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Pharmaceuticals, Inc.

Sunesis Pharmaceuticals

July 2018

Safe Harbor Statement

This presentation contains forward-looking statements, including statements related to the continued development and commercialization of vecabrutinib (SNS-062), vosaroxin and other product candidates, the timing of our Phase 1b/2 trial of vecabrutinib, and the sufficiency of Sunesis' cash and funding into 2019. These forward-looking statements are based upon Sunesis' current expectations. Forward-looking statements involve risks and uncertainties. Sunesis' actual results and the timing of events could differ materially from those anticipated in such forward-looking statements as a result of these risks and uncertainties, which include, without limitation, the risk that Sunesis' development activities for vecabrutinib and other product candidates could be otherwise halted or significantly delayed for various reasons, risks related to Sunesis' need for substantial additional funding to complete the development and commercialization of vecabrutinib and other product candidates, including the risk that Sunesis' clinical studies for vecabrutinib and other product candidates may not demonstrate safety or efficacy or lead to regulatory approval, the risk that data to date and trends may not be predictive of future data or results, risks related to the timing or conduct of Sunesis' clinical trials, and risks related to Sunesis' ability to raise the capital that it believes to be accessible and is required to fully finance the development and commercialization of vecabrutinib and other product candidates. These and other risk factors are discussed under "Risk Factors" and elsewhere in Sunesis' Quarterly Report on Form 10-Q for the quarter ended March 31, 2018, Sunesis' Prospectus Supplement filed pursuant to Rule 424(b)5 on June 25, 2018, and Sunesis' other filings with the Securities and Exchange Commission. Sunesis expressly disclaims any obligation or undertaking to release publicly any updates or revisions to any forward-looking statements contained herein to reflect any change in Sunesis' expectations with regard thereto or any change in events, conditions or circumstances on which any such statements are based.



Sunesis Overview

- Developing targeted cancer therapies
- Vecabrutinib: next-generation BTK inhibitor for B-cell malignancies
 - Non-covalent inhibitor, maintains activity in ibrutinib-resistant mutations
 - Phase 1b/2 ongoing
 - Recommended Phase2 Dose in Fall 2018
- Pipeline of other kinase inhibitors for cancer
 - SNS-510: first-in-class PDK1 inhibitor
 - TAK-580: pan-RAF inhibitor partnered with Takeda
- Funded through key milestones

Strong Kinase-Inhibitor Pipeline

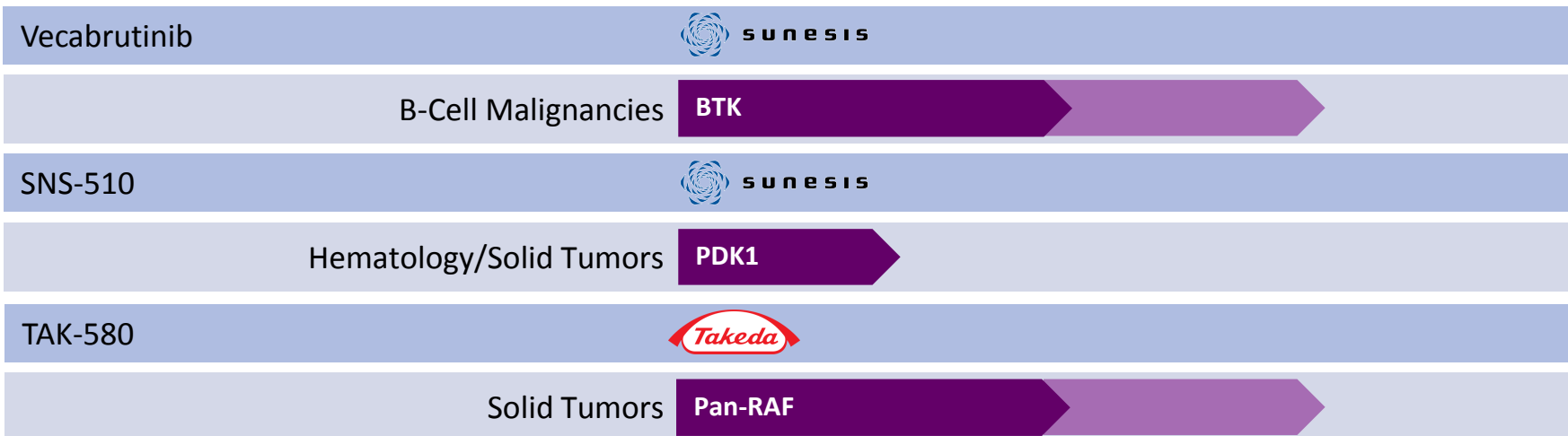
Kinase Inhibitor Pipeline

Pre Clinical

Phase 1

Phase 2

Phase 3



Anticancer Quinolone Derivative



Vecabrutinib

Non-covalent BTK inhibitor



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Vecabrutinib: Potential Next-Generation BTK Inhibitor

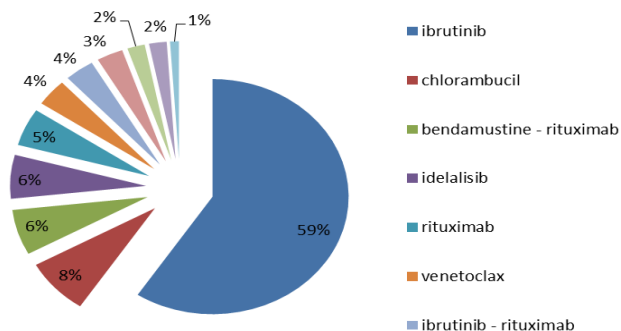
- BTK inhibitors: large and growing market
- Resistance to covalent BTK inhibitors is a growing concern
 - Mutation at covalent binding site (C481)
- **Vecabrutinib: *non-covalent*** BTK inhibitor
 - Inhibits wild-type and C481S-mutant BTK
 - Favorable PK/PD profile
- Phase 1b/2 B-Cell Malignancies Trial ongoing
 - Recommended P2 Dose expected in Fall 2018



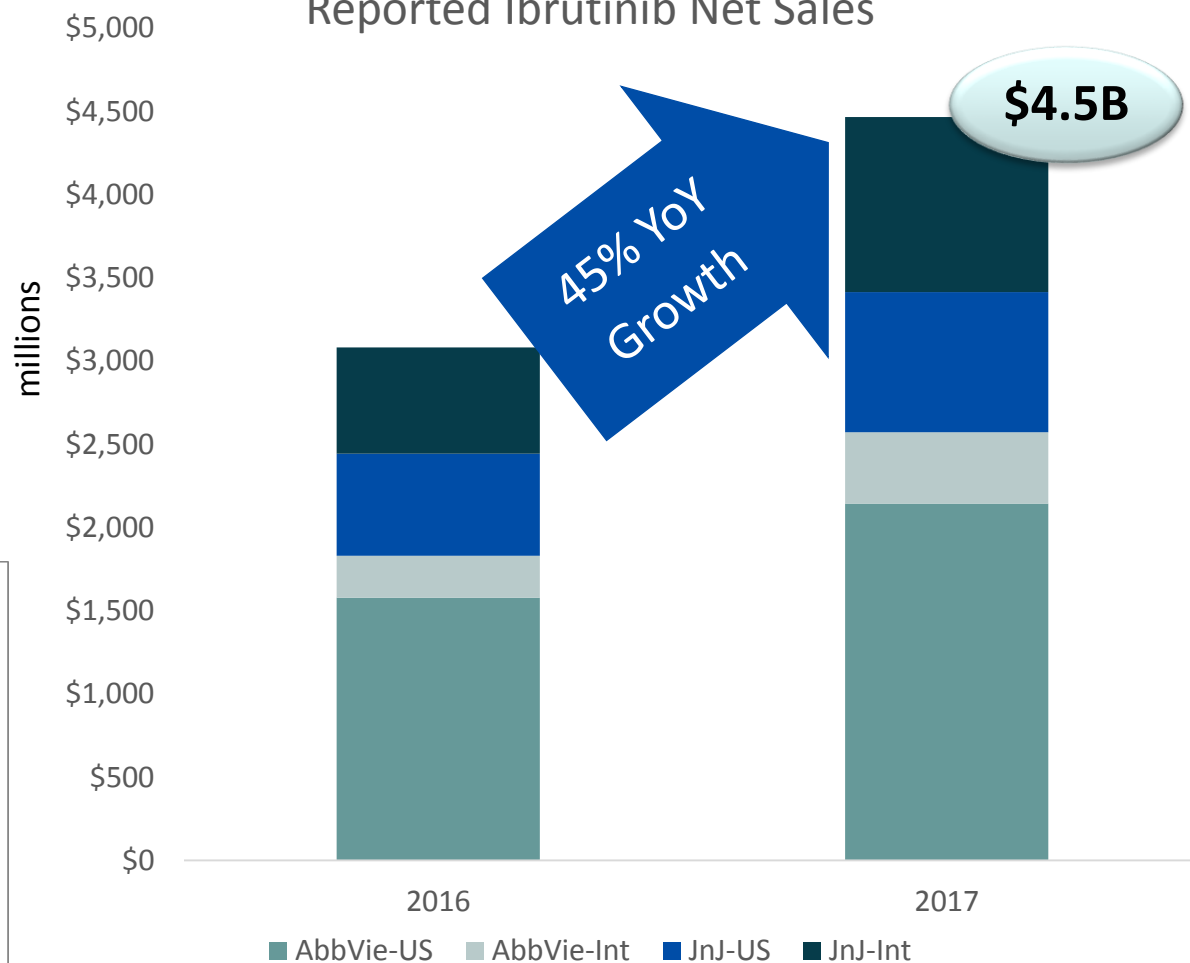
BTK-Inhibition Market is Growing Quickly

- Imbruvica® is leading frontline treatment for CLL
- Approved for a growing number of B-cell malignancies
- 70% market share in second-line

12 Month Average MS
2nd Line CLL



Reported Ibrutinib Net Sales

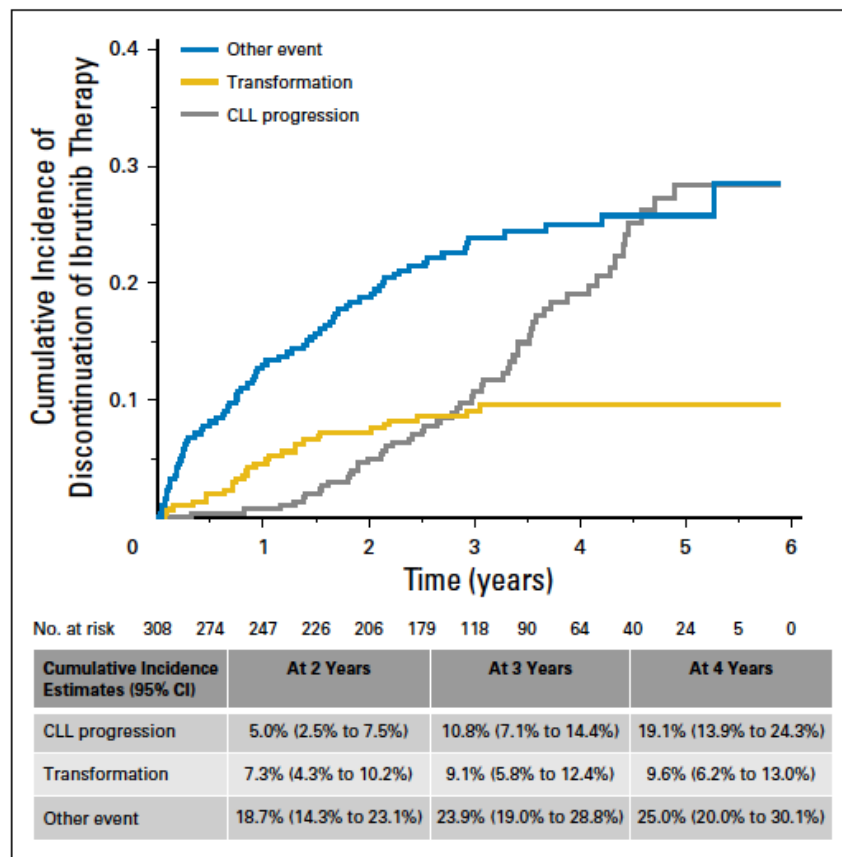


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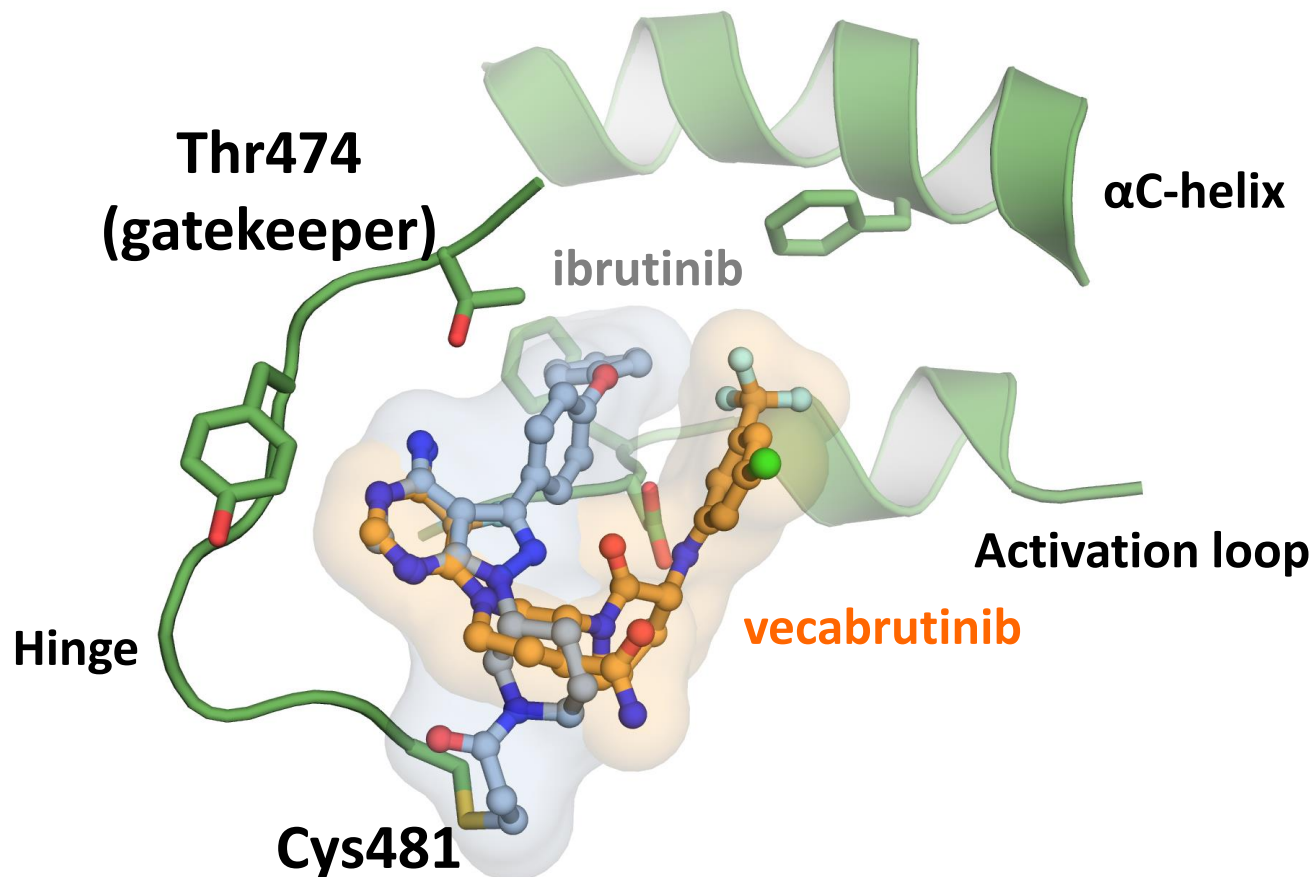
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Ibrutinib-Treated CLL Patient Landmark Analysis Supports BTK C481S as Emerging Resistance Mechanism at Relapse

- 308 ibrutinib-treated CLL patients followed at The Ohio State University
- Nearly 20% had progressive CLL by year 4
- **67% of CLL progressors sequenced had mutations in BTK C481 only**
 - C481 mutation appeared ~9 months prior to progression



Vecabrutinib BTK Kinase Domain Interaction is Differentiated from Ibrutinib

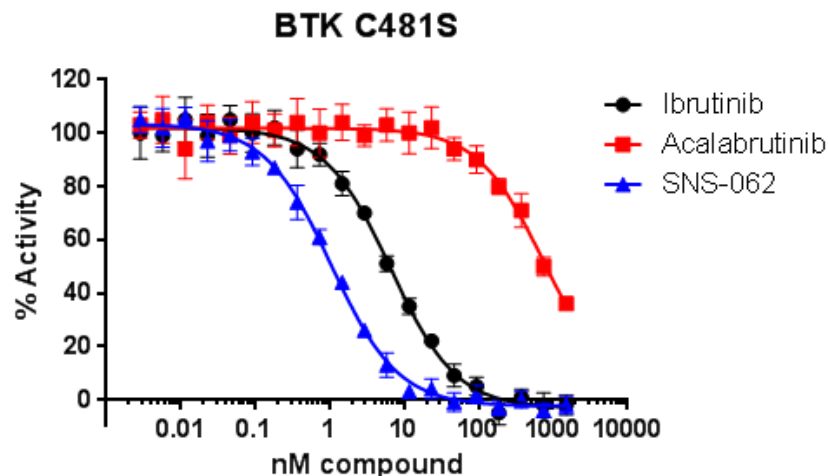


- Vecabrutinib interacts with a distinct set of residues in the α C-helix

Vecabrutinib Active in Wild-type and C481S Mutant BTK

| BTK Inhibition – Kinase Assays | | | |
|--------------------------------|--------|-----------|-------------|
| IC ₅₀ nM | WT BTK | C481S BTK | Fold Change |
| Vecabrutinib | 4.6 | 1.1 | 0.2 |
| Ibrutinib | 0.1 | 6.6 | 66 |
| Acalabrutinib | 4.2 | 707 | 168 |

[ATP] = 50 μ M.



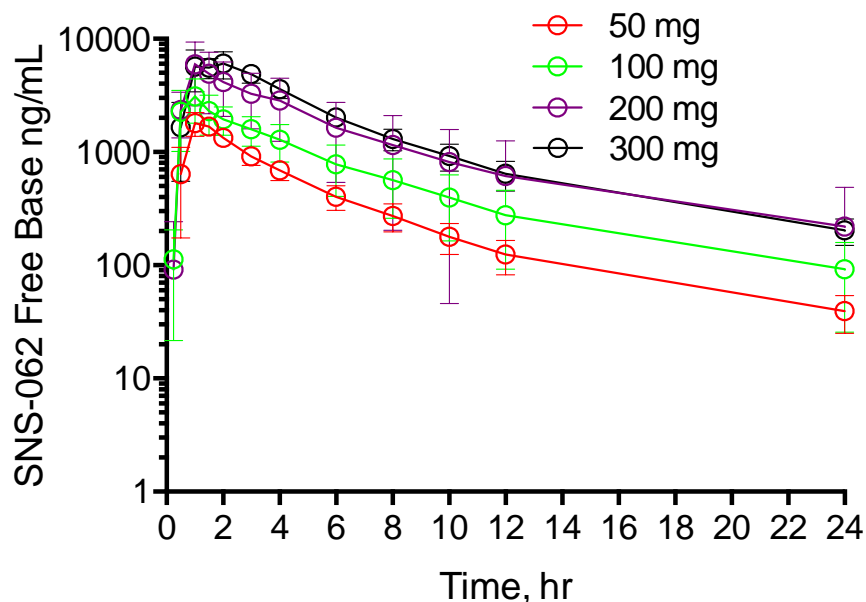
- Activity of ibrutinib and acalabrutinib is profoundly affected
 - Poor PK also contributes to resistance in patients with BTK C481 mutations
- Vecabrutinib activity unaffected by C481S mutation and has improved PK properties
- Vecabrutinib overcomes resistance conferred by C481S mutation and induces apoptosis in BTK-dependent lymphoma cell lines*

AACR-NCI-EORTC Conference, November 2015

* "Preclinical Validation of Vecabrutinib Efficacy Against BTK C481S Mutated Lymphomas";
EHA Poster, June 2018

Phase 1A: Favorable Pharmacokinetic Properties

- Vecabrutinib was rapidly absorbed with high exposure across all dose cohorts



| | Cohort 1 50 mg (n=6) | Cohort 2 100 mg (n=6) | Cohort 3 200 mg (n=6) | Cohort 4 300 mg (n=6) |
|-----------------------------------|----------------------------|-----------------------------|-----------------------------|-----------------------------|
| C _{max} (ng/mL) | 1913 | 3404 | 5956 | 6795 |
| AUC ₀₋₂₄ (ng*hr/mL) | 7826 | 14505 | 29904 | 35406 |
| T _{max} (hr) | 1.2 | 1.3 | 1.0 | 1.5 |
| CL/F (mL/hr) | 6139 | 7162 | 7886 | 7615 |
| Vd/F (mL) | 69580 | 72948 | 117823 | 177190 |
| t _{1/2} (hr) | 8.1 | 7.4 | 10.5 | 17.0 |

Vecabrutinib's Superior PK Profile Critical for Non-covalent BTK Activity

| | C_{max} (ng/mL) | AUC_{0-24} (ng*hr/mL) | $t_{1/2}$ (hr) |
|--|----------------------|----------------------------|-------------------|
| Vecabrutinib 50 mg (single dose) ¹ | 1913 | 7826 | 8.15 |
| Ibrutinib 560 mg (single dose) ^{2,3} | 141 | 682 | 4 to 6 |
| Acalabrutinib 100 mg BID (steady-state) ⁴ | 827 | 1850 | 1.13 |
| BGB-3111 160 mg (single dose) ⁵ | ~300 | | |

¹ Neuman et al., ASH 2016; Vecabrutinib values are for the free base

² IMBRUVICA® (ibrutinib) capsules, for oral use [prescribing information]. Sunnyvale, CA: Pharmacyclics LLC; 2017.

³ Center for Drug Evaluation and Research. 205552 Clinical pharmacology review (Imbruvica™). July 30, 2013.

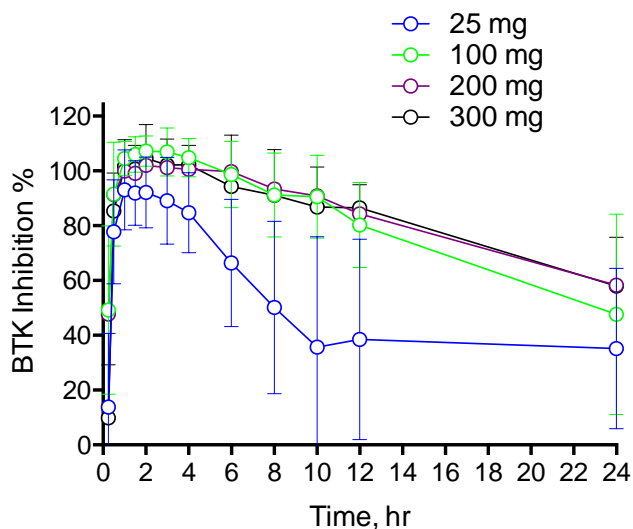
⁴ Byrd et al., *N Engl J Med.* 2016;374:323-32.

⁵ Tam et al., ASH 2015



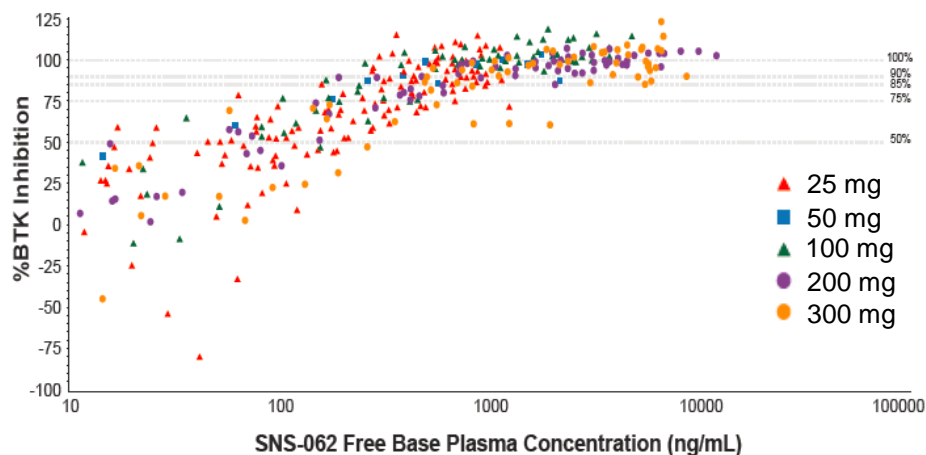
Phase 1A: Profound and Sustained Target Inhibition

Percent BTK Inhibition Over Time



- Vecabrutinib demonstrated rapid, near complete inhibition of pBTK at all dose levels
- Prolonged inhibition seen at dose levels of 100 mg and higher, supporting BID dosing

BTK Inhibition vs Vecabrutinib Plasma Levels



- pBTK inhibition $\geq 85\%$ observed at vecabrutinib concentrations ≥ 200 ng/mL
 - As concentrations increased, inhibition increased and became less variable
 - Clinical activity anticipated with sustained inhibition of pBTK of $\geq 85\%$

Phase 1A: Safety Outcome from Dose Escalation

- No obvious pattern of dose-dependent toxicity
- All AEs were transient and low grade (all Grade 1 except one Grade 2 headache and fatigue)
- No clinically meaningful AEs, laboratory abnormalities, ECG, or telemetry findings
- No serious AEs

| | Cohort 1 50 mg (n=6) | Cohort 2 100 mg (n=6) | Cohort 3 200 mg (n=6) | Cohort 4 300 mg (n=6) | Total Active (n=24) | Placebo (n=8) |
|------------------------------------|----------------------------|-----------------------------|-----------------------------|-----------------------------|---------------------------|------------------|
| Subjects with AEs n (%) | 4 (67%) | 1 (17%) | 1 (17%) | 2 (33%) | 8 (33%) | 3 (38%) |
| Treatment related | | | | | | |
| Headache | 4 (67%) | 0 | 0 | 1 (17%) | 5 (21%) | 2 (25%) |
| Diarrhea | 0 | 0 | 0 | 0 | 0 | 1 (13%) |
| Nausea | 0 | 0 | 1 (17%) | 0 | 1 (14%) | 2 (25%) |
| Supraventricular tachycardia (SVT) | 0 | 0 | 0 | 1 (17%) | 1 (4%) | 0 |
| Treatment unrelated | | | | | | |
| Constipation | 0 | 1(17%) | 0 | 0 | 1(4%) | 0 |
| Fatigue | 0 | 0 | 0 | 1 (17%) | 1 (4%) | 0 |
| Orthostatic hypotension | 0 | 0 | 0 | 1 (17%) | 1 (4%) | 0 |
| Bronchitis | 0 | 0 | 0 | 1 (17%) | 1 (4%) | 0 |

Unique and Differentiated Clinical-Stage BTK Inhibitor

- Commercial and late-stage BTK Inhibitors are ***covalent/irreversible inhibitors***
- Vecabrutinib (SNS-062) is ***non-covalent/reversible inhibitor***
 - With reported human PK/PD clinical data
 - With selective kinase activity including potent BTK and ITK inhibition

| BTK-Inhibitor | Company | Stage - CLL | Binding Profile |
|---------------------|--------------------|-------------------|----------------------------|
| Ibrutinib | AbbVie/Janssen | Marketed | <i>Covalent</i> |
| Acalabrutinib | AstraZeneca/Acerta | Phase 3 | <i>Covalent</i> |
| BGB-3111 | Beigene | Phase 2 | <i>Covalent</i> |
| Vecabrutinib | Sunesis | Phase 1b/2 | <i>Non-covalent</i> |
| ARQ 531 | Arqule | Phase 1 | <i>Non-covalent</i> |
| Loxo-305 | Loxo (Redx) | Preclinical | <i>Non-covalent</i> |



Vecabrutinib is a Selective Kinase Inhibitor

| | TEC Family Kinases | | | | | Other Kinases | | | | | |
|---------------------|--------------------|-----|------------------|------|------|------------------|-------|-------------|-----|--------|-------|
| | BTK | ITK | TEC [†] | TXK* | BMX* | LCK [†] | cSRC* | SRC (1-530) | BLK | NEK11* | EGFR* |
| IC ₅₀ nM | 3 | 14 | 14 | 474 | 224 | 8 | 84 | 30 | 23 | 90 | >6000 |

*Determined with vecabrutinib free base.

[†]Activated.

- Inhibits BTK with nanomolar potency
- Highly selective: only 4 non-Tec kinases inhibited with IC₅₀ < 100 nM in 234-kinase kinome screen
- Lack of EGFR inhibition: 1000-fold less potent than ibrutinib
- **Emerging ITK role in CLL:**
 - Improves T cell number and function¹
 - Both BTK and ITK inhibition contribute to ibrutinib activity in chronic graft-versus-host disease²

¹ Long et al., JCI 2017

² Ibrutinib prescribing information 2017; Miklos et al., Blood 2017; Schutt et al., PLoS 2015



Kinase Inhibition Profile of BTK Inhibitor Pipeline

| | TEC Family Kinases | | | | | Other Kinases | | | | |
|--------------|----------------------------|------|------------------|------------------|------------------|------------------|------------------|-------|-------------------|-------|
| | BTK | ITK | Tec [#] | TXK [*] | BMX [*] | LCK [#] | SRC [*] | BLK | EGFR [*] | |
| covalent | Ibrutinib ² | 0.5 | 10.7 | 78 | 2.0 ³ | 0.8 | 33.2 | 171 | 10.7 | 5.6 |
| | Acalabrutinib ³ | 5.1 | >1000 | 93 | 368 | 46 | >1000 | >1000 | >1000 | >1000 |
| | BGB-3111 ⁴ | 0.22 | 30 | 1.9 | n/a | n/a | n/a | n/a | n/a | n/a |
| non-covalent | Vecabrutinib ¹ | 3 | 14 | 14 | 474 | 224 | 8 | 84 | 14 | >6000 |
| | ARQ 531 ⁵ | 4.23 | >10000 | 5.8 | 36.4 | 5.23 | 3.86 | 57.7 | 9.71 | 290 |
| | Loxo-305 ⁶ | 8.7 | >15597 | 181 | 220 | 1410 | 433 | 581 | 1589 | 959 |

n/a=not available

* Determined with vecabrutinib free base

Activated

¹ Neuman et al., ASH 2016

² Honigberg et al., PNAS 2010

³ Byrd et al., NEJM 2016

⁴ Tam et al., ASH 2016

⁵ Eathiraj et al., Pan Pacific Lymphoma Conference 2016

⁶ Guisot et al., ASH2016 and Furman, CLL Expert Forum 2017

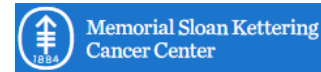


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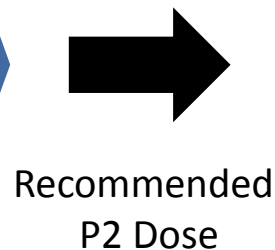
Vecabrutinib Phase 1b/2 B-Cell Malignancy Study

- Objectives:
 - Establish recommended Phase 2 dose (RP2D)
 - Assess safety & activity in CLL/SLL, MCL, WM, DLBCL-ABC, &FL
- Key features:
 - Patients with relapsed disease after 2 or more lines of treatment
 - CLL/SLL, MCL, WM patients must have failed a covalent BTK
 - Phase 1b: 3+3 dose escalation to identify RP2D
 - Dose cohorts: 25mg, 50mg, 100mg, 200mg, 300mg, 400mg, 500mg given BID
 - Phase 2 expansion cohort(s) to establish safety / efficacy
- Phase 1b conducted in US; Phase 2 expansion in US and EU
 - Sites include: Dana Farber, Cornell, MD Anderson, UC Irvine, Swedish, & MSKCC



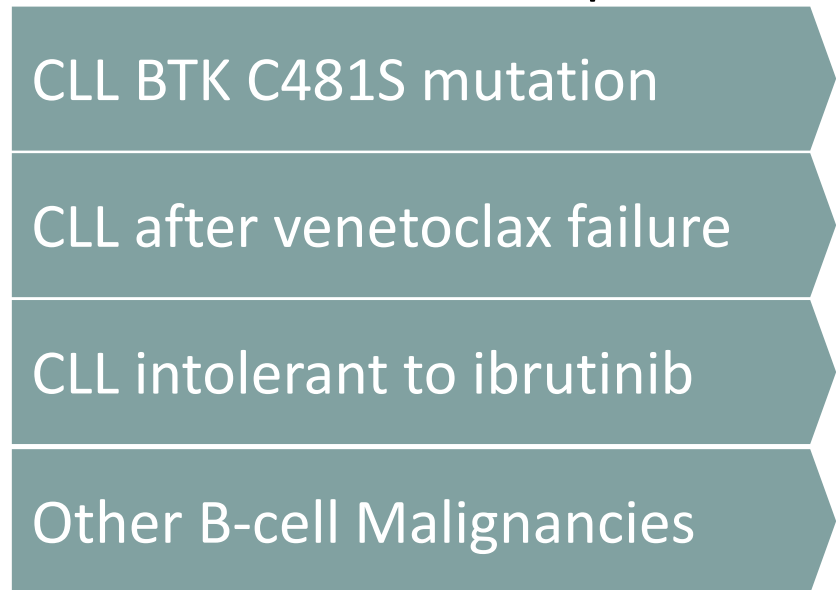
Vecabrutinib Phase 1b/2 Study Schema

Phase 1b Dose Escalation



*CLL/SLL, MCL, WM, DLBCL-ABC, FL

Phase 2 Cohort Concepts#



R/R disease

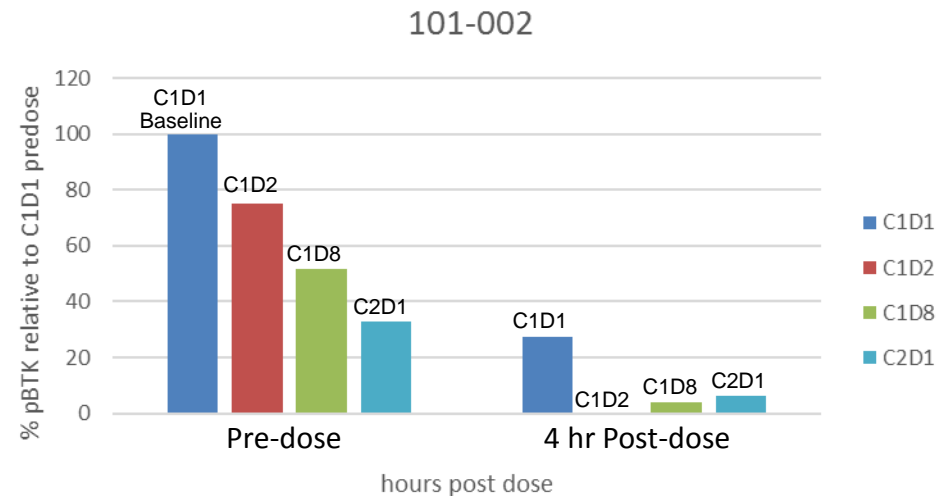
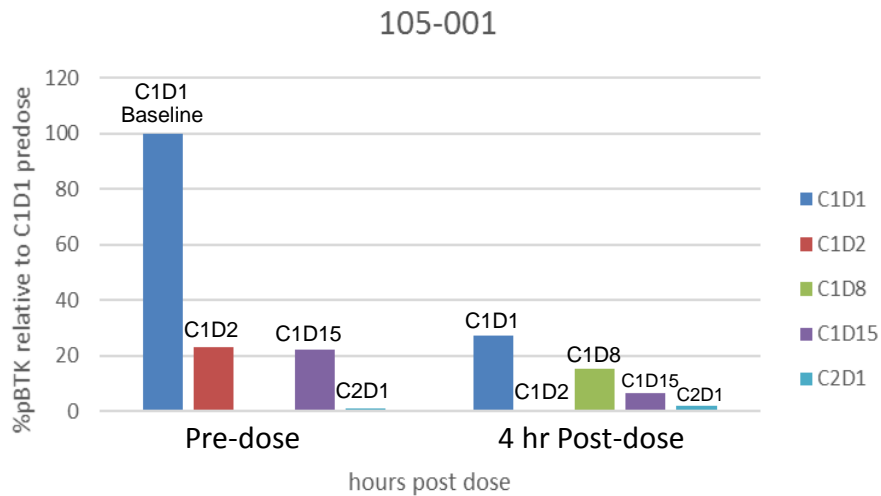
Preliminary Data – 25 mg BID Cohort

| | 101-001 (DFCI) | 101-002 (DFCI) | 105-001 (MDACC) |
|-----------------------------------|--|--|--|
| Age (years). Sex | 47, Male | 75, Female | 72, Female |
| Diagnosis | CLL | CLL | CLL |
| Molecular Profile | C481S, 13q del | 17p del | C481S, 17p del |
| Prior Therapy – (Response) | <ul style="list-style-type: none"> • 12/07 – 06/08: FCR (PR) • 06/11 – 11/16: bendamustine, rituximab, ibrutinib, then ibrutinib (PR) • 11/16 – 07/17: idelalisib + ACY-1215 (SD) | <ul style="list-style-type: none"> • 09/13 – 11/13: BR (unknown) • 2014: High dose methylprednisone (PR) • 04/14 – 11/16: ibrutinib (unknown) • 11/16 – 09/17: venetoclax (PR) | <ul style="list-style-type: none"> • 11/12 – 03-13: alemtuzumab (CR) • 02/14 – 09/17: BR (PR) • 03/14 – 09/17: acalabrutinib (PR) |



Preliminary Data – 25 mg BID Cohort

BTK Phosphorylation Inhibition from Ongoing Ph1b/2 Study



- Clear pBTK inhibition observed in subjects 105-001 and 101-002
- Pharmacokinetics consistent with Phase 1a healthy volunteer study

Proprietary Pipeline

SNS-510, *PDK-1 inhibitor*

Vosaroxin, *Topoisomerase II inhibitor*



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SNS-510 PDK1 Inhibitor: Exciting Preclinical Cancer Program

| | |
|-------------------------------------|---|
| Modality | Small-molecule, oral inhibitor of PDK-1 |
| Molecular Hypothesis | <ul style="list-style-type: none">• Major mediator of PI3K signaling and activator of AKT• PI3K-independent activator of RSK and S6K kinases |
| Therapeutic Hypothesis | Potentially broader activity than PI3K/AKT inhibitors both as a single agent and in combination |
| Potential Target Indications | <ul style="list-style-type: none">• <i>Hematologic tumors</i> including CLL, AML, and BCL• <i>Solid tumors</i> including breast, prostate, stomach, lung, colon and pancreatic |
| Program Status | IND filing expected in 2019 |



Vosaroxin Strategic Out-licensing Opportunity

- Positive confirmatory Phase 3 trial could unlock global revenue
- Relapsed/refractory AML in older patients is a true unmet need

VALOR Phase 3 Trial

- Overall ITT showed borderline improvement in overall survival
- Strongest results in *pre-specified* subgroup of patients ≥ 60 years, *with early and sustained separation of the survival curves*
- Reaching CR provides better outcomes (with or without transplant)

| VALOR Results from ≥ 60 yo Early Relapse and Refractory Patients (n=364) | | |
|---|------------------------------|--------------------------|
| Result | Vosaroxin Treated n = 182 | Placebo Group n = 182 |
| CR Rate | 25% | 10% |
| 2-Year Survival | 16% | 5% |
| 3-Year Survival | 13% | 4% |
| Median OS | 6.5 months | 3.9 months |



2018 Milestones



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Potential Pipeline Milestones

| PROGRAM | UPDATE | TIMEFRAME |
|---------------------|---|-----------|
| Vecabrutinib BTK | Interim Data Updates | 2H 2018 |
| | Recommended Phase 2 dose | Fall 2018 |
| SNS-510 PDK1 | Submit IND | 2019 |
| Vosaroxin | Explore strategic out-licensing opportunities | 2018 |



Financial Position and Capitalization

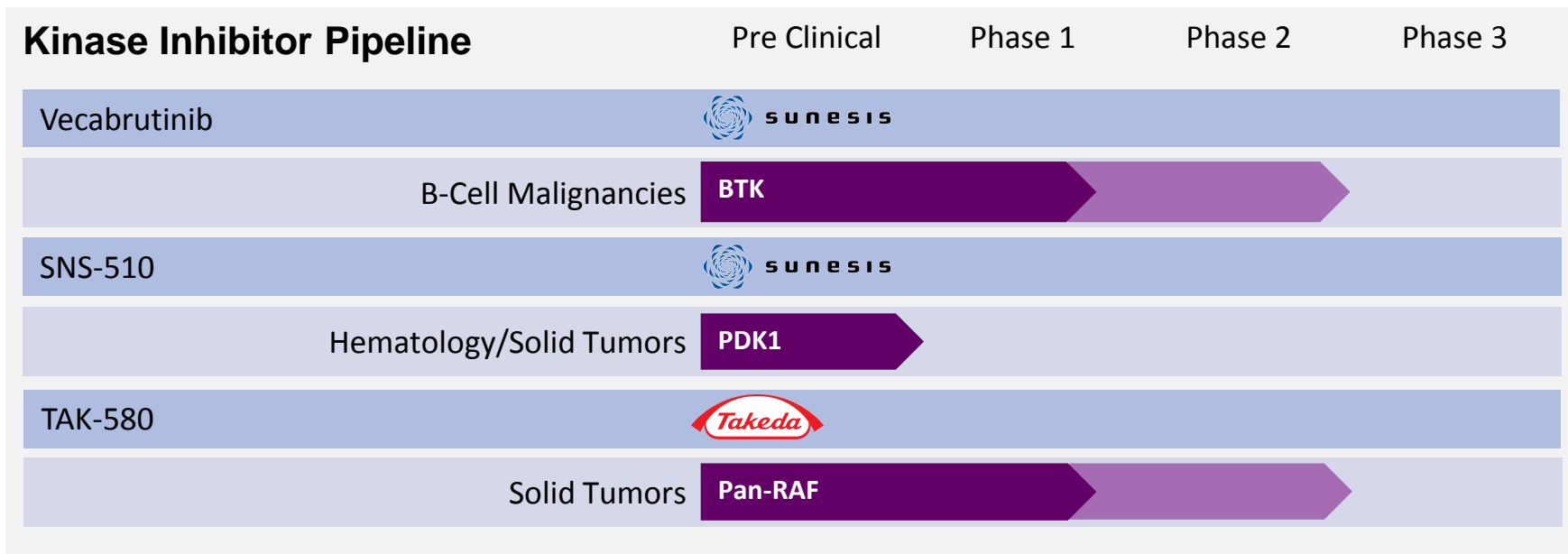
| Ticker | SNSS (NASDAQ) |
|----------------------|--|
| Cash & Equivalents | \$25.4 million ¹ |
| Debt | \$7.5 million ¹ |
| Shares | 41.1 million ² |
| Warrants (lenders) | 0.2 million @ avg \$3.72 ¹ |
| Warrants (investors) | 5.0 million @ \$3.00 ³ |
| Stock Options | 3.3 million @ avg \$5.82 ¹ |
| Top Shareholders | BVF, MPM, Aisling, Eventide, Balyasny, Boxer Capital, Bay City, NEA, Vanguard |
| Covering Analysts | Jim Birchenough (Wells Fargo Securities) Hartaj Singh (Oppenheimer & Co) Marc Frahm (Cowen & Co) |

¹ As of 3/31/18

² As of 6/25/18; includes common (34.4M), preferred as converted (6.3M), and common issued to Aspire (0.4M)

³ As of 3/31/18; 5M warrants expire 10/27/18, cash exercise only

Summary



- Funded through key milestones
- Potential upside via vosaroxin out-licensing



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Thank You