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

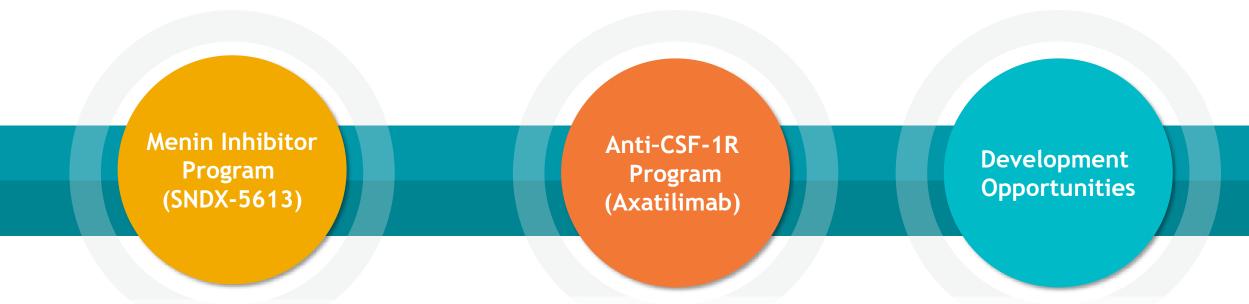


4020 EARNINGS PRESENTATION

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This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Words such as "may," "will," "expect," "plan," "anticipate" and similar expressions (as well as other words or expressions referencing future events or circumstances) are intended to identify forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding future operations, financial results and the financial condition of Syndax Pharmaceuticals, Inc. ("Syndax" or the "Company"), including financial position, strategy and plans, the progress, timing, clinical development and scope of clinical trials and the reporting of clinical data for Syndax's product candidates, and Syndax's expectations for liquidity and future operations, are forward-looking statements. Many factors may cause differences between current expectations and actual results, including unexpected safety or efficacy data observed during preclinical or clinical studies, clinical site activation rates or clinical trial enrollment rates that are lower than expected, changes in expected or existing competition, failure of our collaborators to support or advance collaborations or product candidates and unexpected litigation or other disputes. Moreover, Syndax operates in a very competitive and rapidly changing environment. Other factors that may cause our actual results to differ from current expectations are discussed in Syndax's filings with the U.S. Securities and Exchange Commission, including the "Risk Factors" sections contained therein. New risks emerge from time to time. It is not possible for Syndax's management to predict all risks, nor can Syndax assess the impact of all factors on its business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in any forward-looking statement. In light of these risks, uncertainties and assumptions, the forward-looking events and circumstances discussed in this presentation may not occur and actual results could differ materially and adversely from those anticipated or implied. Except as required by law, neither Syndax nor any other person assumes responsibility for the accuracy and completeness of the forward-looking statements. Syndax undertakes no obligation to update publicly any forward-looking statements for any reason after the date of this presentation to conform these statements to actual results or to changes in Syndax's expectations.

Syndax pipeline targets indications with significant unmet need



- Target validated for acute leukemias
- Ph 1 data expected late 1Q/early 2Q
- Ph 2 initiation expected 2Q
- Accelerated path to approval

- Macrophage driven diseases
- Ph 1 data validates target for cGVHD, additional inflammatory / fibrotic opportunities
- Pivotal trial ongoing (AGAVE-201)

 Focused on expanding clinical pipeline through development and in-licensing

SNDX-5613: Breakthrough targeted therapy for acute leukemia

Advantages

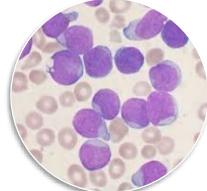
Strong target validation

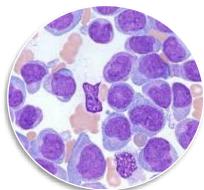
Precise patient selection

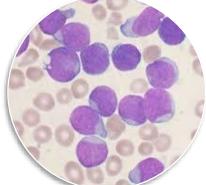
Big effect in small studies

Molecular markers of disease status

Potential for rapid regulatory path







Cre recombinase (Ma

conditional knock-in o

Namle mutant mice with Dnmt3crRicesi

We confirmed Hox or

ferent hematopoietic

nmt3a mutant mic

tion of the knock-in

At this time, mutant

leukemia and had nor

rease in granulocyt

nutant cells showed

Hozza9 mRNA expressi

atopoietic stem cells (L)

ties (Fig. 1A). Npm1c or.

cells maintained inap

Hom9 across the differ (Fig. 1A), RNA sequen-

4 weeks after activat revealed that half of t

genes in Npmlc GMI The HSC-enriched Lin

(LSK) showed much le

to their high baselin genes (Fig. 1B and tab sion programs induced

were also enriched fo

NPMIc mutant AML si

Hoza/b genes and Me

basis of these gene ex

itor (GMP) frequence

expression of genes associated with normal stem cell self-renewal, such as Hoza/h cluster genes, throughout myeloid differentiation.

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

Javant Y. Gadrev^{1,2}, Andrei V. Krivtsov^{1,2}, Frank G. Rücker³, Konstanze Döhner³, Ross L. Levine⁵, Lars Bullinger⁶, George S. Vassiliou⁷⁸, Scott A. Armstrong^{1,2}

The initiating mutations that contribute to cancer development are sometime cells. Whether therapies targeting these mutations can eradicate premalignar myeloid leukemia (AML) is an attractive system for investigating the effect of because this disease is often preceded by a premalignant state (clonal hematop syndrome). In Npm2c/Dnmt3a mutant knock-in mice, a model of AML developme by a period of extended myeloid progenitor cell proliferation and self-renewal. We f can be reversed by oral administration of a small molecule (VTP-50469) that chromatin complex. These preclinical results support the hypothesis that individ developing AML might benefit from targeted epigenetic therapy in a preventativ

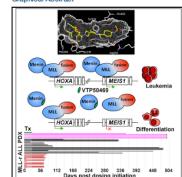
cleophosmin (NPMI) mutant acute | To identify the leui nyeloid leukemia (AML) is one of the population in NPMIc A ost common types of AML (1-3). Despite its high prevalence, the mechaism of leukemogenesis is still poorly tood, and targeted therapy options are lacking (4), NPM1 gene mutations (NPMIc) induce cytoplasmic localization of NPMI and often co-occur with other mutations in genes such as DNA methultransferase 3A (DNMT3ARS801 NPMIc leukemias express a distinctive stem cell-like gene expression pattern that includes homeobox cluster A and B (HOXA/B) genes and their DNA-binding cofactor MEIS1 (5-8). In humans, DNMT3A mutations are detected in the most primitive hematopoietic stem cell compartment, often long before the development of leukemia, a condition often referred to as clonal hematopoiesis of indeterminate potential (CHIP) (9). NPMI mutations are found in committed progenitors and differentiated myeloid cells in AML but are absent from the stem cell and lymphoid compartments (9, 10). This suggests that NPMIc may induce selfrenewal in myeloid progenitors as a critical step in the development of AML and that this aberrant progenitor self-renewal may represent a critical step in the progression from

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Cancer Cell

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

Graphical Abstract



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

Article

Correspondence

scott_armstrong@dfci.harvard.edu

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 MLLrearranged leukemia.

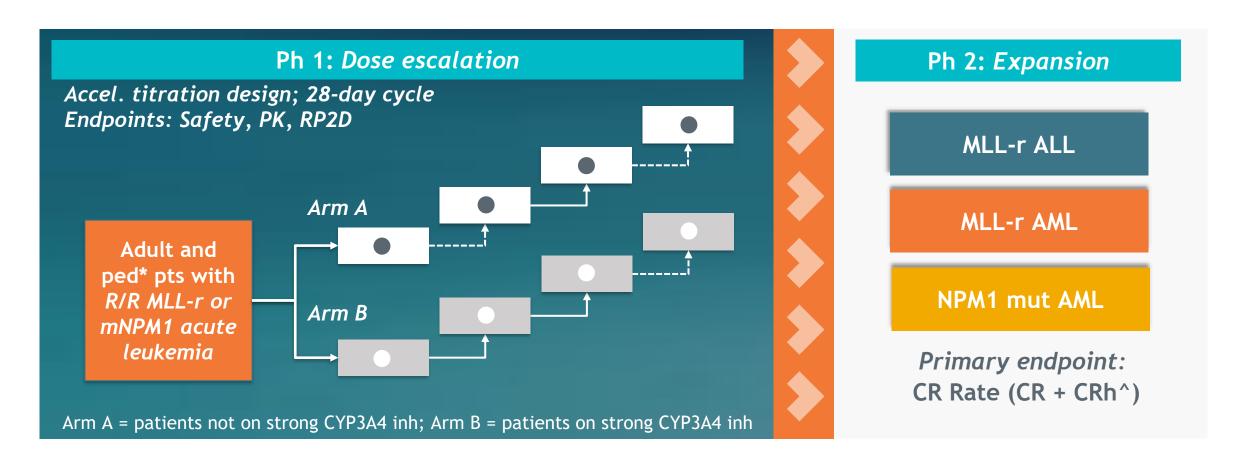
- A selective, orally bioavailable Menin-MLL inhibitor, VTP50469, is developed
- Displacement of Menin from chromatin leads to loss of MLL from specific loci
- Treatment with VTP50469 leads to suppression of a subset of MLL fusion target genes
- Treatment with VTP50469 improves survival in PDX models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



Krivtsov et al., 2019, Cancer Cell 36, 660-673



AUGMENT-101: Ph 1/2 trial testing SNDX-5613 in MLL-r and NPM1 acute leukemia



Ph 1 data late 1Q/early 2Q, Ph 2 initiation expected in 2Q

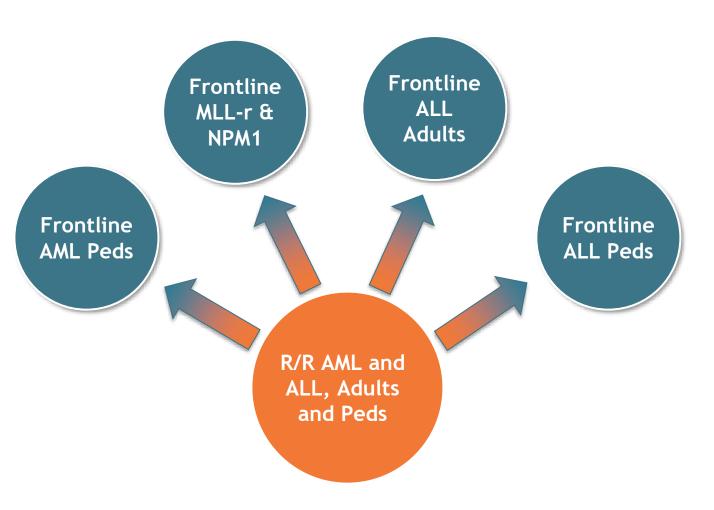
^{*} Allows patients ≥ 30 days of age; MLL-r - mixed lineage leukemia rearranged; NPM = nucleophosmin; CR = Complete response; CRh = CR with partial hematologic recovery

Multiple commercial opportunities in acute leukemias

 Potential fast to market regulatory path in R/R disease

 Subsequent approvals prioritized by medical need and commercial opportunity

 Collaborate and broaden utilization through combo and investigator-initiated trials





AGAVE-201: global pivotal trial for Axatilimab, a CSF-1R mAB, in chronic GVHD

Inclusion criteria:

- 6 years and older
- Recurrent or refractory active cGVHD after at least 2 lines of systemic therapy



Primary Endpoint: Objective Response Rate (ORR) by 2014 NIH GVHD Criteria Key Secondaries: Duration of response, improvement in modified Lee Symptom Scale

Proven ability to build the pipeline

Business development continues to be a core strength of our business

Clinical development leadership enables competitive advantage

Established relationships enhance identification and access to quality assets

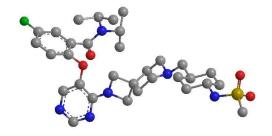
From UCB

Axatilimab



From Allergan/Vitae

Menin-MLL inhibitors



Financial highlights and FY 2021 financial guidance

Ticker		SNDX (NASDAQ)
Cash and short-term investments (at December 31, 2020)		\$293.1 million
Shares Outstanding* (at December 31, 2020)		51.4 million
1Q and 2021 Operating Expense Guidance		
	1Q 2021	FY 2021
Research and Development	\$25-30 million	\$90-100 million
Total Operating Expenses^	\$30-35 million	\$110-120 million

^{*} Includes 47.9 million common shares and pre-funded warrants to purchase 3.6 million common shares;

[^] Includes ~\$2.0 million non-cash stock compensation expense per quarter

