UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 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): **January 8, 2016**

Verastem, Inc.
(Exact Name of Registrant as Specified in Charter)

Delaware	**001-35403**	**27-3269467**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

117 Kendrick Street, Suite 500, Needham, MA	**02494**
(Address of Principal Executive Offices)	(Zip Code)

Registrant's telephone number, including area code: **(781) 292-4200**

(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 (see General Instruction A.2. below):

o Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

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

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

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

Item 7.01 Regulation FD Disclosure.

On January 7, 2016, Verastem, Inc. ("Verastem") updated its corporate presentation, a copy of which is furnished as Exhibit 99.1 hereto.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	**Description**
99.1	Slide presentation of Verastem, Inc. dated January 7, 2016.

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.

VERASTEM, INC.

Date: January 8, 2016

By: /s/ John B. Green
John B. Green
Chief Financial Officer

Exhibit 99.1

CORPORATE OVERVIEW

NASDAQ: VSTM

January 7, 2016

FORWARD-LOOKING STATEMENTS

This presentation, and other matters discussed today, or answers that may be given to questions asked, includes forward-looking statements about the Company's strategy, future plans and prospects, including statements regarding the development and activity of the Company's product candidates, including VS-6063, VS-4718 and VS-5584, and the Company's FAK, PI3K/mTOR and diagnostics programs generally, the timeline for clinical development and regulatory approval of the Company's product candidates, the expected timing for the enrollment and the reporting of data from on-going trials, the structure of the Company's planned or pending clinical trials, additional planned studies, the Company's rights to develop or commercialize its product candidates and the ability of the Company to finance contemplated development activities and fund operations for a specified period. The words "anticipate," "appear," "believe," "estimate," "expect," "intend," "may," "plan," "predict," "project," "target," "potential," "will," "would," "could," "should," "continue," and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Each forward-looking statement is subject to risks and uncertainties that could cause actual results to differ materially from those expressed or implied in such statement. Applicable risks and uncertainties include the risks that the preclinical testing of the Company's product candidates and preliminary or interim data from clinical trials may not be predictive of the results or success of ongoing or later clinical trials, that data may not be available when we expect it to be, that enrollment of clinical trials may take longer than expected, that the Company's product candidates will cause unexpected safety events, that the Company will be unable to successfully initiate or complete the clinical development of the Company's product candidates, that the development of the Company's product candidates will take longer or cost more than planned, and that the Company's product candidates will not receive regulatory approval or become commercially successful products. Other risks and uncertainties include those identified under the heading "Risk Factors" in the Company's Annual Report on Form 10-K for the year ended December 31, 2014, the Company's Quarterly Report on form 10-Q for the quarter ended September 30, 2015 and in any subsequent SEC filings. The forward-looking statements contained in this presentation reflect the Company's current views with respect to future events, and the Company does not undertake and specifically disclaims any obligation to update any forward-looking statements.

THE VERASTEM OPPORTUNITY

- Multi-faceted approach to improving outcomes for patients with cancer
  - Reduce cancer stem cells
  - Boost immune attack
  - Reduce stromal density

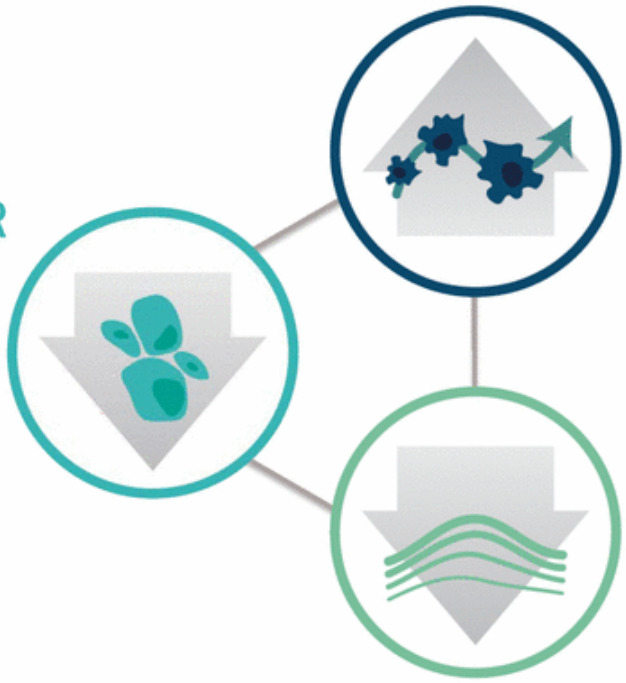
- Two programs with clinical-stage oral kinase inhibitors targeting multiple tumor types
  - ***FAK*** – VS-6063 and VS-4718
  - ***PI3K/mTOR*** – VS-5584
- Well capitalized
  - $120.1M in cash and cash equivalents as of Sept. 30, 2015
  - Sufficient operating capital into 2018
- Strong IP (Composition of Matter)
  - VS-6063: 2028
  - VS-4718: 2028
  - VS-5584: 2029
- Experienced management and Board

CHALLENGES IN IMPROVING OUTCOMES FOR PATIENTS WITH CANCER

Resident cancer stem cells, limited immune response, and dense stroma contribute to the ineffectiveness of current treatments.

MULTI-FACETED APPROACH TO IMPROVING CANCER PATIENT OUTCOMES

REDUCE CANCER STEM CELLS
to prevent recurrence and metastasis[1,2]



BOOST IMMUNE ATTACK
to increase proportion of responders and duration of response[3]

REDUCE STROMAL DENSITY
to increase drug and immune system penetration into tumors[4]

1. Kolev VN et al. **FAK inhibition targets cancer stem cells.** EORTC 2015
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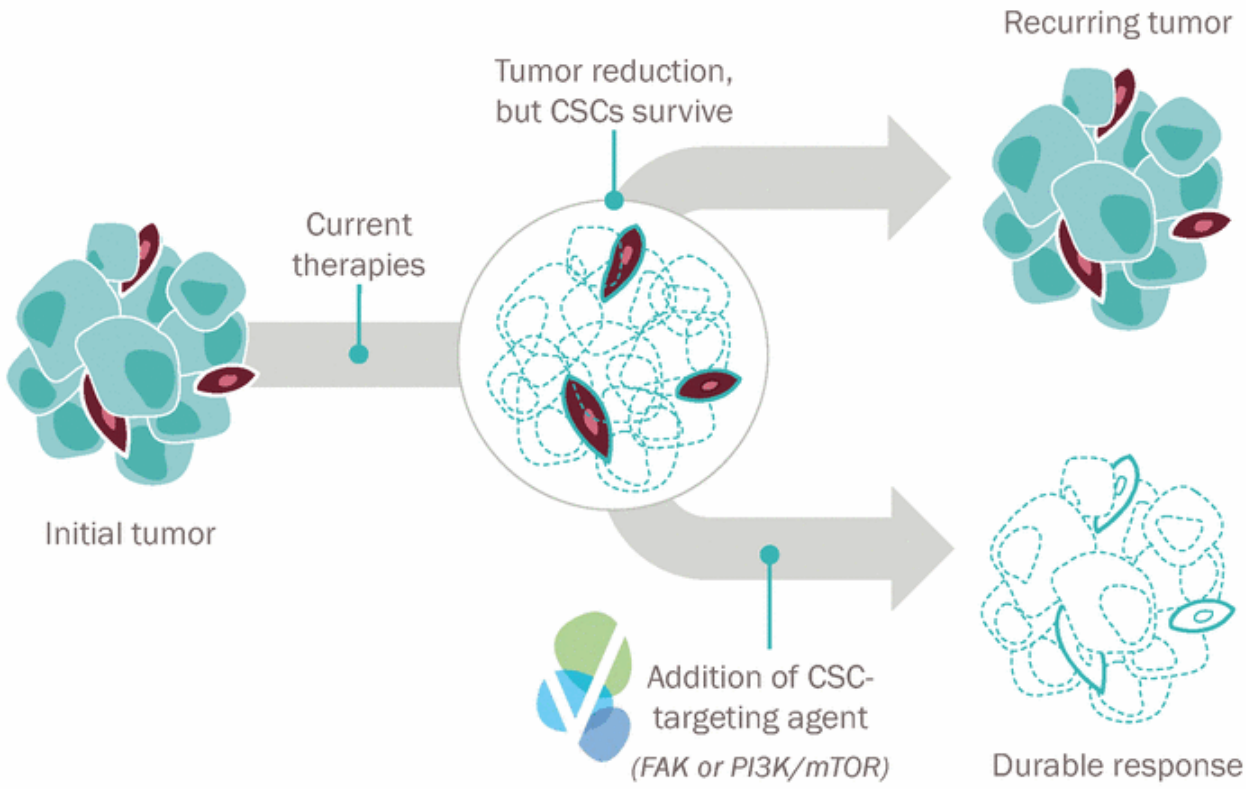
3. Serrels et al. **Nuclear FAK controls chemokine transcription, Tregs, and evasion of anti-tumor immunity.** Cell. 2015
4. Stokes JB et al. **Inhibition of Focal adhesion Kinase by PF-562,271 inhibits the growth and metastasis of pancreatic cancer concomitant with altering the tumor microenvironment.** Mol Cancer Ther. 2011

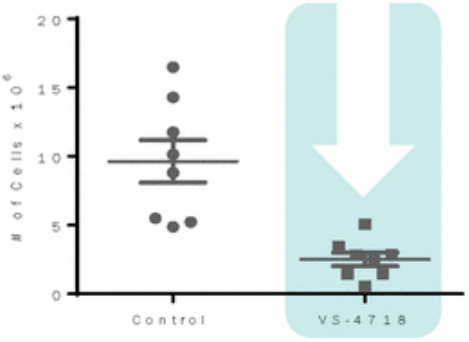
REDUCING CANCER STEM CELLS (CSC) AND BULK TUMOR TO IMPROVE DURATION OF RESPONSE



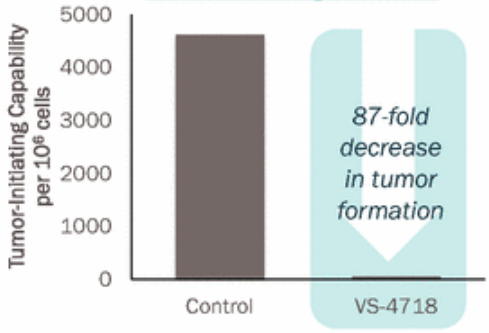
FAK INHIBITORS SHOWN TO REDUCE CANCER STEM CELLS

Ovarian mouse model (ID8)

In vivo tumor cells in ascites

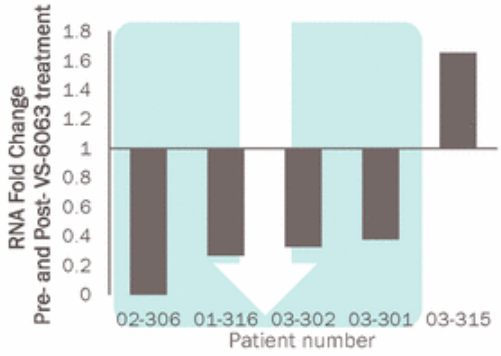


In vivo limiting dilutions

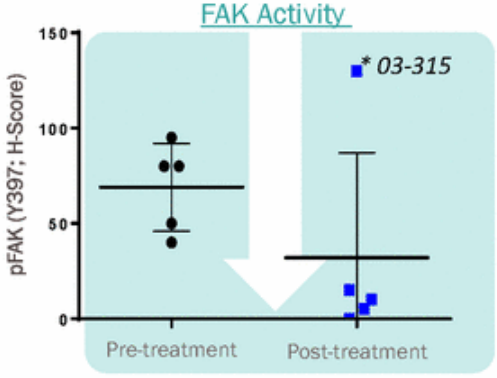


Ovarian patients, paired biopsies

SOX2 RNA (CSC marker)



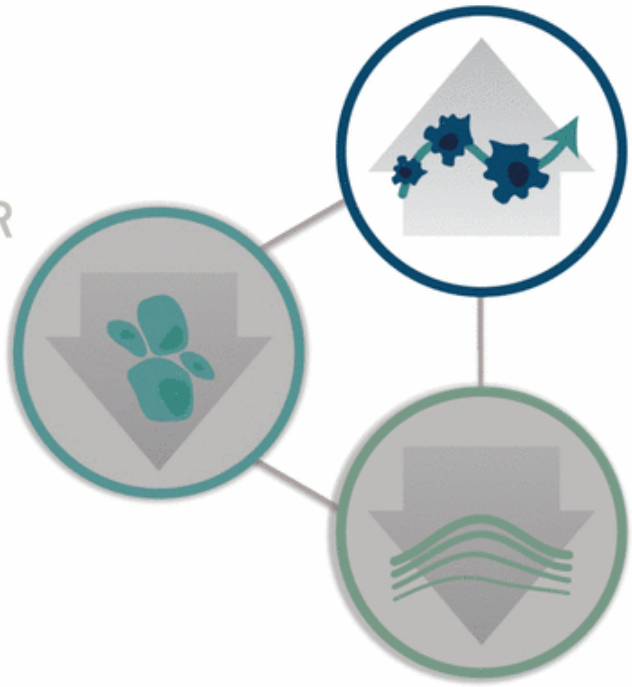
FAK Activity



 Source: Verastem data from preclinical and clinical studies

Verastem

MULTI-FACETED APPROACH TO IMPROVING CANCER PATIENT OUTCOMES



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BOOSTING IMMUNE ATTACK BY COMBINING FAK INHIBITION WITH IMMUNOTHERAPIES

Treatment with FAK inhibitor

DECREASE

Immuno-Suppressive Cells
Myeloid-derived suppressor cells, T-regs, M2 tumor-associated macrophages

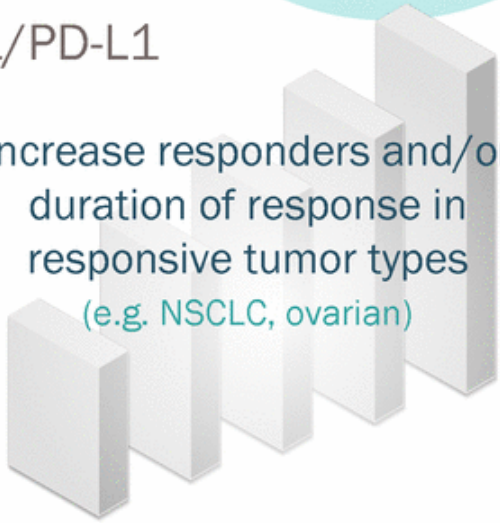
INCREASE

Cytotoxic CD8+ T cells
Tumor infiltration & tumor cell killing

Checkpoint inhibitor target presentation
PD-L1

+ anti-PD-1/PD-L1

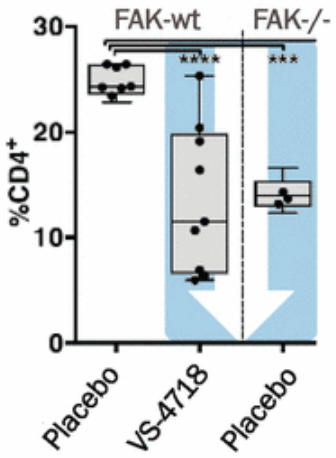
Opens up potential for targeting refractory or "cold" tumor types
(e.g. pancreatic)



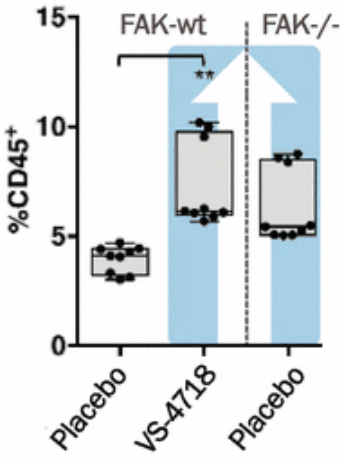
Increase responders and/or duration of response in responsive tumor types
(e.g. NSCLC, ovarian)

FAK INHIBITORS PRODUCE FAVORABLE CHANGES TO IMMUNE SYSTEM TO DECREASE TUMOR BURDEN

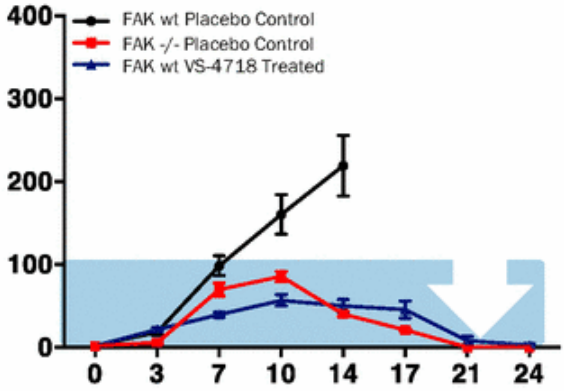
Immunosuppressive T-regs



Cytotoxic CD8+ T cells



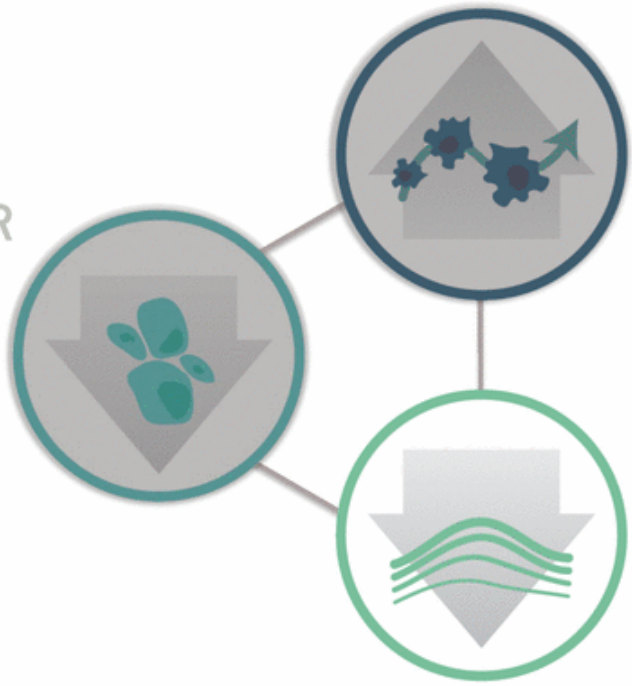
Tumor burden



Small-molecule inhibition or genetic knockout of FAK eliminates tumors via T cell attack

Serrels et al. **Cell** 163: 160, 2015
VS-4718 treatment vs. FAK knock out, SCC 7.1 model
T-regs: CD4+ FOXP3+ CD25+; CD8+ T cells: CD45+ CD3+ CD4- CD8+
p-values: ** = $p < 0.01$; *** = $p < 0.001$; **** = $p < 0.0001$

MULTI-FACETED APPROACH TO IMPROVING CANCER PATIENT OUTCOMES



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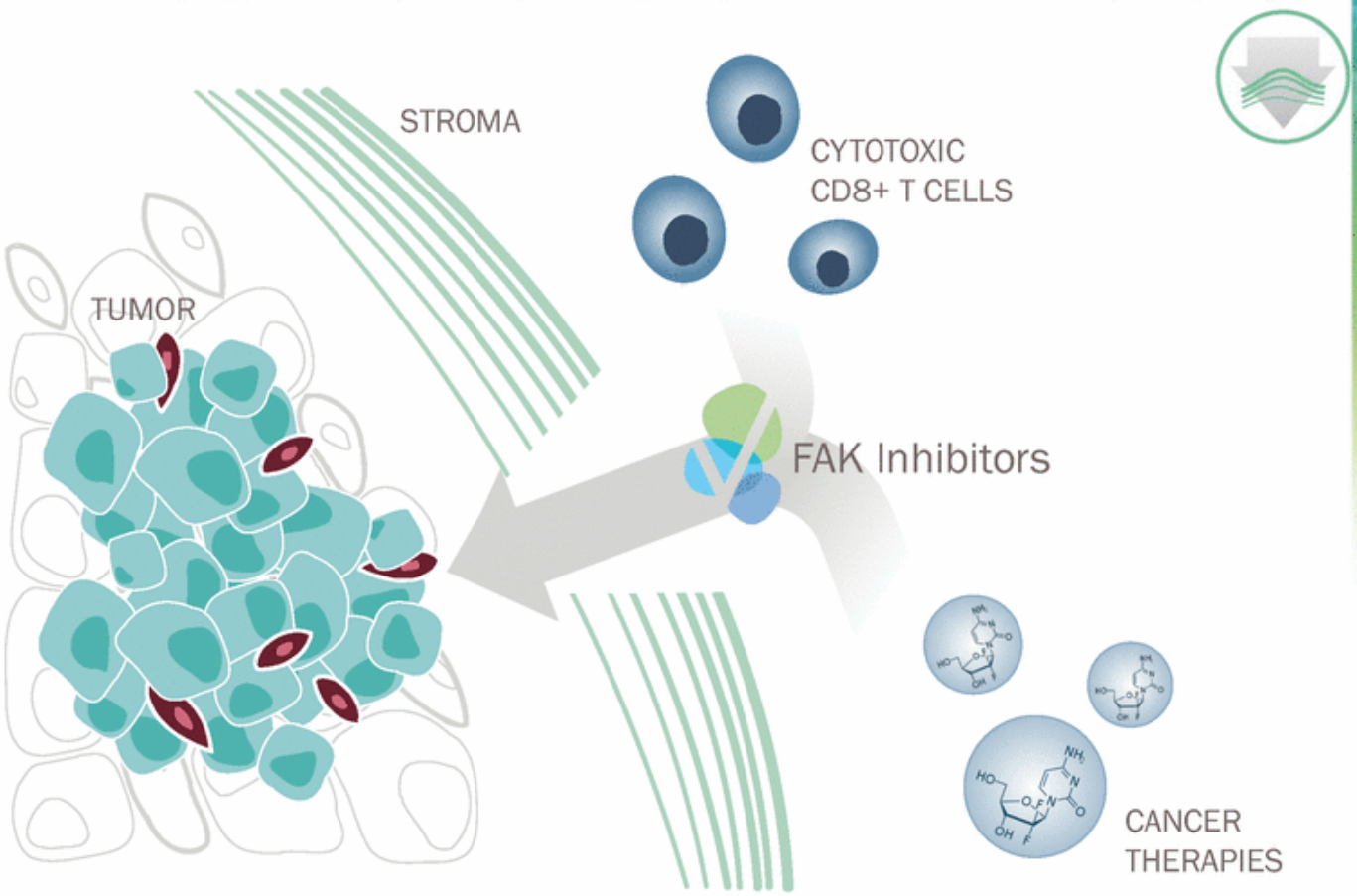
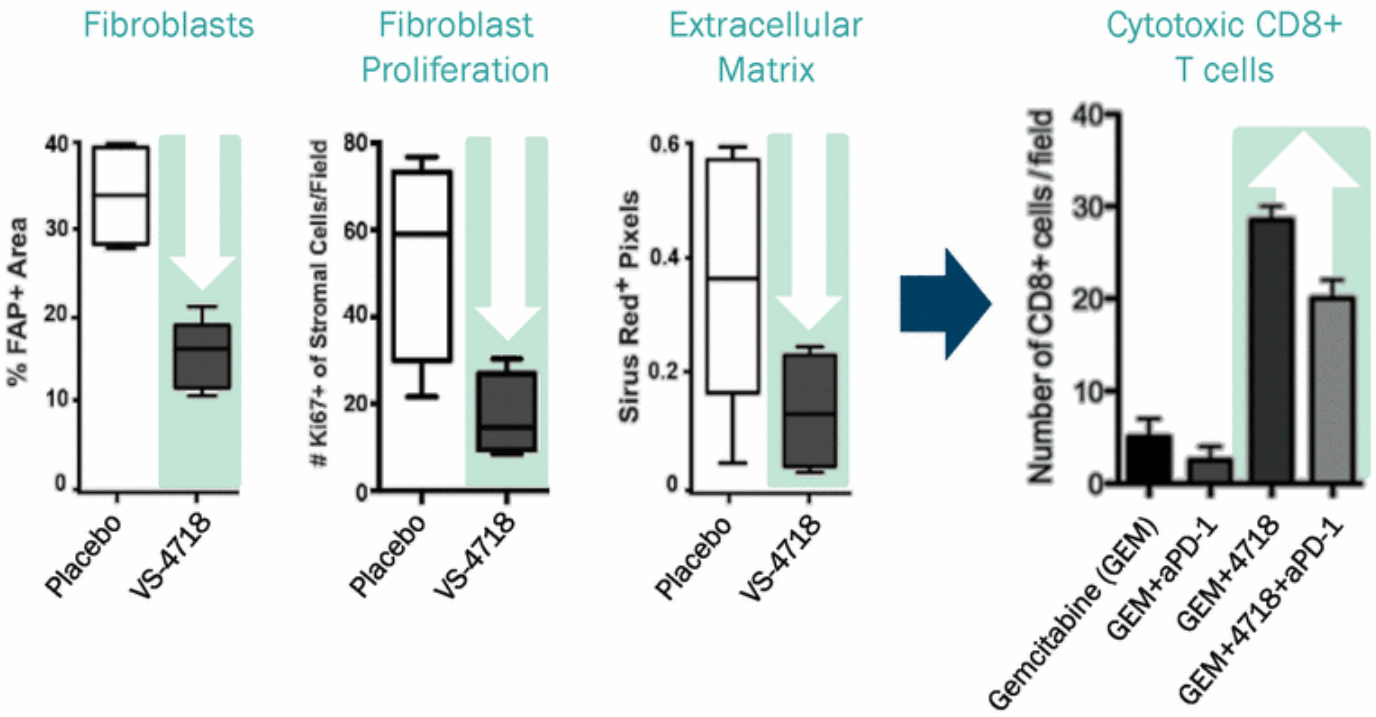
FAK INHIBITION REDUCES STROMAL DENSITY ENABLING THERAPIES & IMMUNE CELLS TO PENETRATE TUMORS



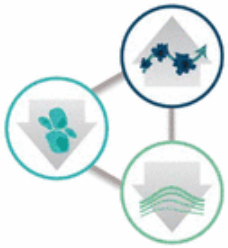
Stromal density = Tumor-associated fibroblasts + extracellular matrix proteins

FAK INHIBITION REDUCES STROMAL DENSITY AND BOOSTS T CELL ENTRY INTO THE TUMOR



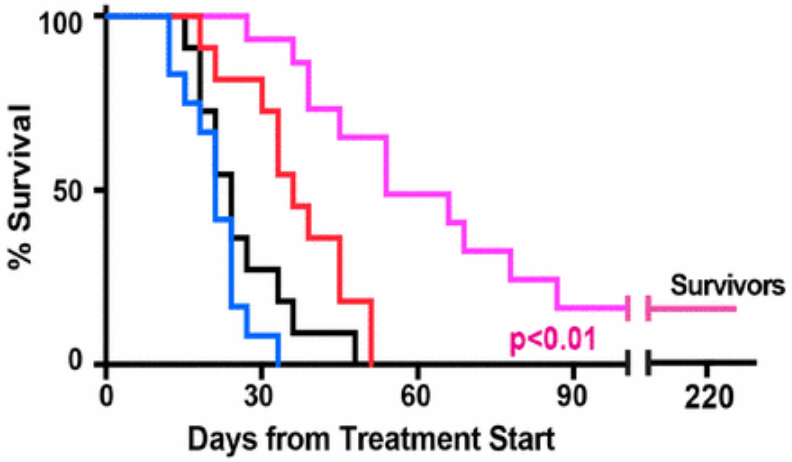
D. Denardo, Wash U; Jiang H et al. **Focal adhesion kinase inhibition enables efficacy of checkpoint immunotherapy in pancreatic cancer** EORTC 2015
ECM, FAP+ Fibroblasts, Fibroblast proliferation: PDAC transgenic pancreatic model, VS-4718 treatment
CD8+ T cells: Kras/p53 pancreatic tumors, Gem +/- anti-PD-1 +/- VS-4718 treatment

PROOF OF CONCEPT: MULTI-FACETED APPROACH TO IMPROVE SURVIVAL IN PANCREATIC CANCER



1. TARGET CSCs
2. BOOST IMMUNE CELL RESPONSE
3. REDUCE STROMAL DENSITY

FAK Inhibitors Combined with Immuno-Oncology Therapies Improves Long term Survival in Aggressive Pancreatic Cancer



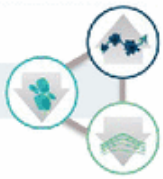
Transgenic Kras/p53 pancreatic model

"Immuno" = anti-PD-1 + anti-CTLA-4 + GEM (25 mg/kg)

D. Denardo, Wash U; Jiang H et al. **Focal adhesion kinase inhibition enables efficacy of checkpoint immunotherapy in pancreatic cancer** EORTC 2015

FAK PROGRAM: NEWLY INITIATED COMBINATION STUDIES IN PANCREATIC CANCER

Phase 1/1b, VS-4718 + Gemcitabine + Abraxane



DOSE ESCALATION (Ongoing)

- Advanced solid tumors
- 28-day cycles of VS-4718 (oral BID) + Gem/Abraxane
- 3+3 escalation

RP2D

EXPANSION, 1ST LINE PANCREATIC (Planned)

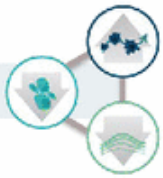
1st line pancreatic

RANDOMIZATION

- **Arm 1:** VS-4718 + Gem/Abraxane (n = 10)
- **Arm 2:** Gem/Abraxane (n = 10)

Safety
ORR
PFS

Phase 1/1b, VS-6063 + Pembrolizumab (Merck anti-PD-1) + Gemcitabine



DOSE ESCALATION (Ongoing)

- Advanced solid tumors
- 21-day cycles of VS-6063 (oral BID) + pembrolizumab (IV on Day 1) + gemcitabine (IV on Days 1 and 8)
- 3+3 escalation

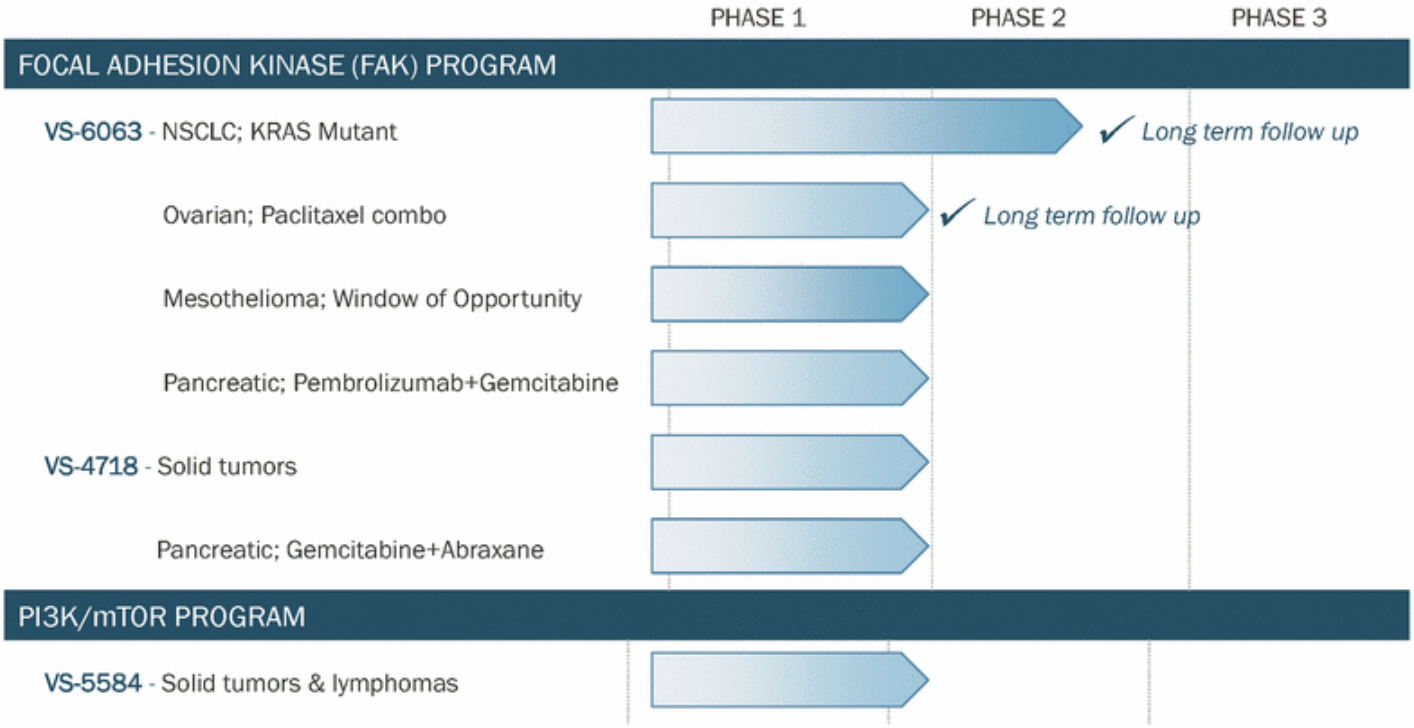
RP2D

EXPANSION, ADVANCED PANCREATIC (Planned)

- **Arm 1:** VS-6063 + Pembrolizumab + Gem
  Stable on front-line Gem/Abraxane (n = 10)
- **Arm 2:** VS-6063 + Pembrolizumab + Gem
  1 – 2 prior lines of therapy (n = 10)

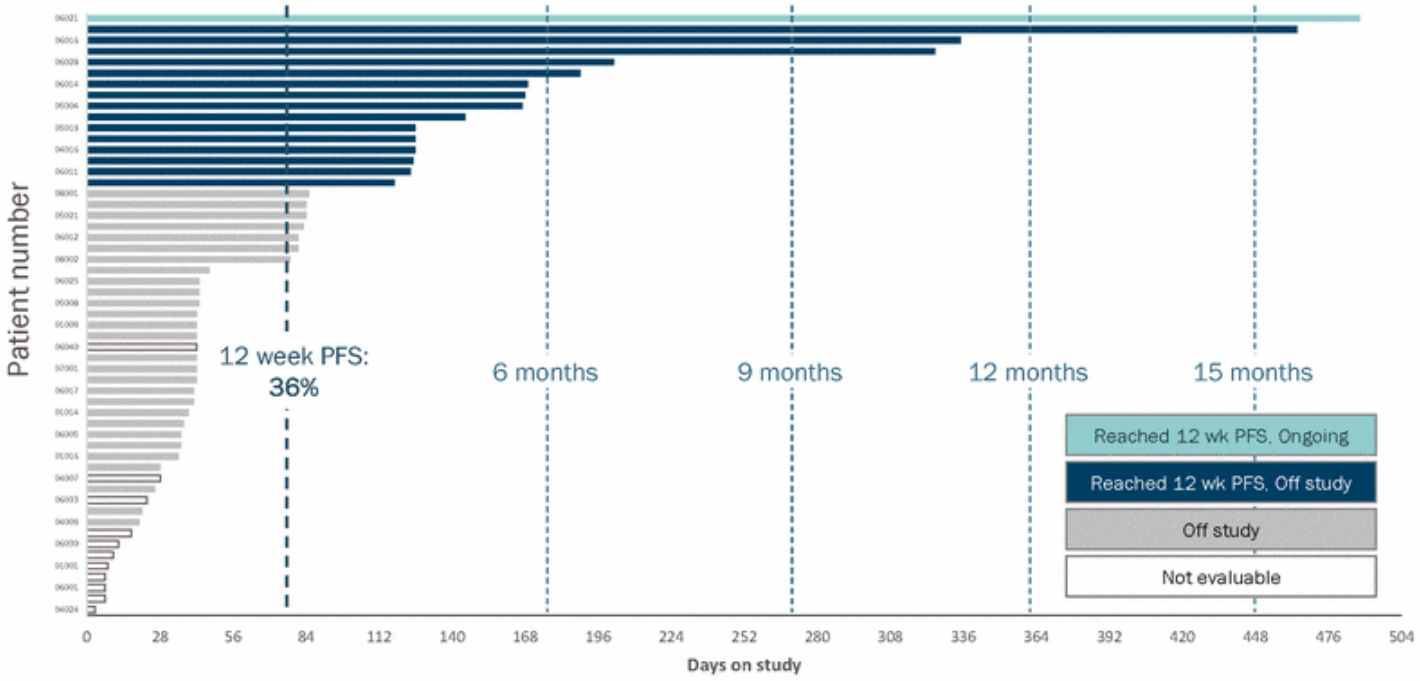
Safety
ORR
PFS
OS

VERASTEM PORTFOLIO OF CANCER PROGRAMS



VS-6063 MONOTHERAPY IN KRAS-MUTANT NSCLC SHOWED ENCOURAGING PROGRESSION-FREE SURVIVAL RATES

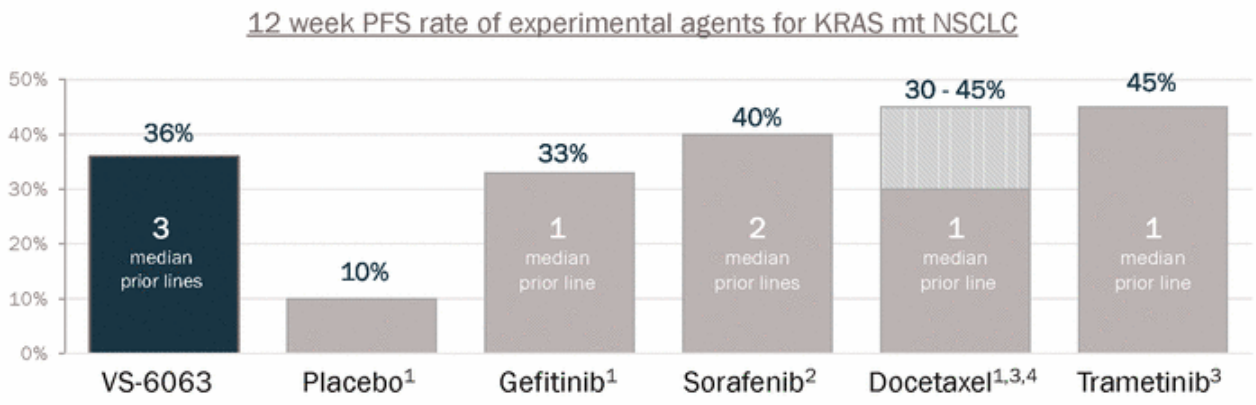
- ✓ 16/44 (36%) evaluable patients alive and progression free at 12 weeks
- ✓ Best overall response (RECIST): PR = 1; SD = 26; PD = 15; NE = 2
- ✓ Median PFS 11.7 weeks, with 6 patients on study for > 6 months and 1 patient continuing on study > 15 months



 Source: Unlocked, in-progress data as of 12/21/2015

Verastem

VS-6063 AS SINGLE AGENT IS COMPARABLE TO TARGETED AGENTS AND DOCETAXEL



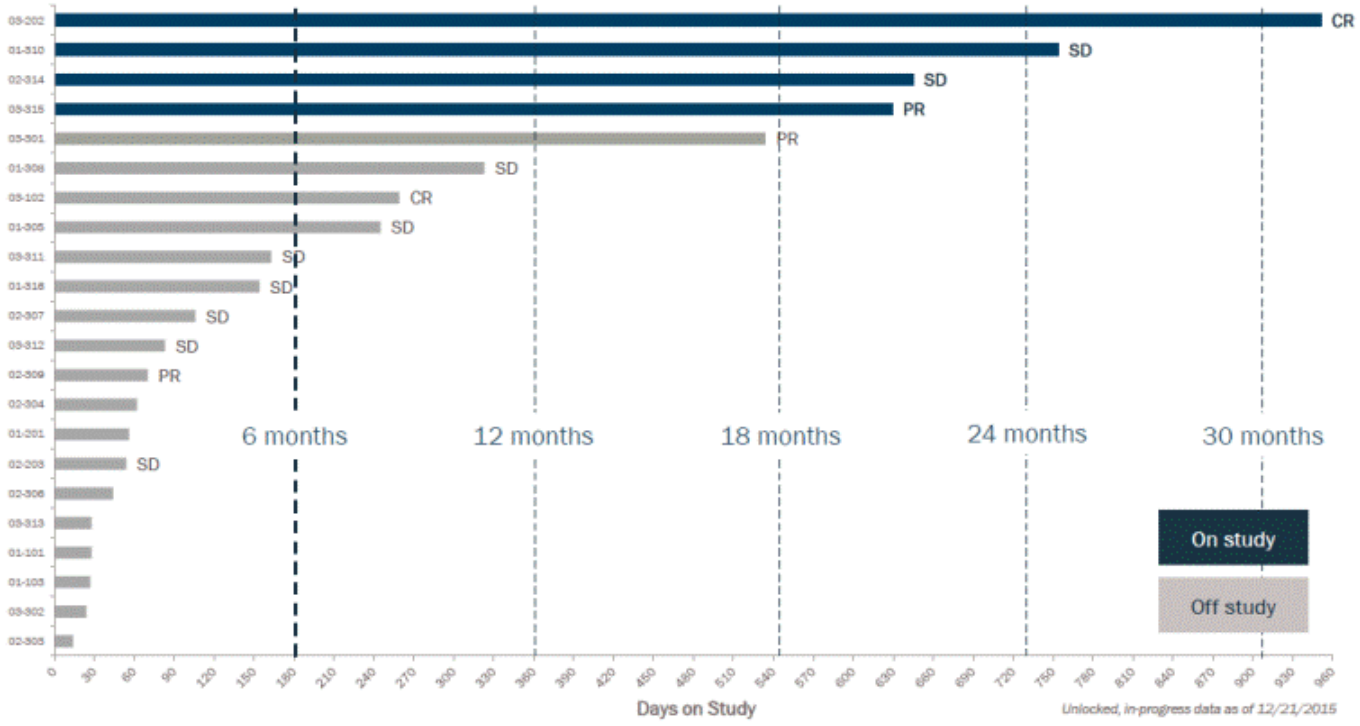
*"VS-6063 was generally well tolerated and suitable for long-term dosing. In this cohort of heavily pretreated patients, there were signs of single-agent activity comparable to other targeted agents and docetaxel. **Future directions include possible combination studies with existing standard and emerging therapies, including checkpoint inhibitors.**"*

Dr. David Gerber, IASLC 2015

1. Phase 3 INTEREST, Douillard et al., JCO 2010
2. Phase 3 MISSION, Mok et al., ESMO 2012
3. Phase 2, Blumenschein et al., Ann Oncol 2015
4. Phase 2, Janne et al., Lancet 2013

VS-6063 PLUS PACLITAXEL SHOWED PROMISING DURATION OF RESPONSES IN OVARIAN CANCER

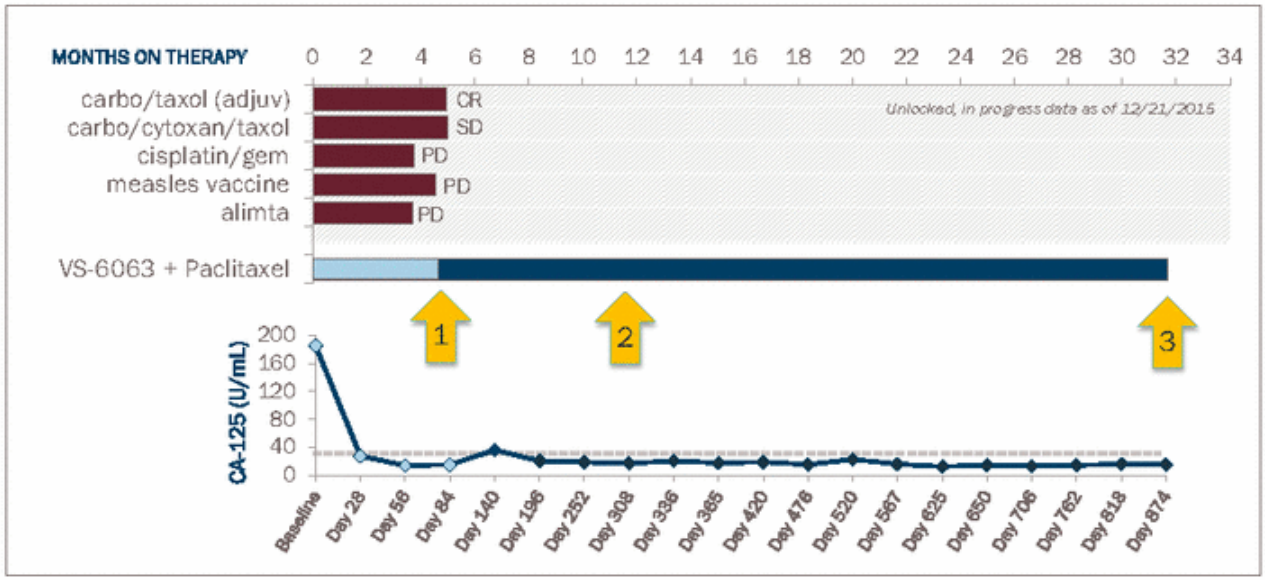
- ✓ VS-6063 can be safely combined with paclitaxel
- ✓ 41% (9/22) disease control (objective response or SD ≥ 6 months; 2 CR; 3 PR; 4 SD ≥ 6 months)



DURABLE COMPLETE RESPONSE SHOWS POTENTIAL FOR FAK INHIBITION TO "FINISH THE JOB"

Patient 03-202: Stage IV platinum-resistant serous ovarian cancer with 5 prior lines of therapy

Stable disease (SD) with combination treatment → 1 VS-6063 monotherapy at 4.5 months → 2 Complete response (CR) at 11.8 months → 3 Continues to tolerate VS-6063 well



Potential next steps: *Combine VS-6063 with checkpoint inhibitors*

VS-5584, A PAN-PI3K/MTOR INHIBITOR, WILL BE MOVING SHORTLY INTO OVARIAN AND HEME EXPANSION COHORTS

- VS-5584 is equipotent against mTORC1, mTORC2 and all four PI3K class I isoforms
- Combined inhibition of PI3K and mTOR is critical to CSC targeting[1]

mTOR IC50 (nM)	PI3K isoform IC_{50} (nM)			
	Alpha	Beta	Delta	Gamma
3.4	2.6	21	3.0	2.7

Phase 1: Ongoing dose escalation of oral, intermittent dosing in advanced non-hematologic malignancies or lymphoma

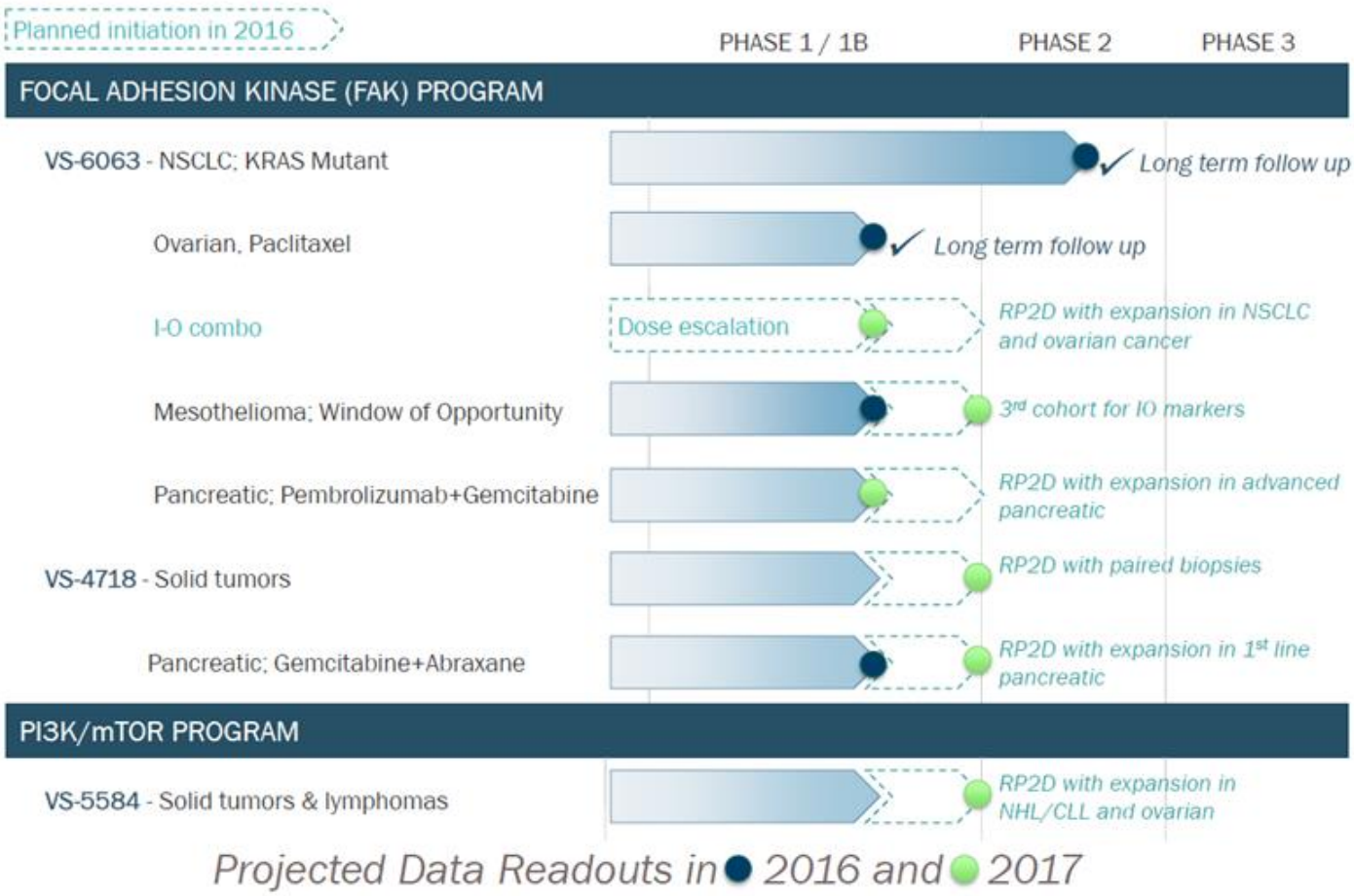
- ✓ Continues to be safe and well tolerated
- ✓ 5-75mg dose range. MTD reached
- ○ Ongoing RP2D confirmation cohort at 55 mg dose

Planned expansion cohorts at RP2D in ovarian & NHL/CLL

Potential for PARPi combination in ovarian & endometrial

[1]Kolev VN et al. **PI3K/mTOR dual inhibitor VS-5584 preferentially targets cancer stem cells.** Cancer Res. 2015

2016 MILESTONES



EXECUTIVE MANAGEMENT

Robert Forrester
President/CEO, BOD
CEO/CFO, CombinatoRx/COLY
MeesPierson, Barclays, UBS

Daniel Paterson
Chief Operating Officer
CEO: The DNA Repair Co. (now On-Q-ity)
PharMetrics (now IMS), Axion

Steven Bloom
VP, Corporate Development
VP, Commercial Strategy and Business Development
Ziopharm, PharMetrics (now IMS) , Eli Lilly and Company

Jonathan Pachter, Ph.D.
VP, Head of Research
Head of Cancer Biology, OSI (now Astellas)
Schering-Plough (now Merck)

Jack Green
Chief Financial Officer
CFO, Genzyme Transgenics Corporation (GTC)

Lou Vaickus, M.D., FACP
Interim Chief Medical Officer
VP, Head of Clinical Development Vertex
Tolerx, Sunovion, EMD Serono

BOARD OF DIRECTORS

Timothy Barberich
Former CEO/Chair Sepracor (SEPR)

Henri Termeer
Lead Director
Former CEO/Chair Genzyme

Alison Lawton
Former Genzyme (now Sanofi)

Paul Friedman, M.D.
Former President/CEO Incyte (INCY)

Christoph Westphal, M.D., Ph.D.
Cofounder/CEO: MNTA, ALNY, XLRN, SIRT, VSTM
Cofounder: Alnara (now Lilly), OvaScience (OVAS)

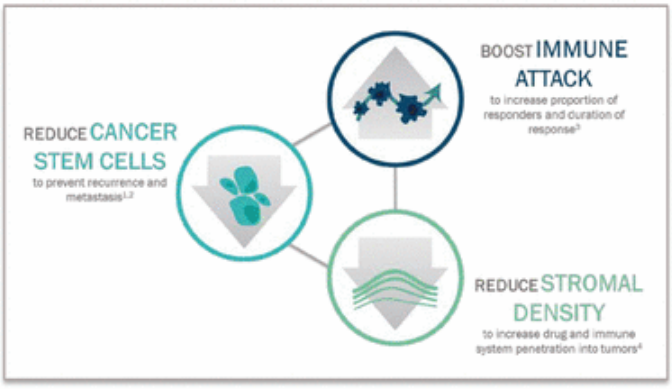
Louise Phanstiel
BOD: Cedars Sinai, MYGN

Michael Kauffman, M.D., Ph.D.
CEO Karyopharm (KPTI), former CMO Onyx

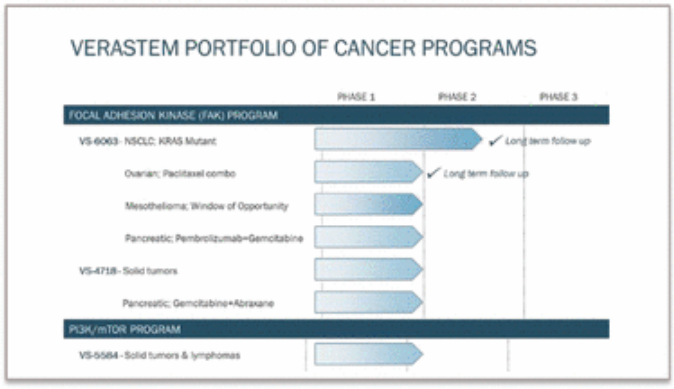
Stephen Sherwin, M.D.
BOD: BIIB; NBIX, RIGL

THE VERASTEM OPPORTUNITY

Multi-faceted approach to improving outcomes



Clinical programs targeting multiple cancers



Experienced team

EXECUTIVE MANAGEMENT

Robert Forrester
President/CEO, BOD
CEO/CFO, Constellation/COLY
Mees Pierson, Barclays, UBS

Daniel Paterson
Chief Operating Officer
CEO: The DNA Repair Co. (now On-Q-ity)
PharMetrics (now IMS), Avion

Steven Bloom
VP, Corporate Development
VP, Commercial Strategy and Business Development
Zoptarel, PharMetrics (now IMS), Eli Lilly and Company

Jonathan Pachter, Ph.D.
VP, Head of Research
Head of Cancer Biology, OSI (now Astellas)
Schering-Plough (now Merck)

Jack Green
Chief Financial Officer
CFO, Genzyme Transgenics Corporation (GTC)

Lou Vaickus, M.D., FACP
Interim Chief Medical Officer
VP, Head of Clinical Development Vertex
Tolaris, Sunovion, EMD Serono

BOARD OF DIRECTORS

Timothy Barberich
Former CEO/Chair Sepracor (SEPR)

Henri Termeer
Lead Director
Former CEO/Chair Genzyme

Alison Lawton
Former Genzyme (now Sanofi)

Paul Friedman, M.D.
Former President/CEO Incyte (INCY)

Christoph Westphal, M.D., Ph.D.
Cofounder/CEO: MNTA, ALNY, XLRN, SIRT, VSTM
Cofounder: Alnara (now Lilly), OvaScience (OVAS)

Louise Phanstiel
BOD: Gelters Sinai, MYGN

Michael Kauffman, M.D., Ph.D.
CEO Karyopharm (KPTI), former CMO Onyx

Stephen Sherwin, M.D.
BOD: BIIB, HRX, RHIS

Well capitalized with strong IP

$120.1M

IN CASH AND CASH EQUIVALENTS
AS OF SEPT. 30, 2015
Sufficient operating capital into 2018

Intellectual Property (Composition of Matter)
VS-6063: 2028
VS-4718: 2028
VS-5584: 2029