sunesis | Pharmaceuticals, Inc.

March 2017

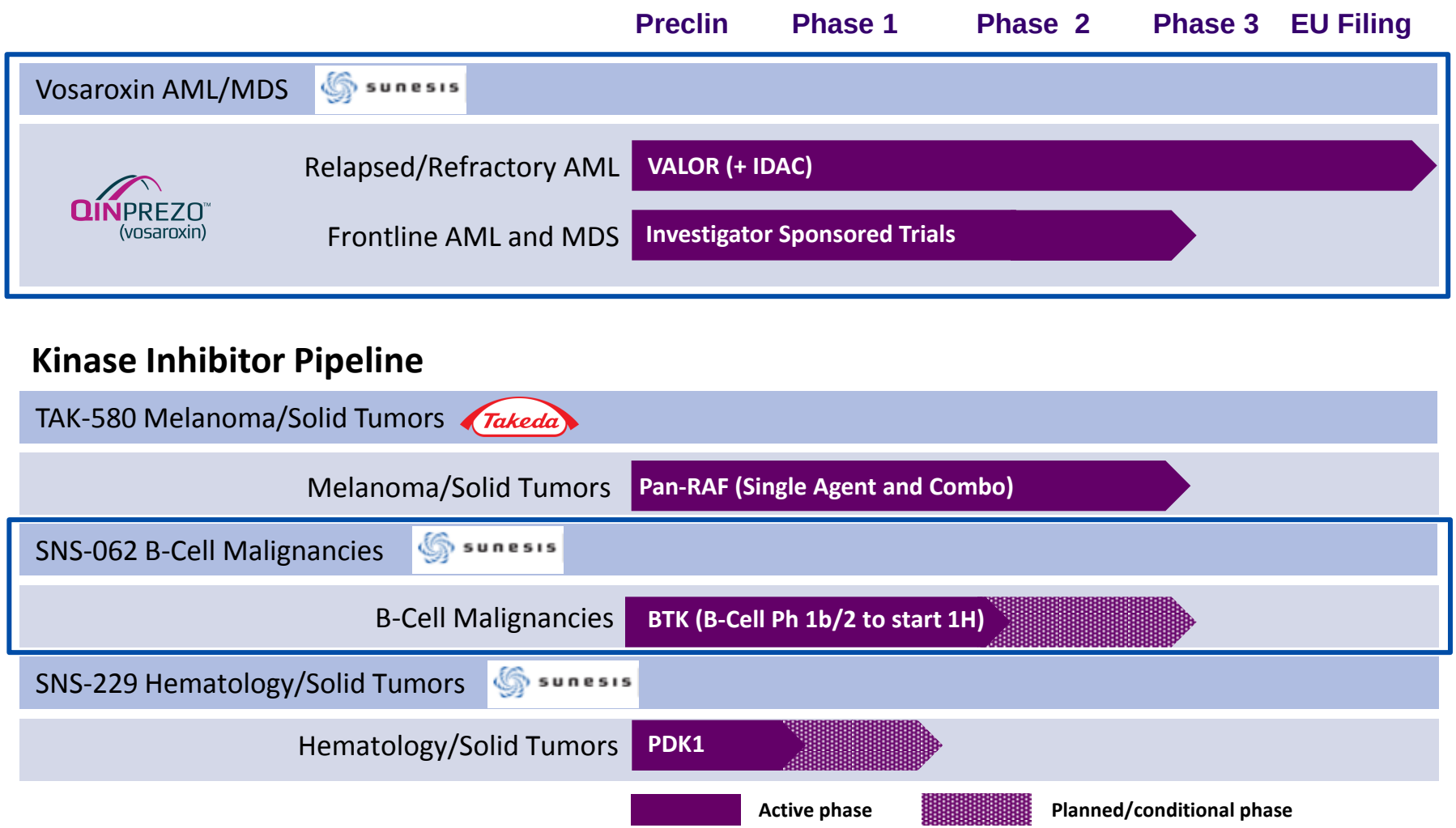
Safe Harbor Statement

This presentation contains “forward-looking” statements – statements that are not historical facts and that involve risks, uncertainties and assumptions, including statements related to Sunesis' overall strategy; outcome of the marketing authorization filing with the EMA; future regulatory interactions with the FDA; Sunesis’ analyses, assessments and conclusions of the results of the VALOR trial; the efficacy and commercial potential of vosaroxin and SNS-062, and outcomes of the ongoing and future in-human studies of SNS-062. 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. These statements are based on estimates and information available to us at the time of this presentation and are not guarantees of future performance. Actual results could differ materially as a result of many factors, including without limitation, risks related to Sunesis' need for substantial additional funding to complete the development and commercialization of vosaroxin and SNS-062, the risk that unfavorable economic and market conditions may make it more difficult and costly to raise additional capital, the risk that Sunesis' development activities for vosaroxin or SNS-062 could be halted or significantly delayed for various reasons, the risk that Sunesis' clinical studies for vosaroxin 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, the risk that Sunesis' proprietary rights may not adequately protect vosaroxin or SNS-062, and the risk that Biogen Idec and Takeda/Millennium may abandon development of the product candidates that are the subject of our collaborations. Additional information concerning these and other risk factors are described under “Risk Factors” and elsewhere in Sunesis’ Form 10-Q for the quarter ended September 30, 2016 and other filings with the Securities and Exchange Commission. You are advised that any forward-looking statements you see or hear during this presentation speak only as of the date of this presentation. We assume no obligation and do not intend to publicly update or revise these forward-looking statements for any reason.

Sunesis Pharmaceuticals, Inc.

- Focused on the development and commercialization of innovative products for the treatment of cancer
- SNS-062 (BTK) and other Kinase Inhibitor Pipeline
  - SNS-062 is a clinical-stage, non-covalent/reversible BTK inhibitor
    - Favorable PK/PD profile from completed Healthy Volunteer study
    - Activated IND with cancer patient Ph1b/2 study to start in 1H
    - Near-term opportunity to address ibrutinib-acquired resistance
  - TAK-580 (Pan-Raf) is an oral once-weekly therapy in clinic partnered with Takeda
  - SNS-229/-510 (PDK1) are in pre-clinical development
- Vosaroxin is a unique Anti-Cancer Quinolone Derivative (AQD)
  - Pivotal Phase 3 VALOR trial in relapse/refractory AML basis for MAA filing
  - Received MAA 180-day list of outstanding issues
  - Potential EU approval and partnership by midyear
  - Investigator-sponsored trials ongoing in AML and MDS
- Experienced management team backed by a strong investor group

Product Pipeline



IDAC = intermediate-dose cytarabine

Kinase Inhibitors: Rapidly Emerging Pipeline

SNS–062 (BTK)

- Potent, selective non-covalent reversible oral inhibitor
- Completed Healthy Volunteer Study and presented at ASH
- Potential in B-cell malignancies and beyond
- Worldwide development and commercial rights

SNS–229/-510 (PDK1)

- Master kinase; central mediator of PI3K and AKT signaling
- Also demonstrates activity independent of AKT pathway
- Potential in both hematologic and solid tumor indications
- Worldwide development and commercial rights

TAK–580 (Pan-RAF)

- Single-agent MTD identified (ASCO 2013) with dose expansion data presented in melanoma patients (EORTC 2014 and ECCO 2015)
- Combo study underway:
  a) MLN0128 (mTORC 1/2),
  b) Alisertib (Aurora A),
  c) Paclitaxel
  d) Cetuximab
  e) Irinotecan
  f) Nivolumab (PD1)

SNS-062: Potential Next-Generation BTK Inhibitor

- Important pathway and leading treatment for B-Cell Malignancies

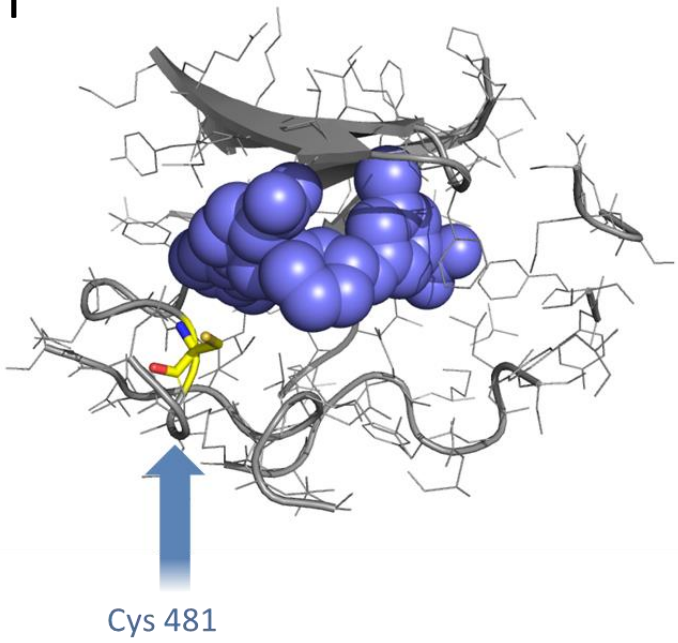
abbvie / pharmacyclics®

Acquired | $21Bn (2015)

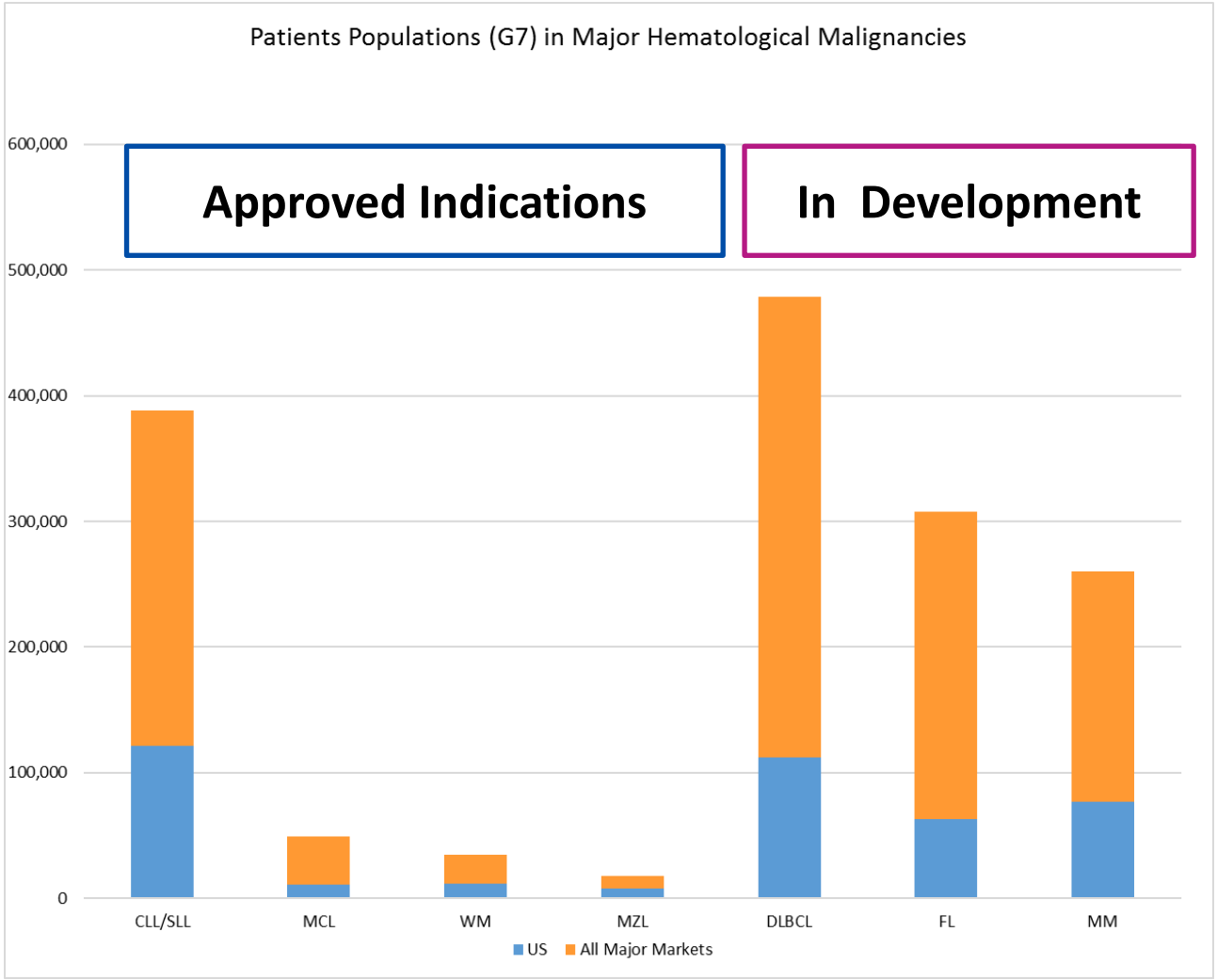
AstraZeneca / AcertaPharma INNOVATE DEVELOP CURE

55% Stake | $4Bn (2015)

- Ibrutinib resistance is a growing concern
  - Caused by acquired mutation at binding site (C481S)
- SNS-062: Potent oral, ***non-covalent reversible*** BTK inhibitor
- Clinical study completed in Healthy Volunteers with excellent PK/PD profile
- IND active with Phase 1b/2 dosing expected in H1 2017



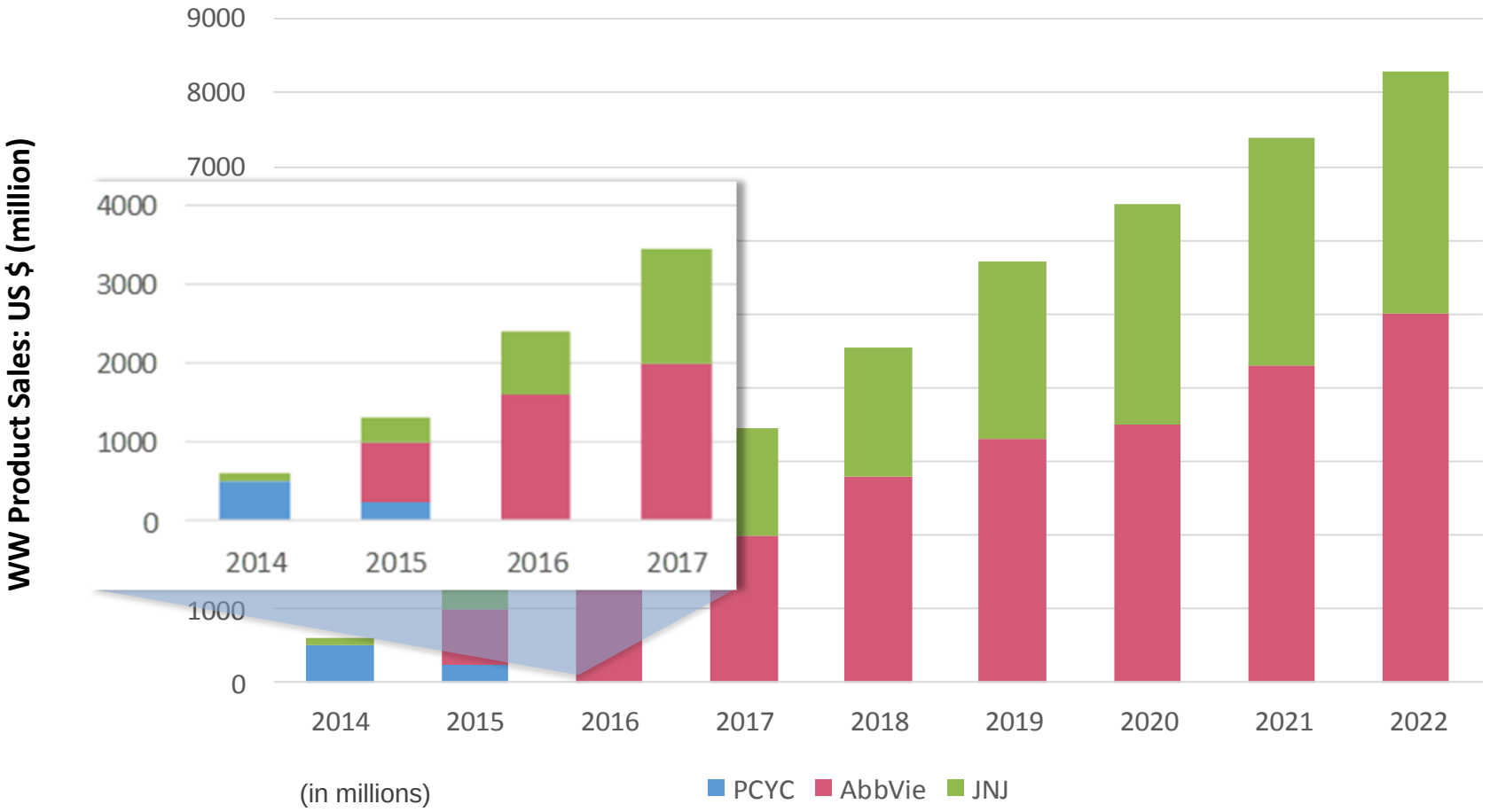
Large Unmet Need for Patients with B-Cell Neoplasms



Sources:

4WM Foundation estimates

Significant Commercial Sales Achieved with Launch of BTK-Inhibitor (ibrutinib)



Source: EvaluatePharma

Unique and Differentiated Clinical-Stage BTK-Inhibitor

- Other clinical BTK Inhibitors are ***covalent/irreversible inhibitors***
- SNS-062 is ***non-covalent/reversible*** with the potential to treat resistant disease

BTK-Inhibitor	Company	Stage	Binding Profile
ibrutinib	Abbvie/Janssen Pharmacyclics	Marketed	***Covalent***
acalabrutinib	AstraZeneca/ Acerta	Phase 3	***Covalent***
BGB-3111	Beigene	Phase 2 (CLL)	***Covalent***
ONO-4059	Gilead/Ono	Phase 1	***Covalent***
SNS-062	Sunesis	Phase 1B/2	***Non-covalent***

Emerging C481S Resistance to Covalent BTK Inhibitors

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Resistance Mechanisms for the Bruton's Tyrosine Kinase Inhibitor Ibrutinib

Jennifer A. Woyach, M.D., Richard R. Furman, M.D., Ta-Ming Liu, M.S., Hatice Gulcin Ozer, Ph.D., Marc Zapatka, Ph.D., Amy S. Ruppert, M.A.S., Ling Xue, Ph.D., Daniel Hsieh-Hsin Li, Ph.D., Susanne M. Steggerda, Ph.D., Matthias Versele, Ph.D., Sandeep S. Dave, M.D., Jenny Zhang, B.S., Ayse Selen Yilmaz, M.S., Samantha M. Jaglowski, M.D., M.P.H., Kristie A. Blum, M.D., Arletta Lozanski, M.S., Gerard Lozanski, M.D., Danelle F. James, M.D., Jacqueline C. Barrientos, M.D., Peter Lichter, Ph.D., Stephan Stilgenbauer, M.D., Joseph J. Buggy, Ph.D., Betty Y. Chang, Ph.D., Amy J. Johnson, Ph.D., and John C. Byrd, M.D.

RESULTS

We identified a cysteine-to-serine mutation in *BTK* at the binding site of ibrutinib in five patients and identified three distinct mutations in *PLCγ2* in two patients. Functional analysis showed that the C481S mutation of *BTK* results in a protein that is only reversibly inhibited by ibrutinib. The R665W and L845F mutations in *PLCγ2* are both potentially gain-of-function mutations that lead to autonomous B-cell–receptor activity. These mutations were not found in any of the patients with prolonged lymphocytosis who were taking ibrutinib.

CONCLUSIONS

Resistance to the irreversible BTK inhibitor ibrutinib often involves mutation of a cysteine residue where ibrutinib binding occurs. This finding, combined with two additional mutations in *PLCγ2* that are immediately downstream of BTK, underscores the importance of the B-cell–receptor pathway in the mechanism of action of ibrutinib in CLL. (Funded by the National Cancer Institute and others.)

N ENGL J MED 370;24 NEJM.ORG JUNE 12, 2014

SNS-062 Retains Activity Against BTK C481S Mutant Cells

Inhibition of Kinase Activity

IC50 (nM)	WT BTK	C481S BTK	Fold Change
Ibrutinib	0.58	25.2	43.4
SNS-062	2.9	4.5	1.6

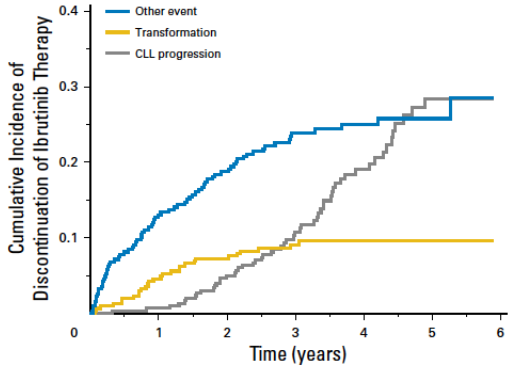
Inhibition of pBTK Levels

IC50 (μM)	WT BTK	C481S BTK	Fold Change
Ibrutinib	0.016	25.5	>1,000
SNS-062	0.57	0.8	1.4

- Activity of ibrutinib is profoundly affected
  - Significant loss of potency combined with poor PK that contribute to a loss of efficacy in presence of BTK C481S mutation
- SNS-062's activity is unaffected by C481S mutation and has favorable PK

Recent JCO Publication: ~90% of CLL Relapse with C481S

- Ohio State recently reported results of a landmark analysis in JCO of 308 CLL patients treated with ibrutinib
- Median follow-up of 3.4 years:
  - 51% discontinued treatment of ibrutinib
- Reasons for discontinuation include:
  - Disease progression 52.5%
  - Adverse events 47.5%
- An estimated 19.1% had progressive CLL by year 4
  - By year 5, ~25-30% with progressive CLL
- Of these, 77.5% had mutations in BTK C481 only
- An additional 15% had mutations in both BTK C481 and PLCG2



Cumulative Incidence Estimates (95% CI)	At 2 Years	At 3 Years	At 4 Years
CLL progression	5.0% (2.5% to 7.5%)	10.8% (7.1% to 14.4%)	19.1% (13.9% to 24.3%)
Transformation	7.3% (4.3% to 10.2%)	9.1% (5.8% to 12.4%)	9.6% (6.2% to 13.0%)
Other event	18.7% (14.3% to 23.1%)	23.9% (19.0% to 28.8%)	25.0% (20.0% to 30.1%)

Source: BTK^{C481S}-Mediated Resistance to Ibrutininb in CLL; Jennifer A. Woyach; J Clin Oncol 35, February 2017

SNS-062 Is a Selective Kinase Inhibitor

	TEC Family Kinases						Other Kinases				
	BTK	ITK	TEC†	BLK	TXK*	BMX*	LCK†	cSRC*	SRC (1-530)	NEK11*	EGFR*
IC_{50} nM	3	14	14	23	474	224	8	84	30	90	6644

*Determined with SNS-062 free base.
†Activated.

- Inhibits BTK potently and shows selective activity
- In 234 kinome screen, only 4 non-Tec kinases inhibited with IC_{50} < 100nM
- 1000-fold less potent against EGFR compared with ibrutinib
- Kinase selectivity and reversible binding profile may provide safety and tolerability benefits

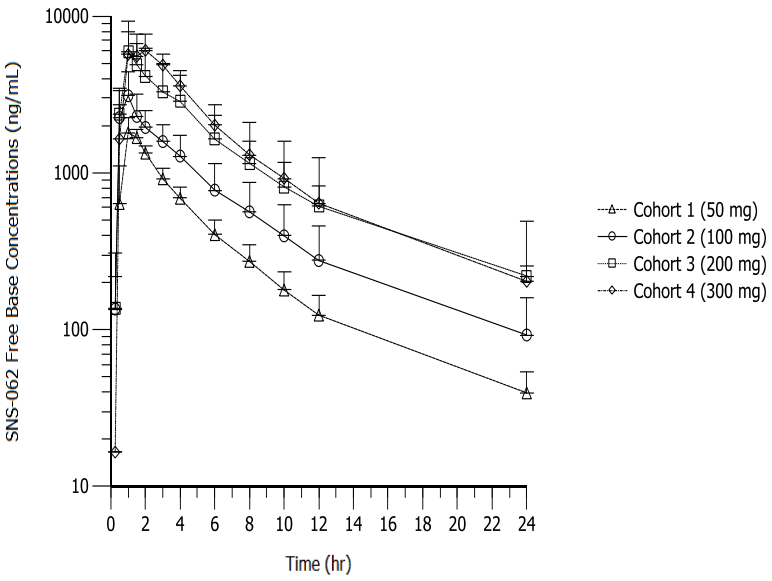
Phase 1A Healthy Volunteers Study: Objectives

- Objectives: evaluate safety, pharmacokinetics (PK), and pharmacodynamics (PD) after a single dose of SNS-062 administered to healthy volunteers
- Dosing began for cohort 1 in March 2016
- First-in-human Phase 1a randomized, double-blind, placebo-controlled, single dose study, conducted in 3 stages
  - Stage 1 (completed):
    - SNS-062 dosing: progressively higher single oral administrations at doses of **50, 100, 200, and 300 mg**
  - Stages 2 and 3 (completed)
    - Evaluated effects of food and CYP3A4 inhibition, respectively, on the PK of SNS-062

Ward R, et al. Presented at 2nd International Conference on New Concepts in B-Cell Malignancies; 9-11 September 2016; Estoril, Portugal.

Phase 1A: Favorable Pharmacokinetic Properties

- SNS-062 was rapidly absorbed, with a median T_{max} = 1 hour (range: 0.5-3.0)
- Mean $t_{1/2}$ values across all dose cohorts ranged from 7.4 to 17 hours
- SNS-062 concentrations declined in a multiphasic manner
- Total exposure (AUC and C_{max}) increased ~proportionally with dose



	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

Data reported as mean, SD

Ward R, et al. Presented at 2nd International Conference on New Concepts in B-Cell Malignancies; 9-11 September 2016; Estoril, Portugal.

Phase 1A: Improved PK Profile to Covalent Binders

- Mean SNS-062 exposure at the lowest dose of 50 mg exceeded exposures reported for both ibrutinib[1-3] and acalabrutinib[4] when administered at their respective recommended dose levels

	C_{max} (ng/mL)	AUC_{0-24} (ng*hr/mL)
SNS-062 50 mg (single dose)	1913	7826
Acalabrutinib 100 mg BID (steady-state)[4]	827	1850
Ibrutinib 560 mg (single dose)[3]	141	682

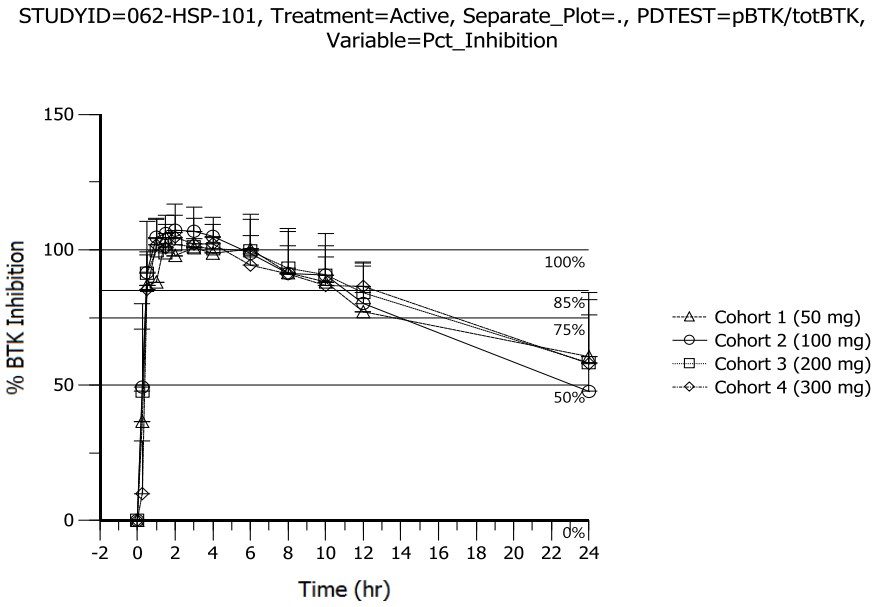
1. Binnerts ME, et al. Mol Cancer Ther. 2015;14 (12 Suppl 2).
2. IMBRUVICA® (ibrutinib) capsules, for oral use [prescribing information]. Sunnyvale, CA: Pharmacyclics LLC; 2016.
3. Center for Drug Evaluation and Research. 205552 Clinical pharmacology review (Imbruvica™). July 30, 2013.
4. Byrd JC, et al. *N Engl J Med*. 2016;374:323-32.

Ward R, et al. Presented at 2nd International Conference on New Concepts in B-Cell Malignancies; 9-11 September 2016; Estoril, Portugal.

Phase 1A: Profound and Sustained Target Inhibition

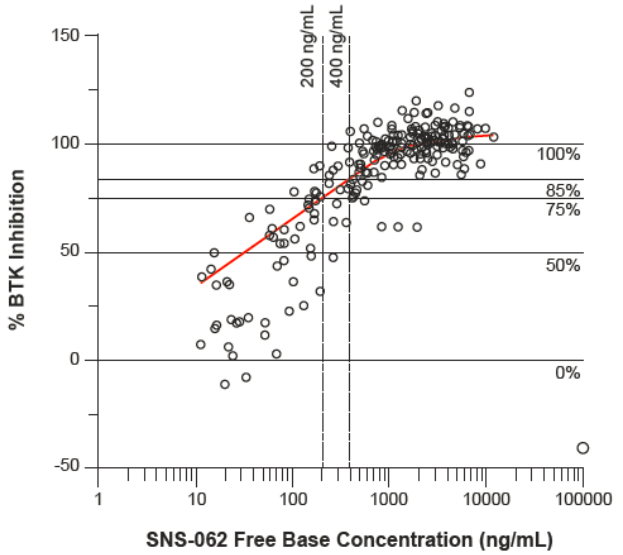
Percent BTK Inhibition Over Time

- SNS-062 demonstrated rapid, profound (~100%), and prolonged (>12 hour) target inhibition



BTK Inhibition vs SNS-062 Plasma Concentration

- It is expected that 85% inhibition is sufficient for clinical activity[1]



% BTK inhibition determined by assessment of pBTK as described in methods; SNS-062 free base concentration determined by LC-MS/MS
BTK, Bruton's tyrosine kinase; pBTK, phosphorylated BTK.

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

Ward R, et al. Presented at 2nd International Conference on New Concepts in B-Cell Malignancies; 9-11 September 2016; Estoril, Portugal.

Phase 1A: Safety Outcomes from Dose Escalations

- Investigators were blinded as to treatment arm when determining relatedness of AE
- No obvious pattern of dose-dependent toxicity
- All AEs were transient and low grade
- 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

AE, adverse event; TEAE, treatment-emergent adverse event.

Ward R, et al. Presented at 2nd International Conference on New Concepts in B-Cell Malignancies; 9-11 September 2016; Estoril, Portugal.

Conclusions from Phase 1A Study

- Well tolerated in healthy subjects following single-dose administration
- Rapidly absorbed and orally bioavailable
- PK properties improved over ibrutinib and acalabrutinib
- PK and PD profiles support twice daily dosing
- Rapid and near complete inhibition of pBTK observed at all dose levels
- PK/BTK inhibition profile suggests potential for clinical activity in B-lymphoid malignancies with either mutant C481 or wild-type BTK
- May be administered with/without food; is a sensitive substrate of CYP3A4
- Results support planned Phase 1b/2 study in advanced B-cell malignancies after prior ibrutinib exposure

Phase 1b/2 Cancer Study to Initiate 1H

- U.S. IND Active (January 2017)
- Open-label trial initially at 5 top U.S. centers
- Treat patients with progressed B-cell malignancies (Q2)
  - CLL
  - Mantle Cell
  - Waldenstrom's
- Dose-escalation to identify RP2 dose followed by Ph2 cohort expansions:
  - 3 + 3 design for Ph1b in "all comers"
  - In Ph2 at recommended dose:
    - CLL progressed with documented C481S mutations
    - Plus cohorts in other malignancies
- To evaluate:
  - Safety
  - Pharmacokinetics (PK) and pharmacodynamics (PD)
  - Antitumor activity (ORR)

PDK1: Exciting New Cancer Program (Preclinical)

Modality	Small-molecule, selective oral inhibitor of PDK-1
Molecular Hypothesis	Major mediator of PI3K signaling with unique, additional effects on survival and invasion beyond PI3K and AKT pathways
Therapeutic Hypothesis	Potentially broader activity that PI3K/AKT inhibitors both single-agent and combination activity
Potential Target Indications	• *Hematologic tumors* including CLL and AML • *Solid tumors* including breast, prostate, stomach, lung, colon and pancreatic
Program Status	• Identified Development Candidates (SNS-229/-510) • Toxicology evaluation and testing ongoing

TAK-580: Oral Pan-Raf Kinase Inhibitor in Clinic

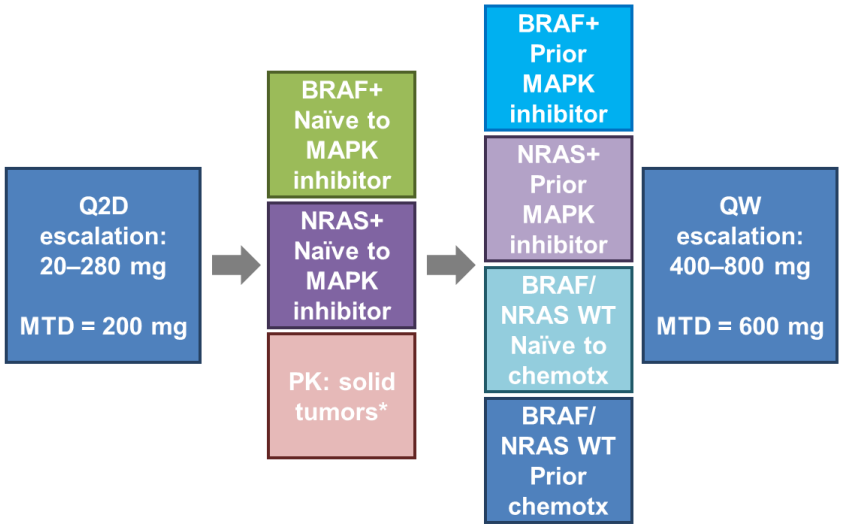
TAK-580 (MLN2480)

- Raf kinases (A-Raf, B-Raf and C-Raf) are key regulators of cell proliferation and survival
- TAK-580 is a pan-Raf kinase inhibitor which has exhibited a promising profile

Opportunity

- Up to $57.5 million in pre-commercialization milestones
- Royalty payments on net sales
- Option to co-develop/promote potentially in 2017

C28002: Multi-arm, open-label Phase 1B study in adult patients with advanced non-hematologic malignancies



Initial safety profile and efficacy of once weekly oral dosing being explored in novel combination strategies in solid tumors:

- TAK-580 + nivolumab(PD1) - melanoma
- TAK-580 + TORC1/2i (TAK-128)
- TAK-580 + alisertib (Aurora A)
- TAK-580 + paclitaxel
- TAK-580 + cetuximab
- TAK-580 + irinotecan

ESMO 2015, ClinicalTrials.gov

QINPREZO™ (Vosaroxin)

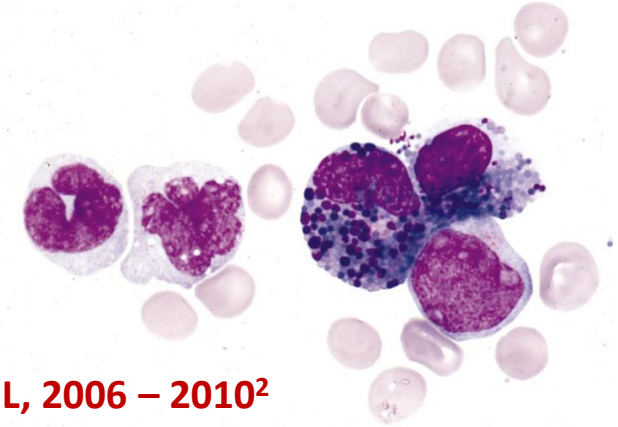
Initial Target Indication for Treatment of Relapsed/Refractory AML

- **Anti-Cancer Quinolone Derivative: First-in-Class Therapeutic**
- Intercalates DNA and inhibits topoisomerase II
- Replication-dependent, site-selective DNA damage, G2 arrest and apoptosis
- Evades common drug resistance pathways of P-gp and p53
- Low risk of drug-drug interaction
- Lower potential for off-target organ damage

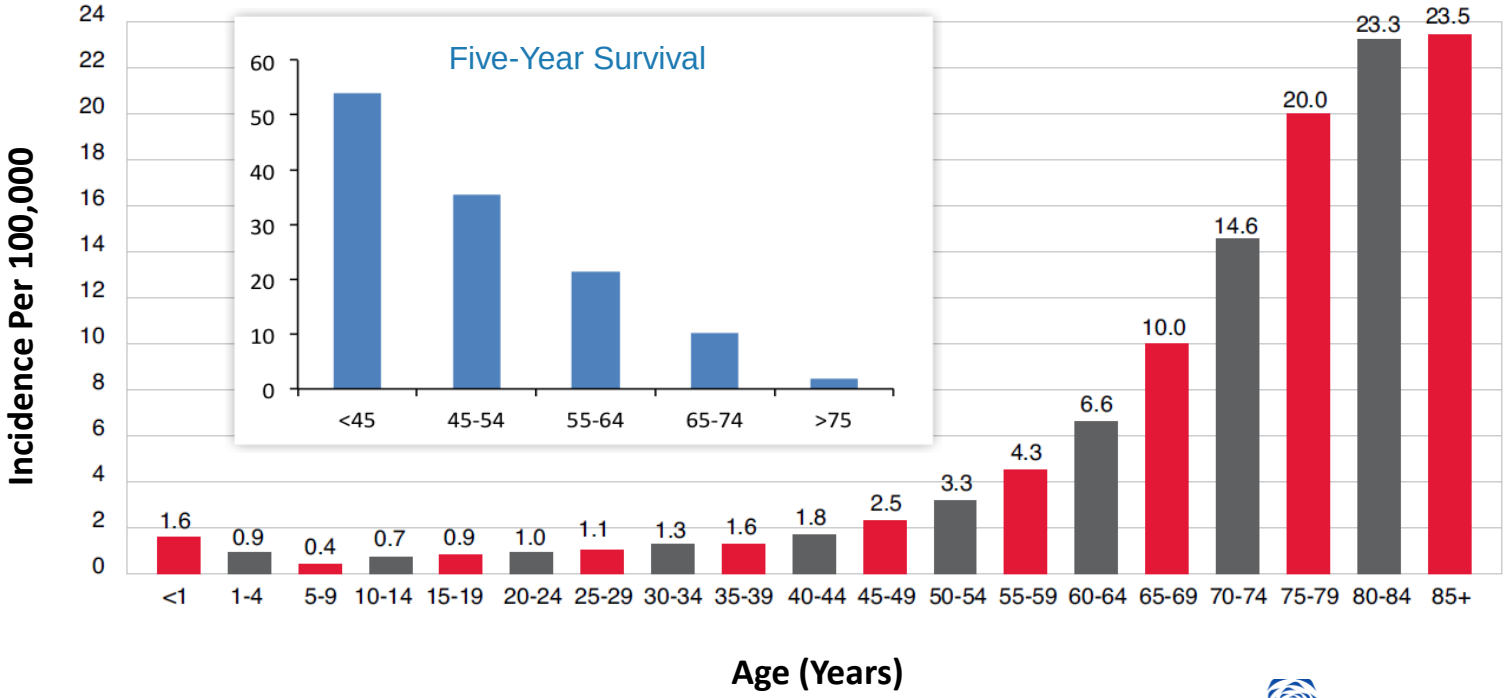
Drug Metab Disp 2009; Cancer Chemother Pharmacol 2009, 2010; Clin Cancer Res 2010; PLoS One 2010; Oncotarget 2010; Haematologica 2011

AML Overview: Unmet Need Greatest in Older Patients

- AML is a rapidly progressing cancer of the blood characterized by the uncontrolled proliferation of immature blast cells in the bone marrow.
- Incidence of 19,950 in the US in 2016[1]



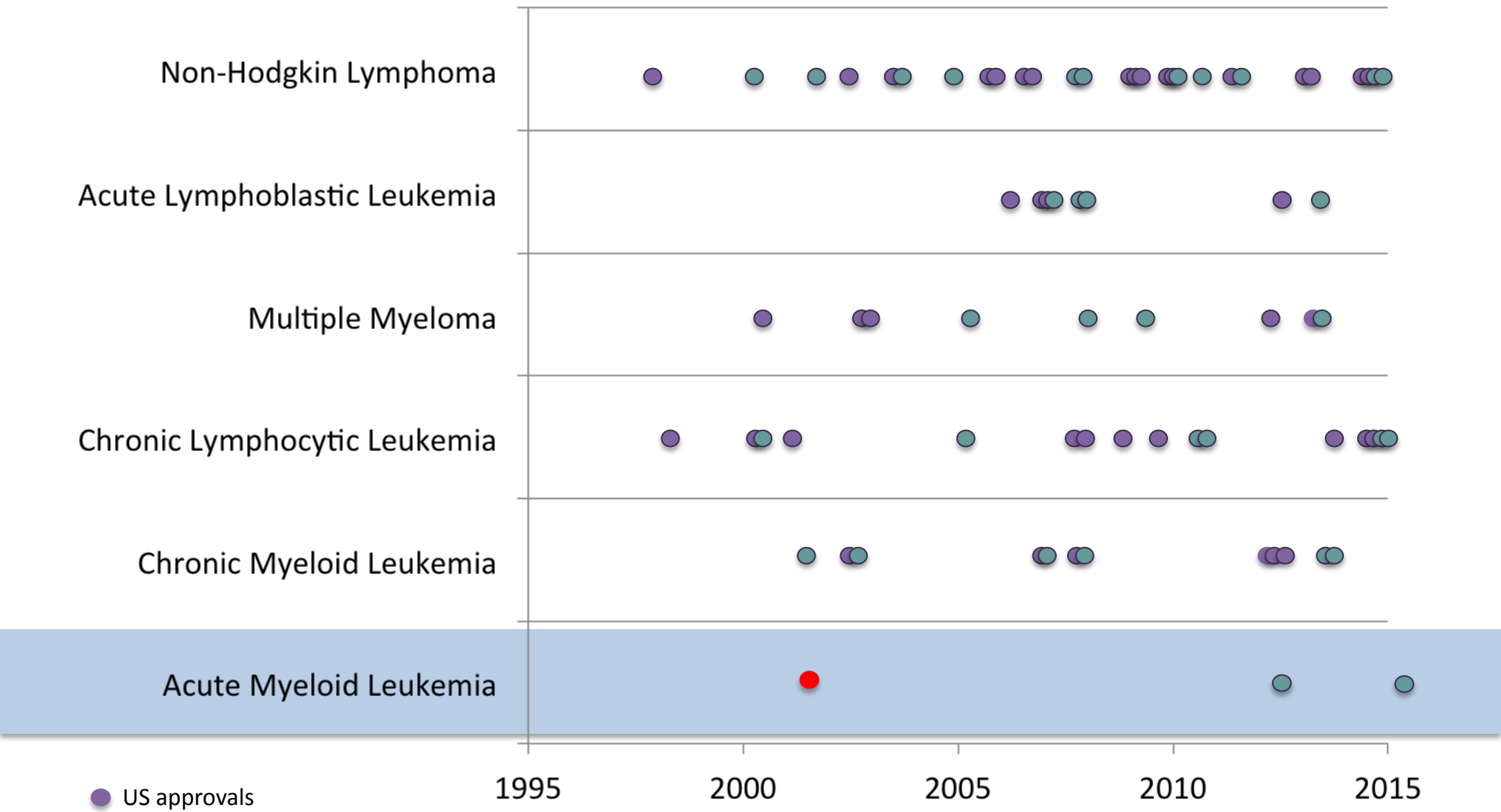
Age-Specific Incidence Rates for AML, 2006 – 2010[2]



1 http://www.cancer.org/cancer/leukemia-acutemyeloidaml/detailedguide/leukemia-acute-myeloid-myelogenous-key-statistics

2 Leukemia and Lymphoma Society. Facts 2014-15.

Approved Treatment Options



Vosaroxin in Europe – Regulatory Timeline

Proposed Indication: Treatment of Adult Patients ≥ 60 Years of Age with Relapsed/Refractory AML

European Commission

2016	2017
MAA filed	
GCP Inspections & GMP	
D120 List of Outstanding Issues	
D120 Response	
D180 List of OIs	D180 Response
	SAG-O Review
	CHMP Decision
	Named Patient Program
	Launch w/ Partner

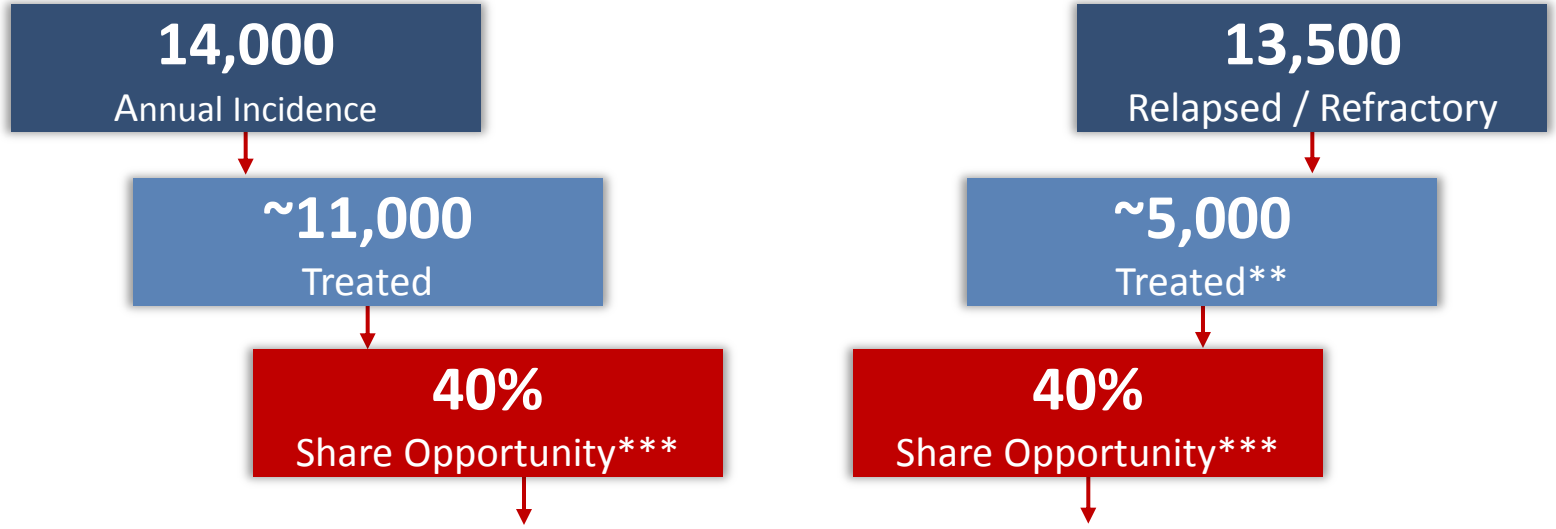
= Achieved = Anticipated

sunesis Pharmaceuticals, Inc.

Europe is a Significant Commercial Opportunity

Initial indication in Relapsed/Refractory Disease

2017 EU11 AML Treatment–Eligible Patients ≥ 60 Years[(1)]



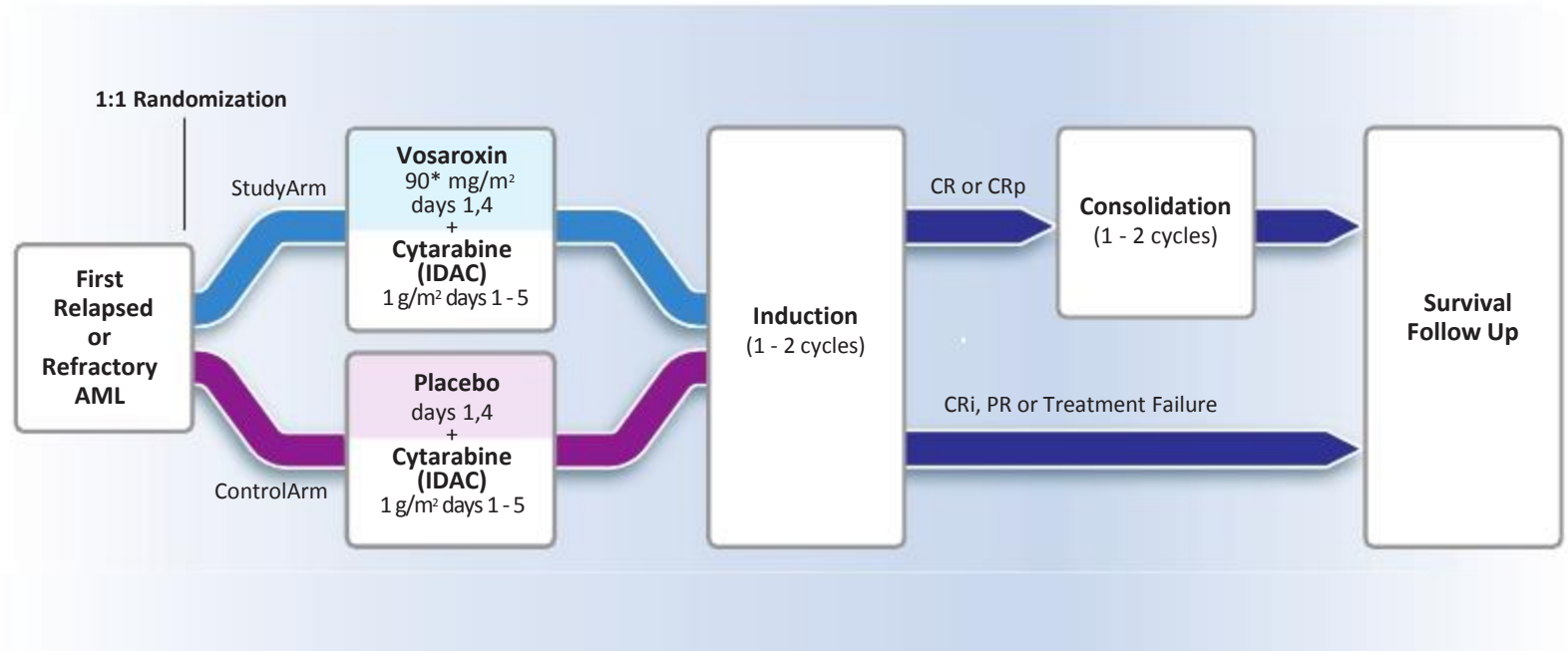
Vosaroxin EU11 AML Revenue Opportunity

Active Partnership Discussions Ongoing for European Rights

[1]Based on company projections;
** Intensive, HMAs and LoDAC;
*** New entrant opportunity with substantial clinical benefit.

VALOR Pivotal Trial Conducted in 15 Countries

VALOR



* After cycle 1, all subsequent cycles at 70 mg/m^2 vosaroxin on days 1 and 4.

VALOR Data Summary

	Median Survival (Months)		p Value
	Placebo/IDAC	Vosaroxin/IDAC	
OS Intent to treat (n=711)	6.1	7.5	0.061* 0.024**
OS 60 years and older (n=451)	5.0	7.1	0.003
CR Rate ITT	16.3%	30.1%	<0.0001
Responder Median OS Analysis			
CR on study (n=94)		21.2	<0.0001
Non-CR on study (n=191)		7.3	
30-day mortality ITT	6.6%	7.9%	
60-day mortality ITT	19.4%	19.7%	
60-day mortality ≥ 60 years	22.6%	20.4%	

The Lancet Oncology Volume 16, Issue 9, Sept. 2015
Chemotherapy Foundation, November 2015
* Unstratified log-rank; ** Stratified log-rank

VALOR Updated Survival Analysis: ASH 2016 Oral

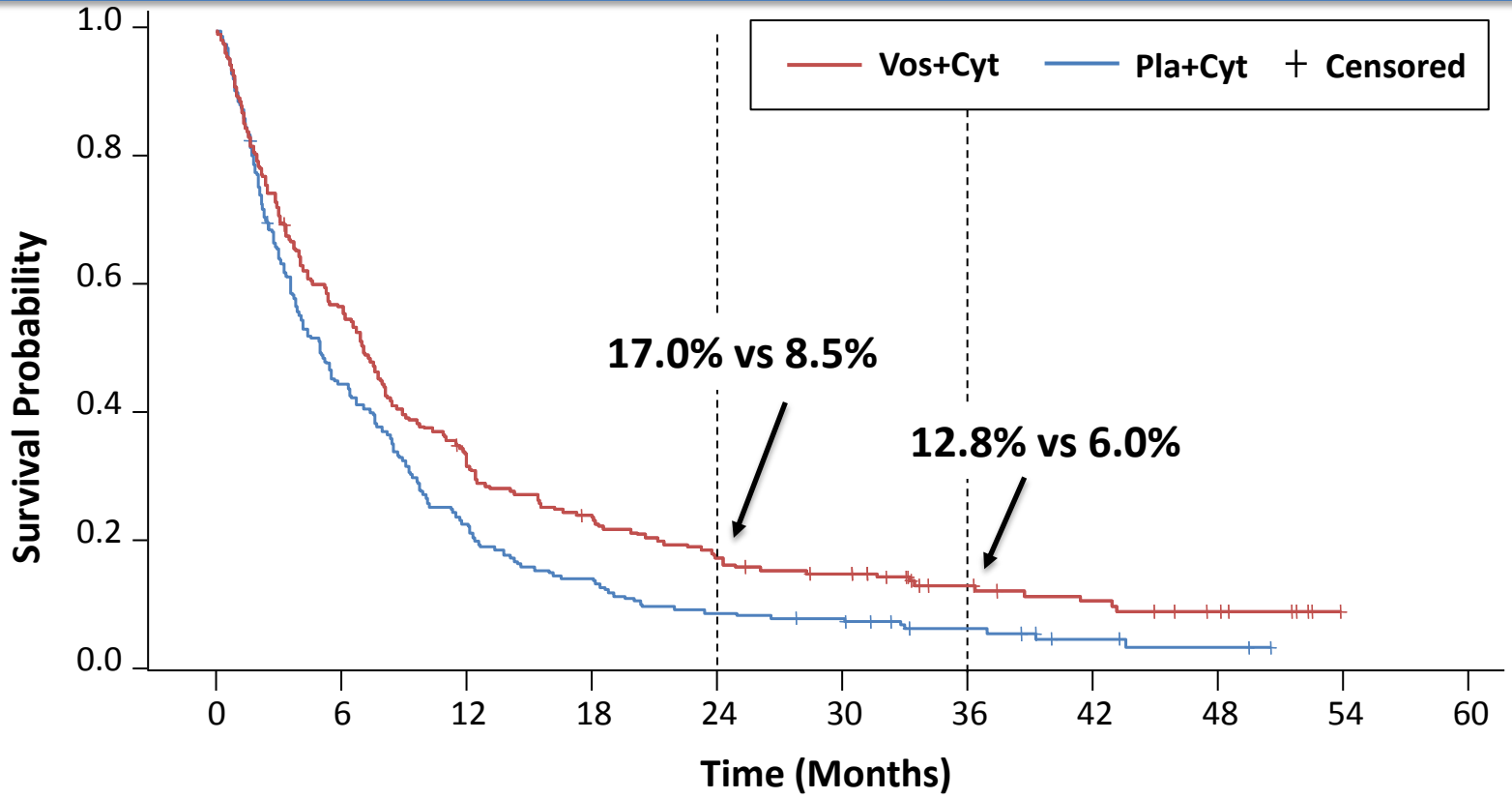
- Updated with 16 months longer follow-up
- Twice as many patients alive on the vosaroxin treatment arm

	Primary Analysis (September 2014)	Updated Survival Analysis (January 2016)
Median duration of follow-up, months[a]	24.4	39.9
Patients ≥ 60 years remaining in follow-up, n (%)		
All patients ≥ 60 years (N = 451)	63 (14)	33 (7)
Treated with vos/cyt (n = 226)	42 (19)	23 (10)
Treated with pla/cyt (n = 225)	21 (9)	10 (4)

[a] Estimated by the reverse Kaplan-Meier method.

Ravandi, ASH 2016

Durable Survival Benefit in Patients ≥ 60 Years



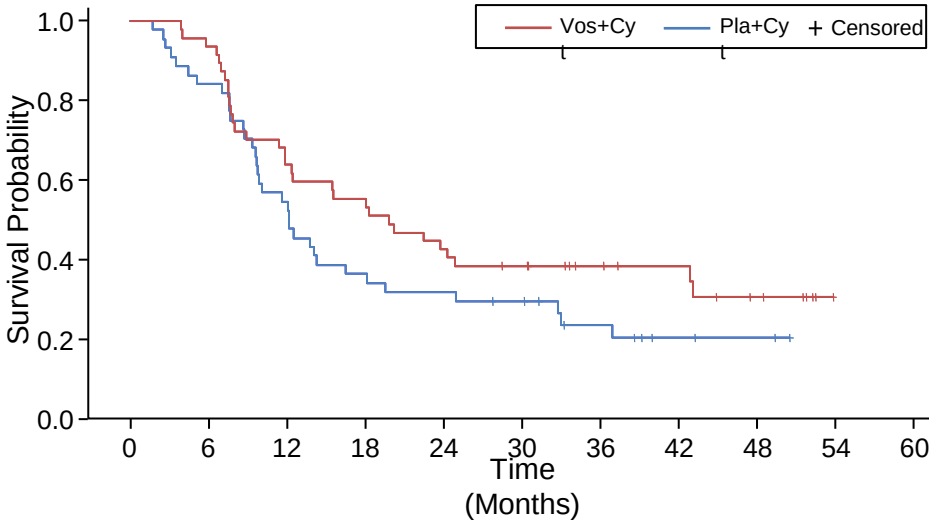
Treatment arm	Patients, n	Median OS (95% CI)	HR (95% CI)	*P* value
Vos+Cyt	226	7.1 (5.8-8.1)	0.75 (0.62-0.91)	0.002
Pla+Cyt	225	5.0 (3.8-6.4)		

Ravandi, ASH 2016

Survival ± Post-Treatment Transplantation

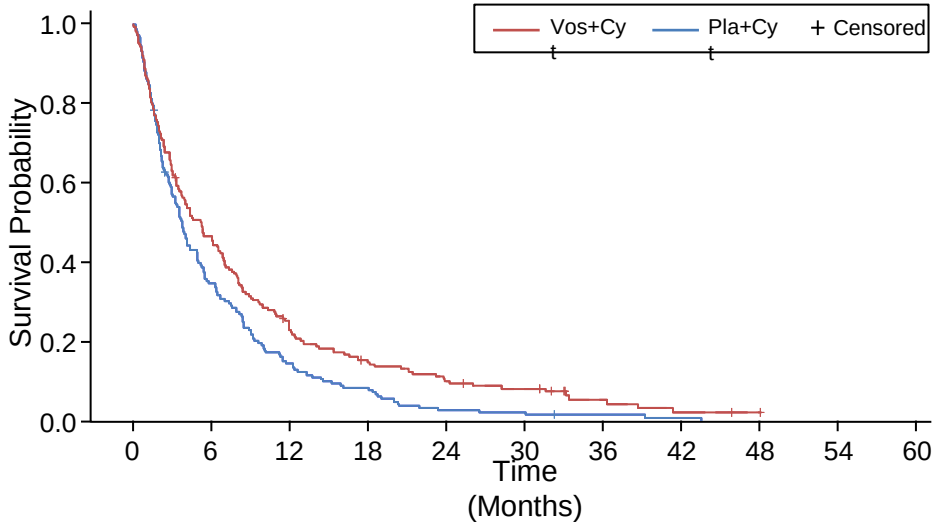
- Survival benefit demonstrated in both transplanted and non-transplanted patients

Patients ≥ 60 Years With Subsequent Transplantation (n = 91)



HR = 0.70 (95% CI: 0.43-1.13)

Patients ≥ 60 Years Without Subsequent Transplantation (n = 360)



HR = 0.75 (95% CI: 0.60-0.92)

Ravandi, ASH 2016

Treatment Effect Improved with IPCW Analysis

- Use crossover methodology to estimate treatment effect in the absence of transplant

IPCW Analysis	Patients ≥ 60 Years		
	HR for OS	95% CI	*P* value
Final model adjusted for baseline covariates	0.63	0.48-0.82	**< 0.001**

IPCW: Inverse Probability of Censoring Weights

Ravandi, ASH 2016

Consistent Survival Benefit in Age Subgroups

- OS benefit with the addition of vosaroxin was observed **in all age subsets above 60 years**

Patient Age	Median OS, months		HR (95% CI)
	Vosaroxin/ Cytarabine	Placebo/ Cytarabine	
60-64 years (n = 124)	8.1	5.2	**0.72 (0.49-1.06)**
65-74 years (n = 293)	7.0	5.0	**0.76 (0.60-0.97)**
75-84 years (n = 34)	5.5	3.3	**0.72 (0.36-1.45)**

Ravandi, ASH 2016

Grade ≥ 3 Adverse Events*

System Organ Class Preferred Term	Vosaroxin/Cytarabine (n = 226), n (%)	Placebo/Cytarabine (n = 221), n (%)
Any grade ≥ 3 AE	213 (94)	189 (86)
Blood and lymphatic system disorders		
Febrile neutropenia	96 (43)	67 (31)
Thrombocytopenia	55 (24)	56 (25)
Anemia	52 (23)	54 (24)
Neutropenia	42 (19)	31 (14)
Gastrointestinal disorders		
Stomatitis	36 (16)	9 (4)
Diarrhea	12 (5)	6 (3)
Infections and infestations		
Pneumonia	24 (11)	18 (8)
Sepsis	28 (12)	13 (6)
Bacteremia	21 (9)	9 (4)
Metabolism and nutrition disorders		
Hypokalemia	33 (15)	15 (7)
Hypophosphatemia	17 (8)	9 (4)
Decreased appetite	14 (6)	3 (1)
Vascular disorders		
Hypertension	15 (7)	10 (5)

Ravandi, ASH 2016

*Grade ≥ 3 events occurring in at least 5% of patients.

VALOR: Conclusions from ASH Presentation

- At median follow-up of 39.9 months, OS in patients ≥ 60 years of age significantly improved with vosaroxin/cytarabine
- Sensitivity analyses in patients ≥ 60 years show an OS benefit
- Significant interaction between age and treatment effect
  - OS benefit in the ITT population driven by patients ≥ 60 years
  - Consistent OS benefit among all older patients, including those ≥ 75 years of age
- **These data support vosaroxin/cytarabine as a treatment option in patients ≥ 60 years of age with R/R AML**

Ravandi, ASH 2016

Vosaroxin Life Cycle Plan Ongoing

QINPREZO™ (vosaroxin)

Multiple Opportunities in Frontline AML and Myelodyplastic Sydrome (MDS)

		Phase 1	Phase 2
Frontline AML	Vanderbilt University	**Vosaroxin + Infusional Cytarabine (VITAL) – Phase 2** Previously Untreated AML	
Frontline AML*	MD Anderson Cancer Center	**Vosaroxin + Decitabine - Phase 1/2** AML & High Risk MDS; Age 60 or Older	
MDS	Weill Cornell Medical College	**Vosaroxin – Phase 1/2** Intermediate 2/High-Risk MDS After Failure with Hypomethylating Agents	
	Washington University in St. Louis School of Medicine	**Vosaroxin + Azacitidine - Phase 1** Intermediate & High-Risk MDS	

*Includes 7/63 patients with MDS having 10-20% blast counts (borderline AML)

Impressive Synergistic Activity in MDACC Frontline Study

Oral Presentation/Update

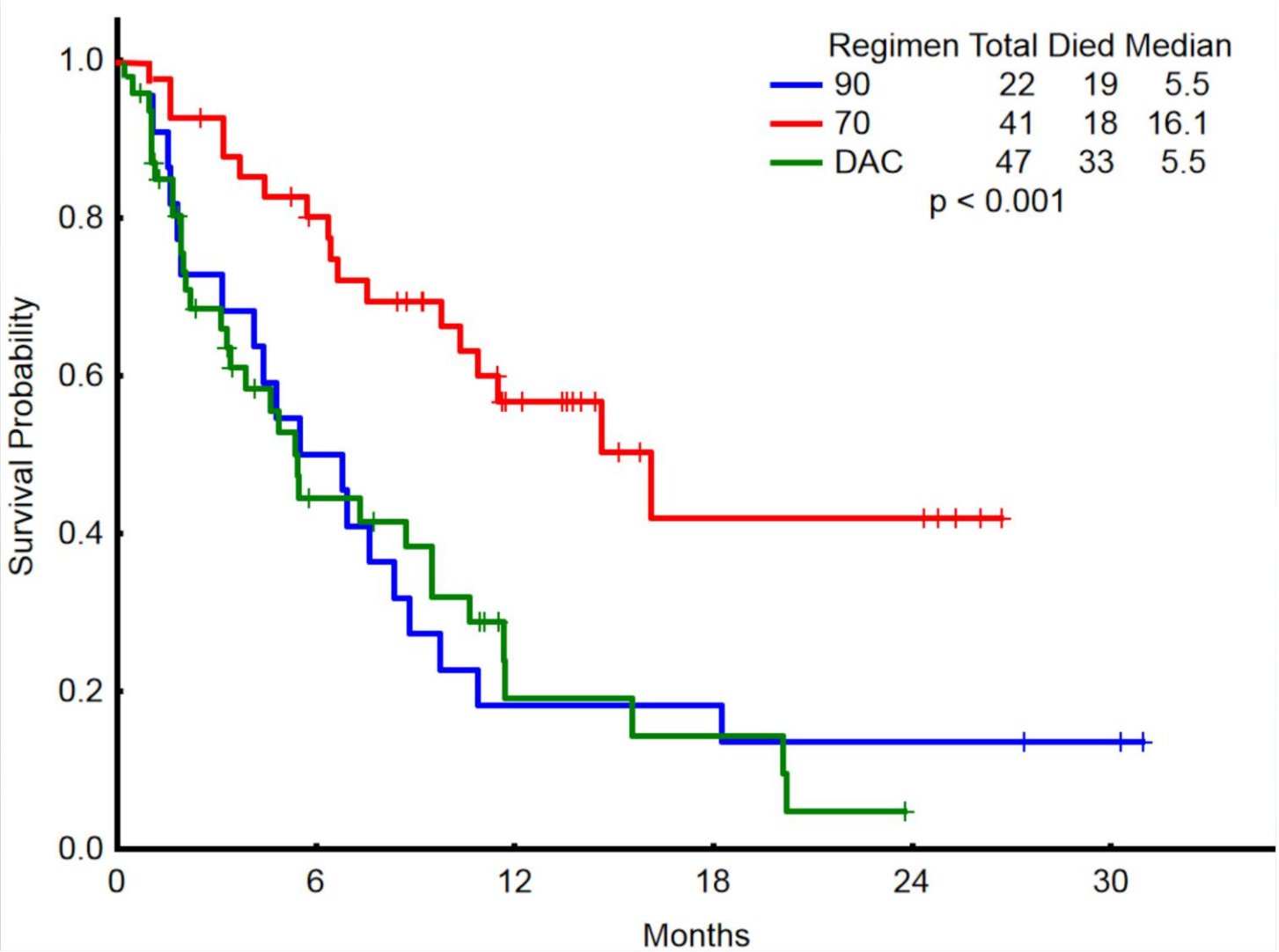
European Hematology Association on June 11, 2016

MD Anderson Cancer Center

Phase I/II Study of Vosaroxin and Decitabine in Frontline AML and Myelodyplastic Sydrome

Response / Outcome	N (%)
Evaluable (median age = 69 years)	63
CRc (CR + CRp + CRi)	47 (75%)
CR	31 (49%)
CRc Complex Karyotype & TP53 Mutated	68%/77%
Early Death ≤ 4 weeks	0 (0)
Early Death ≤ 8 weeks	8 (13%)
MRD (-) by Multi-Parameter Flow	22/34 (65%)

Promising Survival from Optimized 70 mg/m^2 Cohort



Significant Layers of Exclusivity for Vosaroxin Franchise

- Data exclusivity 10+ years in Europe, 7 years in the U.S. and 10 years in Japan
- E.U. issued patent protection currently to 2027+
- U.S. issued patent protection to 2030+
- Additional patents pending ex-US to 2030

+ Potential for further extension through patent-term restoration

Financial Position and Capitalization

Ticker	SNSS (NASDAQ)
Cash & Equivalents	$50.4 million[1]
Debt O / S	$15.0 million[1]
Shares O / S	25.1 million[2]
Warrants O/S Stock Options O/S	269,000 @ avg $4.51 2,641,000 @ avg $13.50
Top Shareholders	BVF Partners, Great Point, NEA, Aisling, Bay City, Palo Alto Investors, Dafna, Cormorant, Baker Brothers, First Eagle, Tavistock, Vivo, Driehaus
Covering Analysts	Mara Goldstein (Cantor Fitzgerald) Eric Schmidt (Cowen & Co) Maxim Jacobs (Edison Research) Jim Birchenough (Wells Fargo Securities)

[1] As of September 30, 2016. Cash is pro-forma, including proceeds from October financings.

[2] Based on treasury method as of October 24, 2016; does not include out-of-the-money warrants and options; includes preferred as converted.

Potential Pipeline Milestones

PROGRAM	UPDATE	TIMEFRAME
SNS-062	**Activate IND**	✓
	Initiate Phase 1B/2 B-Cell Malignancies trial with prior ibrutinib, including CLL relapsed patients with C481S mutations	1H 2017
Vosaroxin QINPREZO™ (vosaroxin)	**Submit Response to CHMP Day 180 List of Outstanding Issues**	1Q 2017
	Participation in Scientific Advisory Group, Oncology (SAG-O) CHMP	April 2017
	CHMP Opinion	Mid-Year
	Chargeable Named Patient Access Program	2H 2017
	Qinprezo Launch with Partner in Europe	2H 2017
TAK-580 & PDK	**TAK-580: Present combination data from ongoing study**	2017
	SNS-229/510: Complete toxicology/pre clinical assessment	2H 2017