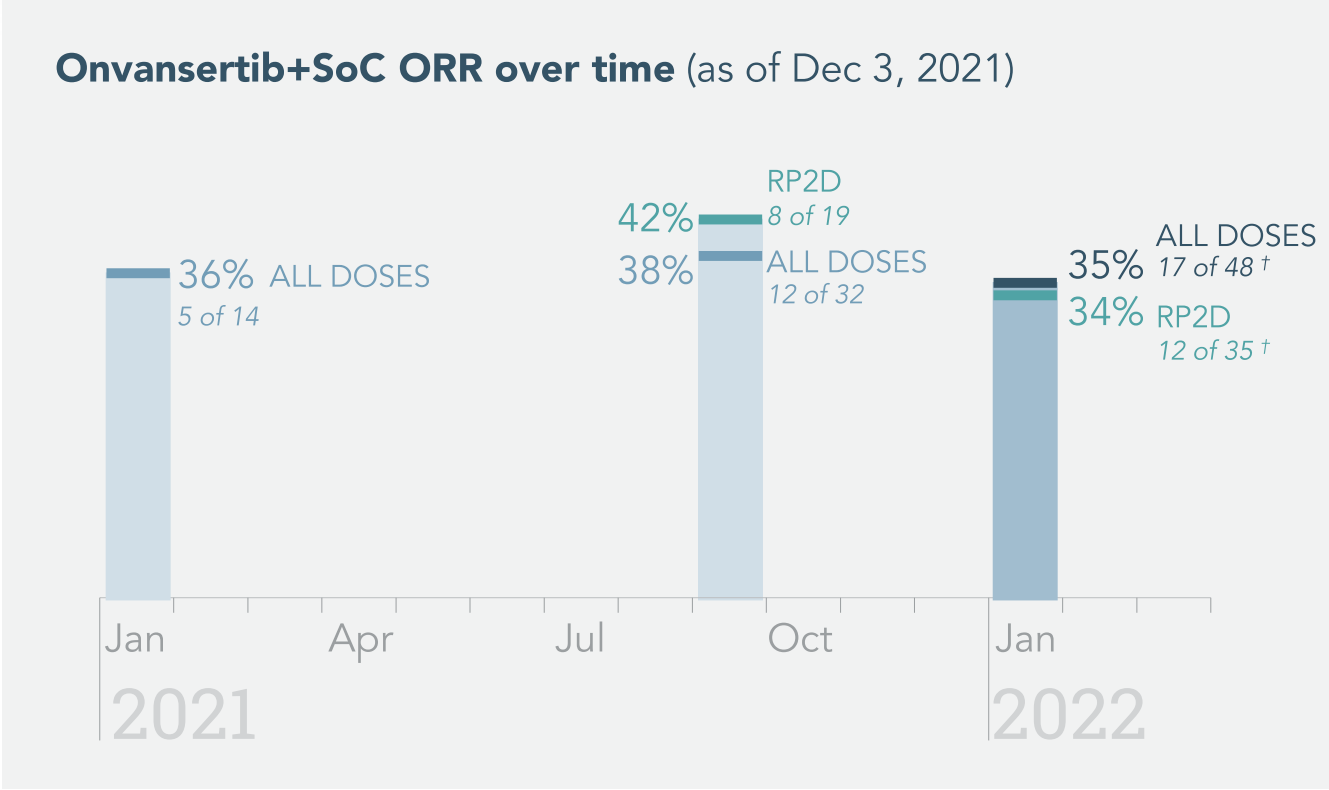
KRAS Variant	CR+PR	SD	PD	Total
G12D	6*	7*	1	14
G12V	1	8	1	10
G13D	4	2		6
G12A	3	3		6
A146T	1	3		4
G12S		3	1	4
G12C	1	1	1	3
Q61H	1			1
Total	17*	27*	4	48

1. Jones R et al. Br J Cancer. 2017 Mar 28;116(7):923-929

* Patient 01-046 (with a G12D mutation) achieved a PR after the Dec 3, 2021 data cutoff date and is included in the table above as a 6th PR in the G12D line and 17th PR in the total line

Objective response rate for mCRC trial exceeds SoC over time



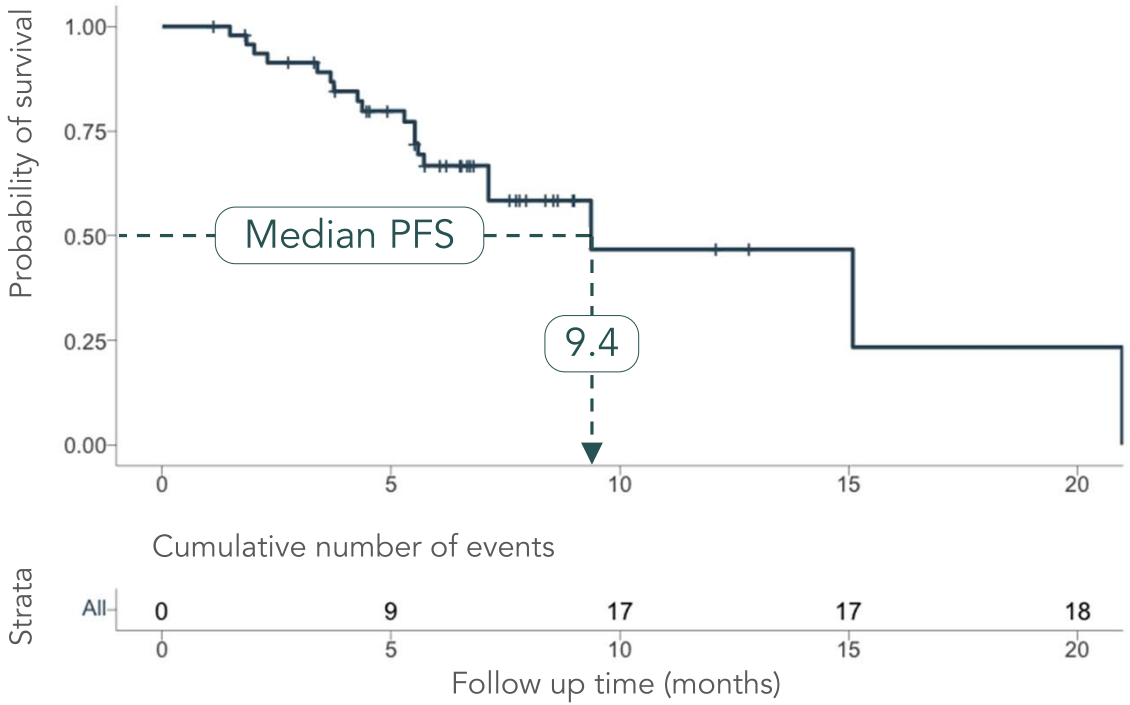


† Jan 2022 ORR are interim data as of Dec 3, 2021 from an ongoing trial and unlocked database, and includes one subsequent PR achieved on follow up through Jan 18, 2022 press release

* 2008: Bennouna et al., Lancet Oncol 2013; 14: 29–37; 2013: Giessen et al., Acta Oncologica, 2015, 54: 187-193; 2017: Cremolini et al., Lancet Oncol 2020, 21: 497–507; and Antoniotti et al., Correspondence Lancet Oncol June 2020. SoC: Standard-of-care

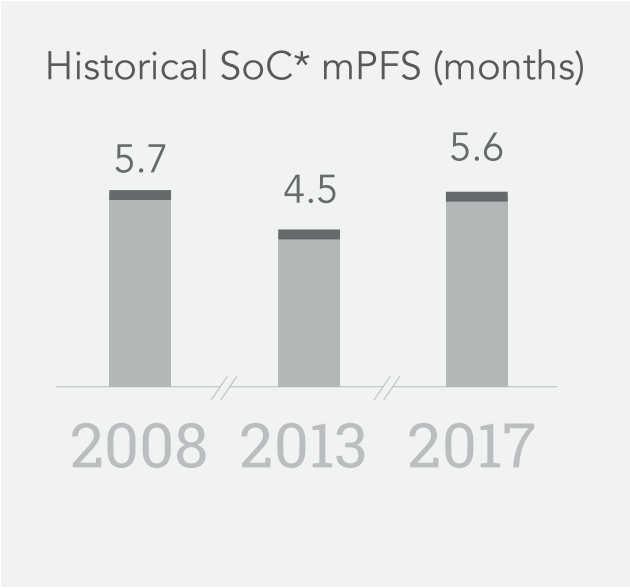
Median progression free survival for mCRC trial exceeds SoC over time

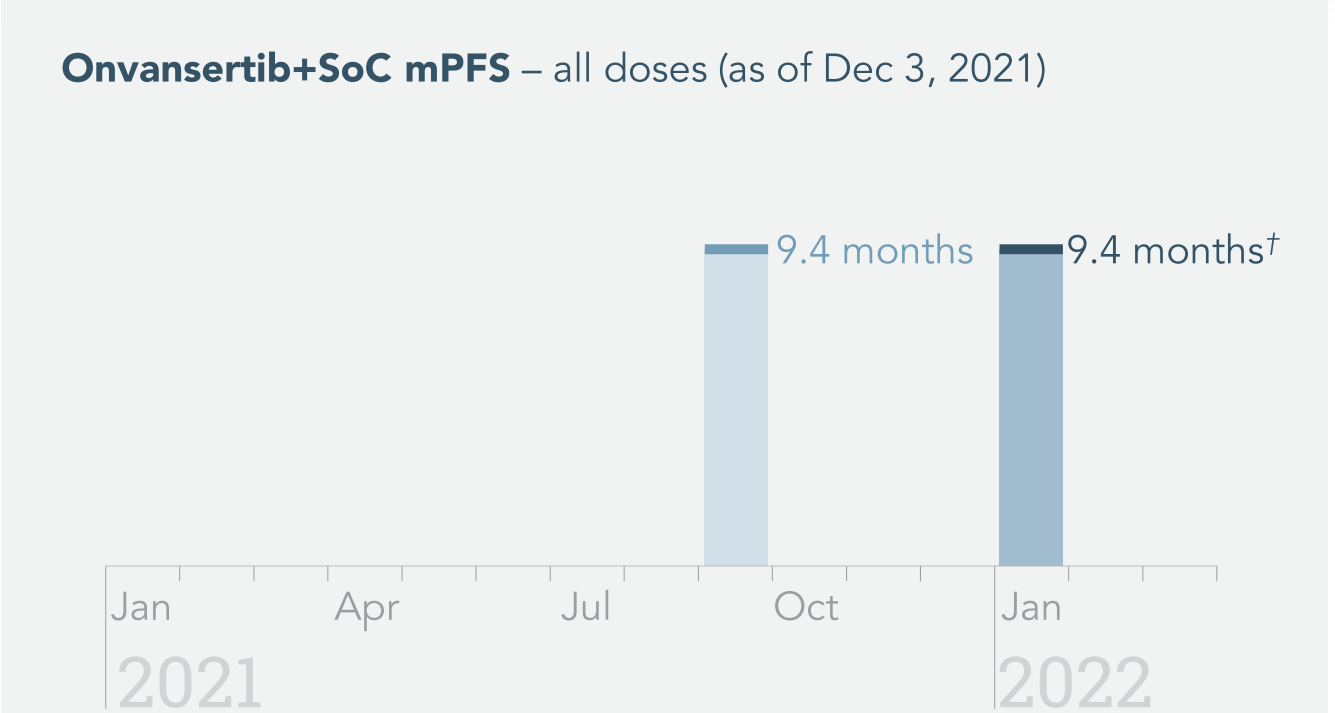
Progression free survival* – all doses (as of Dec 3, 2021)



* mPFS is interim data as of Dec 3, 2021 from an ongoing trial and unlocked database. mPFS for the RP2D is not yet reached as of Dec 3, 2021

Median progression free survival for mCRC trial exceeds SoC over time





† Jan 2022 PFS are interim data as of Dec 3, 2021 from an ongoing trial and unlocked database

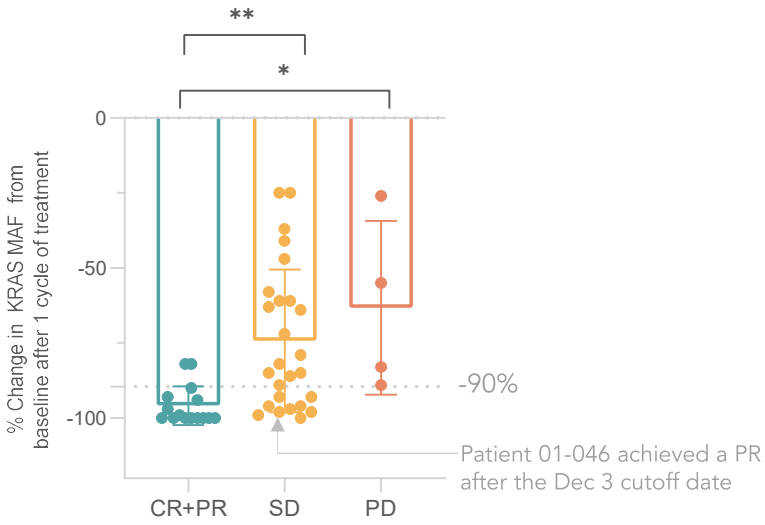
* 2008: Bennouna et al., Lancet Oncol 2013; 14: 29–37; 2013: Giessen et al., Acta Oncologica, 2015, 54: 187-193; 2017: Cremolini et al., Lancet Oncol 2020, 21: 497–507; and Antoniotti et al., Correspondence Lancet Oncol June 2020. SoC: Standard-of-care. mPFS: median progression free survival

Early KRAS MAF ctDNA decrease correlates with radiographic response

Predictive response biomarker

- 87% (13/15) of PR patients had ≥90% decrease in KRAS MAF after the 1st cycle
- 35% (9/26) of SD patients and none of the PD patients (n=4) had such a decrease

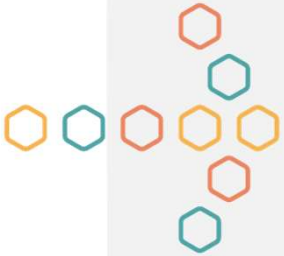
% KRAS Mutant Allelic Frequency (MAF)*
decrease after one 28-day treatment cycle
(as of Dec 3, 2021)



	CR+PR	SD	PD
Mean	-96	-74	-63

One way ANOVA, **p<0.01, *p<0.05

* KRAS MAF measured by droplet digital PCR (ddPCR) at baseline (day 1 of cycle 1, pre-dose) and on-treatment (day 1 of cycle 2). 1 PR and 2 SD patients had undetectable KRAS at baseline. KRAS MAF plot reflects interim data as of Dec 3, 2021 from an ongoing trial and unlocked database, and indicates one subsequent PR achieved on follow up through Jan 18, 2022 press release



WHAT	Onvansertib has achieved
WHY	Onvansertib works
WHERE	Cardiff Oncology can go

To date, toxicity has prevented regulatory approval of PLK1 inhibitors

Onvansertib's safety profile

eclipses that of its most promising predecessor

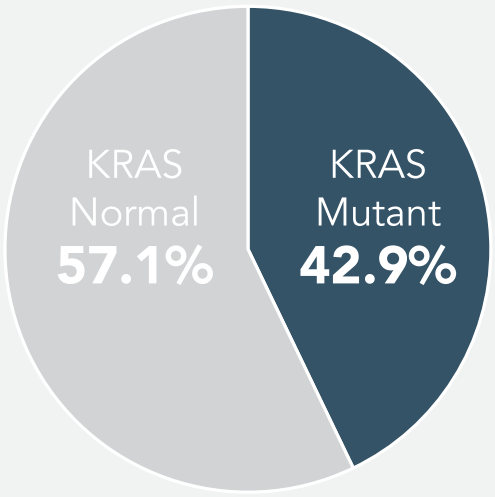
	Onvansertib	Volasertib[1]
Selectivity for PLK1	Exclusive for PLK1	Pan-inhibitor for PLK1, 2, and 3
Dosing	Oral	IV
Half-life	1 day	~5 days
Safety and tolerability	Well tolerated in ~200 patients	Pivotal trial suspended at 371 patients: toxicity

1. Boehringer Ingelheim was developing volasertib plus LDAC for the treatment of AML which did not meet the primary endpoint of ORR (EHA 2016). The data showed an unfavorable overall survival trend with the safety profile of volasertib plus LDAC considered as the main reason. Schoffski et al; European Journal of Cancer 48(2012); 179-186

Onvansertib's PLK1 inhibition is a two-pronged attack of tumor cells

KRAS HYPERSENSITIVITY[1]

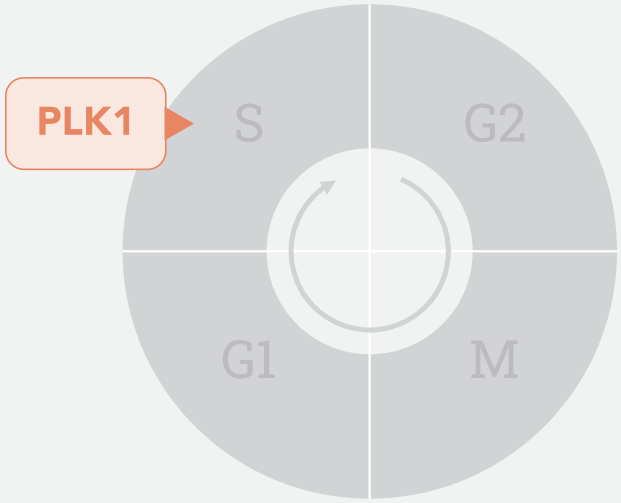
Cells with KRAS mutation are hypersensitive to inhibition of PLK1



MOA 1

SYNERGY WITH CHEMO

Inhibiting PLK1 increases the efficacy of chemotherapy drugs



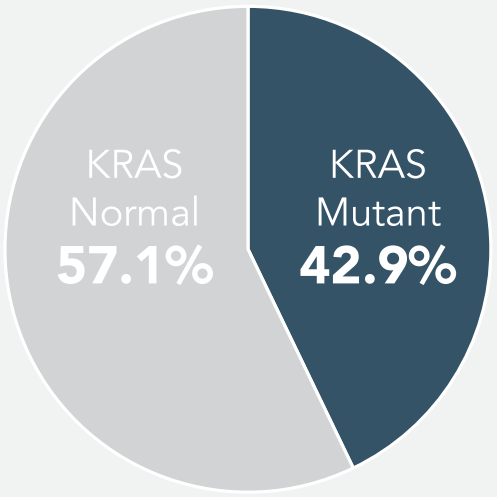
MOA 2

1. Jones R et al. Br J Cancer. 2017 Mar 28;116(7):923-929

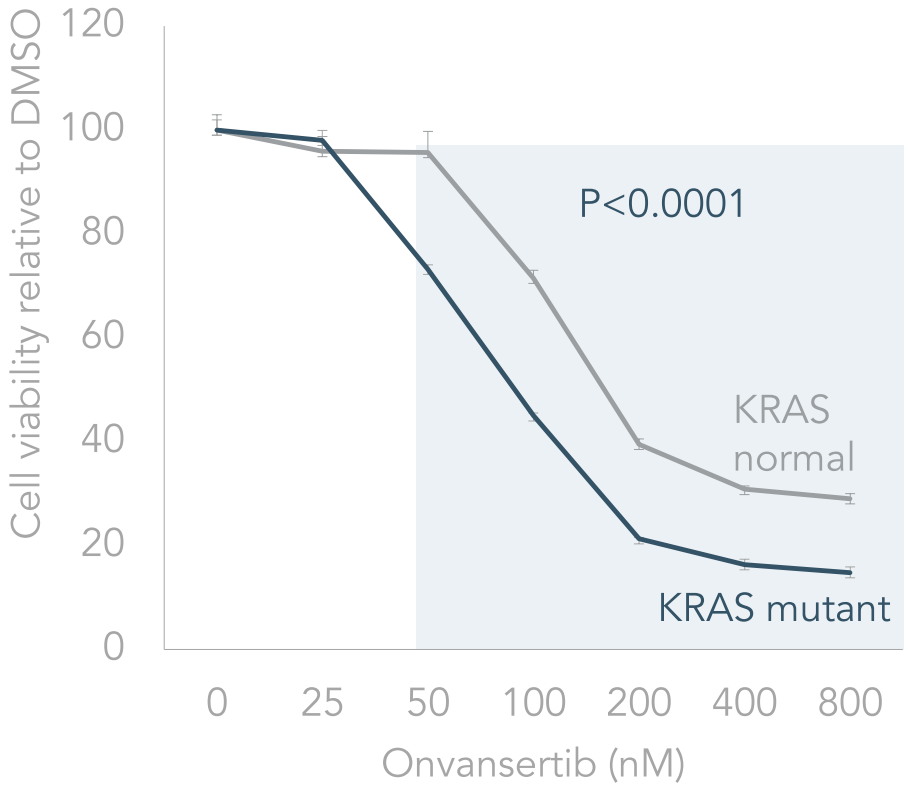
Onvansertib's PLK1 inhibition is a two-pronged attack of tumor cells

KRAS HYPERSENSITIVITY[1]

Cells with KRAS mutation are hypersensitive to inhibition of PLK1



MOA 1



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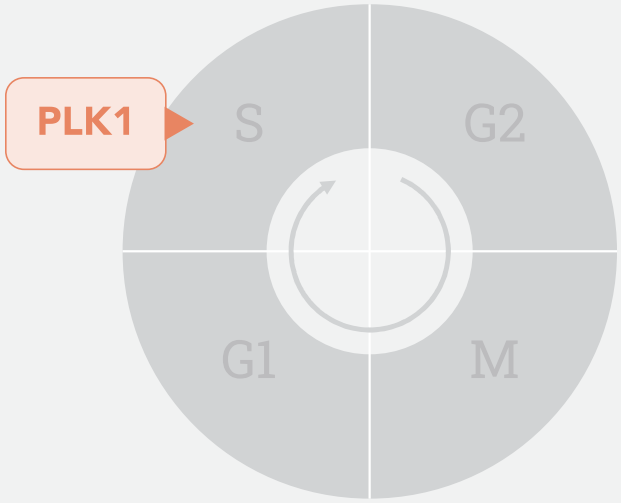
MOA 1

SYNERGY WITH CHEMO

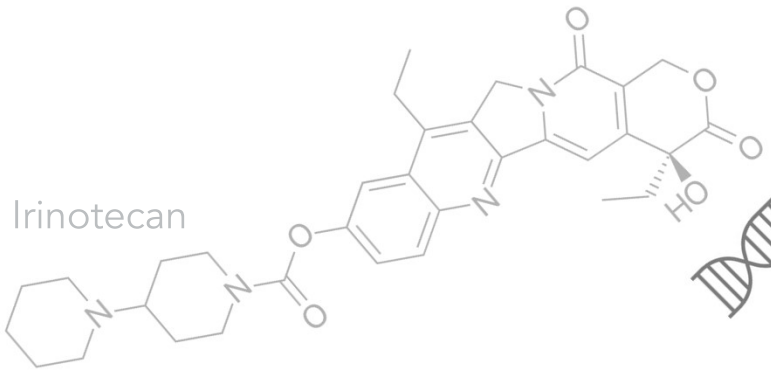
Inhibiting PLK1 increases the efficacy of chemotherapy drugs



MOA 2

Chemotherapy drugs damage tumor DNA to prevent cell proliferation

DNA Damaging Agent

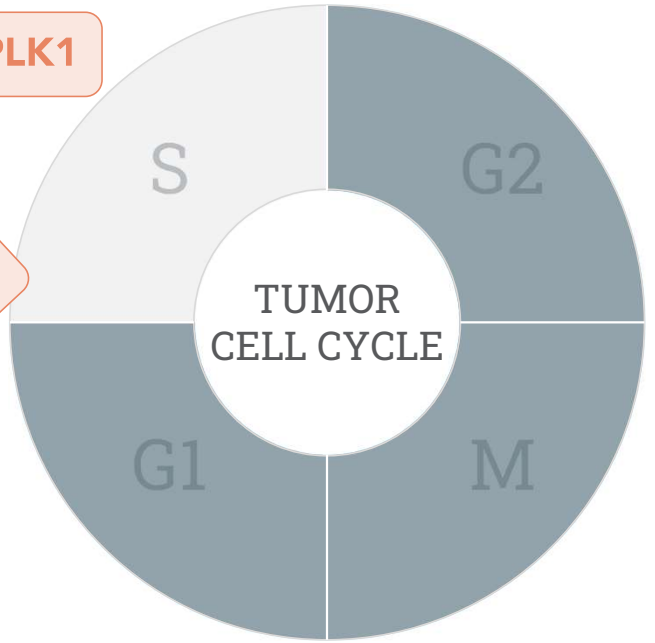


Irinotecan

DNA REPLICATION PHASE

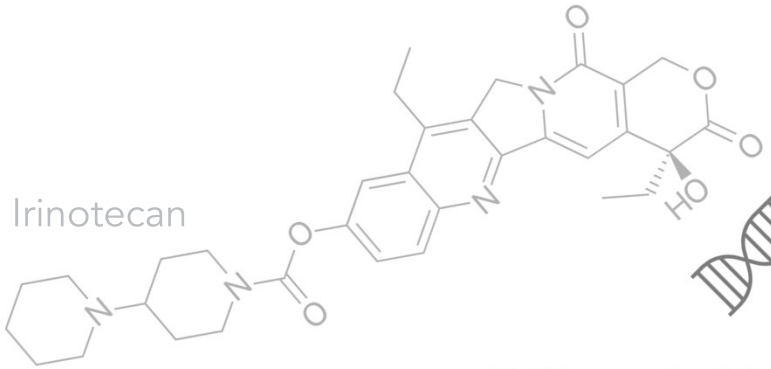
PLK1

PLK1

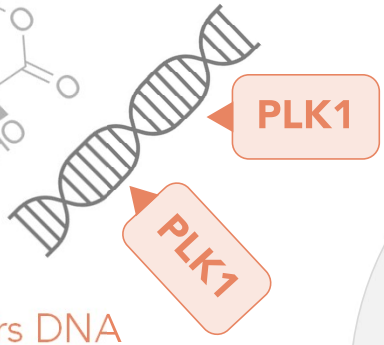


PLK1's repair of DNA interferes with chemotherapy drugs

DNA Damaging Agent



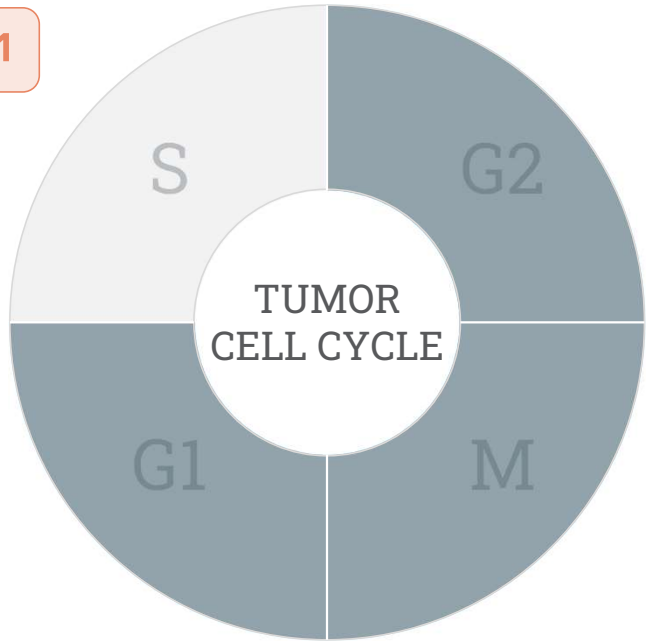
Irinotecan



PLK1 repairs DNA
enabling progression to G2

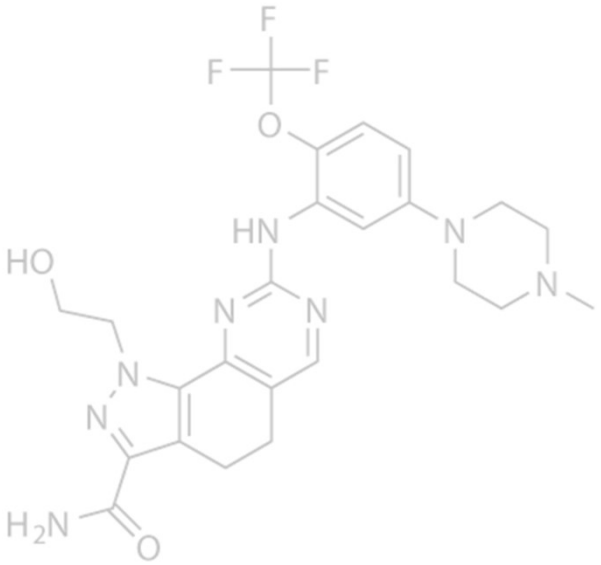
DNA REPLICATION PHASE

CELL GROWTH PHASE

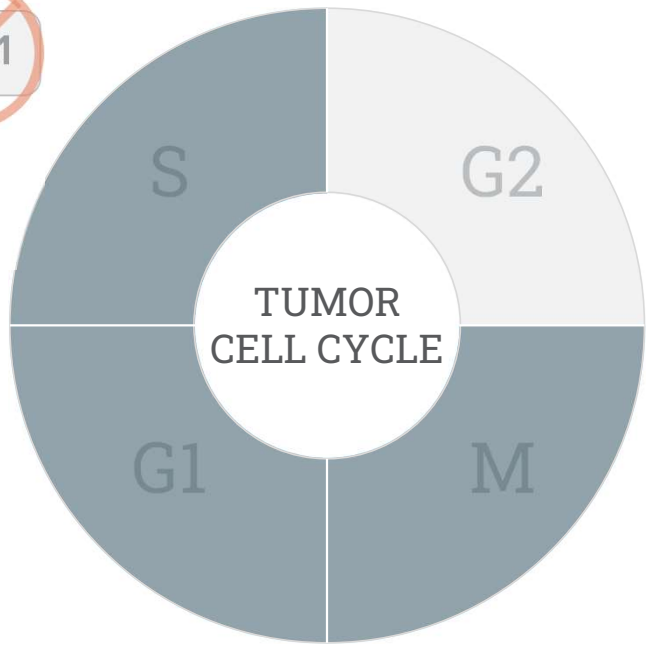


Inhibiting PLK1 prevents DNA repair and halts the cell cycle

Onvansertib inhibits PLK1 preventing DNA repair

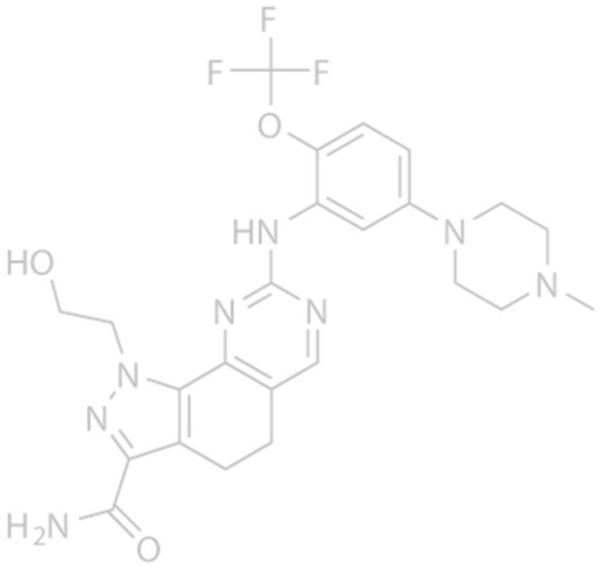


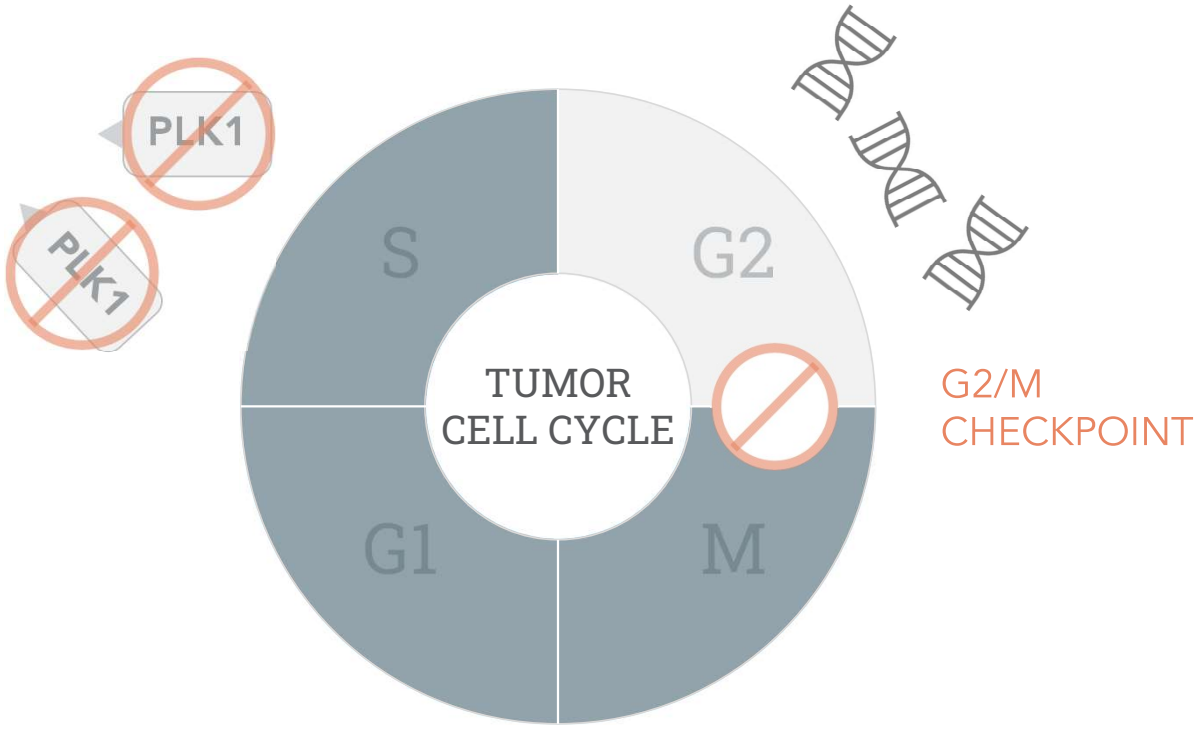
CELL GROWTH PHASE



Inhibiting PLK1 prevents DNA repair and halts the cell cycle

Onvansertib inhibits PLK1 preventing DNA repair and progression from G2 to M

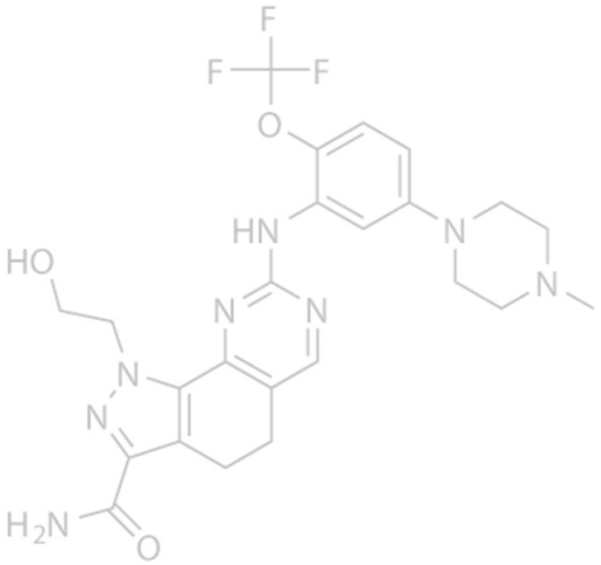


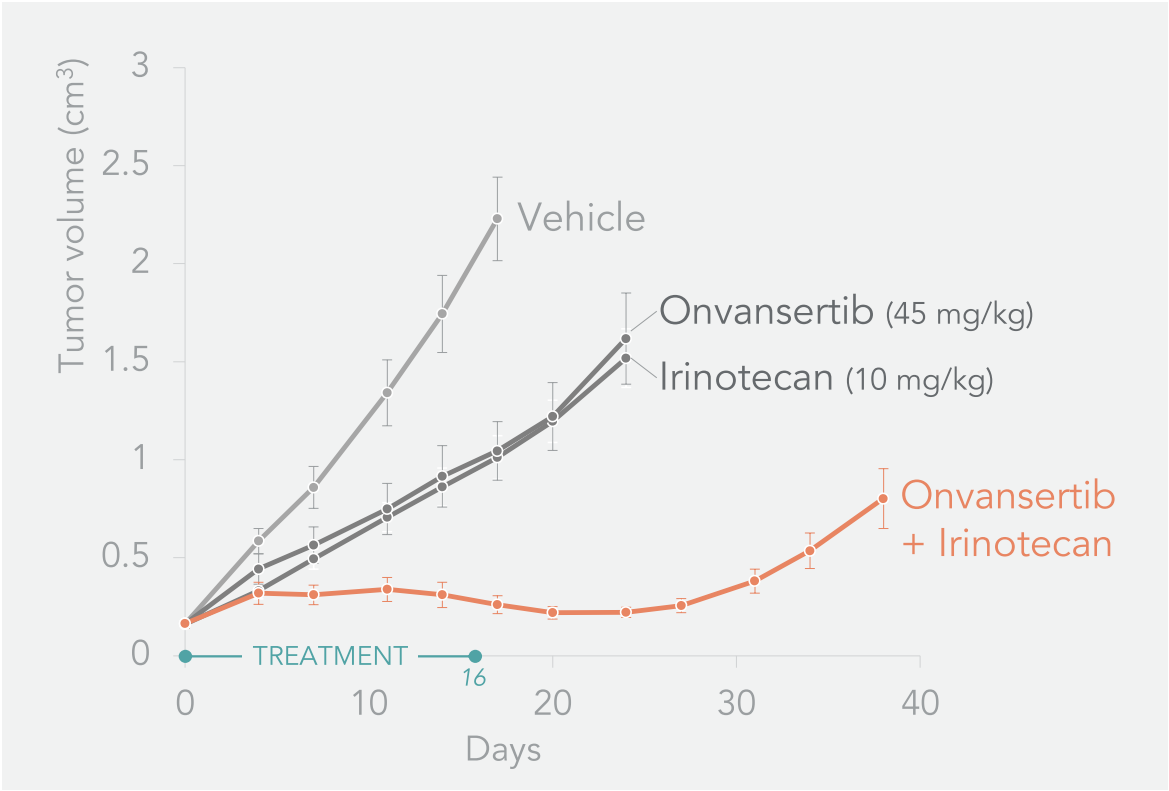


Combined, onvansertib and irinotecan are profoundly more effective

Onvansertib + Irinotecan[1]

in HCT-116 (with G13D KRAS mutation)





1. Mice were treated with vehicle, onvansertib (4 cycles of 1OFF/3ON), etirinotecan pegol NKTR-102 (days 1, 8, and 15) or the combination

WHAT	Onvansertib has achieved
WHY	Onvansertib works
WHERE	Cardiff Oncology can go

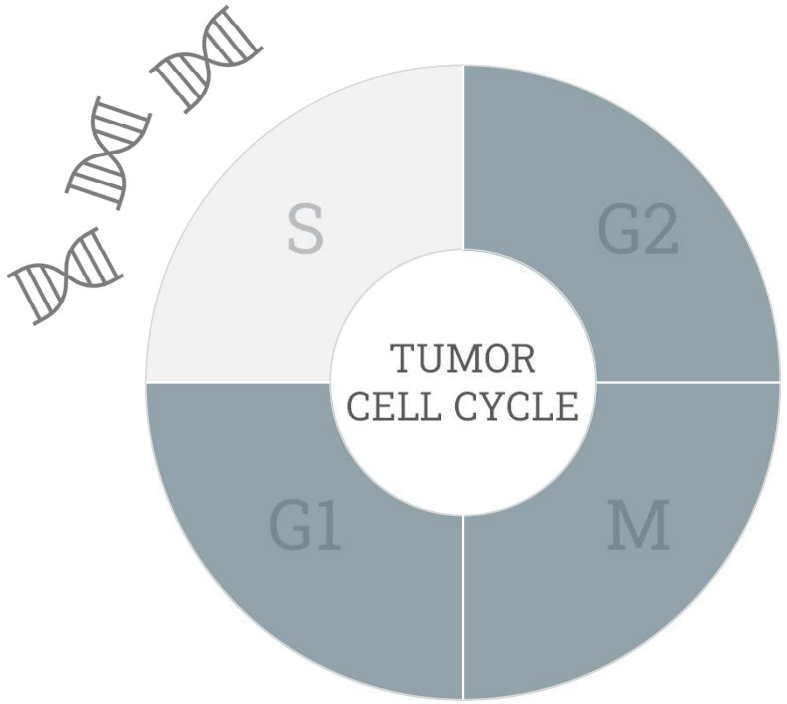
Onvansertib's MOA allows combinations with current therapies

DNA DAMAGING AGENTS

Inhibit the ability of PLK1 to repair DNA

	CHEMOTHERAPY	PARP INHIBITORS
mCRC	Phase 1b/2 Trial	
mPDAC	Phase 2 Trial	⬡⬡⬡
mCRPC	⬡⬡⬡	⬡⬡⬡
Ovarian		⬡⬡⬡
Breast		⬡⬡⬡
SCLC	⬡⬡⬡	⬡⬡⬡
Medulloblastoma		

⬡⬡⬡ = Cardiff Oncology potential



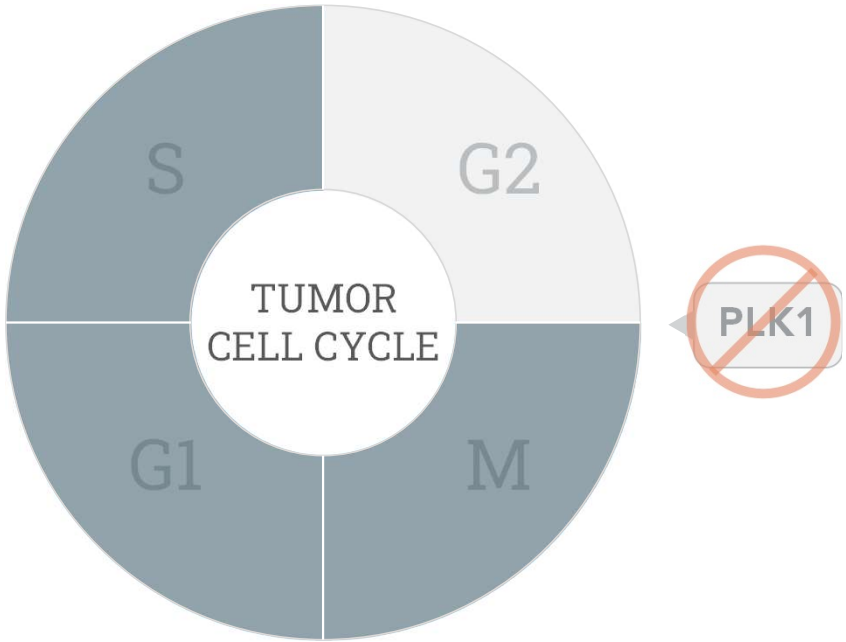
Onvansertib's MOA allows combinations with current therapies

DNA DAMAGING AGENTS

Inhibit the ability of PLK1 to promote progression to M phase

	RADIATION
mCRC	
mPDAC	
mCRPC	
Ovarian	
Breast	
SCLC	
Medulloblastoma	⬡⬡⬡

⬡⬡⬡ = Cardiff Oncology potential



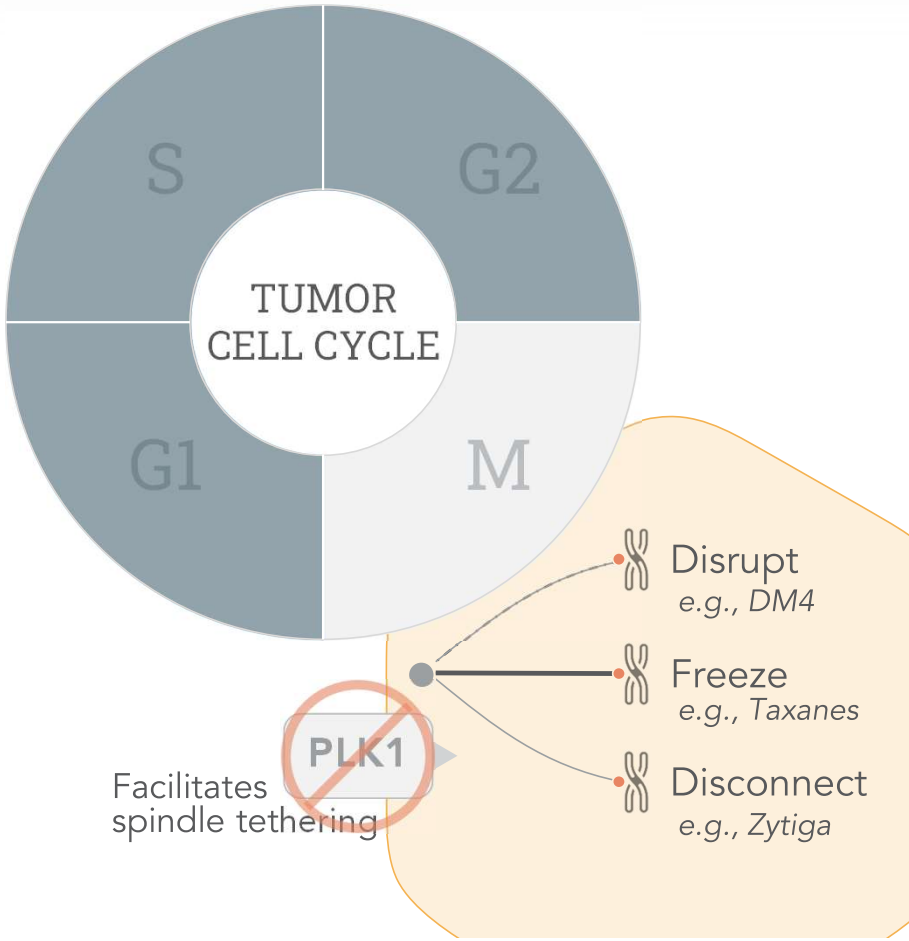
Onvansertib's MOA allows combinations with current therapies

MICROTUBULE TARGETING AGENTS

Inhibit the ability of PLK1 to promote cell division

	DISRUPT	FREEZE	DISCONNECT
mCRC			
mPDAC		⬡⬡⬡	
mCRPC			Phase 2 Trial
Ovarian	⬡⬡⬡	⬡⬡⬡	
Breast		⬡⬡⬡	
SCLC		⬡⬡⬡	
Medulloblastoma			

⬡⬡⬡ = Cardiff Oncology potential



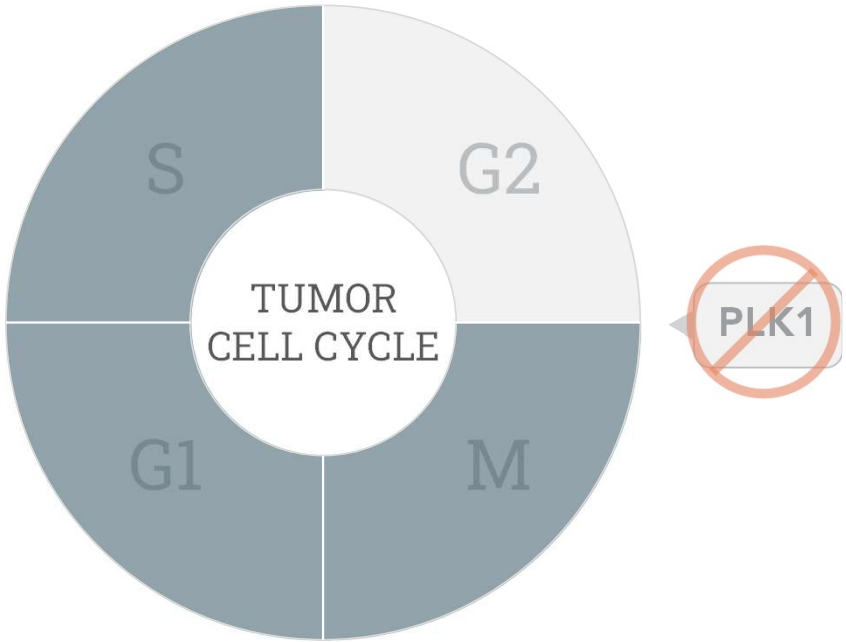
Onvansertib's MOA allows combinations with current therapies

EPIGENETICS

Inhibit PLK1's promotion to M and increase tumor cell's vulnerability to G2/M arrest

	LSD1 INHIBITORS
mCRC	
mPDAC	
mCRPC	⬡⬡⬡
Ovarian	
Breast	
SCLC	⬡⬡⬡
Medulloblastoma	

⬡⬡⬡ = Cardiff Oncology potential



Collectively, Cardiff Oncology has many attractive options

	DNA DAMAGING AGENTS			MICROTUBULE TARGETING			EPIGENETICS
	CHEMOTHERAPY	PARP INHIBITORS	RADIATION	DISRUPT	FREEZE	DISCONNECT	LSD1 INHIBITORS
mCRC	Phase 1b/2 Trial						
mPDAC	Phase 2 Trial	⬡⬡⬡			⬡⬡⬡		
mCRPC	⬡⬡⬡	⬡⬡⬡				Phase 2 Trial	⬡⬡⬡
Ovarian		⬡⬡⬡		⬡⬡⬡	⬡⬡⬡		
Breast		⬡⬡⬡			⬡⬡⬡		
SCLC	⬡⬡⬡	⬡⬡⬡			⬡⬡⬡		⬡⬡⬡
Medullo-blastoma			⬡⬡⬡				

⬡⬡⬡ = Cardiff Oncology potential

Our pipeline opens many attractive opportunities for onvansertib

		Preclinical	IND En.	Ph 0/1	Ph 2	Status	Partners
mCRC	FOLFIRI/bev					Enrolling	
mPDAC	Onivyde/5-FU					Enrolling	
mCRPC	Abiraterone					Enrolling	
PDAC	Biomarker					Target Q1 '22	Oregon Health & Science University
TNBC	Combo w/ Paclitaxel					Development	Dana-Farber Cancer Institute
SCLC	Single agent					Development	UPMC
CMML	Single agent					Development	Mayo Clinic
Medullo-blastoma	Combo w/ radiation					Development	University of Denver
Ovarian	PARP inhibitors					Preclinical	Dana-Farber Cancer Institute

Anticipated catalysts over the next twelve months

2022

CLINICAL PROGRAMS

Mid 2022

mCRC Phase 1b/2 data release

Launch pivotal trial

mPDAC Phase 2 data release

mCRPC Phase 2 data release

OTHER COMBINATIONS

- **Ovarian** cancer with PARPi
- **Breast** cancer with paclitaxel
- **Medulloblastoma** with radiation (pediatric)

We believe Pfizer relationship validates the opportunity for onvansertib

Pfizer BREAKTHROUGH GROWTH INITIATIVE

- Onvansertib program validation
- Scientific Advisory Board expertise: Adam Schayowitz, PhD
- Financial investment

SUMMARY TERMS

Announced November 18, 2021

- Pfizer invested a total of $15M at $6.22 per share (a 19% premium over prior closing price) with a 180-day lockup
- Right of First Access:
  Pfizer sees onvansertib data 2 days before release

Cardiff Oncology at a glance

Clinical-stage biotech company developing onvansertib, an oral, highly-selective PLK1 inhibitor, across a range of cancers

Exchange	Nasdaq: CRDF
Cash, Cash Equivalents and Investments[1]	$140.8M
Cash used in Operating Activities[1] (FY 2021)	$23.0M
Headquarters	San Diego, CA

1. As of 12/31/21. The above financial information is derived from our audited financials in Form 10K filed on 2/24/22.

KRAS-Mutated Metastatic Colorectal Cancer (mCRC)

Phase 1b/2 mCRC trial patient enrollment and demographics

Enrollment*

Number of Patients (N)	Phase 1b, Dose Level 0 Onvansertib 12 mg/m^2	Phase 1b, Dose Level +1 Onvansertib 15 mg/m^2	Phase 1b, Dose Level +2 Onvansertib 18 mg/m^2	Phase 2 RP2D Onvansertib 15 mg/m^2	Total Patients All Doses
Treated	6	6	6	32	50
Currently on treatment	0	1	0	15	16

Total Patients N=50	Median [range] or n (%)
Age (years)	61 [35-83]
Sex	
Male	28 (56%)
Female	22 (44%)
ECOG[1]	
0	33 (69%)
1	15 (31%)
Primary tumor site[2]	
Colon	27 (55%)
Rectum	17 (35%)
Other	5 (10%)

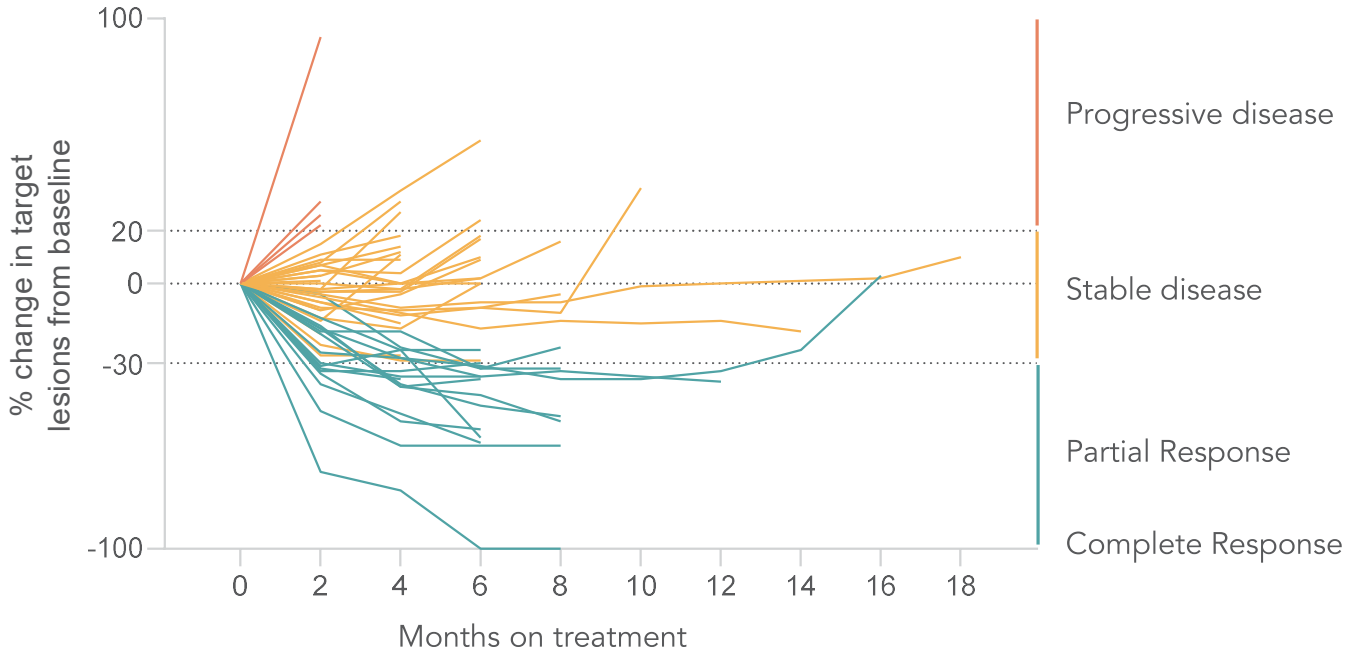
Total Patients N=50	Median n (%)
Liver metastasis[3]	
None	11 (22%)
Liver and other	29 (59%)
Liver only	9 (18%)
Number of metastatic organs[4]	
1	17 (35%)
≥2	32 (65%)
Prior bevacizumab treatment[5]	
Yes	33 (67%)
No	16 (33%)

as of 03-Dec-2021

* Jan 2022 data are interim as of Dec 3, 2021 from an ongoing trial and unlocked database. 1. ECOG not reported for two patients; 2. Primary tumor site not reported for one patient; 3. Liver metastasis presence not reported for one patient; 4. Number of metastatic organs not reported for one patient; 5. Prior bevacizumab treatment not reported for one patient

Deepening of responses observed as patients remain on treatment

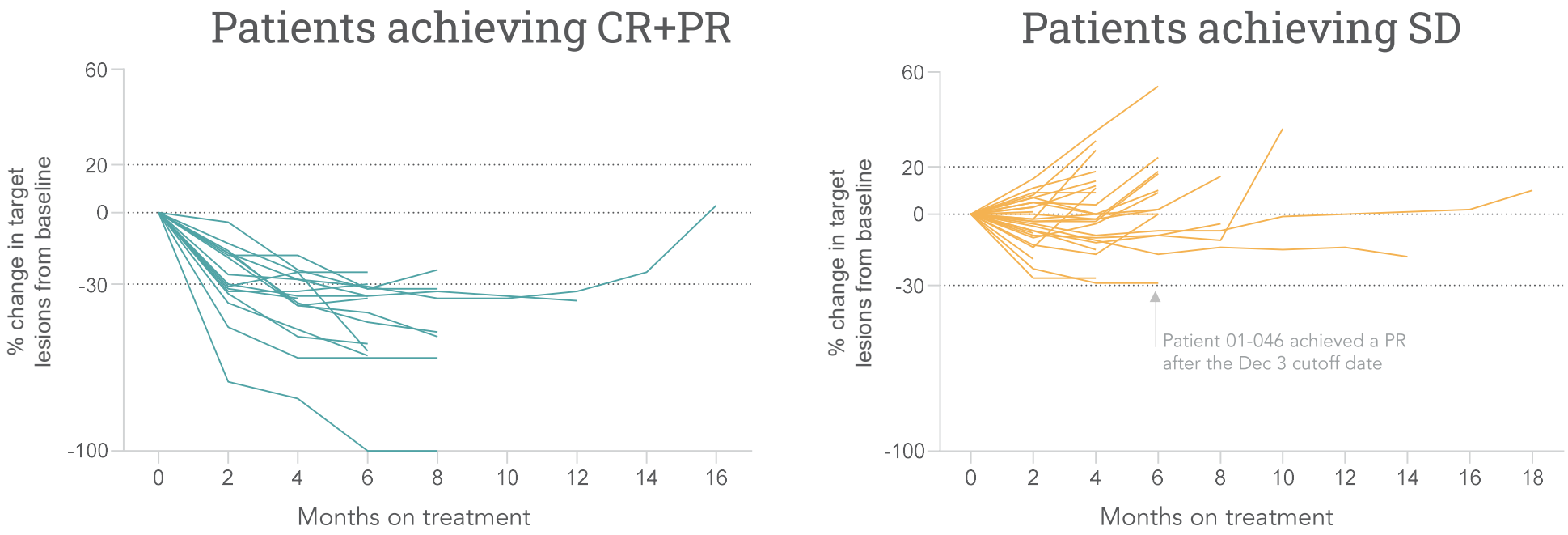
Change in tumor size from baseline* – all doses (as of Dec 3, 2021)



* Spider plot reflects interim data as of Dec 3, 2021 from an ongoing trial and unlocked database

Deepening of responses observed as patients remain on treatment

Change in tumor size from baseline* – all doses (as of Dec 3, 2021)



* Spider plots reflect interim data as of Dec 3, 2021 from an ongoing trial and unlocked database, and indicates one subsequent PR achieved on follow up through Jan 18, 2022 press release

Summary of onvansertib mCRC Ph1b/2 trial data over time

	ASCO GI Jan 2021	KOL Event Sept 2021		ASCO GI Jan 2022				Investor Webcast Jan 2022	
				Abstract		Poster			
Data Cutoff Date	Nov 1, 2020*	July 2, 2021*		Sep 16, 2021		Dec 3, 2021		Dec 3, 2021 + efficacy follow up through Jan. 18	
	All Doses	All Doses	RP2D	All Doses	RP2D	All Doses	RP2D	All Doses	RP2D
						At data cutoff		After data cutoff	
Evaluable Patients	14	32	19	44	31	48	35	48	35
ORR (CR+PR)	36% (5)	38% (12)	42% (8)	36% (16)	35% (11)	33% (16)	31% (11)	35% (17)	34% (12)
Confirmed PRs	29% (4)	31% (10)	37% (7)	Data not disclosed in abstract		27% (13)	29% (10)	1 patient waiting for confirmatory scan	
mPFS	-	9.4 mo	NR			9.4 mo	NR	No change from poster	
Disease control rate (CR+PR+SD)	86% (12)	94% (30)	100% (19)			92% (44)	94% (33)		

* Data release include certain follow up data. "Investor Webcast Jan 2022" column reflects interim data as of Dec 3, 2021 from an ongoing trial and unlocked database, and indicates one subsequent PR achieved on follow up through Jan 18, 2022 press release. NR: Not reached

KRAS-mutated mCRC: Cardiff Oncology's next steps

Cardiff Oncology management has decided to expand the activated Phase 2 trial and enroll ~40-50 additional patients as we prepare for initiation of the pivotal trial

- Obtain additional patient data:
  - Safety
  - Efficacy
  - Pharmacokinetic/pharmacodynamic (biomarkers)
- Continue assessing the value of KRAS response biomarker
- Keep current sites activated to lead into pivotal trial

Progression-free survival has ranged from 4.5 – 5.7 months

HISTORICAL REFERENCE

PFS	OS		
5.7	11.2	2006 – 2008	ML18147 Phase 3 Registrational Trial FOLFIRI + bev in second-line[1]
4.5	11.5	2000 – 2013	Systematic Literature-Based Analysis of 23 Randomized Trials (10,800 Patients) in Second-Line mCRC[2]
5.6	— Not reported for 2nd line	2015 – 2017	TRIBE2 Randomized Phase 3 Trial: SOC arm FOLFIRI + bev in Second-line following FOLFOX + bev First-line[3,4]

1. Bennouna et al., Lancet Oncol 2013; 14: 29–37; 2. Giessen et al., Acta Oncologica, 2015, 54: 187-193; 3. Cremolini et al., Lancet Oncol 2020, 21: 497–507; 4, Antoniotti et al., Correspondence Lancet Oncol June 2020. mCRC: metastatic colorectal cancer

Metastatic Pancreatic Adenocarcinoma (mPDAC)

Our Ph 2 trial in KRAS-mutated mPDAC combines onvansertib w/ SoC

One Cycle = 14 Days

WEEKS 1–2

2nd-Line SoC: Nal-IRI
+ Leucovorin + 5-FU

11 12 13 14

ONVANSERTIB

WEEKS 3–4

2nd-Line SoC: Nal-IRI
+ Leucovorin + 5-FU

25 26 27 28

ONVANSERTIB

Our Ph 2 trial in KRAS-mutated mPDAC combines onvansertib w/ SoC

EFFICACY END POINTS

1. Primary: Objective Response Rate (ORR) in patients who receive ≥28-days of treatment
2. Secondary: Duration of Response (DOR) and median overall survival (mOS)
3. Exploratory: Identification of biomarkers related to sensitivity and resistance to treatment using patient-derived organoids, blood samples, and archival tissue biopsies.

End-points measured tumor response and duration of response

HISTORICAL RESPONSE RATE

7.7%	ORR

PROGRESSION-FREE SURVIVAL

3.1 mo

DETAIL

PROOF OF CONCEPT CRITERIA

20% ORR

≥6 mo PFS

Metastatic Castrate Resistant Prostate Cancer (mCRPC)

Our Ph 2 study in mCRPC combines onvansertib with abiraterone

One cycle = 21 days

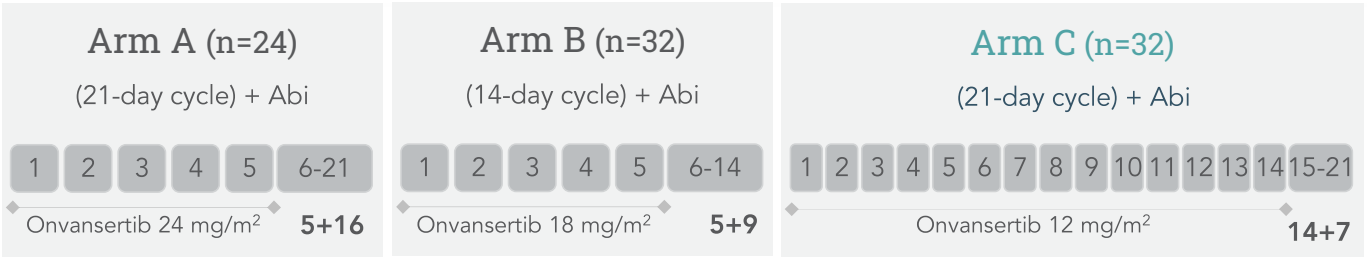
Arm C in our Ph 2 trial provides the greatest onvansertib dose density

Key Eligibility Criteria

- Initial signs of abiraterone resistance defined as 2 rising PSAs; one rise of ≥0.3 ng/mL separated by one week

Key Exclusion Criteria

- Prior treatment with either enzalutamide or apalutamide
- Rapidly progressing disease or significant symptoms related to disease progression



Number of patients (N)	Arm A (5+16)	Arm B (5+9)	Arm C (14+7)
Treated	24	20	18
Completing 12-weeks	14	14	10
Currently on Treatment	0	5	11

Enrollment as of August 2021

mCRPC: Metastatic castrate-resistant prostate cancer; PSA: Prostate specific antigen

Endpoints measure disease control and time to PSA progression

EFFICACY ENDPOINTS

1. Primary: Proportion of patients achieving disease control after 12-weeks of treatment, as defined by lack of PSA progression (per PCWG3 criteria)
2. Secondary: Radiographic response (per RECIST v1.1); time to PSA progression
3. Exploratory: Target inhibition of PLK1 and relevant biomarkers correlated with patient response

End-points measure disease control and time to PSA progression

HISTORICAL RESISTANCE TO ARSi

9-15 mo Resistance to ARSi

OVERALL SURVIVAL BENEFIT

~4 mo

One cycle = 21 days

PROOF OF CONCEPT CRITERIA

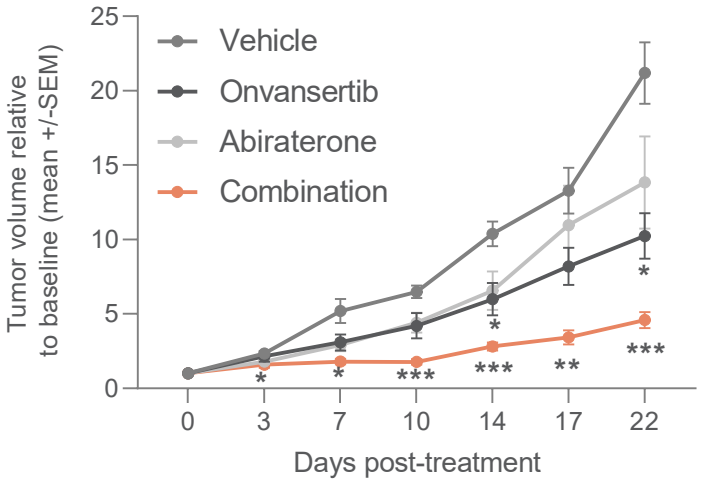
30% disease control rate

after 12-weeks of treatment

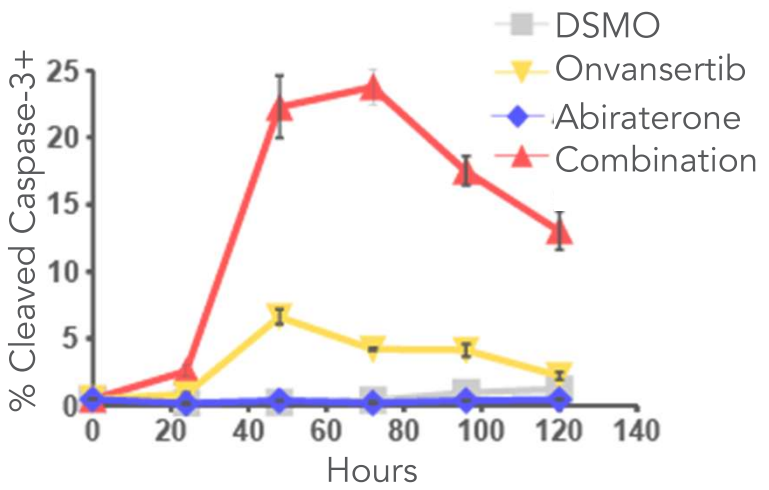
≥6 mo PFS

Onvansertib demonstrates synergy in an abiraterone-resistant setting

Onvansertib + Abiraterone Demonstrate Synergy in Abi-Resistant Model (LVCaP2CR)[1]



Onvansertib + Abiraterone Significantly Increases Apoptotic Cell Death[1]



- PLK1 is overexpressed in prostate cancer and linked to higher tumor grades[2]
- PLK1 inhibition + abiraterone demonstrated synergy in mCRPC *in vitro* and *in vivo* models: combination induced increased mitotic arrest and apoptosis in comparison with single agents alone
- Preclinical studies suggest that abiraterone sensitizes cells to onvansertib through regulation of mitotic processes

1. Data on-file. In collaboration with Michael Yaffe (MIT) and Jun Luo (John Hopkins University); [2]Weichert et al., Prostate 2004;60(3):240-5
Abi: Abiraterone; mCRPC: metastatic castrate-resistant prostate cancer

mCRPC trial baseline characteristics and safety (02-Jul-21)

Baseline Characteristics

Total patients N=51	Median [range] or n (%)
Age, years	72 [51-87]
Nonwhite ethnicity	7 (14%)
ECOG	
0	43 (84%)
1	7 (14%)
Years since diagnosis	4 [1-28]
Grade groups 4 and 5	29 (57%)
De novo metastatic disease	19 (37%)
Presence of bone metastasis	42 (82%)
Presence of visceral metastasis	18 (35%)
Baseline PSA, ng/mL	11.4 [0.6-515]
AR-V7+ at baseline*	10 (20%)
Baseline CTC/7.5 mL of blood**	15.8 [0-653]

Safety Assessment

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

Most Common Treatment-Emergent Adverse Events in Treated Patients (≥10% of patients)

Adverse Events n=number of patients (total N=51)	Grade 1	Grade 2	Grade 3	Grade 4	All Grades
Anemia	10 (20%)	6 (12%)	1 (2%)		17 (33%)
Fatigue	10 (20%)	3 (6%)			13 (25%)
Thrombocytopenia	11 (22%)	1 (2%)			13 (25%)
Neutropenia	1 (2%)	1 (2%)	7 (14%)		12 (24%)
Hypophosphatemia	3 (6%)	3 (6%)	4 (8%)		10 (20%)
WBC decrease	3 (6%)	2 (4%)	3 (6%)	2 (4%)	10 (20%)
Back pain	4 (8%)	3 (6%)			7 (14%)
Hypokalemia	3 (6%)	1 (2%)	1 (2%)		5 (10%)

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

mCRPC: Metastatic castrate-resistant prostate cancer; ECOG: Eastern Cooperative Oncology Group; PSA: Prostate specific antigen; AR-V7: androgen receptor variant 7, CTC: circulating tumor cells; WBC: white blood cells; AE: Adverse event

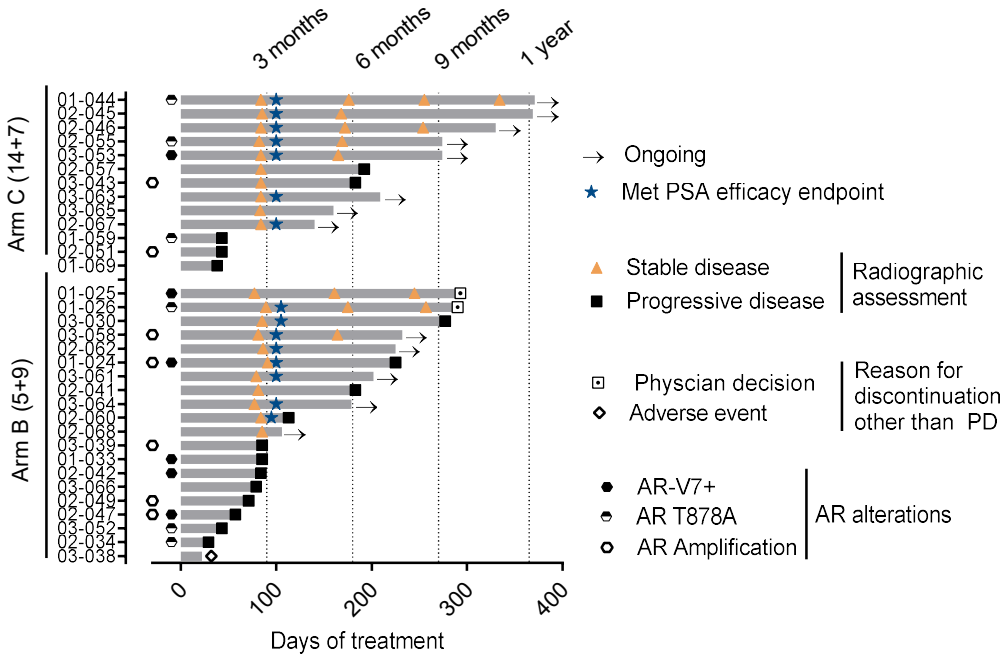
Arm C of our Ph 2 trial provides the greatest disease control

Preliminary Data Summary for Evaluable Patients

	Arm A (5+16)	Arm B (5+9)	Arm C (14+7)
Evaluable for efficacy*	17	20	13
Completed ≥ 12 weeks of treatment	14	16	10
Had radiographic or clinical progression within 12 weeks	3	4	3
Disease control at 12 weeks**	5 (29%)	8 (40%)	**7 (54%)**
Radiographic SD at 12 weeks	9 (53%)	11 (55%)	**10 (77%)**
Median progression-free survival (months)	4.8	6	**Not reached**

Increase in rate of patients achieving PSA stabilization and radiographic SD achieved with greater dose-density schedule in Arm C

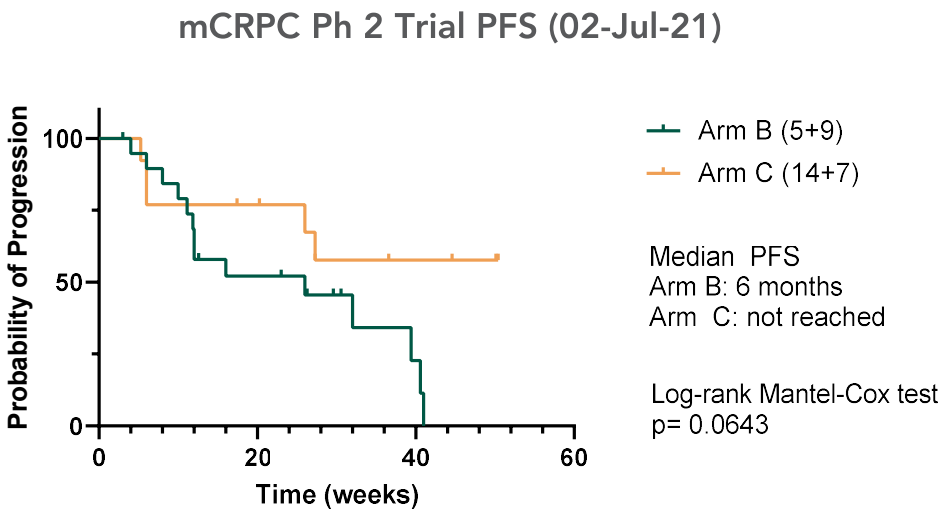
Treatment Response and Duration (02-Jul-21)



Efficacy was observed in patients harboring AR alterations across all 3 arms

* Completed at least 12 weeks of treatment or had radiographic/clinical progression within 12 weeks; **Defined as prostate specific antigen (PSA) stabilization or decline (PSA rise <25% over baseline); SD: Stable disease; DCR: Disease control rate; AR: Androgen receptor

Greater dose density of onvansertib Arm C trending to longer PFS

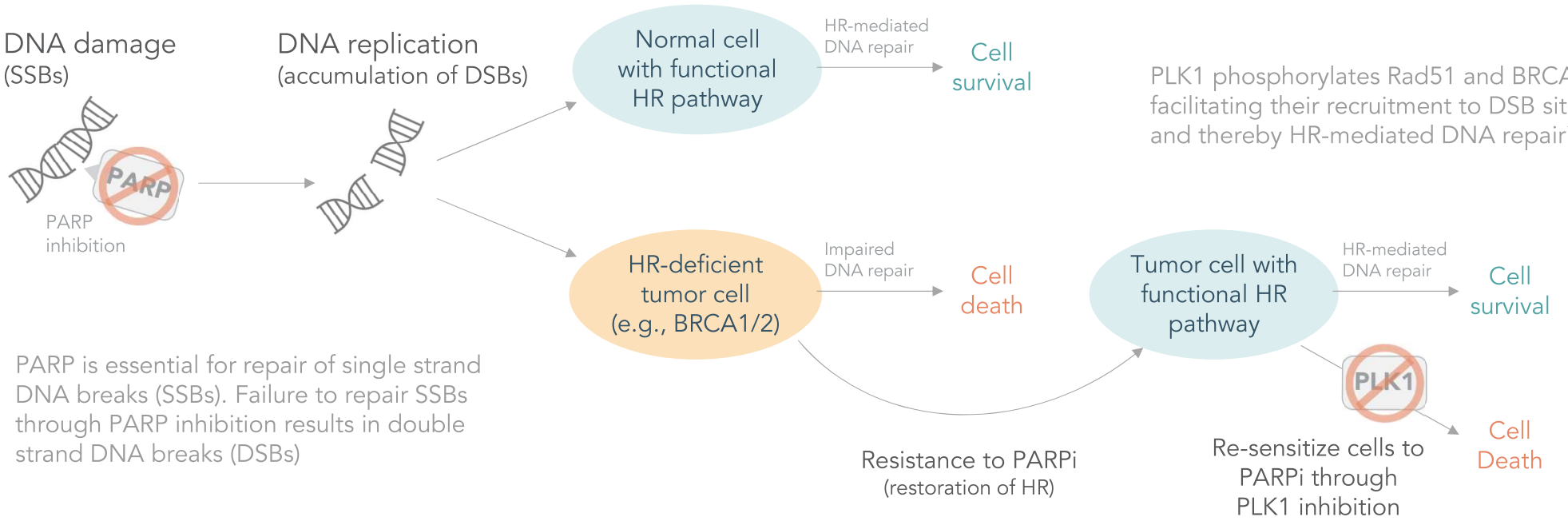


Arm	Median Survival	Lower 95% CI	Upper 95% CI
B	5.98	2.76	NA
C	Not reached	5.98	NA
Log-rank P-value		0.064	

PARPi Pre-Clinical Data

PLK1 inhibition re-sensitizes tumor cells to PARP inhibition

Onvansertib + PARP inhibitors



PLK1 phosphorylates Rad51 and BRCA1, facilitating their recruitment to DSB sites and thereby HR-mediated DNA repair[1]

PARP is essential for repair of single strand DNA breaks (SSBs). Failure to repair SSBs through PARP inhibition results in double strand DNA breaks (DSBs)

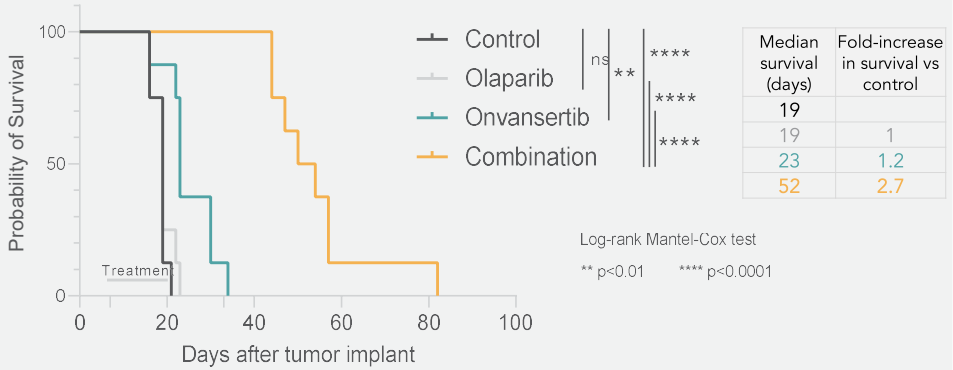
1. Yata et al. Mol. Cell 45, 371-383, 2012; Chabalier-Taste et al., Oncotarget 2016 Jan 19; 7(3): 2269-83; Peng et al., NAR 2021,49(13):7554-7570. HR: Homologous recombination; PARPi: PARP inhibitor

Preclinical studies demonstrate the benefit of PLK1 + PARP inhibitors

Onvansertib + PARP inhibitors*

Ovarian BRCA1 mutant PARPi-resistant PDX models

HGSOC, PDX#HOC22 (BRCA1 mutant)

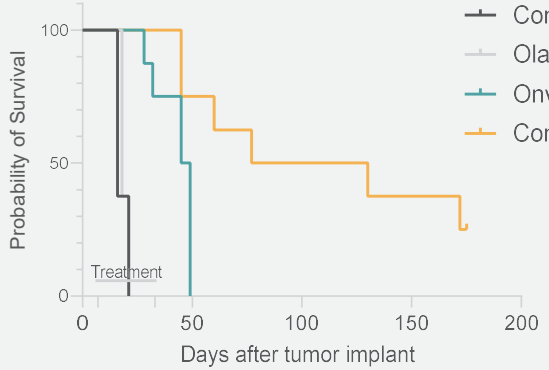


Median survival (days)	Fold-increase in survival vs control
19	
19	1
23	1.2
52	2.7

Log-rank Mantel-Cox test

** p<0.01 **** p<0.0001

HGSOC, PDX#HOC266 (BRCA1 mutant)



Median survival (days)	Fold-increase in survival vs control
16	
18	1.1
47	2.9
103	6.5

Log-rank Mantel-Cox test

** p<0.01 **** p<0.0001

2/8 mice were still alive at day 175

* Tumor cells (#HOC22 and #HOC266) were intraperitoneally transplanted and mice were treated for 4 weeks with vehicle, onvansertib, olaparib or the combination of onvansertib + olaparib. In collaboration with Giovanna Damia (IRFM, Italy). HGSOC: high grade serious ovarian cancer; PARPi: PARP inhibitor