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): **April 27, 2020**

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:

☐ Written communications 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))

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

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common stock, $0.0001 par value per share	VSTM	The Nasdaq Global Market

Indicate by check mark whether the registrant is an emerging growth company 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 2.02 Results of Operations and Financial Condition.

On April 27, 2020, Verastem, Inc. (the “Company”) announced that net product revenue for the first fiscal quarter of 2020 was $5.0 million dollars. The Company’s actual results may differ from these estimates due to the completion of the Company’s closing procedures with respect to the quarter ended March 31, 2020, final adjustments and other developments that may arise between now and the time the financial results for the fiscal quarter are finalized. A full text of the American Association for Cancer Research (AACR) 2020 Virtual Annual Meeting I presentation (the “Presentation”) in connection with the announcement is furnished as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated by reference into this Item 2.02.

This information contained in this Current Report on Form 8-K, including Exhibit 99.1 attached hereto, is being furnished and shall not be deemed filed for any purpose, and shall not be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended (the “Securities Act”), or the Securities Exchange Act of 1934, as amended (the “Exchange Act”), except as expressly set forth by specific reference in such filing.

Item 7.01 Other Events.

On April 27, 2020, the Company issued a press release and posted the Presentation announcing preliminary data from its ongoing investigator-initiated Phase 1 clinical study investigating VS-6766, its RAF/MEK inhibitor, in combination with defactinib, its FAK inhibitor, in patients with KRAS mutant advanced solid tumors. Copies of the presentation and press release are attached hereto as Exhibits 99.1 and 99.2, respectively. The information in this report, including Exhibits 99.1 and 99.2, is being furnished pursuant to Item 7.01 and shall not be deemed filed for purposes of Section 18 of the Exchange Act, or otherwise subject to the liabilities of that section, nor will it be incorporated by reference in any filing under the Securities Act or in any filing under the Exchange Act, except as expressly set forth by specific reference in such filing.

Note Regarding Forward-Looking Statements

This Current Report on Form 8-K includes forward-looking statements about Verastem Oncology’s strategy, future plans and prospects, including statements related to the potential clinical value of the RAF/MEK/FAK combination and the timing of commencing a registration-directed trial for the RAF/MEK/FAK combination. The words "anticipate," "believe," "estimate," "expect," "intend," "may," "plan," "predict," "project," "target," "potential," "will," "would," "could," "should," "continue," “can,” “promising” and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words.

Applicable risks and uncertainties include the risks and uncertainties, among other things, regarding: the success in the development and potential commercialization of our product candidates, including defactinib in combination with VS-6766; the occurrence of adverse safety events and/or unexpected concerns that may arise from additional data or analysis or result in unmanageable safety profiles as compared to their levels of efficacy; our ability to obtain, maintain and enforce patent and other intellectual property protection for our product candidates; the scope, timing, and outcome of any legal proceedings; decisions by regulatory authorities regarding labeling and other matters that could affect the availability or commercial potential of our product candidates; whether preclinical testing of our product candidates and preliminary or interim data from clinical trials will be predictive of the results or success of ongoing or later clinical trials; that the timing, scope and rate of reimbursement for our product candidates is uncertain; that third-party payors (including government agencies) may not reimburse; that there may be competitive developments affecting our product candidates; that data may not be available when expected; that enrollment of clinical trials may take longer than expected; that our product candidates will experience manufacturing or supply interruptions or failures; that we will be unable to successfully initiate or complete the clinical development and eventual commercialization of our product candidates; that the development and commercialization of our product candidates will take longer or cost more than planned; that we or Chugai Pharmaceutical Co., Ltd. will fail to fully perform under the VS-6766 (CH5126766) license agreement; that we may not have sufficient cash to fund our contemplated operations; that we may be unable to make additional draws under our debt facility or obtain adequate financing in the future through product licensing, co-promotional arrangements, public or private equity, debt financing or otherwise; that we will be unable to execute on our partnering strategies for defactinib in combination with VS-6766; that we will not pursue or submit regulatory filings for our product candidates, and that our product candidates will not receive regulatory approval, become commercially successful products, or result in new treatment options being offered to patients.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Verastem, Inc. Presentation for American Association for Cancer Research (AACR) 2020 Virtual Annual Meeting I
99.2	Verastem, Inc. Press Release, dated April 27, 2020

SIGNATURES

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.

Dated: April 27, 2020

By: /s/ Brian M. Stuglik
Brian M. Stuglik
Chief Executive Officer

Exhibit 99.1


Verastem
Oncology
Addressing RAS Pathway Blockade & Resistance
VS-6766 & Defactinib Combination Data in KRAS Mutant Solid Tumors
Investor Conference Call and Webcast
NASDAQ: VSTM
April 27, 2020

Speakers

Verastem Oncology

Brian Stuglik
CEO

Jon Pachter
CSO

Dan Paterson
COO

Rob Gagnon
CFO

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Lead Investigator

Udai Banerji,
MBBS, MD, DNB, PhD, FRCP

Professor Udai Banerji is the deputy head of the Drug Development Unit where he is involved in running the portfolio of more than 40 Phase I trials. He plays a key role bridging pre-clinical and clinical drug discovery by designing and conducting Phase I studies.

In addition to clinical trials, Professor Banerji leads the Clinical Pharmacodynamics Biomarker Group and the Clinical Pharmacology – Adaptive Therapy Groups at The Institute of Cancer Research. His laboratory interests include anticancer drug resistance and pharmacological aspects of cancer evolution.

Professor Banerji holds a PhD from The Institute of Cancer Research and completed his medical oncology training at The Royal Marsden Hospital.

Agenda

Topic	Presenter
• Introduction	• Brian Stuglik
• RAS Pathway: Current Approaches and Unmet Needs • RAS Pathway Blockade: Bypass Mechanisms and Resistance • VS-6766 and Defactinib	• Jon Pachter
• Phase 1 Combination Data	• Udai Banerji
• Next Steps • Concluding Remarks	• Dan Paterson & Brian Stuglik

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Safe Harbor Statement

This presentation includes forward-looking statements about, among other things, Verastem Oncology's products and product candidates, including anticipated regulatory submissions, approvals, performance and potential benefits of Verastem Oncology products and product candidates, that are subject to substantial risks and uncertainties that could cause actual results to differ materially from those expressed or implied by such statements.

Additional information regarding these factors can be found in Verastem Oncology's Annual Report on Form 10-K for the fiscal year ended December 31, 2019 and in any subsequent filings with the SEC, including in the sections thereof captioned "Risk Factors" and "Forward-Looking Information and Factors that May Affect Future Results," as well as in our subsequent reports on Form 8-K, all of which are filed with the U.S. Securities and Exchange Commission (SEC) and available at www.sec.gov and www.verastem.com.

The forward-looking statements in this presentation speak only as of the original date of this presentation, and we undertake no obligation to update or revise any of these statements.

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We are a biopharmaceutical company committed to developing and commercializing new medicines for patients battling cancer

Verastem Oncology

New lead clinical program has best-in-class potential	VS-6766 (RAF/MEK) and defactinib (FAK) inhibition clinically active against KRAS mutant variants, especially KRAS G12V & G12D
Rapid development pathway to market	Clinical proof-of-concept achieved in KRAS mutant low-grade serous ovarian cancer (LGSOC); goal to initiate registration-directed trial in 2020
Significant downstream market opportunity and blockbuster potential	30% of all human cancers are driven by mutations in RAS family of genes; VS-6766 combinations poised to fuel the future pipeline
Strong balance sheet and investor syndicate	Cash runway into the fourth quarter of 2021; recent financing funded by several premier life science investors
Revenue-generating commercial asset with multiple planned indication expansion opportunities	COPIKTRA® (duvelisib) generated $12.3M in 2019 and $5.0M in 1Q20 in approved indications; actively working toward label expansions in PTCL and other hematologic malignancies

Key Pipeline Programs Aligned with New Strategic Direction

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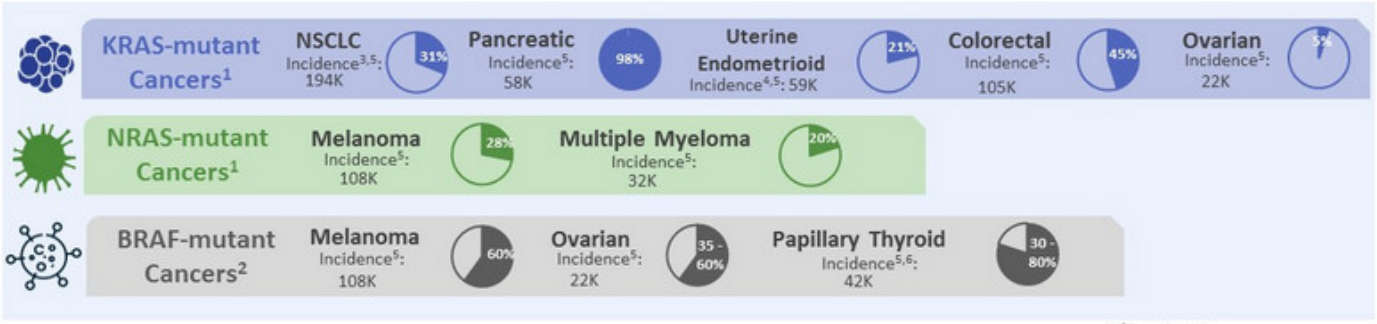
RAS Pathway: Current Approaches and Unmet Needs

Jon Pachter, PhD

Verastem Oncology

High Unmet Needs in RAS/RAF/MEK/ERK-Driven Cancers



Breadth of potential opportunity

- 30% of all human cancers are driven by mutations of the RAS family of genes

Established prognostic significance

- Patients with mutations of the RAS family have an overall worse prognosis

Challenges with conventional approaches

- Modest progress; limited number of approved therapies
- Single agent therapies (e.g. MEK inhibitors) associated with resistance
- Tolerable combination regimens with MEK inhibitors have been challenging

Incidence Sources:

[1]Reference for RAS mt frequencies– Cox et al. Nature Reviews 13: 828, 2014
[2]Reference for BRAF mt frequencies – Turski et al. Mol Cancer Ther 15: 533, 2016
[3]85% of lung cancer is NSCLC (Lu et. al. Cancer Manag Res. 2019)
[4] 90% of all uterine cancers are of the endometrial type (ACS)
[5] Cancer Statistics 2020, Siegel et. al. CA Cancer J Clin 2020;70:7-30
[6] 8 out of 10 thyroid cancers are of the papillary type (ACS)

References:
McCormick F Clin Cancer Res 15April2015
Adderley H et al. EBioMedicine 01Mar2019
Papke B et al. Science 17Mar2017
Ryan M et al. Nature Reviews Clinical Oncology 01Oct2018
NIH cancer.gov/research/key-initiatives/ras

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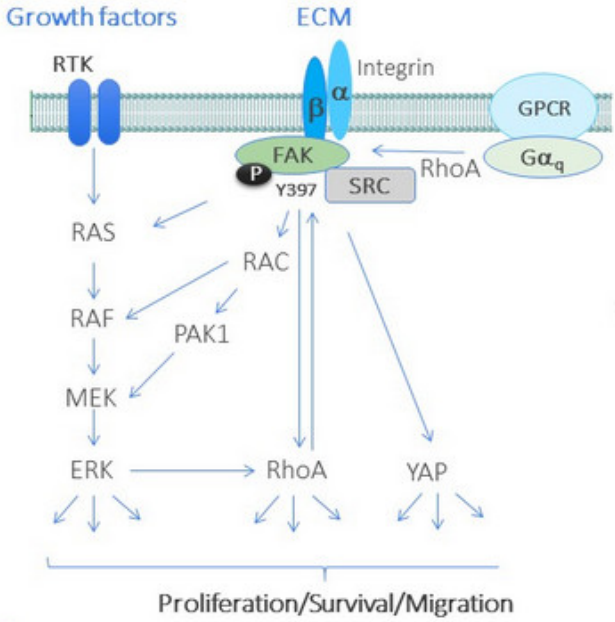
RAS Pathway Blockade: Bypass Mechanisms and Resistance

Jon Pachter, PhD

Verastem Oncology

Overcoming Key Resistance Mechanisms to MEK Inhibitors



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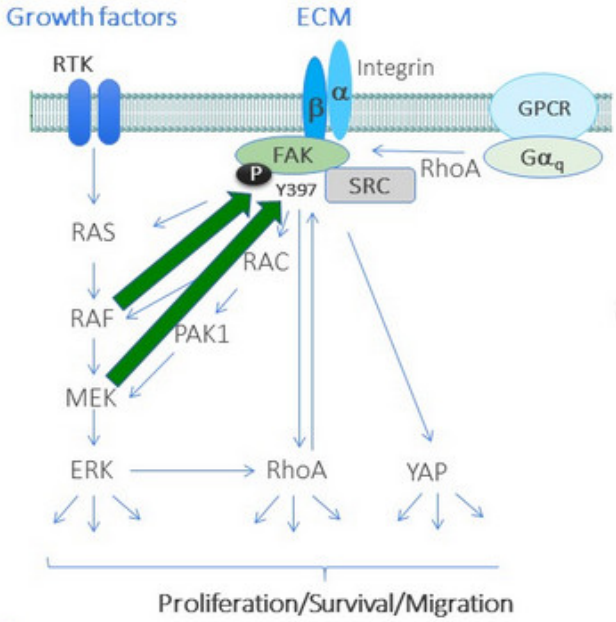
References:
Banerji, BTOG Dublin, Jan 23, 2019
Slack-Davis, JCB 162:281, 2003
Feng, Cancer Cell, 2019
Konstantinou, Cancer Discovery 3:444, 2013
Hirata, Cancer Cell 27:574, 2015

Overcoming Key Resistance Mechanisms to MEK Inhibitors



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    - Combination with defactinib reduced this compensatory pFAK signal

➡ = Feedback Reactivation

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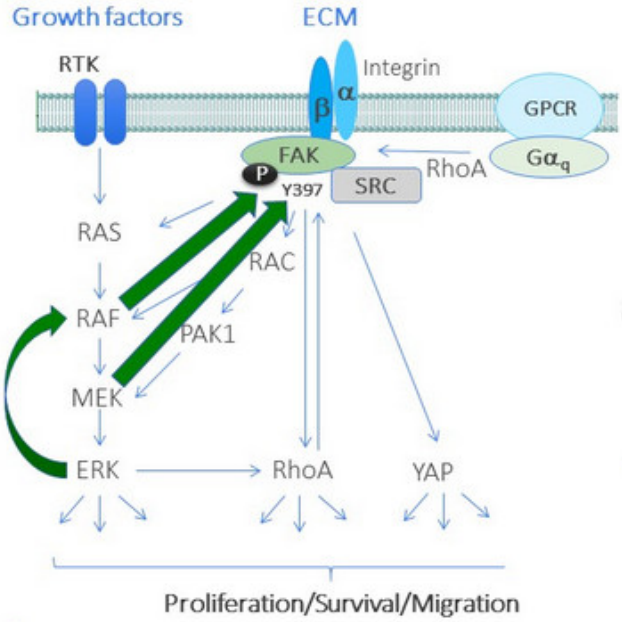
References:
1. Chen, Mol Cancer Res 2018
2. Banerji, BTOG Dublin, Jan 23, 2019

More Complete Shutdown requires Addressing Multiple Resistance Mechanisms



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- Upon MEK blockade, ERK feeds back to activate RAF kinase[3]

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References:
[1]Chen, Mol Cancer Res 2018
[2]Banerji, BTOG Dublin, Jan 23, 2019
[3]Ishii et al., *Cancer Res*, 2013

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VS-6766 and Defactinib

Jon Pachter, PhD

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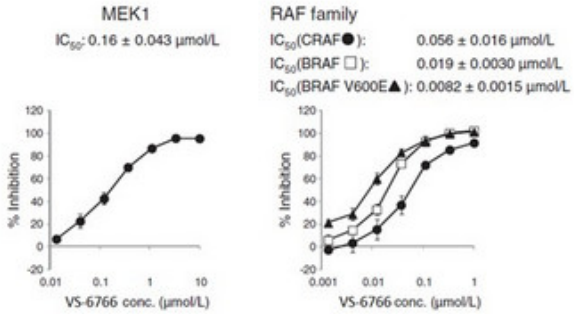
VS-6766 is a Unique Small Molecule RAF/MEK Inhibitor

- VS-6766 inhibits both MEK & RAF kinase activities
- Standard MEK inhibitors paradoxically induce MEK phosphorylation (pMEK) by relieving ERK-dependent feedback inhibition of RAF



- By inhibiting RAF phosphorylation of MEK, VS-6766 has advantage of not inducing pMEK
- VS-6766 inhibits ERK signaling more completely; may confer enhanced therapeutic activity

Reference:
Ishii et al., *Cancer Res*, 2013; Lito et al., Cancer Cell, 2014; Blasco, R. B. *et al. Cancer Cell* (2011); Sanclemente, M. *et al. Cancer Cell* (2018)

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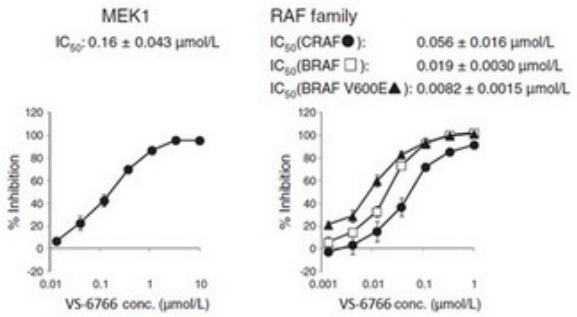

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VS-6766 inhibits CRAF

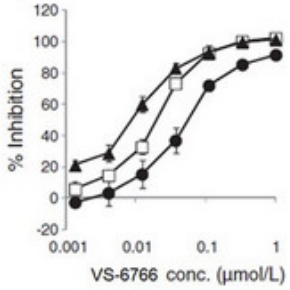
A central mediator of KRAS-G12V driven NSCLC

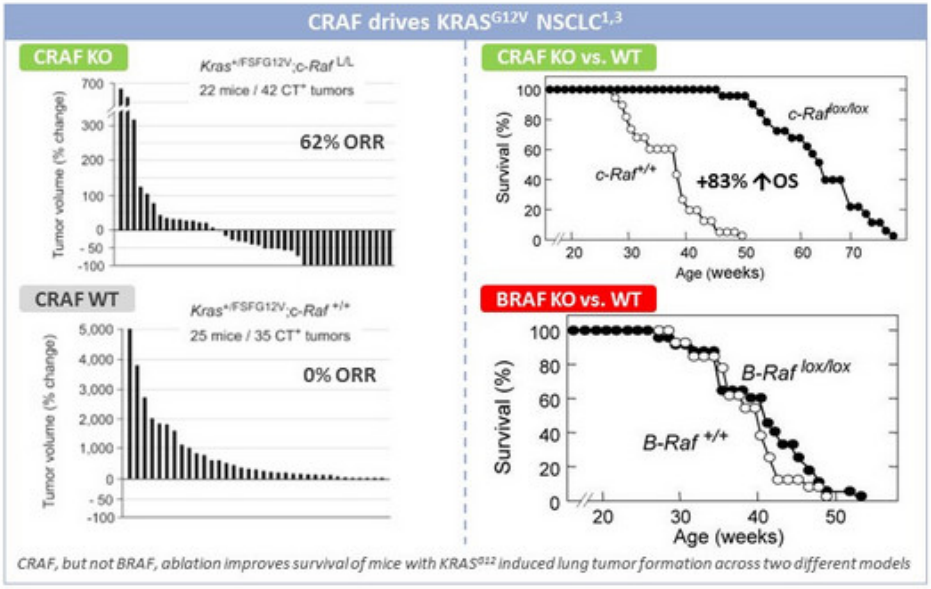
RAF family

IC_{50}(CRAF●): 0.056 ± 0.016 µmol/L

IC_{50}(BRAF □): 0.019 ± 0.0030 µmol/L

IC_{50}(BRAF V600E▲): 0.0082 ± 0.0015 µmol/L





CRAF, but not BRAF, ablation improves survival of mice with KRAS^{G12} induced lung tumor formation across two different models

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Source: Ishii et al. *Cancer Res* (2013), Blasco, R. B. *et al. Cancer Cell* (2011), Lito, P. *et al. Cancer Cell* (2014), Sanclemente, M. *et al. Cancer Cell* (2018)

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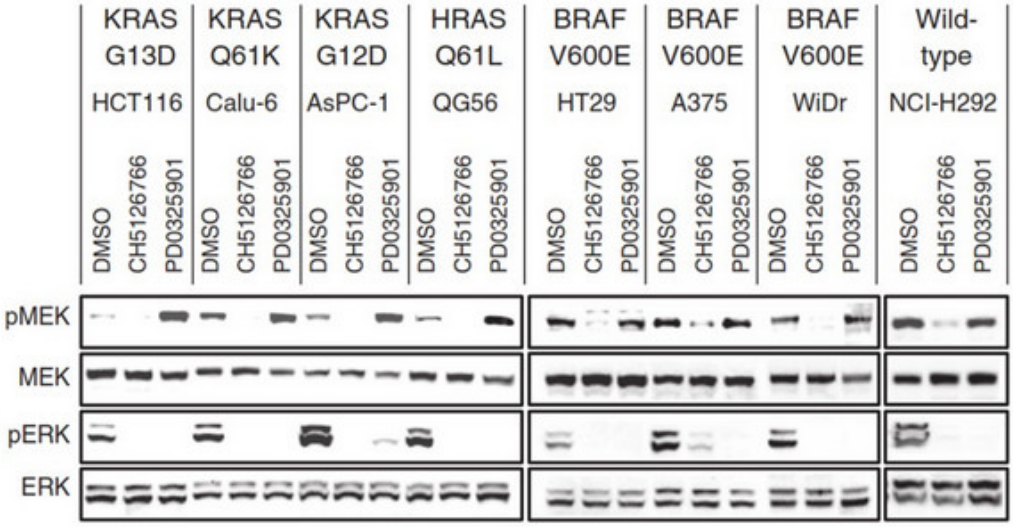
VS-6766 is Effective against Multiple RAS & RAF Mutations*

Potential to act more broadly or be combined with agents targeting specific mutations only



*Preclinical Reference:
Ishii et al., Cancer Research, 2013

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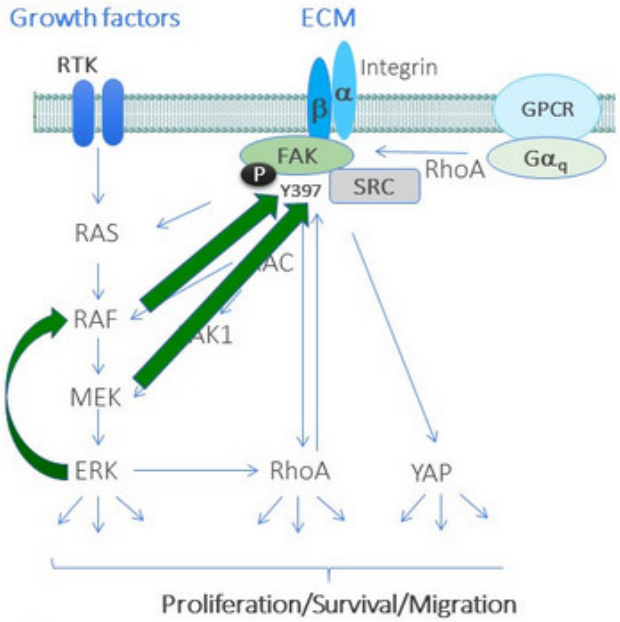
CH5126766 = VS-6766
PD0325901 (mirdametinib) is a conventional MEK inhibitor

More Complete Shutdown requires Addressing Multiple Resistance Mechanisms



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➡ = Feedback Reactivation

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References:
1. Chen, Mol Cancer Res 2018
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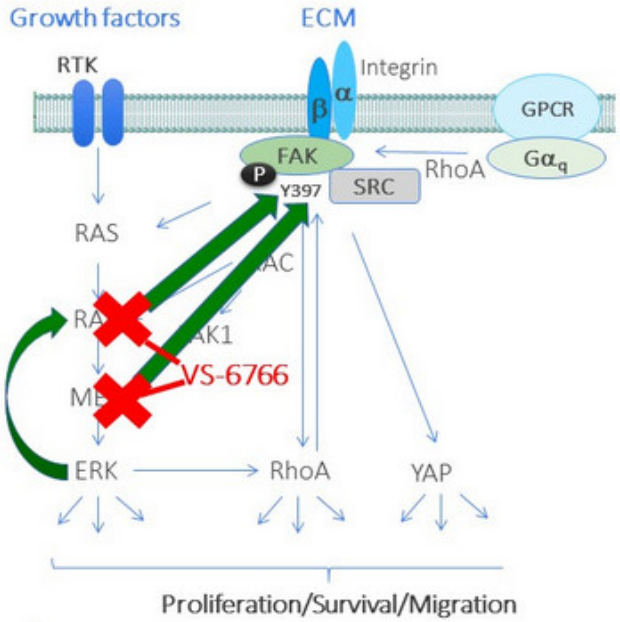

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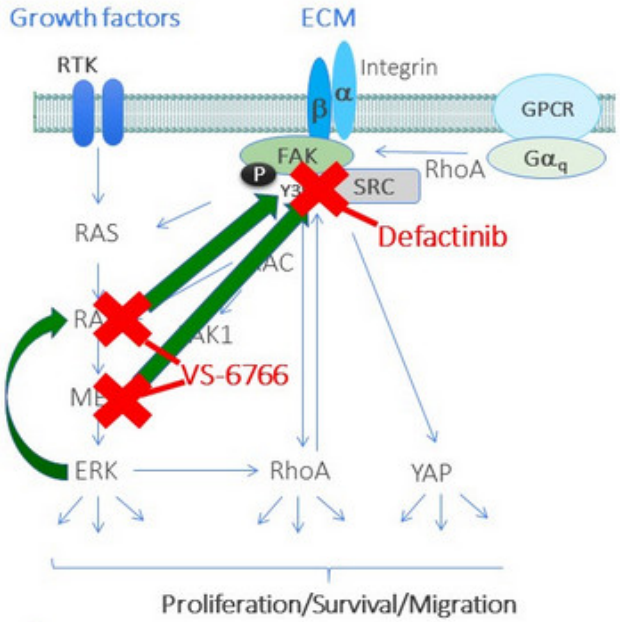

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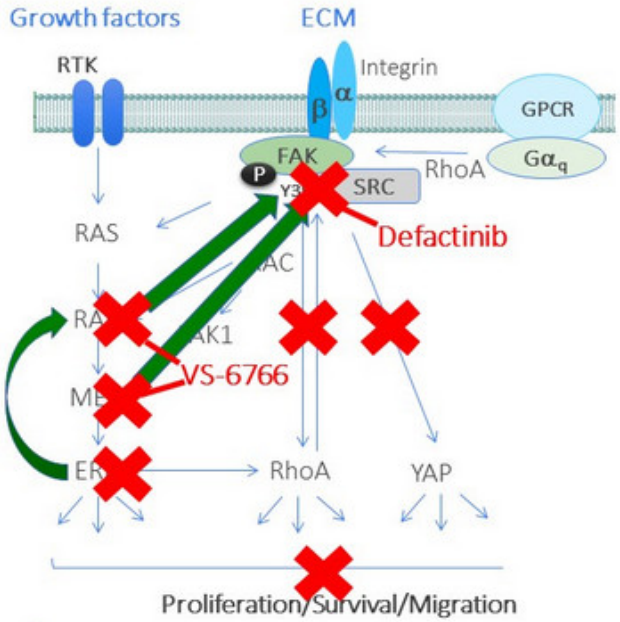

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Background

- In view of promising activity, a different trial design was investigated to mitigate toxicity
- Mean terminal $t_{1/2}$ of ≈ 60 hours
  - 2x-weekly and 3x-weekly scheduling, in 4 week cycles
- Led by the Drug Development Unit at RMH/ICR

Martinez-Garcia et al. ClinCancerRes. 2012 Sep1;18(17):4806-19

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Adverse Events

Adverse event details	Expansion: 4mg 2x weekly n=26		Martinez-Garcia *et al.* CCR 2012 Patient treated at OD MTD n=6
	All grades	≥ Gr. 3	≥ Gr. 3
Rash-related	22 (84.6 %)	5 (**19.2 %**)	3 (**50.0 %**)
CK elevation	15 (57.7 %)	2 (**7.6%**)	1 (**16.7 %**)
Blurred vision	13 (50 %)	0	0
Peripheral oedema	10 (38.5 %)	0	0
Diarrhoea	9 (34.1 %)	1 (3.8 %)	0
Mucositis/Mouth ulcer	8 (30.8 %)	1 (3.8 %)	0
Fatigue	6 (23.1 %)	1 (3.8 %)	0
Nausea	5 (19.2 %)	0	0

Martinez-Garcia et al. Clin Cancer Res. 2012 Sep 1;18(17):4806-19

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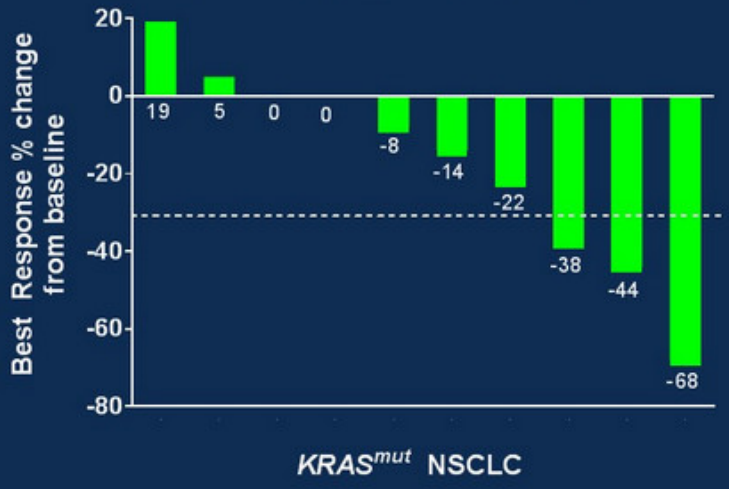
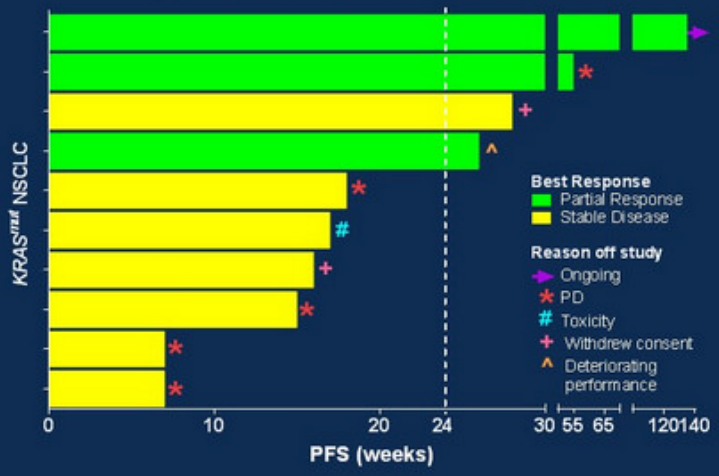
Results: *KRAS*^{mut} NSCLC - Adenocarcinoma

Best response by RECIST v1.1



Progression Free Survival



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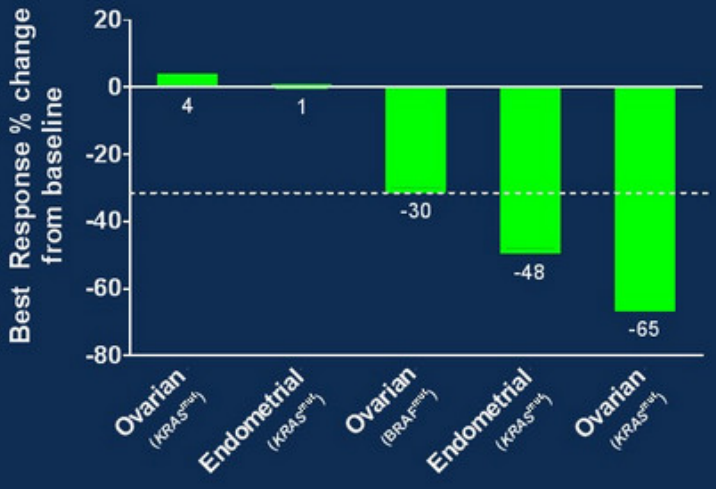
Results: Gynaecological cancers

Best response by RECIST v1.1



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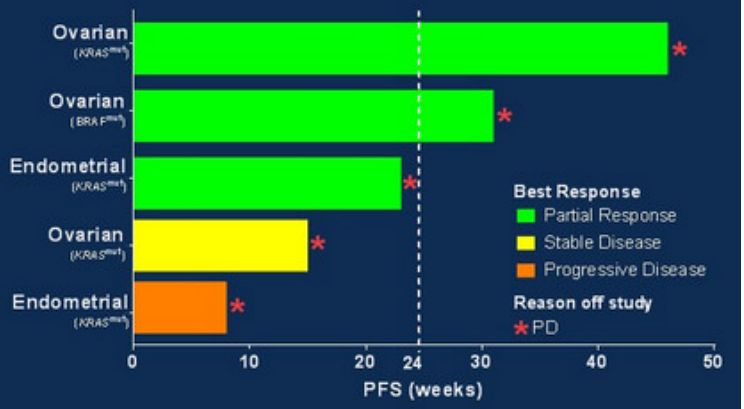

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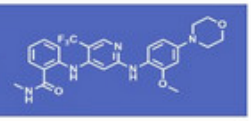
Defactinib: Selective FAK inhibitor

Defactinib 

Focal Adhesion Kinase (FAK)

- Non-receptor tyrosine kinase: Mediates signaling downstream of integrins & growth factor receptors
- Key roles in drug resistance
  - RAF & MEK inhibitors
  - Chemotherapy
- Immuno-Oncology/Tumor Microenvironment
  - FAK inhibition reduces stromal density: ↑ entry of cytotoxic T cells into tumor
  - FAK inhibition reduces immuno-suppressive Tregs, M2 macrophages & MDSCs

Defactinib (VS-6063)

- Selective inhibitor of FAK & related kinase PYK2
- Good pFAK target inhibition in tumors of patients following oral defactinib administration
- Early signs of clinical efficacy
- Studied in 500+ patients with good safety profile observed to date
  - Only ≥Gr 3 toxicity over 2.5% was hyperbilirubinemia – Not associated liver AEs
- Preliminary results show it is generally well-tolerated in combination
  - MEK/RAF, PD-1, Chemo

Reference:
Jones, Invest New Drugs, 2015; Kang, J Natl Cancer Inst. 2013; Diaz Osterman, Elife 2019; Tong, Respiratory Res 2019, Serrels Cell 2015; Jiang et al Nat Med 2016; Banerji, BTOG Dublin, Jan 23, 2019; Data on file

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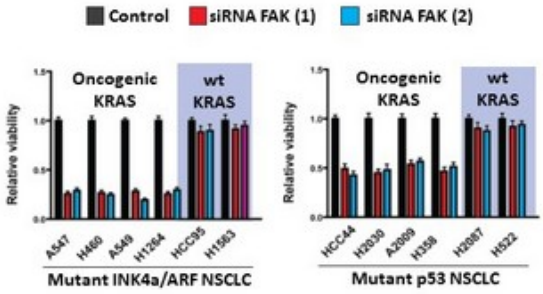
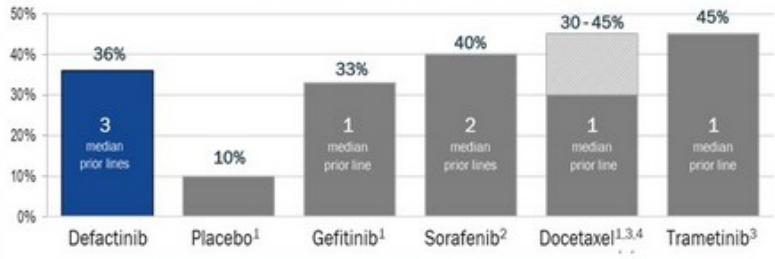
Defactinib Monotherapy Shows Clinical Activity in KRAS Mutant NSCLC

KRAS mt is necessary for sensitivity to FAK inhibition in NSCLC cell lines



Reference:
Konstantinidou G et al. Cancer Discovery 2013;3:444-57

12-week PFS rate of experimental agents for KRAS mt NSCLC



"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; Lung Cancer 2020

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

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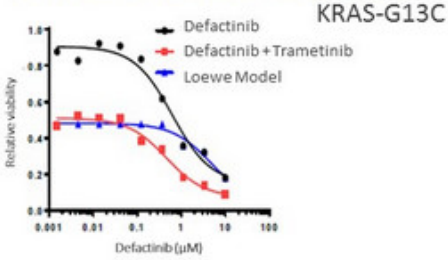
Screen for Synergy with Defactinib Identified MEK Inhibitors (& VS-6766) as Top Hit

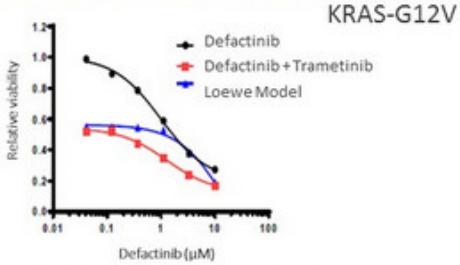
TOV-21G CELLS KRAS-MUTANT OVARIAN CANCER

KRAS-G13C

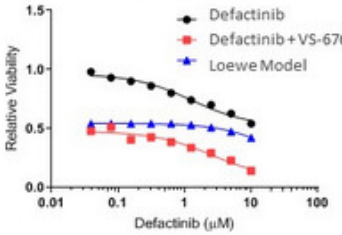


H441 CELLS KRAS-MUTANT NON-SMALL-CELL LUNG CANCER

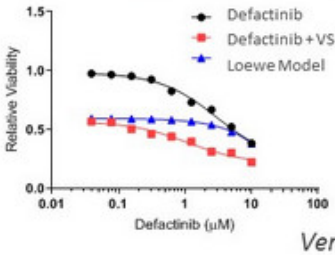
KRAS-G12V



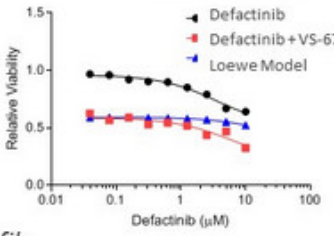
SW982 CELLS SARCOMA BRAF:pV600E



MERO-14 CELLS MESOTHELIOMA



CAL-51 CELLS TRIPLE NEGATIVE BREAST CANCER



Verastem, data on file

Verastem issued patent on FAK/MEK inhibitor combinations

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Phase 1 VS-6766 and Defactinib Combination Data in KRAS Mutant Solid Tumors – Initial Results

Udai Banerji, MBBS, MD, DNB, PhD, FRCP

Verastem Oncology

Ongoing Investigator-Sponsored Basket Study of VS-6766 + Defactinib in KRAS^{M} Cancers

*Refractory to conventional treatment or for which no conventional treatment exists

Dr. Udai Banerji
Royal Marsden Hospital

Phase I

Advanced Solid Cancers

- VS-6766 oral twice wkly x 3 wks every 4 wks
- Defactinib oral BID daily x 3 wks q 4 wks
- 3 cohorts with increasing doses explored
  - Cohort 1: VS-6766 3.2 mg & Defactinib 200 mg
  - Cohort 2a: VS-6766 4 mg & Defactinib 200 mg
  - Cohort 2b: VS-6766 3.2 mg & Defactinib 400 mg

Advanced NSCLC KRAS mutant* (N=20)	*14 enrolled by March 2020* *Median prior lines = 2* *Majority prior PD-(L)1 treatment*
LGSOC* (N=20)	*9 enrolled by March 2020* *Median prior lines ≥2* *Prior MEKi allowed*
Advanced CRC RAS mutant* (N=10)	*10 enrolled by March 2020* *Median prior lines = 2-3* *Prior VEGFi allowed*
Advanced Solid Tumors Enriched for RAS* (Biopsy Amenable) (N=6)	*6 enrolled by March, 2020*

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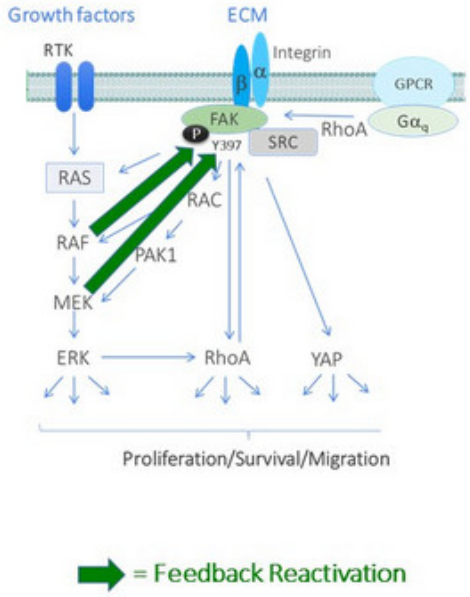
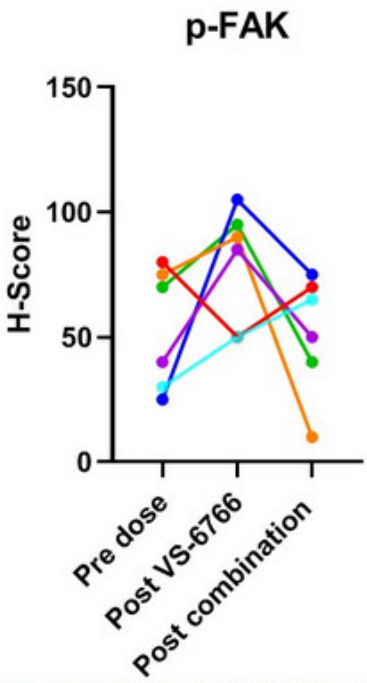
References: Banerji, AACR VM 1, April 27, 2020, CT143; Data on file

Overcoming Key Resistance Mechanisms to MEK Inhibitors





- MEK inhibition induces compensatory activation of pFAK preclinically and clinically
  - Trametinib induced ↑ pFAK (Y397) preclinically in KRAS mt NSCLC cell lines
  - Also observed in patients
    - VS-6766 induced ↑ pFAK (Y397) as a potential resistance mechanism in the majority of patients
    - Combination with defactinib reduced this compensatory pFAK signal

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References:
Banerji, BTOG Dublin, Jan 23, 2019
Banerji, AACR VM 1, April 27, 2020, CT143

VS-6766 3.2 mg + Defactinib 200 mg Selected as RP2D

The ROYAL MARSDEN NHS Foundation Trust

Treatment Related Adverse Events Occurring in ≥ 10 Patients (Total) Q4 2019 Update

	Dose Escalation Phase						Dose Expansion Phase				Total N=46
	VS-6766 3.2mg Def 200mg Cohort 1 n=3		VS-6766 4mg Def 200mg Cohort 2a n=6		VS-6766 3.2mg Def 400mg Cohort 2b n=3		VS-6766 3.2mg Def 200mg Cohort 1 n=17		VS-6766 4mg Def 200mg Cohort 2a n=17		
Adverse Event Details*	Gr1/2	Gr3/4	Gr1/2	Gr3/4	Gr1/2	Gr3/4	Gr1/2	Gr3/4	Gr1/2	Gr3/4	
Rash	2		6		3		16		12	3	42
CK elevation	2		2	1	1		7	1	8	3	25
AST elevation			1		1		5		10	1	18
Hyperbilirubinemia	1	1	1	1	1		7		6		18
Visual disturbance			1		2		5		8		16
ALT elevation			1		1		3		8		13
Diarrhoea	2		1		1		4		5		13
Fatigue			2				3		8		13
Oral Mucositis^							4		6	2	12
Nausea	1		3		2				6		12
Peripheral Edema							4		6		10

- Most Adverse Events (AE) were Grade 1/2
  - All changes were reversible
- No DLTs in Cohort 1 or 2a
- DLTs Cohort 2b: Gr 2 rash in 2/3 of patients; MTD not reached
- Due to chronic Grade 2 AEs in patients on treatment > 6 months

RP2D

- **VS-6766 3.2 mg** oral twice wkly (3 wks of every 4 wks)
- **Defactinib 200 mg** oral BID (3 wks of every 4 wks)

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References: Banerji, AACR VM 1, April 27, 2020, CT143; Data on file

*AEs were graded by NCI CTC v4; highest grade only recorded for each patient; data preliminary and subject to change; ^also includes glossitis/mouth ulcers

Pharmacokinetic Profiles of VS-6766 + Defactinib in Combination Similar to that seen in Single Agent Studies

VS-6766

Cohort	Dose (mg)	N	Subject	$AUC_{0\text{-}24h}$ (h*ng/mL)	C_{max} (ng/mL)
1	3.2 (with 200mg VS)	3	Mean	6179	354
			CV%	32.1	30.4
2a	4 (with 200mg VS)	5	Mean	5353	289
			CV%	15.8	16.0
2b	3.2 (with 400mg VS)	1	FRA101-007	3302	229

Defactinib

Cohort	Dose (mg)	N	Subject	AUClast (h*ng/mL)	Cmax (ng/mL)
1	200 (with 3.2mg RO)	3	Mean	2071	273
			CV%	103	80
2a	200 (with 4mg RO)	5	Mean	2252	318
			CV%	124	117
2b	400 (with 3.2mg RO)	3	Mean	2807	360
			CV%	31	32

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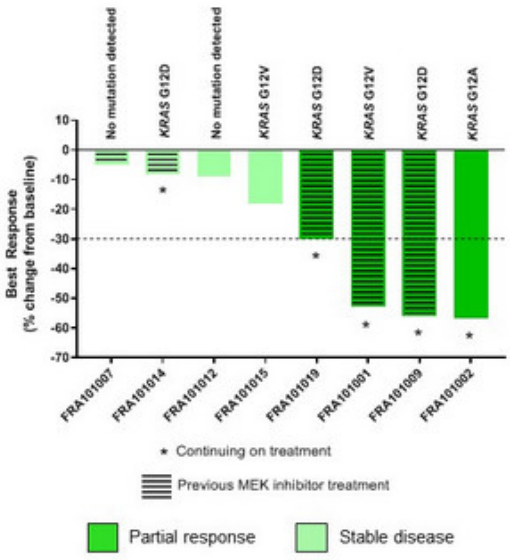
Reference: Banerji, AACR VM 1, April 27, 2020, CT143

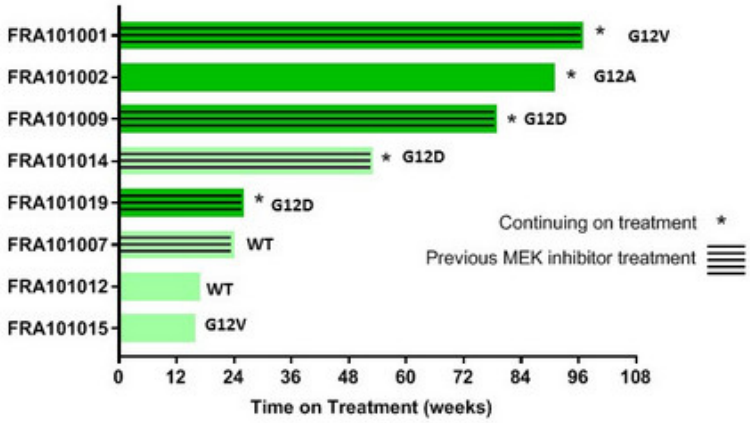
Efficacy – Low Grade Serous Ovarian Cancer

Best response by RECIST



- All PRs confirmed with subsequent scan per RECIST

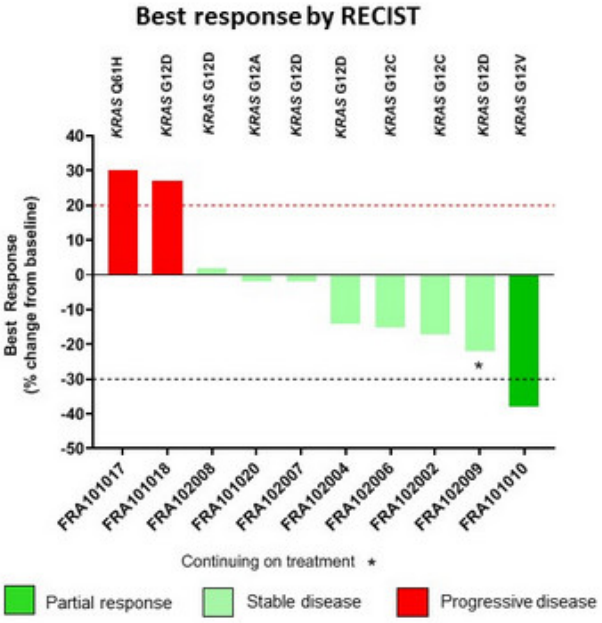
Time on treatment

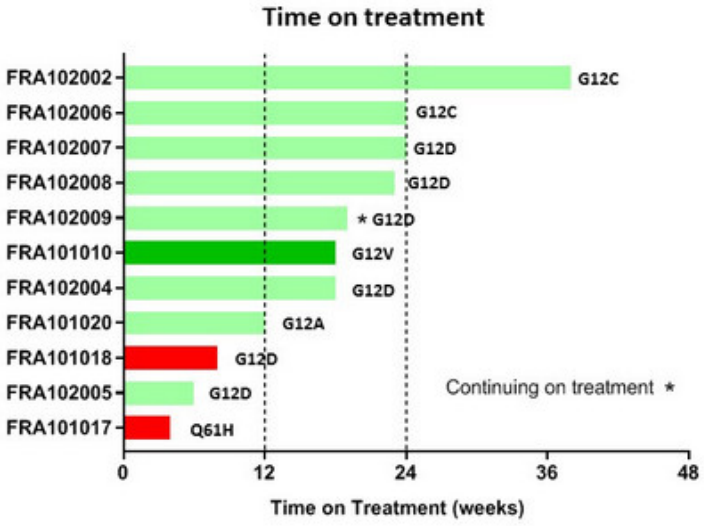


- Response rates: LGSOC $KRAS^M$ = 67% (4/6); All LGSOC = 50% (4/8)
  - Also, 1 patient with KRAS mutant mucinous ovarian cancer had PR (> 60% reduction) with > 1 year on therapy
- ORR for LGSOC in the current literature is <10 % chemotherapy, 13% letrozole, 26% for trametinib, 24% for binimetinib, 15% for selumetinib

References: Annals of Oncology, 10/2019, V30, v897-898; Journal of Clinical Oncology 2015 33:15_suppl, TPS5610; Farley, J. *et al. Lancet Oncol.* (2013); Banerji, AACR VM 1, April 27, 2020, CT143

Efficacy – KRAS mutant NSCLC





- 3 patients received treatment for 24 weeks
- Median time on treatment for this cohort was approximately 18 weeks (range 4-38 weeks)

14/20 pts enrolled in KRAS mt NSCLC cohort;
1 additional confirmed PR in KRAS-G12V mutant patient

Reference: Banerji, AACR VM 1, April 27, 2020, CT143

Summary: VS-6766 + Defactinib

Rationale

- VS-6766 & defactinib have shown single agent clinical activity in KRAS mt cancer
- RAS pathway blockage activates FAK as potential resistance mechanism preclinically & clinically
- FAKi and MEKi are synergistic in reducing viability of cancer cell lines *in vitro* & *in vivo* in multiple models

FRAME study shows promising results & continues to enroll

- Most adverse events were grade 1 / 2 with the Intermittent dosing of VS-6766 + defactinib (no PK interaction observed)
- VS-6766 + defactinib combination shows clinical promise in heavily pre-treated refractory patients with KRAS mt disease
  - 67% ORR in KRAS mt LGSOC, including patients progressing on prior MEK inhibitors
  - High rate of disease control and tumor regression in NSCLC with several patients out to 24 weeks
  - The study continues to enroll with additional responses in LGSOC, NSCLC and colorectal since Nov cut off

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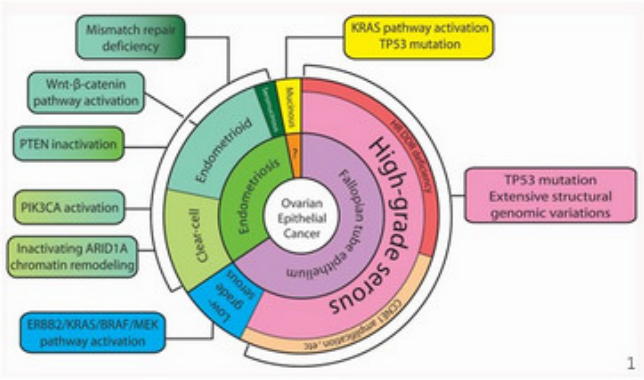
Next Steps and Closing Remarks

Dan Paterson / Brian Stuglik

Verastem Oncology

LGSOC – Strong Proof of Concept, High Unmet Need



Am J Pathol. 2016 Apr;186(4):733-47

	Incidence	10 y Prevalence
Worldwide	~13,000	~80,000
US	~1,000	~6,000

*Based on LGSOC representing 5% of epithelial ovarian cancer

- LGSOC represents ~4-10% of epithelial ovarian cancer[2]
- Long survival results in high prevalence rate
- RAS pathway mt frequency 50%[3]
- No FDA-approved therapy

Combination of VS-6766 + Defactinib offers potential for:

- Long duration of therapy
- High market share
- Speed to market opportunity
- Two product revenue streams

In LGSOC, G12V & G12D are the dominant KRAS mutations, and G12V confers a more aggressive phenotype (Tsang et al., J. Pathol 231: 449, 2013)

[1] http://www.gynecologycancer.org/content
[2] SEER data, 2011-2016
[3] http://molecularcasestudies.cshlp.org/content/3/6/a004341.full

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Clinical Activity in Discrete KRAS Codon 12 Variants (G12V, G12D)

Summary: VS-6766 + Defactinib in KRAS mt ovarian & lung cancers

Tumor Type		G12V	G12D	G12A	G12C	Q61H	WT
Ovarian	# patients	3	3	1	0	0	2
	PR	2 (67%)	2 (67%)	1 (100%)			0 (0%)
	Disease Control	3 (100%)	3 (100%)	1 (100%)			2 (100%)
	≥6 months time on therapy	2 (67%)	2 (67%)	1(100%)			0 (0%)
Lung	# patients	1	6	1	2	1	0
	PR	1 (100%)	1 (17%)*	0 (0%)	0 (0%)	0 (0%)	
	Disease Control	1 (100%)	4 (67%)	1 (100%)	2 (100%)	0 (0%)	
	≥3 months time on therapy	1 (100%)	4 (67%)	1 (100%)	2 (100%)	0 (0%)	

*22% reduction & still on treatment

November 2019 data cut

Includes 1 patient with KRAS-G12V mt mucinous ovarian cancer

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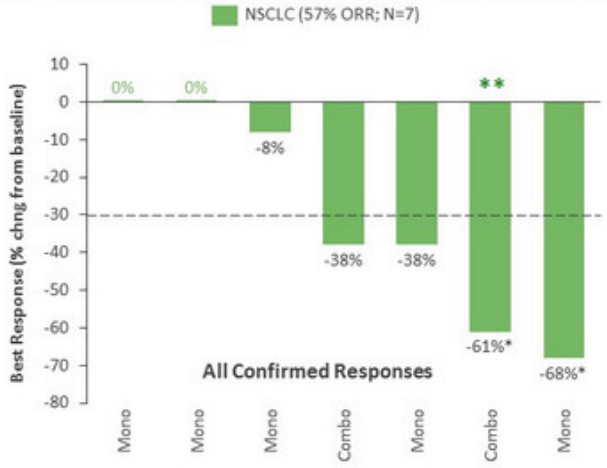
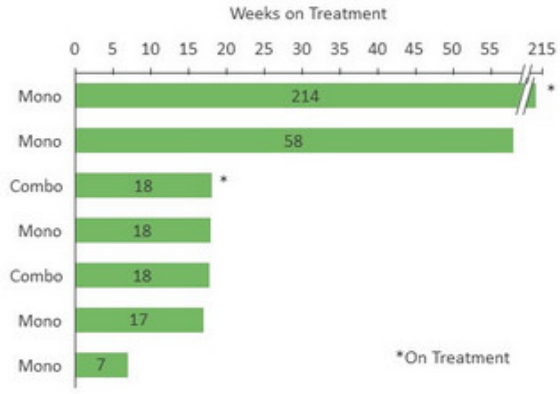
VS-6766 ± Defactinib has a Confirmed 57% ORR in KRAS^{G12V} NSCLC

Best Response by RECIST in KRAS^{G12V} NSCLC



Time on Treatment for KRAS^{G12V} NSCLC



**March 2020

- All PRs confirmed with subsequent scan per RECIST

Mono = VS-6766 Monotherapy[1]
Combo = VS-6766 + Defactinib

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Source: (1) Martinez-Garcia, M. *et al. Clin. Cancer Res.* (2012)

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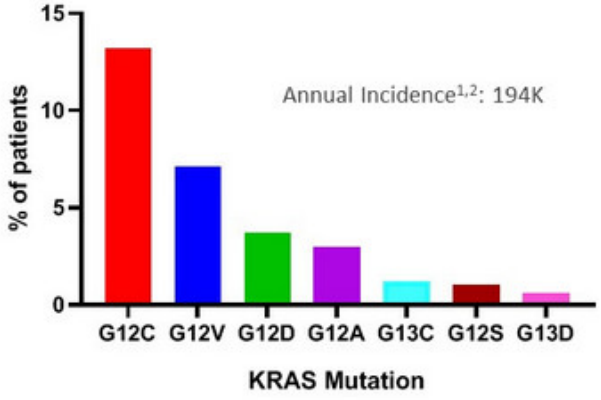
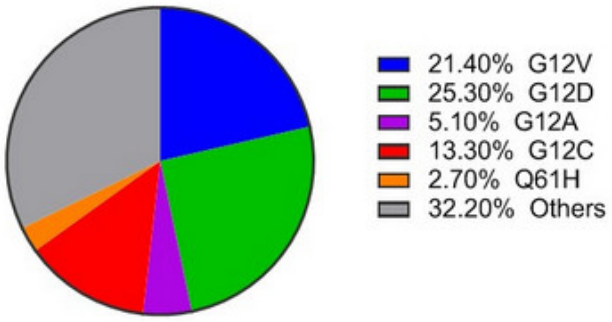
KRAS G12V Represents a Large Opportunity in NSCLC and across Tumors

NSCLC Adenocarcinoma[3]



% Frequency in Total of 780 Cancer Patients with KRAS mts[3]



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[1] 85% of lung cancer is NSCLC (Lu et. al. Cancer Manag Res. 2019)
[2] Cancer Statistics 2020, Siegel et. al. CA Cancer J Clin 2020;70:7-30
[3] TCGA PanCancer Atlas (cBioPortal analysis)

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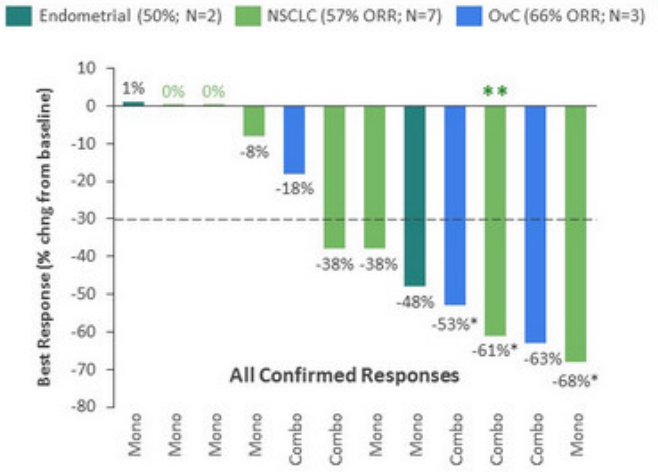
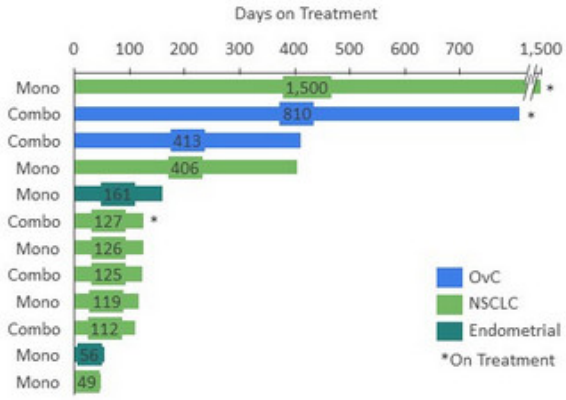
VS-6766 ± Defactinib has a Confirmed 58% ORR in KRAS^{G12V} Tumors

Best Response by RECIST in KRAS^{G12V} Tumors



Time on Treatment for KRAS^{G12V} Tumors



**March 2020

- All PRs confirmed with subsequent scan per RECIST

Mono = VS-6766 Monotherapy[1]
Combo = VS-6766 + Defactinib

PROPERTY OF VERASTEM, INC.

Source: (1) Martinez-Garcia, M. *et al. Clin. Cancer Res.* (2012)

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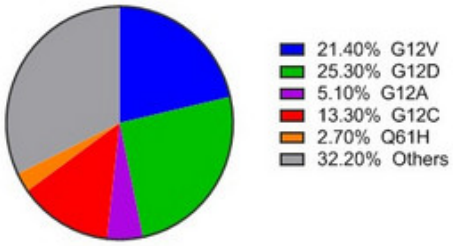
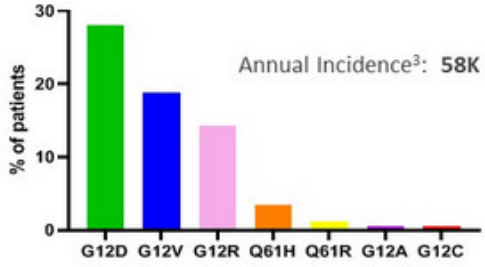
KRAS G12V and G12D Represent ~50% of KRAS Mutations across Human Cancers

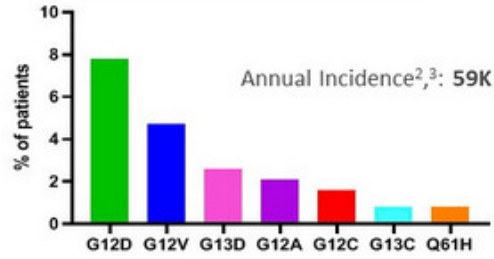
% frequency in a total of 780 cancer patients with KRAS mutations[1]



Pancreatic Adenocarcinoma[1]



Uterine Endometrioid Carcinoma[1]



[1] TCGA PanCancer Atlas (cBioPortal analysis)
[2] 90% of all uterine cancers are of the endometrial type (ACS)
[3] Cancer Statistics 2020 (Siegel et al. CA Cancer J Clin 2020; 70:7-30)

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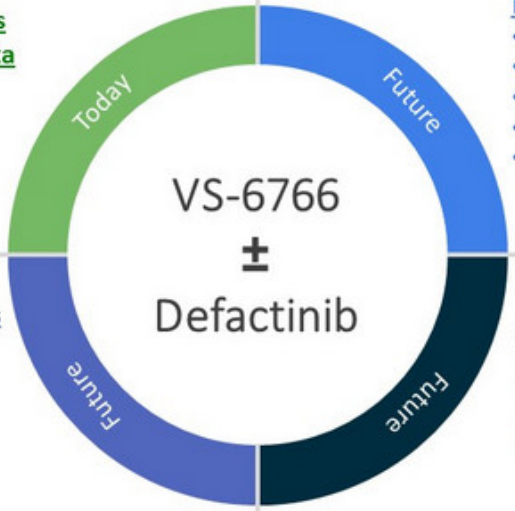
Focusing on High Priority Indications with Significant Opportunities for Growth

High Priority Indications Supported by Initial Data

- LGSOC[1,2]
- KRAS-G12V NSCLC[1,2]
- Pancreatic[1,2]

Expansion Opportunities

- Additional G12V & G12D mt cancers[1]
- Uveal Melanoma[2]
- BRAF mt melanoma[1,2]
- BRAF mt colorectal
- BRAF mt prostate[2]



Other Mutation Opportunities

- GNAQ mutations in uveal melanoma[2]
- NF1 mutations in melanoma
- MAP3K1 mutations in breast cancer

Other Combinations

- KRAS G12C inhibitors
- EGFR inhibitors
- Everolimus[2]
- Anti-PD-1[1,2]

[1] Supported by clinical data
[2] Supported by preclinical data

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Strong Patent Protection for VS-6766 ± Defactinib

- COM for VS-6766 to 2027 & defactinib to 2028, Hatch Waxman should extend to 2032
- VS-6766 intermittent dosing regimen until 2038 if granted
- FAK/MEK combination to 2035
- VS-6766/defactinib combination until 2040 if granted
- Method of manufacture for VS-6766 to 2032
- Other activity related to patent protection is ongoing and will continue into the future

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Potential Blockbuster Opportunity with VS-6766 + Defactinib

Key mechanistic attributes

- Potential Best in class RAF/MEK & FAK inhibitors
- More complete RAS pathway shut down addressing key resistance mechanisms
- Uniquely targeting CRAF to shut down KRAS-G12V

Significant commercial potential

- First in class approach to KRAS-G12V & G12D
- No approved therapies in LGSOC
- 30% of all human cancers driven by RAS family mutations

Early clinical experience

- All-oral combination regimen with non-overlapping safety profile
- Initial clinical data with the combination are encouraging including both objective response rate and durability
- KRAS-G12V mutant cancers appear to be particularly responsive to VS-6766 ± defactinib

Next steps

- Goal to initiate LGSOC registration-directed study in 2020
- Complete expansion cohorts in ongoing investigator initiated Phase 1 combination study
- KRAS-G12V & G12D expansion cohorts in NSCLC & pancreatic
- Explore BRAFm-driven indications
- Combinations with KRAS-G12Ci & anti-PD-1

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Oncology
Q&A

Exhibit 99.2

Verastem Oncology Announces Preliminary Data from Investigator-initiated Study Highlighting Clinical Activity of RAF/MEK and FAK Combination in KRAS Mutant Tumors Presented at the American Association for Cancer Research 2020 Virtual Annual Meeting I

Combination of VS-6766 and Defactinib Demonstrates 67% (4/6 Patients) Overall Response Rate in KRAS Mutant Low-Grade Serous Ovarian Cancer in Phase 1 Trial

Subsequent Combined Analysis (VS-6766 Monotherapy and Defactinib Combination) Demonstrates 57% (4/7 Patients) Overall Response Rate in KRASG12V Non-Small Cell Lung Cancer

Management to Host Investor Conference Call Today at 8:00 AM ET

BOSTON--(BUSINESS WIRE)--Apr. 27, 2020-- Verastem, Inc. (Nasdaq:VSTM) (also known as Verastem Oncology), a biopharmaceutical company committed to developing and commercializing new medicines for patients battling cancer, today announced results from the ongoing investigator- initiated Phase 1 clinical study investigating VS-6766, its RAF/MEK inhibitor, in combination with defactinib, its FAK inhibitor, in patients with KRAS mutant advanced solid tumors. The data will be presented as a virtual poster today at the American Association for Cancer Research (AACR) 2020 Virtual Annual Meeting I.

This ongoing study is an open label, dose escalation and expansion study. The expansion cohorts are currently ongoing in patients with advanced solid tumors, including low-grade serous ovarian cancer (LGSOC), KRAS mutant non-small cell lung cancer (NSCLC) and KRAS mutant colorectal cancer (CRC). In the LGSOC cohort, among the patients with KRAS mutant tumors (n=6), 4 patients responded, for an overall response rate (ORR) of 67%. Median time on treatment was 20.5 months. In the KRAS mutant NSCLC cohort (n=10), 1 patient achieved a partial response and 8 patients achieved disease control. In this cohort, 70% of patients continued on treatment at least 12 weeks and 30% of patients continued on treatment at least 24 weeks.

Based on an observation of higher response rates seen in patients with KRASG12V mutations in the investigator-initiated Phase 1 combination study, we conducted a combined analysis with data from the combination study and the prior single-agent study that utilized a twice-weekly dosing schedule of VS-67661 to get a more complete picture of activity in KRASG12V mutations. The subsequent, combined analysis (VS-6766 monotherapy and defactinib combination) showed a 57% ORR (4/7 patients); as a single agent (2/5 patients) and in combination with defactinib (2/2 patients) in KRASG12V mutant NSCLC. Similarly, the combined analysis showed a 60% ORR (3/5 patients); as a single agent (1/2 patients) and in combination with defactinib (2/3 patients) in KRASG12V mutant gynecologic cancers. These additional analyses were conducted by Verastem Oncology to understand the impact that various KRAS variants may have had on response to identify potential signals to pursue in future prospective studies. This additional analysis was not part of the AACR 2020 poster presentation.

“Earlier research has demonstrated MEK inhibitors can cause upregulation of FAK in KRAS mutant tumors, which are notoriously difficult to treat and quite common across solid tumors. The combination of a RAF/MEK and FAK inhibitor can potentially overcome this challenge and opens up an exciting new pathway for treatment,” stated Professor Udai Banerji, Professor of Molecular Cancer Pharmacology at The Institute of Cancer Research, London, and Honorary Consultant in Medical Oncology, MBBS, MD, DNB, PhD, FRCP at The Royal Marsden NHS Foundation Trust, London, and lead investigator of the clinical study. “We found that the combination of VS-6766 and defactinib in low-grade serous ovarian cancer (LGSOC) was well tolerated by the patients in the trial and shows promising clinical activity, including durable response that is associated with clinically meaningful benefit. The study continues to enroll additional patients into the ovarian, lung and colorectal expansion cohorts with additional responses seen in all cohorts.”

“We are encouraged by these early response rates in KRAS mutant LGSOC and in KRAS G12V mutant tumors as they underscore the significant potential of this novel approach in areas of high unmet medical need,” said Brian Stuglik, Chief Executive Officer of Verastem Oncology. “The potential of the combination of VS-6766 and defactinib is rapidly evolving as we continue to gain more insights and analyze the data. We plan to initiate discussions with regulatory authorities as soon as possible to define a path forward, with the goal of commencing a registration-directed clinical trial during 2020.”

Initial Results from the Phase 1 Study Investigating the Combination of VS-6766 and Defactinib in Patients with KRAS Mutant Cancers and Subsequent Analyses

The poster presentation describes safety and dose response data from the dose-escalation portion and expansion cohorts from an open-label, investigator-initiated Phase 1 study conducted in the United Kingdom assessing the combination of RAF/MEK and FAK inhibitor therapy in patients with LGSOC and KRAS mutant NSCLC. The study evaluated the combination of VS-6766 and defactinib. VS-6766 was administered using a twice- weekly dose escalation schedule and was administered 3 out of every 4 weeks. Defactinib was administered using a twice-daily dose escalation schedule, also 3 out of every 4 weeks. Dose levels were assessed in 3 cohorts: cohort 1 (VS-6766 3.2mg, defactinib 200mg); cohort 2a (VS-6766 4mg, defactinib 200mg); and cohort 2b (VS-6766 3.2mg, defactinib 400mg).

In the patients with LGSOC (n=8), the ORR was 50% (n=4). Among the patients with KRAS mutant LGSOC (n=6), the ORR was 67% (n=4). Of the 4 patients who have responded, 3 had a prior MEK inhibitor and as of November 2019 had been on study for a median of 20.5 months (range 7-23 months). In the patients with NSCLC (n=10), all of which had KRAS mutations, 1 patient achieved a partial response and 1 patient with a 22% tumor reduction still on treatment as of November 2019. Median time on treatment for this cohort was approximately 18 weeks.

Based on an observation of higher response rates seen in patients with KRASG12V mutations in the investigator-initiated Phase 1 combination study, we conducted a combined analysis with data from the combination study and the prior single-agent study that utilized a twice-weekly dosing schedule of VS-67661 to get a more complete picture of activity in KRASG12V mutations. The subsequent, combined analysis (VS-6766 monotherapy and defactinib combination) showed a 57% ORR (4/7 patients); as a single agent (2/5 patients) and in combination with defactinib (2/2 patients) in KRASG12V mutant NSCLC. Similarly, the combined analysis showed a 60% ORR (3/5 patients); as a single agent (1/2 patients) and in combination with defactinib (2/3 patients) in KRASG12V mutant gynecologic cancers. These additional analyses were conducted by Verastem Oncology to understand the impact that various KRAS variants may have had on response to identify potential signals to pursue in future prospective studies. This additional analysis was not part of the AACR 2020 poster presentation.

The most common side effects seen in the Phase 1 study were rash, creatine kinase elevation, nausea, hyperbilirubinemia and diarrhea, most being NCI CTC Grade 1/2 and all were reversible. The recommended Phase 2 dose was determined to be cohort 1 (VS-6766 3.2mg, defactinib 200mg).

The preliminary data reported in the study suggest that a novel intermittent dosing schedule of RAF/MEK and FAK inhibitor combination therapy has promising clinical activity in patients with KRAS mutant LGSOC and KRASG12V mutant NSCLC, including patients previously treated with a MEK inhibitor. Expansion cohorts remain ongoing.

Details for the AACR 2020 Virtual Meeting I presentation are as follows:

Title: Phase 1 study of the combination of a RAF-MEK inhibitor CH5126766 (VS-6766) and FAK inhibitor defactinib in an intermittent dosing schedule with expansions in KRAS mutant cancers
Lead author: Udai Banerji, The Institute of Cancer Research and The Royal Marsden
Poster #: CT143
Session: VPO.CT01 - Phase I Clinical Trials
Date and Time: Monday, April 27, 2020; 9:00 a.m. to 6:00 p.m. ET
URL: https://www.abstractsonline.com/pp8/#!/9045/presentation/10642

Conference Call and Webcast Information

The Verastem Oncology management team will host a conference call and webcast on Monday, April 27, 2020, at 8:00 AM ET to discuss the Phase 1 RAF/MEK/FAK combination data. The call can be accessed by dialing (877) 341-5660 (U.S. and Canada) or (315) 625-3226 (international), five minutes prior to the start of the call and providing the passcode 8390795.

The live, listen-only webcast of the conference call can be accessed by visiting the investors section of the Company's website at www.verastem.com. A replay of the webcast will be archived on the Company's website for 90 days following the call.

About VS-6766

VS-6766 (formerly known as CH5126766, CKI27 and RO5126766) is a unique inhibitor of the RAF/MEK signaling pathway. In contrast to other MEK inhibitors in development, VS-6766 blocks both MEK kinase activity and the ability of RAF to phosphorylate MEK. This unique mechanism allows VS-6766 to block MEK signaling without the compensatory activation of MEK that appears to limit the efficacy of other inhibitors. The combination of VS-6766 and the focal adhesion kinase (FAK) inhibitor defactinib is currently being investigated in an investigator-initiated Phase 1 dose escalation and expansion study. The expansion cohorts are currently ongoing in patients with KRAS mutant advanced solid tumors, including low grade serous ovarian cancer (LGSOC), non-small cell lung cancer (NSCLC) and colorectal cancer (CRC).2 The ongoing clinical study of the VS-6766/defactinib combination is supported by single-agent Phase 2 studies which investigated defactinib in KRAS mutant NSCLC3 and VS-6766 in KRAS mutant NSCLC and LGSOC.1

About Defactinib

Defactinib is an oral small molecule inhibitor of FAK and PYK2 that is currently being evaluated as a potential combination therapy for various solid tumors. The Company has received Orphan Drug designation for defactinib in ovarian cancer and mesothelioma in the US, EU and Australia.

Preclinical research by Verastem Oncology scientists and collaborators at world-renowned research institutions has described the effect of FAK inhibition to enhance immune response by decreasing immuno-suppressive cells, increasing cytotoxic T cells, and reducing stromal density, which allows tumor-killing immune cells to enter the tumor.4,5 Additionally, in both preclinical and clinical studies, FAK activation has been shown to occur as a potential resistance mechanism in response to MEK inhibitor treatment, and synergy of a FAK inhibitor with a RAF/MEK inhibitor has been shown in several preclinical models. The combination of defactinib and VS-6766 is currently being investigated in an investigator-initiated Phase 1 dose escalation and expansion study. The expansion cohorts are currently ongoing in patients with KRAS mutant advanced solid tumors, including low grade serous ovarian cancer (LGSOC), non-small cell lung cancer (NSCLC) and colorectal cancer (CRC).2 The ongoing clinical study of the VS-6766/defactinib combination is supported by single-agent Phase 2 studies which investigated defactinib in KRAS mutant NSCLC3 and VS-6766 in KRAS mutant NSCLC and LGSOC.4 Defactinib is also in clinical testing in combination with pembrolizumab for treatment of patients with pancreatic cancer, NSCLC and mesothelioma.6

About Verastem Oncology

Verastem Oncology (Nasdaq: VSTM) is a commercial biopharmaceutical company committed to the development and commercialization of new medicines to improve the lives of patients diagnosed with cancer. Our pipeline is focused on novel small molecule drugs that inhibit critical signaling pathways in cancer that promote cancer cell survival and tumor growth, including phosphoinositide 3-kinase (PI3K), focal adhesion kinase (FAK) and RAF/MEK inhibition.

Our first FDA approved product is available for the treatment of patients with certain types of indolent non-Hodgkin’s lymphoma (iNHL).

For more information, please visit www.verastem.com.

Forward-Looking Statements Notice

This press release includes forward-looking statements about Verastem Oncology's strategy, future plans and prospects, including statements related to the potential clinical value of the RAF/MEK/FAK combination and the timing of commencing a registration-directed trial for the RAF/MEK/FAK combination. The words "anticipate," "believe," "estimate," "expect," "intend," "may," "plan," "predict," "project," "target," "potential," "will," "would," "could," "should," "continue," "can," "promising" 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 and uncertainties, among other things, regarding: the success in the development and potential commercialization of our product candidates, including defactinib in combination with VS-6766 (; the occurrence of adverse safety events and/or unexpected concerns that may arise from additional data or analysis or result in unmanageable safety profiles as compared to their levels of efficacy; our ability to obtain, maintain and enforce patent and other intellectual property protection for our product candidates; the scope, timing, and outcome of any legal proceedings; decisions by regulatory authorities regarding labeling and other matters that could affect the availability or commercial potential of our product candidates; whether preclinical testing of our product candidates and preliminary or interim data from clinical trials will be predictive of the results or success of ongoing or later clinical trials; that the timing, scope and rate of reimbursement for our product candidates is uncertain; that third-party payors (including government agencies) may not reimburse; that there may be competitive developments affecting our product candidates; that data may not be available when expected; that enrollment of clinical trials may take longer than expected; that our product candidates will experience manufacturing or supply interruptions or failures; that we will be unable to successfully initiate or complete the clinical development and eventual commercialization of our product candidates; that the development and commercialization of our product candidates will take longer or cost more than planned; that we or Chugai Pharmaceutical Co., Ltd. will fail to fully perform under the VS-6766 (CH5126766) license agreement; that we may not have sufficient cash to fund our contemplated operations; that we may be unable to make additional draws under our debt facility or obtain adequate financing in the future through product licensing, co-promotional arrangements, public or private equity, debt financing or otherwise; that we will be unable to execute on our partnering strategies for defactinib in combination with VS-6766; that we will not pursue or submit regulatory filings for our product candidates, and that our product candidates will not receive regulatory approval, become commercially successful products, or result in new treatment options being offered to patients.

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, 2019 as filed with the Securities and Exchange Commission (SEC) on March 11, 2020 and in any subsequent filings with the SEC. The forward-looking statements contained in this press release reflect Verastem Oncology's views as of the date hereof, and the Company does not assume and specifically disclaims any obligation to update any forward-looking statements whether as a result of new information, future events or otherwise, except as required by law.

References

[1] Chénard-Poirier, M. et al. Results from the biomarker-driven basket trial of RO5126766 (CH5127566), a potent RAF/MEK inhibitor, in RAS- or RAF-mutated malignancies including multiple myeloma. Journal of Clinical Oncology 2017: 35. 10.1200/JCO.2017.35.15_suppl.2506.

[2] https://clinicaltrials.gov, NCT03875820

[3] Gerber D. et al. Phase 2 study of the focal adhesion kinase inhibitor defactinib (VS-6063) in previously treated advanced KRAS mutant non-small cell lung cancer. Lung Cancer 2020: 139:60-67.

[4] Jiang H et al. Targeting focal adhesion kinase renders pancreatic cancers responsive to checkpoint immunotherapy. Nat Med 2016: Aug 22(8) 851-60.

[5] Sulzmaier F.J. et al. FAK in cancer: mechanistic findings and clinical applications. Nature Rev Cancer. 2014 14: 598-610.

[6] www.clinicaltrials.gov, NCT02758587

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