UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, DC 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): **September 21, 2020**

Cardiff Oncology, Inc.
(Exact name of registrant as specified in its charter)

Delaware	**001-35558**	**27-2004382**
(State or other jurisdiction of incorporation or organization)	(Commission File Number)	IRS Employer Identification No.)

11055 Flintkote Avenue
San Diego, CA 92121
(Address of principal executive offices)

Registrant's telephone number, including area code: **(858) 952-7570**

Trovagene, Inc
(Former name or former address, if changed since last report)

Securities registered pursuant to Section 12(b) of the Act:

Title of each class:	Trading Symbol(s)	Name of each exchange on which registered:
Common Stock	CRDF	Nasdaq Capital Market

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communication pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter). Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure

Cardiff Oncology, Inc. (the "Company") intends to conduct meetings with third parties in which its corporate slide presentation ("Company Presentation") will be presented. The Company Presentation is attached to this Current Report on Form 8-K as Exhibit 99.1 and incorporated into this Item 7.01 by reference.

In accordance with General Instruction B.2 of Form 8-K, the information furnished under this Item 7.01 of this Current Report on Form 8-K and the exhibit attached hereto are deemed to be "furnished" and shall not be deemed "filed" for the purpose of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section, nor shall such information and exhibit be deemed incorporated by reference into any filing under the Securities Act of 1933, as amended, or the Securities Exchange Act of 1934, as amended.

Item 9.01. Financial Statements and Exhibits

(d) Exhibits.

99.1	Company Presentation

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Dated: September 21, 2020

CARDIFF ONCOLOGY, INC.

By: /s/ Mark Erlander
Mark Erlander
Chief Executive Officer

Cardiff Oncology™

Turning the Tide on Cancer

September 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 Cardiff Oncology's expectations, strategy, plans or intentions.

These forward-looking statements are based on Cardiff Oncology'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 Cardiff Oncology does not undertake any obligation to update publicly such statements to reflect subsequent events or circumstances.

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 & Clinical Trial Funding*	$30.5M
Common Stock Outstanding**	25.0M
• Convertible Preferred	3.5M
• Outstanding Options – weighted avg. exercise price / share $7.65	1.9M
• Option Pool (available for grant)	0.3M
• Outstanding Warrants – weighted avg. exercise price / share $4.23	8.7M
Total Fully Diluted Shares Outstanding	39.4M
Quarterly Cash Burn	1H'2020 – $3.8M/qtr. average
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

Experienced Management Team With Drug Development and Biomarker Technology Expertise

Mark Erlander, PhD
Chief Executive Officer

Vicki Kelemen
Chief Operating Officer

Brigitte Lindsay
Vice President of Finance

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

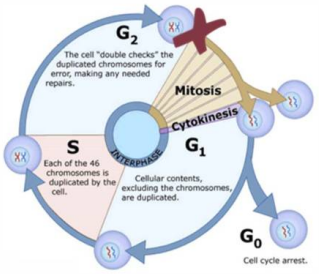
Cardiff Oncology™

Onvansertib

3rd generation, 1st in class, oral and highly selective Polo-like Kinase 1(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

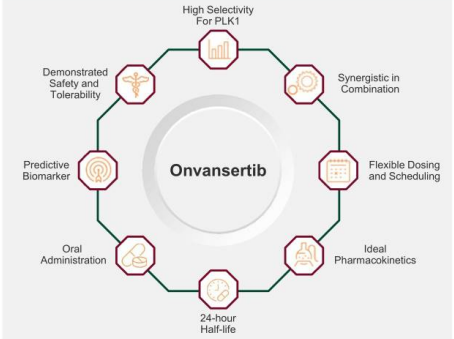


Inhibition of PLK1 causes arrest of cell division and subsequent cell death[1]

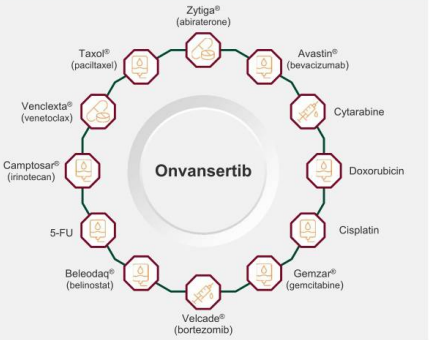
[1]Zitouni et al., Nat Rev Mol Cell Biol. 2014 Jul;15(7):433-52; PLK1: Polo-like kinase 1

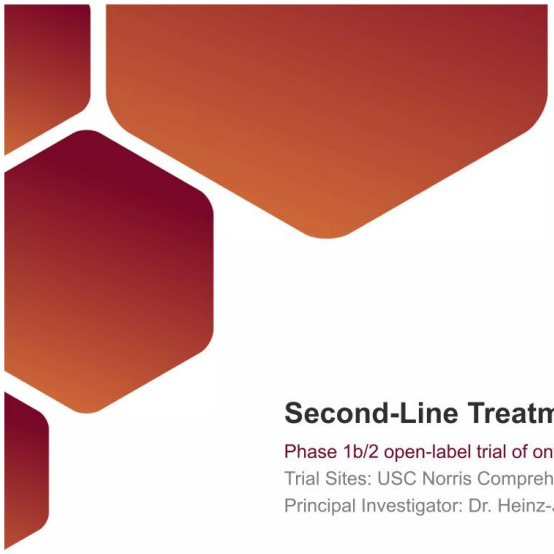
Onvansertib has Best-in-class Drug Properties and Synergistically Combines with Standard-of-Care Therapies

Best-in-Class Drug Properties



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





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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)

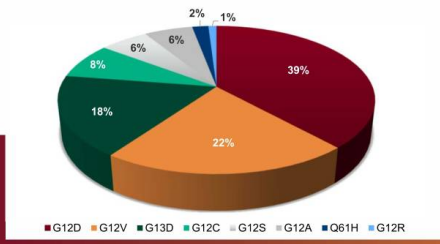
Current second-line standard-of-care treatment in KRAS-mutated mCRC has an overall response rate of 4% and progression-free survival (PFS) of 5.5 months[1]

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

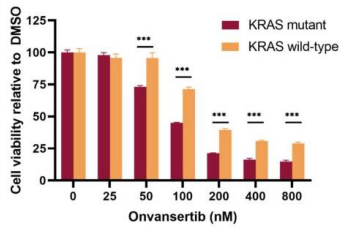
Synthetic Lethality: Cells with KRAS Mutations are Hypersensitive to Inhibition of PLK1

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

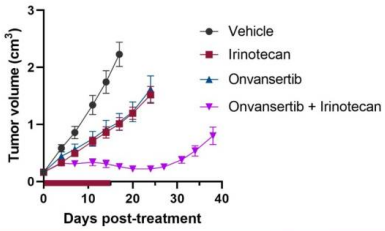


PLK1: Polo-like Kinase 1; mCRC: Metastatic colorectal cancer

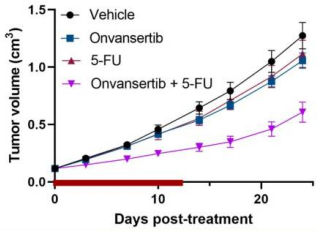
Synergy: Onvansertib in Combination with SOC Irinotecan and 5-FU

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

Synergy in Combination with Irinotecan

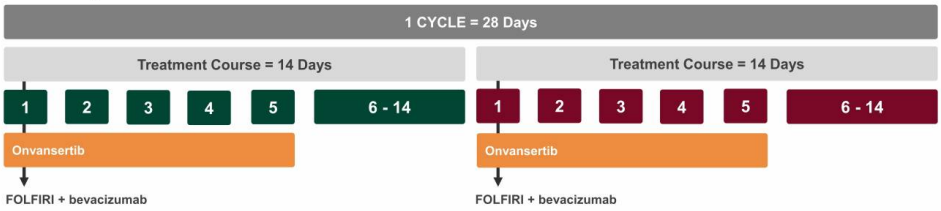


Synergy in Combination with 5-FU



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

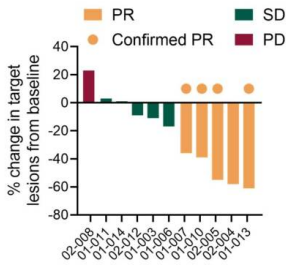
- ≥ 5 of 26 (~20%) patients achieve clinical response confirmed by radiographic scan
- Achieve median progression-free survival of ≥ 6 months

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

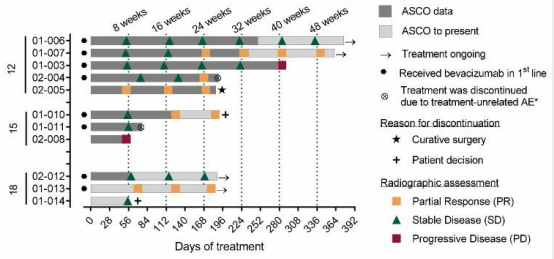


Response to Treatment Confirmed by Progression-Free Survival

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

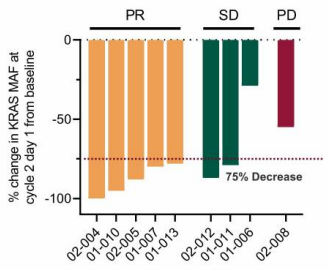


Serial Monitoring of KRAS is Predictive of Radiographic Scan Response

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 for a total of 20 patients
- 11 sites participating across the US
- 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)
- All 11 patients treated to-date 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
11	11	9

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

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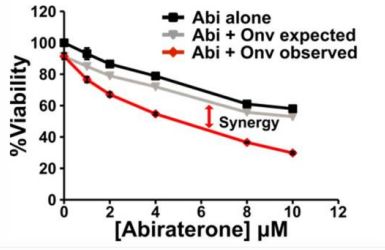
[1]Antonarakis, Emmannel – Current Understanding of Resistance to Abiraterone and Enzalutamide in Advanced Prostate Cancer; Clinical Advances in Hematology & Oncology – May 2016 – Volume 14, Issue 5; [2]Armstrong et al., 2019, JCO 37: 1120-1129; SOC: Standard-of-care; mCRPC: Metastatic castration resistant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

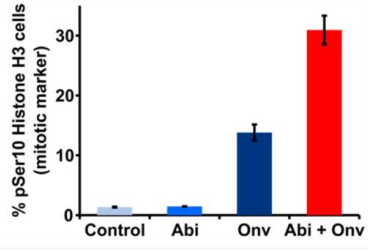
Onvansertib Extends the Response to Androgen Receptor Signaling Inhibitors

Onvansertib works synergistically in combination with abiraterone and significantly increases mitotic arrest

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



Onvansertib + Abiraterone 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 1 (n = 24)	Onvansertib 24mg/m^2 Days 1-5 (21-day cycle) + Zytiga® (Abiraterone)	4 Cycles = 12 Weeks	Disease Control (PSA Stabilization or Decline)
Cohort 2 (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 3 (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

- ≥6 of 32 (~20%) patients achieve primary efficacy endpoint of disease control at 12 weeks (PSA stabilization or decrease); confirmed by scan
- Achieve median radiographic PFS of ≥6 months

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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

Phase 2 Data Demonstrate the Efficacy of Onvansertib in mCRPC

Arm A: 17 patients were evaluable for efficacy

- 5 (29%) patients achieved disease control (DC)
- 9 (53%) had radiographic stable disease (SD) including 4 with durable SD (range 8 months – 1.7 years)

Arm B: 9 patients were evaluable for efficacy

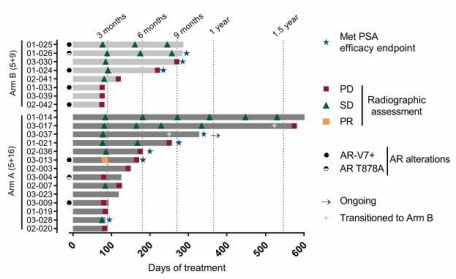
- 3 (33%) patients achieved DC
- 5 (55%) had SD including 4 with durable SD >7 months

Arm C: 3 patient safety lead-in completed

Efficacy demonstrated in patients with **AR alterations**

- N = 8 [AR-V7 (6), AR T878A (2)]
- 3 (37%) achieved DC
- 4 (50%) had SD; 3 durable (range 7-9 months)

Treatment Response and Duration

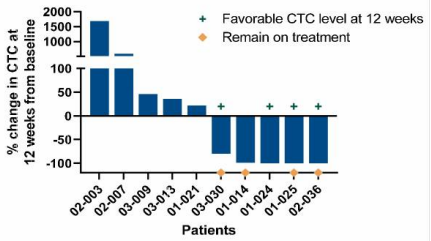


Onvansertib-Induced Circulating Tumor Cell Decrease is Associated with Progression-Free Survival

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, 25 (78%) patients had unfavorable CTC count
- 10 patient with unfavorable CTC count were re-analyzed 12 weeks post-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
  - Median time on treatment for patients with CTC decrease (n=5) is 7 months to-date, with 4 patients remaining on treatment
  - Conversely, median time on treatment for patients with CTC increase (n=5) was 5 months

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



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)

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New Clinical Programs Planned

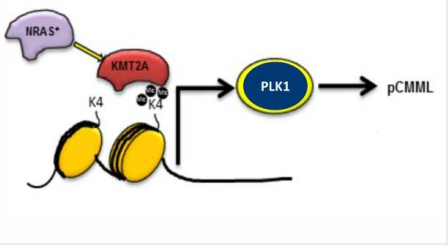
Chronic Myelomonocytic Leukemia (CMML)
Pancreatic Ductal Adenocarcinoma (PDAC)

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 onvanserib, a novel oral PLK1 inhibitor in RAS-pathway mutant chronic myelomonocytic leukemia

Trial Design:

	Dosing Schedule	Duration	Efficacy Endpoint
Arm A (n = 38)	Onvansertib 24 mg/m^2 days 1-5 (14-day cycle)	4 cycles monotherapy (option to add decitabine at cycle 5 if lack of efficacy with single agent)	Interim analysis of first 18 patients after 4 cycles to evaluate objective response
Arm B (n = 38)	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

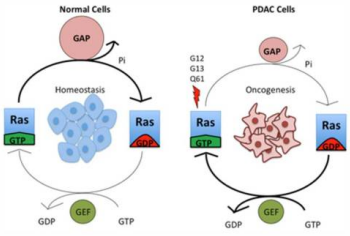
- ≥4 of 32 (12.5%) patients with an objective response to single agent treatment with Onvansertib in the first 4 or the first 3 cycles of treatment (Arm A or B, respectively)

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

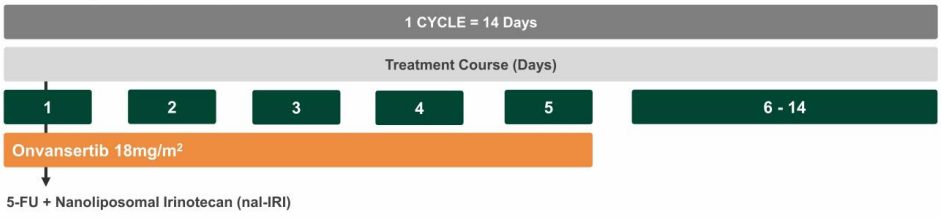
- KRAS is the most common oncogene mutated in pancreatic adenocarcinoma, which is present in ~95% of tumors
- Mutant KRAS is essential for PDAC growth, where the constitutive activated RAS proteins contribute to tumorigenesis, treatment resistance and metastases
- No effective RAS inhibitors have been 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 (35 patients):



Eligibility Criteria

- Patient with tumors harboring a confirmed KRAS mutation
- Patient that have not had prior irinotecan or nal-IRI

Efficacy Endpoints

- Overall response in patients who receive ≥2 cycles of treatment
- Progression-free survival at 6 months
- Decreases in KRAS mutation burden and response to treatment

What is Clinical Trial Success

- Achieve ≥10 of 35 (~30%) patients achieve clinical response confirmed by radiographic scan
- Achieve median progression-free survival of ≥ 6 months

Cardiff Oncology™

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

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 & Clinical Trial Funding*	$30.5M
Common Stock Outstanding**	25.0M
• Convertible Preferred	3.5M
• Outstanding Options – weighted avg. exercise price / share $7.65	1.9M
• Option Pool (available for grant)	0.3M
• Outstanding Warrants – weighted avg. exercise price / share $4.23	8.7M
Total Fully Diluted Shares Outstanding	39.4M
Quarterly Cash Burn	1H'2020 – $3.8M/qtr. average
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

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