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): July 17, 2018

Trovagene, 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

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

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.

Trovagene, Inc. intends to conduct meetings with third parties in which its corporate slide presentation will be presented. A copy of the presentation materials is attached as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

The information in this Item 7.01 and the document attached as Exhibit 99.1 are being furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities and Exchange Act of 1934, as amended (the "Exchange Act"), nor otherwise subject to the liabilities of that section, nor incorporated by reference in any filing under the Securities Act of 1933 or the Exchange Act, except as shall be expressly set forth by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits

(d) Exhibits.

99.1 Trovagene, Inc. Corporate 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: July 17, 2018

TROVAGENE, INC.

By: /s/ Thomas Adams
Thomas Adams
Interim Chief Executive Officer

Exhibit 99.1

Using Our Precision Medicine Strategy to Develop Oncology Drugs That Target Mitosis

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JULY 2018

Forward-Looking Statements

Certain statements in this presentation are forward-looking within the meaning of the Private Securities Litigation Reform Act of 1995. These statements may be identified by the use of words such as "anticipate," "believe," "forecast," "estimated" and "intend" or other similar terms or expressions that concern Trovagene's expectations, strategy, plans or intentions.

These forward-looking statements are based on Trovagene's current expectations and actual results could differ materially. There are a number of factors that could cause actual events to differ materially from those indicated by such forward-looking statements. While the list of factors presented in the 10-K is considered representative, no such list should be considered to be a complete statement of all potential risks and uncertainties. Unlisted factors may present significant additional obstacles to the realization of forward-looking statements. Forward-looking statements included herein are made as of the date hereof, and Trovagene does not undertake any obligation to update publicly such statements to reflect subsequent events or circumstances.

Strategy for Oncology Drug Development

- Taking a precision medicine approach to developing PCM-075 by integrating a highly-selective antimitotic drug with a biomarker strategy
- Leveraging a proven cancer target, PLK1, that is highly expressed in tumor cells and integral to cell division (mitosis)
- Developing a first-in-class, 3rd generation PLK1 inhibitor, benefiting from prior drug class clinical experience, including efficacy, safety and single vs combination therapy trial design
- Combining PCM-075 with already approved drugs that have demonstrated synergy in combination:
  - Phase 1b/2 trial of PCM-075 + cytarabine or decitabine in Acute Myeloid Leukemia (AML)
  - Phase 2 trial of PCM-075 + abiraterone acetate (Zytiga®) in metastatic Castration-Resistant Prostate Cancer (mCRPC)
- Pursuing partnership opportunities with Japanese companies to expand development of PCM-075

Our PCM-075 Oncology Pipeline
Opportunities in Solid Tumors and Leukemias/Lymphomas

	Preclinical	Phase 1	Phase 2
Leukemias & Lymphomas	Acute Myeloid Leukemia – Orphan Drug Designation in the U.S. *Phase 1b/2 trial in combination with low-dose cytarabine (LDAC) or decitabine*		
	Non-Hodgkin Lymphoma		
Solid Tumor Cancers	Metastatic Castration-Resistant Prostate *Phase 2 trial in combination with with abiraterone acetate (Zytiga®)/prednisone*		
	Lung		
	Colon		
	Ovarian		
	Others (adrenocortical, sarcomas, head and neck, skin, liver, pancreatic, ampullary)		
	Triple Negative Breast		

Trovagene's Management Team
Proven Leadership in Oncology

Tom Adams, PhD
Chairman of the Board
Interim CEO

Iris · Genta · SYNERGY PHARMACEUTICALS · CHUGAI · GEN-PROBE

Mark Erlander, PhD
Chief Scientific Officer

BIOMÉRIEUX · Johnson & Johnson PHARMACEUTICAL RESEARCH & DEVELOPMENT, L.L.C. · THE SCRIPPS RESEARCH INSTITUTE

Sandra Silberman, MD, PhD
Chief Medical Advisor

DukeHealth · ARCHER · Moleculin · QUINTILES · NOVARTIS

George Samuel, Esq.
VP, General Counsel

Cooley LLP · DLA PIPER

Vicki Kelemen
VP, Corporate Communications

AMYLIN · Agouron Pharmaceuticals, Inc. A Pfizer Company · MAXIM PHARMACEUTICALS · Dura Pharmaceuticals · Bayer HealthCare Pharmaceuticals

Scientific Advisors
Principal Investigators and Collaborators

- **Jorge Cortes, MD – MD Anderson**
  - Deputy Chair, Professor of Medicine, Department of Leukemia and Director of CML and AML programs
- **Glenn Bubley, MD – Beth Israel Deaconess Medical Center**
  - Director, Multidisciplinary Genitourinary Cancer Program
- **David Einstein, MD – Beth Israel Deaconess Medical Center**
  - Principal Investigator, mCRPC Phase 2 Trial
- **Filip Janku, MD, PhD – MD Anderson**
  - Associate Professor, Investigational Cancer Therapeutics (Phase 1 Clinical Trials Program)
- **Michael Yaffe, MD, PhD – MIT**
  - Director, MIT Center for Precision Cancer Medicine, Professor of Biology and Biological Engineering
- **Amer Zeidan, MBBS, MHS – Yale**
  - Assistant Professor of Medicine

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center

UCLA Jonsson Comprehensive Cancer Center

Massachusetts Institute of Technology

DANA-FARBER CANCER INSTITUTE

Beth Israel Deaconess Medical Center

MGH 1811 MASSACHUSETTS GENERAL HOSPITAL

Yale CANCER CENTER
A Comprehensive Cancer Center Designated by the National Cancer Institute

Licensed Drug Candidate from NMS
PCM-075 – Polo-like Kinase 1 (PLK1) Inhibitor

NERVIANO MEDICAL SCIENCES

Oncology Drug Discovery

- Largest oncology research and development company in Italy
- Developed anthracycline class of drugs (doxorubicin)
- Leader in protein kinase drug development (Polo-like Kinase Inhibitors)
- Identification and validation of molecular targets focused on driver oncogenes, cell-cycle regulation and DNA repair, cancer metabolic pathways and immune oncology
- Excellent track record licensing innovative drugs to pharma/biotech companies including: Genentech (Roche), Ignyta (Roche), Novartis

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Developing Oncology Drugs That Target Mitosis

- Licensed global development and commercialization rights for PCM-075
- Nerviano will continue manufacturing GMP API and finished drug
- Two active INDs in place with the FDA (solid tumor and hematologic cancers)
- Partnering discussions ongoing with several Japanese companies to expand development of PCM-075
- Financing in place to advance clinical trial program into mid-2019

IND = Investigational New Drug

Leveraging Proven Cancer Target

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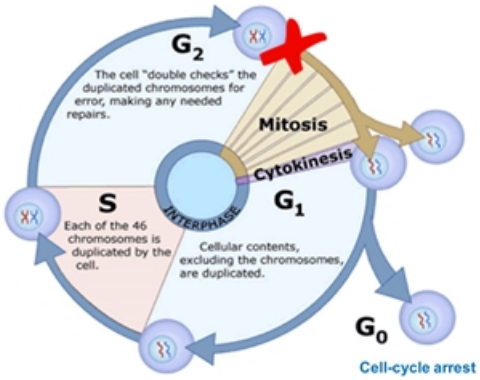
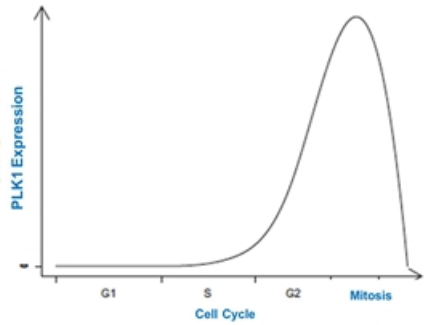
PLK1: Established Target for Cancer Therapy

PLK1 Plays a Critical Role in Initiation, Maintenance and Completion of Mitosis





- PLK1 belongs to a family of kinases (PLK1, 2,4,4 and 5)
- Dysfunction of PLK1 promotes cancerous formation and drives cancer progression
- PLK1 is over-expressed in dividing cells in numerous cancers and is associated with poor patient prognosis[1]
- Inhibition of PLK1 interferes with multiple stages of mitosis and leads to cancer cell death

[1]Liu et al- PLK1, A Potential Target for Cancer Therapy; Translational Oncology – Vol. 10 – pp. 22-32; February 2017

PLK1 is Over-Expressed in Multiple Cancers

PLK1 Inhibitors in Cancer Therapy: From Laboratory to Clinics

"In our view, combined therapies targeting other relevant pathways together with Plk1 may be vital to combat issues observed with monotherapy, especially resistance."
July 2016

Randomized, Phase 2 Trial of Low-Dose Cytarabine with or without Volasertib in AML patients not suitable for Induction Therapy

"By adding volasertib to LDAC, the overall response was more than doubled, with 31% vs 13% for LDAC alone."
August 2014

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

"Our results offer a strong mechanistic rationale to evaluate PLK1 inhibitors in combination drug trials to enhance the efficacy of Androgen Signaling Inhibitors in mCRPC."
September 2014

Overexpression of PLK1 Observed in Numerous Cancers	
Tumor Type	**PLK1 Fold Change Over-Expression**
AML	13.0
B-cell Lymphoma	56.3
Prostate	3.3
Adrenocortical	4.5
Lung Adeno	9.7
Lung Squamous	20.8
Breast	11.3
Esophageal	10.2
Stomach	4.8
Colon	2.5
Head & Neck	4.2
Pancreatic	2.2
Ovarian	31.7
Glioblastoma	12.4
Kidney	4.7
Liver	11.7
Uterine	21.3
Bladder	9.1

[1]Liu et al- PLK1, A Potential Target for Cancer Therapy; Translational Oncology – Vol. 10 – pp. 22-32; February 2017

Developing First-in-Class 3rd Generation PLK1 Inhibitor

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PLK Inhibitor Landscape

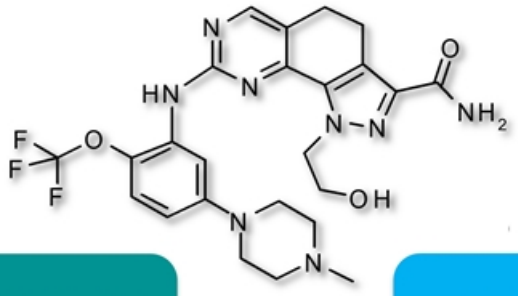
- 1st Generation PLK inhibitors: BI-2536, TAK-960, GW843682
  - Pan inhibitors: No selectivity between PLK1, 2, & 3[1]
  - TAK-960, GW843682 (toxicity issues)
  - Phase 1 & 2 results (BI-2536)
    - Tolerable and reversible hematological toxicities[2-4]
    - Limited efficacy as single agent in AML[3], lymphoma[2], and solid tumors[4]
- 2nd Generation PLK inhibitor: BI-6727
  - Pan inhibitor for PLK 1, 2 & 3
  - Long half-life (~5 days) and I.V. (intravenous) formulation
  - Phase 2 results
    - Significant efficacy demonstrated in AML for the combination BI-6727 + cytarabine[5]
    - Limited efficacy as single agent observed in ovarian cancer[6]

[1]https://www.medchemexpress.com; [2]Vose et al., Leukemia & Lymphoma 2013 54:4, 708-713;[3]Müller-Tidow et al., Br J Haematol. 2013 Oct;163(2):214-22; [4]Awad et al., Lung Cancer. 2017 Feb;104:126-130;[5]Dohner et al., Blood, 2014 124:9, 1426-1433; [6]Pujade-Lauraine, E et al., J Clin Oncol. 2016 Mar 1;34(7):706-13

PCM-075: 3rd Generation PLK1
Best-in-Class Attributes

Highly Selective

Orally Available



Ideal Pharmacokinetics

Favorable Safety and Tolerability

Synergistic in Combination

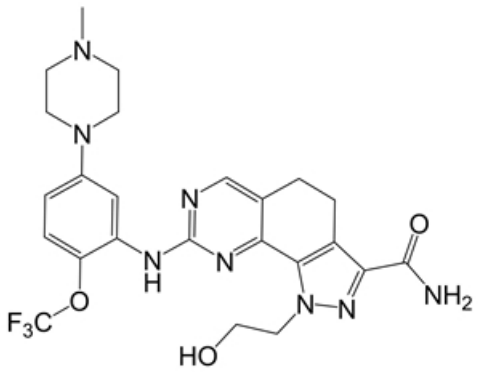
PCM-075: Characteristics

- PCM-075 is a small molecule, selective PLK1 inhibitor (MW 650 Daltons)[1]
  - Half-life of ~24 hours
  - Formulated as 5 mg and 20 mg hard gelatin capsules
  - 4-year shelf-life when stored at 5°C ± 3°C (36°F to 46°F)
  - Nerviano manufacturer for GMP API and finished goods

- ADME Profile[2]
  - Highly stable in human hepatocytes
  - Plasma protein binding ranging from 83% to 93% in the different species
  - No Cytochrome P450 inhibition observed at therapeutic concentrations
  - Good oral bioavailability, low-medium clearance and high-volume of distribution in all tested species in single and repeated pharmacokinetic studies

[1]Data on File, Trovagene, Inc.; [2]ADME = Absorption, Distribution, Metabolism, Excretion

PCM-075 Intellectual Property



- Four worldwide patent families
  - Genus, Compound, Combinations, Salt
- Mature portfolio
  - Granted in most major jurisdictions
- Patent term 2030 plus up to 5 years extension

PCM-075: Selective 3rd Generation PLK1

Selective PLK1 Inhibitor

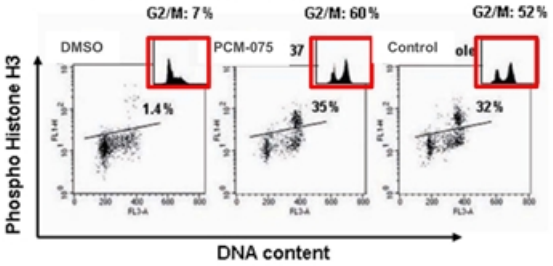
- Tested against >260 kinases and PLK1 was the only active target (IC_{50} of 2nM)
- Selectivity driven by polar interaction with the carboxyl side chain of Glutamate 140 position of PLK1[1]

PLK Member	PCM-075 IC50* (µM)
PLK1	0.002
PLK2	> 10
PLK3	> 10

Induces tumor cell death by G_2M cell cycle arrest

- Treatment of cells with PCM-075 resulted in a clear mitotic block accompanied by an increase of the G2/M population (4N DNA content)

AML-NS8 Patient-Derived Cells Treated with 200 nM PCM-075 for 24 Hrs[1]



[1]Data on File, Trovagene, Inc.

Phase 1 Safety Trial in Solid Tumors[1]
Favorable First-in-Human Data

Phase 1 Dose Escalation Trial in Patients with Advanced or Metastatic Solid Tumors

Phase 1 Trial Design

Open-label dose escalation trial in patients with solid tumor malignancies

19 of 21 patients enrolled administered PCM-075 orally, once daily for 5 consecutive days, every 3 weeks

Included: colorectal, pancreatic, lung, sarcomas, hepatocellular, ampullary, prostate, ovarian, skin

PCM-075 Trial Results

1. Established safety of PCM-075 and identified a recommended Phase 2 dose of 24 mg/m^2/day
2. 16 of the 19 patients (84.2%) treated with PCM-075, were evaluable for efficacy, with stable disease at any dose observed in 5 (31.2%) of the patients
3. Reversible, on-target thrombocytopenia and neutropenia, consistent with the expected mechanism of action, were the primary adverse events
4. No GI disorders, mucositis, or alopecia was observed, confirming that bone marrow cells are the most sensitive to PCM-075 inhibition with the applied dosing schedule

[1]Weiss G et al., Phase I dose escalation study of NMS-1286937, an orally available Polo-like Kinase 1 inhibitor, in patients with advanced or metastatic solid tumors – Invest. New Drugs DOI 10.1007/s10637-017-0491-7

Benefiting From Drug Class Experience

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2nd Generation PLK Inhibitor (BI-6727) Randomized Phase 2 Clinical Trial in AML

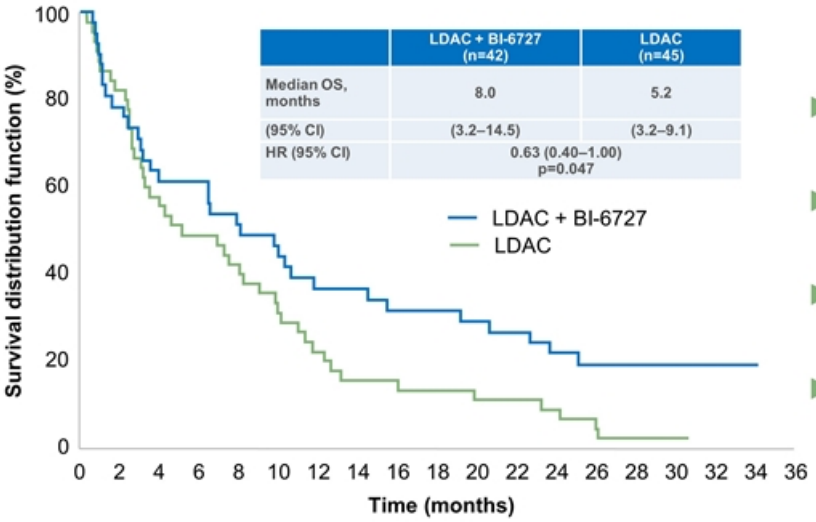
Randomized Phase 2 Trial of BI-6727 + LDAC vs LDAC Alone in Acute Myeloid Leukemia

- Adults with previously untreated AML
- Ineligible for intensive therapy

n=42: LDAC 2x20 mg/day sc, Days 1–10 + BI 6727 50 mg Days 1 + 15 Every 28 days

n=45: LDAC 2 x 20 mg/day sc, Days 1–10 Every 28 days

4-week cycles until progression, relapse, intolerance, patient/ or investigator requested



	LDAC + BI-6727 (n=42)	LDAC (n=45)
Median OS, months	8.0	5.2
(95% CI)	(3.2–14.5)	(3.2–9.1)
HR (95% CI)	0.63 (0.40–1.00) p=0.047	

Results

- Increased response rate with BI-6727 + LDAC (31.0%) vs LDAC alone (13.3%)
- Significant survival benefit in elderly patients ineligible for induction therapy
- Response across all AML genetic subgroups
- Clinically manageable safety profile

2nd Generation PLK Inhibitor (BI-6727)
Randomized Phase 3 Clinical Trial in AML

- Primary analysis for efficacy included 371 of a total 666 patients randomized
- Results: BI-6727 + LDAC vs placebo + LDAC
  - Increase in remission rate (CR + CRi):
    - 25.2% vs 16.8% (central assessment) – Overall Response 1.659; p=0.071
    - 29.7% vs 19.2% (investigator assessment) – Overall Response 1.757; p=0.034
  - Negative overall survival trend (HR: 1.26; p=0.1129)
  - Increased frequency of fatal AEs (fatal infections being the major contributor)
- Trial was unblinded after the primary analysis
  - Patients remaining on treatment were allowed to continue based on individual benefit-risk evaluations

HR = Hazard Ratio.

PCM-075 Clinical Development
Benefiting From Class Experience

Product Attributes	1st and 2nd Generation PLK Inhibitors	3rd Generation PCM-075
Selectivity for PLK1	• panPLK inhibition of PLK1,2,3*	• Highly-selective only for PLK1
Antileukemic Activity	• Phase 2 & 3 trial results indicate activity • Improved response rates	• Early data from Phase 1b/2 trial indicates activity • Biomarker strategy to identify patients most likely to respond
Dosing and Schedule	• Fixed treatment schedule • Fixed dose for all patients	• Treatment schedule flexibility • Dose determined based by BSA
Tolerability	• Insufficient time between treatment cycles negatively impacted tolerability/survival	• Time allotted between cycles for patient recovery from drug-induced neutropenia
Infection Prophylaxis	• Increased rate of fatal infections in patients not given prophylactic antibiotics	• Protocols require mandatory prophylactic antibiotics

*PLK2-PLK5 have properties more consistent with tumor suppressor genes and are not essential for cell division; BSA = Body Surface Area.

Combination Therapy Approach

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PCM-075: Combination Therapy Strategy

- Combination therapy is considered the cornerstone of precision cancer medicine
- Demonstrated synergy in combination with chemotherapies and targeted therapeutics
- Enhances efficacy compared to monotherapy approach because it targets key pathways in a characteristically synergistic or additive manner
- Approach potentially reduces drug resistance, while simultaneously providing therapeutic benefits, including:
  - Reducing tumor growth and metastatic potential
  - Arresting mitotically active cells
  - Inducing apoptosis (programmed cell death)

[1]Mokhtari, R et al - Combination Therapy in Combatting Cancer – Oncotarget, 2017, Vol. 8 (No. 23), pp: 38022-38043

PCM-075: Synergistic in Combination

- High PLK1 expression is associated with the most aggressive forms of solid tumor cancers, leukemias and lymphomas
- Synergistic activity of PCM-075 may enhance efficacy of standard-of-care therapies

Potentially Synergistic Drugs[1,2]
Abiraterone acetate
Bevacizumab
Bortezomib
Cisplatin
Cytarabine
Doxorubicin
FLT3 Inhibitors (Quizartinib)
Gemcitabine
HDAC Inhibitors (Belinostat)
Paclitaxel

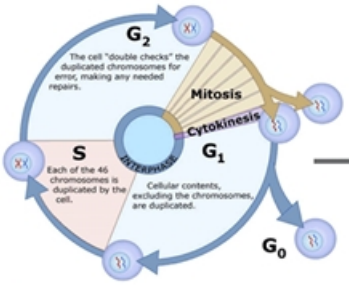
Associated Cancers[2]
Leukemias/Lymphomas:
• Acute Myeloid Leukemia • Acute Lymphocytic Leukemia • Non-Hodgkin Leukemia • Multiple Myeloma
Solid Tumor Cancers
• Castration-Resistant Prostate • Adrenocortical Carcinoma • Triple Negative Breast • Sarcomas • Small Cell Lung • Colon

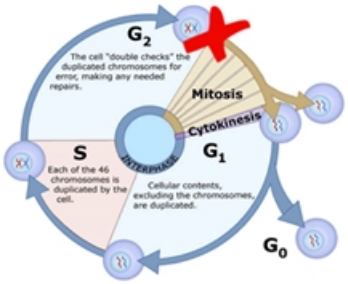
[1]Alphabetical order. [2]Preclinical data on file with PCM-075 and these combined therapeutics

PCM-075: Rationale for Combination with DNA Damaging Agents[1,2]

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

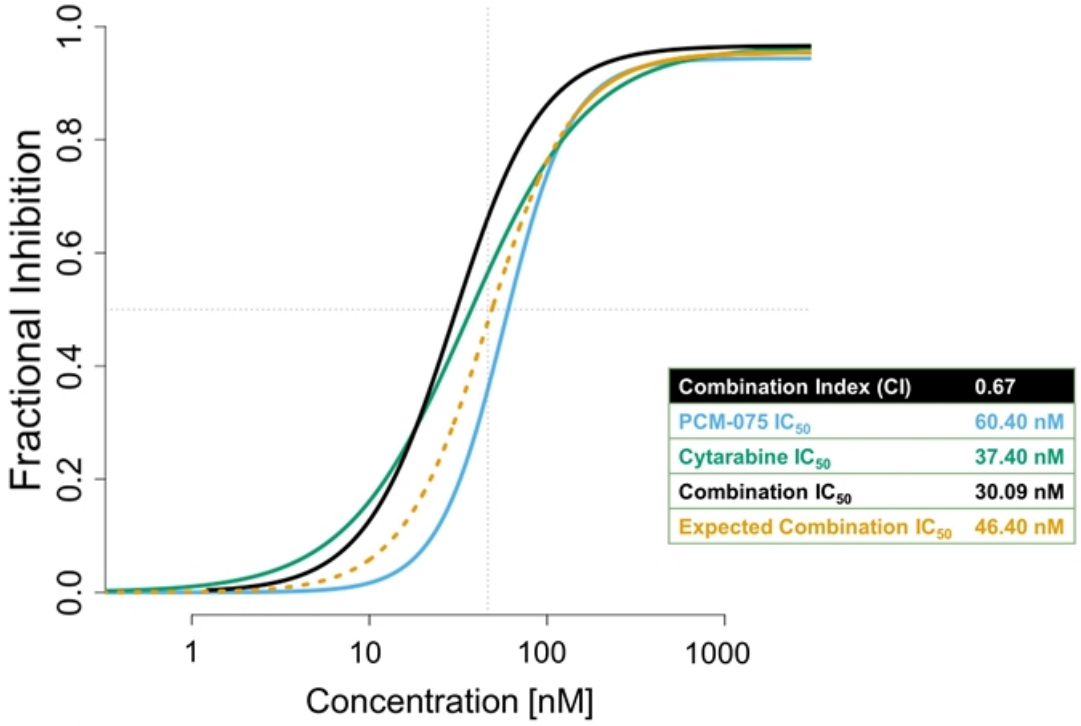
DNA Damaging Agents

- Cytarabine
- Doxorubicin
- Cisplatin

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

Synergy: PCM-075 + Cytarabine
Acute Myeloid Leukemia (AML) Cell Line (HL-60)

Greatest Synergy Observed with Highest % Cell Death



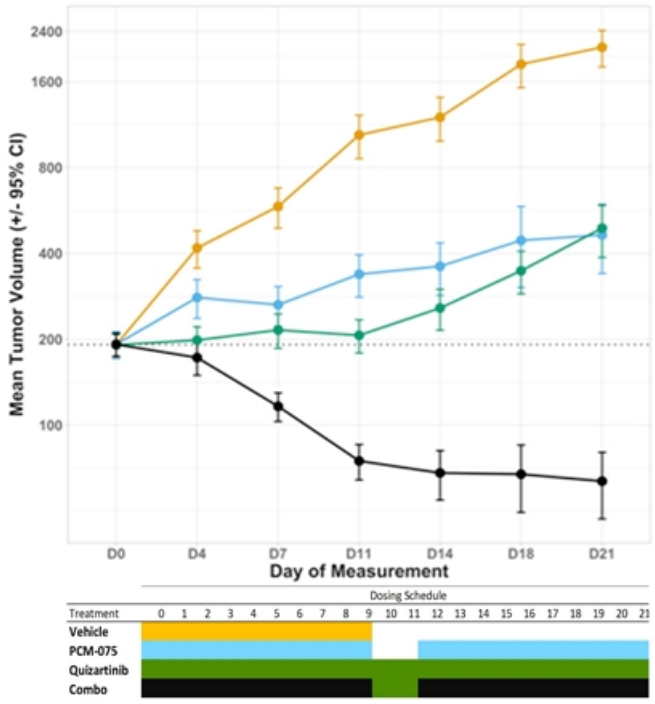
Combination Index (CI)	0.67
PCM-075 IC_{50}	60.40 nM
Cytarabine IC_{50}	37.40 nM
Combination IC_{50}	30.09 nM
Expected Combination IC_{50}	46.40 nM

Data: Nerviano Medical Sciences

Synergy: PCM-075 + FLT3 Inhibitor
Acute Myeloid Leukemia (AML)

- 30% of AML patients harbor a FLT3 mutation[1]
- Midostaurin (FDA approved); 3 additional FLT3 inhibitors, including quizartinib, are currently in Phase 3 clinical development[2]
- The combination of PCM-075 plus quizartinib demonstrated 97% tumor growth inhibition and regression in FLT3 AML xenograft model[3]

Evaluation of Efficacy of PCM-075 for MV-4-11 Human Acute Myeloid Leukemia (AML) Xenograft Model in NOD.SCID Mice

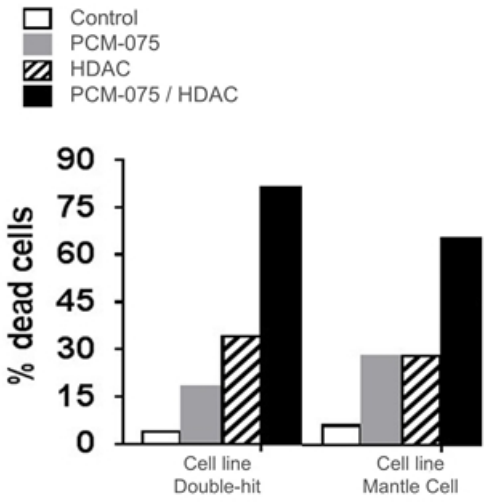


[1]Kindler et al, Blood 2010; 116:5089-10. [2]Stone et al, N Engl J Med 2017; 377:454-64.
[3]Data on File at Trovagene, Inc.

Synergy: PCM-075 + HDAC* Inhibitor
Non-Hodgkin Lymphoma (NHL)

- Aggressive NHL progresses rapidly; accounts for 60% of cases in the U.S.
- Subtypes:
  - Diffuse large B-cell lymphoma (DLBCL), including double-hit
  - Mantle cell lymphoma
  - Peripheral T-cell lymphoma (PTCL)
- Demonstrated synergy of PCM-075 in combination with a HDAC inhibitor in double-hit, mantle-cell[2] and T-cell lymphoma cell lines[3]
- Medical need for improved duration of HDAC inhibitor response

PCM-075 Combined with HDAC Inhibitor Reduces Survival of Double-Hit (DLBCL) and Mantle Cell Lymphoma Cell Lines[1]



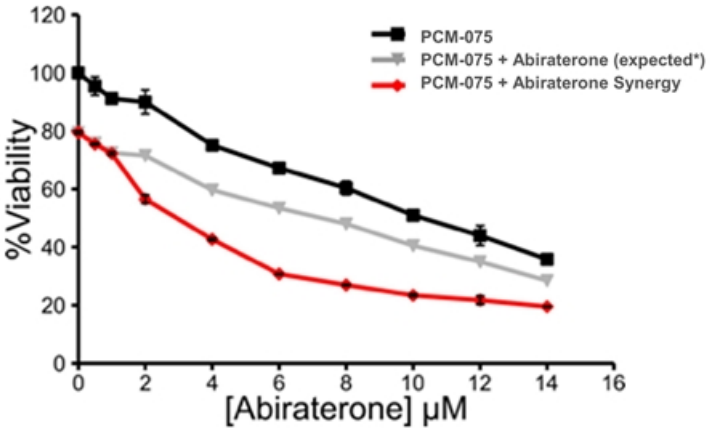
HDAC inhibitors are approved for NHLs, peripheral and cutaneous t-cell

*HDAC – Histone Deacetylases
[1] Steven Grant, MD, Virginia Commonwealth University, Massey Cancer Center; [2]Unpublished Research Data; [3]Data on File

Synergy: PCM-075 + Abiraterone (Zytiga®)
Metastatic Castration-Resistant Prostate Cancer

- PCM-075 in combination with abiraterone demonstrated synergy with decreased viability of mCRPC tumor cells[1]
- Combination appears to enhance the PCM-075 mechanism of action of arresting cells during mitosis[1]
- Medical need to extend the duration of response to anti-androgen therapeutics

C4-2 Castration-Resistant Prostate Cancer Cells Increased Sensitivity to Abiraterone in the Presence of PCM-075



*Expected = the calculated value of the effect of the addition of each drug as calculated by Michael Yaffe, MD - MIT

[1]Yaffe, Michael, MD and Trovagene, 2017

PCM-075 Clinical Trials

Phase 1b/2 in Acute Myeloid Leukemia (AML)

Phase 2 in metastatic Castration-Resistant Prostate Cancer (mCRPC)

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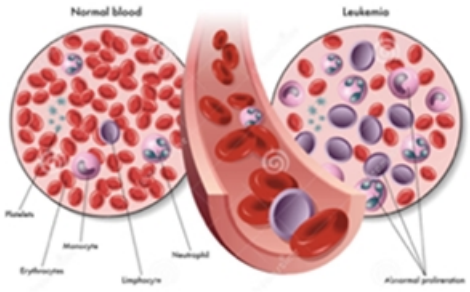
Acute Myeloid Leukemia[1]
Significant Need for New Treatment Options

- AML: aggressive hematologic malignancy of immature blood cells
- Incidence: 20,000* new cases and 10,400 deaths annually in the U.S.
- Prognosis: 5 year survival rate is 25%
- Treatment options vary based on patient condition / age, but can include:
  - Chemotherapy
  - Radiation
  - Stem cell transplant
- Genetically diverse landscape:
  - PLK1 selectivity presents opportunity across patient sub-populations

Acute Myeloid Leukemia



*Orphan Drug Designation granted by the FDA September 28, 2017; [1]National Cancer Institute SEER 2016

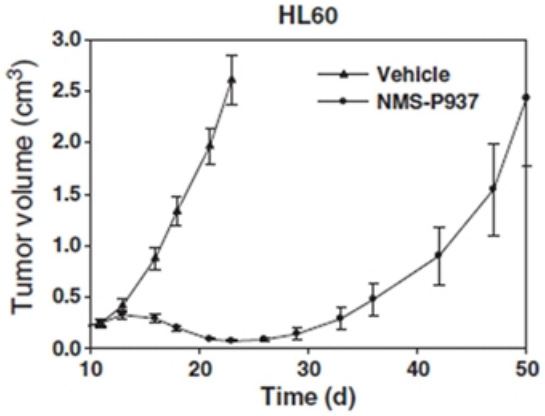
Scientific Rationale for PCM-075
Clinical Development in AML

- ***in-vitro* studies[1]**
  - High sensitivity of hematological tumor cell lines to PCM-075 (IC50 < 200nM in 38 out of 42 tested cell lines)
- ***in-vitro and in-vivo* mode of action (MoA) studies[2]**
  - Cell lines studies indicated G2/M arrest within cell cycle with dose dependent increase in 4N DNA after 24hrs
  - Xenograft model demonstrates dose dependent inhibition of PLK1 activity and G2/M arrest by pharmacodynamic biomarkers (pTCTP, pNPM, pHistone H3)
- ***in-vivo* efficacy in AML xenograft models[2]**
  - Dose dependent efficacy of PCM-075 in
    - HL60 promyelocytic leukemia xenograft
    - Disseminated AML patient derived xenografts (AML-PS)
  - Increased survival with PCM-075 vs cytarabine in disseminated AML patient xenograft (AML-NS8)
  - Combination of PCM-075 + cytarabine has greater survival than either agent alone (AML-PS)

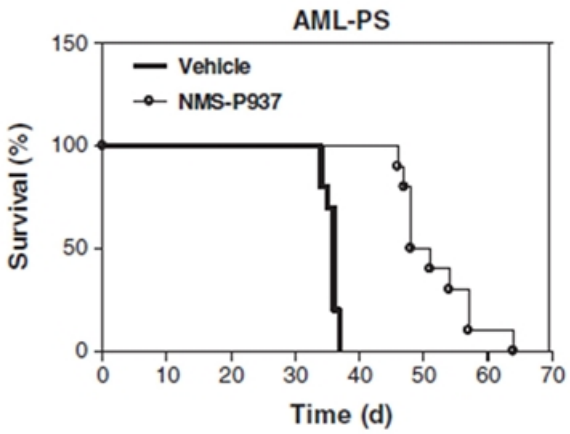
[1]Source: Report No. N-0018670 Antiproliferative activity of NMS-1286937 in a panel of cell lines;[2]Valsasina et al., Mol Cancer Ther; 11(4) April 2012; [3]ClinicalTrials.gov, NCT03303339: PCM-075 in Combination With Either Low-dose Cytarabine or Decitabine in Adult Patients With Acute Myeloid Leukemia (AML) - Data-on-file, Trovagene 2018

PCM-075: *in-vivo efficacy* in AML Models[1]

HL60 Promyelocytic Leukemia Xenograft



Disseminated AML Patient-Derived Xenografts (AML-PS

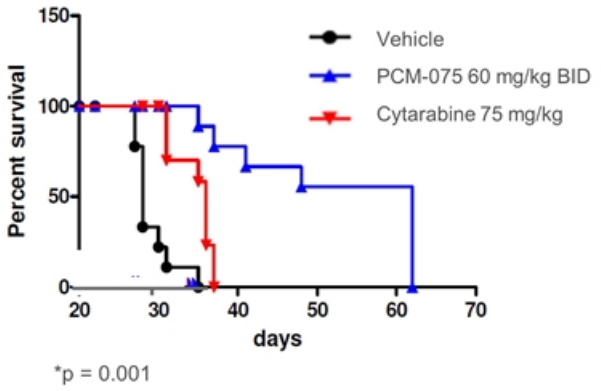


- PCM-075 is efficacious in subcutaneously implanted promyelocytic leukemia (HL60)
- PCM-075 is active in AML patient derived disseminated primary cells (AML-PS) and showed increased survival in comparison with vehicle

[1]Valsasina et al. (2012), Mol Cancer Ther 11(4)

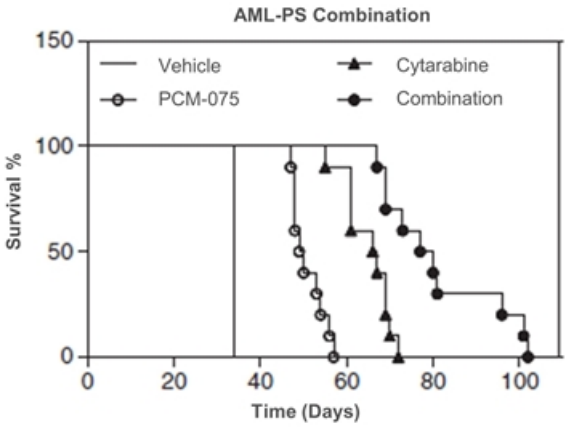
PCM-075: Comparative and Combination with Cytarabine in AML Models[1,2]

In Vivo Disseminated Leukemia Models



*p = 0.001

- PCM-075 60 mg/kg BID (Days 1-2 with 5-day rest) + cytarabine 75 mg/kg IP Injection (Days 1-5 with 5-day rest)
- PCM-075 120 mg/kg for 2 days repeated for 4 cycles with a 10-day rest

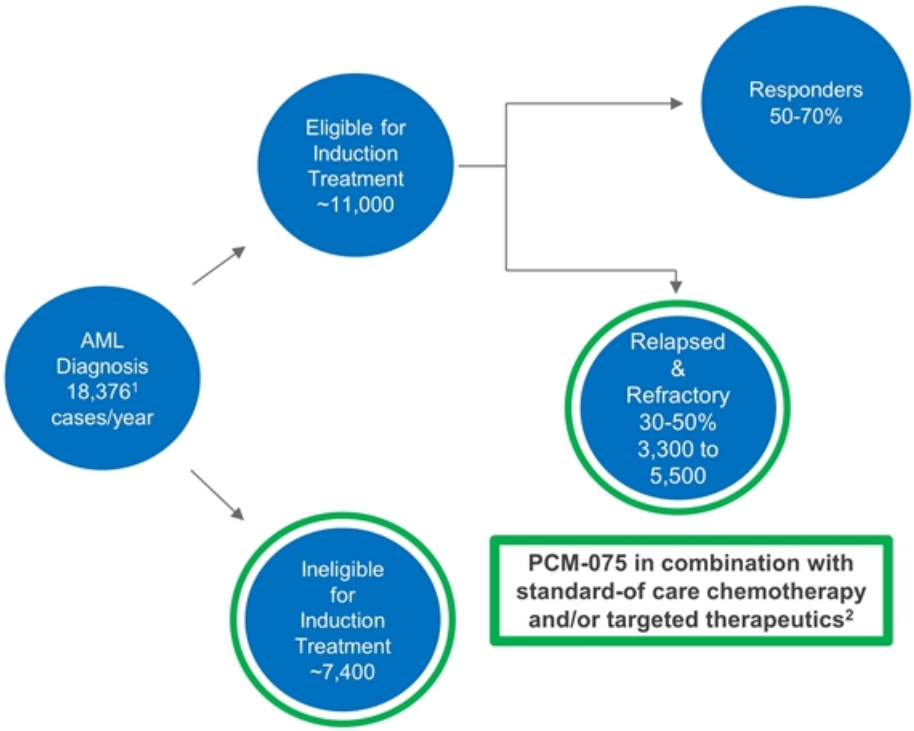


- PCM-075 120 mg/kg for 2 days repeated for 4 cycles with a 10-day rest
- Cytarabine IP at 75mg/kg for 5 cycles of 5 consecutive days with 7-day rest
- The combination was given at the same schedule, doses, and routes of the single agents

- PCM-075 plus cytarabine in combination showed increased survival compared to either agent alone

[1]Casolaro et al. (2013) PLOS One 8(3); [2]Valsasina et al. (2012), Mol Cancer Ther 11(4)

PCM-075: Positioning in AML
Patient Selection Algorithm



- **Positioning of PCM-075:**
  - Targets patient populations in areas of greatest need for new treatment options
  - Prior PLK inhibitors demonstrated efficacy and clinical benefit in these patient populations
  - PCM-075 product profile includes attributes for improved safety and efficacy

[1]Visser et al. (2012), Eur J Cancer (48). Estimated cases in EU27 per year; [2]e.g. Midostaurin for FLT3 mutation

Ongoing Phase 1b/2 Clinical Trial in AML

PCM-075 in Combination with Either Low-Dose Cytarabine (LDAC) or Decitabine in Patients with Acute Myeloid Leukemia (AML)

Study Design: Dose escalation to assess safety and identify recommended Phase 2 dose

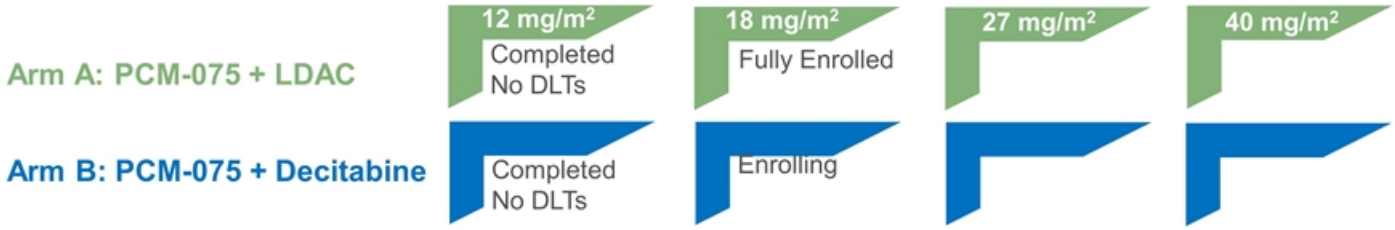
Patient Population: Patients who are Ineligible for induction therapy or have relapsed/refractory disease

Treatment Cycle: PCM-075 administered orally on Days 1-5, in a flexible 21-28 day cycle

Safety Endpoints:

- Assess side effects and tolerability to identify the maximum tolerated dose (MTD) or recommended Phase 2 dose (RP2D) for Phase 2
- Determine whether one combination regimen confers greater benefit and/or if certain patients respond best to one regimen (perhaps based on prior treatment)

Exploratory Endpoints: Evaluate pharmacodynamics and correlative biomarkers



Ongoing Phase 1b/2 Clinical Trial in AML

PCM-075 in Combination with Either Low-Dose Cytarabine (LDAC) or Decitabine in Patients with Acute Myeloid Leukemia (AML)

Phase 2: Assess safety and preliminary antitumor activity

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

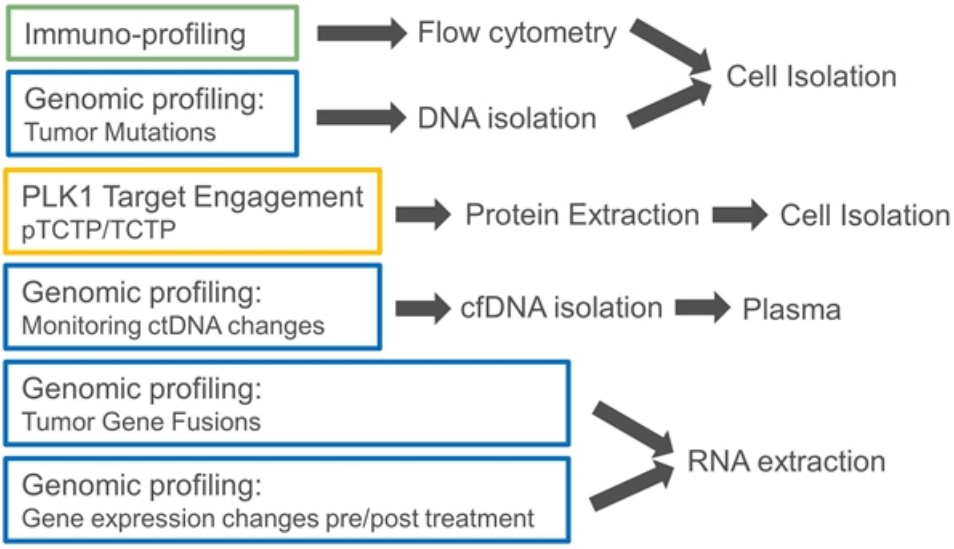
- **Recommended Phase 2 dose (RP2D) identified in Phase 1b**
  - Patients will be treatment naïve or have received no more than one prior regimen

CR:
- Absolute neutrophil count (ANC) of >1000/mm^3
- Platelets of ≥100,000/mm^3
- Patient is independent of transfusions

CRi:
- Meets all criteria for CR except for either neutropenia (ANC <1000/mm^3) or thrombocytopenia (<100,000/mm^3)
- Patient is independent of transfusions

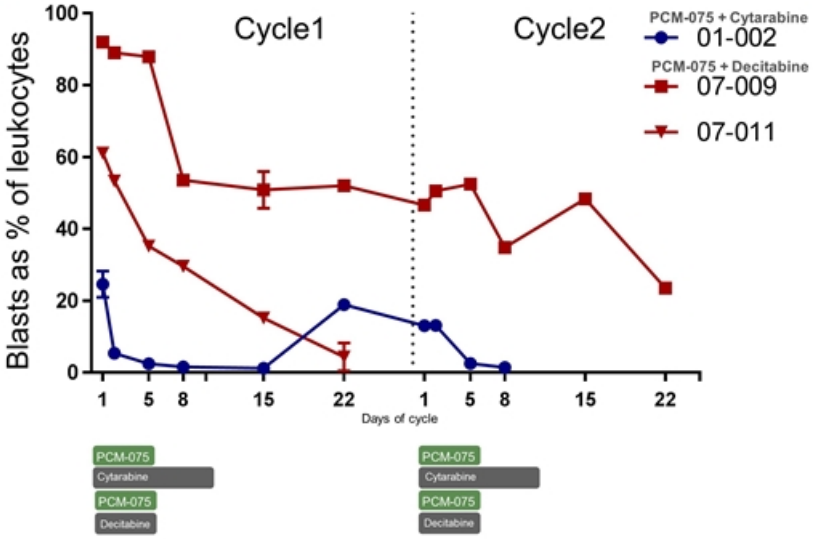
Biomarker Strategy in AML

- Biomarkers will be measured and correlated with pharmacokinetic drug levels to assess:
  - Inhibition of PLK1 by PCM-075 (Target Engagement)
  - Monitoring treatment effects by measuring % blast cells in blood and bone marrow
  - Correlating underlying tumor genetics with treatment response

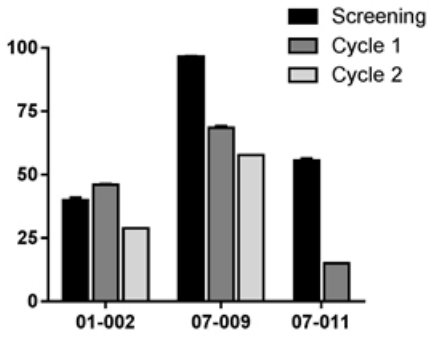


Immuno-Profiling:
Monitoring Leukemic Cells in Response to Treatment

% of Leukemic Cells in Blood



% of Leukemic Cells in Bone Marrow

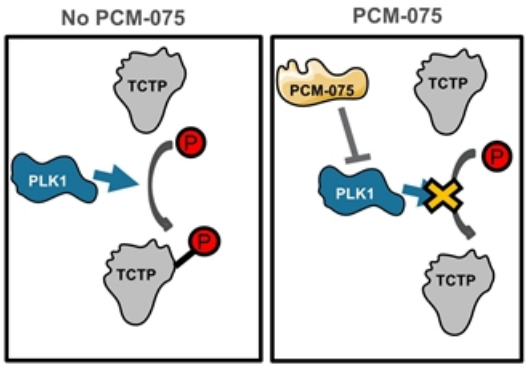


- 3 of 6 patients treated with PCM-075 in combination with low-dose cytarabine (LDAC) or decitabine showed decreases in the percentage of blood leukemic cells
- Bone marrow analysis also showed a decrease in the percentage of leukemic cells for these 3 patients; with 2 patients (07-009 and 07-011) having decreases from 96% to 55% and 55% to 15%, respectively

[1]NCT03303339, ClinicalTrials.gov; "PCM-075 in Combination With Either Low-dose Cytarabine or Decitabine in Adult Patients With Acute Myeloid Leukemia (AML)

Target Engagement:
Monitoring PLK1 Inhibition Upon Treatment

The Translational Control Tumor Protein (TCTP) Identified as Specific Marker for PLK1 Activity In-Vivo[1]

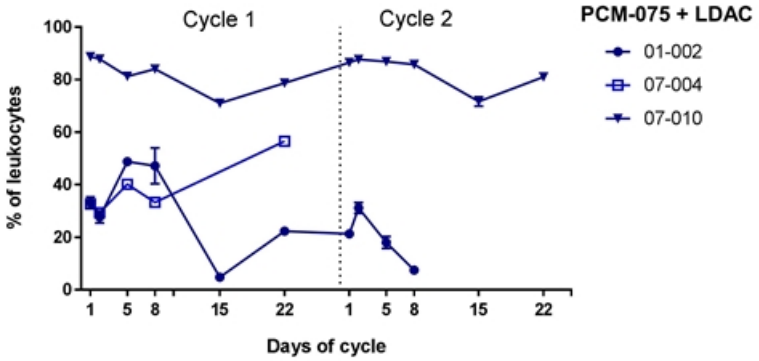


- PCM-075 inhibits PLK1 kinase activity resulting in reduction in PLK1 substrates phosphorylation; Translational Control Tumor Protein (TCTP) is phosphorylated by PLK1
- Inhibition of PLK1 by PCM-075 induces decreases in phosphorylated TCTP (pTCTP) in AML cell lines; pTCTP levels were unaffected by treatment with either cytarabine or decitabine
- PLK1 inhibition was assessed 3-hours following administration of PCM-075 at peak concentration (C_{max})
- PLK1 inhibition, as measured by pTCTP, was observed in 4 of the initial 6 patients treated
- Patients with the greatest target engagement of PCM-075 with PLK1 also had the greatest treatment effect

[1]Cusshi U. et al, Phosphorylation of TCTP as a Marker for Polo-like Kinase 1 Activity In Vivo – Anticancer Research December 2010 vol. 30 no. 12 pp. 4973-4985

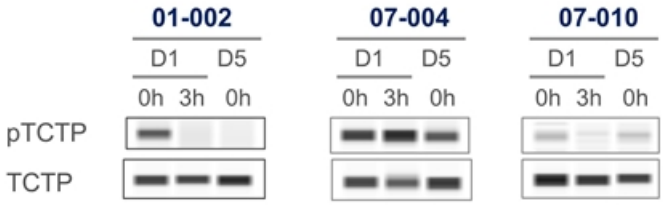
Correlation of Target Engagement and Treatment Response

% of Leukemic Cells in Blood

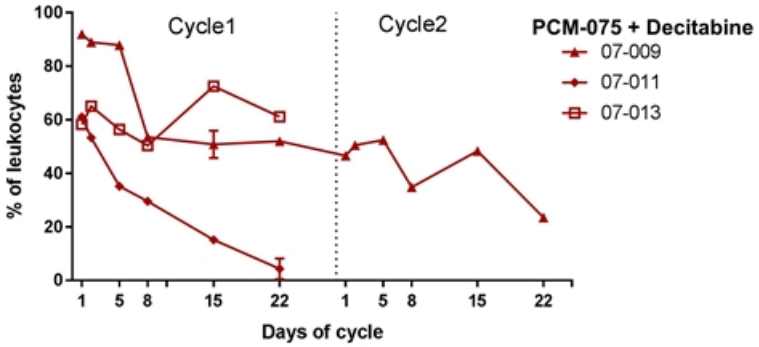


pTCTP status as a surrogate for PLK1 inhibition

PCM-075 12mg/m2 + LDAC



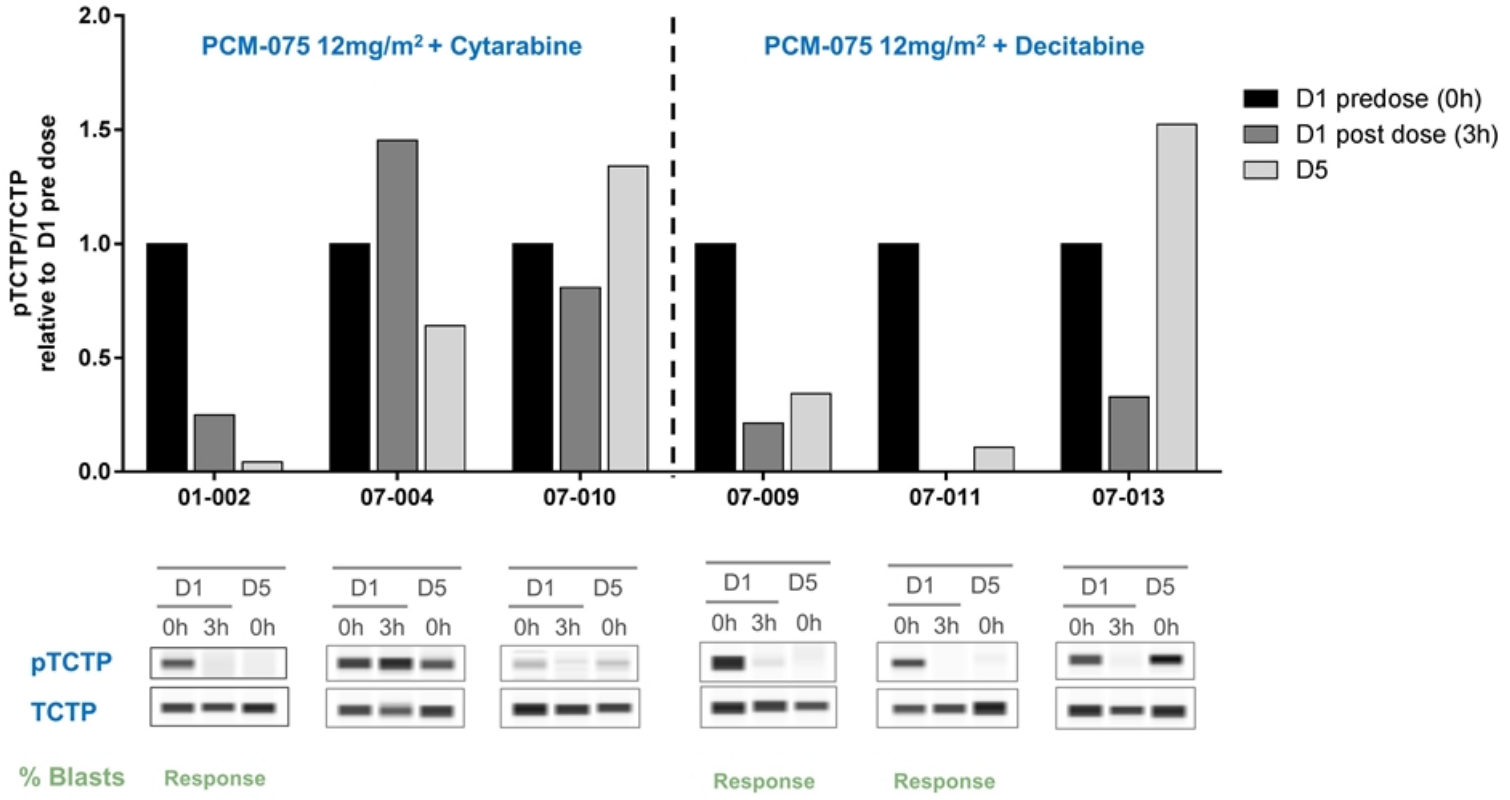
% of Leukemic Cells in Blood



pTCTP status as a surrogate for PLK1 inhibition

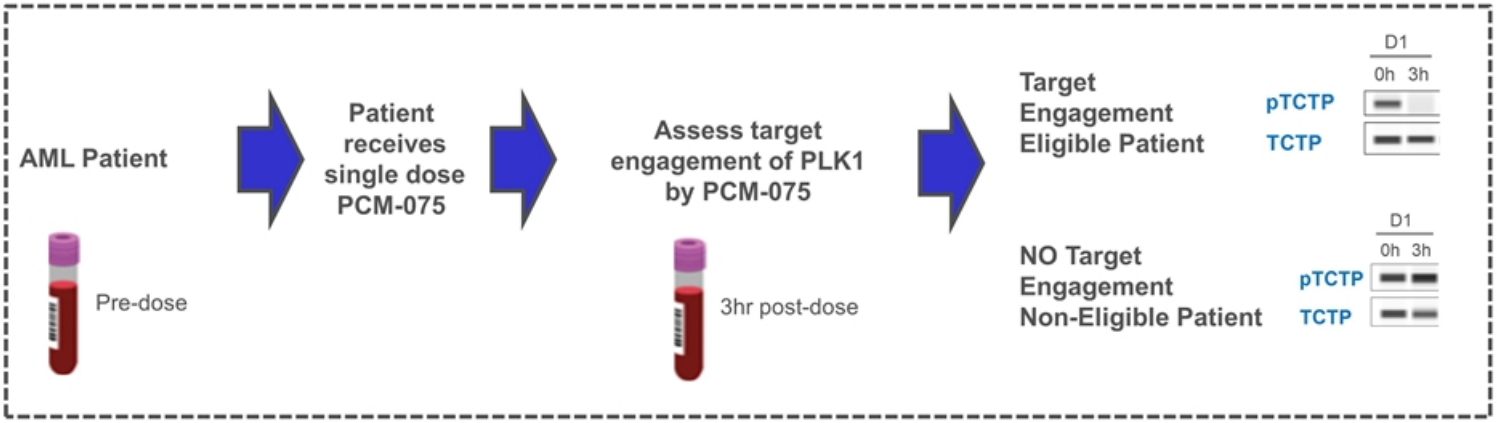
PCM-075 12mg/m2 + Decitabine

Summary of Target Engagement and Correlation to Treatment Response



Predictive Response Strategy

Determining which Patients are Eligible for AML Trial



Molecular Profiling and Patient Response in AML[1]

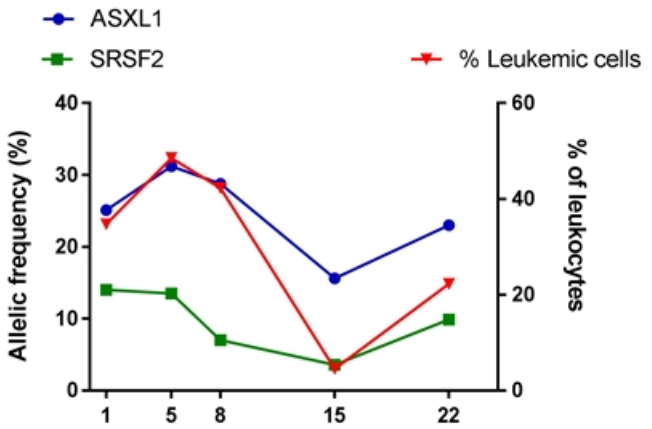
AML Genomic Subgroup	Frequency of Patients	Most Frequently Mutated Genes (%)	DNA Panel	RNA Panel
NPM1 mutation	27%	NPM1(100), DNMT3A(54), FLT3(39), NRAS(19), TET2(16), PTPN11(15)	X	
Mutated chromatin, RNA-splicing genes, or both	18%	RUNX1(39), MLLPTD(25), SRSF2(22), DNMT3A(20), ASXL1(17), STAG2(16), NRAS(16),TET2(15),FLT3ITD(15)	X	
TP53mutations, chromosomal aneuploidy, or both	13%	Complex karyotope(68), -5/5q(47), -7/7q(44), TP53(44), -17/17p(31), +8/8q(16)	X	X
inv(16)(p13.1q22) or t(16;16)(p13.1;q22);CBFB-MYH11	5%	inv(16) (100), NRAS(53), +8/8q(16), KIT(15), FLT3TKD(15)	X	X
biallelic CEBPA mutations	4%	CEBPAbiallelic(100), NRAS(30), WT1(21), GATA2(20)	X	
t(15;17)(q22;q12); PML-RARA	4%	t(15;17) (100), FLT3 ITD(35), WT1(17)	X	X
t(8;21)(q22;q22); RUNX1-RUNX1T1	4%	t(8;21) (100), KIT(38), -Y(33), -9q(18)	X	X
MLL fusion genes; t(x;11)(x;q23)	3%	t(x;11q23) (100), NRAS(23)	X	X
inv(3)(q21q26.2) or t(3;3)(q21;q26.2); GATA2,MECOM(EVI1)	1%	inv(3) (100), -7(85), KRAS(30), NRAS(30), PTPN11(30), ETV6(15), PHF6(15), SF3B1(15)	X	X
IDH2R172 mutations and no other class-defining lesions	1%	IDH2R172(100), DNMT3A(67), +8/8q(17)	X	
t(6;9)(p23;q34); DEK-NUP214	1%	t(6;9) (100), FLT3ITD(80), KRAS(20)	X	X

[1]Papaemmanuil et al. Genomic classification and prognosis in acute myeloid leukemia; NEJM 2016;374:2209-2221

Genomic Profiling: Correlation of Mutation Detected in Blood and % Leukemic Cells

Patient	Mutations detected
01-002	ASXL1 c.1926_1927insG p.G646fs*12 SRSF2 c.284C>G p.P95R
07-004	TP53 c.955 A>T p.Lys319Ter
07-010	SRSF2 c.284C>G p.Pro95Arg RUNX1 c.511G>A p.Asp171Asn RUNX1 c.250A>C p.Thr84Pro TET2 c.3633T>A p.Cys1211Ter
07-009	SF3B1 c.1998G>T p.Lys666Asn FLT3 c.250G>T p.Asp835Tyr RUNX1 c.984_985delAG p.Ala329fs GATA2 c.829A>G p.Ser277Gly
07-011	TP53 c.773A>C p.Glu258Ala
07-013	PHF6 c.955C>T p.Arg319Ter GATA2 c.962T>C p.Leu321Pro

Mutations allelic frequencies and leukemic cells level in blood samples of patient 01-002

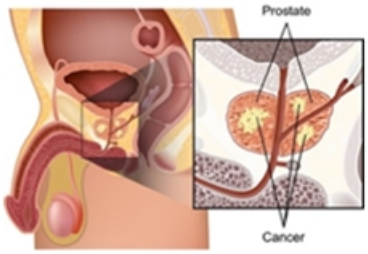


- Genomic analysis was performed on bone marrow and blood samples
- Mutations detected in bone marrow and blood were identical for all patients examined
- The mutation allelic frequencies detected in blood correlates with % of circulating leukemic cells

Metastatic Castration-Resistant Prostate Cancer

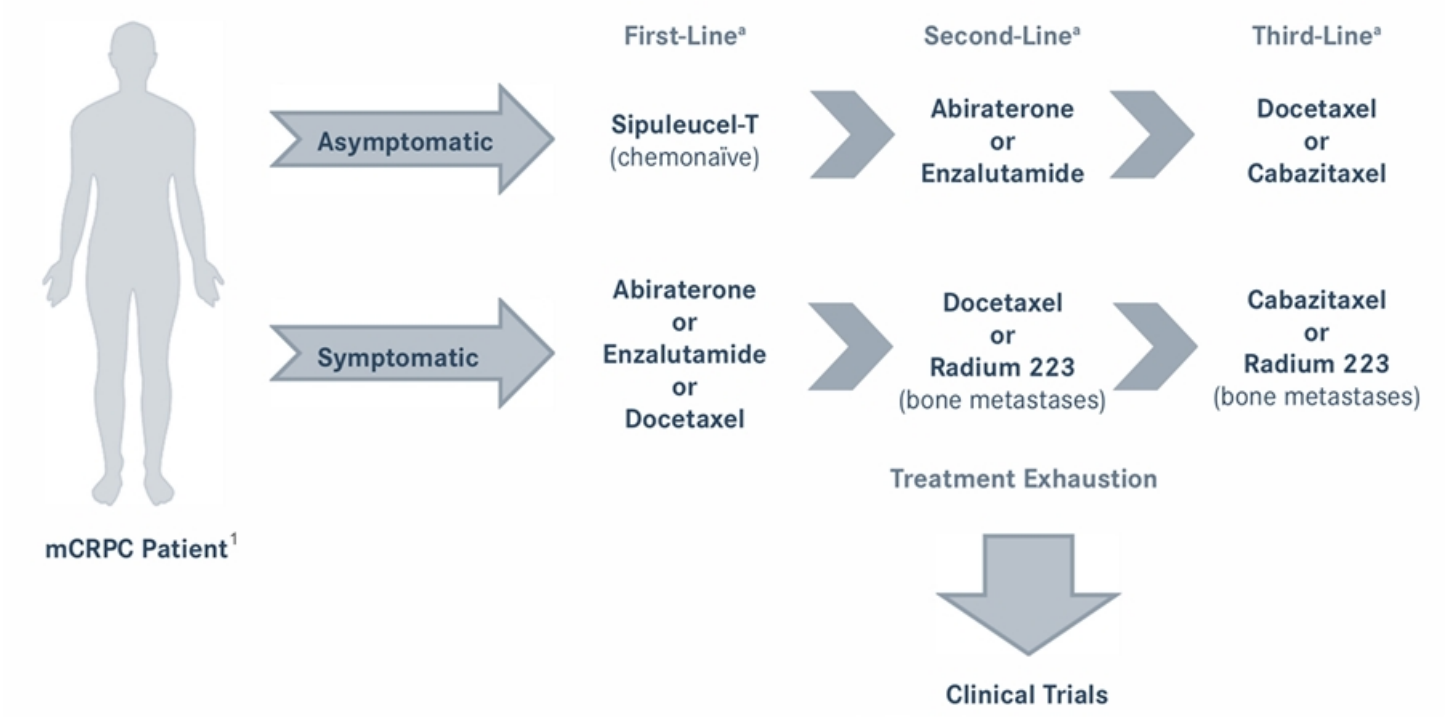
- 25,000 men progress to metastatic prostate cancer resistant to standard androgen-deprivation therapy, anually[1]
  - Five-year survival rate of 37%[2]
  - Risk of metastases increases as the disease progresses; most common metastases are adrenal gland, bone, and lung[3]
- Treatments
  - Abiraterone acetate (Zytiga® – Johnson & Johnson) and prednisone
  - Enzalutamide (Xtandi® – Astellas/Pfizer)
  - Docetaxel (Docefrez, Taxotere) and prednisone
- Ongoing need to increase duration of response for mCRPC patients
  - Patients develop resistance to abiraterone and enzalutamide (within 9-15 months)[4] and do not respond well to subsequent therapies

Prostate Cancer



[1]2017 Annual Report on Prostate Disease – Harvard Health Publications; [2]GlobalData. Prostate Cancer—Global Drug Forecast and Market Analysis to 2023. Apr, 2015; [3] National Cancer Institute Metastatic cancer. Mar, 2013. Available at: http://www.cancer.gov/about-cancer/what-is-cancer/metastatic-fact-sheet; [4]GAntonarakis, Emmannel – Current Understanding of Resistance to Abiraterone and Enzalutamide in Advanced Prostate Cancer; Clinical Advances in Hematology & Oncology – May 2016 – Volume 14, Issue 5

PCM-075 + Abiraterone Combination
Opportunity to Extend Response to Abiraterone



[1]Yadi, Li et al; An Investigative Approach for Sequencing Therapies in Metastatic Prostate Cancer - AJHO. 2017;13(12):26-31
[A] All treatment options should include androgen deprivation therapy (surgical/medical orchiectomy)

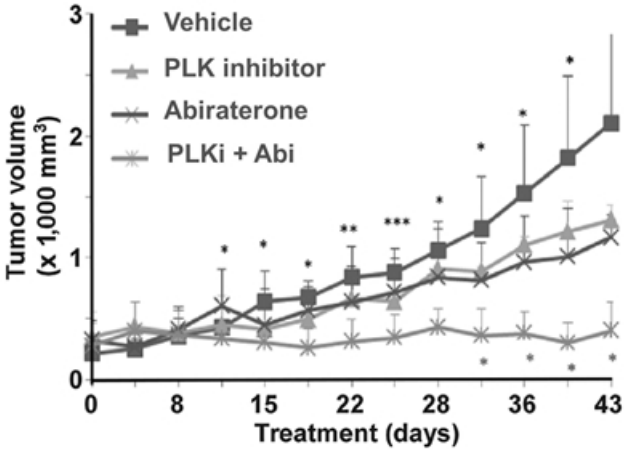
PLK1, Abiraterone and Castration-Resistant Prostate Cancer

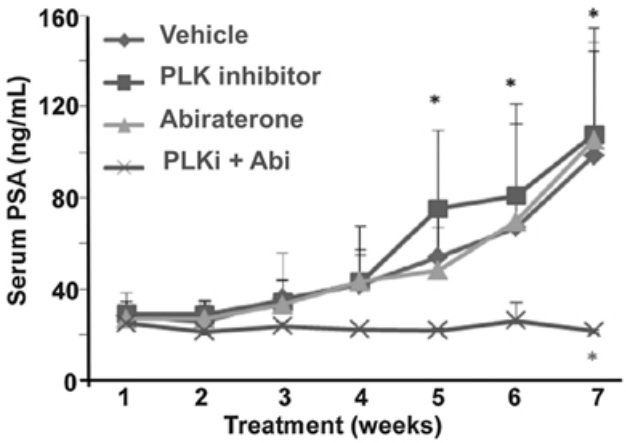
- All metastatic prostate cancer patients become castration-resistant
- PLK1 dependent microtubule dynamics promotes androgen receptor (AR) signaling in prostate cancer[1,2]
- PLK1 inhibition improves abiraterone acetate efficacy[3]
- Inhibition of PLK1 represses androgen signaling pathway in castration resistant prostate cancer[4]
- PLK1 inhibitors are anticipated to add important therapeutic benefit for the treatment of castrate resistant prostate cancer patients[5]

[1]Xianzeng, Hou, Zhiguo, Li – PLK1-Dependent Microtubule Dynamics Promotes Androgen Receptor Signaling in Prostate Cancer; Prostate. 2013 September; 73(12): 1352–1363. doi:10.1002/pros.22683; [2] Arpaporn, Deeraksa, Jing, Pan - Plk1 is upregulated in androgen-insensitive prostate cancer cells and its inhibition leads to necroptosis; Oncogene. 2013 June 13; 32(24): 2973–2983. doi:10.1038/onc.2012.309; [3]Clemens, Thoma – Prostate Cancer: PLK-1 Inhibition Improves Abiraterone Efficacy; Nature Reviews Urology volume11, page603 (2014); [4]Zhang Z1, Chen L – Inhibition of PLK1 Represses Androgen Signaling Pathway in Castration-Resistant Prostate Cancer; Cell Cycle. 2015;14(13):2142-8. doi: 10.1080/15384101.2015.1041689; [5]Klaus, Strebhardt - Drugging Plk1: An attractive approach to inhibit androgen receptor signaling; Cell Cycle. 2015 Jul 18; 14(14): 2193–2194

PSA Tracks Tumor Response in mCRPC[1]

Patient-Derived Xenograft (PDX) Model of CRPC[1] in Abiraterone-Resistant Cell Line





- Tumor volume was significantly reduced in mice treated with the combination of PLK inhibitor and abiraterone versus either drug alone
- Serum levels of PSA correlate with efficacy for the combination versus each drug alone

[1]Zhang et al, Cancer Res 2014; 74(22)

Ongoing Phase 2 Clinical Trial in mCRPC

PCM-075 in Combination with Zytiga® (abiraterone acetate) and Prednisone in Patients with Metastatic Castration-Resistant Prostate Cancer (mCRPC)

Dosing Regimen	Duration	Evaluation
PCM-075 – 24 mg/m^2 Days 1-5 (21-Day Cycle) + Zytiga®/prednisone daily	4 Cycles = 12 Weeks	Disease Control based on PSA level

Patient Population: Patients on abiraterone showing early signs of disease progression

Treatment Cycle: PCM-075 administered orally on Days 1-5 in a 21-day cycle

Efficacy Endpoints: Effect of PCM-075 in combination with Zytiga®/prednisone on disease control assessed by prostate-specific antigen (PSA) decline or stabilization pre- and post-treatment

Safety Endpoint: Safety of PCM-075 in combination with Zytiga®/prednisone

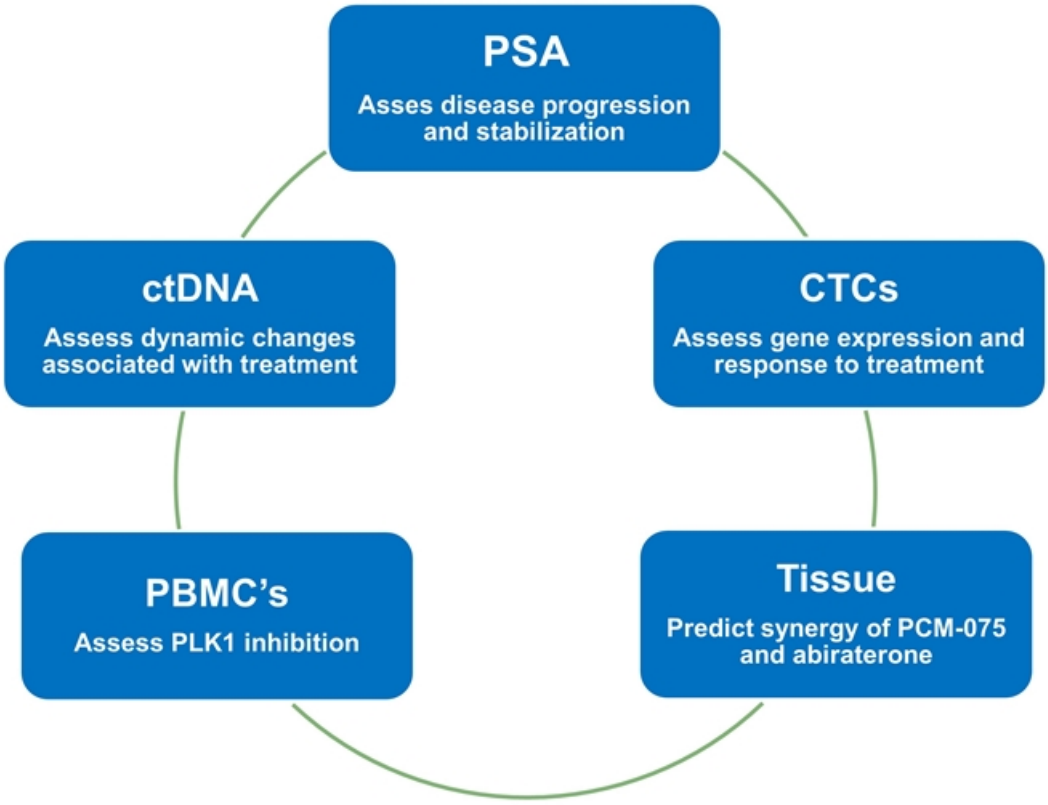
Exploratory Endpoint: Target inhibition of PLK1, evaluation of relevant biomarkers and correlation with patient response and genomic profile

PSA: NCCN Recommended Biomarker
Trial Eligibility and Efficacy for mCRPC[1]

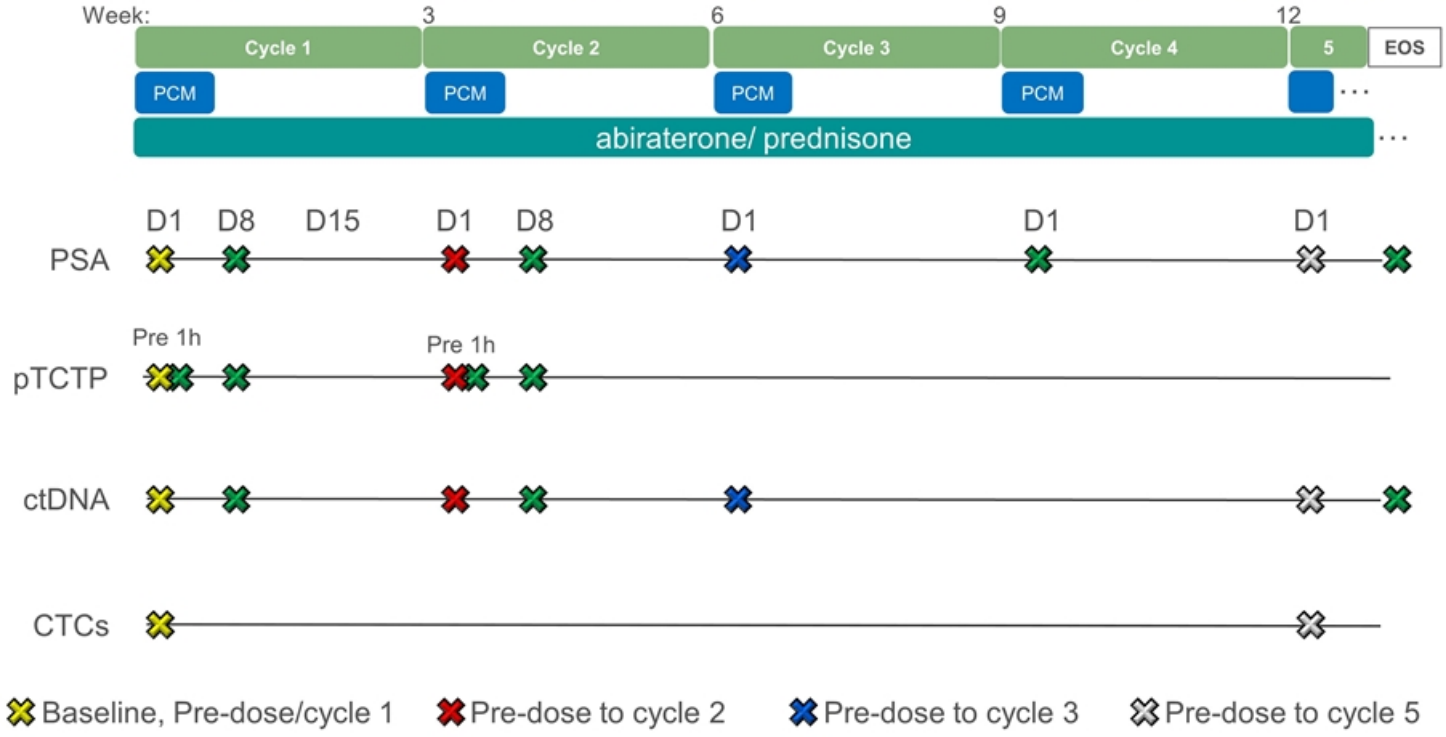
- Prostate Specific Antigen (PSA) is a validated biomarker that shows breakthrough in treatment
- Prostate Cancer Clinical Trials Working Group (PCWG)[1] has set criteria for the use of blood PSA levels for both trial eligibility (progression) and initial assessment of efficacy
  - Trial Eligibility (defining progression)
    - Obtain sequence of two rising PSA values separated by at least 1 week
  - Initial Assessment of Efficacy
    - Initial efficacy endpoint = percent of patients with disease control as defined as a lack of PSA progression at 12 weeks
    - PCWG2/3 defines PSA progression as the date that an increase of ≥25% and absolute increase of 2 ng/mL or more from the baseline/nadir[1]

[1]PCWG2: Sher et al, JCO, 2008, PCWG3: Sher et al, JCO, 2016

Biomarker Strategy in mCRPC



Biomarker Assessment Schedule



- Primary Endpoint: Proportion of patients achieving disease control after 12 weeks of study treatment, as defined by lack of PSA progression

Summary

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Milestones
Significant Value Creation in 2018

	Q1 18	Q2 18	Q3 18	Q4 18
Leukemias & Lymphomas	AML Phase 1bTrial Enrolling ✓	AACR Data Presentation ✓	Successful completion of 1st dosing cohort in AML trial (Arm B) ✓	ASH Data Presentation
	First Patient Successfully Completes Cycle 1 in AML Trial ✓	Successful completion of 1st dosing cohort in AML trial (Arm A) ✓	Successful completion of 2nd dosing cohort in AML trial	MTD / RP2D Identified for AML Phase 2 Trial
		Preliminary clinical data from 1st dosing cohort demonstrating durable treatment effect ✓	Initiation of 3rd dosing cohort in AML trial	Topline AML safety and efficacy data
Solid Tumor Cancers	mCRPC Phase 2 Trial Clears FDA Review Window ✓	mCRPC Phase 2 trial sites activated and recruiting ✓	3 safety lead-in patients enrolled	Continued active enrollment in mCRPC trial
	ASCO GU Data Presentation ✓		Safety and efficacy data on 3 lead-in patients	Continued efficacy data readouts

Summary

- Precision Medicine Approach
  - Developing PCM-075 by integrating a highly-selective antimitotic drug with a biomarker strategy
- Leveraging a proven cancer target
  - PLK1 is highly expressed in tumor cells and integral to mitosis
- PCM-075 – first-in-class, 3rd generation PLK1 inhibitor
  - Benefiting from prior drug class clinical experience, including efficacy, safety and single vs combination therapy trial design
- Synergy strategy
  - Combining PCM-075 with already approved drugs that have demonstrated synergy in combination
- Actively pursuing partnering opportunities with Japanese companies

For additional information or questions please contact:
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