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

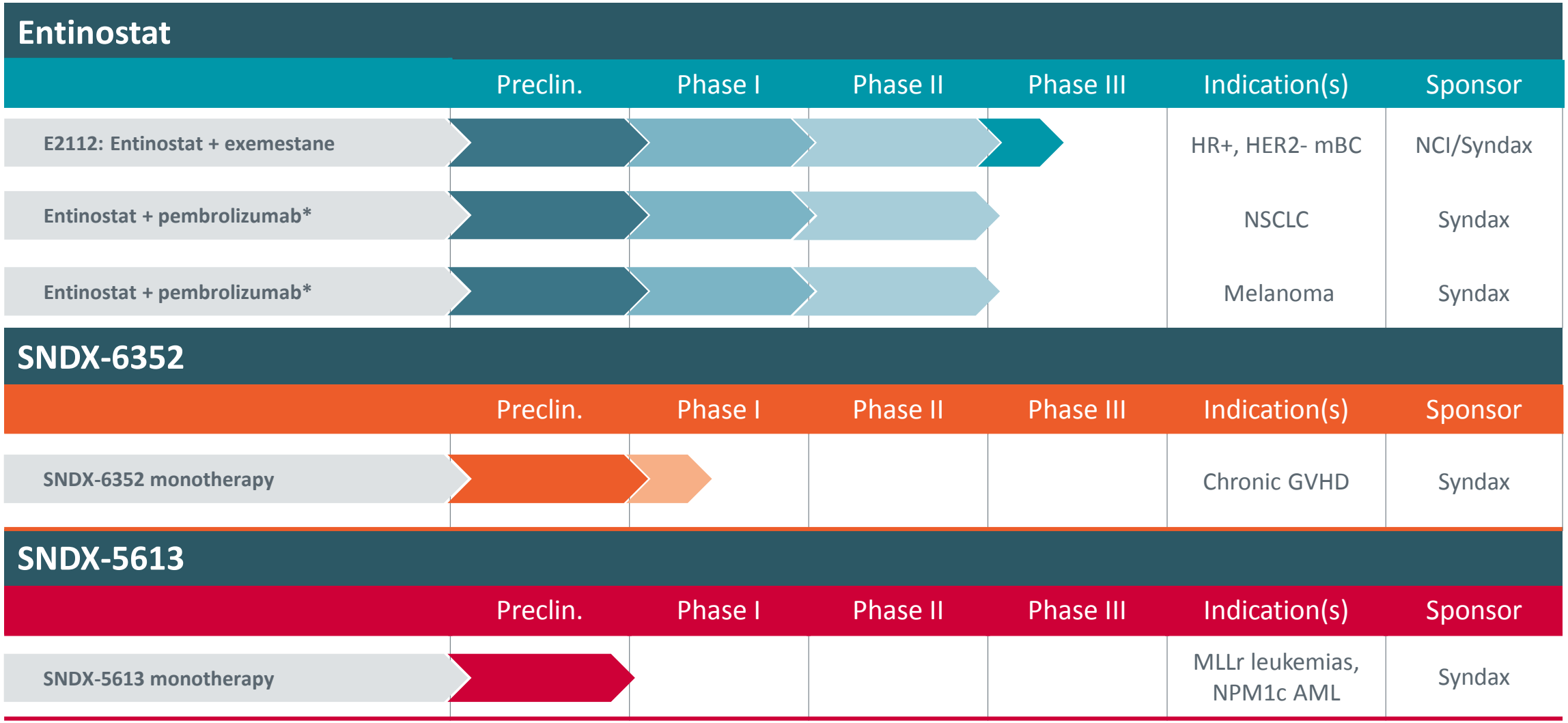
Syndax

CORPORATE PRESENTATION | JUNE 2019

Forward-looking statements disclosure

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 Current Pipeline



* Development on hold pending positive E2112 OS trial results

2019: Portfolio prioritization to drive value

Entinostat + exemestane

FDA BTD

Oral, Class I HDAC in HR+ mBC

- Positive OS data possible 2H19
- NDA filing anticipated in 2021
- Efficacy in CDK4,6 treated patients
- Blockbuster potential

Would-be first combo to demonstrate survival benefit

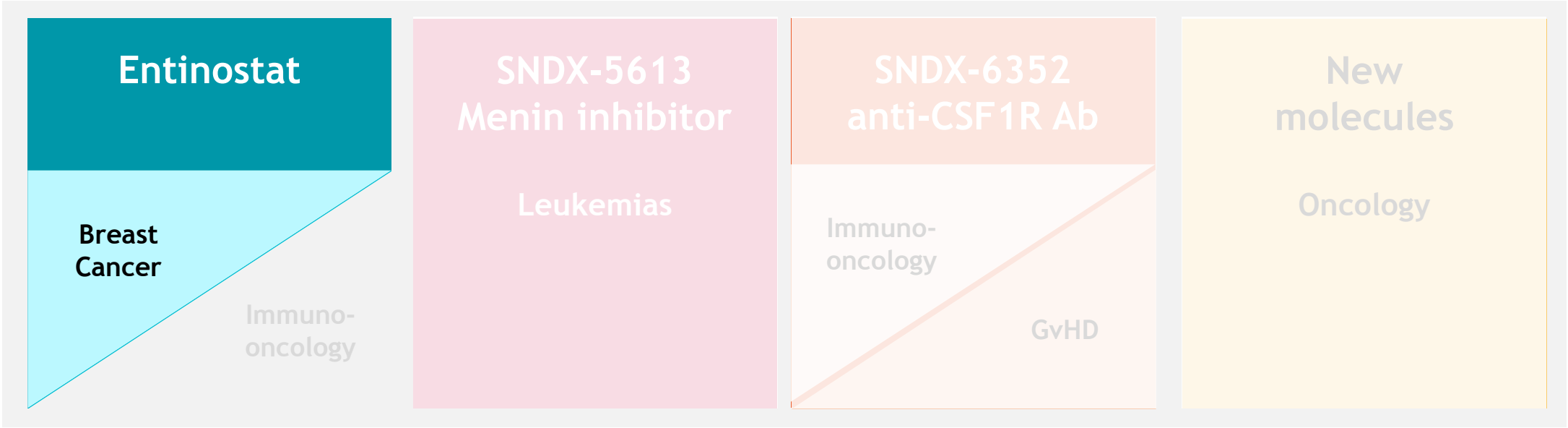
SNDX-5613

Oral, Menin inhibitor

- Blocks activity of MLL-fusion proteins
- IND filing est. 2Q, clinical data ‘19/‘20
- Benefit expected in high need AML, ALL
- Blockbuster potential

Targeted therapy provides fast to market opportunity

HR+ mBC – hormone receptor positive metastatic breast cancer; MLL – mixed lineage leukemia; AML – acute myeloid leukemia; ALL – acute lymphoblastic leukemia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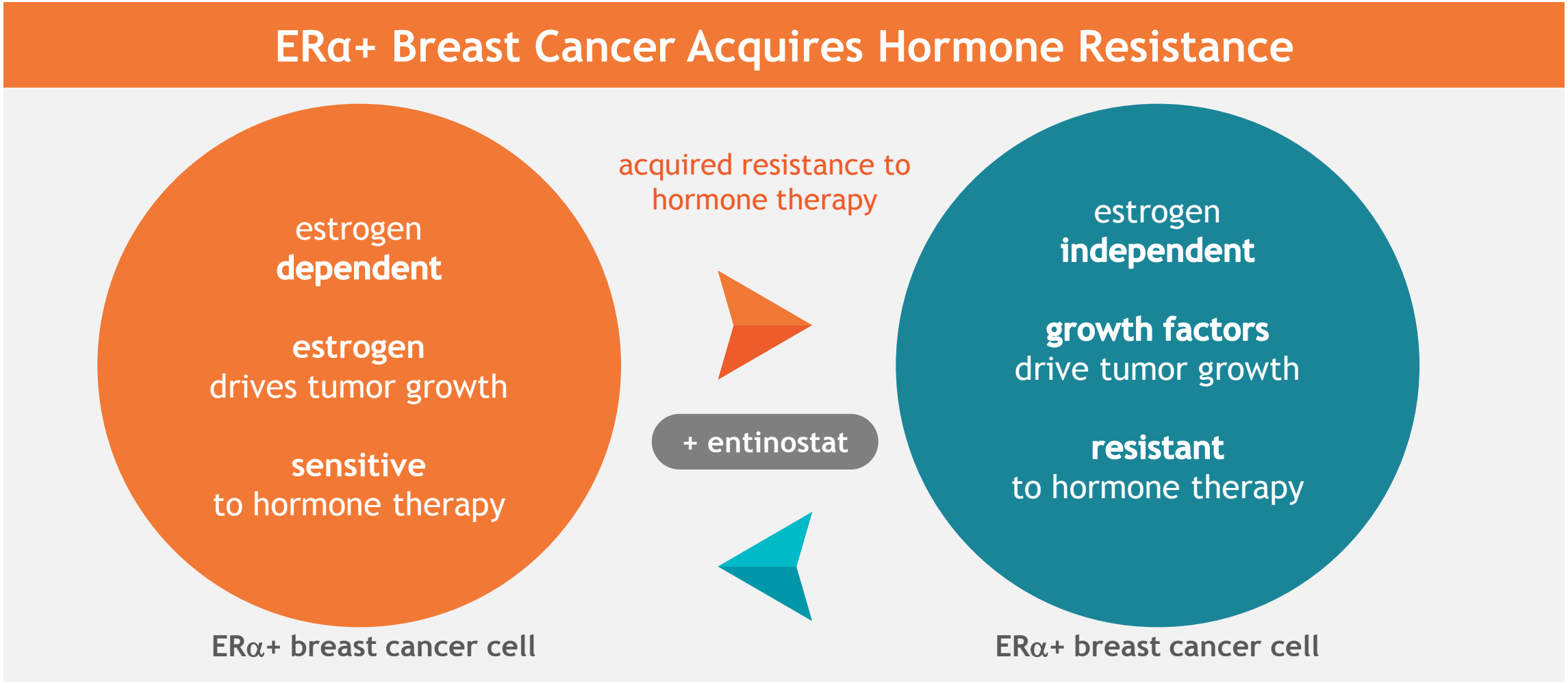
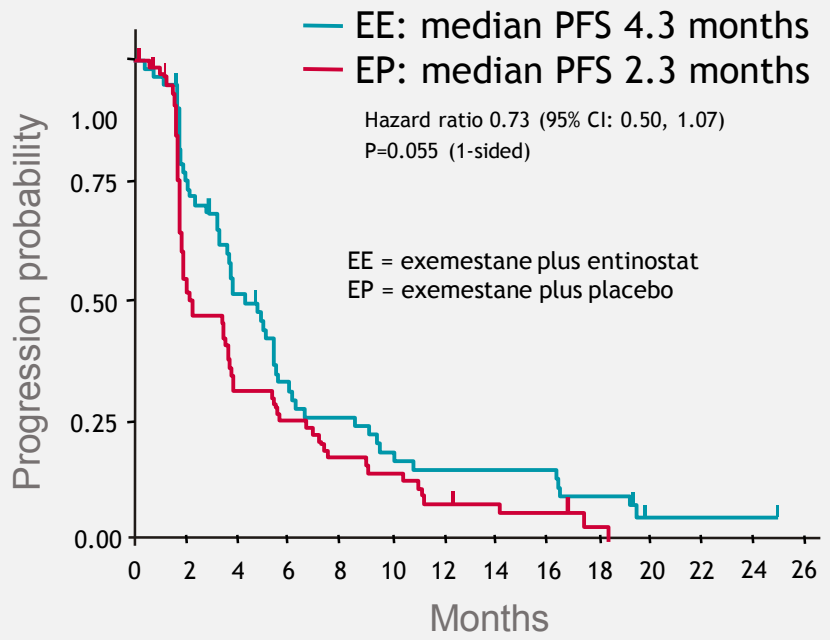
Entinostat re-sensitizes cancer cells



Phase 2 trial resulted in breakthrough therapy designation

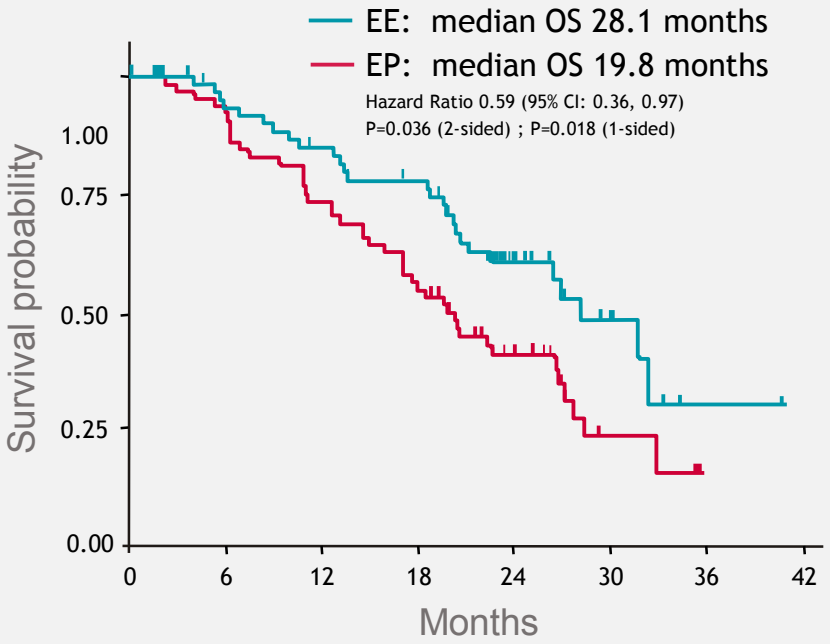
Progression-free Survival



Placebo	31/66	13/33	4/20	5/16	2/11	4/9	0/5	1/4	1/3	1/1	0/0	0/0	0/0
Entinostat	15/64	14/45	11/29	3/17	4/14	2/10	0/8	0/8	3/8	2/5	0/1	0/1	0/1

(#events / #at risk)

Overall Survival



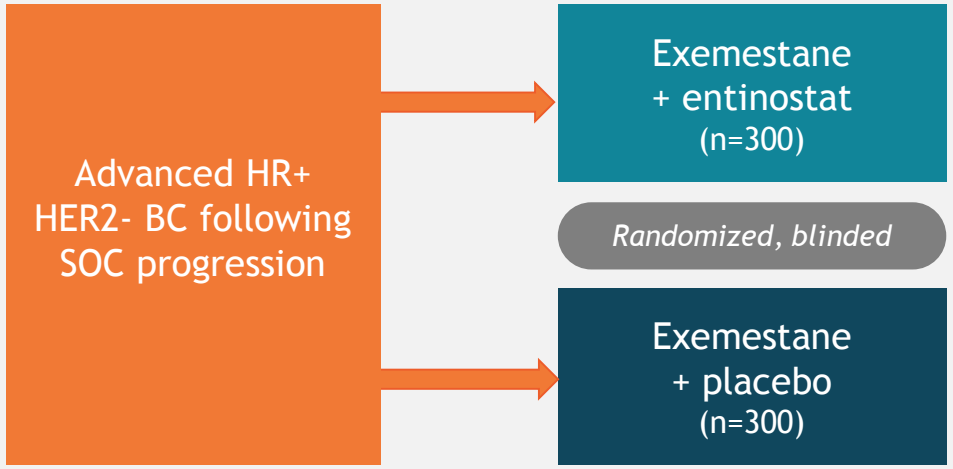
Placebo	4/66	13/60	12/47	8/35	5/18	1/3	0/0
Entinostat	4/64	5/55	4/49	9/43	3/21	2/9	0/1

(#events / #at risk)

Source: Yardley, Denise A., et al. *Journal of Clinical Oncology* 31.17 (2013): 2128-2135

Phase 3 E2112: Focused on overall survival

E2112: Exemestane +/- entinostat



Primary endpoint: OS

FDA BTD

NATIONAL CANCER INSTITUTE

ECOG-ACRIN cancer research group
Reshaping the future of patient care

Syndax

E2112 Trial Milestones

- ✓ **4Q18:** Accrual completed (n=608), PFS and interim OS analyses shared
- ✓ **2Q19:** Passed interim OS futility
- **4Q19:** Next interim OS analysis
- **2Q20:** Final OS analysis (if needed)

Expect to file NDA ~6 months after positive OS data

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

Anastrozole or letrazole +/- CDK4,6 inhibitor

2nd/3rd/4th line hormone Tx

Anastrazole, Faslodex +/- CDK4,6 inhibitor or Afinitor-exemestane

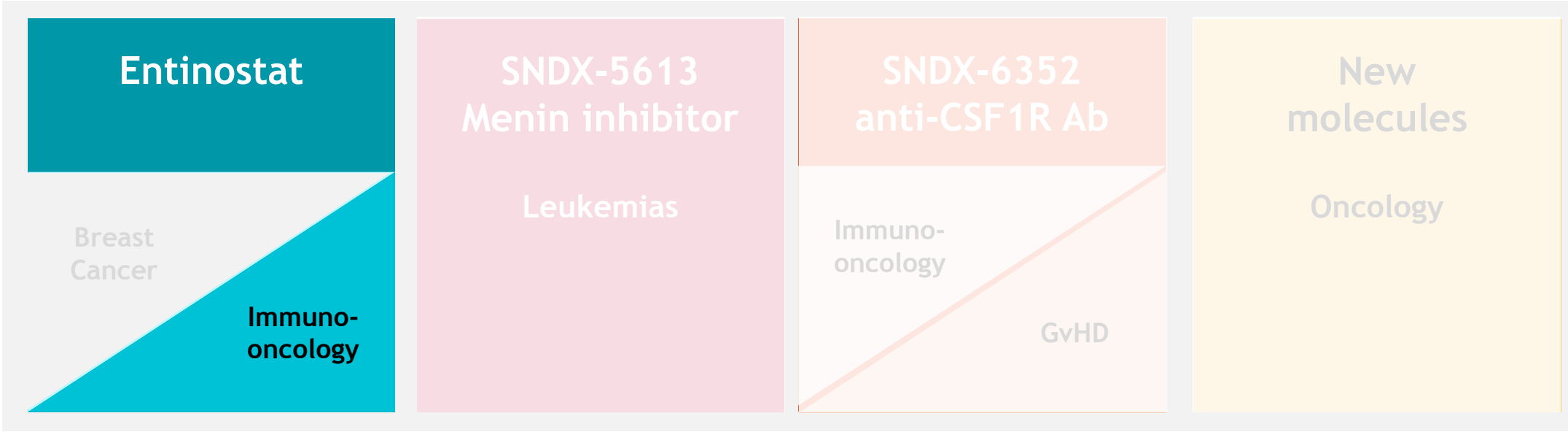
34,000 pts

Entinostat-exemestane target population

Chemo-Tx

Capecitabine, gemcitabine, eribulin

Source: DataMonitor 2016 Breast cancer: HR+/HER2- Disease Coverage Report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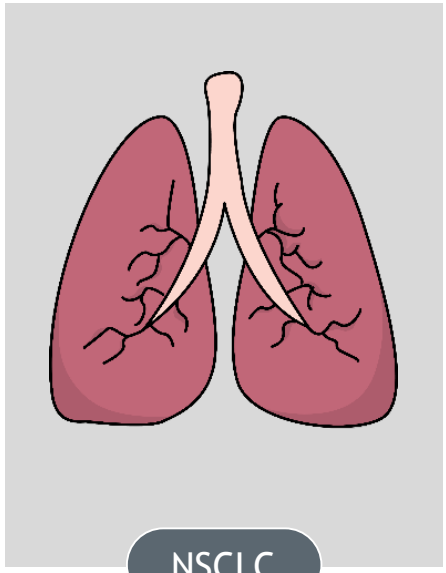
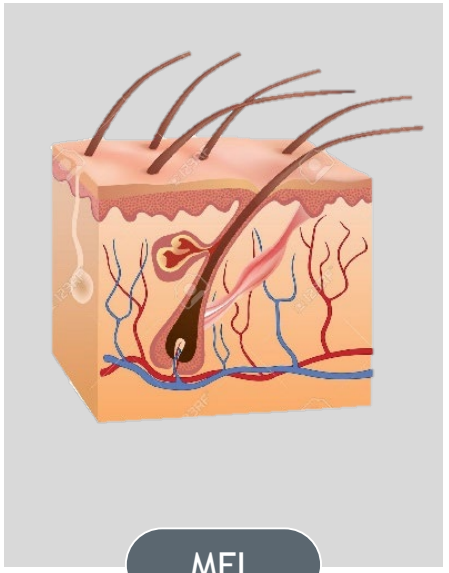
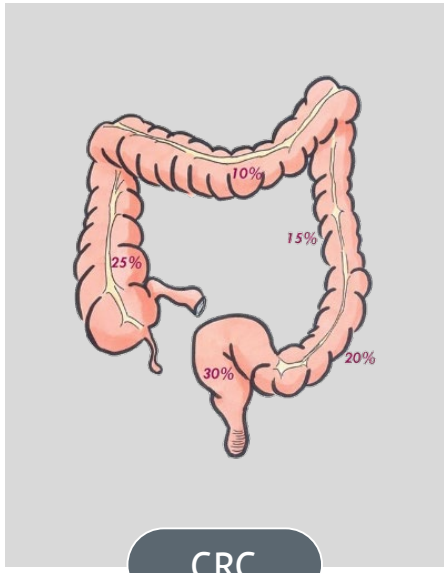
ENCORE Clinical Trial Program: Entinostat demonstrates potential to enhance PD1 efficacy



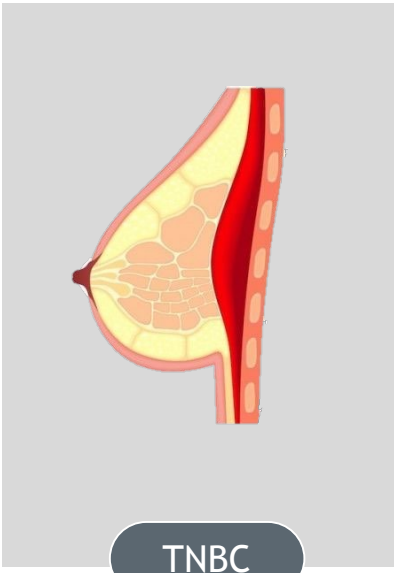
NSCLC



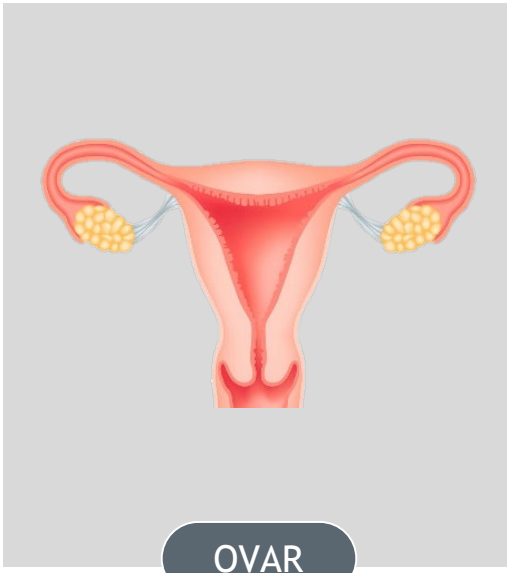
MEL



CRC



TNBC



OVAR

PD-(L)1	Immune cells	Tumor mutational burden (TMB)	Nanostring

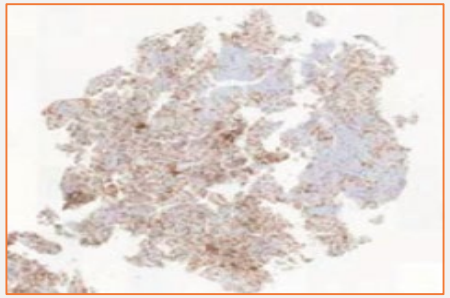
Focused on early signs of efficacy and biomarkers that predict clinical benefit

ENCORE program testing combos across immune phenotypes

Responds to PD-(L)1

Convert to “inflamed” with combinations

Inflamed



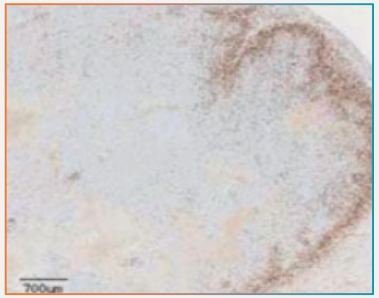
- *Abundance of TILs*
- *CD8+ Tcells, INFγ*
- *PD-(L)1 expression*

ENCORE 601

NSCLC and Melanoma

MERCK

Excluded



- *Angiogenesis*
- *MDSCs*
- *Reactive Stroma*

ENCORE 601	ENCORE 602
CRC	TNBC
MERCK	Genentech

Non-Inflamed

- *Highly proliferating tumor cells*
- *Low Tcell infiltrate*

ENCORE 603

Ovarian

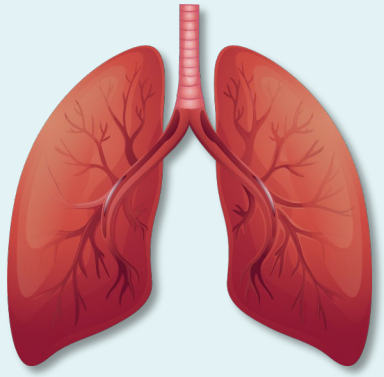
MERCK Pfizer

Source: Hedge, et al. *Clin Cancer Res*; 22.8 (2016): 1865-1874.

ENT-Keytruda shown to reverse resistance to anti-PD-1 Tx in NSCLC and MEL

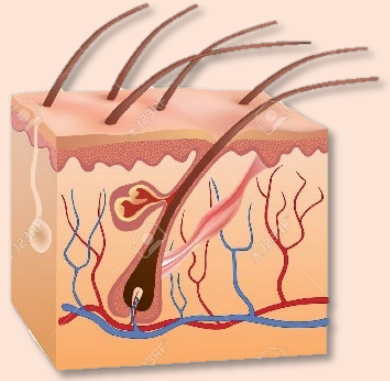
Trial cohorts enrolled patients whose disease had progressed on/after anti-PD-1 therapy

Biomarker selected NSCLC



ENT shown to down regulate myc activity; re-sensitizing pts to PD1 w/8 months mDoR

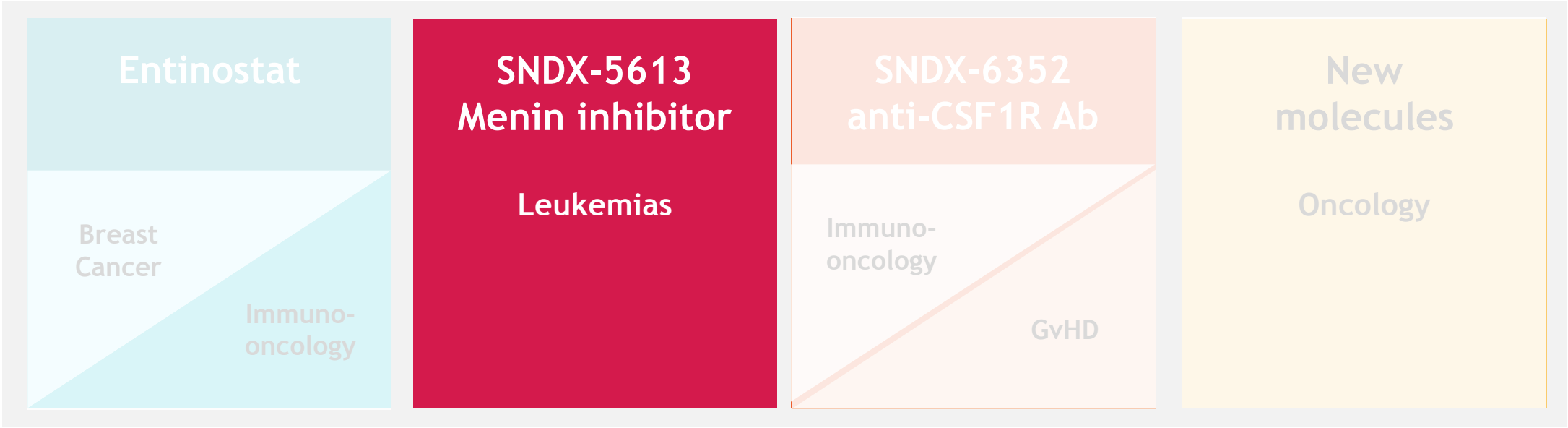
Melanoma



19% ORR, 36% CBR*; 13mo mDOR, 4.2mo mPFS similar results in pts who had prior CTLA4

"The overall medical benefit is impressive, the study is very positive for seeing the potential role for epigenetic therapy in the setting of immunotherapy." – Dr. S. Baylin (AACR 2019 oral presentation discussant)

CBR – Clinical Benefit Rate includes patients with CR, PR or SD >6 months; Source: Ramalingam, S, et al; AACR Annual meeting 2019; Sullivan, R, et al; AACR Annual meeting 2019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SNDX-5613 targets novel fusion protein: Fusion proteins proven to be good candidates for targeted therapies

Advantages

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

Therapies targeting fusion proteins

BCR-ABL

Tasigna (nilotinib)
SPRYCEL dasatinib 100 mg tablets
ICLUSIG (ponatinib) tablets
gleevec (imatinib mesylate) tablets 100mg, 400mg

EML4-ALK

ZYKADIA ceritinib 150 mg capsules
ALUNBRIG BRIGATINIB 30 mg TABLETS
ALECENSA alectinib 150 mg capsules
XALKORI CRIZOTINIB

NTRK Fusions

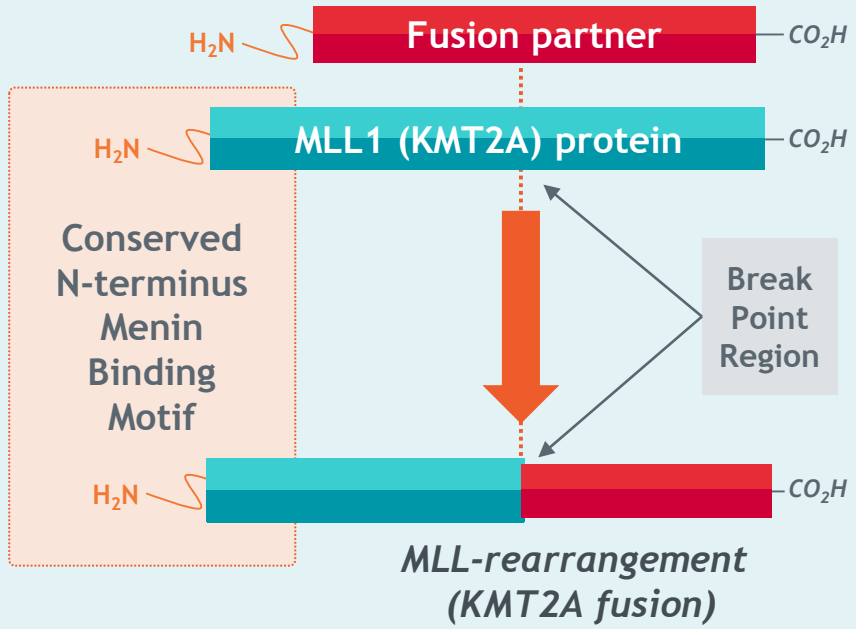
VITRAKVI (larotrectinib) 25-mg/100-mg CAPSULES 20-mg/mL ORAL SOLUTION
entrectinib (Ignyta/Roche)

RET Fusions

LOXO-292 (Loxo/Lilly)
ALECENSA alectinib 150 mg capsules
BLU-667 (Blueprint)

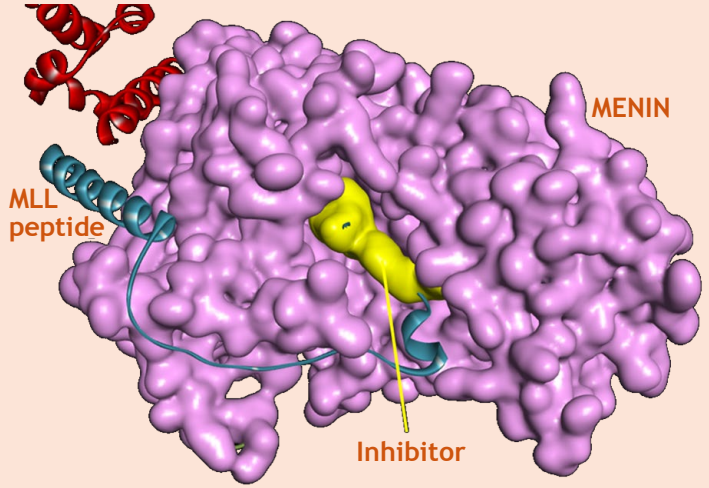
In MLLr, leukemic transformation is highly dependent on the Menin-MLL interaction

MLL1 Transformations



MLL1 fusion partner recruits oncogenic factors to transcription site[1]

Menin

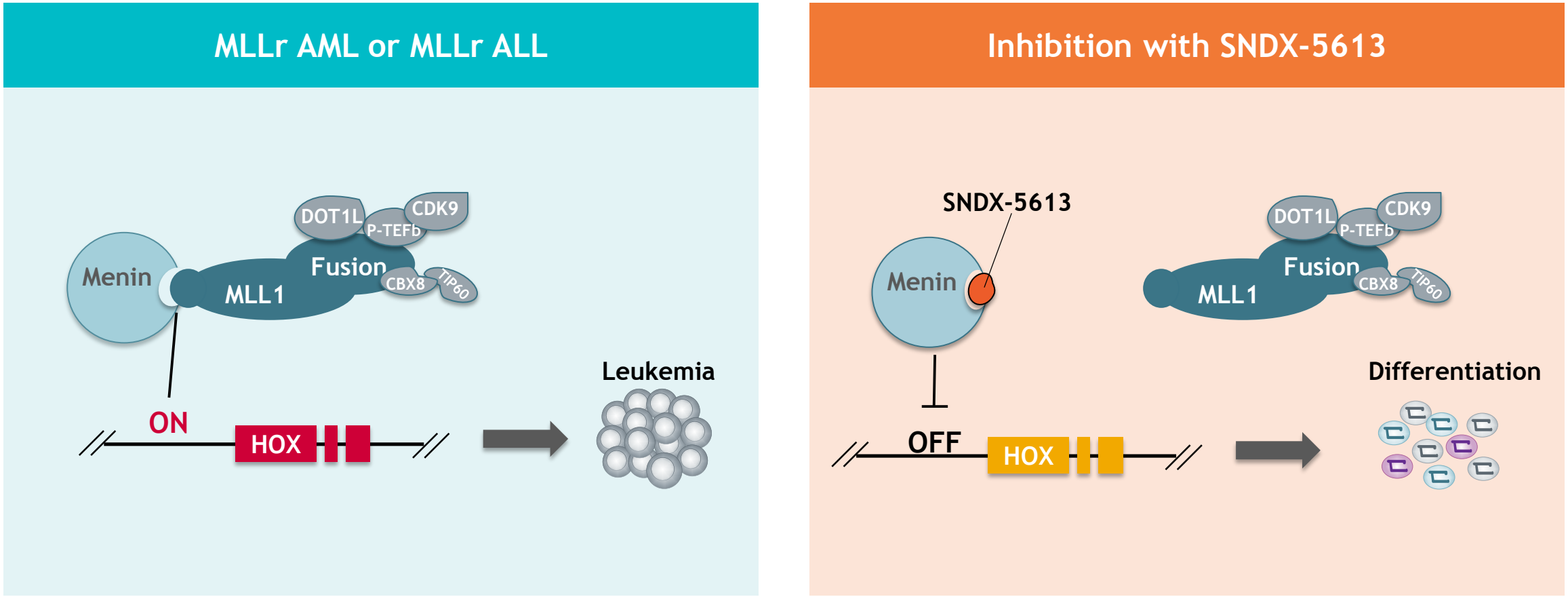


- Menin is a scaffold protein involved in gene transcription
- MLL1 N-terminus binds w/in conserved pocket on Menin

SNDX-5613 designed to block site of interaction

Source: Yokoyama A, Cell. 2005 Oct 21;123(2):207-18; Caslini C, *Cancer Res. 2007* Aug 1;67(15):7275-83.

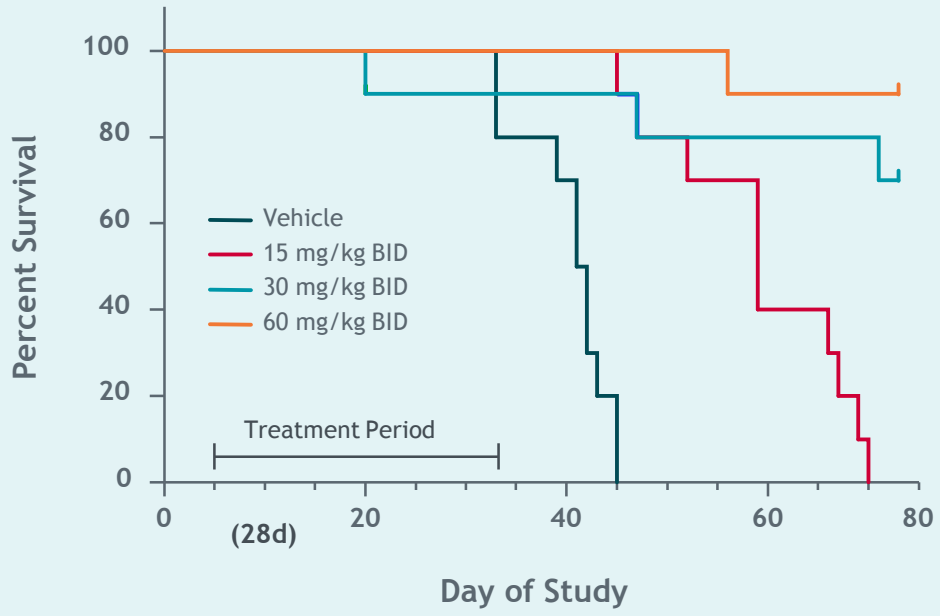
Binding of Menin to MLL1 leads to upregulation of HOX gene transcription and leukemia in MLLr AML and MLLr ALL



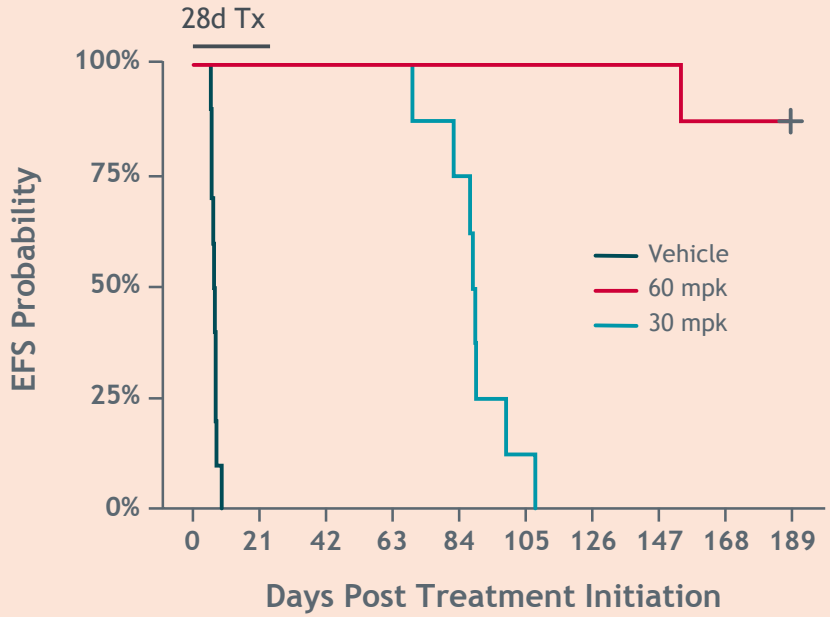
Adopted from: Uckelmann HJ, et al. Presented at ASH Annual Meeting, 2018.

Menin-MLL inhibition significantly prolongs survival in MLLr xenograft models

MLLr Cell Line Xenograft (MV4;11) (SNDX-469)



Patient Derived Xenograft (PDX) MLL-AF4 (SNDX-469)

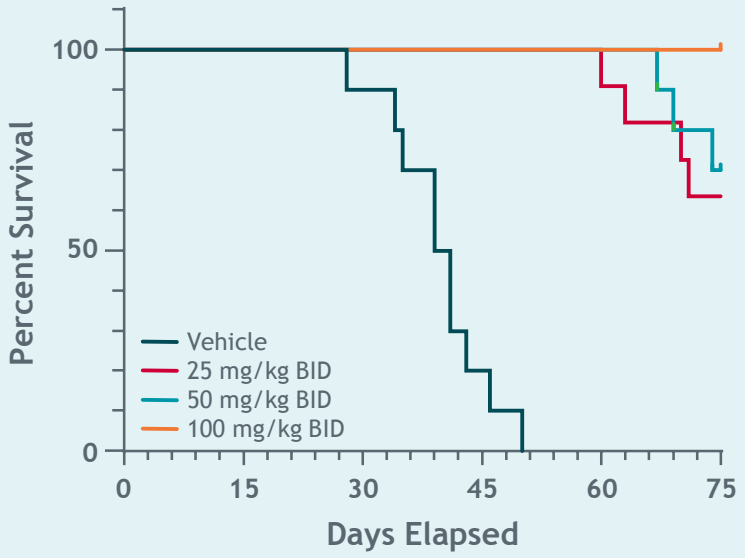


SNDX-469 shows profound, single agent treatment benefit in multiple models

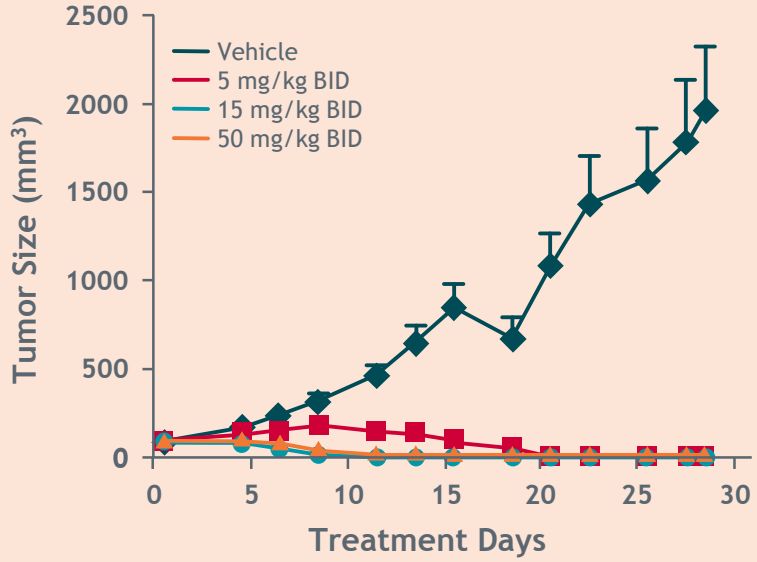
Source: Kristov, A., 2018 American Association for Cancer Research annual meeting

SNDX-5613 selected from Menin-MLL portfolio

MV4;11 Leukemia, NSG mice (SNDX-5613)



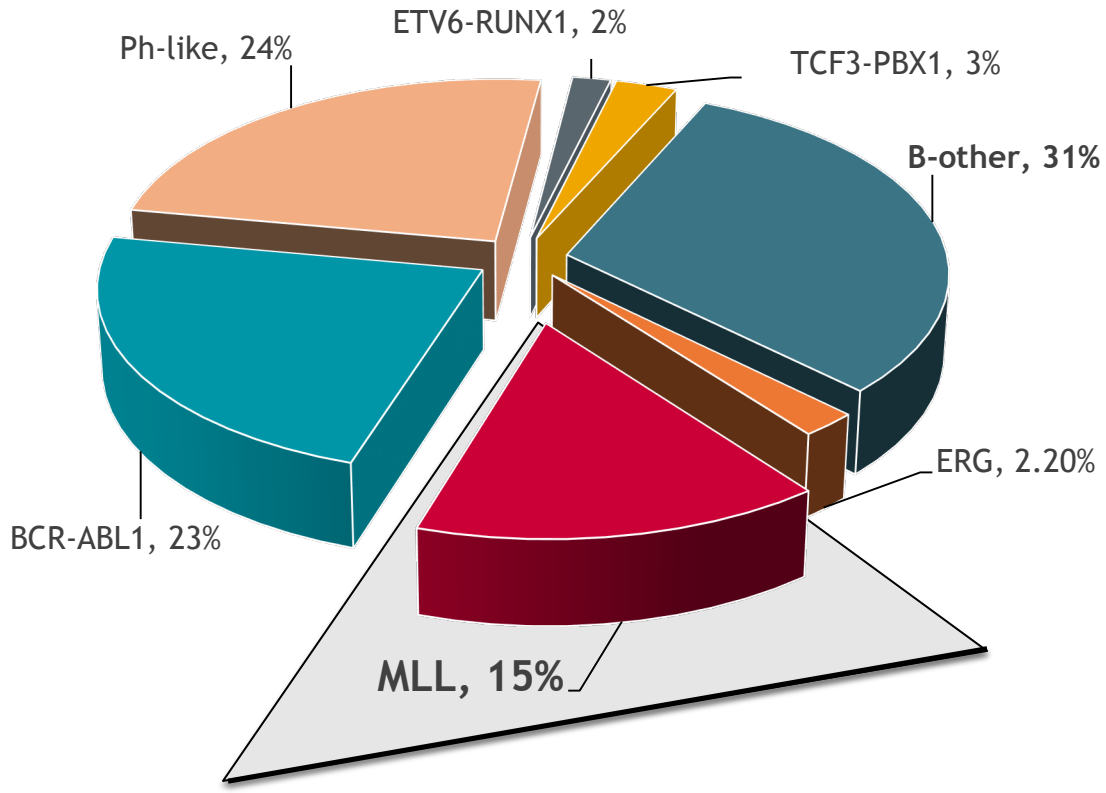
MV4;11 s.c. Xenograph, nude rat (SNDX-5613)



SNDX-5613 shows dose dependent effect on tumor growth and survival across a range of xenografts harboring MLL-r fusions and has the most potent anti-tumor activity among tested inhibitors

Source: Syndax data on file

SNDX-5613 potentially effective in MLLr - ALL; distinct molecular subtype of ALL conferring a worse prognosis



5-year survival

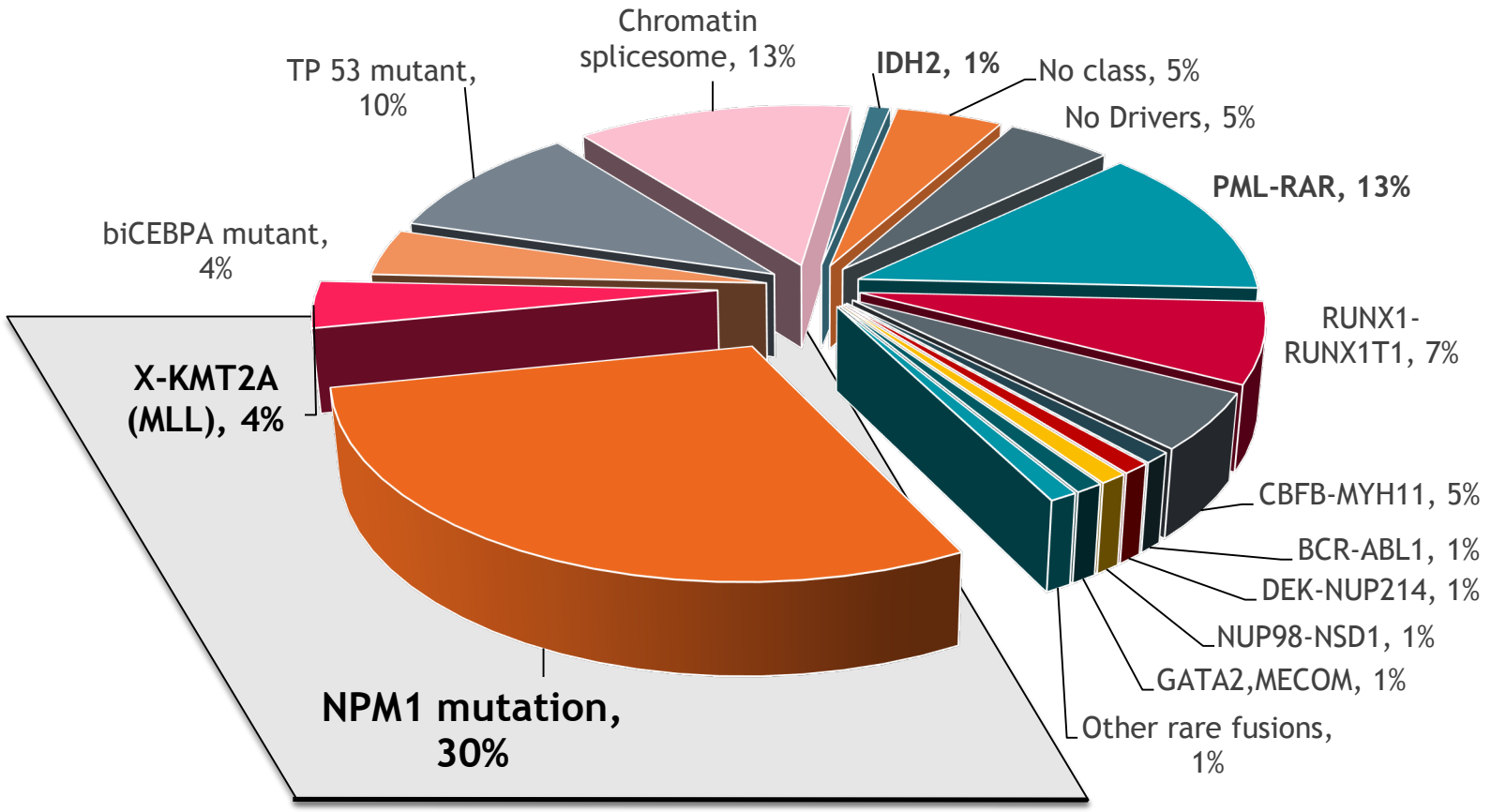
Pediatric ALL: 75%-90%

MLLr ALL: ~50% for infants and ~60% >1 yr

WW incidence ~1,000/yr
10-15% ALL, 80% infant ALL

Adopted from: Shah, B. and Nasello, D. Jan 2019; NCCN conference and meetings: Update on Management of Acute Lymphoblastic Leukemia.

SNDX-5613 poised to target MLLr and NPM1 classes of AML; distinct subsets representing ~34% of AML



WW Incidence

MLLr AML (4 - 10% AML)
~3,000 patients / year

NPM1 AML (30% AML)
~20,000 patients / year

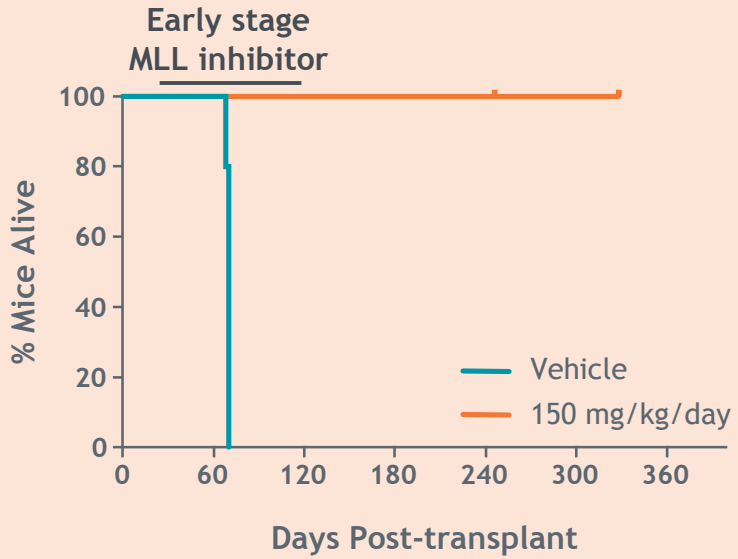
AML 5 yr survival 5% - 55%

Adopted from: Dohner, H. et al. Blood, 2017; 129(4):424-447

Preclinical models of NPM1 AML reveal profound single agent activity of Menin inhibition

- NPM1 mutation is the most frequent molecular alteration in AML
- Like MLLr, NPM1 AML depends on genes known to be sensitive to Menin-MLL interaction
- Standard AML screening identifies NPM1 mutation today

NPM1c; FLT3 ITD; PHF6 mice
(treated with SNDX-469 or vehicle)

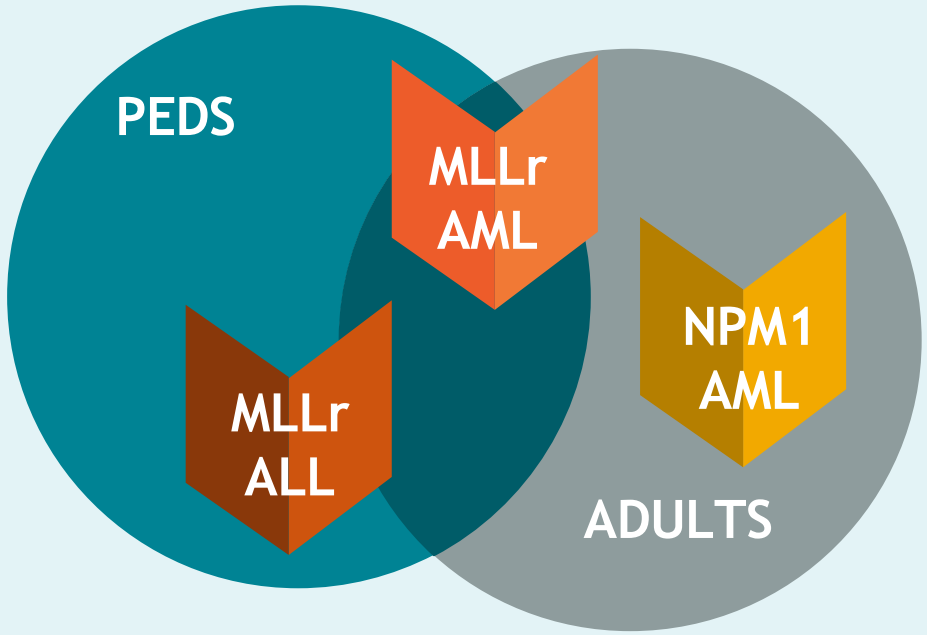


NPM1 transfected mice showed profound single agent survival benefit with SNDX-469 in multiple PDX models

Source: Kühn MW, *Cancer Discov*. 2016 Oct;6(10):1166-1181; Kristov, A., 2018 American Association for Cancer Research annual meeting

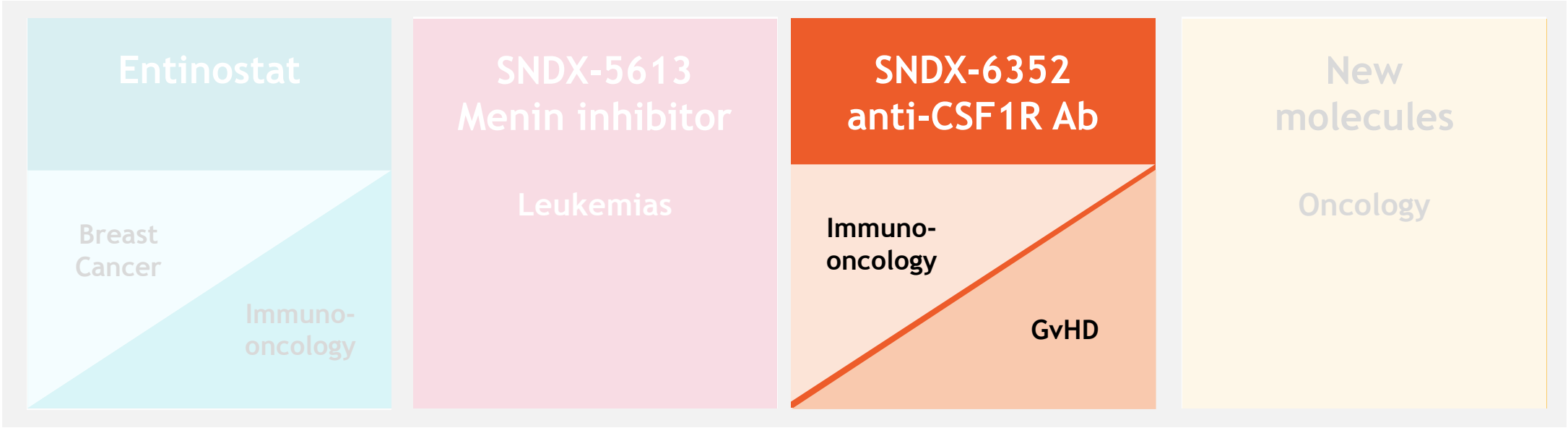
SNDX-5613: potential best-in-class, targeted, oral agent with single agent activity and fast to market potential

Phase 1/2 trial population:
MLLr adult, MLLr peds, NPM1 mut AML



Defined fast to market pathway

- **IND filing est. 2Q19; Phase 1 to follow**
  - Early efficacy possible as early as year-end 2019
- **MLLr and NPM1 identified today with standard screening protocols**
- **No approved therapies targeting MLLr or NPM1 acute leukemias**
  - $$B commercial opportunity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New molecules
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Update on SNDX-6352: pursuing novel indication

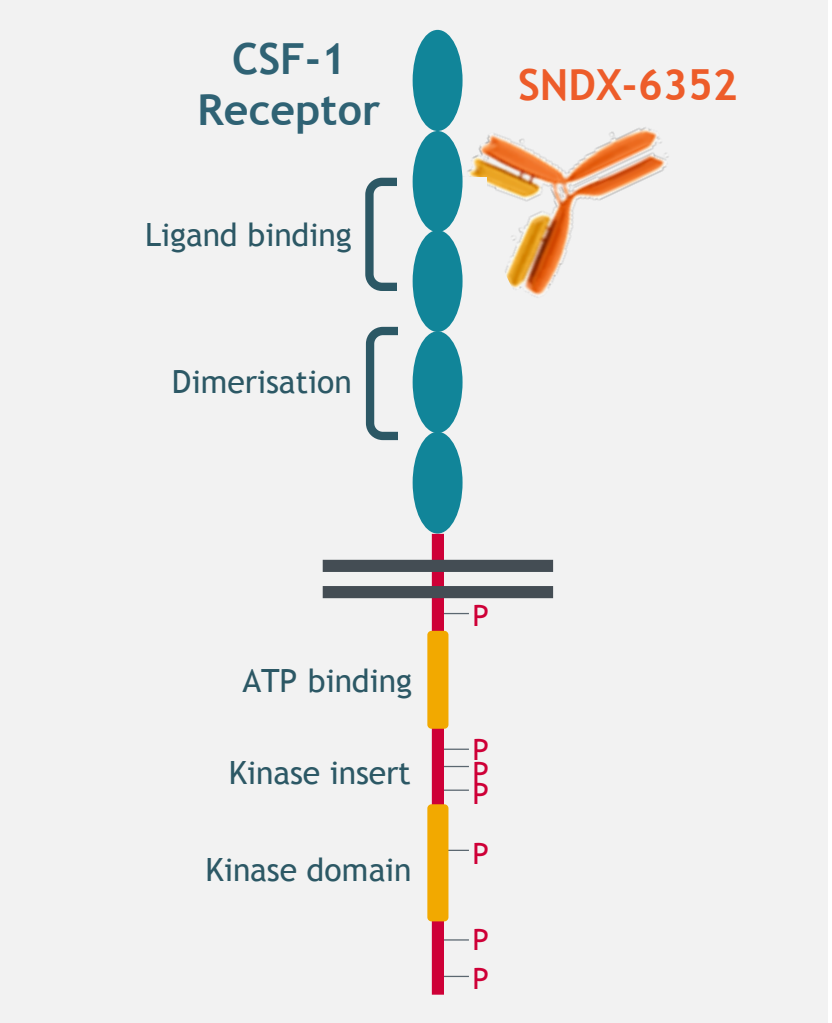
High affinity, IgG4 (K_D = 4-8 pM)

✓ Chronic graft versus host disease (cGVHD) study initiated

- RP2D expected in 2H19

✓ Multiple ascending dose studies ongoing

- Monotherapy (solid tumors) and IMFINZI (durvalumab, AZ) combo
- RP2D expected in 2Q19

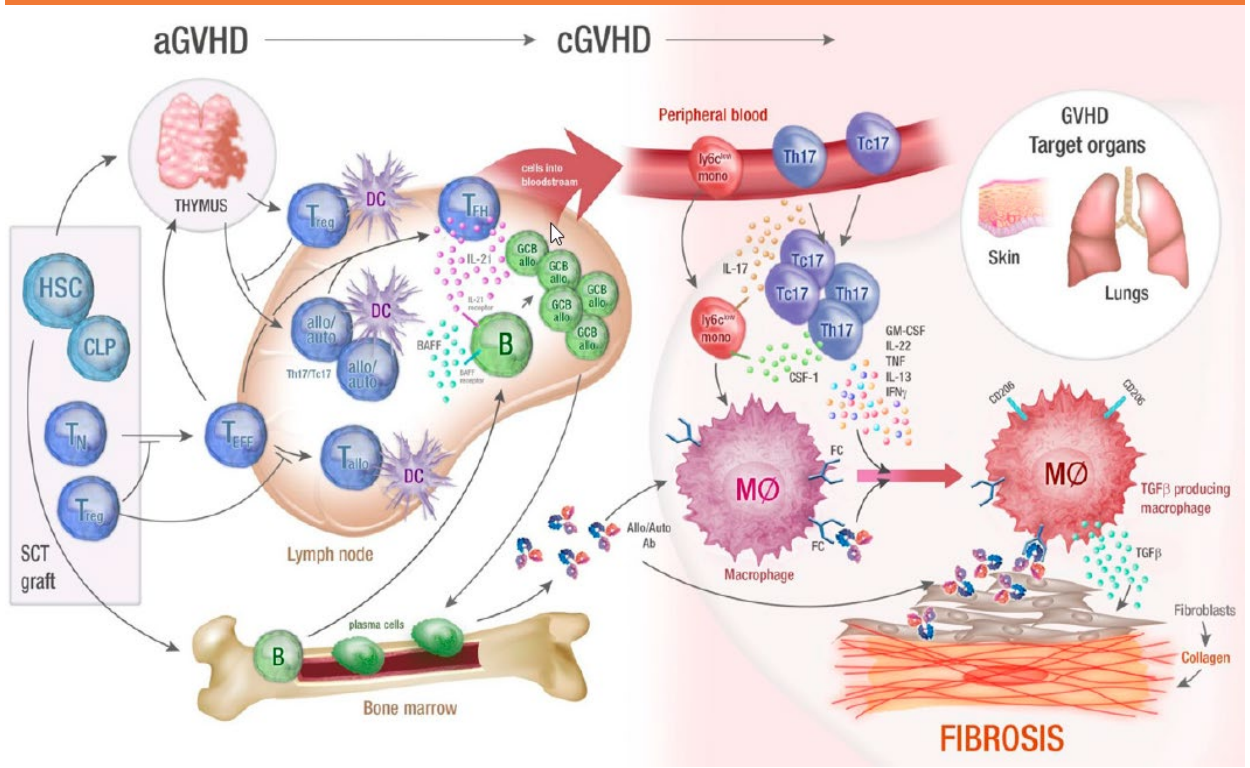


CSF-1R – colony stimulating factor -1 receptor; RP2D recommended Phase 2 dose.
Source : Ordentlich, P. et al SITC 2016.

CSF-1 pathway may play a meaningful role in cGVHD

- Preclinical data implicates CSF-1 in cGVHD
- cGVHD develops in 30-70% of HCST[1]
  - US 5,000
  - Global 12,500
- Phase 1 data expected 3Q19
  - Primary outcome measures:
    - Progression (2014 NIH GVHD criteria)
    - Optimal biologic dose (OBD)
    - RP2D

Cellular and molecular mediators of GVHD pathology[2]



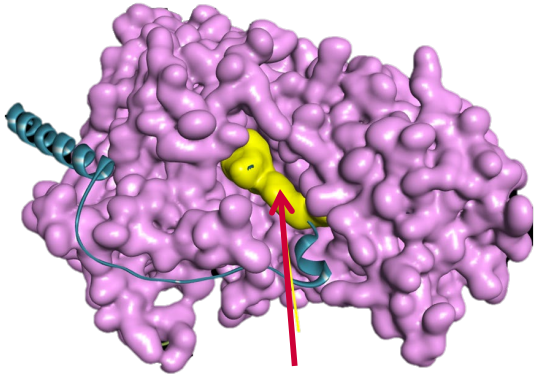
1. https://www.fda.gov/newsevents/newsroom/pressannouncements/ucm569710.htm.
2. MacDonald, K.P.A. et al., *BLOOD*, 5 (129) 13-21.

Proven ability to build the pipeline

3Q16: UCB

SNDX-6352

4Q17: Allergan/Vitae



Menin-MLLr inhibitors

- 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

March 2019 financing: $27.4 million net proceeds extends cash runway

- **Completed deal with key investors, led by BVF**
- **Issued 4.6M shares and prefunded warrants @ $6.00 (premium to market) and 4.6 M series warrants priced at $12 and $18**
  - Warrants expire on the earlier of E2112 positive OS data + 3 months or Dec 31, 2020
- **31.6 million total shares outstanding post financing**

Q1 2019 financial highlights and 2Q, full-year 2019 guidance

Ticker	SNDX (NASDAQ)	
As of March 31, 2019		
Cash and short-term investments	$92.7 million	
Shares Outstanding*	31.6 million	
2019 2Q and full year Operating Expense Guidance		
	2Q 2019	**2019**
Research and Development	$9 – 10 M	$46 - 50 M
Total Operating Expenses^	$13 – 14 M	$60 – 64 M

** Includes 27.1 million common shares and pre-funded warrants to purchase 4.5 million common shares*

^ Includes $1.5 and $6 million non-cash stock compensation expense for 2Q 2019 and for 2019, respectively

Upcoming milestones

ENTINOSTAT (Class 1 specific HDAC inhibitor)	2Q19	3Q19	4Q19	1H20
E2112 – upcoming OS analyses*			●	●

* Final 1H20 OS analysis will only be conducted if needed

SNDX-5613 (Menin inhibitor)	2Q19	3Q19	4Q19	1H20
Investigational New Drug (IND) application	●			
Potential for early efficacy in relapsed refractory AML			▬	▬

SNDX-6352 (anti-CSF-1R mAB)	2Q19	3Q19	4Q19	1H20
Identify recommended Phase 2 dose and schedule	●			
Preliminary efficacy in chronic GVHD		●		

Significant value drivers in 2019

Entinostat - exemestane

FDA BTD

Oral, Class I HDAC in HR+ mBC

- Positive OS data possible 2H19
- NDA filing anticipated in 2021
- Efficacy in CDK4,6 treated patients
- Blockbuster potential

Would-be first combo to demonstrate survival benefit

SNDX-5613

Oral, Menin inhibitor

- Blocks activity of MLL-fusion proteins
- IND filing est. 2Q, clinical data ‘19/‘20
- Benefit expected in high need AML, ALL
- Blockbuster potential

Targeted therapy provides fast to market opportunity

HR+ mBC – hormone receptor positive metastatic breast cancer; MLL – mixed lineage leukemia; AML – acute myeloid leukemia; ALL – acute lymphoblastic leukemia

Thank you. Questions?

Syndax