Safe Harbor Statement

This presentation includes forward-looking statements about, among other things, Verastem Oncology’s programs and product candidates, including anticipated regulatory submissions, approvals, performance and potential benefits of Verastem Oncology’s 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. 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; or our ability to obtain, maintain and enforce patent and other intellectual property protection for our product candidates.

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, 2020 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.
# Verastem Oncology
## Well Positioned to Capitalize on Growth Opportunities

<table>
<thead>
<tr>
<th><strong>New lead clinical program has best-in-class potential</strong></th>
<th>VS-6766 (RAF/MEKi) and defactinib (FAKi) are clinically active against RAS mutant cancers</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rapid development paths to market</strong></td>
<td>FDA Breakthrough Therapy Designation in LGSOC; Supported by clinical results achieved in low-grade serous ovarian cancer (LGSOC), strong signal in KRAS G12V mutant NSCLC; registration-directed trials initiated in 4Q 2020</td>
</tr>
<tr>
<td><strong>Significant downstream market opportunity and blockbuster potential</strong></td>
<td>30% of all human cancers are driven by mutations in RAS: VS-6766 combinations potentially broadly applicable across a variety of tumor types. <strong>Clinical collaboration with Amgen</strong> evaluating combination with sotorasib in KRAS G12C mutant NSCLC supported by strong pre-clinical rationale</td>
</tr>
<tr>
<td><strong>Strong balance sheet</strong></td>
<td>Monetization of COPIKTRA® (duvelisib) provides funding of current programs until at least 2024</td>
</tr>
</tbody>
</table>

- Cash Balance of $103.4 million, as of September 30, 2021
- Debt reduced from approx. $185M to $0M (2019-2021)
- Annual operating expense forecast of approximately $55-60 million
Verastem Oncology Strategic Transformation

Q1 2020: In-licensed global rights to VS-6766, best-in-class RAF/MEK inhibitor, from Chugai PIPE financing based on data for new clinical program

Q3 2020: Divested global rights to Copiktra to Secura Bio

Q4 2020: Initiated registration-directed ph. 2 study in LGSOC
Initiated registration-directed ph. 2 study in NSCLC

Q1 2021: LGSOC study updated to include KRAS wild type patients

Q2 2021: FDA Breakthrough Therapy Designation granted for VS-6766 + Defactinib in LGSOC

Q3 2021: Remaining outstanding debt retired
VS-6766 + sotorasib Collaboration agreement with Amgen
VS-6766 RAF/MEK Inhibitor Program Overview
VS-6766 is a differentiated, best-in-class asset potentially applicable across multiple patient populations

- Unique dual RAF/MEK targeting mechanism of action
- Best-in-class safety & tolerability profile relative to marketed MEK inhibitors, with potential for combinability with agents from multiple target classes
- Novel intermittent dosing schedule; convenient oral regimen
- Promising signals of clinical activity in various RAS-driven cancers, including in patients whose tumors previously progressed on other MEK inhibitors
- Preclinical anti-proliferative activity across multiple MAPK pathway alterations (e.g. KRAS, NRAS, BRAF, NF1 mt) and multiple solid tumor indications
- Strong preclinical combination data with other agents targeting RAS pathway and parallel pathways
High Priority Lead Indications with Multiple Growth Opportunities

High Priority Registration Indications
Registration-Directed Trials Initiated in 4Q20
- LGSOC\textsuperscript{1,2}
- KRAS\textsuperscript{G12V} mt NSCLC\textsuperscript{1,2}

RAS Pathway Dependent Cancers
- Gynecological\textsuperscript{1,2}
- NSCLC\textsuperscript{1,2}
- Colorectal\textsuperscript{1,2}
- Melanoma\textsuperscript{1,2}
- Pancreatic\textsuperscript{2}

Signal Finding
- VS-6766 + G12Ci KRAS\textsuperscript{G12C} mt NSCLC\textsuperscript{2}
- Pancreatic\textsuperscript{1,2} (10 pt cohort initiated)
- KRAS mt endometrioid\textsuperscript{1} (10 pts initiated)
- Uveal Melanoma\textsuperscript{2} (IST initiated)
- VS-6766 + Everolimus KRAS mt NSCLC\textsuperscript{1,2}

Rational Combinations
- Anti-EGFR\textsuperscript{2}
- SOS1 or SHP2 inhibitor\textsuperscript{2}
- CDK4/6 inhibitor\textsuperscript{2}
- Anti-PD-1\textsuperscript{1,2}
- G12Ci\textsuperscript{1,2}
- Everolimus\textsuperscript{1,2}

Biomarker Selection
- KRAS mt\textsuperscript{1,2}
- BRAF mt (V600 & non-V600)\textsuperscript{1,2}
- NRAS mt\textsuperscript{1,2}
- CRAF mt/fusions\textsuperscript{2}

\textsuperscript{1} Supported by clinical data
\textsuperscript{2} Supported by preclinical data
Robust Pipeline Targeting the RAS Pathway and Multiple Growth Opportunities

### VS-6766 + DEFACTINIB

<table>
<thead>
<tr>
<th>Study/Combinations</th>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Market</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAMP-201&lt;sup&gt;1&lt;/sup&gt;</td>
<td>KRAS mt/wt LGSOC</td>
<td>✔️</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
</tr>
<tr>
<td>RAMP-202&lt;sup&gt;1&lt;/sup&gt;</td>
<td>KRAS mt G12V NSCLC</td>
<td>✔️</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
</tr>
<tr>
<td>FRAME study</td>
<td>Advanced LGSOC</td>
<td>✔️</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
</tr>
<tr>
<td>FRAME study</td>
<td>Advanced KRAS mt NSCLC</td>
<td>✔️</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
</tr>
<tr>
<td>FRAME study</td>
<td>Advanced CRC</td>
<td>✔️</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
</tr>
<tr>
<td>FRAME study</td>
<td>Advanced KRAS-G12V mt NSCLC</td>
<td>✔️</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
</tr>
<tr>
<td>FRAME study</td>
<td>Advanced pancreatic cancer</td>
<td>✔️</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
</tr>
<tr>
<td>FRAME study</td>
<td>Advanced KRAS mt endometrioid cancer</td>
<td>✔️</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
</tr>
<tr>
<td>Metastatic uveal melanoma</td>
<td>✔️</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
<td></td>
</tr>
</tbody>
</table>

*Pre-clinical studies ongoing in multiple KRAS mutant tumors

FDA Breakthrough Therapy Designation for VS-6766 + defactinib

### VS-6766 + OTHER COMBINATIONS

<table>
<thead>
<tr>
<th>Study/Combinations</th>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Market</th>
</tr>
</thead>
<tbody>
<tr>
<td>KRAS mt NSCLC</td>
<td>VS-6766 + everolimus (mTORi)</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
<td></td>
</tr>
<tr>
<td>KRAS mt NSCLC</td>
<td>VS-6766 + sotorasib (G12C)</td>
<td>✔️</td>
<td>🟨</td>
<td>✔️</td>
<td></td>
</tr>
</tbody>
</table>

<sup>1</sup> Registration-directed trial

<sup>2</sup>Pre-clinical studies ongoing in multiple KRAS mutant tumors

RAMP 201 study = NCT04625270
RAMP 202 study = NCT04620330
FRAME study = NCT03875820
VS-6766 is a Unique Small Molecule RAF/MEK Inhibitor

- VS-6766 inhibits both MEK & RAF kinase activities
- 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

References: Ishii et al., Cancer Res, 2013; Lito et al., Cancer Cell, 2014
VS-6766 inhibits cell proliferation across multiple MAPK pathway alterations and multiple solid tumor indications.

![Graph showing IC50 values for VS-6766 across various cell lines and indications.]

- **Indication**: NSCLC, Panc, CRC, Melanoma, Other
- **KRAS/BRAF/NRAS/NF1 status**: KRAS G12C, G12D, G12V, KRAS mt, BRAF mt, NRAS mt, ARAF mt, ERK2 mt, NRAS mt, Other mt


**Key**: KRAS G12C, KRAS G12D, KRAS G12V, Other, KRAS mt, BRAF mt, NRAS mt, ARAF mt, ERK2 mt, Other mt.
Vertical Blockade: Establishing VS-6766 as the backbone of therapy for RAS pathway-driven tumors

- **Current Challenges**
  - Blocking any single target in the pathway is insufficient for maximum depth and duration of anti-tumor efficacy
    - e.g., SHP2i, KRAS-G12Ci, RAFi, MEKi, ERKi
  - Vertical blockade concept is now well established
    - Necessary to block more than 1 target in the pathway
    - Many of these agents (e.g., SHP2i, MEKi) have poor tolerability as monotherapy and in combination

- **Solutions offered by VS-6766**
  - Vertical blockade (RAF and MEK blockade) in a single drug
  - Best-in-class tolerability with established twice weekly dosing regimen
    - Should enable tolerable combinations
  - Compelling synergy data (preclinical) for VS-6766 combinations (e.g., with KRAS-G12C inhibitors) supporting clinical combinations

References:
Parallel Pathway Inhibition: Establishing VS-6766 as the backbone of therapy for RAS pathway-driven tumors

- **Current Challenges**
  - Blocking Ras pathway can be circumvented through parallel pathways
    - e.g., PI3K/AKT/mTOR, FAK, RhoA, YAP
  - Combinations of MEKi + AKTi have shown poor tolerability

- **Solutions offered with VS-6766**
  - Good tolerability with twice weekly VS-6766 opens up intermittent dosing options for combinations
  - Compelling preclinical synergy data with VS-6766 in combination with FAK inhibition and with AKT pathway inhibition (e.g., everolimus)
  - RP2D established for VS-6766 + defactinib and for VS-6766 + mTORi (everolimus) with twice weekly regimen

References: ¹ Chen, Mol Cancer Res 2018; ² Banerji, BTOG Dublin, Jan 23, 2019
VS-6766 +/- Defactinib in Low-Grade Serous Ovarian Cancer
Favorable Tolerability Profile with Novel Intermittent Dosing Regimen

Summary of Adverse Events Grade ≥ 3 Occurring in ≥ 5% of patients

<table>
<thead>
<tr>
<th>Treatment Related Adverse Event</th>
<th>Grade ≥3</th>
<th>Grade ≥3</th>
<th>Grade ≥3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rash</td>
<td>3 (50%)</td>
<td>5 (19%)</td>
<td>2 (5%)</td>
</tr>
<tr>
<td>CK elevation (Creatine phosphokinase)</td>
<td>1 (17%)</td>
<td>2 (8%)</td>
<td>2 (5%)</td>
</tr>
</tbody>
</table>

Summary of FRAME Safety Profile
Most Adverse Events (AE) were Grade 1/2
Few patients have discontinued due to AEs in the study

References: Banerji, Q4 2020 report; Data on file
RP2D: recommended phase 2 dosing
Favorable Tolerability Profile at Recommended Phase 2 dose for VS-6766 plus defactinib combination regimen

<table>
<thead>
<tr>
<th>Treatment Related Adverse Events Details* (≥10% patients in cohort)</th>
<th>VS-6766 4mg Twice Weekly (4 wks of every 4 wks)(^1) n=22</th>
<th>VS-6766 3.2mg Twice Weekly Def 200mg BID (3 wks of every 4 wks)(^2) n=38</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gr1/2</td>
<td>Gr3/4</td>
<td>Gr1/2</td>
</tr>
<tr>
<td>Rash</td>
<td>15</td>
<td>5</td>
</tr>
<tr>
<td>CK Elevation</td>
<td>13</td>
<td>2</td>
</tr>
<tr>
<td>AST Elevation</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>Hyperbilirubinemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visual Disturbance</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>ALT Elevation</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Fatigue</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Oral Mucositis(^\wedge)</td>
<td>7</td>
<td>1</td>
</tr>
<tr>
<td>Nausea</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Vomiting</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Peripheral Edema</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Paronychia</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pruritus</td>
<td>3</td>
<td>0</td>
</tr>
</tbody>
</table>

Summary of FRAME Safety Profile

- Most Adverse Events (AE) were Grade 1/2
- Few patients have discontinued due to AEs in the study

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)

\(^{*}\)AEs were graded by NCI CTC v4; highest grade only recorded for each patient; AEs presented in ≥10% Patient (cohort 3.2mg 6766 and Def 200mg) data preliminary and subject to change;

\(^{\wedge}\)also includes glossitis/mouth ulcers

References: 1 Data on file VS-6766 Investigator’s Brochure; 2 Banerji, Q4 2020 report
VS-6766 in Combination with Defactinib Shows Promising ORR with Durability in Refractory LGSOC with Expanded Number of Patients (n=24)

- Overall response rate (ORR) = 46% (11 confirmed PRs/24)
  - KRAS mutant ORR = 64% (7 confirmed PRs/11)
  - KRAS wild-type ORR = 44% (4 confirmed PRs/9)
  - KRAS status undetermined (1 unconfirmed PR/4)
- Response too early to determine for 2 pts on study for ≤5 months
- Responses in patients previously treated with MEKi
- 54% (13/24) patients still on treatment
- 1 patient discontinuing for adverse events as of April 2021
- Median PFS 23 months (95% CI 10.6-NR) across all LGSOC

Data cut off April 2021
PFS: Progression free survival
NR: Not reached

Reference: Banerjee et al., ESMO Sept 2021
LGSOC: Limited Treatment Options with High Unmet Need

Low-Grade Ovarian Cancer – Treatment Algorithm

**Stage IA-IB**
- Observe only

**Stage IC**
- Pt Chemo Combo: Carbo-Pt + Paclitaxel (preferred) + Beva for Stage II-IV (incl maintenance Beva) OR Hormonal Tx (2B)

**Stage II-IV**
- Pt-Sensitive
  - Pt-Chemo combo +/- Beva
  - Trametinib
  - Fulvestrant

- Pt-Resistance
  - Taxane or gemcitabine, or doxorubicine, or topotecan +/- Beva
  - Trametinib
  - Fulvestrant

**Recurrence**
- Recurrence

<table>
<thead>
<tr>
<th>Therapy</th>
<th>Response Rate ORR</th>
<th>Median PFS Months (95% CI)</th>
<th>Discontinuation Rate due to AEs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard of Care¹</td>
<td>6%</td>
<td>7.2 (5.6-9.9)</td>
<td>12 %</td>
</tr>
<tr>
<td>Trametinib¹</td>
<td>26%</td>
<td>13.0 (9.9-15.0)</td>
<td>35%</td>
</tr>
<tr>
<td>Standard of Care²</td>
<td>13%</td>
<td>10.6 (9.2 to 14.5)</td>
<td>17%</td>
</tr>
<tr>
<td>Binimetinib²</td>
<td>16%</td>
<td>9.1 (7.3-11.3)</td>
<td>31%</td>
</tr>
</tbody>
</table>

References:

¹ NCCN guidelines

2 Monk et al., J Clin Oncol 2020.

Standard of Care = letrozole, tamoxifen, chemotherapy

PFS = Progression free survival

CI = confidence interval
70% of LGSOC tumors driven by mutations in the RAS pathway

- LGSOC is a type of ovarian cancer that disproportionately affects younger women
- 1,000 to 2,000 patients in the U.S. and 15,000 to 30,000 worldwide diagnosed with LGSOC each year
- A slow growing cancer; that has a median survival of almost 10 years, so patients remain in treatment for a long time (10-yr prevalence ~80,000 worldwide, ~6,000 US)
- Patients often experience significant pain and suffering from their disease over time
- Most prior research has focused on high grade serous ovarian cancer (HGSOC). However, LGSOC is clinically, histologically and molecularly unique from HGSOC with limited treatments available

~30% of LGSOC Patients Have KRAS mt
~70% of LGSOC Shows RAS Pathway-Associated mts

References: AACR Project GENIE Cohort v9.0-public and Verastem unpublished analysis

RAMP 201: KRAS Mutated (mt) and Wild Type (wt), Phase 2, Recurrent LGSOC Adaptive Design for Potential Accelerated Approval

**Selection Phase***
- Defactinib+VS-6766
  - KRAS-mt
  - (n=16)
- VS-6766 Mono
  - KRAS-mt
  - (n=16)

**Expansion Phase**
- Selected Regimen based on ORR
  - Add ~20-30 patients with KRAS mt
  - Add ~20-40 patients with KRAS wt
- Total Expected Range of Patients: 104-134

- New cohorts added via protocol amendment

**Primary Endpoint ORR**
- Hierarchical evaluation of:
  1) In KRAS mt patients
  2) All patients (KRASmt & wt)

---

**FDA Was Supportive of Development Strategy, Adaptive Design, and Addition of KRAS wt to Selection Phase**

Registration-directed Study Commenced in Nov. 2020 with estimated Primary Completion Date for the Expansion Phase of June 2023 (clinicaltrials.gov)

---

*Dosing: Defactinib + VS-6766 combo: Defactinib 200mg PO BID: 21/28 days + VS-6766 3.2mg PO 2x/wk 21/28 days; VS-6766 monotherapy: VS6766 4.0 mg PO 2x/wk 21/28 days

**Expansion Phase** – final sample size to be adjusted based on adaptive design
LGSOC market opportunity larger or comparable to other high unmet need KRAS opportunities

Patient-months of Therapy Per Year\(^2\) (across all 2L+ patients)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Prevalence</th>
<th>Patient-months of Therapy Per Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSCLC KRAS G12C(^3)</td>
<td>~50,000</td>
<td></td>
</tr>
<tr>
<td>Pancreatic(^3)</td>
<td>~100,000</td>
<td></td>
</tr>
<tr>
<td>LGSOC(^3)</td>
<td>~150,000</td>
<td></td>
</tr>
<tr>
<td>Endometrioid(^3)</td>
<td>~4k</td>
<td></td>
</tr>
<tr>
<td>Metastatic uveal melanoma(^3)</td>
<td>~2k</td>
<td></td>
</tr>
</tbody>
</table>


\(^2\) Patient-months of Therapy metric calculated by multiplying relevant incidence/prevalence rate times estimated duration of therapy; represents US market opportunity only; patient population estimates from Globocan 2020, American Cancer Society 2021, AACR Genie Cohort V9.0 public, and scientific publications. Duration of therapy estimates from clinical studies and clinician experience. Patient-months on therapy is for 2nd-line+ patients

\(^3\) NSCLC KRAS G12C 2nd line patients (incidence); Pancreatic RAS/RAF mutant 2nd-line patients (incidence); LGSOC KRAS mutant and wild-type patients (prevalence); Endometrioid RAS/RAF mutant 2nd-line patients (incidence); Uveal melanoma RAS/RAF mutant 2nd-line patients (incidence)
VS-6766 +/- Defactinib in NSCLC
High Unmet Need in Refractory KRAS mt NSCLC Adenocarcinoma

NSCLC Adenocarcinoma

<table>
<thead>
<tr>
<th>KRAS Mutation</th>
<th>% of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>G12C</td>
<td>15</td>
</tr>
<tr>
<td>G12V</td>
<td>10</td>
</tr>
<tr>
<td>G12D</td>
<td>5</td>
</tr>
<tr>
<td>G12A</td>
<td>5</td>
</tr>
<tr>
<td>G13C</td>
<td>3</td>
</tr>
<tr>
<td>G12S</td>
<td>3</td>
</tr>
<tr>
<td>G13D</td>
<td>1</td>
</tr>
</tbody>
</table>

KRAS Mutations Represent 25% of Lung Cancer Adenocarcinoma (EGFR 17%, ALK 7%)\(^4\)

Advanced or Metastatic NSCL Cancer Recommend Histologic and Molecular Subtyping\(^5\)

- EGFR/ALK/ROS1/BRAF (targeted)
- Non-targeted PD-(L)\(1 \geq 1\%\)
- Non-Targeted PD-(L)\(1 < 1\%\)

Appropriate targeted agent

PD-(L)\(1\) single agent or PD-(L)\(1\) + chemo

Chemo ± PD-(L)\(1\)

Recurrence

Prior PD-(L)\(1\)
- Chemotherapy
  - Docetaxel
  - Gemcitabine
  - Pemetrexed

No Prior PD-(L)\(1\)
- PD-(L)\(1\)

Chemotherapy or clinical trials

• SOC in recurrent disease is chemotherapy
• Pre-PD-(L)\(1\) era, chemotherapy response rate ~10% in recurrent disease; 12w PFS of 30–45%

References:
1. Globocan, 2018
3. TCGA PanCancer Atlas (cBioPortal analysis)
5. Adapted from NCCN Non-small cell lung cancer guidelines Version 3.2020
VS-6766 Inhibits CRAF - The key driver of KRAS-G12V mutant NSCLC

A Precision Approach to KRAS-G12V Driven NSCLC

- KRAS$^{G12V}$ signals mainly through RAF/MEK in contrast to other variants, such as KRAS-G12D, which signal more through PI3K/AKT
- KRAS$^{G12V}$ models are especially dependent on CRAF

CRAF drives KRAS G12V mt NSCLC

CRAF KO shows strong efficacy

BRAF KO has no effect

VS-6766 +/- FAKi induces significant tumor regression in KRAS G12V mt NSCLC in vivo model, with clear differentiation from trametinib

**Doses Tested**
- Trametinib: 0.1 mg/kg QD (5 days/week)
- VS-6766: 0.1 mg/kg QD (5 days/week)
- FAKi: 50 mg/kg BID (5 days/week)

**KRAS G12V mutant; Tp53 KO NSCLC**

- VS-6766 monotherapy caused tumor regression
- VS-6766 + FAKi showed stronger regression
- No significant anti-tumor effect of trametinib at same dose level

**4 weeks of treatment**

Statistics: Mann-Whitney test

**Collaboration with Mariano Barbacid**

Reference: Coma et al. AACR 2021
Case Study: Response to VS-6766 + defactinib in a patient with KRAS G12V mutant NSCLC

May 2019: Diagnosed with NSCLC

June 2019 - Sept 2019: Treated with first line Carboplatin + Pemetrexed + Pembrolizumab

Oct 2019: Progression, palliative RT to right hip

Nov 2019 – present: On treatment in FRAME study VS-6766 + Defactinib

Reference: Krebs et al. AACR 2021
Strong Signal Identified in KRASG12V to Be Further Validated

VS-6766 ± Defactinib Has a Confirmed 57% ORR in KRASG12V mt NSCLC in Integrated Analysis

- Preclinical evidence suggests combination with Defactinib may improve efficacy in KRASG12V mt NSCLC
- Activity of VS-6766 as a single agent and in combo with Defactinib in KRASG12V mt NSCLC

NSCLC Clinical Strategy: KRAS Mutant (mt), Enriched G12V, Phase 2, Recurrent NSCLC for Potential Accelerated Approval

Selection Phase
- Defactinib + VS-6766¹
  - KRAS mt G12V
  - N=16

- VS-6766²
  - KRAS mt G12V
  - N=16

Expansion Phase
- KRAS Mutant – G12V
  - Selected Regimen based on ORR

- KRAS Mutant – non-G12V
  - Exploratory mutation-specific cohort analyses for ORR

This Registration-directed Phase 2 Study commenced December 2020 with an estimated Primary Completion Date for the Expansion Phase of March 2023 (clinicaltrials.gov)

References:
¹ Defactinib 200 mg PO BID (21/28 days) + VS-6766 3.2 mg PO 2x/wk (21/28 days)
² VS-6766 4.0 mg PO 2x/wk (21/28 days)
Preclinical synergy of VS-6766 + G12C inhibitors in KRAS G12C mt models

Synergy of VS-6766 + G12C inhibitor AMG 510 across G12C mutant NSCLC, CRC & Pancreatic cancer cell lines

<table>
<thead>
<tr>
<th>Cell line</th>
<th>Indication</th>
<th>Sensitivity to G12C inhibitors</th>
<th>VS-6766 + AMG 510</th>
<th>VS-6766 + MRTX849</th>
</tr>
</thead>
<tbody>
<tr>
<td>H2122</td>
<td>NSCLC</td>
<td>Moderately sensitive</td>
<td>44.7</td>
<td>44.6</td>
</tr>
<tr>
<td>H1373</td>
<td>NSCLC</td>
<td>Sensitive</td>
<td>10.0</td>
<td>3.4</td>
</tr>
<tr>
<td>SW1573</td>
<td>NSCLC</td>
<td>Insensitive</td>
<td>8.6</td>
<td>12.0</td>
</tr>
<tr>
<td>H358</td>
<td>NSCLC</td>
<td>Sensitive</td>
<td>6.9</td>
<td>5.4</td>
</tr>
<tr>
<td>H2030</td>
<td>NSCLC</td>
<td>Moderately sensitive</td>
<td>5.1</td>
<td>ND</td>
</tr>
<tr>
<td>SW837</td>
<td>CRC</td>
<td>Sensitive</td>
<td>16.1</td>
<td>18.5</td>
</tr>
<tr>
<td>MIAPACA2</td>
<td>Panc</td>
<td>Sensitive</td>
<td>2.3</td>
<td>5.3</td>
</tr>
</tbody>
</table>

ND: not determined

Combination of AMG 510 potentiates VS-6766 efficacy in KRAS G12C mutant NSCLC in vivo; tumor regression in all mice with triple combination

Doses Tested
- Trametinib: 0.3 mg/kg QD
- VS-6766: 0.3 mg/kg QD
- FAKi: 50 mg/kg BID
- AMG 510: 30 mg/kg QD


tumor volume (mm³ ± SEM)

Days after cell inoculation

H2122 KRAS G12C mutant NSCLC

Concentrations Tested
- AMG 510: 100 nM
- VS-6766: 100 nM

Reference: Coma et al., AACR 2021
Acquired resistance mechanisms to KRAS G12Ci treatment in patients further support combination of KRAS G12Ci with VS-6766

Summary of Putative Mechanisms of Acquired Resistance to Adagrasib Treatment

- Mechanisms of acquired resistance to KRAS G12Ci adagrasib treatment in patients recently reported\(^1,2\)
- The main resistance alterations occurred in
  - RTK mts or amplifications
  - KRAS mts or amplification
  - NRAS mt
  - BRAF V600E mt, BRAF or CRAF fusions
  - MAP2K1 (MEK1) mt/deletion
- VS-6766 is expected to be effective against these KRAS, NRAS, BRAF and CRAF modifications

We have just initiated a clinical collaboration with Amgen to explore the combination of VS-6766 + sotorasib in NSCLC KRAS G12C mt patients.

- Patients must have known G12C KRAS mutation determined using validated test
- Treatment with at least 1 but no more than 3 prior systemic regimens, for Stage 3B-C or 4 NSCLC
- Patient may have previously received adjuvant chemotherapy for earlier-stage disease
- Measurable disease according to RECIST 1.1
- ECOG performance status ≤ 1

**Part A: Dose Evaluation (3+3 DLT Assessment)**

- Dose Level 1 (N= 3 to 6)
  - VS-6766 3.2 mg BIW + Sotorasib 960mg QD

**Part B: Dose Expansion at RP2D (Primary endpoint ORR)**

- Cohort 1
  - Patients without Prior KRAS G12C Inhibitor Treatment
  - Stage 1: ~20 patients
  - Stage 2: expand

- Cohort 2
  - Patients whose NSCLC Progressed on KRAS G12C Inhibitor Treatment
  - Stage 1: ~20 patients
  - Stage 2: expand

**Part A (Dose Evaluation) portion of study expected to be initiated in 4Q 2021**
Future Opportunities: VS-6766 as Backbone of RAS Therapy
Vertical Blockade: Preclinical synergy in combination with several promising targets

Reference: Coma et al., AACR 2021
Parallel Pathway Inhibition: Two synergistic combinations already progressed to clinical stage

Reference: Coma et al., RAS-Targeted Drug Discovery, Feb 2021
Corporate
Key VSTM Milestones 2020-2022

**Corporate**
- 1H2020: VS-6766 In-Licensed from Chugai
- 2H2020: PIPE Financing for New Clinical Program
- 1H2021: Copiktra divested to Secura Bio
- 2H2021: Retired Outstanding Debt
- 1H2022: 

**LGSOC**
- 2H2020: RAMP-201 Study Initiated
- 1H2021: RAMP-201 Amended to Include KRAS wt patients in Selection Phase
- 2H2021: FDA Breakthrough Therapy Designation
- 1H2022: Updated data from FRAME LGSOC cohort Presenting at ESMO

**NSCLC**
- 1H2020: RAMP-202 Study Initiated
- 2H2020: Updated data from FRAME NSCLC cohort Presented at AACR
- 1H2021: Updated data from FRAME NSCLC cohort Presented at AACR
- 2H2021: VS-6766 + Sotorasib Collaboration w/Amgen RAMP-203 Study Initiated
- 1H2022: RAMP-202 Top-Line Data from Selection Phase & Initiate Expansion Phase
### Key Financial Statistics

**As of September 30, 2021**

<table>
<thead>
<tr>
<th>Cash, cash equivalents &amp; investments</th>
<th>$103.4M</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shares fully diluted</td>
<td>196.9M</td>
</tr>
<tr>
<td>Insider ownership (outstanding / vested)</td>
<td>8.1% / 5.1%</td>
</tr>
</tbody>
</table>

* The 2018 Notes have an initial conversion rate of 139.5771 shares of Common Stock per $1,000 which translates to an initial conversion price of $7.16 per share of Common Stock.
Backup Slides
High Unmet Needs in RAS/RAF/MEK/ERK-Driven Cancers

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>NRAS-mutant Cancers¹</td>
<td>Melanoma Incidence²: 108K</td>
<td>Multiple Myeloma Incidence³: 32K</td>
<td>Papillary Thyroid Incidence⁴: 42K</td>
<td>Ovarian Incidence¹: 22K</td>
<td></td>
</tr>
<tr>
<td>BRAF-mutant Cancers²</td>
<td>Melanoma Incidence²: 108K</td>
<td>Ovarian Incidence³: 22K</td>
<td>Papillary Thyroid Incidence³: 42K</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Breadth of potential opportunity
- 30% of all human cancers are driven by mutations of the RAS family of genes⁶

### 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
- Current RAS inhibitors in development address only a minority of all RAS mutated cancers

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

% frequency in a total of 780 cancer patients with KRAS mutations\(^1\)

References:
\(^1\) 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)
VS-6766 and FAK inhibitor combination leads to more robust anti-tumor efficacy in vivo

**KRAS\textsuperscript{mt} Ovarian TOV-21G in vivo Model\textsuperscript{1}**

- Tumor growth over time for different treatment groups:
  - Vehicle
  - Trametinib 1.5 mg/kg QD
  - FAKi 50 mg/kg BID
  - VS-6766 1.5 mg/kg QD
  - VS-6766 + FAKi

**KRAS\textsuperscript{mt} NSCLC H358 in vivo Model\textsuperscript{2}**

- Tumor growth over time for different treatment groups:
  - Vehicle
  - Trametinib 0.3 mg/kg QD
  - FAKi 50 mg/kg BID
  - VS-6766 0.3 mg/kg QD
  - VS-6766 + FAKi

References: \textsuperscript{1} Coma AACR 2021; \textsuperscript{2} Krebs AACR 2021
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
Pharmacokinetic Profiles of VS-6766 + Defactinib in Combination Similar to that seen in Single Agent Studies

### VS-6766

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Dose (mg)</th>
<th>N</th>
<th>Subject</th>
<th>AUC$_{0-24h}$ (h*ng/mL)</th>
<th>C$_{max}$ (ng/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3.2 (with 200mg VS)</td>
<td>3</td>
<td></td>
<td>Mean 6179</td>
<td>354</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CV% 32.1</td>
<td>30.4</td>
</tr>
<tr>
<td>2a</td>
<td>4 (with 200mg VS)</td>
<td>5</td>
<td></td>
<td>Mean 5353</td>
<td>289</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CV% 15.8</td>
<td>16.0</td>
</tr>
<tr>
<td>2b</td>
<td>3.2 (with 400mg VS)</td>
<td>1</td>
<td>FRA101-007</td>
<td>3302</td>
<td>229</td>
</tr>
</tbody>
</table>

### Defactinib

<table>
<thead>
<tr>
<th>Cohort</th>
<th>Dose (mg)</th>
<th>N</th>
<th>Subject</th>
<th>AUC$_{last}$ (h*ng/mL)</th>
<th>C$_{max}$ (ng/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>200 (with 3.2mg RO)</td>
<td>3</td>
<td></td>
<td>Mean 2071</td>
<td>273</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CV% 103</td>
<td>80</td>
</tr>
<tr>
<td>2a</td>
<td>200 (with 4mg RO)</td>
<td>5</td>
<td></td>
<td>Mean 2252</td>
<td>318</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CV% 124</td>
<td>117</td>
</tr>
<tr>
<td>2b</td>
<td>400 (with 3.2mg RO)</td>
<td>3</td>
<td></td>
<td>Mean 2807</td>
<td>360</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CV% 31</td>
<td>32</td>
</tr>
</tbody>
</table>

Reference: Banerji, AACR VM 1, April 27, 2020, CT143
NSCLC Responses with VS-6766 + Defactinib Combination (n=20)

Confirmed responses in 2/2 patients with KRAS G12V mt NSCLC

Tumor reduction in 4/6 patients with KRAS G12C mt NSCLC

**Best response by RECIST in KRAS mt NSCLC**

- ORR = 15% (3/20)
- ORR in G12V mt = 100% (2/2)
- DCR = 65% (13/20)
- 3/20 (15%) still on study
- 7 pts on treatment ≥ 24 weeks

**Data cut off March 5, 2021**

**Time on Treatment**

- Continuing on treatment
- Unconfirmed PR
- Time to response

**Best Response (%) change from baseline**

- Continuing on treatment
- Partial response
- Progressive disease
- Stable disease

Reference: Krebs et al. AACR 2021
Target exposure for preclinical tumor regression is covered by twice weekly dosing of 4 mg VS-6766 3 wks on/1 wk off.

- Modeling of PK for 4 mg VS-6766 2/wk, 3 wks on/1 wk off, based on 4 mg single dose PK data (study NO21895)
- Relationship to average exposure for tumor regression in KRAS G12V mt NSCLC mouse model

References: Martinez-Garcia et al., Clin Cancer Res 2012; Coma et al. AACR 2021
Status: Combination of VS-6766 with Everolimus (mTOR inhibitor)

- Synergy of VS-6766 + everolimus observed broadly across cancer cell lines with various KRAS mutation variants
- A well-tolerated RP2D for VS-6766 + everolimus has been established with intermittent dosing of both agents (twice weekly; 3 wks on/1 wk off)
- KRAS mutant NSCLC expansion cohort is currently ongoing with VS-6766 + everolimus

Reference: Coma et al., RAS-Targeted Drug Discovery, Feb 2021
Combination of VS-6766 with anti-EGFR mAb induces tumor regression in a KRAS mt Colorectal PDX model

- VS-6766 + anti-EGFR (panitumumab) induces tumor regression in a KRAS G12V mt CRC patient-derived xenograft model
- G12Ci + anti-EGFR (sotorasib + panitumumab and adagrasib + cetuximab) have shown partial responses in KRAS G12C mt CRC (Fakih et al. ESMO 2021; Weiss et al. ESMO 2021)
- These data support clinical testing of VS-6766 + anti-EGFR for treatment of KRAS mt CRC
VS-6766 monotherapy has shown clinical activity in several cancer indications, including NSCLC

Best Response

Guo et al., Lancet Oncology 2020
VS-6766 upregulates MHC Class I antigens on tumor cells: a mechanism for potentiation of I/O efficacy

<table>
<thead>
<tr>
<th>Cell Line</th>
<th>Tumor type</th>
<th>RAS/RAF mutation status</th>
</tr>
</thead>
<tbody>
<tr>
<td>A549</td>
<td>Lung</td>
<td>KRASmt G12S</td>
</tr>
<tr>
<td>TOV21g</td>
<td>Ovarian</td>
<td>KRASmt G13C</td>
</tr>
<tr>
<td>SKMEL5</td>
<td>Melanoma</td>
<td>BRAFmt V600E</td>
</tr>
<tr>
<td>IGR-1</td>
<td>Melanoma</td>
<td>BRAFmt V600E</td>
</tr>
<tr>
<td>WM115</td>
<td>Melanoma</td>
<td>BRAFmt V600E</td>
</tr>
</tbody>
</table>

VS-6766 @ 1 µM (except SKMEL5 and IGR-1, 300 nM)

Reference: Pachter, RAS-Targeted Drug Development, Sept 2020
VS-6766 enhances tumor growth inhibition when combined with anti-PD-1 in the CT26 KRAS (G12D) syngeneic model

**Reference:** Pachter, RAS-Targeted Drug Development, Sept 2020
# LGSOC Market Opportunity – Reference Calculations

<table>
<thead>
<tr>
<th></th>
<th>Number of Patients (2L+)</th>
<th>Average months on Therapy (per patient)</th>
<th>Patient-months of Therapy Per Year (across all 2L+ patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NSCLC KRAS G12C</strong></td>
<td>10,000</td>
<td>5.00</td>
<td>50,000</td>
</tr>
<tr>
<td></td>
<td>20,000</td>
<td>10.00</td>
<td>100,000</td>
</tr>
<tr>
<td></td>
<td>30,000</td>
<td>15.00</td>
<td>150,000</td>
</tr>
<tr>
<td></td>
<td>40,000</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Pancreatic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>LGSOC</strong></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Endometrioid</strong></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Metastatic uveal melanoma</strong></td>
<td>1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


2 Patient-months of Therapy metric calculated by multiplying relevant incidence/prevalence rate times estimated duration of therapy; represents US market opportunity only; patient population estimates from Globocan 2020, American Cancer Society 2021, AACR Genie Cohort 9.0 public, and scientific publications. Duration of therapy estimates from clinical studies and clinician experience. Number of patients and months on therapy are for 2nd-line+ patients

3 NSCLC KRAS G12C 2nd line patients (incidence); Pancreatic RAS/RAF mutant 2nd-line patients (incidence); Endometrioid RAS/RAF mutant 2nd-line patients (incidence); Uveal melanoma RAS/RAF mutant 2nd-line patients (incidence)
A drug with a Breakthrough designation will have(1)…

• Increased communication with FDA during drug development and review

• FDA guidance to ensure that the design of clinical trials are as efficient as practicable

• A cross-disciplinary project lead assigned to the FDA review team and increased involvement of senior managers and experienced review staff

• Based on the criteria for the grant of breakthrough designation, may represent substantial improvement over existing clinical therapies

Reference: US FDA website
## List of Oncology Drugs that Received Breakthrough Therapy Designation

<table>
<thead>
<tr>
<th>Sr no.</th>
<th>Proprietary Name</th>
<th>Established Name</th>
<th>Current Approval Status</th>
<th>Company</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Zykadia</td>
<td>Ceritinib</td>
<td>Approved</td>
<td>Novartis</td>
</tr>
<tr>
<td>2.</td>
<td>Ibrance</td>
<td>Palbociclib</td>
<td>Approved</td>
<td>Pfizer</td>
</tr>
<tr>
<td>3.</td>
<td>Keytruda</td>
<td>Pembrolizumab</td>
<td>Approved</td>
<td>Merck</td>
</tr>
<tr>
<td>4.</td>
<td>Opdivo</td>
<td>Nivolumab</td>
<td>Approved</td>
<td>Bristol Myers Squibb</td>
</tr>
<tr>
<td>5.</td>
<td>Tagrisso</td>
<td>Osimertinib</td>
<td>Approved</td>
<td>Lilly</td>
</tr>
<tr>
<td>6.</td>
<td>Alecensa</td>
<td>Alectinib</td>
<td>Accelerated Approval</td>
<td>Genentech</td>
</tr>
<tr>
<td>7.</td>
<td>Xalkori</td>
<td>Crizotinib</td>
<td>Approved</td>
<td>Pfizer</td>
</tr>
<tr>
<td>8.</td>
<td>Lenvima</td>
<td>Lenvatinib</td>
<td>Approved</td>
<td>Eisai</td>
</tr>
<tr>
<td>9.</td>
<td>Tecentriq</td>
<td>Atezolizumab</td>
<td>Approved</td>
<td>Genentech</td>
</tr>
<tr>
<td>10.</td>
<td>Rubraca</td>
<td>Rucaparib</td>
<td>Approved</td>
<td>Clovis Oncology</td>
</tr>
<tr>
<td>11.</td>
<td>Kisqali</td>
<td>Ribociclib</td>
<td>Approved</td>
<td>Novartis</td>
</tr>
<tr>
<td>12.</td>
<td>Zejula</td>
<td>Niraparib</td>
<td>Approved</td>
<td>GSK</td>
</tr>
<tr>
<td>13.</td>
<td>Alunbrig</td>
<td>Brigatinib</td>
<td>Accelerated Approval</td>
<td>Takeda</td>
</tr>
<tr>
<td>14.</td>
<td>Kisqali Femara Co-Pack</td>
<td>Letrozole &amp; Ribociclib</td>
<td>Approved</td>
<td>Novartis</td>
</tr>
<tr>
<td>15.</td>
<td>Tafinlar</td>
<td>Dabrafenib</td>
<td>Approved</td>
<td>Novartis</td>
</tr>
<tr>
<td>16.</td>
<td>Mekirist</td>
<td>Trametinib</td>
<td>Approved</td>
<td>Novartis</td>
</tr>
<tr>
<td>17.</td>
<td>Verzenio</td>
<td>Abemaciclib</td>
<td>Approved</td>
<td>Lilly</td>
</tr>
<tr>
<td>18.</td>
<td>Imfini</td>
<td>Durvalumab</td>
<td>Approved</td>
<td>AstraZeneca</td>
</tr>
<tr>
<td>19.</td>
<td>Yervoy</td>
<td>Ipilimumab</td>
<td>Approved</td>
<td>Bristol Myers Squibb</td>
</tr>
<tr>
<td>20.</td>
<td>Azaleda</td>
<td>Iobenguane</td>
<td>Approved</td>
<td>Progenics Pharmaceuticals</td>
</tr>
<tr>
<td>21.</td>
<td>Lorbrena</td>
<td>Lorlatinib</td>
<td>Approved</td>
<td>Pfizer</td>
</tr>
<tr>
<td>22.</td>
<td>Kadcyla</td>
<td>Ado-trastuzumab emtansine</td>
<td>Approved</td>
<td>Genentech</td>
</tr>
<tr>
<td>23.</td>
<td>Padcev</td>
<td>Enfortumab vedotin-afv</td>
<td>Approved</td>
<td>Astellas Pharma</td>
</tr>
<tr>
<td>24.</td>
<td>Enhertu</td>
<td>Fam-trastuzumab deruxtecan-ixli</td>
<td>Approved</td>
<td>Daichi-Sankyo</td>
</tr>
<tr>
<td>25.</td>
<td>Jelmyto</td>
<td>Mitomycin</td>
<td>Approved</td>
<td>UroGen Pharma</td>
</tr>
<tr>
<td>26.</td>
<td>Tukysha</td>
<td>Tucatinib</td>
<td>Approved</td>
<td>Seagen</td>
</tr>
<tr>
<td>27.</td>
<td>Trodelvy</td>
<td>Sactituzumab Govitecan-hzly</td>
<td>Approved</td>
<td>Gilead</td>
</tr>
<tr>
<td>28.</td>
<td>Tabrecta</td>
<td>Cemotinib</td>
<td>Approved</td>
<td>Novartis</td>
</tr>
<tr>
<td>29.</td>
<td>Retevmo</td>
<td>Selpercatinib</td>
<td>Approved</td>
<td>Lilly</td>
</tr>
<tr>
<td>30.</td>
<td>Gavreto</td>
<td>Pralatrexib</td>
<td>Approved</td>
<td>Blueprint medicines</td>
</tr>
<tr>
<td>31.</td>
<td>N/A</td>
<td>VXS105/Darofectinb</td>
<td>Not yet approved</td>
<td>Verastem</td>
</tr>
<tr>
<td>32.</td>
<td>Lumakras</td>
<td>Sotorasib</td>
<td>Accelerated Approval</td>
<td>Amgen</td>
</tr>
<tr>
<td>33.</td>
<td>N/A</td>
<td>177Lu-PSMA-617</td>
<td>Not yet approved</td>
<td>Novartis</td>
</tr>
<tr>
<td>34.</td>
<td>Ayvakit</td>
<td>Ansprentinb</td>
<td>Approved (Mast Cell Leukemia)</td>
<td>Blueprint Medicines Corp</td>
</tr>
<tr>
<td>35.</td>
<td>N/A</td>
<td>Adagrasib</td>
<td>Not yet approved (NSCLC)</td>
<td>Mirati Therapeutics, Inc.</td>
</tr>
</tbody>
</table>

Reference: US FDA website. Covers drugs approved from inception of BTD program through July 16, 2021. Approvals include both full and accelerated approvals.
Strong Patent Protection

• 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
Experienced Senior Management Team

Brian Stuglik
Chief Executive Officer
- Global VP & Chief Marketing Officer – Lilly Oncology
- Founding Member – Proventus Health Solutions

Cathy Carew
Chief Organizational Effectiveness Officer
- Principal – HR Collaborative
- Ironwood, ActiveBiotics, Dynogen, Tufts Health Plan

Hagop Youssoufian, MSc, M.D.
Head of Medical Strategy
- CMO, BIND Therapeutics, EVP, Progenics
- CMO & EVP, Ziopharm Oncology, SVP, Imclone

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

Jonathan Pachter, Ph.D.
Chief Scientific Officer
- Head of Cancer Biology – OSI (now Astellas)
- Schering-Plough

Rob Gagnon
Chief Business and Financial Officer
- CFO – Harvard Bioscience, Clean Harbors
- VP of Finance – Biogen Idec

Louis Denis, M.D.
Chief Medical Officer
- CMO, Asana BioSciences
- Boehringer-Ingelheim, Pfizer