KRONOS·BIO

Corporate Presentation

May 2021

KRONOS·BIO

Forward-Looking Statements

This presentation includes certain projections and forward-looking statements as of the date of this presentation provided by Kronos Bio, Inc. (the “Company”). The information in this presentation is current only as of its date and may have changed since that date. These projections and forward-looking statement include, but are not limited to, those regarding the Company’s future financial position, the Company’s strategy, intellectual property matters, the Company’s clinical development plans and timelines, regulatory matters, market size and opportunity and the Company’s estimates regarding expenses capital requirements and needs for additional financing. These projections and forward-looking statements are based on the beliefs of the Company’s management as well as assumptions made and information currently available to the Company. Such statements reflect the current views of the Company with respect to future events and are subject to business, regulatory, economic and competitive risks, uncertainties, contingencies and assumptions about the Company, including, among other things, the development of its business, trends in the industry, the legal and regulatory framework for the industry and future expenditures. In light of these risks, uncertainties, contingencies and assumptions, the events or circumstances referred to in the forward-looking statements may not occur. None of the future projections, expectations, estimates or prospects in this presentation should be taken as forecasts or promises nor should they be taken as implying any indication, assurance or guarantee that the assumptions on which such future projections, expectations, estimates or prospects have been prepared are correct or exhaustive or, in the case of the assumptions, fully stated in the presentation. The actual results may vary from the anticipated results and the variations may be material.

This presentation discusses product candidates that are under clinical study and which have not yet been approved for marketing by the U.S. Food and Drug Administration. No representation is made as to the safety or effectiveness of these product candidates for the use for which such product candidates are being studied.

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KRONOS·BIO

- **Introduction**
- Targeting Oncogenic TRNs
- Lead Programs
  - SYK
  - CDK9
- The Kronos Bio Opportunity

~~IM~~POSSIBLE.
~~UN~~DRUGGABLE.
~~UN~~ACHIEVABLE.

Kronos Bio Investment Highlights

- Pioneering a new approach to target a potentially large market opportunity – dysregulated transcription factors and their associated TRNs
- SYK inhibitor program including registrational Phase 3 trial to begin in mid-2021
- Highly differentiated CDK9 program targeting MYC-amplified tumors
- Proprietary product engine to drive accelerated expansion of product candidates focused on high-value targets
- Highly experienced management team and support from leading investors

Dedicated to Transforming Patient Outcomes in Cancer by Targeting Dysregulated Transcription

Demonstrated leadership advancing transformative therapies

Leadership Team

Norbert Bischofberger, PhD.
President and Chief Executive Officer

Barbara Kosacz
Chief Operating Officer and General Counsel

Jorge DiMartino, M.D., Ph.D.
Chief Medical Officer and Executive Vice President, Clinical Development

Yasir Al-Wakeel, BM BCh
Chief Financial Officer and Head of Corporate Development

Christopher Dinsmore, Ph.D.
Chief Scientific Officer

Board of Directors

Arie Belldegrun, M.D., FACS
Co-Founder and Chair

Norbert Bischofberger, Ph.D.
President and Chief Executive Officer

Marianne De Backer, Ph.D., MBA
Bayer AG

Joshua Kazam
Co-Founder, Two River

Jakob Loven, Ph.D.
Nextech

Elena Ridloff
Acadia Pharmaceuticals

Otello Stampacchia, Ph.D.
Omega Funds

David Tanen
Two River

Taiyin Yang, Ph.D.
Gilead Sciences

Kronos Bio leadership track record:

Successfully commercialized over 25 therapeutic products across multiple indications

Arie Belldegrun, M.D., FACS

Co-founder & Chair

- Chair, Two River & Co-founder, Vida Ventures
- Founder of Kite Pharma (acquired by Gilead), Cougar Biotechnology (acquired by J&J) and Agensys (acquired by Astellas)
- Professor of Urology, and Director of the UCLA Institute of Urologic Oncology

Norbert Bischofberger, Ph.D.

President & CEO

- Former CSO and EVP of R&D at Gilead Sciences
- Oversaw development and NDA approvals of more than 25 therapeutic products for a range of serious conditions

Truvada®

ATRIPLA

Letairis® ambrisentan 5 mg and 10 mg Tablets

Zytiga® (abiraterone acetate) 250 mg, 500 mg tablets

YESCARTA® (axicabtagene ciloleucel) Suspension for IV infusion

COMPLERA® emtricitabine 200mg/rilpivirine 25mg/ tenofovir disoproxil fumarate 300mg tablets

Zydelig™ (idelalisib) 150 mg 100 mg tablets

STRIBILD® 100 elvitegravir 150mg/ cobicistat 150mg/ emtricitabine 200mg/ tenofovir disoproxil fumarate 300mg tablets

Viread® 300 mg tablets tenofovir disoproxil fumarate

EPCLUSA® sofosbuvir/velpatasvir 400 mg/100 mg tablets

Genvoya® elvitegravir 150mg/cobicistat 150mg/emtricitabine 200mg/tenofovir alafenamide 10mg tablets

BIKTARVY® bictegravir 50mg/emtricitabine 200mg/ tenofovir alafenamide 25mg tablets

Tamiflu® oseltamivir phosphate Prescription for flu

HARVONI® ledipasvir/sofosbuvir 90 mg/400 mg tablets

SOVALDI® SOFOSBUVIR

Advancing a robust pipeline of targeted oncology programs

Product Candidate	Discovery	IND-Enabling Studies	Phase 1 Trial	Phase 2 Trial	Phase 3 Trial	Next Anticipated Milestones
Entospletinib (SYK Inhibitor)	HOXA9/MEIS1 - AML					• Mid-2021: Initiate NPM1 mt AML registrational Phase 3 clinical trial
Entospletinib or Lanraplenib (SYK Inhibitor)	FLT3 mt - AML					• 2021: Initiate FLT3 mt AML Phase 1/2 clinical trial in combination with a FLT3 inhibitor
KB-0742 (CDK9 Inhibitor)	MYC – Amplified Solid Tumors					• Q4 2021: Initial safety, PK and PD data from Phase 1/2 clinical trial dose escalation cohorts

TRN	Indication	Discovery	IND-Enabling Studies	Phase 1 Trial	Phase 2 Trial	Phase 3 Trial	Next Anticipated Milestone
MYB	**AML**						First IND filing in 2022 among these programs*
ARv7	**Prostate Cancer**						
IRF4	**Multiple Myeloma**						
ASCL1	**Small Cell / Neuroendocrine Cancers**						

*We may not be successful in identifying product candidates that can selectively modulate the specific oncogenic TRNs associated with such programs.

KRONOS·BIO

- Introduction
- **Targeting Oncogenic TRNs**
- Lead Programs
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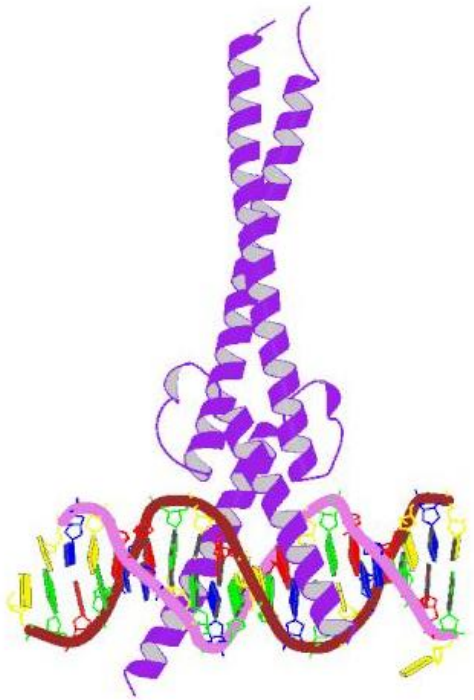
  - CDK9
- The Kronos Bio Opportunity

Transcription factors (TFs) are high-value but historically challenging targets

> 500 human TFs
> 50 known oncogenic role

MYC oncogenic TF

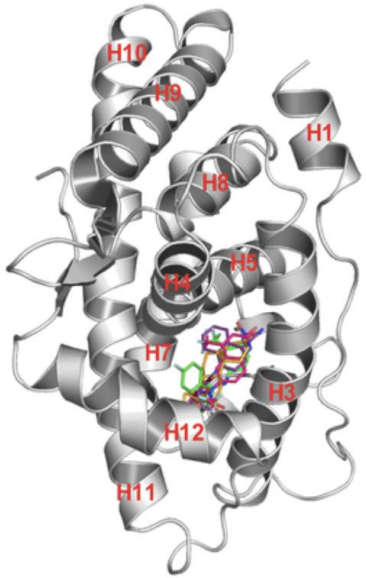


Existing TF therapies (< 10 TFs drugged)

Androgen and estrogen receptor

Xtandi™ (enzalutamide)

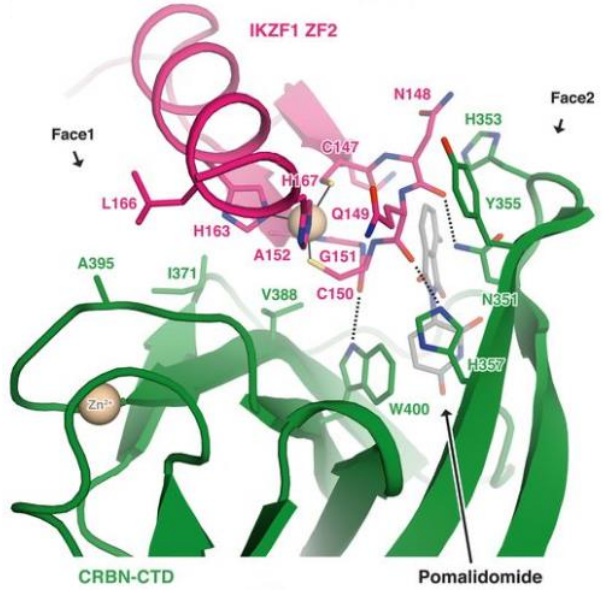
Faslodex™ (fulvestrant)



IMiDs degrade Ikaros/Aiolos TFs

Pomalyst™ (pomalidomide)

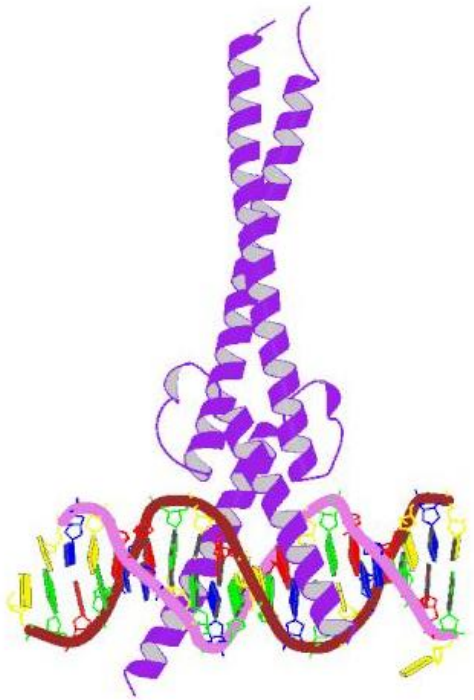
Revlimid™ (lenalidomide)



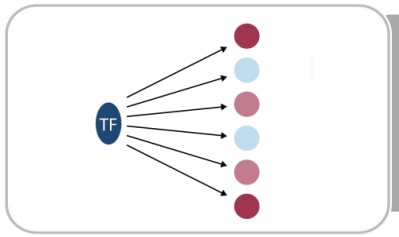
Transcription factors (TFs) are high-value but historically challenging targets

> 500 human TFs
> 50 known oncogenic role

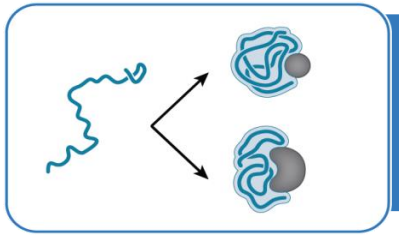
MYC oncogenic TF



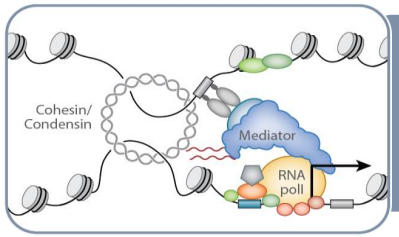
CHALLENGES



Context-dependent **activity**



Context-dependent **structure**

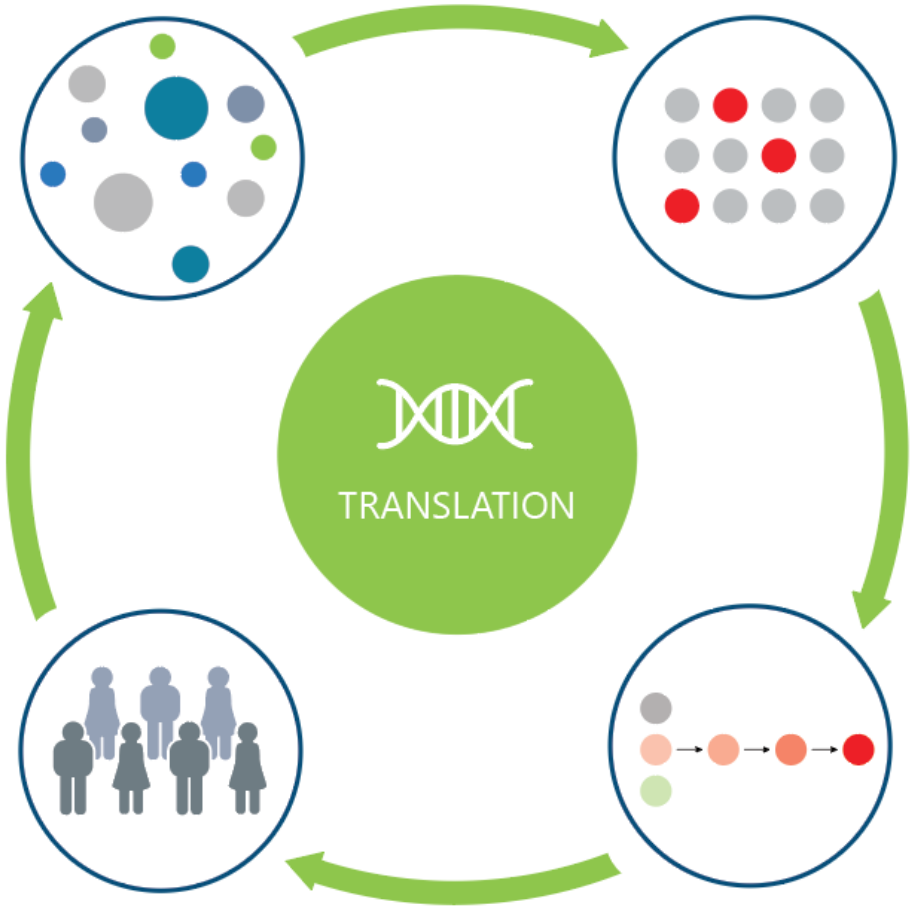


Context-dependent **complexes**

Proprietary product engine to systematically target dysregulated transcription factors and associated TRNs

MAP

Identify gene expression signature of selective TRN modulation



SCREEN

Conduct high throughput SMM screens in tumor cell lysates to identify selective TRN modulators

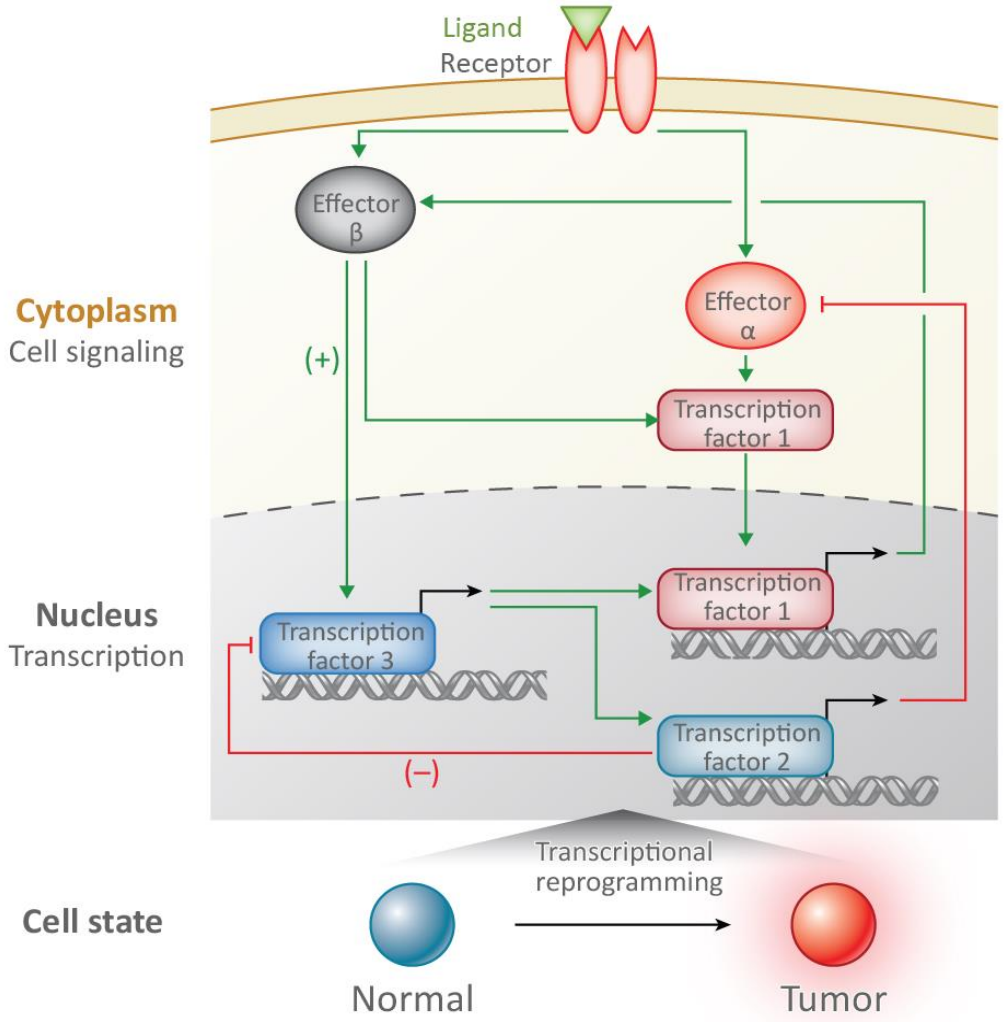
VALIDATE

Hypothesis driven clinical trials to deliver proof of concept early in the development process

OPTIMIZE

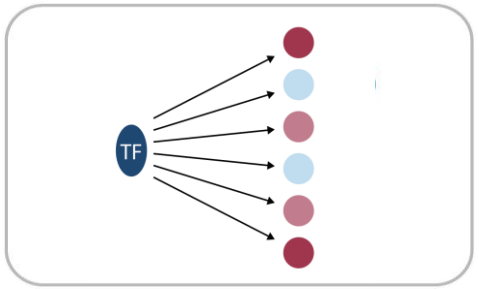
Refine pharmacological properties to yield attractive product development candidates

Map gene expression signatures and critical nodes in oncogenic TRNs



- Dynamic
- Interdependent
- Bi-directional

ACTIVITY



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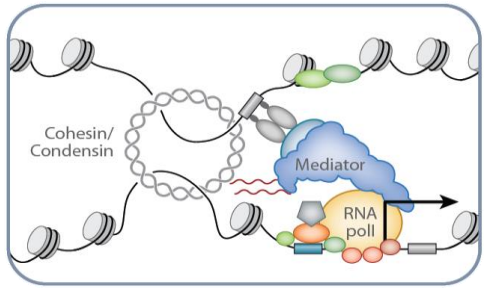
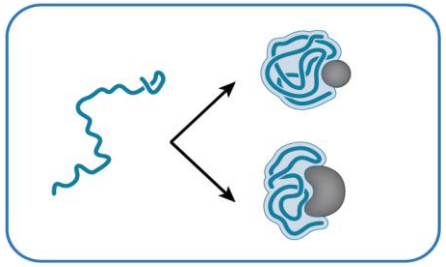


Screen using small molecule microarray (SMM) platform

STRUCTURE



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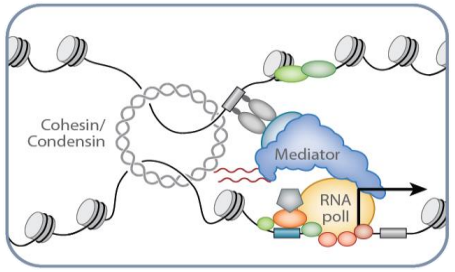
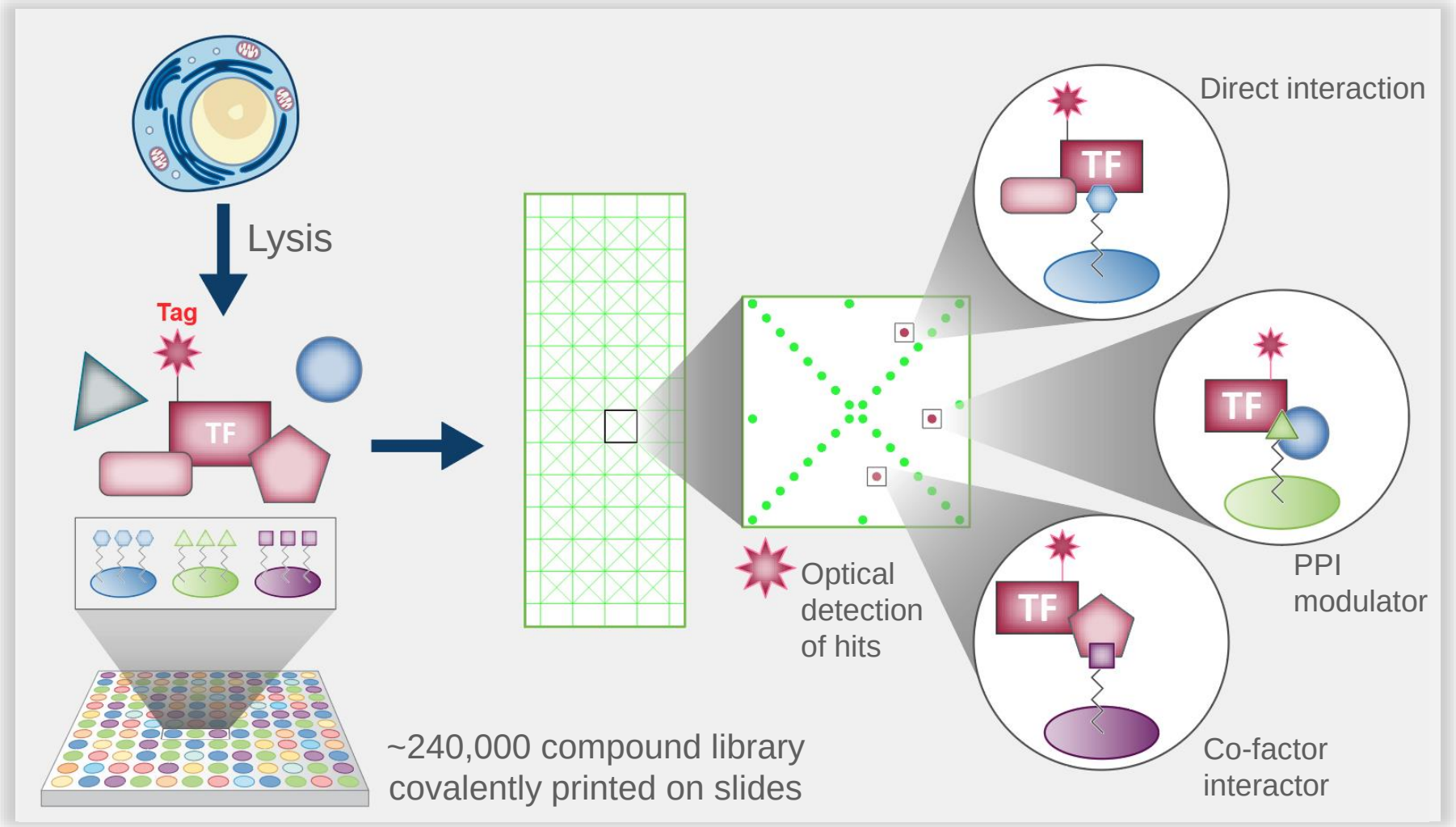
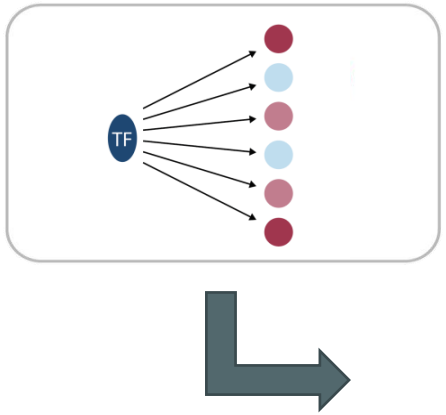


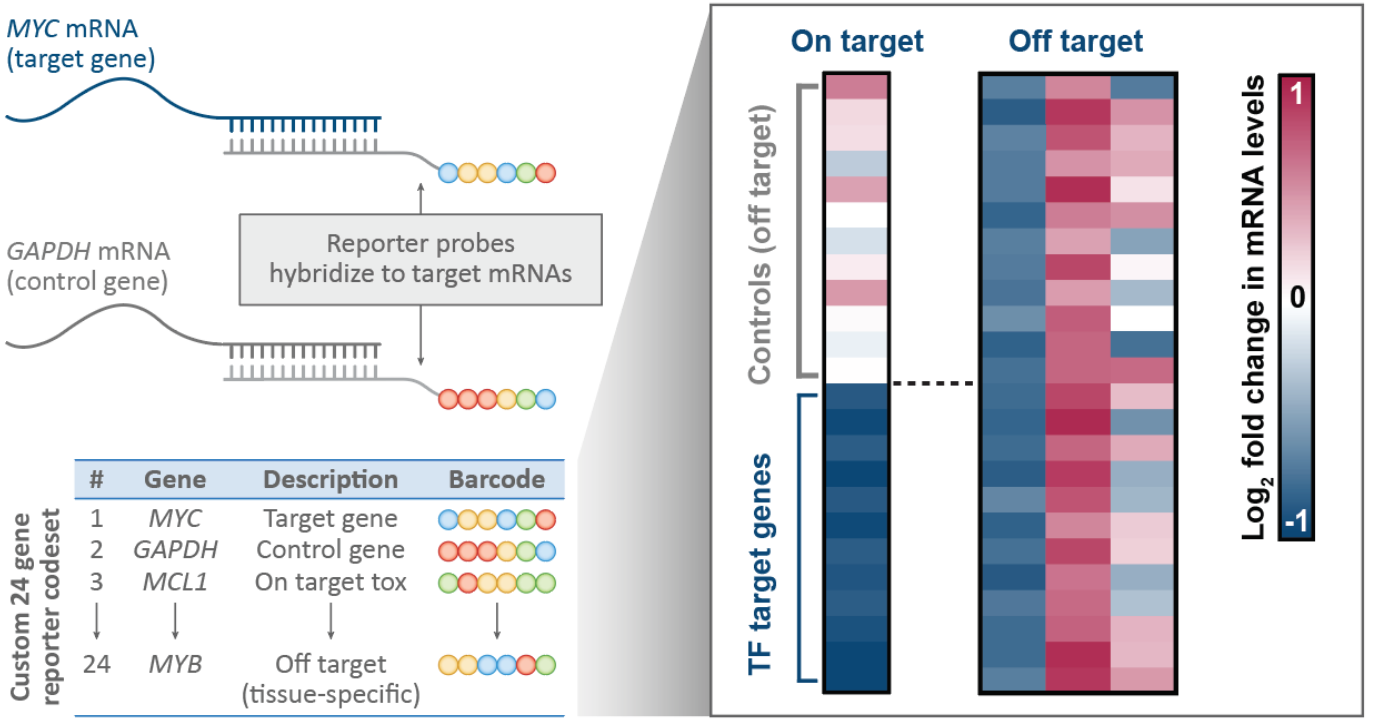


Screen

Prioritize hits based on gene expression signature

TF ACTIVITY SIGNATURE





- Evaluate **context-dependent** transcriptomic effects in relevant cancer lines
- Identify hits that **selectively** perturb the oncogenic TRN
- Drive hit-to-lead and lead optimization of **transcription factor modulators**

Optimize product candidates and aim to validate through hypothesis driven clinical trials

OPTIMIZE

- Identify relationships between molecular structure and target engagement
- Tailor structure-activity relationship studies to optimize to a specific transcriptional signature
- Refine pharmacological properties to match desired clinical product profile

VALIDATE

- Identify predictive biomarkers for drug response to enable precision medicine strategy
- Characterize PK/PD relationship to define optimal dose and schedule in patients
- Potentially rapidly achieve clinical proof of concept to inform registrational trial design

Current discovery programs and future opportunities

Hematologic Malignancies	• HOXA9/MEIS1 • MYB • IRF4
Small cell / neuroendocrine	• ASCL1 • Novel E3 (overexpressed in SCLC)
Prostate cancer	• ARv7 • Novel E3 (drives AR/SRC3 overexpression)
MYC-driven cancers	• MYC/MAX • Novel E3 (drives MYC overexpression)

Future opportunities

- KRAS mutant
- β-catenin
- p53 deficient tumors
- HPV-driven cancers
- FOXP3 (I/O, Immunology)

KRONOS·BIO

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  - **SYK**
  - CDK9
- The Kronos Bio Opportunity

SYK inhibitor program: Entospletinib

Entospletinib
SYK Inhibitor
HOXA9/MEIS1 AML

Selective inhibitor targeting Spleen Tyrosine Kinase (SYK) with ~7 years of clinical data in over 1,300 human subjects

SYK has been implicated as a target in biomarker-defined subsets of AML patients

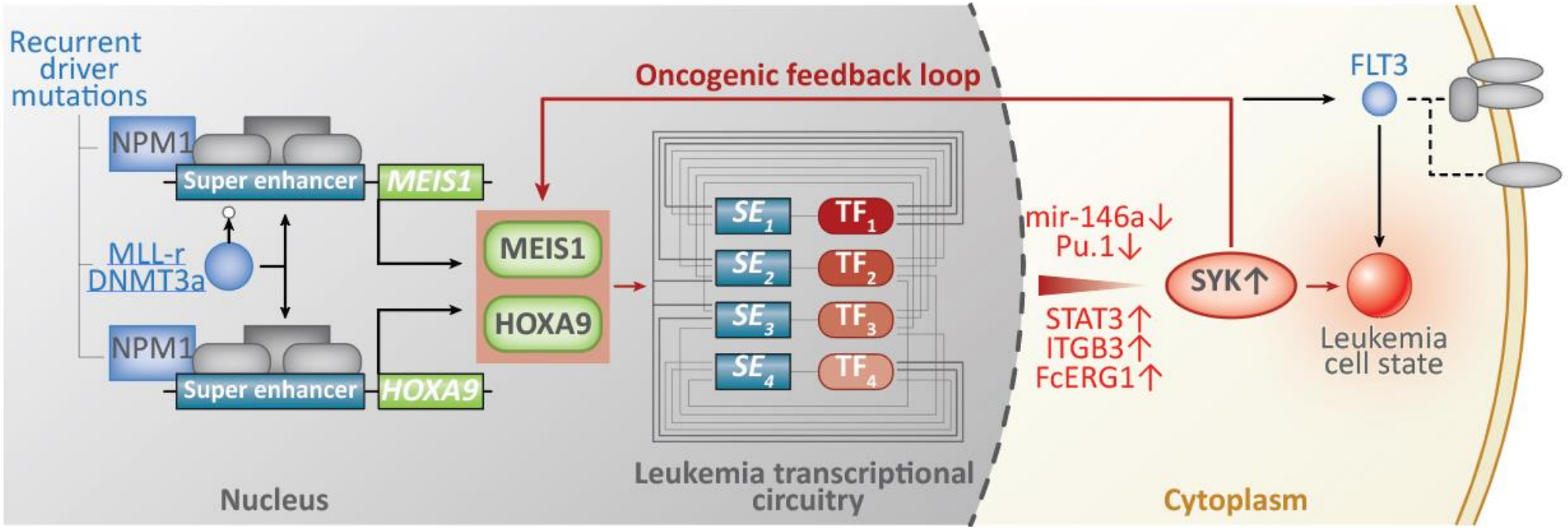
Clinical trials show encouraging activity in patients with HOXA9/MEIS1 high AML (associated with NPM1 mutation and MLL rearrangement)

Registrational Phase 3 trial to support potential accelerated approval in patients newly diagnosed with NPM1-mutated AML planned to begin in mid-2021; pivotal data expected in H2 2023

Additional opportunities in elderly / unfit AML patients and FLT3 mutated AML

SYK is a critical dependency in HOXA9/MEIS1-high AML

Elevated HOX/MEIS results in increased quantity and activity of SYK as part of an oncogenic TRN



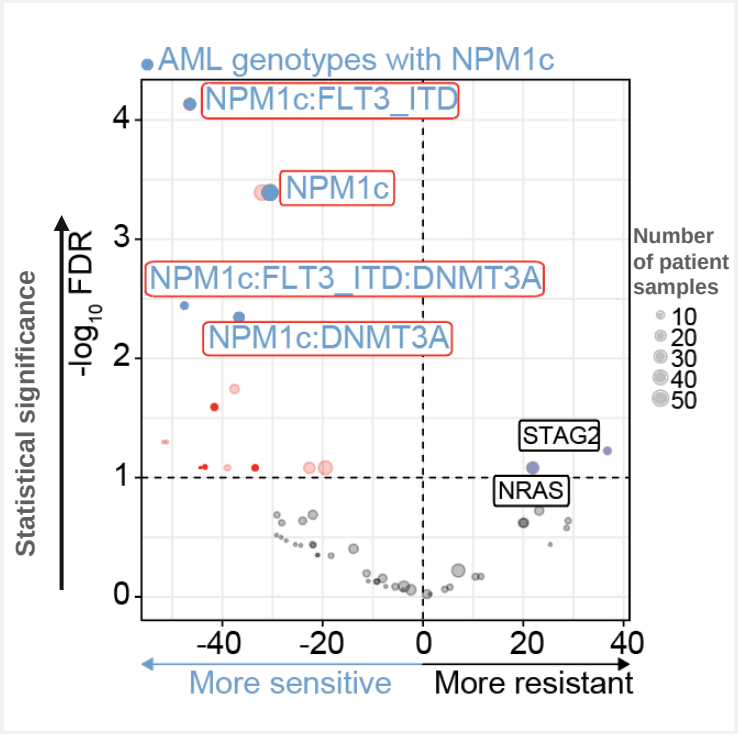
Adapted from Mohr et al. 2017. Cancer Cell

- SYK stabilizes the HOX/MEIS TRN via a positive feedback loop
- SYK signaling contributes to leukemogenesis, in part, through FLT3

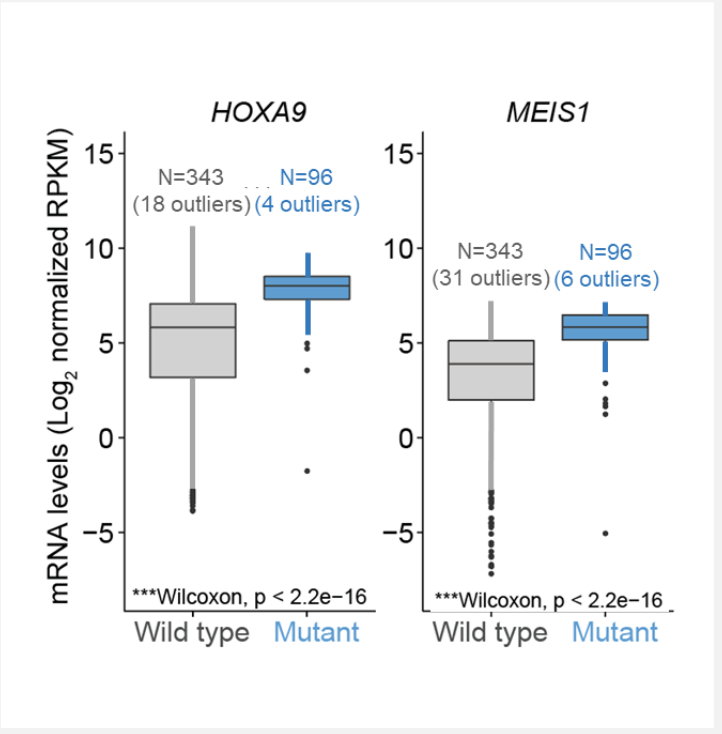
NPM1 mutant AML samples: Correlation with high H/M and sensitivity to Entospletinib

Leukemia and Lymphoma Society's BEAT AML program: bone marrow from 562 AML patients

NPM1 MT AML SAMPLES ARE MORE SENSITIVE TO ENTO



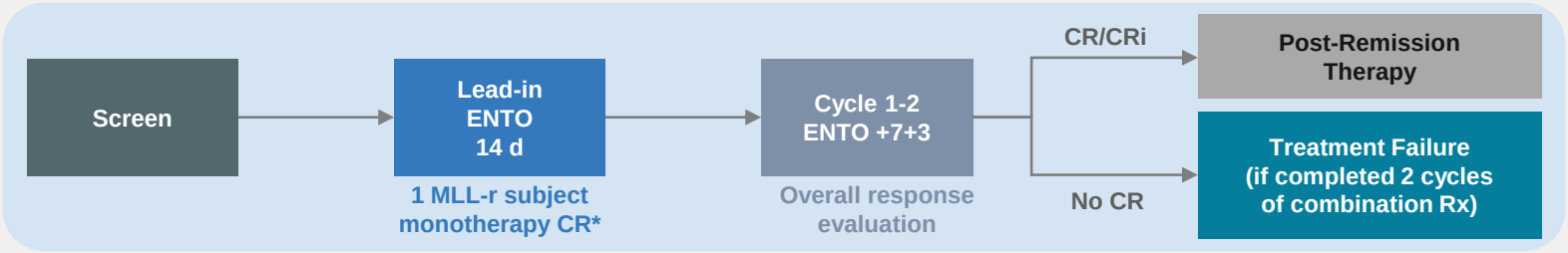
NPM1 MUTATION PREDICTS HIGH H/M

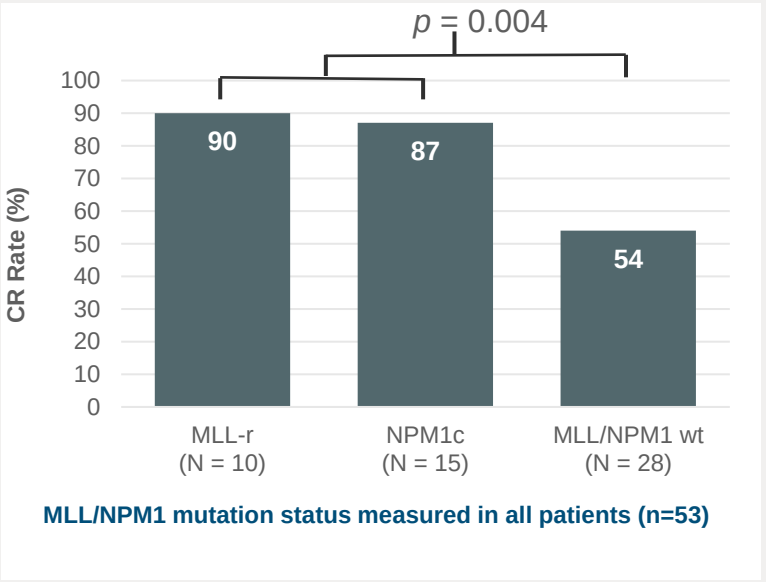


Internal analysis of Beat AML gene expression dataset *(n=672; Tyner et al. 2018. Nature)*

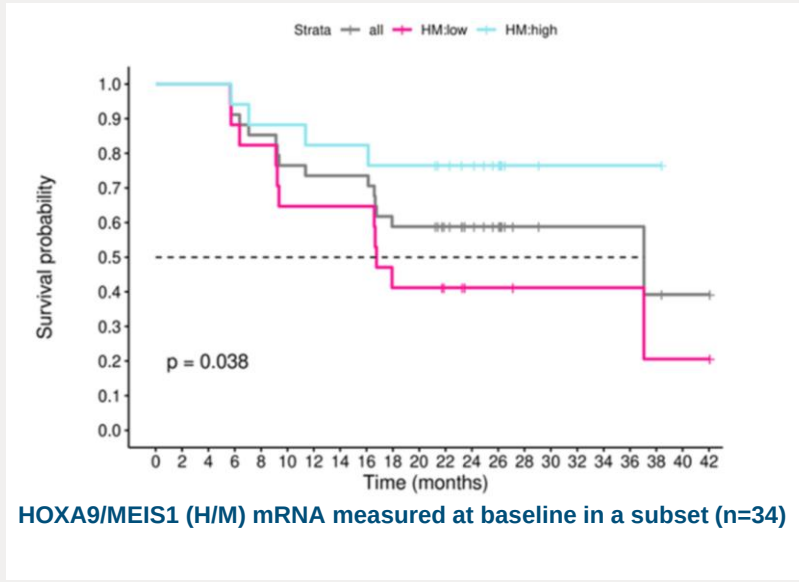
ENTO Phase 1b/2 clinical trial data shows activity in defined AML subsets

PHASE 1b/2 | PHASE 3





MLL/NPM1 mutation status measured in all patients (n=53)



HOXA9/MEIS1 (H/M) mRNA measured at baseline in a subset (n=34)

- 53 newly diagnosed AML patients
- 14-day ENTO monotherapy followed by combination induction
- 1 of 10 MLL-r patients had a CR with ENTO monotherapy

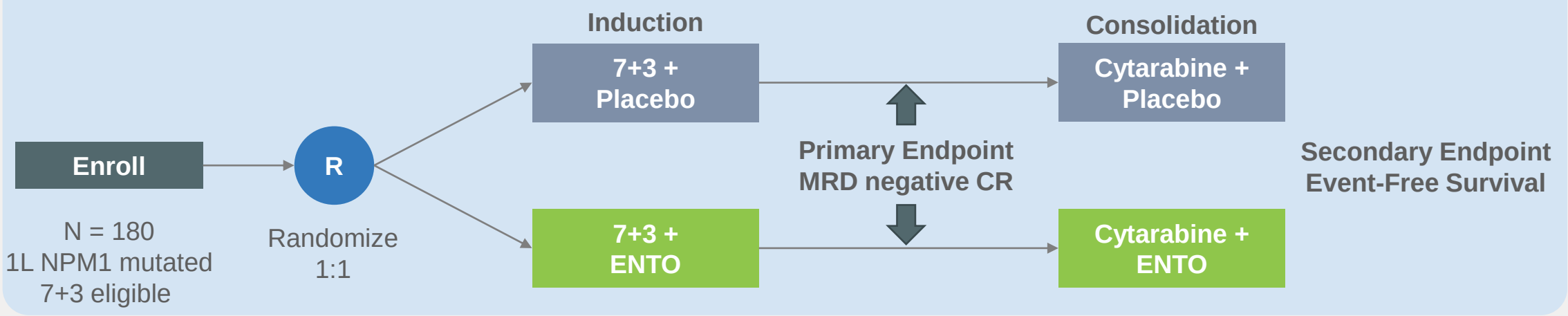
Phase 1b/2 data are consistent with the dependency between SYK and HOX/MEIS high AML subsets

Walker et al. 2018. Clin Cancer Res PMID: 32820015
Online ahead of print

Planned Phase 3 trial in NPM1-mutated AML leverages MRD endpoint

Trial follows positive End-of-Phase 2 meeting with FDA

PHASE 1b/2 | PHASE 3



- Patient enrollment based on existing clinical assays for NPM1 mutation
- Validate one assay to meet FDA label requirements for CDx in parallel with trial conduct
- Primary endpoint of MRD negative CR after two cycles of induction

Trial initiation planned for mid-2021, with pivotal data expected in H2 2023

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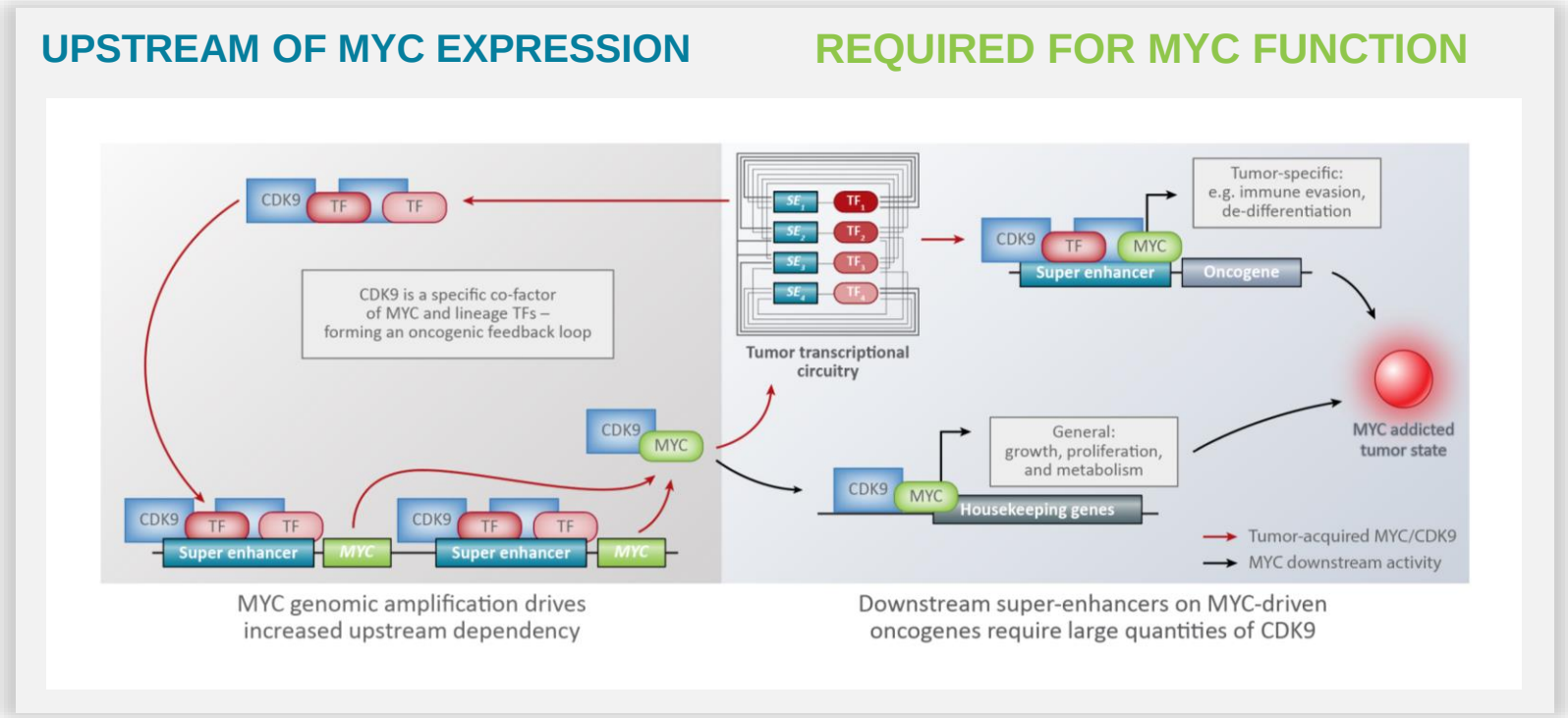
  - SYK
  - **CDK9**
- The Kronos Bio Opportunity

CDK9 Inhibitor Program: KB-0742

KB-0742
CDK9 Inhibitor
Solid Tumors

- **CDK9 is a global regulator of transcription and a critical node in multiple oncogenic TRNs**
- **KB-0742 originated from proprietary SMM screen**
- **Differentiated selectivity profile, oral bioavailability and other attractive pharmacologic properties**
- **Demonstrated dependence on CDK9 in MYC amplified tumors**
- **Dose escalation stage of Phase 1/2 clinical trial underway**

CDK9 is a global transcription elongation factor and an essential co-factor for the MYC TRN

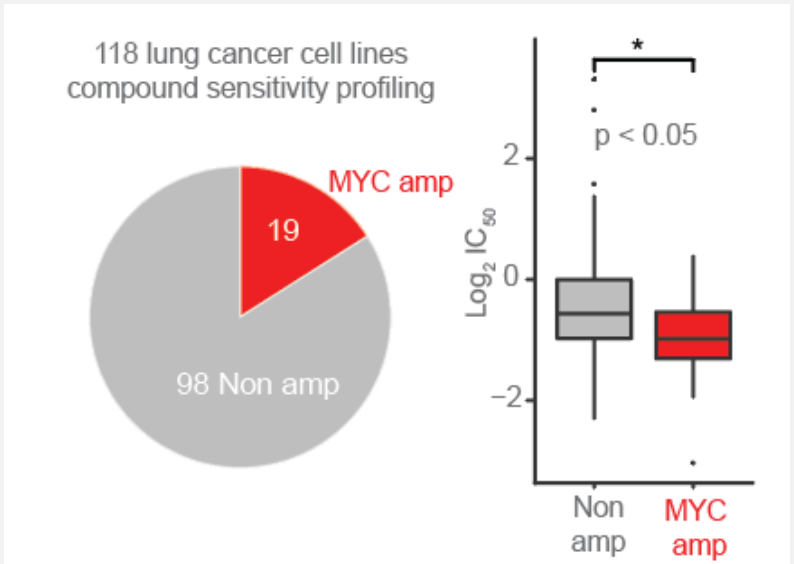


- CDK9 phosphorylates RNA pol II, allowing transcription to proceed
- MYC requires CDK9 to maintain its own mRNA expression
- MYC requires CDK9 to drive expression of its target genes
- Tumors that are addicted to MYC have heightened sensitivity to CDK9 inhibition
- Intermittent, partial inhibition of CDK9 can be active without causing unacceptable toxicity

CDK9 is an attractive target in transcriptionally addicted cancers, and specifically MYC-amplified solid tumors, due to its essential role in transcriptional elongation

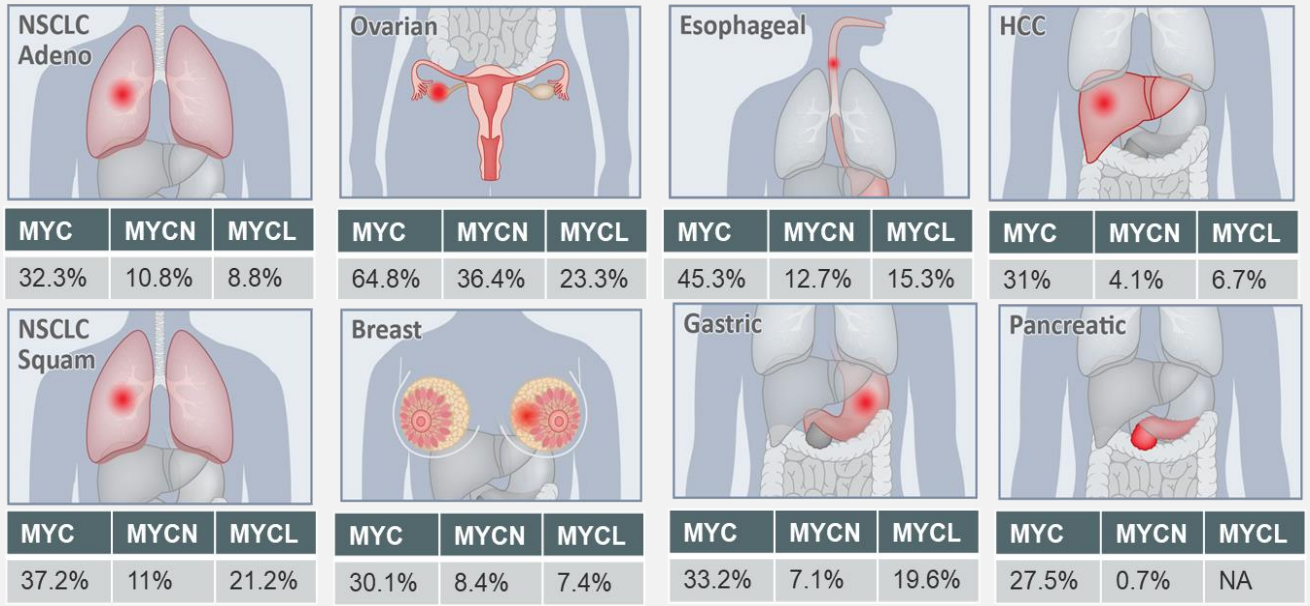
MYC amplification defines a subset of tumors with increased sensitivity to KB-0742

MYC AMPLIFIED NSCLC IS MORE SENSITIVE TO KB-0742



BROAD INSTITUTE

MYC AMPLIFICATION IS FOUND IN ~30% OF COMMON CANCER TYPES



NSCLC Adeno		
MYC	MYCN	MYCL
32.3%	10.8%	8.8%

Ovarian		
MYC	MYCN	MYCL
64.8%	36.4%	23.3%

Esophageal		
MYC	MYCN	MYCL
45.3%	12.7%	15.3%

HCC		
MYC	MYCN	MYCL
31%	4.1%	6.7%

NSCLC Squam		
MYC	MYCN	MYCL
37.2%	11%	21.2%

Breast		
MYC	MYCN	MYCL
30.1%	8.4%	7.4%

Gastric		
MYC	MYCN	MYCL
33.2%	7.1%	19.6%

Pancreatic		
MYC	MYCN	MYCL
27.5%	0.7%	NA

Percentage of tumors in the TCGA dataset with copy number gains of MYC, MYCN or MYCL (Schaub et al, 2018. Cell Systems 6: 282 – 300)

We believe focusing on tumors with heightened sensitivity to CDK9 inhibition increases the likelihood of achieving anti-tumor activity without unacceptable toxicity

Pharmacologic properties of KB-0742 are conducive to defining therapeutic index in patients

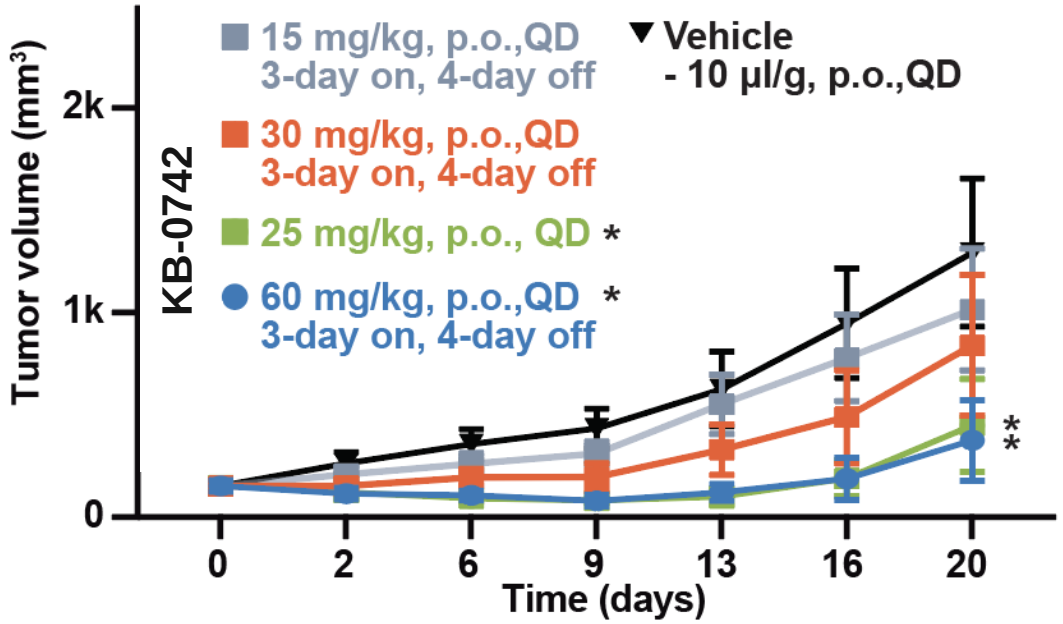
- Inhibition of cell cycle CDKs leads to off-target toxicity
- KB-0742 has demonstrated improved selectivity for CDK9 over cell cycle CDKs
- Optimal schedule of dosing is key to managing toxicity while maintaining efficacy
- Oral bioavailability, potential long half-life provide flexibility to explore different intermittent dosing schedules

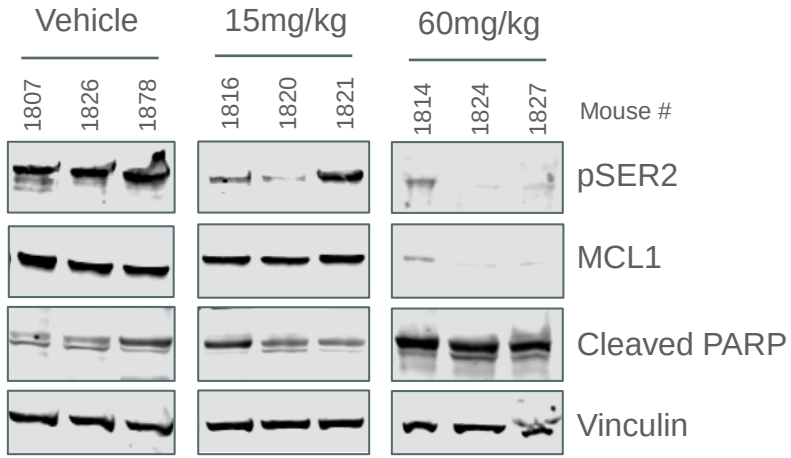
Compound		KB-0742
Potency (biochemical IC_{50})	**CDK9**	**6 nM**
Fold Selectivity CDK9 vs. other CDK family members	CDK8	>1000x
	CDK7	252x
	CDK6	658x
	CDK5	303x
	CDK4	522x
	CDK3	237x
	CDK2	66x
	CDK1	497x
Route of administration		**Oral**

Transcriptional CDK

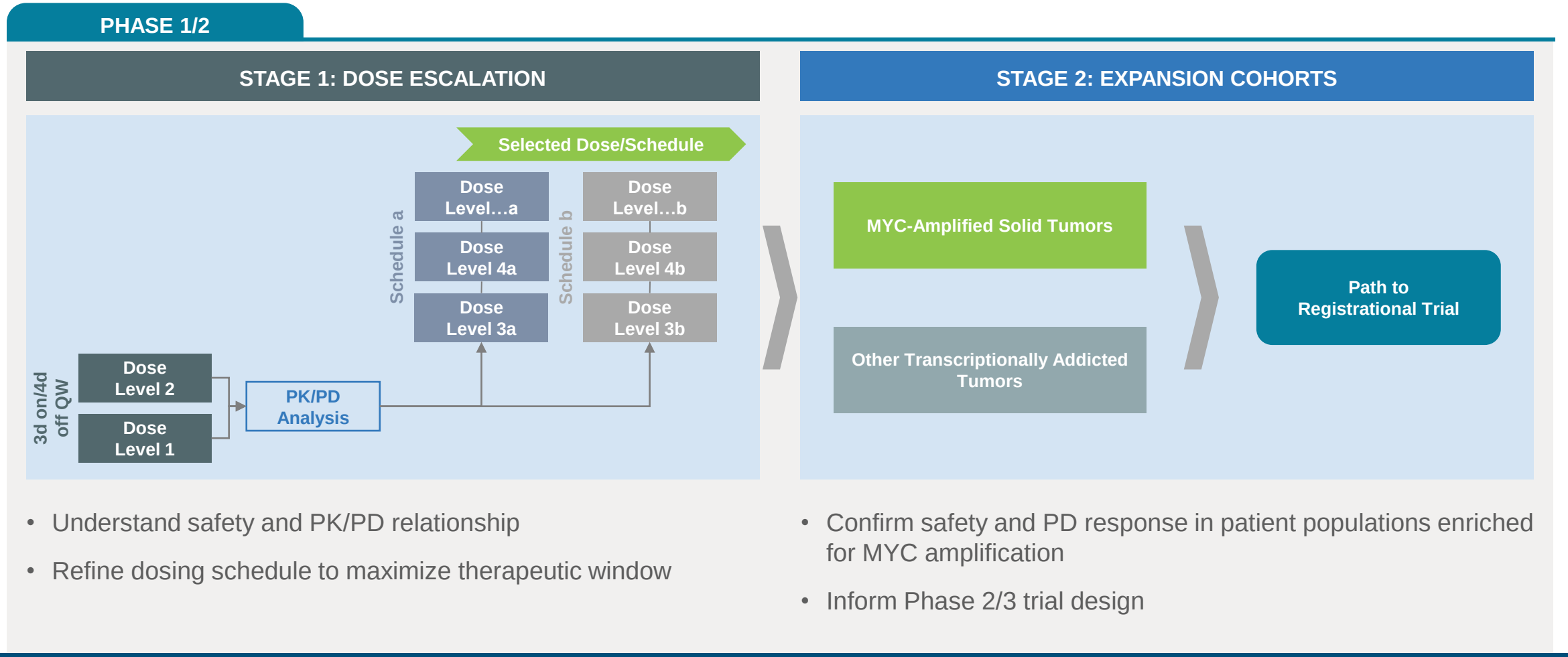
Cell cycle CDK

Intermittent dosing is as efficacious as continuous dosing in a MYC-driven AML xenograft model





KB-0742 Phase 1/2 trial design



- Understand safety and PK/PD relationship
- Refine dosing schedule to maximize therapeutic window

- Confirm safety and PD response in patient populations enriched for MYC amplification
- Inform Phase 2/3 trial design

Initial safety, PK and PD data from dose escalation cohorts expected in Q4 2021

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- **The Kronos Bio Opportunity**

Strong financial profile

~$441M

cash, cash equivalents and investments

(unaudited, as of March 31, 2021)

Cash runway at least into

2024

~56M

shares outstanding

Company financed through multiple potential value catalysts

Q1 2021

Initiate Phase 1/2 trial for KB-0742

Q1 2021

FDA End-of-Phase 2 Meeting for ENTO

Mid-2021

Initiate NPM1+ AML Phase 3 trial for ENTO

Q4 2021

Initial PK/PD and safety data for KB-0742

Q4 2021

Initiate FLT3+ AML PoC trial for ENTO or LANRA

2022

Efficacy signal for KB-0742

H2 2023

Pivotal data NPM1+ AML Phase 3 trial for ENTO

~~IM~~POSSIBLE.
~~UN~~DRUGGABLE.
~~UN~~ACHIEVABLE.

Kronos Bio Investment Highlights

- Pioneering a new approach to target a potentially large market opportunity – dysregulated transcription factors and their associated TRNs
- SYK inhibitor program including registrational Phase 3 trial to begin in mid-2021
- Highly differentiated CDK9 program targeting MYC-amplified tumors
- Proprietary product engine to drive accelerated expansion of product candidates focused on high-value targets
- Highly experienced management team and support from leading investors

Dedicated to Transforming Patient Outcomes in Cancer by Targeting Dysregulated Transcription

KRONOS·BIO

Efforts guided by scientific advisory board comprised of leading KOLs

Owen Witte, M.D.
Scientific Advisory Board Chairman
UCLA

Myles Brown, M.D.
Dana-Farber Cancer Institute
Brigham and Women's Hospital
Harvard Medical School

David Chang, M.D., Ph.D.
President, Chief Executive Officer and Co-Founder of Allogene Therapeutics

Robert Eisenman, Ph.D.
Fred Hutchinson Cancer Research Center
University of Washington School of Medicine

Angela Koehler, Ph.D.
Scientific Founder
Associate Professor, Koch Institute for Integrative Cancer Research (MIT)

Roger D. Kornberg, Ph.D.
Winzer Professor in Medicine, Stanford University School of Medicine
Nobel Laureate 2006