Determined to realize a future in which people with cancer live longer and better than ever before

Syndax

1Q20 EARNINGS PRESENTATION

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Syndax pipeline addresses key areas of unmet need in cancer

Entinostat
Class I
HDAC Inhibitor
Breast Cancer

- Targeting endocrine therapy resistance in HR+ met breast cancer
- Ph 3 data expected 2Q20
- Potential approval 2Q21

SNDX-5613
Menin Inhibitor
Leukemias

- Targeting acute leukemias
- Ph 1 data provides initial clinical evidence of efficacy
- Ph 1 data expected 4Q20

Axatilimab
anti-CSF1R mAb
Chronic GVHD

- Targeting macrophage driven diseases
- Clinical POC for chronic GVHD achieved
- Ph 1/2 data expected 4Q20

Phase 3 E2112: Focused on overall survival

E2112: Exemestane +/- entinostat

Primary endpoint: OS

E2112 Trial Assumptions

80% power to detect HR = 0.75

Minimal HR detectable = 0.82

Equates to a clinically meaningful risk reduction and ~5 mo mOS benefit

2Q20: Final OS analysis anticipated

A positive OS result allows filing for full regulatory approval

Blockbuster potential as 2nd/3rd line agent

Leading treatment options - HR+, HER2- advanced breast cancer

1st line hormone Tx	2nd/3rd/4th line hormone Tx	Chemo-Tx
Anastrozole or letrozole +/- CDK 4/6 inhibitor	Anastrozole, Faslodex +/- CDK 4/6 inhibitor or Afinitor-exemestane	Capecitabine, gemcitabine, eribulin
	34,000 pts Entinostat-exemestane target population	

U.S. commercial launch preparation underway

Source: Kantar 2019 Breast cancer Epidata; IQUVIA Monthly treatment report (2018)

Recent market research with U.S. and EU physicians demonstrated favorable reaction to entinostat TPP

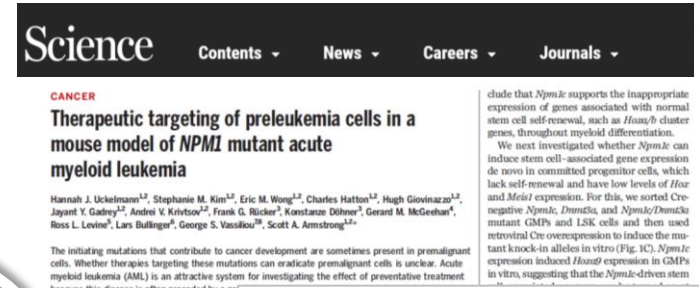
Attribute		Key Feedback
Indication		• High unmet need in 2L, especially after CDK 4/6 treatment
Mechanism of Action		• Re-sensitization to endocrine therapy seen as positive, providing rationale for other entinostat -- ET combinations
Efficacy		• OS benefit viewed as most important efficacy measure; • Positive QoL benefit could offset smaller PFS benefit
Route of Administration		• Oral administration reduces cost and burden of hospital visits
Safety & Tolerability		• AE profile viewed as favorable vs SOC and alpelisib

Source: 2019 HR+/HER2- breast cancer Physician market research

SNDX-5613: Breakthrough targeted therapy for acute leukemia

Advantages

- Strong target validation
- Precise patient selection
- Big effect in small studies
- Molecular markers of disease status
- Potential for rapid regulatory path



Science Contents News Careers Journals

CANCER

Therapeutic targeting of preleukemia cells in a mouse model of *NPM1* mutant acute myeloid leukemia

Hannah J. Uckelmann[1,2], Stephanie M. Kim[1,2], Eric M. Wong[1,2], Charles Hatton[1,2], Hugh Giovinazzo[1,2], Jayant Y. Gadrey[1,2], Andrei V. Krivtsov[1,2], Frank G. Rücker[3], Konstanze Döhner[3], Gerard M. McGeehan[4], Ross L. Levine[5], Lars Bullinger[6], George S. Vassiliou[7,8], Scott A. Armstrong[1,2]*

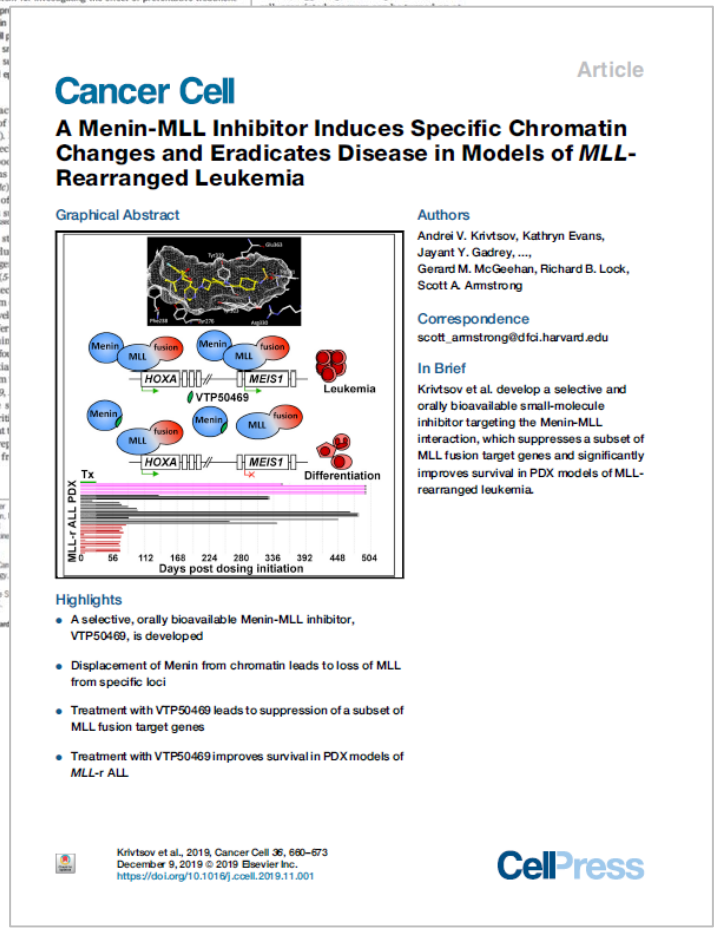
The initiating mutations that contribute to cancer development are sometimes present in premalignant cells. Whether therapies targeting these mutations can eradicate premalignant cells is unclear. Acute myeloid leukemia (AML) is an attractive system for investigating the effect of preventative treatment because this disease is often preceded by a pr… syndrome). In *Npm1c/Dnmt3a* mutant knock-in … by a period of extended myeloid progenitor cell … be reversed by oral administration of a s… matin complex. These preclinical results s… oping AML might benefit from targeted e…

…leophosmin (*NPM1*) mutant ac… …loid leukemia (AML) is one of … common types of AML (*1*–*3*). … its high prevalence, the mec… … of leukemogenesis is still poo… …, and targeted therapy options … *NPM1* gene mutations (*NPM1c*) …asmic localization of NPM1 and of… …ith other mutations in genes s… *ethyltransferase 3A* (*DNMT3A*[Ross]) … …ukemias express a distinctive s… …gene expression pattern that inclu… …box cluster A and B (*HOXA/B*) ge… …eir DNA-binding cofactor MEIS1 (*5*… …mans, *DNMT3A* mutations are detec… …he most primitive hematopoietic stem … …compartment, often long before the devel… …ment of leukemia, a condition often refer… …to as clonal hematopoiesis of indetermin… potential (CHIP) (*9*). *NPM1* mutations are fo… …in committed progenitors and differentia… …myeloid cells in AML but are absent from … …tem cell and lymphoid compartments (*9*, … …is suggests that *NPM1c* may induce s… …wal in myeloid progenitors as a crit… …n the development of AML and that t… …t progenitor self-renewal may reg… …itical step in the progression fr… …AML.

…Pediatric Oncology, Dana-Farber Cancer … …vision of Hematology/Oncology, Boston, … …Children's Hospital and Harvard Medical … …MA, USA. [7]Department of Internal Medicine … …ital of Ulm, Ulm, Germany. [8]Syndax … …ls, Inc., Waltham, MA, USA. [9]Center for … …leukemias, Memorial Sloan Kettering Can… …ork, NY, USA. [10]Department of Hematology … …d Tumor Immunology, Charité University … …erlin, Germany. [11]Wellcome-MRC Cambridge S… …e, University of Cambridge, Cambridge, UK. … …e Trust Sanger Institute, Cambridge, UK. …onding author. Email: scott_armstrong@dfci.harvard…

clude that *Npm1c* supports the inappropriate expression of genes associated with normal stem cell self-renewal, such as *Hoxa/b* cluster genes, throughout myeloid differentiation.

We next investigated whether *Npm1c* can induce stem cell–associated gene expression de novo in committed progenitor cells, which lack self-renewal and have low levels of *Hoxa* and *Meis1* expression. For this, we sorted Cre-negative *Npm1c*, *Dnmt3a*, and *Npm1c/Dnmt3a* mutant GMPs and LSK cells and then used retroviral Cre overexpression to induce the mutant knock-in alleles in vitro (Fig. 1C). *Npm1c* expression induced *Hoxa9* expression in GMPs in vitro, suggesting that the *Npm1c*-driven stem



Article

Cancer Cell

A Menin-MLL Inhibitor Induces Specific Chromatin Changes and Eradicates Disease in Models of *MLL*-Rearranged Leukemia

Graphical Abstract

Authors

Andrei V. Krivtsov, Kathryn Evans, Jayant Y. Gadrey, ..., Gerard M. McGeehan, Richard B. Lock, Scott A. Armstrong

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In Brief

Krivtsov et al. develop a selective and orally bioavailable small-molecule inhibitor targeting the Menin-MLL interaction, which suppresses a subset of MLL fusion target genes and significantly improves survival in PDX models of MLL-rearranged leukemia.

Highlights

- A selective, orally bioavailable Menin-MLL inhibitor, VTP50469, is developed
- Displacement of Menin from chromatin leads to loss of MLL from specific loci
- Treatment with VTP50469 leads to suppression of a subset of MLL fusion target genes
- Treatment with VTP50469 improves survival in PDX models of *MLL*-r ALL

Krivtsov et al., 2019, Cancer Cell 36, 660–673
December 9, 2019 © 2019 Elsevier Inc.
https://doi.org/10.1016/j.ccell.2019.11.001

CellPress

Projecting pre-clinical PK/PD to target clinical exposure

Target PK Profile Requirements

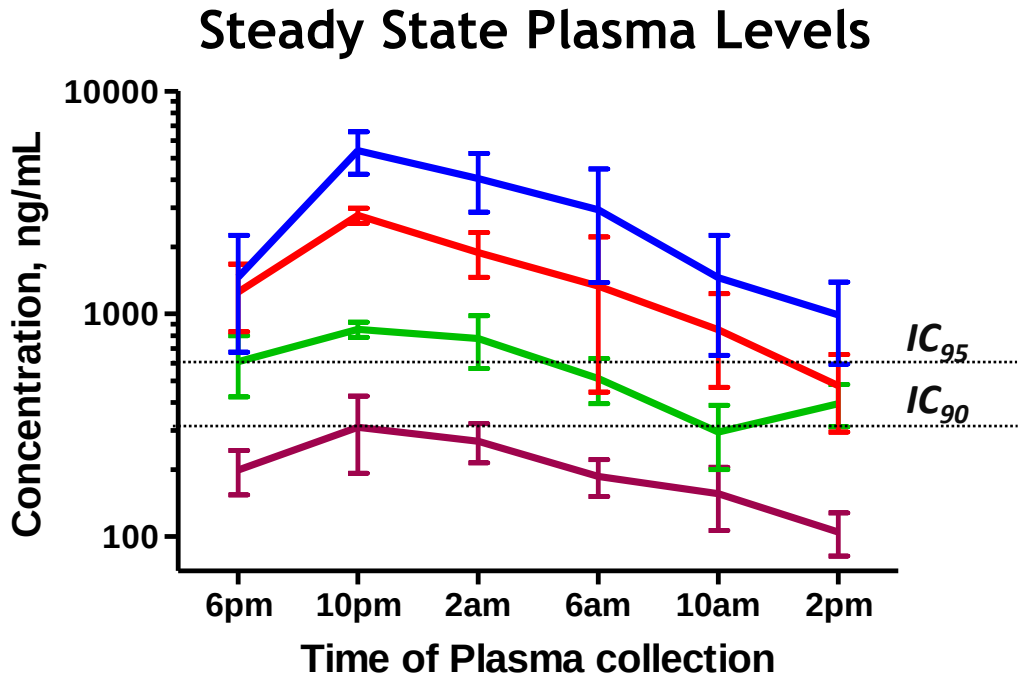
Maintain steady state levels above IC_{95} (~600 ng/mL) for most of dosing interval

AND

Maintain C_{min} level above projected IC_{90} (~300 ng/mL)

AND

Minimum 24 h AUC of ~30,000 ng*h/mL

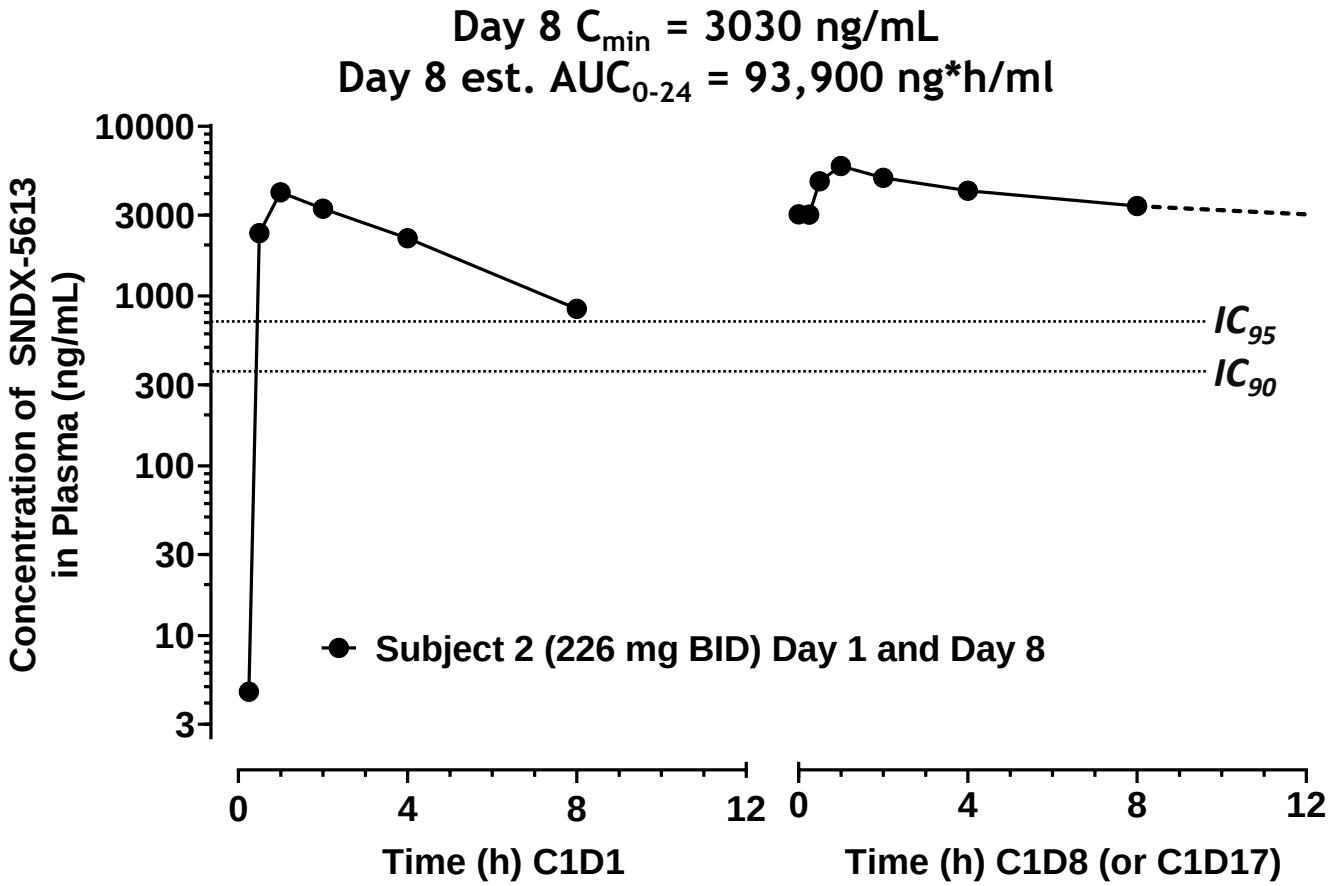


DOSE STRENGTH %	AVE CONC ng/ml	AUC_{0-24} ng*hr/ml
0.025	203	4900
0.05	573	13700
0.10	1425	34200
0.20	2713	65100

Patient #2: 226 mg PO q12h

Patient Characteristics

Gender, Age	Female, 69 yr old
Diagnosis	Refractory MPAL
Mutational status	MLL-TET1 fusion FLT3 ITD
Prior lines of therapy	2 (chemo, gilteritinib)
SNDX-5613 dose	226 mg PO q12 h
DLT period	No DLTs; Grade 2 QTc → resolved with dose reduced to 113 mg q12h
Day 28 response	CRi; beyond DLT period has improved to CR while on reduced dose



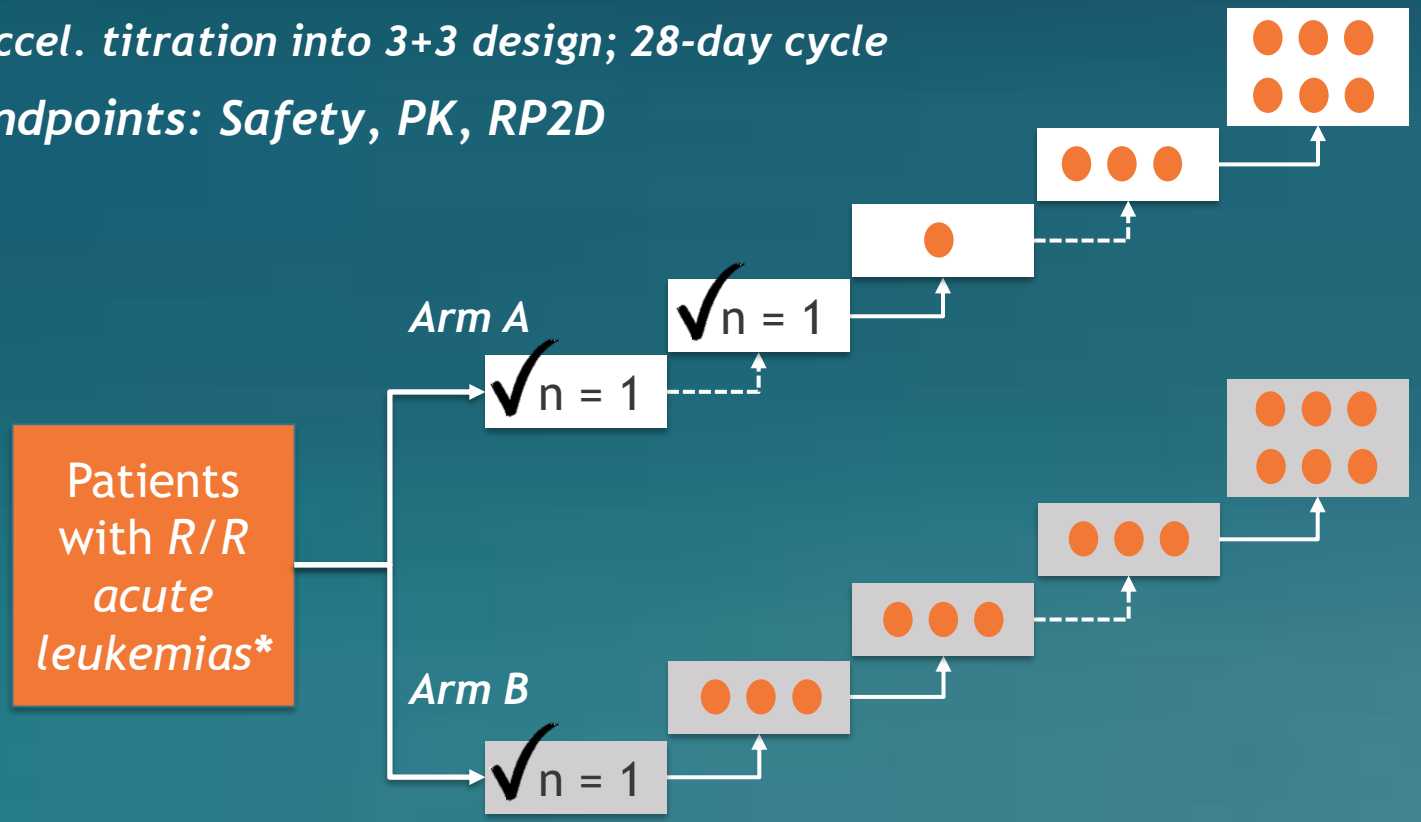
CR = Complete response, CRh = Complete response with partial hematologic recovery,
CRi = Complete response with incomplete hematologic recovery

AUGMENT-101: Phase 1/2 trial of SNDX-5613, in patients with acute leukemia

Phase 1: *Dose escalation*

Accel. titration into 3+3 design; 28-day cycle

Endpoints: Safety, PK, RP2D



Arm A = patients not on a strong CYP3A4 inh. Arm B = patients on a strong CYP3A4 inh.

Phase 2: *Expansion*

- Adult MLL-r ALL
- Adult MLL-r AML
- Adult NPM1 mut AML

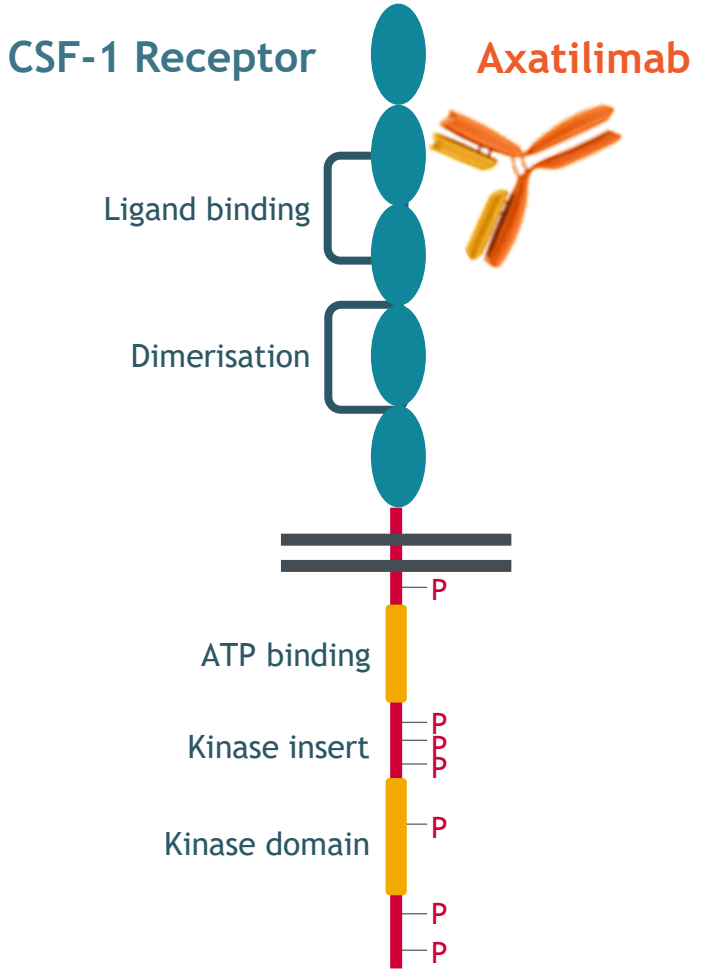
Primary endpoint:
CR Rate (CR + CRh^)

* Unselected population; ^ CR = Complete response, CRh = Complete response with partial hematologic recovery; MLL-r – mixed lineage leukemia rearranged; NPM = nucleophosmin

Axatilimab: CSF-1R mAB with potential best-in-class profile

- Axatilimab Phase 1 MAD data presented at AACR Virtual Meeting I
  - Showed rapid and sustained depletion of circulating pro-inflammatory monocytes observed at all doses
  - Identified RP2D as Monotherapy agent in patients with solid tumors
  - Identified RP2D in combo with IMFINZI® (durvalumab, AZ)
  - Demonstrates tolerability and robust PD biomarker modulation

CSF-1R – colony stimulating factor -1 receptor; RP2D recommended Phase 2 dose.
Source : Ordentlich, P. et al SITC 2016.



High affinity, IgG4 (K_D = 4-8 pM)

Axatilimab: significant potential in cGVHD

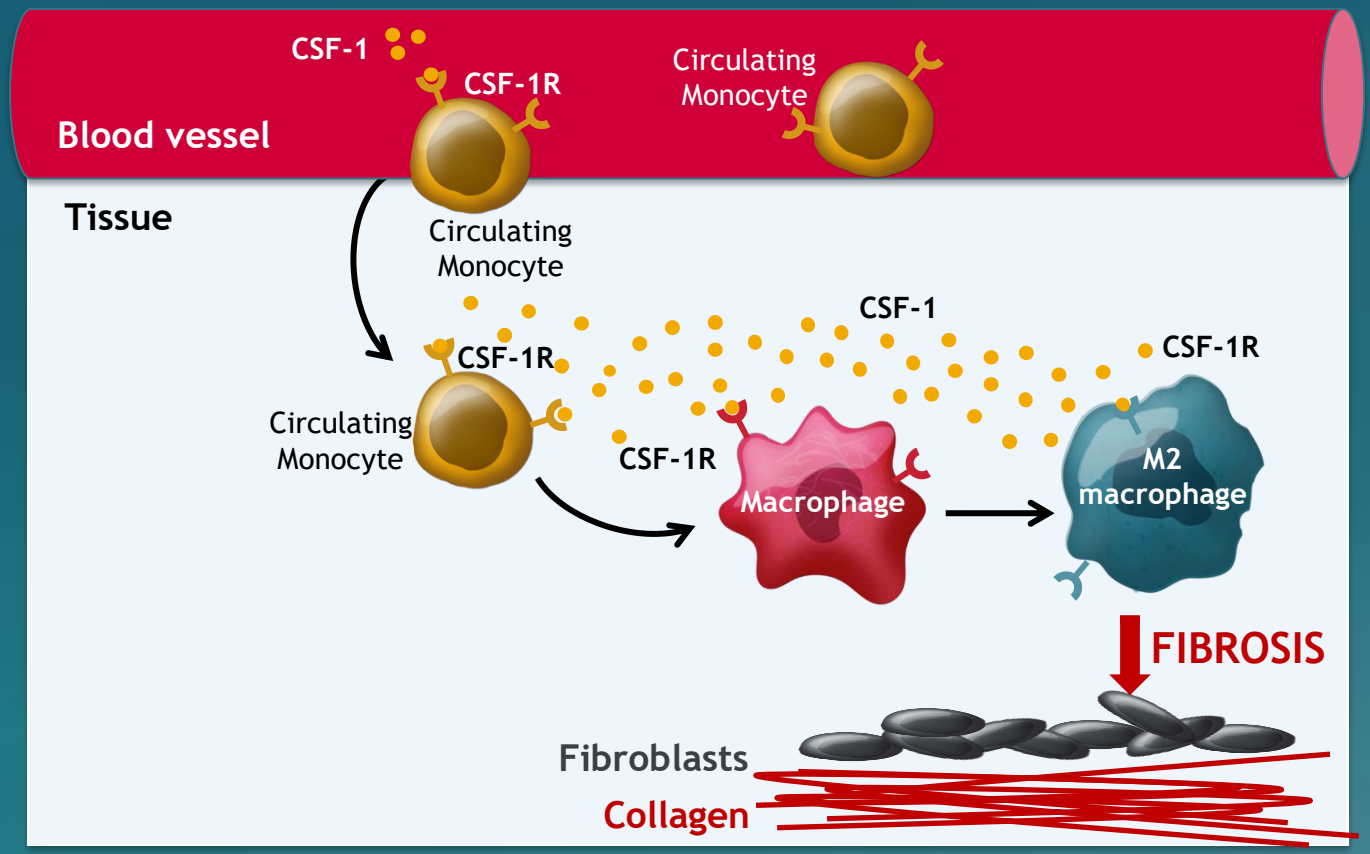
cGVHD develops in 40% of HSCT[1,2]

- **US prevalence ~14,000[2]**

Pre-clinical evidence suggest CSF-1-dependant macrophages mediate cGVHD[3]

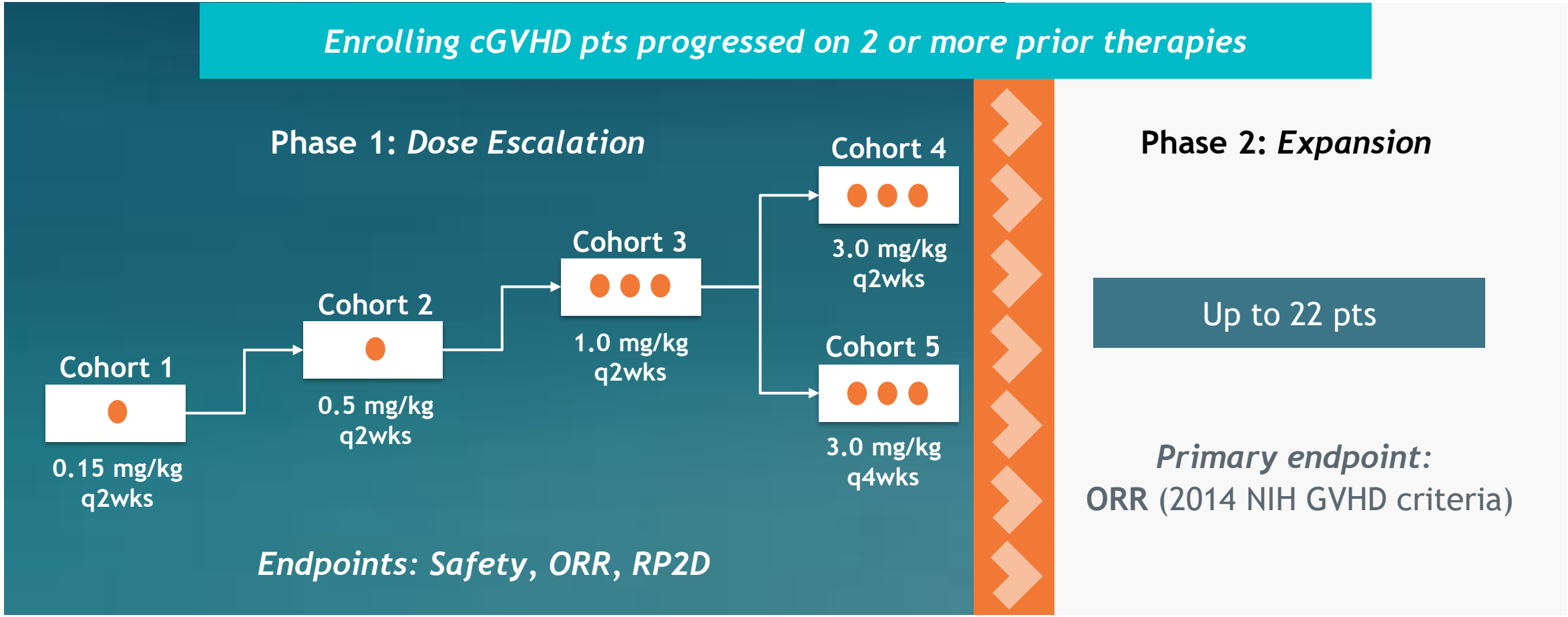
Phase 1/2 trial enrolling; data 2H20

Signaling through CSF-1R may play a meaningful role in the management of chronic GVHD



Source: 1. SmartAnalyst 2017 SmartImmunology Insights chronic GVHD report.; 2. Bachier, CR. et al. ASH annual meeting 2019; abstract #2109 Epidemiology and Real-World Treatment of cGVHD Post Allogeneic HSCT: A U.S. Claims Analysis.; 3.Alexander, KA et al., J Clin Invest. 2014;124(10):4266-4280. Figure Adopted from MacDonald, K.P.A. et al., BLOOD, 5 (129) 13-21; HSCT - Hematopoietic stem cell transplantation

Axatilimab: Phase 1 established early proof of concept in cGVHD, Phase 2 designed to identify optimal dose



Proven ability to build the pipeline

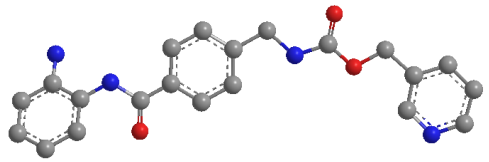
Established relationships enhance identification and access to quality assets

Clinical development leadership enables competitive advantage

Business development continues to be a core strength of our business

From Bayer

Entinostat

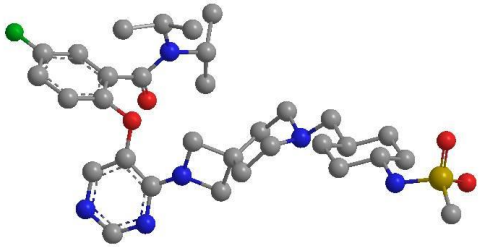


From UCB

Axatilimab

From Allergan/Vitae

Menin-MLL inhibitors



Financial highlights and 1H 2020 financial guidance

1Q 2020: Raised $35 million at a 20% premium to market, closed on $20 million debt
2Q 2020: Equity offering at $18.00, net proceeds of $93.7

Ticker	SNDX (NASDAQ)
Cash and short-term investments (at Mar 31, 2020)	$99.0 million
Cash proceeds from Q2 2020 offering	$93.7 million
Shares Outstanding* (at May 6, 2020)	41.7 million
1H 2020 Operating Expense Guidance**	
	2Q 2020
Research and Development	$12-14 M
Total Operating Expenses^	$18-20 M

** Includes 36.1 million common shares and pre-funded warrants to purchase 5.6 million common shares; ^ Includes $1.5 million non-cash stock compensation expense for 2Q 2020; ** Financial guidance for 2H 2020 will be issued following results of the E2112 trial*

Key upcoming milestones

Entinostat (Class 1 specific HDAC inhibitor)	1Q20	2Q20	3Q20	4Q20
E2112 - Final Overall Survival analysis expected		●		

SNDX-5613 (Menin inhibitor)	1Q20	2Q20	3Q20	4Q20
Data presentation from AUGMENT-101 trial (in R/R acute leukemias)				●

Axatilimab (anti-CSF-1R mAB)	1Q20	2Q20	3Q20	4Q20
Data presentation from Phase 1/2 chronic GVHD trial				●

Thank you. Questions?

Syndax