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

Syndax

4Q19 EARNINGS PRESENTATION | MARCH 2020

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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
- Ph3 data expected 2Q20
- Potential approval 2Q21

SNDX-5613
Menin inhibitor
Leukemias

- Targeting Acute Leukemias
- Recent publications in Cancer Cell and Science endorse rationale for treating MLLr, NPM1 mut
- Ph1 data expected 4Q20

Axatilimab*
anti-CSF1R mAb
Chronic GVHD

- Targeting macrophage driven diseases
- Clinical proof-of-concept for chronic GVHD achieved
- Ph1 data expected 4Q20

** recently granted generic name for SNDX-6352*

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 letrazole +/- CDK4,6 inhibitor	Anastrazole, Faslodex +/- CDK4,6 inhibitor or Afinitor-exemestane	Capecitabine, gemcitabine, eribulin
	34,000 pts Entinostat-exemestane target population	

US commercial launch preparation underway

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

Recent market research with US and EU physicians demonstrated favorable reaction to entinostat TPP

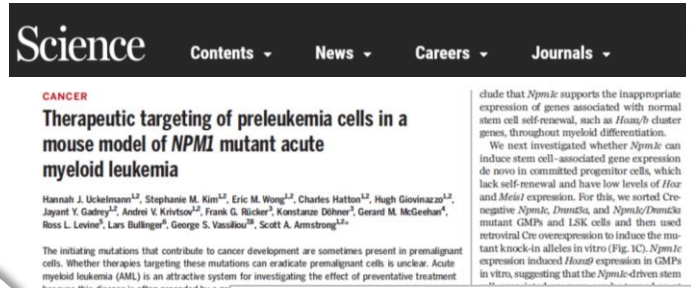
Attribute		Key Feedback
Indication		• High unmet need in 2L, especially after CDK 4/6 treatment
Mechanism of Action		• Re-sensitization to endocrine therapy seen as positive, providing rationale for other entinostat -- ET combinations
Efficacy		• OS benefit viewed as most important efficacy measure; • 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
- 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,*]

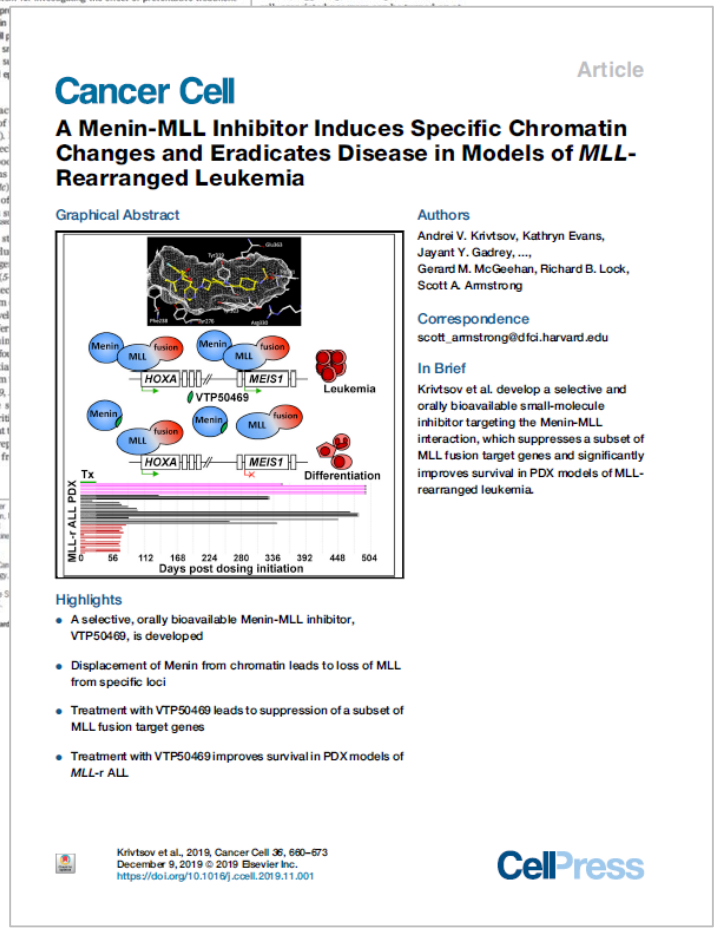
The initiating mutations that contribute to cancer development are sometimes present in premalignant cells. Whether therapies targeting these mutations can eradicate premalignant cells is unclear. Acute myeloid leukemia (AML) is an attractive system for investigating the effect of preventative treatment 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 … other mutations in genes s… *…ethyltransferase 3A* (*DNMT3A*[Rose… …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 … stem cell and lymphoid compartments (*9*, … suggests that *NPM1c* may induce s… …wal in myeloid progenitors as a crit… …the development of AML and that t… …t progenitor self-renewal may re… …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 … …s, Inc., Waltham, MA, USA. [9]Center for … …leukemias, Memorial Sloan Kettering Can… …ork, NY, USA. [10]Department of Hematology, … …Tumor Immunology, Charité University … …erlin, Germany. [11]Wellcome-MRC Cambridge S… …e, University of Cambridge, Cambridge, UK. … 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

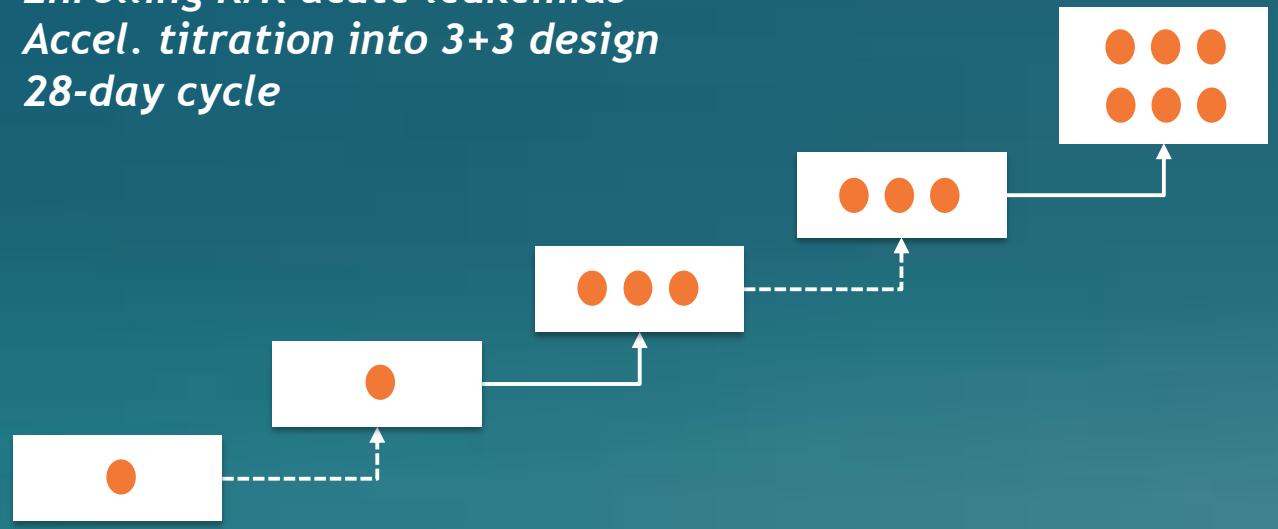
Krivtsov et al., 2019, Cancer Cell 36, 660–673
December 9, 2019 © 2019 Elsevier Inc.
https://doi.org/10.1016/j.ccell.2019.11.001

CellPress

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

Phase 1: *Dose escalation*

*Enrolling R/R acute leukemias**
Accel. titration into 3+3 design
28-day cycle



Endpoints: Safety, PK, RP2D

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 monoclonal antibody

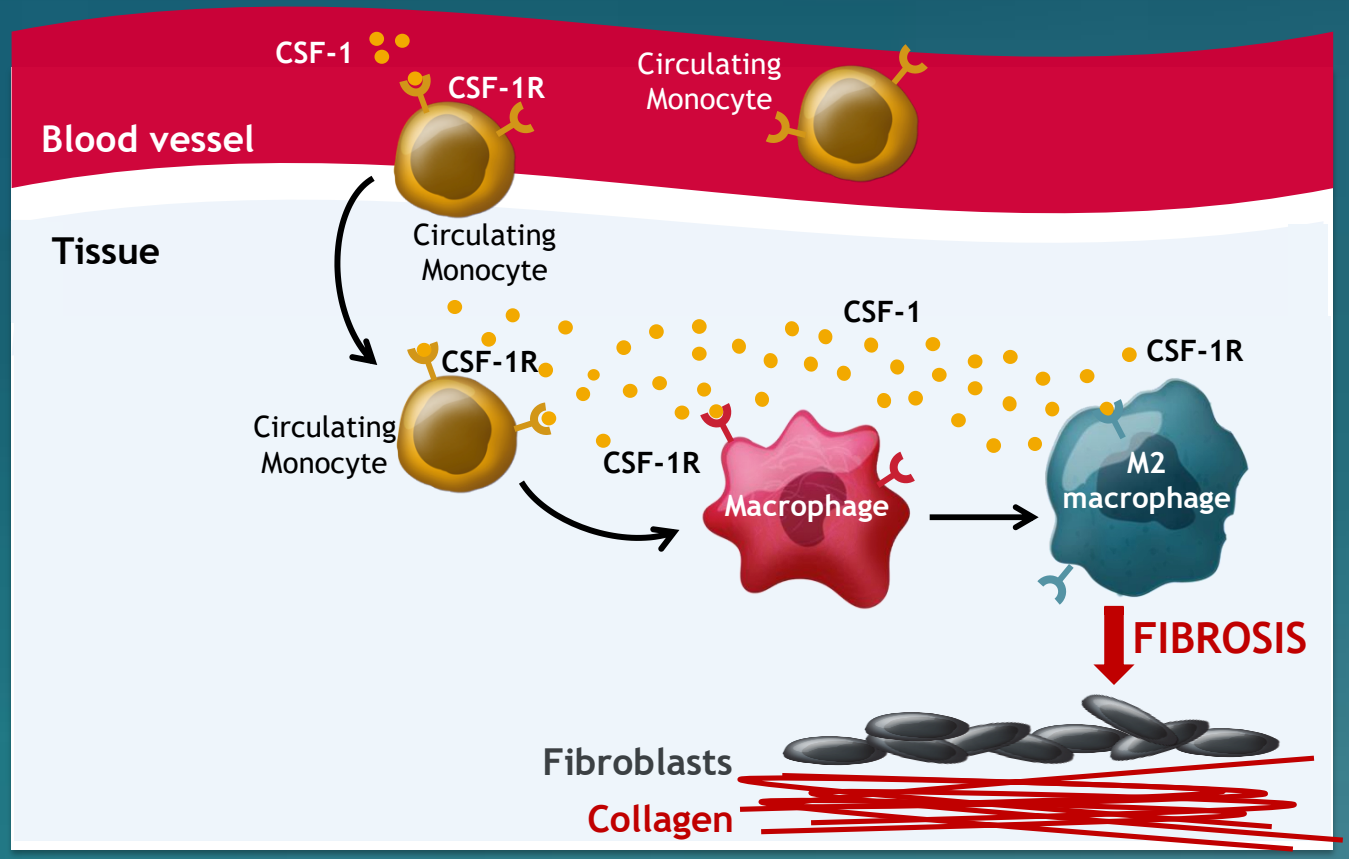
cGVHD develops in 40% of HSCT[1,2]

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

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

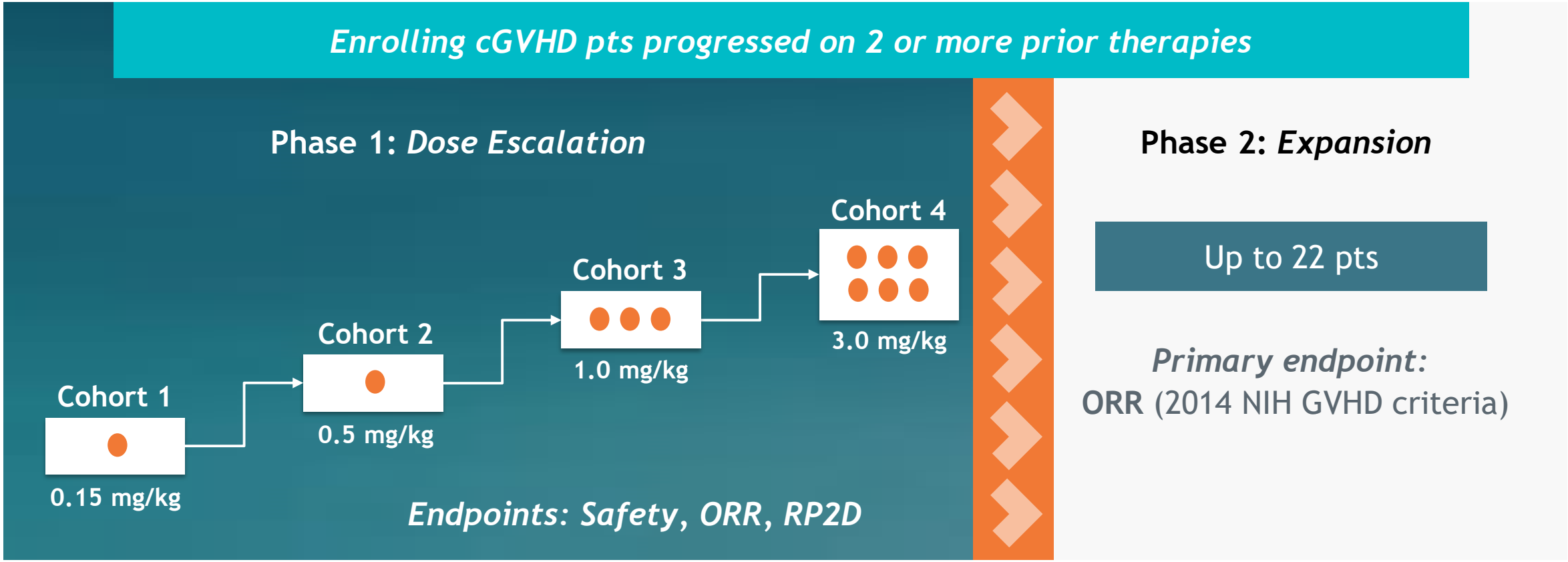
Phase 1/2 trial enrolling; data 2H20

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 designed to identify optimal Phase 2 dose



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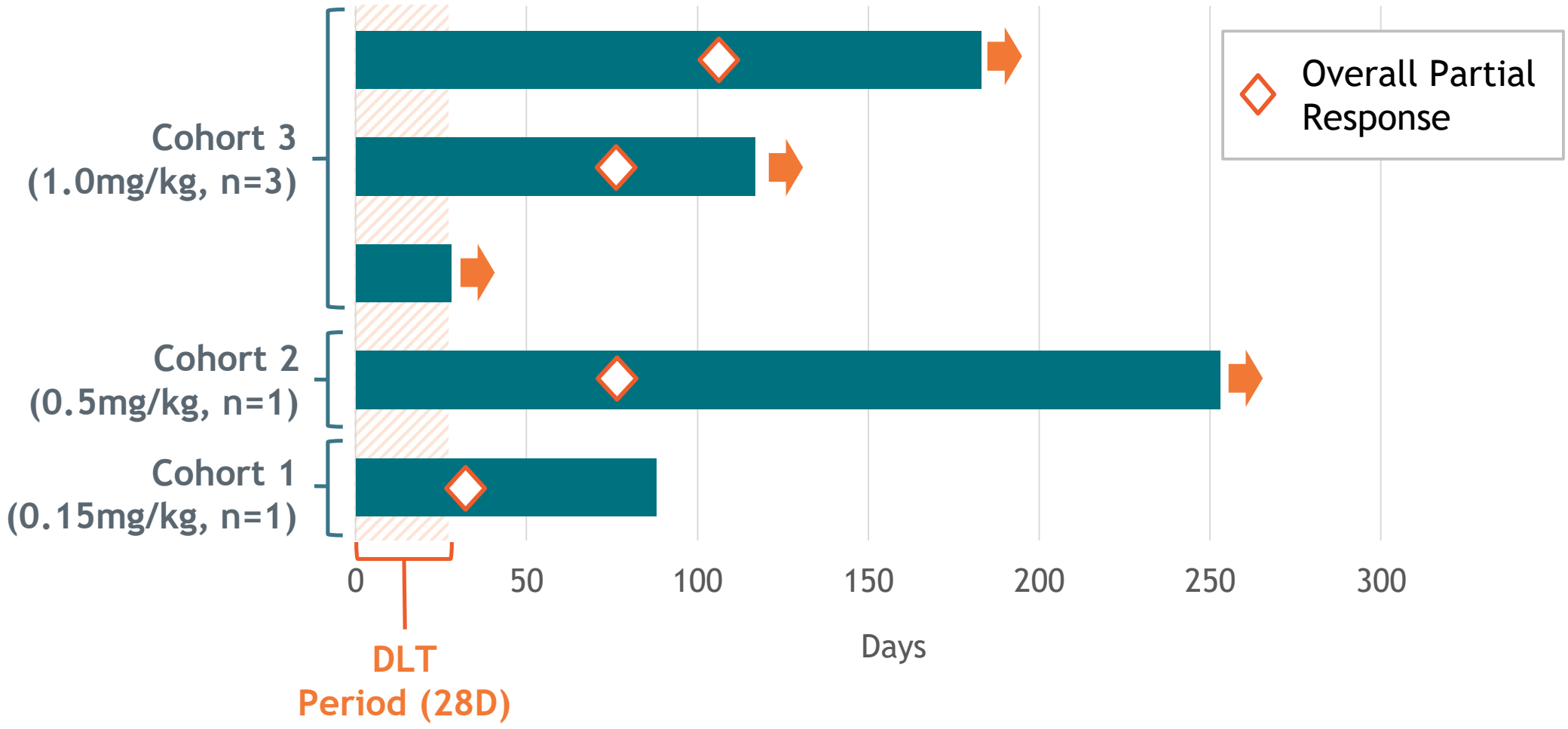
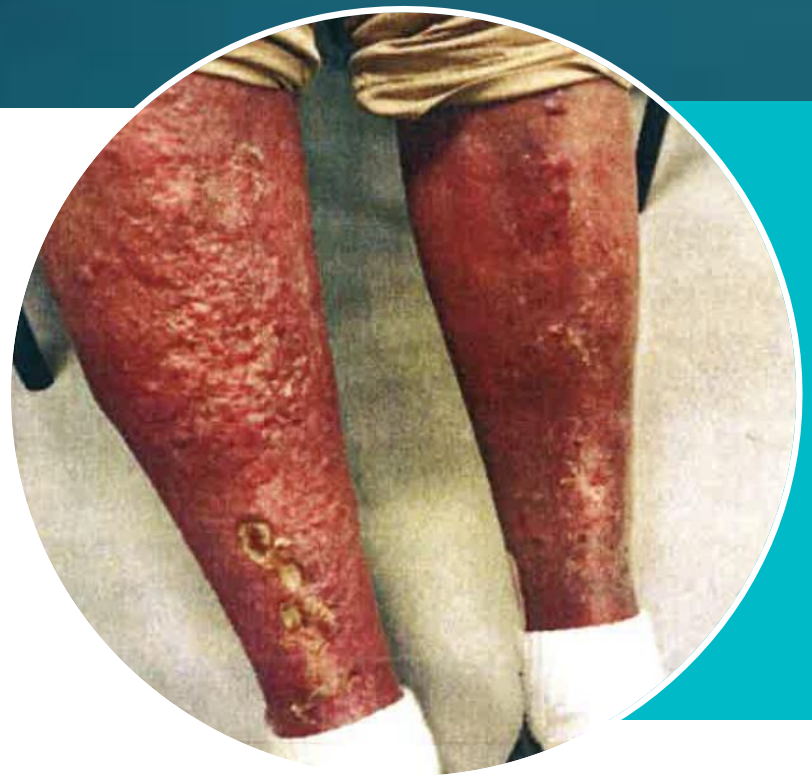
Responses observed in all evaluable patients*



*As of Dec 2019

First evidence of CSF-1R inhibition inducing responses in cGVHD

- *Patient experienced chronic condition unresponsive to prior therapies*
- *Treatment with 1mg/kg Q2W axatilimab led to significant improvement in ulceration*



1mg/kg Q2W axatilimab
initiated 6/12/19

5/15/19

9/18/19

Proven ability to build the pipeline

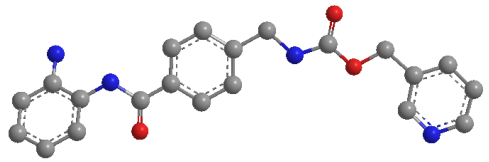
Established relationships enhance identification and access to quality assets

Clinical development leadership enables competitive advantage

Business development continues to be a core strength of our business

From Bayer

Entinostat

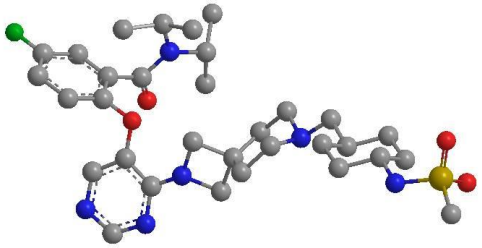


From UCB

Axatilimab

From: Allergan/Vitae

Menin-MLL inhibitors



Financial highlights and 1H 2020 financial guidance

1Q20: Raised $35 million at a 20% premium to market, closed on $20 million debt

Ticker	SNDX (NASDAQ)
Cash and short-term investments (at Dec 31, 2019)	$60 million
Cash proceeds from Q1 2020 offering and debt	$55 million
Shares Outstanding* (at Mar 3, 2020)	36 million

1H 2020 Operating Expense Guidance**	1Q 2020	2Q 2020
Research and Development	$12-13 M	$13-14 M
Total Operating Expenses^	$17-18 M	$18-19 M

* *Includes 30.2 million common shares and pre-funded warrants to purchase 5.8 million common shares*
** *Financial guidance for 2H 2020 will be issued following results of the E2112 trial*
^ *Includes $3 million non-cash stock compensation expense for 1H 2020*

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?

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