Trovagene, Inc.
Form FWP
June 04, 2018

Issuer Free Writing Prospectus dated June 4, 2018 Relating to Prospectus dated June 4, 2018 Filed Pursuant to Rule 433 Registration Statement No. 333-224808 Transforming Patient Care with Targeted Cancer Therapeutics june, 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.

Free Writing Prospectus This presentation highlights basic information about Trovagene, Inc. and the offering. Trovagene, Inc. has filed a registration statement on Form S-1 (Registration No. 333-224808) (including a prospectus) with the U.S. Securities and Exchange Commission ("SEC") for the offering to which this presentation relates. The registration statement has not yet become effective. Before you invest, you should read the prospectus in the registration statement (including among other things, the risk factors described therein) and other documents the issuer has filed with the SEC for more complete information about Trovagene, Inc. and this offering. The preliminary prospectus dated June 4, 2018 and subsequent amendments are available at the SEC website. You may get these documents for free by visiting EDGAR on the SEC website at www.sec.gov. Alternatively, Trovagene, Inc. or any underwriter or any dealer participating in the offering will arrange to send you the prospectus if you request it by contacting ThinkEquity, a division of Fordham Financial Management, Inc., 17 State Street, 22nd Floor, New York, New York 10004, via email at prospectus@think-equity.com or via telephone at (646) 968-9355. This presentation contains statistics and other data that has been obtained from or compiled from information made available by third parties service providers. Trovagene, Inc. has not independently verified such statistics or data. The information contained in this presentation is as of June 4, 2018, unless indicated otherwise.

Issuer Trovagene, Inc. Listing / Symbol NASDAQ: TROV Expected Offering Size Approximately $15,000,000 (100% Primary) Over-Allotment Option 15% (100% Primary) Securities Offered Class A Unit of Common Stock and Warrants Class B Unit, for 4.99% or greater shareholders, of Preferred Stock and Warrants Use of Proceeds Fund research and development activities, working capital and general corporate purposes Sole Book-Runner ThinkEquity, a division of Fordham Financial Management, Inc. Public Offering Summary

Trovagene Snapshot Oncology Focus Clinical-stage therapeutics company developing therapies for hematologic and solid tumor cancers Lead drug candidate, PCM-075, a highly-selective Polo-like Kinase 1 (PLK1) inhibitor Phase 1 safety trial in advanced/metastatic solid tumor cancers published in 2017 Two Investigational New Drug (IND) applications in place to develop PCM-075 for hematologic and solid tumor cancers Phase 1b/2 trial in Acute Myeloid Leukemia (AML) ongoing Orphan Drug Status received from FDA September, 2017 Phase 2 trial in metastatic Castration-Resistant Prostate Cancer (mCRPC), goal to activate in 2Q 2018 Preclinical data in other hematologic and solid tumor cancers, including: Non-Hodgkin Lymphoma Lung Cancer Triple Negative Breast

Key Advisors and Collaborators Jorge Cortes, MD – MD Anderson Cancer Center 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 Medical Oncologist, genitourinary cancers Sandra Silberman, MD – Duke VA Hematologist, former VP and Global Head of Translational Medicine at Quintiles Filip Janku, MD, PhD – MD Anderson Cancer Center Associate Professor, Investigational Cancer Therapeutics (Phase 1 Clinical Trials Program) Michael Yaffe, MD, PhD – MIT Director, Clinical Research at Koch Institute for Integrative Cancer Research Confidential

PCM-075 Pipeline Assets Indication (Target) Preclinical Phase 1 Phase 2 Non-Hodgkin Lymphoma Phase 1 Advanced or Metastatic Solid Tumors Published in Investigational New Drugs Metastatic Castration-Resistant Prostate Phase 2 open-label clinical trial in combination with abiraterone acetate (Zytiga®)/prednisone planned for initiation Lung Cancer Triple Negative Breast Acute Myeloid Leukemia – Orphan Drug Designation Phase 1b/2 open-label clinical trial in combination with low-dose cytarabine or decitabine ongoing Completed Ongoing Planned Preclinical

PCM-075 – Polo-like Kinase 1 (PLK1) Inhibitor Lead Drug Candidate

PLK1 – Established Cancer Target PLK1 belongs to a family of kinases (PLK1,2,3,4 and 5) PLK1 is the master regulator of the cell-cycle and only with expression of PLK1 are cells able to pass-through the last checkpoint (G2Mitosis) and divide1-6 PLK2-PLK5 have properties more consistent with tumor suppressor genes and are not essential for cell division7 Over-expression of PLK1 is observed in numerous cancer types and associated with poor patient prognosis8 Depletion of PLK1 induces cell death in tumor cells9 1Takai N, et al. Oncogene 2005;24:287–91; 2Rudolph D, et al. Clin Cancer Res 2009;15:3094–102; 3Chopra P, et al. Expert Opin Investig Drugs 2010;10:27–43; 4Strebhardt K. Nat Rev Drug Discov 2010;9643–60; 5Zitouni S, et al. Nat Rev Mol Cell Biol 2014;15:433–52; 6Takaki T, et al. Curr Opin Cell Bio 2008;20:650–60; 7Bahasa EM. Polo-like kinases and DNA damage checkpoint: beyond the traditional mitotic functions. ExpBiol and Medicine 2011; 236: 648-657; 8Data derived from The Tumor Genome Atlas, http://togadata.nci.nih.gov/docs/publications/tcga;,9Liu et al., Mol. Cell. Biol. March 15, 2006; 26:6 2093-2108
PLK1 – Last Checkpoint of Cell Cycle

PLK1 – Overexpressed in Numerous Cancers “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 PLK1 Inhibitors in Cancer Therapy: From Laboratory to Clinics 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 Over-Expression AML 13.0 B-cell Lymphoma 56.3 Adrenocortical 4.5 Lung Adeno 10.5 Lung Squamous 20.9 Breast 14.4 Esophageal 7.8 Stomach 2.2 Colon 2.5 Head & Neck 3.1 Skin Melanoma 18.3 Ovarian 31.7

Clinical Development Program Hematologic and Solid Tumor Cancers PCM-075 in combination with either low-dose cytarabine or decitabine Lead Investigator: Dr. Jorge Cortes – MD Anderson Study to include up to 10 clinical trial sites Phase 1b/2 Open-Label Trial in Acute Myeloid Leukemia (AML) - Ongoing Phase 2 Open-Label Trial in Metastatic Castration-Resistant Prostate Cancer (mCRPC) - Planned Hematologic Cancers Solid Tumor Cancers PCM-075 in combination with Zytiga (abiraterone acetate) / prednisone Lead Investigator: Dr. David Einstein – Beth Israel Deaconess Medical Center Study to include 3 Harvard Medical Cancer Centers

PCM-075 – Highly Selective PLK1 Inhibitor Tested against >260 kinases and PLK1 was the only active target (IC50 of 2nM) Selectivity driven by polar interaction with the carboxyl side chain of Glutamate 140 position of PLK11 1Data on File, Trovagene, Inc. Selective PLK1 Inhibitor Induces tumor cell death by G2M cell cycle arrest Molecular Weight : 648.60 Daltons AML-NS8 Patient-Derived Cells Treated with 200 nM PCM-075 for 24 Hrs1 PCM-075 DMSO PCM-075 Control Treatment of cells with PCM-075 resulted in a clear mitotic block accompanied by an increase of the G2/M population (4N DNA content)

PCM-075 – Oral Administration and Half Life 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) NerPharMa Inc. – contract manufacturer for GMP API and finished goods ADME Profile2 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 1Data on File, Trovagene, Inc.; 2ADME = Absorption, Distribution, Metabolism, Excretion

PCM 075 – Synergistic in Combination High PLK1 expression is associated with the most aggressive forms of hematologic and solid tumor cancers PCM-075's synergistic activity may enhance the efficacy of numerous standard-of-care therapies 1Alphabetical order. 2Preclinical data on file with PCM-075 and these combined therapeutics Potentially Synergistic Drugs1,2 Abiraterone acetate Bortezomib Cisplatin Cytarabine Doxorubicin FLT3 Inhibitors (Quizartinib) Gemcitabine HDAC Inhibitors (Belinostat) Paclitaxel Associated Cancers2 Hematologic Cancers: 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

Synergy: PCM-075 + FLT3 Inhibitor Acute Myeloid Leukemia (AML) 1Kindler et al, Blood 2010; 116:5089-10. 2Stone et al, N Engl J Med 2017; 377:454-64. 3Data on File at Trovagene, Inc. 30% of AML patients harbor a FLT3 mutation1 Midostaurin (FDA approved); 3 additional FLT3 inhibitors, including quizartinib, are currently in Phase 3 clinical development2 The combination of PCM-075 plus quizartinib demonstrated 97% tumor growth inhibition and regression in FLT3 AML xenograft model3 Evaluation of Efficacy of PCM-075 for MV-4-11 Human Acute Myeloid Leukemia (AML) Xenograft Model in NOD.SCID Mice

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-cell2 and T-cell lymphoma cell lines3 Medical need for improved duration of HDAC inhibitor response *HDAC – Histone Deacetylases 1 Steven Grant, MD, Virginia Commonwealth University, Massey Cancer Center; 2Unpublished Research Data; 3Data on File Cell line Double-hit Cell line Mantle Cell Control PCM-075 HDAC PCM-075 / HDAC HDAC inhibitors are approved for NHLs, peripheral and cutaneous t-cell PCM-075 Combined with HDAC Inhibitor Reduces Survival of Double-Hit (DLBCL) and Mantle Cell Lymphoma Cell Lines1

Synergy: PCM-075 + Abiraterone (Zytiga®) Metastatic Castration-Resistant Prostate Cancer (mCRPC) PCM-075 in combination with abiraterone demonstrated synergy with decreased viability of mCRPC tumor cells1 Combination appears to enhance the PCM-075 mechanism of action of arresting cells during mitosis1 Medical need to extend the duration of response to anti-androgen therapeutics 1Yaffe, Michael, MD and Trovagene, 2017 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 * PCM-075 PCM-075 + Abiraterone (expected*) PCM-075 + Abiraterone Synergy

PCM-075 Summary Characteristic Benefit PCM-075 Oral; 24-Hour Half-Life Enables sustaining drug levels without compromising safety Selective for PLK1 Provides highly-targeted therapy for G2/M cell-cycle division checkpoint Reversible, on-target side effects, consistent with the expected mechanism of action3 Synergistic in Combination1 Combination therapies potentially yielding “1+1=3” clinical activity Demonstrated synergy with (e.g., cytarabine, paclitaxel and abiraterone) Enhances mechanism of action without increasing on-target toxicities Tumor Cell Sensitivity Normal cells are 10-fold less sensitive than tumor cells to induced cell death2 No GI disorders, mucositis, or alopecia observed in Phase 1 solid tumor study Resistance Mechanism Ability to induce cell death in tumor cells that express transporters (e.g. MDR1)3 1Preclinical data on file with PCM-075 in combination with chemo and targeted therapeutics; 2Valsasina et al., Molecular Cancer Therapeutics; 11(4) April 2012; 3Investigator Brochure for PCM-075, 22 June 2017 – Data on File

PCM-075 Clinical Trials Phase 1 Advanced/Metastatic Solid Tumors Phase 1b/2 Acute Myeloid Leukemia (AML) Phase 2 metastatic Castration-Resistant Prostate Cancer (mCRPC)

Phase 1 Safety Trial1 Completed in Solid Tumors Phase 1 Trial Design PCM-075 Trial Results Included: colorectal, pancreatic, lung, sarcomas, hepatocellular, ampullary, prostate, ovarian, skin Established safety of PCM-075 and identified a recommended Phase 2 dose of 24 mg/m2/day 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 Reversible, on-target thrombocytopenia and neutropenia, consistent with the expected mechanism of action, were the primary adverse events 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 Phase 1 dose escalation trisl in patients with advanced or metastatic solid tumors 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 1Weiss 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

Acute Myeloid Leukemia1 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 *Orphan Drug Designation granted by the FDA September 28, 2017; 1National Cancer Institute SEER 2016 Acute Myeloid Leukemia

Pre-Clinical Data in AML Compound Median Survival Time (days) Placebo 28 Cytarabine 36 PCM-075 62* Treatment Started 20 Days Post-Inoculation 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) 1Casolaro et al (2013) PLOS One *p = 0.001 Vehicle PCM-075 60 mg/kg BID Cytarabine 75 mg/kg Vehicle PCM-075 60 mg/kg BID Cytarabine 75 mg/kg In Vivo Disseminated Leukemia Model (AML-NS8 Cells) Vehicle PCM-075 60 mg/kg BID Cytarabine 75 mg/kg Vehicle PCM-075 60 mg/kg BID Cytarabine 75 mg/kg

Previous PLKi Clinical Experience in AML Volasertib (Boehringer Ingelheim) Volasertib demonstrated clinical activity in AML Phase 2 and Phase 3 clinical trials Phase 2 trial: Increased response rate (31.0%) vs control (13.3%) and significant survival benefit in elderly patients ineligible for intensive induction therapy. Volasertib demonstrated response across all AML genetic subgroups and had a clinically manageable safety profile Phase 3 trial: Increased response rate (25.2%) vs control (16.8%) in elderly patients ineligible for intensive induction therapy. However, there was a negative OS trend due to higher incidence of severe adverse events (SAEs) with a fatal infection frequency (16.6% volasertib vs 5.1% control) Median dose density was higher in the Phase 3 than the Phase 2 trial. The dosing regimen was considered the main reason for an imbalance in adverse events as well as insufficient anti- infective prophylaxis1 PCM-075 anticipated advantages: Highly-selective PLK1 inhibitor suggesting a more favorable safety profile Five-fold shorter half-life that will allow greater dosing flexibility based on patient response Proactive management of potential infections via prophylactic administration of antibiotics 1Dohner, H., Symeonidis, A. - Phase III Randomized Trial of Volasertib Plus Low-Dose Cytarabine (Idac) Versus Placebo Plus Idac in Patients Aged ≥65 Years with Previously Untreated AML, Ineligible for Intensive Therapy; 21st Congress of the European Hematology Association Copenhagen, Denmark, June 9 - 12, 2016

PCM-075 Highly Selective Short Half-Life Synergistic in Combination Broad Applications Orally Available

Treatment Pathway in Acute Myeloid Leukemia AML Diagnosis 20,0001 cases/year Eligible for Induction Treatment 12,000 Relapse & Refractory 30-50% 4,000 to 6,000 Phase 1b/2 AML Trial: To assess the safety and efficacy of PCM-075 in combination with cytarabine or decitabine Responders 50-70% Ineligible for Induction Treatment ~8,000 Consolidation Treatment 1National Cancer Institute SEER 2016

Ongoing Phase 1b/2 Clinical Trial in AML Current Clinical Trial Sites MD Anderson Yale Cancer Center University of California Los Angeles Kansas University Cancer Center PCM-075 in Combination with Either Low-Dose Cytarabine (LDAC) or Decitabine in Patients with Acute Myeloid Leukemia (AML) Seattle Cancer Center Virginia Cancer Specialists University of Texas Southwestern Virginia Piper Cancer Institute Phase 1b: Dose escalation to assess safety and identify recommended Phase 2 dose Exploratory Endpoints: Evaluation of pharmacodynamic and correlative biomarkers Arm A: PCM-075 + LDAC Arm B: PCM-075 + Decitabine 12 mg/m2 Completed Fully Enrolled Enrolling 18 mg/m2 27 mg/m2 40 mg/m2

Ongoing Phase 1b/2 Clinical Trial in 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 PCM-075 in Combination with Either Low-Dose Cytarabine (LDAC) or Decitabine in Patients with Acute Myeloid Leukemia (AML) 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/mm3 Platelets of ≥100,000/mm3 Patient is independent of transfusions CRi: Meets all criteria for CR except for either neutropenia (ANC <1000/mm3) or thrombocytopenia (<100,000/mm3) Patient is independent of transfusions PCM-075 in combination with either LDAC or Decitabine RP2D 32 patients

Biomarker Assessment in AML Biomarkers will be measured and correlated with pharmacokinetic drug levels to assess: Inhibition of PLK1 Changes in blast cells in blood and bone marrow Underlying tumor genetics Blood sample (baseline) AML Patient + PCM-075 Blood sample (post PCM-075) Pre- and Post-Dose Blood Samples Obtained

Molecular Profiling and Patient Response in AML1 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 1Papaemmanuil et al. Genomic classification and prognosis in acute myeloid leukemia; NEJM 2016;374:2209-2221

Metastatic Castration-Resistant Prostate Cancer 25,000 men progress to metastatic prostate cancer resistant to standard androgen-deprivation therapy, anually1 Five-year survival rate of 37%2 Risk of metastases increases as the disease progresses; most common metastases are adrenal gland, bone, and lung3 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 12017 Annual Report on Prostate Disease – Harvard Health Publications; 2GlobalData. 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; 4GAntonarakis, Emmannel – Current Understanding of Resistance to Abiraterone and Enzalutamide in Advanced Prostate Cancer; Clinical Advances in Hematology & Oncology – May 2016 – Volume 14, Issue 5 Prostate Cancer

PLK1 Sensitivity of AR-Driven Tumors PLK1 dependent microtubule dynamics promotes androgen receptor (AR) signaling in prostate cancer1 PLK1 is upregulated in androgen insensitive prostate cancer cells and its inhibition leads to necroptosis2 PLK1 inhibition improves abiraterone acetate efficacy3 Inhibition of PLK1 represses androgen signaling pathway in castration resistant prostate cancer4 PLK1 inhibitors are anticipated to add important therapeutic benefit for the treatment of prostate cancer patients5 1Xianzeng, 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; 3Clemens, Thoma – Prostate Cancer: PLK-1 Inhibition Improves Abiraterone Efficacy; Nature Reviews Urology volume11, page603 (2014); 4Zhang 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; 5Klaus, 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 mCRPC1 Tumor volume was significantly reduced in mice treated with the combination of PLK inhibitor and abiraterone versus either drug alone 1Zhang et al, Cancer Res 2014; 74(22) Vehicle PLK inhibitor Abiraterone PLKi + Abi Vehicle PLK inhibitor Abiraterone PLKi + Abi Patient-derived xenograft (PDX) model of CRPC1 in abiraterone-resistant cell line Serum levels of PSA correlate with efficacy for the combination versus each drug alone

Planned Phase 2 Clinical Trial in mCRPC 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 PCM-075 in Combination with Zytiga® (abiraterone acetate) and Prednisone in Patients with Metastatic Castration-Resistant Prostate Cancer (mCRPC) Dosing Regimen PCM-075 – 24 mg/m2 Days 1-5 (21-Day Cycle) + Zytiga®/prednisone daily 4 Cycles = 12 Weeks Disease Control based on PSA level Duration Evaluation

Looking Ahead

2018 Goals Completion of dose escalation cohorts in Phase 1b trial1 Safety and preliminary clinical activity (pharmacodynamic markers) Recommended Phase 2 dose1 Initiation of Phase 2 continuation trial1; possible preliminary data Activation of Beth Israel, Dana Farber and Massachusetts General and first patient dosed at all centers Safety and clinical activity on “lead-in” patients (3-6 patients) 10 to 25 patients receive study drug Phase 1b/2 AML Trial Phase 2 mCRPC Trial 1Timing dependent on the number of cohorts required to reach maximum tolerated dose (MTD) / recommended Phase 2 dose

Takeaways PLK1 is an established cancer target; the PLK inhibitor drug class has demonstrated clinical activity in AML PCM-075 is the only oral, highly-selective PLK1 inhibitor in clinical trials Synergy demonstrated with PCM-075 in combination with chemotherapies and targeted therapeutics 2 clinical trials: Phase 1b/2 in AML (ongoing) and Phase 2 in mCRPC (planned) Significant collaborations with leading investigators and institutions, including MD Anderson (AML) and Beth Israel Deaconess (mCRPC) Patent and other marketing exclusivity worldwide through 2030 with opportunity for expansion

Financial Summary and Capitalization Capitalization Table as of April 30, 20181 Common Shares2 4,953,442 70% Warrants Outstanding 1,489,488 21% Options Outstanding3 630,061 9% Restricted stock units 30,919 0% Fully Diluted Shares4 7,103,910 100% 1Q 2018 Financial Summary Cash: $6.7 M Repayment of Equipment Line of Credit on April 6, 2018 in the amount of ~$1.1 M Net Cash used in Operating Activities in 1Q 2018: $(2.9) M 1 April 30, 2018 capitalization numbers have been converted to reflect a 1 for 12 reverse stock split which was effected on June 1, 2018 2 Includes 5,621 shares of Common Stock reflecting Series A Convertible Preferred Stock on an as-converted basis. 3 Weighted average exercise price $29.88 4 Does not include 2014 Employee Incentive Plan pool available for grant

For additional information or questions please contact: ir@trovagene.com