sunesis Pharmaceuticals, Inc.

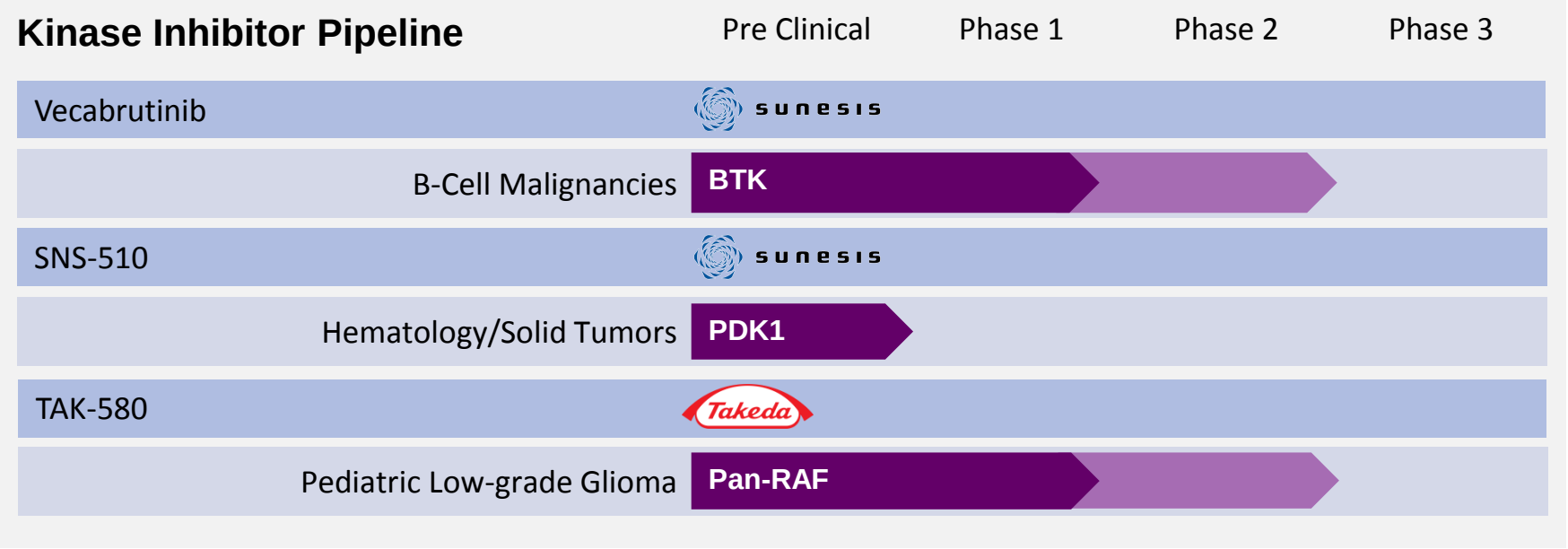
Corporate Update

May 2019

Safe Harbor Statement

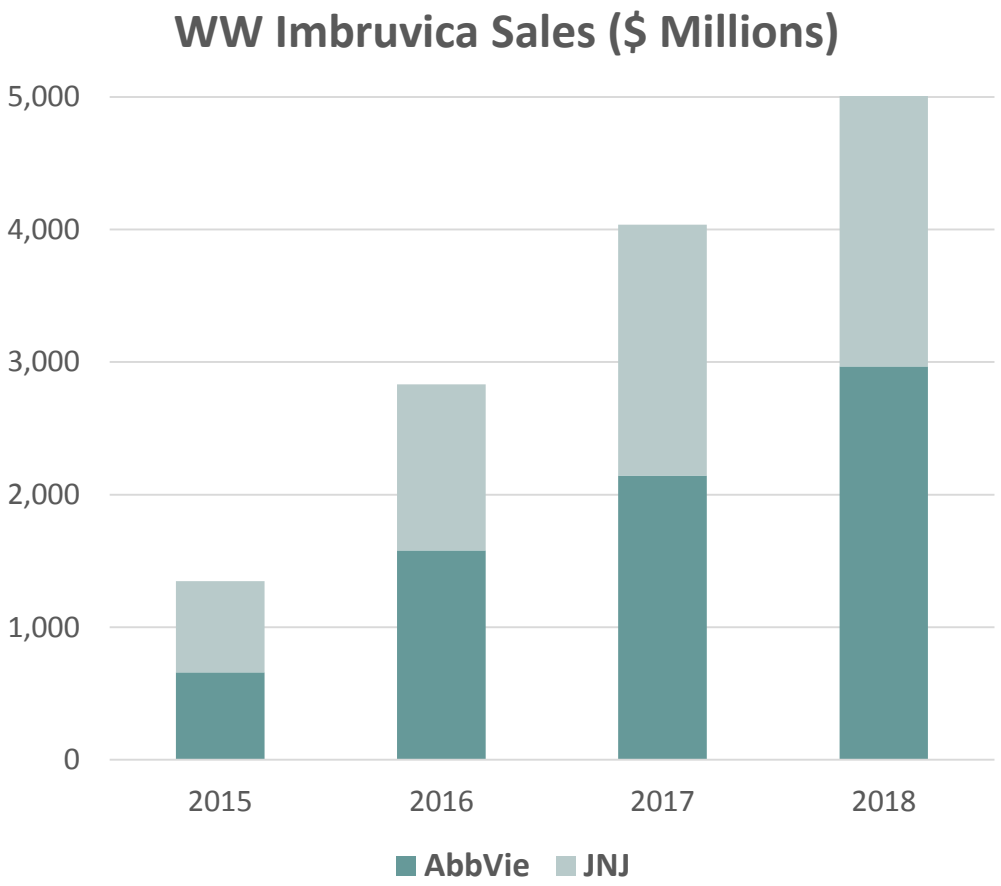
This presentation contains forward-looking statements, including statements related to the continued development of vecabrutinib (SNS-062) and other product candidates, including the timing and preliminary results of Phase 1b/2 trial of vecabrutinib and the therapeutic potential of vecabrutinib, further development and potential of its kinase inhibitor pipeline, and planned development of SNS-510. 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 three months ended March 31, 2019 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



- June 2019: vecabrutinib clinical update at the European Hematology Association annual meeting

Strong Growth Continues for BTK-Inhibitor Market

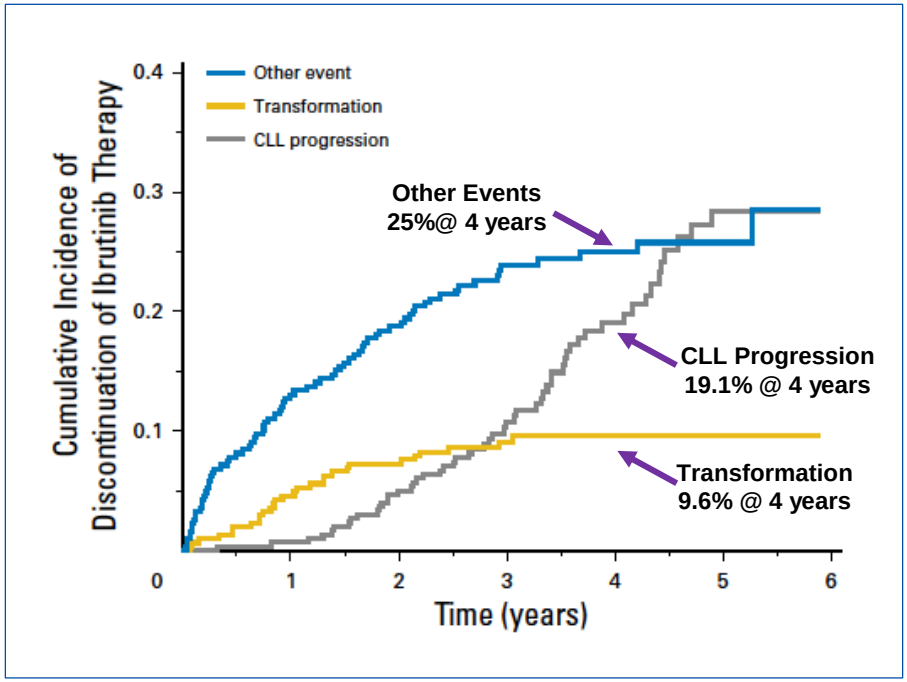


- Imbruvica: first covalent BTK inhibitor approved
- Imbruvica 2018 sales: >$5B
- CLL market share leader (across all lines of therapy)
- EvaluatePharma estimates 2024 sales >$9B

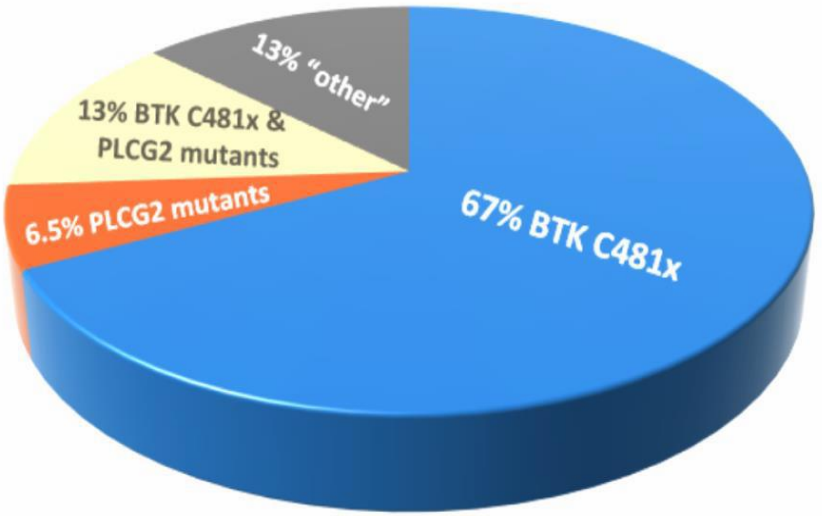
Company annual reports and earnings releases, EvaluatePharma World Preview Report (2018)

Relapse from Ibrutinib Associated with Acquired Resistance Mutations

Landmark Analysis of 308 Ibrutinib-Treated Patients
51% Discontinuation at 4 years



Ibrutinib Acquired Resistance Mutations N=46



- Progression associated with resistance mutations
  - BTK C481x mutations prevent covalent inhibition of BTK
  - BTK C481x mutations appear ~9 months prior to relapse
- Intolerance ("Other event") is a significant source of discontinuation

Woyach et al., J Clin Oncol 2017
Scarfo et al. (ERIC), EHA 2018 and Quinquenel et al. (FILO), ASH 2018

Vecabrutinib

Non-covalent BTK inhibitor

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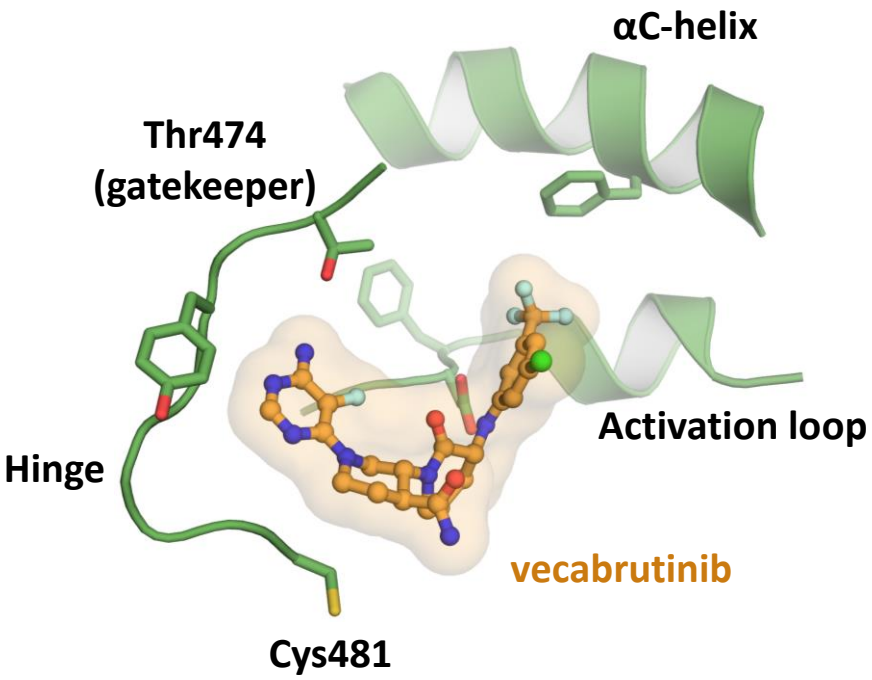
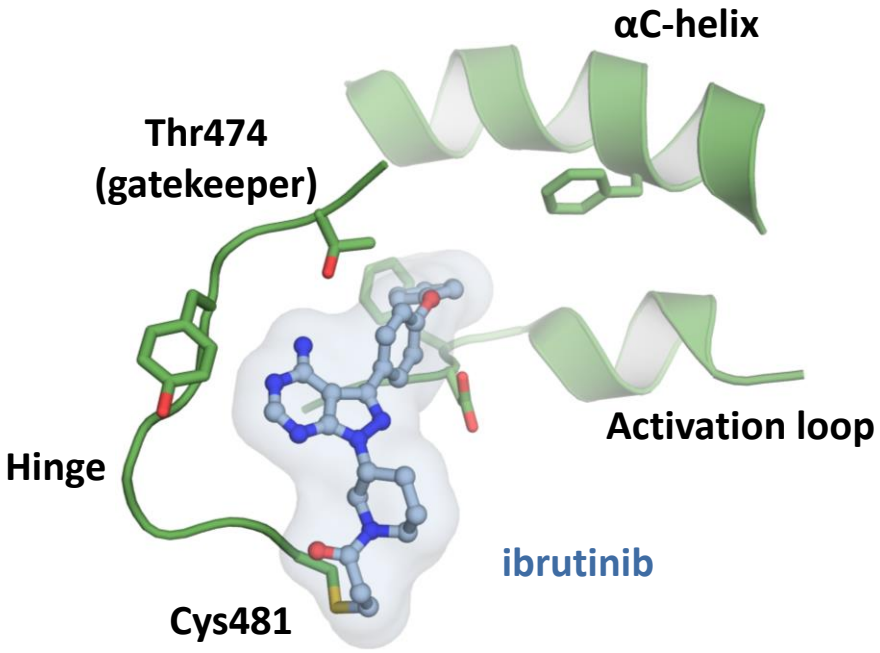
Vecabrutinib BTK Kinase Domain Interaction Differentiated from Ibrutinib





Vecabrutinib distinct kinase domain interaction translates into improved selectivity over ibrutinib and maintained activity against BTK C481S

Vecabrutinib - Selective Kinase Inhibitor

	TEC Family Kinases					Other Kinases				
	BTK	ITK	TEC†	TXK	BMX	LCK†	cSRC	BLK	NEK11	EGFR
IC_{50} nM	3	14	14	474	224	8	84	23	90	>6000

†Activated.

- Inhibits BTK with nanomolar potency
- Highly selective: IC_{50} < 100 nM in only 4 of 234 non-Tec family kinases[1]
- Lack of EGFR inhibition: 1000-fold less potent than ibrutinib
- **Emerging ITK role in CLL and other indications**:
  - Improves T cell number and function[2]
  - Contributes to ibrutinib activity in chronic graft-versus-host disease[3]
  - Improves activity of anti-CD19 CAR-T cells in CLL patients[4]

[1] Sunesis data: 234 kinase kinome screen
[2] Long et al., JCI 2017
[3] Ibrutinib prescribing information 2017; Miklos et al., Blood 2017; Schutt et al., PLoS 2015
[4] Gauthier et al., ASH 2018

Vecabrutinib Differentiated Kinase Inhibition Profile

		TEC Family Kinases				Inhibition of Other Kinases
	IC_{50} (nM)	BTK	ITK	Tec#	TXK*	BMX*	Notable Target Kinases
covalent	**Ibrutinib**[2]	0.5	10.7	78	2.0[3]	0.8	>10 more: EGFR family
	Acalabrutinib[3]	5.1	>1000	93	368	46	Selective
	Zanubrutinib[4]	0.22	30	1.9	n/a	n/a	N/A (not published)
non-covalent	**Vecabrutinib**[1]	3	14	14	474	224	Selective: only 4, including SRC family, NEK11
	ARQ 531[5]	4.23	>10000	5.8	36.4	5.23	>20 more: SRC & TRK families, RAF1, MEK1
	Loxo-305[6]	3.15	>5000	1234	209	1155	Very Selective
	CG-806[7] **(Aptose)**	8.4	4.3	>1000	n/a	14.5	18 w/ IC_{50} <10 nM: FLT3 (wt, ITD) c-MET, TRK family & Aurora kinases

n/a=not available

* Determined with vecabrutinib free base (also relevant for SRC and EGFR)

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; Sun et al., AACR 2014

[5] Eathiraj et al., Pan Pacific Lymphoma Conference 2016

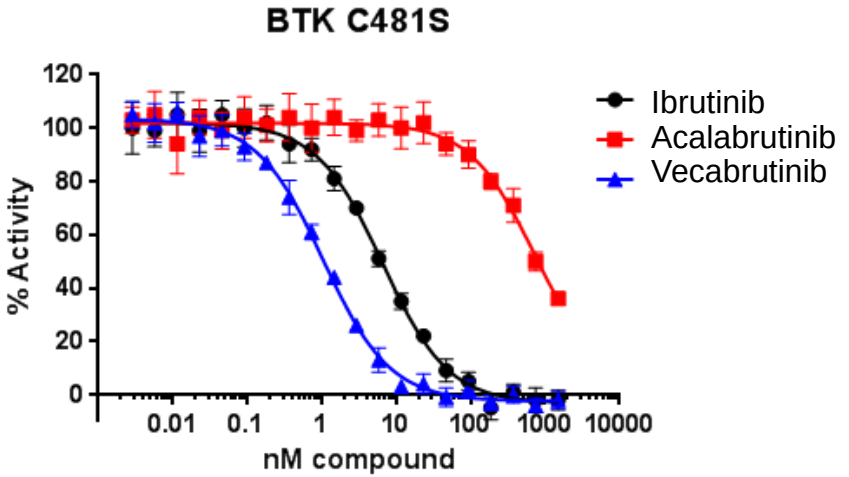
[6] Brandhuber et al., SOHO 2018

[7] Zhang et al., EHA 2018

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

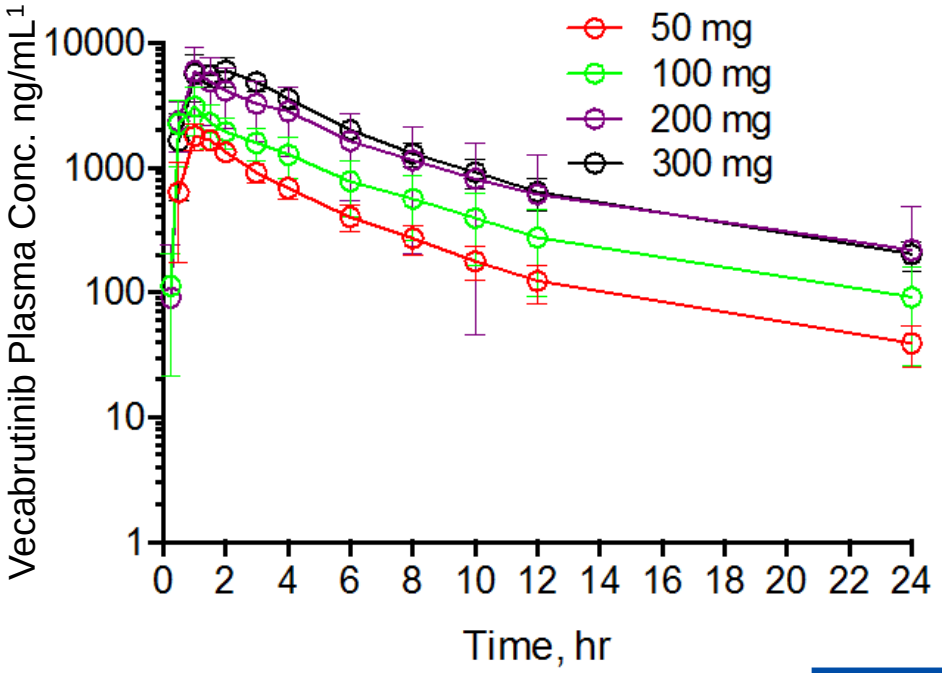
[ATP] = 50 µM

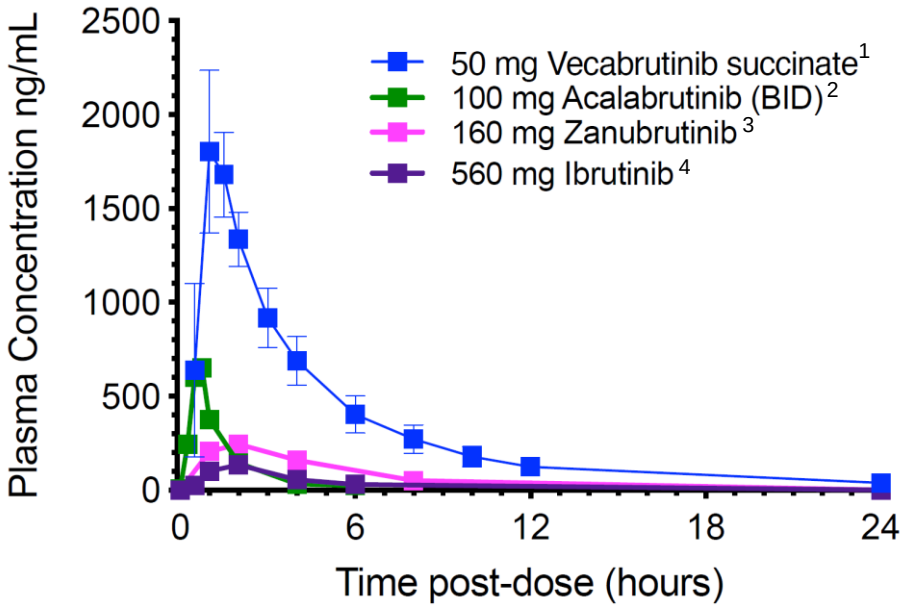


- Activity of ibrutinib and acalabrutinib is profoundly affected[1-4]
  - Poor PK contributes to resistance in patients with BTK C481 mutations
- Vecabrutinib active against BTK C481S mutation and induces apoptosis in BTK-dependent lymphoma cell lines[1-4]
  - Clinical PK profiles suggest target levels that provide sustained BTK inhibition required for clinical activity are achievable[2,5]

[1]Binnerts et al., EORTC-AACR-NCI 2015
[2]Neuman et al., ASH 2016
[3]Fabian et al., AACR 2017
[4]Libre, Salles, Sujobert et al., EHA 2018
[5]062-HEM-102 Sunesis data

Vecabrutinib Superior PK Profile
Critical for Non-covalent BTK Inhibitor Activity





	Vecabrutinib[1]	Ibrutinib[2]	Acalabrutinib[3]
$AUC_{0\text{-}24}$ (ng•hr/mL)	7826	682	1850

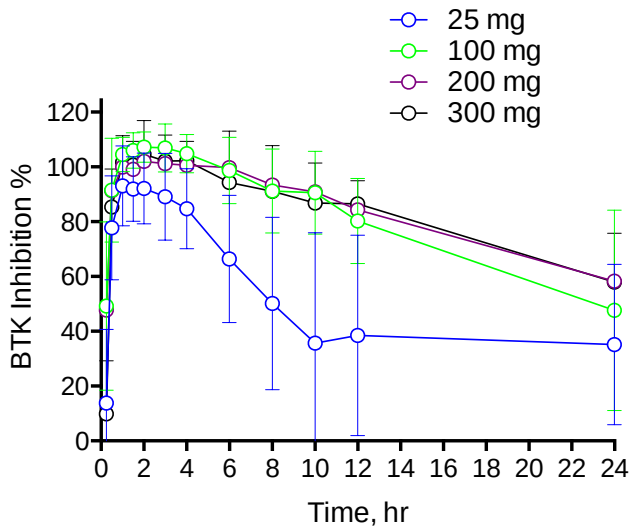
Zanubrutinib AUC is not available

[1] Neuman et al., ASH 2016
[2] Adapted from Byrd et al., NEJM 2016
[3] Adapted from Tam et al., ASH 2015
[4] Adapted from Advani et al., J. Clin. Oncol. 2013
[5] IMBRUVICA® (ibrutinib) prescribing information. Sunnyvale, CA: Pharmacyclics LLC; 2017.

Note: doses are for vecabrutinib succinate; conversion factor to vecabrutinib is 0.82. For example, 100 mg vecabrutinib succinate corresponds to 82 mg vecabrutinib.

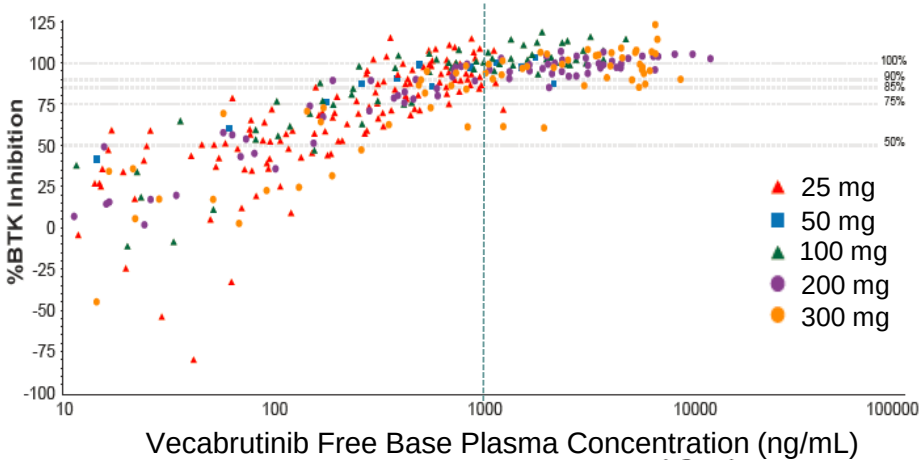
Phase 1a: Sustained Target Inhibition

Percent BTK Inhibition Over Time



- Rapid, near complete inhibition of pBTK at all dose levels
- Prolonged inhibition seen at dose levels of ≥ 100 mg, supporting BID dosing
- Target dose level in patients likely 100-300 mg BID

BTK Inhibition vs Vecabrutinib Plasma Levels



- As concentrations increased, inhibition increased and variability decreased
- Target trough levels adequate to maintain high BTK inhibition over dose interval

Note: doses are for vecabrutinib succinate; conversion factor to vecabrutinib is 0.82. For example, 100 mg vecabrutinib succinate corresponds to 82 mg vecabrutinib.

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Vecabrutinib in B-Cell Malignancies

Phase 1b/2 Trial
Status and Preliminary Data

Vecabrutinib in B-Cell Malignancies
Phase 1b/2 Study Design

Modified 3+3 Design

Or continue escalation

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

- ≥ 2 prior regimens, includes BTK inhibitor if approved for the indication

Phase 2
Potential Expansion Cohorts

- R/R CLL w/ BTK C481x mutation
- R/R CLL no BTK C481x mutation
- R/R CLL venetoclax failure
- CLL ibrutinib intolerant
- B-cell lymphomas

Opportunity to explore areas of high unmet need

Note: doses are for vecabrutinib succinate; conversion factor to vecabrutinib is 0.82. For example, 100 mg vecabrutinib succinate corresponds to 82 mg vecabrutinib.

Vecabrutinib in B-Cell Malignancies
Phase 1b/2 Study Status

Currently open in United States at 9 sites:

Weill Cornell Medical College

Chao Family Comprehensive Cancer Center
University of California · Irvine
A National Cancer Institute-Designated Comprehensive Cancer Center

Memorial Sloan Kettering Cancer Center

DANA-FARBER CANCER INSTITUTE

SWEDISH

UC San Diego Health Sciences

The University of Texas MD Anderson Cancer Center
Making Cancer History®

The US Oncology Network
WVCI

MOFFITT CANCER CENTER

- Status
  - Completed first three cohorts
  - 100 mg cohort:
    - 5 patients completed 28-day safety evaluation period
    - No DLTs or drug-related SAEs
  - 200 mg cohort is enrolling

Note: doses are for vecabrutinib succinate; conversion factor to vecabrutinib is 0.82. For example, 100 mg vecabrutinib succinate corresponds to 82 mg vecabrutinib.

Vecabrutinib Preliminary Data – ASH 2018
Baseline Characteristics

All Subjects N=11[a]	
Indication	7 CLL, 2 MCL, 2 WM
Median age (range) years	66 (47-75)
ECOG PS 0	3 (27%)
ECOG PS 1	8 (83%)
Prior therapies median (range)	5 (2-7)
≥1 Chemotherapy	11 (100%)
BTKi	11 (100%) 10 (ibrutinib) 1 (acalabrutinib)
Venetoclax	5 (45%)
CAR-T	2 (18%)
TP53 mutations/deletions	8 (73%)

CLL Subjects N=7[b]	
Unmutated *IGHV*	5 (71%)
TP53 mutations /deletions	6 (86%)
BTK C481 Mutations[c]	4 (57%)
BTK C481S	3 (43%)
	VAF 16%, 55%, 91%
BTK C481R	1 (14%)
	VAF 77%

All as N (%) unless otherwise noted; VAF=variant allelic frequency

[a] Data available for 11 of 13 subjects

[b] Data available for 7 of 9 CLL subjects

[c] No BTK C481 mutations in WM or MCL, no PCLγ2 mutations at baseline

Allan et al. (ASH 2018 – Poster # 3141)

Vecabrutinib Preliminary Data – ASH 2018
Safety N=10

Adverse Event	All Grades >15% N(%)[a]	Grade ≥ 3 N	Related, Grade ≥ 3 N
Anaemia	7 (70)	6	1
Neutropenia	5 (50)	5	1
Night sweats	5 (50)		
AST increased	4 (40)	1	
Thrombocytopenia	4 (40)	4	
Hypoalbuminaemia	3 (30)		
Hypocalcaemia	3 (30)	1	
Pyrexia	3 (30)		
Abdominal distension	2 (20)		
ALT increased	2 (20)	1	1[b]
Back pain	2 (20)		
Alk phos increased	2 (20)	1	
Cellulitis	2 (20)	1	
Chills	2 (20)		

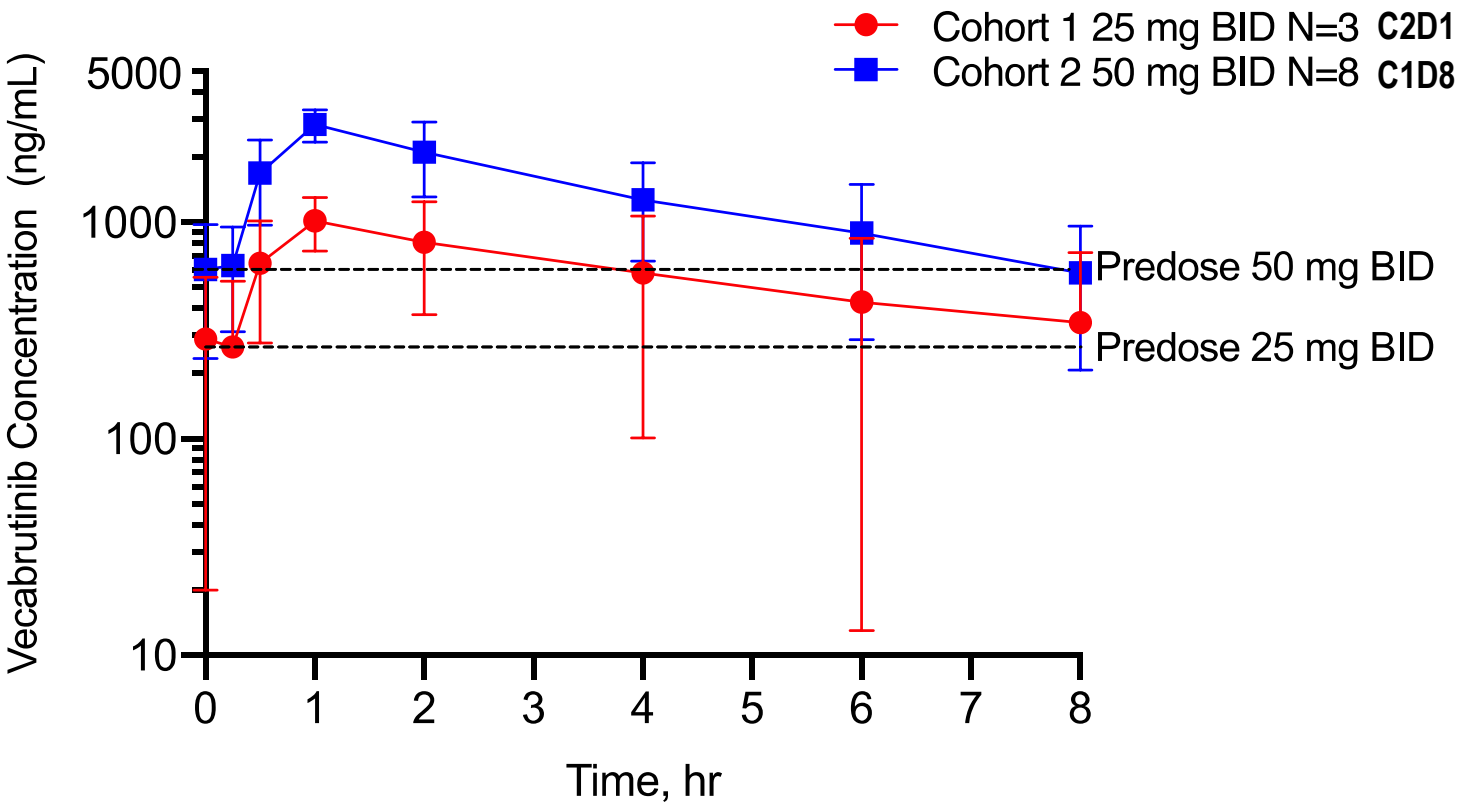
Adverse Event	All Grades >15% N(%) [a]	Grade ≥ 3 N	Related, Grade ≥ 3 N
Constipation	2 (20)		
Cough	2 (20)		
Diarrhea	2 (20)		
Dyspepsia	2 (20)		
Dyspnoea	2 (20)		
Fatigue	2 (20)	1	
Haematuria	2 (20)		
Headache	2 (20)		
Hyperglycaemia	2 (20)	1	
Hyperkalaemia	2 (20)		
Hypermagnesemia	2 (20)		
Hyponatremia	2 (20)	1	
Leukopenia	2 (20)	2	
Lymphopenia	2 (20)	2	

Additional Grade ≥3, N=1: Blood bilirubin increased, Hyperuricaemia, Hypophosphataemia, Intestinal perforation, Leukocytosis (related), Neutrophil count decreased, Pneumonia, Platelet count decreased.

[a]Preliminary safety data available for 10 of 13 treated subjects
[b]One patient in Cohort 2 experienced a DLT of an inadequate number of Cycle 1 doses administered due to a related grade 3 ALT increase

Allan et al. (ASH 2018 – Poster # 3150)

Vecabrutinib Steady-State Mean PK Profiles in B-Cell Malignancy Patients Consistent with Healthy Subjects

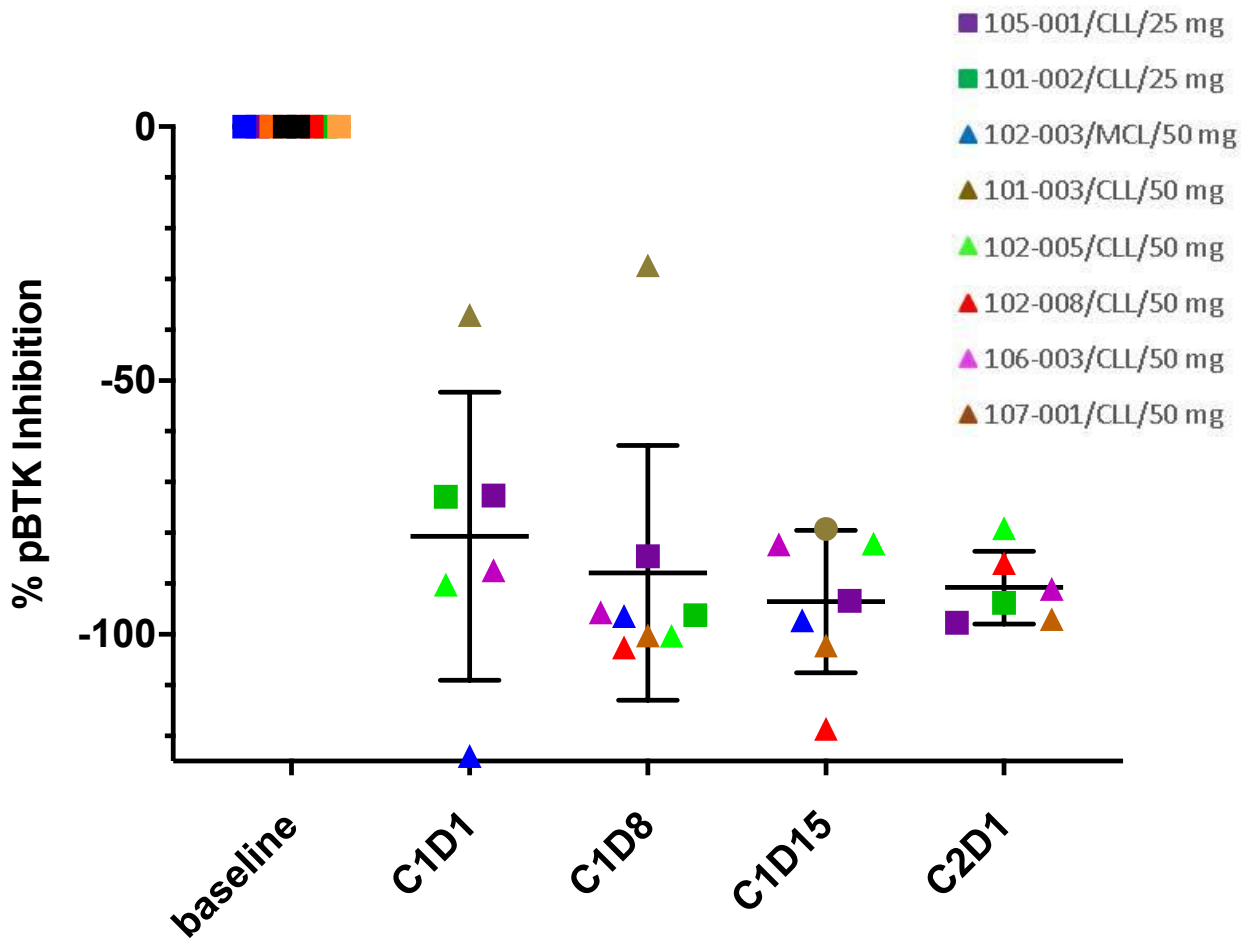


- Generally dose proportional
- Sustained vecabrutinib exposure over dosing interval

Allan et. al. (ASH 2018 – Poster # 3141); Sunesis internal data

Note: doses are for vecabrutinib succinate; conversion factor to vecabrutinib is 0.82. For example, 100 mg vecabrutinib succinate corresponds to 82 mg vecabrutinib.

Vecabrutinib Treatment Induces Rapid and Sustained Inhibition of BTK Phosphorylation

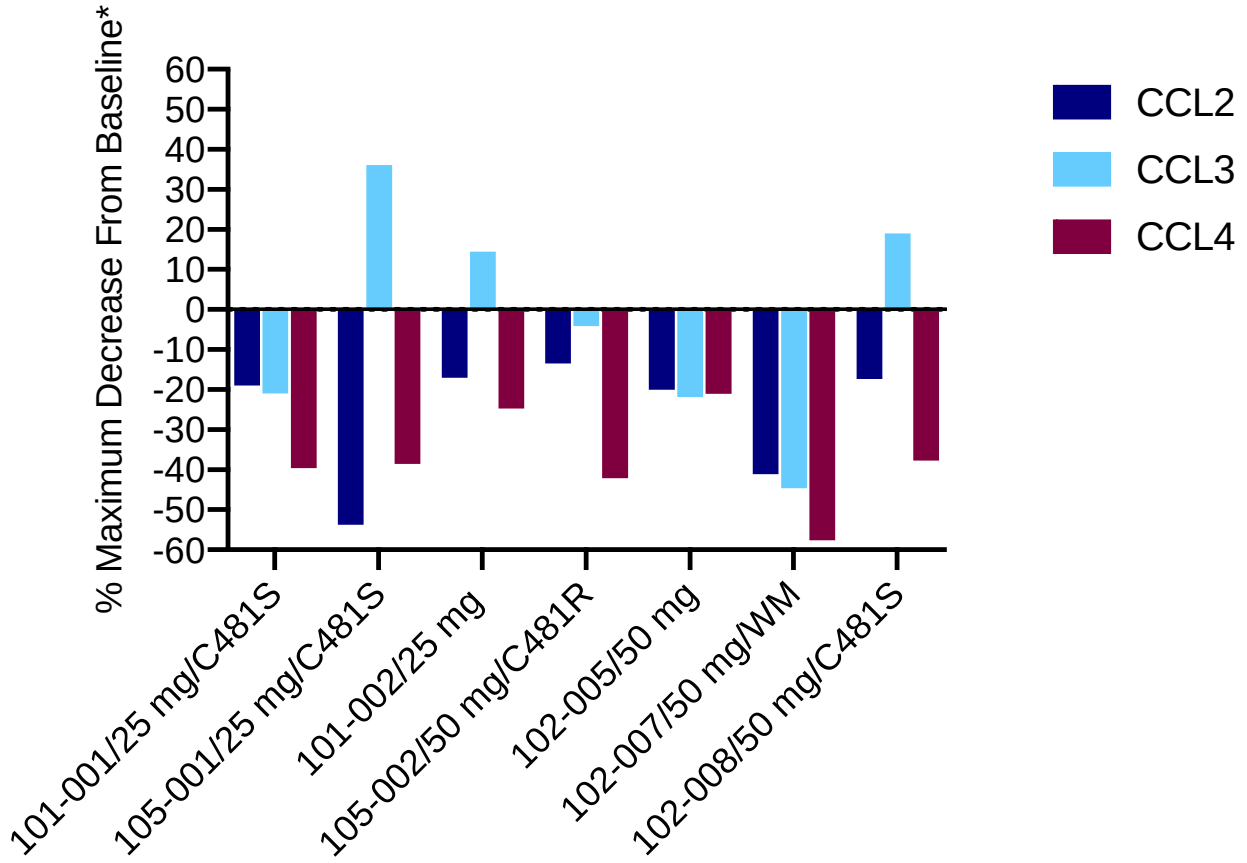


Samples taken at 1 or 4 hours after dosing

Allan et al. (ASH 2018 – Poster # 3141); Sunesis internal data

Note: doses are for vecabrutinib succinate; conversion factor to vecabrutinib is 0.82. For example, 100 mg vecabrutinib succinate corresponds to 82 mg vecabrutinib.

Vecabrutinib Treatment Decreased Serum Cytokines in Subjects Who Completed Cycle 1



- Consistent with inhibition of BTK signaling
- Baseline levels higher than previously reported in CLL (may indicate advanced disease)
  - Median (range) pg/mL: CCL2 545.7 (11.8-2561.9), CCL3 320 (68.6-567), CCL4 1039 (22.5-3874.9)
- 4/11 patients did not complete Cycle 1

Allan et. al. (ASH 2018 – Poster # 3141); Sunesis internal data

Summary: Preliminary Vecabrutinib Profile & Status

- Safety – appears well-tolerated in the context of advanced disease
- Pharmacokinetics – sustained exposure over dosing interval and generally dose proportional
- Pharmacodynamics – inhibition of BTK phosphorylation & cytokine reduction seen in patients (including BTK C481S subjects)
- Activity – evidence of clinical benefit at 50 mg in some subjects
  - WM subject improvement in night sweats and fatigue, hemoglobin stabilized
  - CLL subject improvement in fatigue, decreased tumor burden
- Dose escalation ongoing
  - 200 mg cohort is enrolling
  - Next update at EHA in June 2019

Note: doses are for vecabrutinib succinate; conversion factor to vecabrutinib is 0.82. For example, 100 mg vecabrutinib succinate corresponds to 82 mg vecabrutinib.

Proprietary Pipeline
SNS-510, *PDK-1 inhibitor*

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SNS-510 PDK1 Inhibitor: Exciting Preclinical Cancer Program

Modality	Small-molecule, oral inhibitor of PDK-1
Molecular Hypothesis	• Major mediator of PI3K signaling and activator of AKT • PI3K-independent activator of RSK and SGK
Therapeutic Hypothesis	Potentially broader activity than PI3K/AKT inhibitors both as a single agent and in combination
Potential Target Indications	• *Hematologic tumors* including CLL, AML, and BCL • *Solid tumors* including breast, prostate, stomach, lung, colon and pancreatic
Program Status	Completing pharmacology studies & formulation development

Summary

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Financial Position and Capitalization

Ticker	SNSS (NASDAQ)
Cash & Equivalents	24.8 million[1]
Debt	$5.5 million[2]
Shares	83.9 million[3]
Warrants Stock Options	0.2 million @ avg $3.72[1] 4.0 million @ avg $3.43[1]
Top Shareholders	Aisling, BVF, Eventide, JFL Capital, MPM, Samsara BioCapital, Balyasny, Bay City, NEA, and Palo Alto Investors
Covering Analysts	Jim Birchenough (Wells Fargo Securities) Hartaj Singh (Oppenheimer & Co) Marc Frahm (Cowen & Co) Andrew Fein (H.C. Wainwright)

[1] As of 3/31/19

[2] 4/29/19 SVB Loan

[3] As of 5/1/19: 83.9M includes common (72.5M) plus preferred as converted (11.4M)

Sunesis Highlights

- Large and rapidly growing market for BTK inhibitors
- Validated target, clear mechanism of action
- Covalent BTK inhibitor resistance is a growing problem
- Funded to complete vecabrutinib dose escalation and start P2
- Global rights retained for both programs
  - Vecabrutinib (BTK)
  - SNS-510 (PDK1)

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Thank You