KRONOS·BIO

Corporate Overview

June 2022

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. These risks and uncertainties are described under the "Risk Factor" heading of the Company's Annual Report on Form 10-K dated December 31, 2021 and filed with the U.S. Securities and Exchange Commission on February 24, 2022 and our Form 10-Q dated March 31, 2022 and filed with the SEC on May 4, 2022. 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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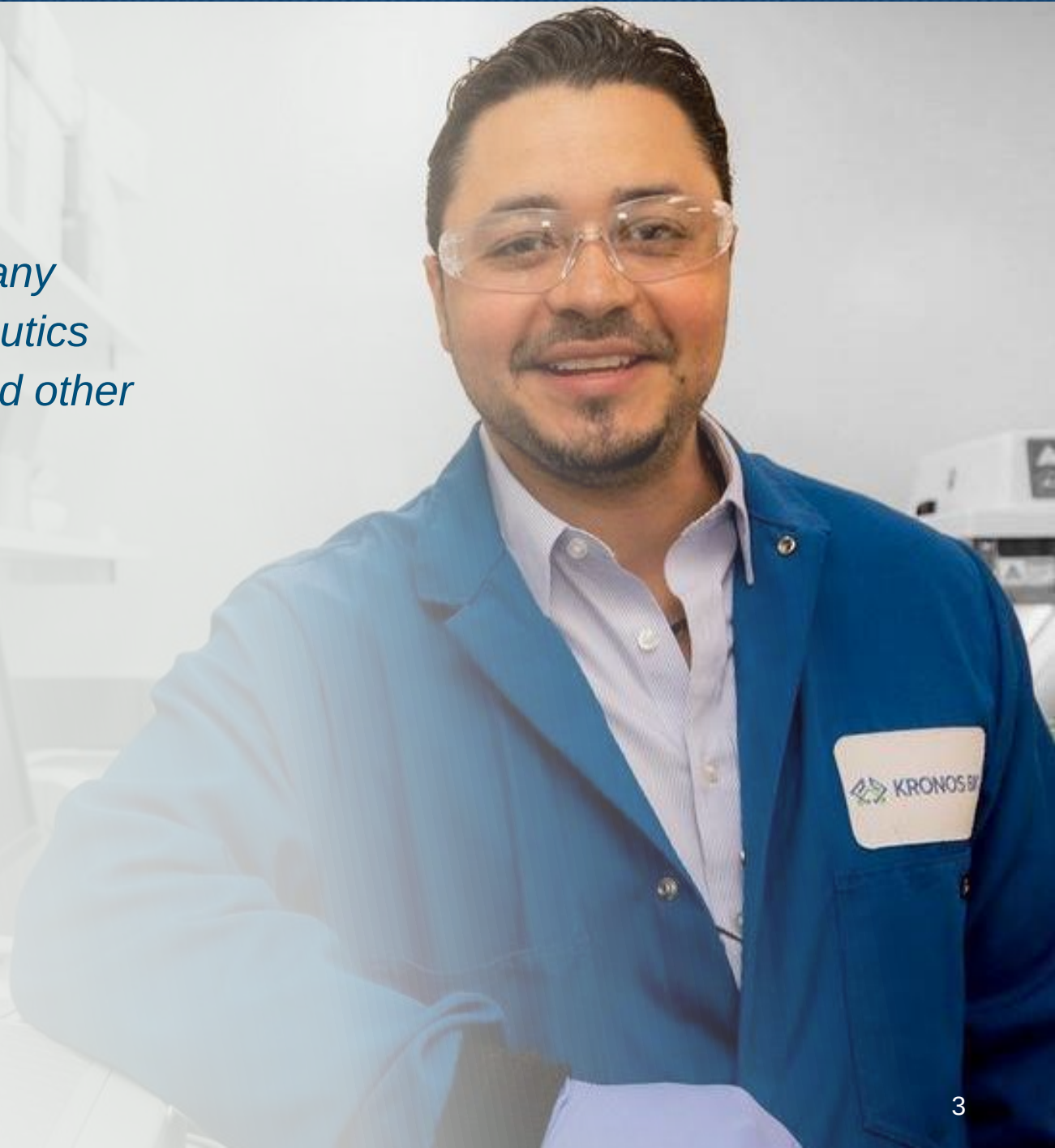
Who We Are

We are a clinical-stage biopharmaceutical company dedicated to discovering and developing therapeutics that target deregulated transcription in cancer and other serious diseases.

We analyze transcription regulatory networks in their entirety.

We are headquartered in San Mateo, Calif., with a research and discovery facility in Cambridge, Mass.



Experienced Leadership With Strong Track Record Across the Industry

LEADERSHIP TEAM

Norbert Bischofberger, Ph.D.
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

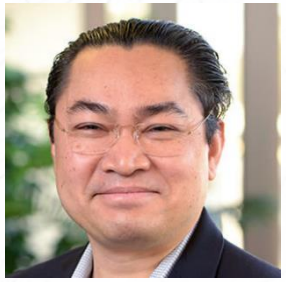
Yasir Al-Wakeel, BM BCh
Chief Financial Officer and
Head of Corporate Development

Christopher Dinsmore, Ph.D.
Chief Scientific Officer

Marni Kottle
Senior Vice President
Corp Communications &
Investor Relations

Charles Lin, Ph.D.
Senior Vice President
Biology

Elizbeth Olek, DO, MPH
Senior Vice President
Clinical Development



Pasit Phiasivongsa, Ph.D.
Senior Vice President
Pharmaceutical Development &
Manufacturing

The Kronos Bio Story

3 Clinical Compounds

- Kronos Bio's lead compound, **entospletinib**, is in a registrational Phase 3 trial (AGILITY) in combination with standard of care chemotherapy as frontline treatment for patients with NPM1-mutated AML
- Sites open for Phase 1b/2 trial of **lanraplenib** in patients with r/r FLT3-mutated AML in combination with gilteritinib
- **KB-0742**, our CDK9 inhibitor, is in ongoing Phase 1/2 study. Initial positive data reported in Q4 2021; additional Phase 1 data expected in Q4 2022

Product Engine

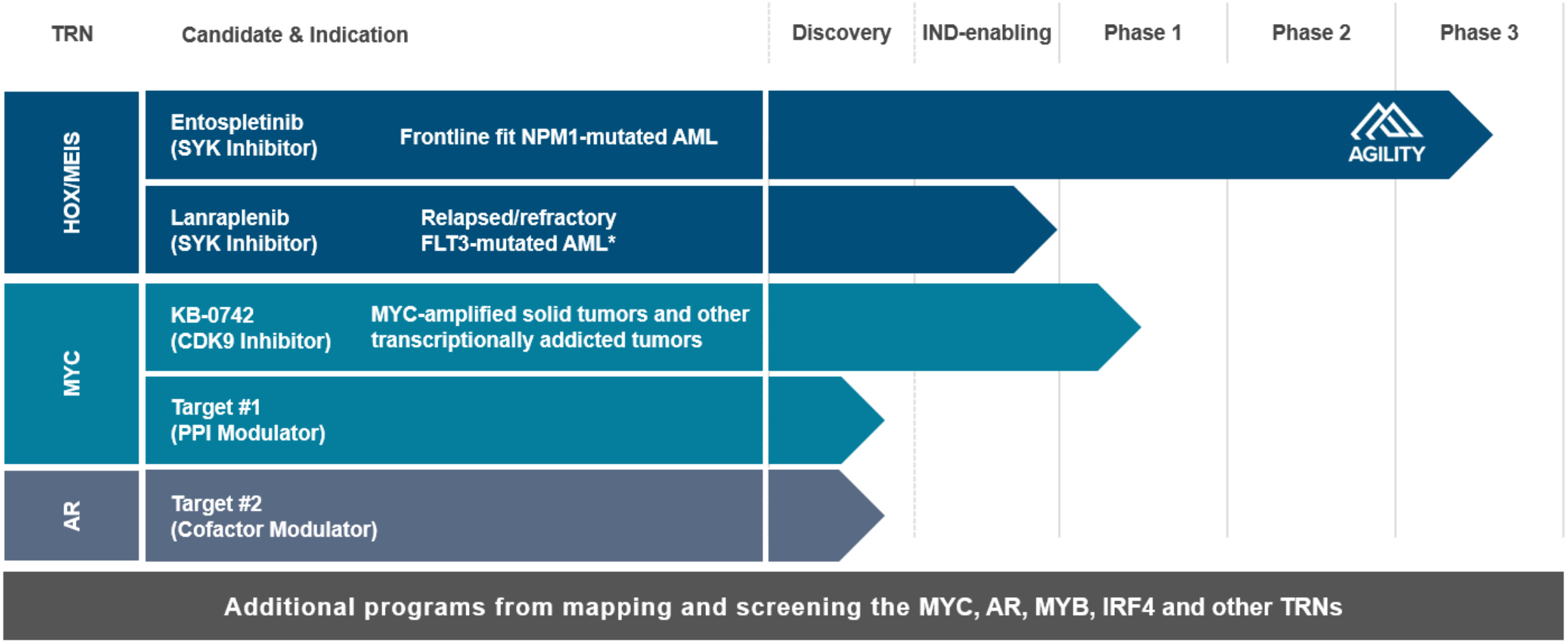
- Capability to map and target transcription regulatory networks (TRNs) in a differentiated manner to enable discovery and translation
- SMM platform to enable screening of TRN in transcriptionally dysregulated environment
- Two new discovery programs announced in November 2021

Human & Financial Capital

- Experienced management team has commercialized more than 25 therapies in oncology and other diseases
- Approx. $315.4 cash, cash equivalents and investments (unaudited, as of March 31, 2022)
- Cash runway into H2 2024

AML: Acute myeloid leukemia. CDK9: Cyclin Dependent Kinase 9. SMM: Small molecule microarray. SYK: Spleen tyrosine kinase.

Advancing Clinical-Stage Programs Across Multiple Oncogenic TRNs



*IND cleared

KRONOS·BIO

01 Introduction

02 **Dysregulated Transcription: A Hallmark of Cancer**

03 Our Clinical Programs

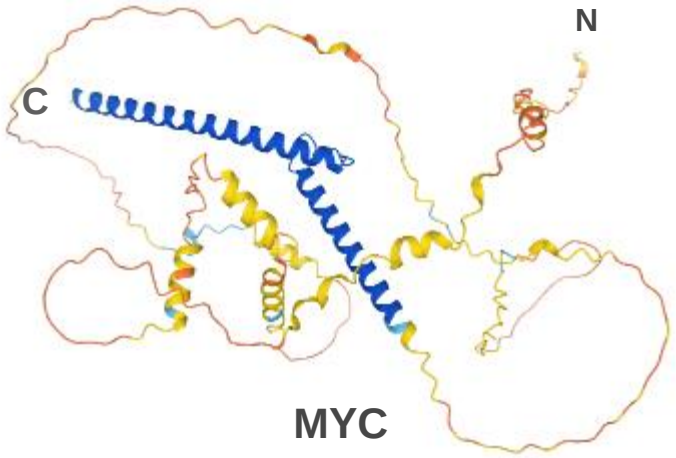
- KB-0742 (CDK9 inhibitor)
- Entospletinib/lanraplenib (SYK inhibitor portfolio)

04 The Kronos Bio Opportunity

- Looking Ahead to Commercialization
- Milestones and Financials

Transcription Factors Have Eluded Traditional Drug Discovery

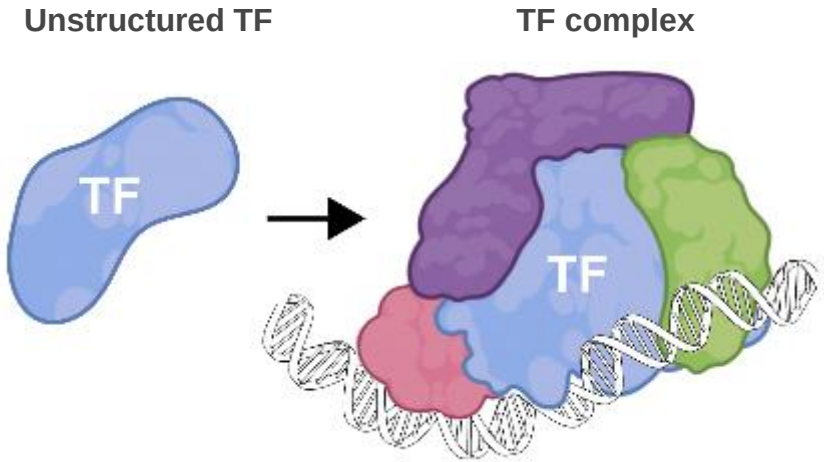
Most TFs are intrinsically disordered in isolation



Less structured — More structured

Alphafold - P01108

In the nucleus, TFs adopt a unique structure via cofactor interactions



TFs act by recruiting cofactors to the genome and regulating transcription

TFs and their cofactors become more druggable in their native complexes

Significant Potential:

Only 7 of the 100+ TFs Implicated in Driving Cancer Have Been Drugged

APPROVED*

- IKZF1/3
- NR (AR)
- ESR1 (ER)
- NR3C1 (GR)
- PPAR
- RARA
- HIF1/2

IN CLINICAL DEVELOPMENT

- IKZF2
- MYC
- NUT
- STAT3
- STAT5
- TP53
- YAP1/TEAD

UNDRUGGED

ASCL1	GLI1	NF1	PRRX1
BCL6	GLI2	NFKB	RUNX1
C19ORF11	HAND1/2	NOTCH1	RUNX2
CEBPa/b	IRF4	NOTCH2	SMADs
CRX	ID1	NOTCH3	SNAI2
E2F	ID2	NR2F2	SOX2
EWS	JUN	NRL	T
ETV1	MAF	OLIG2	TAL1
FLI	MAZ	PAX3	TAL2
FOS	MITF	PAX8	TCF3
FOXL2	MYB	PHOX2A	TCF4
FOXOs	MYCL	PHOX2B	TCF7L2
FOXP3	MYCN	POU2AF1	TWIST1
GATA2/3	NEUROD1	PRDM1	XBP1

*FDA NDA approval of compound drugging this TRN

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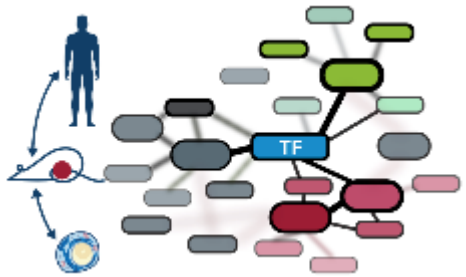
Our Product Engine Allows us to Target Previously Undruggable TRNs

Our Approach

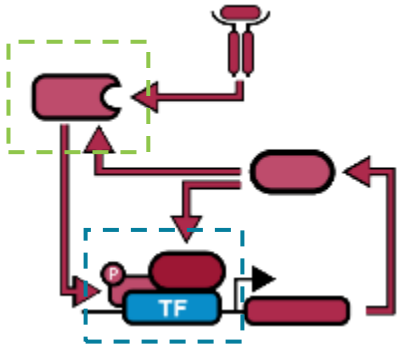
MAP TRN

Integrative networks shaped by real world evidence



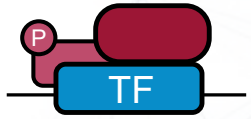
DEFINE DEPENDENCIES

Target and patient selection driven by causal networks



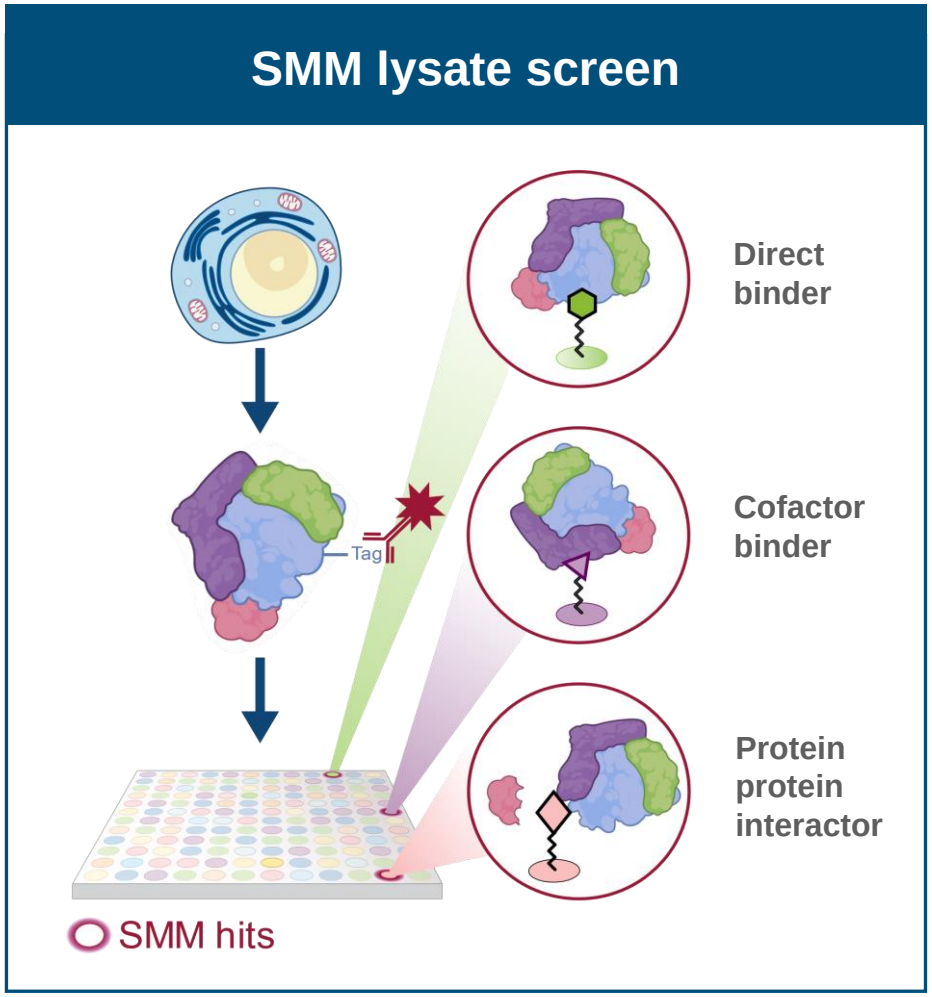
IDENTIFY MODULATORS

Small Molecule Microarray (SMM) e.g., KB-0742



Acquisition/in-licensing or directed internal discovery, e.g., SYK portfolio

Small Molecule Microarray Identifies Binders to TRN Constituents



- Continuous platform development has improved screening throughput, quality and turnaround time
- 240,000 compound diversity library with lead-like properties
- Drives discovery of multi-modality binders:
  - Direct TF binder
  - Cofactor binder
  - Protein protein interactor (PPI)
- Binders can be modulators or elaborated into bifunctional degraders

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- Entospletinib/lanraplenib (SYK inhibitor portfolio)

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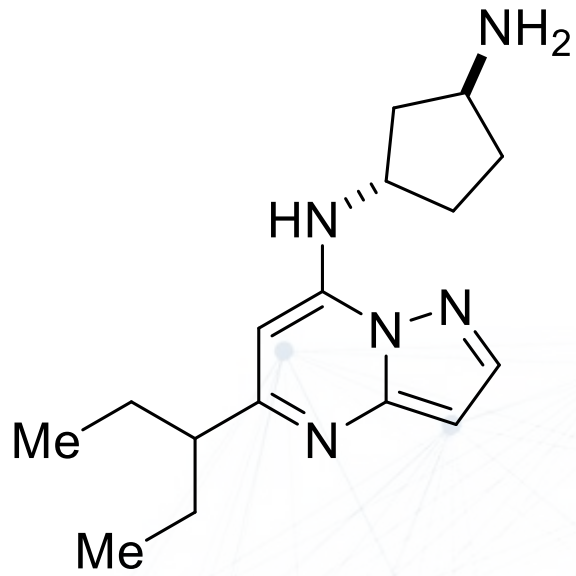
KB-0742:
Internally Discovered CDK9 Inhibitor in Phase 1/2 Study

- CDK9 is a global regulator of transcription and a critical node in multiple oncogenic TRNs; demonstrated dependence on CDK9 in MYC-amplified tumors
- KB-0742 originated from proprietary SMM screen
- Differentiated selectivity profile, oral bioavailability and other attractive pharmacologic properties
- Phase 1/2 trial ongoing, with positive interim data announced in Q4 2021 and additional clinical data anticipated in Q4 2022

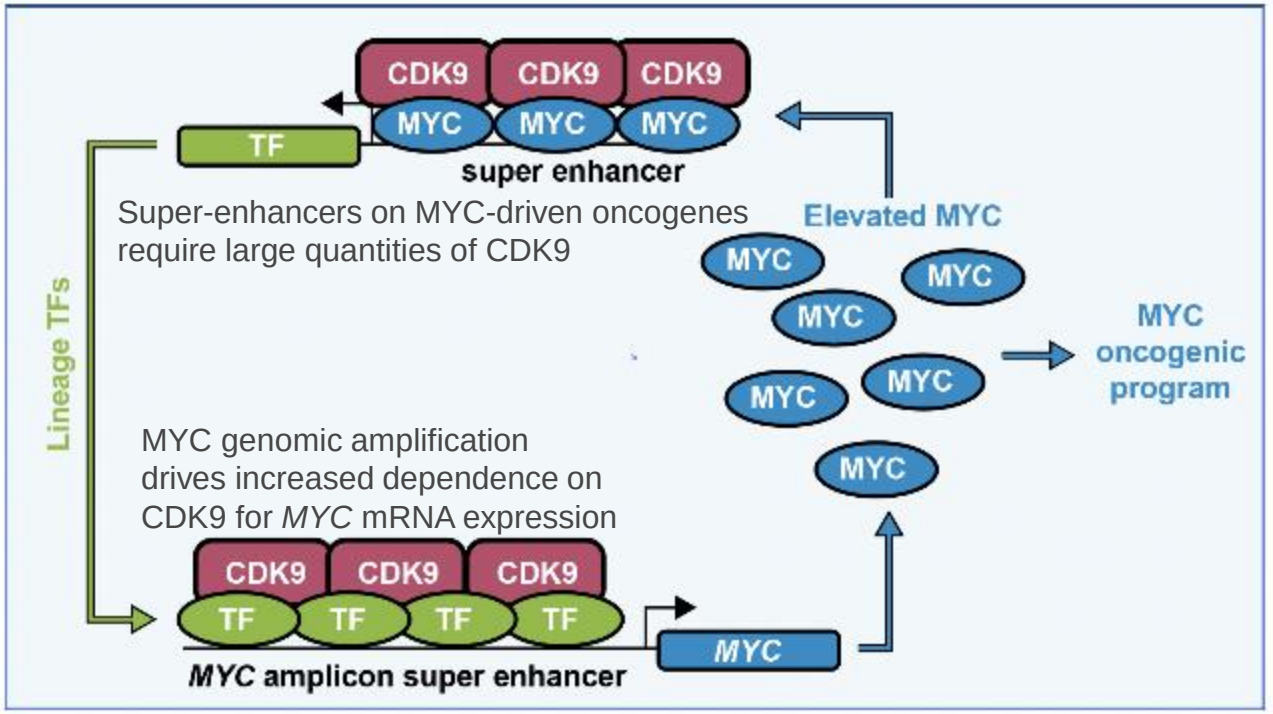


KB-0742

First internally discovered candidate enables differentiated CDK9 clinical approach

CDK9 is a Global Transcription Elongation Factor and Essential Co-Factor for the MYC TRN

CDK9 is required for MYC expression and MYC function



CDK9: Cyclin-dependent kinase 9. TF: Transcription factor. TRN: Transcription regulatory network.

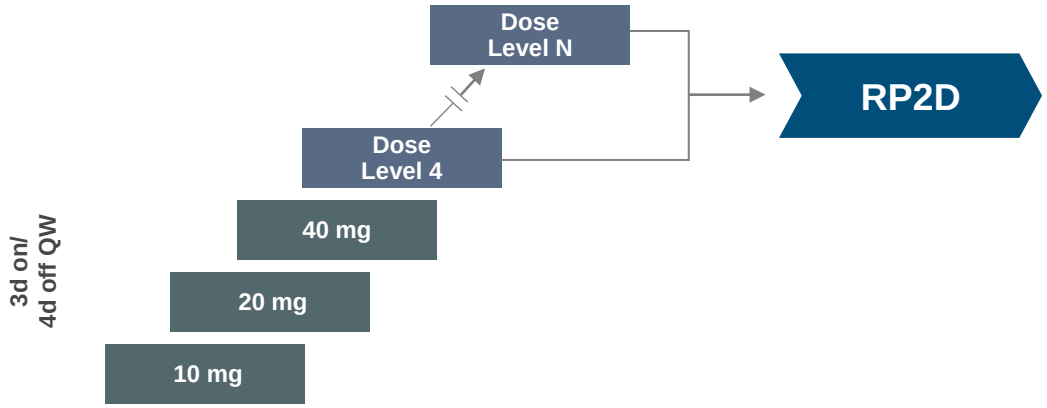
- CDK9 phosphorylates RNA pol II, allowing transcription to proceed driving mRNA expression of MYC itself and its target genes
- Tumors that are addicted to MYC have heightened sensitivity to CDK9 inhibition
- Intermittent, partial inhibition of CDK9 can be active without causing unacceptable toxicity

CDK9 is an attractive target in transcriptionally addicted cancers

Ongoing KB-0742 Phase 1/2 Trial Includes Two Stages

PHASE 1/2

STAGE 1: DOSE ESCALATION



- Relapsed/refractory solid tumor population **not selected for MYC amplification**
- Understand safety, PK and PD **in PBMC**
- Refine dosing schedule to maximize therapeutic window

STAGE 2: EXPANSION COHORTS

Cohort A: MYC-Dependent Solid Tumors

NSCLC, SCLC, TNBC, Ovarian
N=40

Cohort B: Other Transcriptionally Addicted Tumors

Sarcoma, Chordoma
N=30

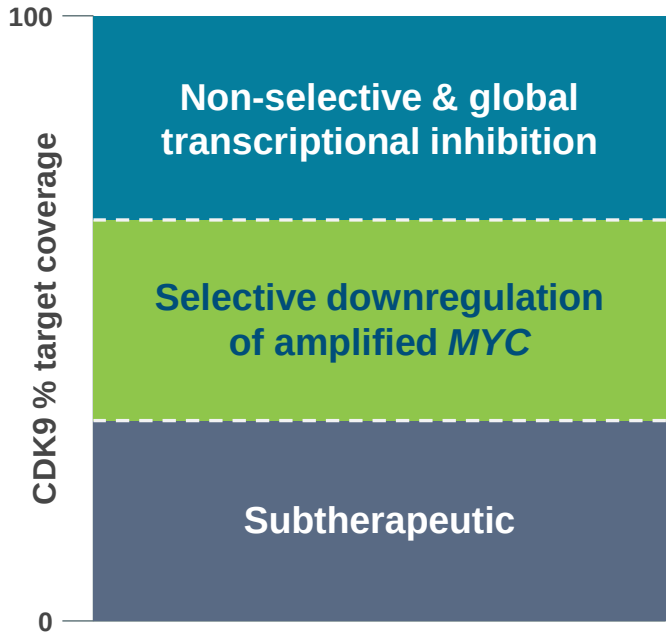
Path to Registrational Trial

- **Biomarker selected** patients most likely to benefit from CDK9 inhibition
- Confirm safety and PD **in tumor tissue**
- Anti-tumor activity in specific tumor types

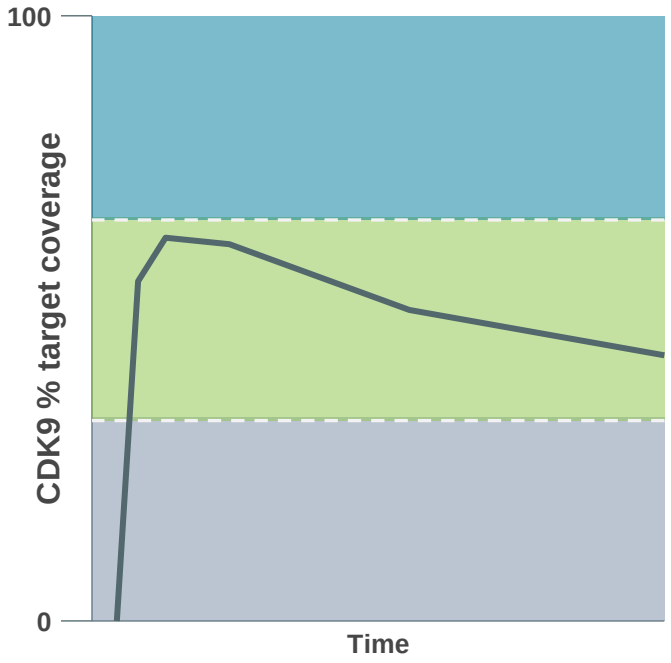
NSCLC: Non-small cell lung cancer. PD: Pharmacodynamics. PK: Pharmacokinetics. QW: Weekly. SCLC: Small cell lung cancer. TNBC: Triple-negative breast cancer. PBMC: Peripheral Blood Mononuclear Cells. RP2D: recommended Phase 2 dose

A Long Plasma Half-Life Provides a Differentiated Opportunity to Establish a Therapeutic Window for CDK9 Inhibition

CDK9 therapeutic index

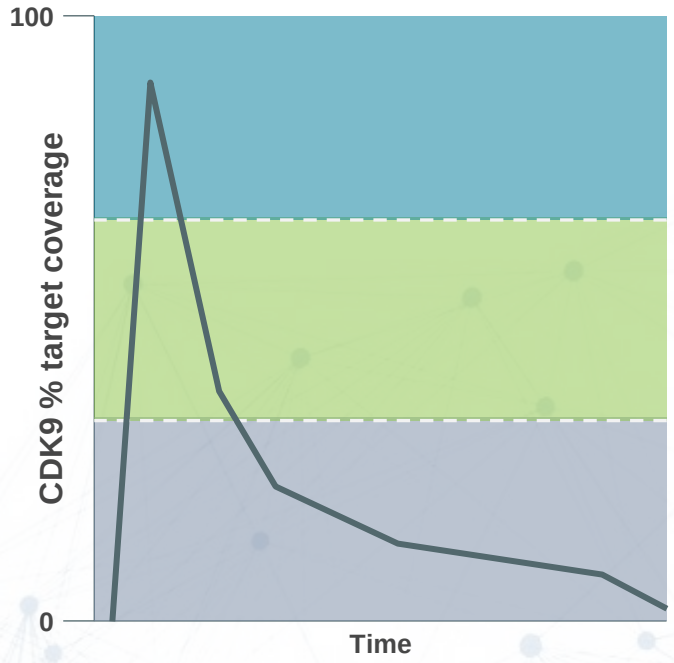


Long half-life CDK9 inhibitor



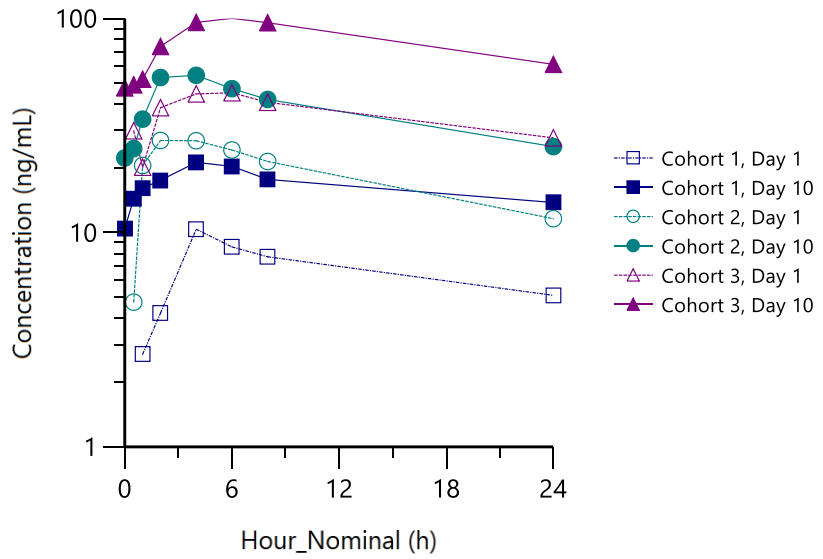
Ideal profile

Short half-life CDK9 inhibitor



KB-0742 has Plasma Half-Life of ~24 Hours After Oral Dosing

CLINICAL PK



- Preliminary PK analysis indicates that KB-0742 exhibited a dose-proportional increase in plasma exposure from 10 to 40 mg
- The t_{max} and half-life appeared independent of dose and time
- KB-0742 plasma half-life is approximately 24 hours, leading to accumulation ratios (AUC Day 10/AUC Day 1) of 2.1 to 2.5

Potential to achieve target engagement without excessive and potentially toxic peak concentrations

A Look Ahead: What's Next for KB-0742

- Anticipated announcement of the recommended Phase 2 dose (RP2D) and updated Phase 1 data in Q4 2022
- We expect to begin the dose expansion Phase 2 stage of the study in Q1 2023 and announce initial data from the Phase 2 stage in H2 2023

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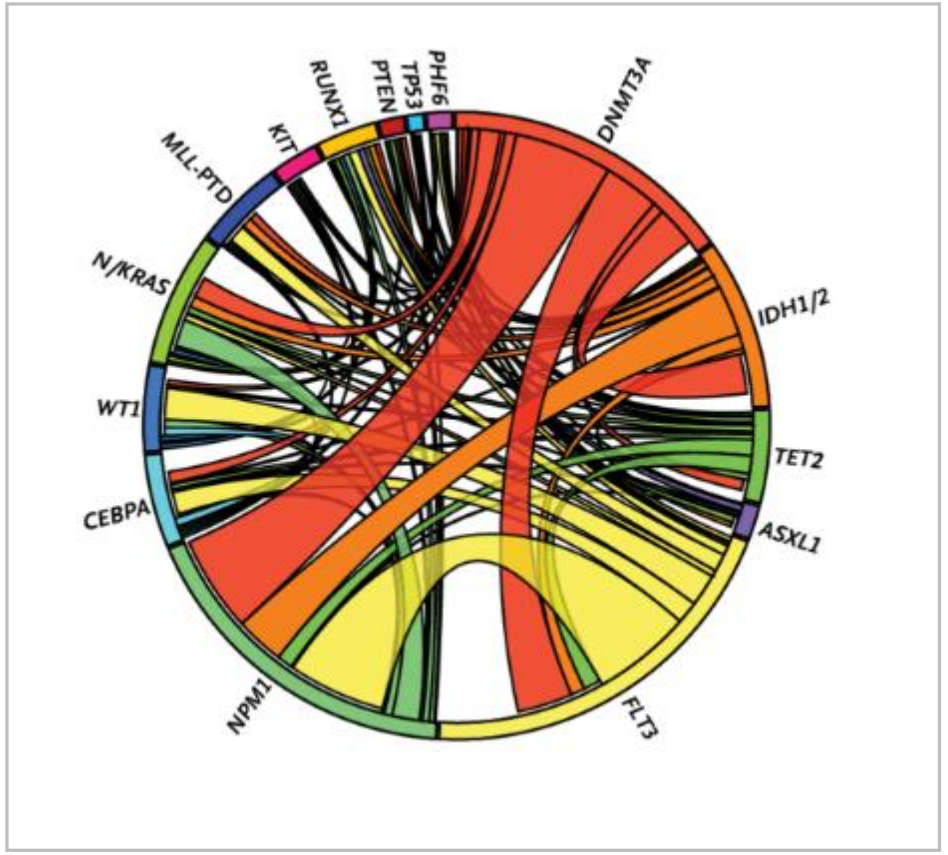
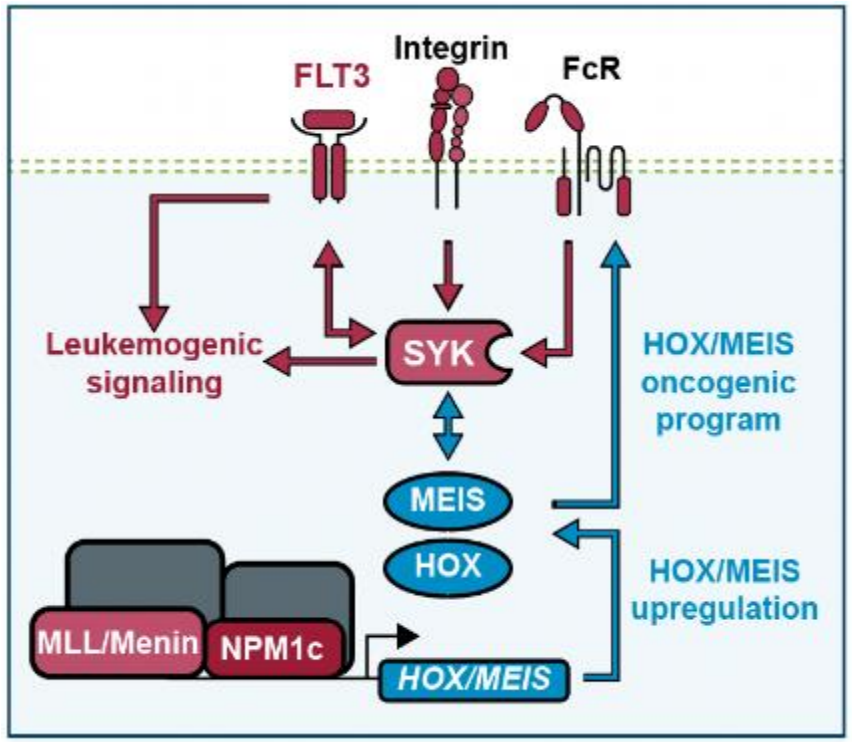
Two Investigational SYK Inhibitors Being Developed for AML

- Acute myeloid leukemia (AML) is a heterogeneous disease driven by recurring mutations – even with a number of approved therapies, unmet need remains high
- Therapies that target the underlying mutational drivers can extend survival
- Kronos Bio has two compounds – **entospletinib** (Phase 3) and **lanraplenib** (IND cleared) – that are targeted at NPM1-mutated and FLT3-mutated AML



Our SYK portfolio has potential to address significant unmet need in patients with AML

SYK is a Critical Dependency in NPM1-Mutated and FLT3-Mutated AML

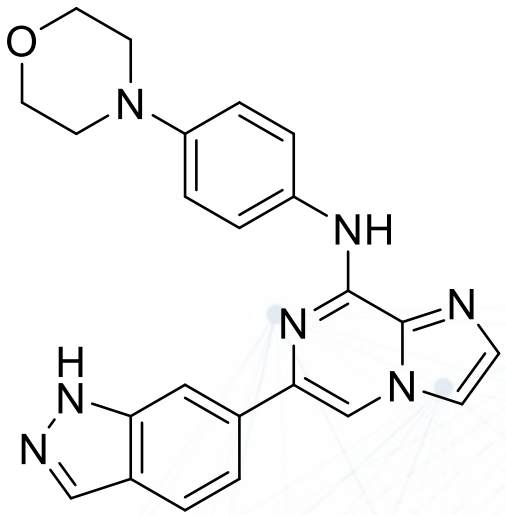


- SYK stabilizes the HOX/MEIS TRN downstream of NPM1 via a positive feedback loop
- SYK phosphorylation of FLT3 is required for FLT3-mutant leukemogenesis

Sources: Mohr et al. 2017. Cancer Cell ; Puissant et al. 2014. Cancer Cell. Figures created with BioRender.com.
AML: Acute myeloid leukemia. DNMT3a: DNA Methyltransferase 3 Alpha. FLT3: Fms like tyrosine kinase 3. MLL-r: Mixed-lineage leukemia rearrangements.
NPM1: Nucleophosmin 1. SYK: Spleen tyrosine kinase. TRN: Transcription regulatory network;

Entospletinib:
Ongoing Phase 3 Study in Patients with NPM1-Mutated AML

- Clinical data in more than 1,300 people
  - Data include more than 700 patients with a variety of hematologic malignancies
- Phase 2 clinical data in 53 patients support ongoing pivotal Phase 3 study in patients newly diagnosed with NPM1-mutated AML in combination with 7+3 chemotherapy
- Readout expected in second half of 2023

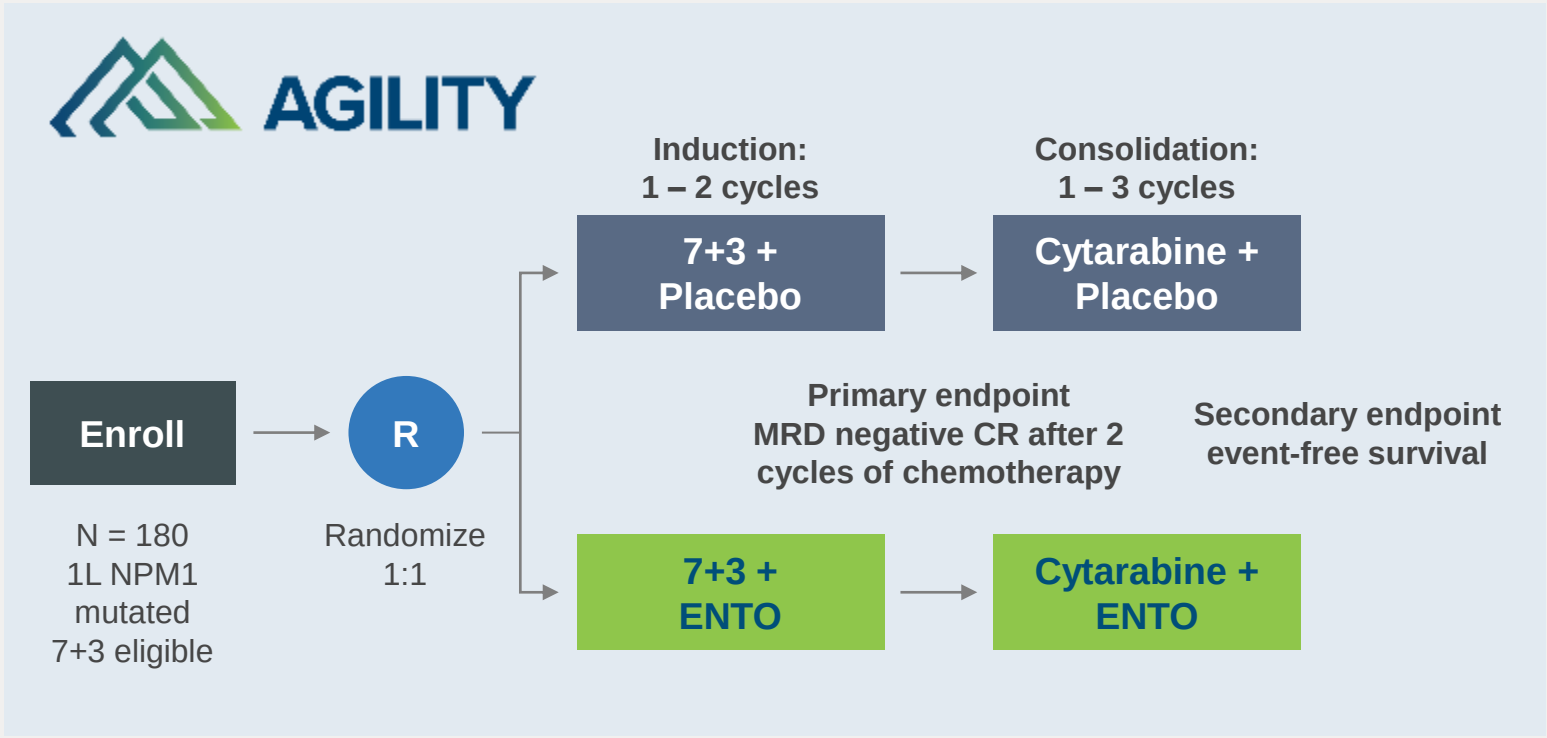


entospletinib

Large clinical database and safety profile position entospletinib to address Patients newly diagnosed with NPM1-mutated AML

Phase 3 AGILITY Trial of Entospletinib with Intensive Induction/Consolidation is Being Studied in Patients with Frontline Fit NPM1-Mutated AML

PHASE 3



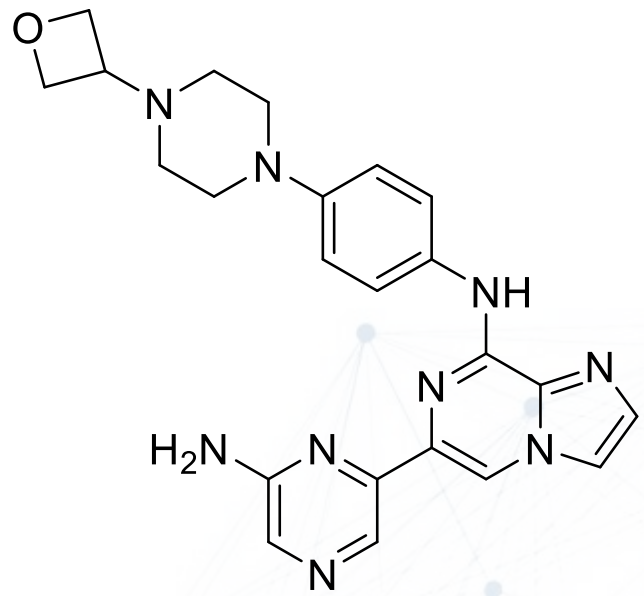
- NPM1 mutation selects for high HOXA9/MEIS1 expression
- Novel use of MRD negative CR as primary endpoint has potential to support accelerated approval
- Key secondary endpoint of EFS supports conversion for full approval

AML: Acute myeloid leukemia. CR: Complete response. CDx: Companion diagnostic. ENTO: Entospletinib. FDA: U.S. Food and Drug Administration. MRD: Measurable residual disease. NPM1: Nucleophosmin 1.

Top-line MRD data expected in H2 2023

Lanraplenib:
Phase 1b/2 Study in Patients with Relapsed/Refractory FLT3-Mutated AML

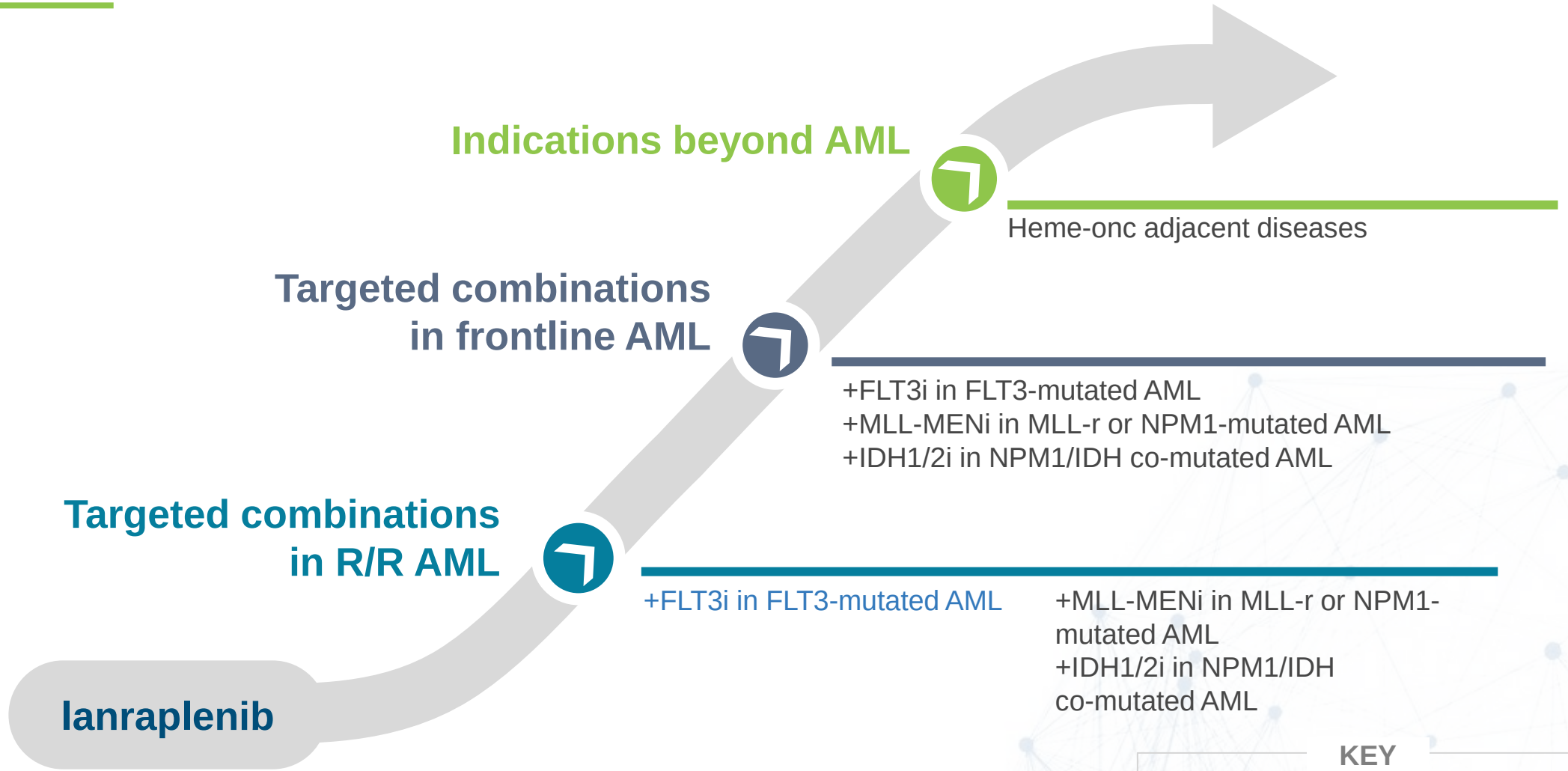
- Prior studies in autoimmune diseases show favorable PK and safety profile that support long-term, maintenance dosing
- Equivalent anti-leukemic activity to entospletinib in primary AML bone marrow samples
- Phase 1b/2 clinical trial to begin in Q2 2022 in relapsed/refractory FLT3-mutated AML in combination with gilteritinib



lanraplenib

Once daily dosing, no food restrictions and PPI compatibility support use of lanraplenib in chronic setting

Long-Term Vision for Lanraplenib: A Cornerstone of Targeted Regimens in Genetically-Defined Subsets of AML



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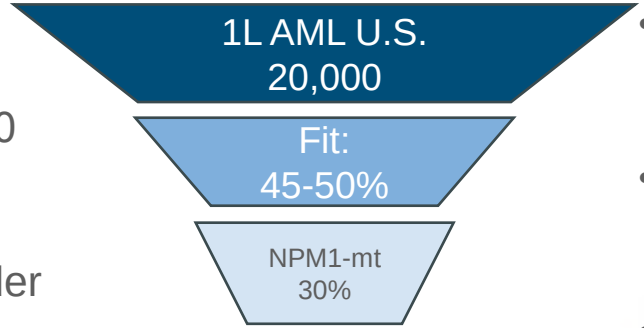
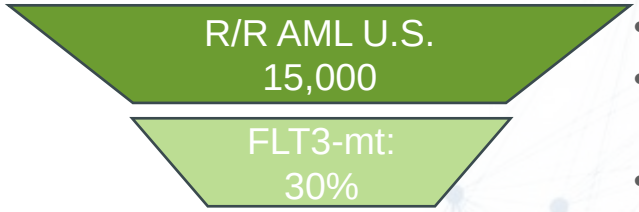
Our SYK Portfolio Addresses Significant Unmet Need for Patients With AML

		Unmet Need	Patient Population	Treatment Goal
ENTOSPLETINIB	**Patients newly diagnosed with NPM1-mutated AML, in comb. with 7+3**	• No targeted therapies approved • 68% CR rate for patients ≥60 years old receiving 7+3[1] • 5-year OS for patients with favorable risk is 64% for under 60; 37% for over 60[2]		• Increase probability of cure; limit need for transplant and associated morbidity • Achieve deepest possible CR (MRD-negative) as to increase OS • Limit added toxicity
LANRAPLENIB	**Patients with R/R, FLT3-mutated AML, in comb. with gilteritinib**	• No therapies approved in combination with standard of care (gilteritinib), partly due to gilteritinib toxicity • Poor prognosis: CR rate of 21.1% and median OS of 9.3 months[3]		• Prolong survival • Continuous disease suppression • Limit added toxicity

[1]Mims et al, 2021; [2]Herold et al, 2020; [3]Perl et al, 2019 (ADMIRAL study)

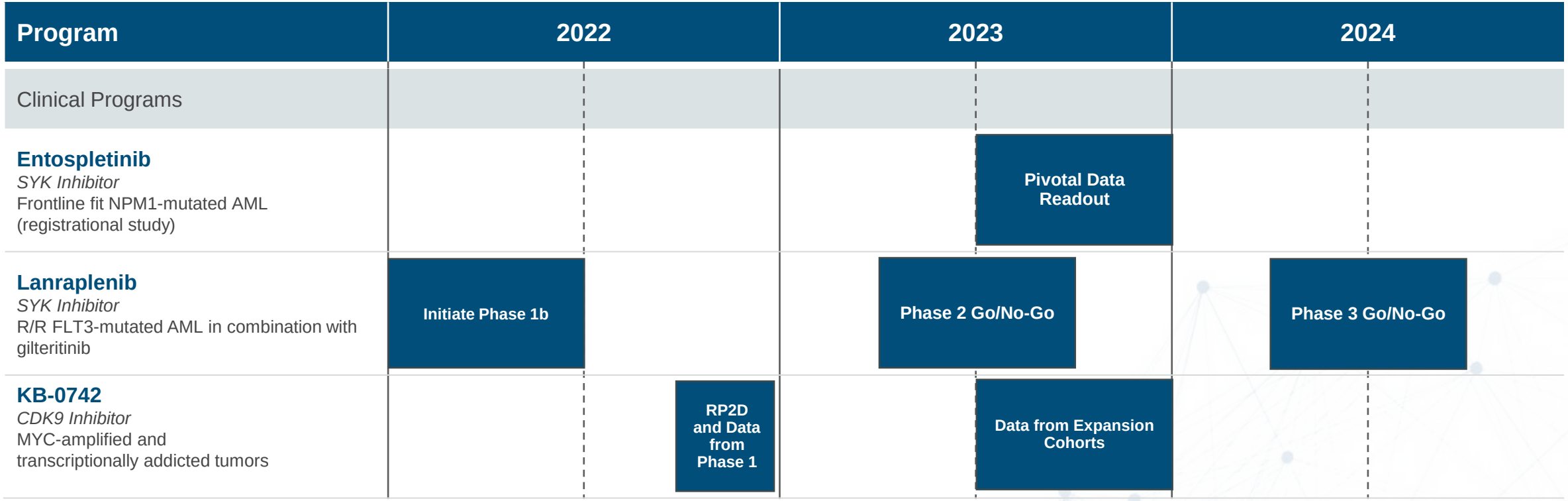
Preparing for Entospletinib:

Setting the Stage for Success by Focusing on Patient Needs

	Map potential patients	Accelerate diagnosis	Timely access to the right treatment	Meaningful patient outcomes
Today's patient experience	Most patients with fit AML are managed at academic centers	Most patients are tested for NPM1-mutation, but turnaround times can be 2-3 weeks	NPM1 targeted therapies needed; opportunity to build on momentum from treating FLT3-mutations	Best chance for a patient to be cured is in the front-line setting
Kronos Bio priority	Target pre-launch efforts at centers with highest patient volumes	Support faster turnaround times	Focus on accelerating access to treatment and patient support	Establish correlation between MRD-negative CR and improved overall survival

Commercial/medical investment in pre-launch phase designed to address patient needs

Multiple Potential Value Catalysts Through the Second Half of 2024



Discovery: Additional programs associated with MYC, AR, MYB, IRF4 and other TRNs

AML: acute myeloid leukemia. AR: androgen receptor. CDK9: cyclin dependent kinase 9. FLT3: Fms-like tyrosine kinase 3.NPM1: nucleophosmin 1. R/R: relapsed/refractory. SYK: Spleen tyrosine kinase. RP2D: recommended Phase 2 dose.

Kronos Bio is Well-Positioned

Strong Financial Position

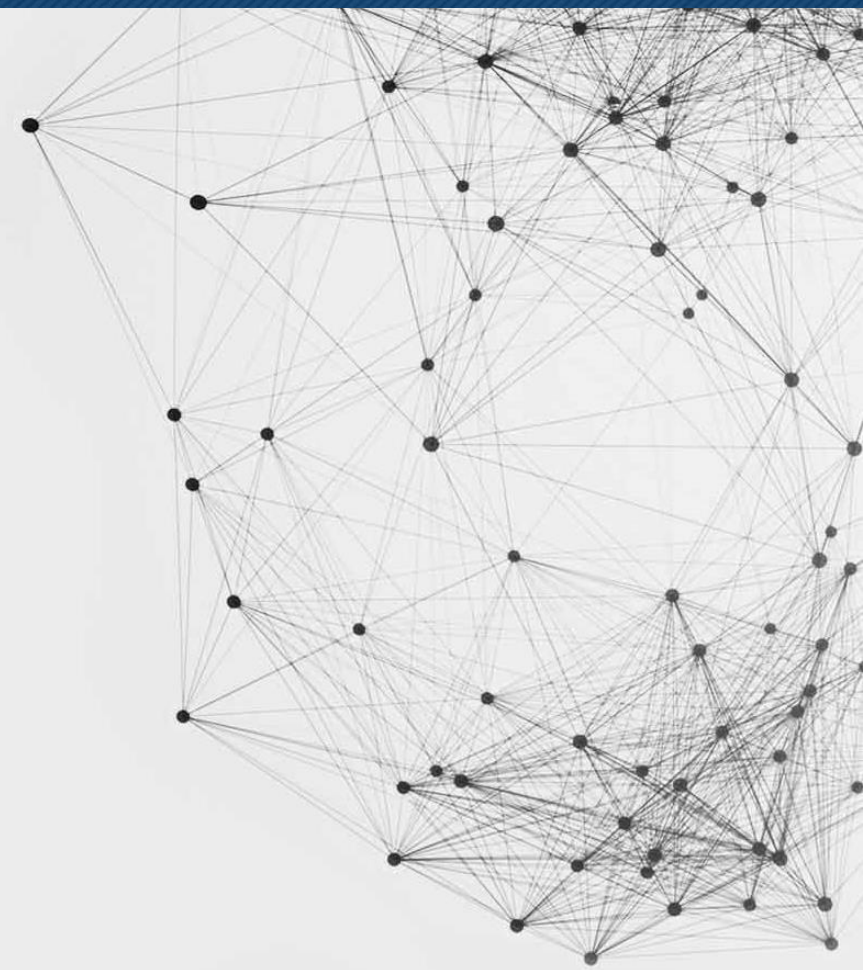
- Approx. $315.4 million in cash, cash equivalents and investments (unaudited, as of March 31, 2022)
- Cash runway into H2 2024
- Approx. 56.7 million shares outstanding (common, as of April 27, 2022)

Experienced Corporate Development Team

- Experienced team, driving collaborations and licensing agreements
- SYK portfolio acquired from Gilead in July 2020, with all rights to entospletinib and lanraplenib retained by Kronos Bio
- Ongoing collaboration with Tempus provides access to real-world and multi-omics data

GILEAD TEMPUS

KRONOS·BIO

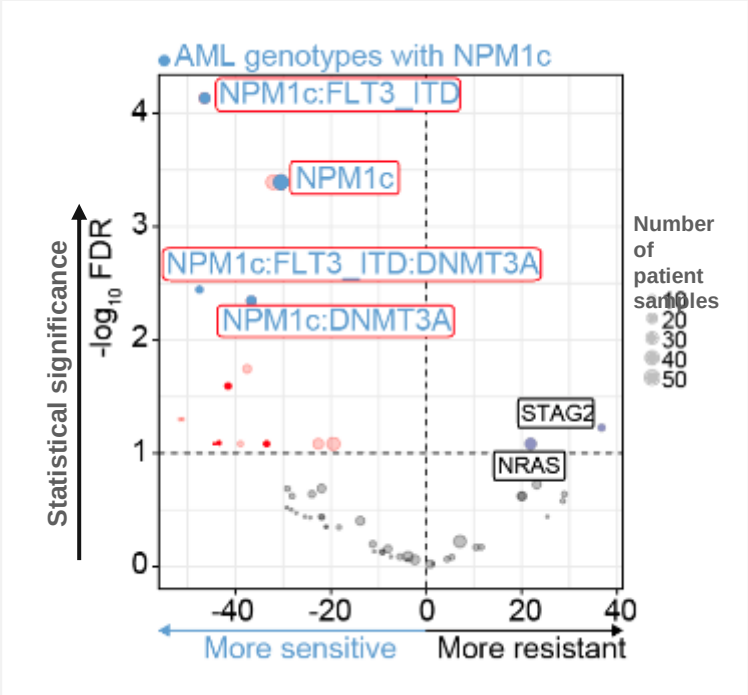


Appendix

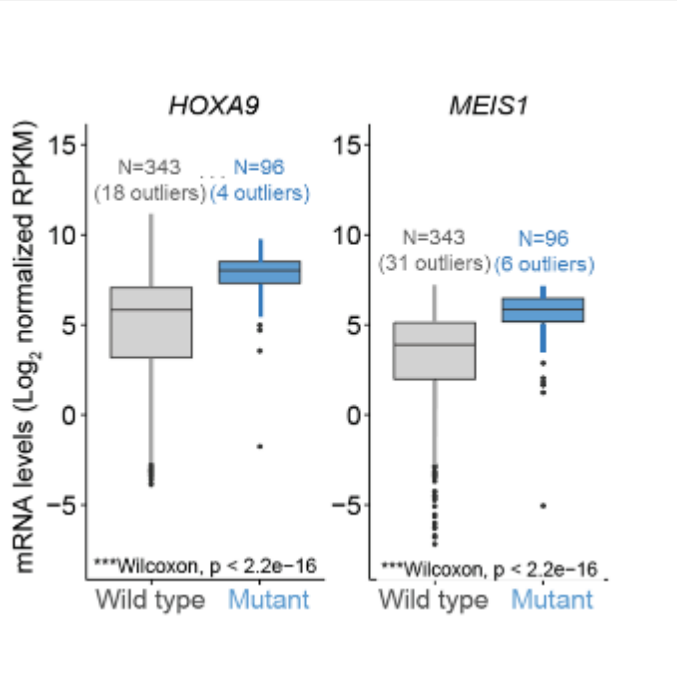
NPM1 Mutation Drives High HOXA9/MEIS1 Expression and Sensitivity to Entospletinib

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

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)*

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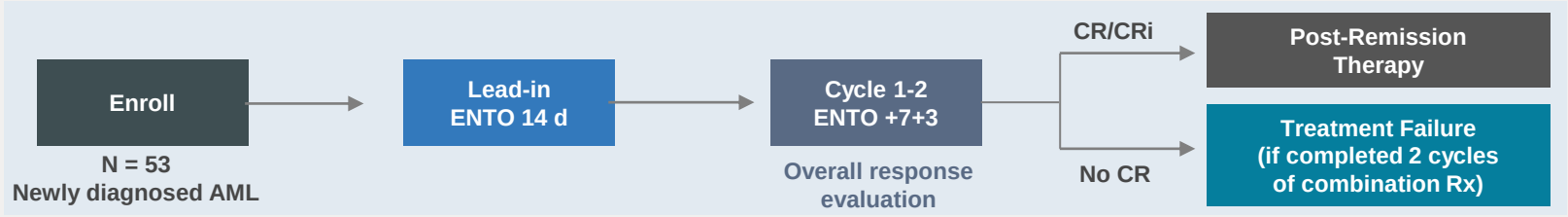
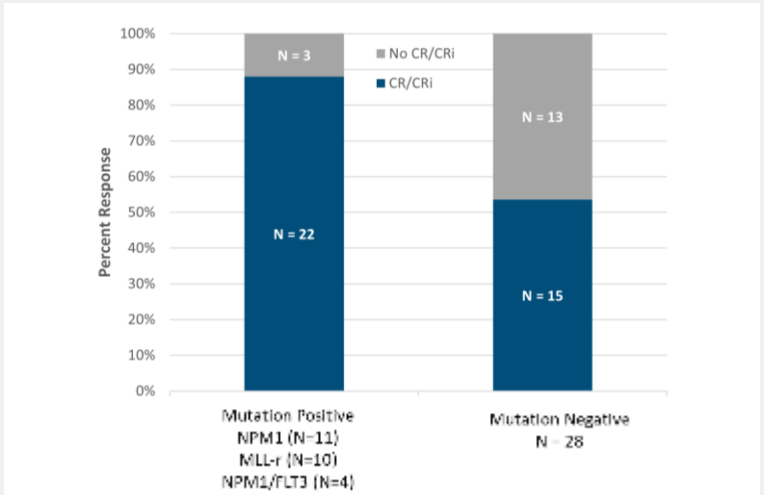
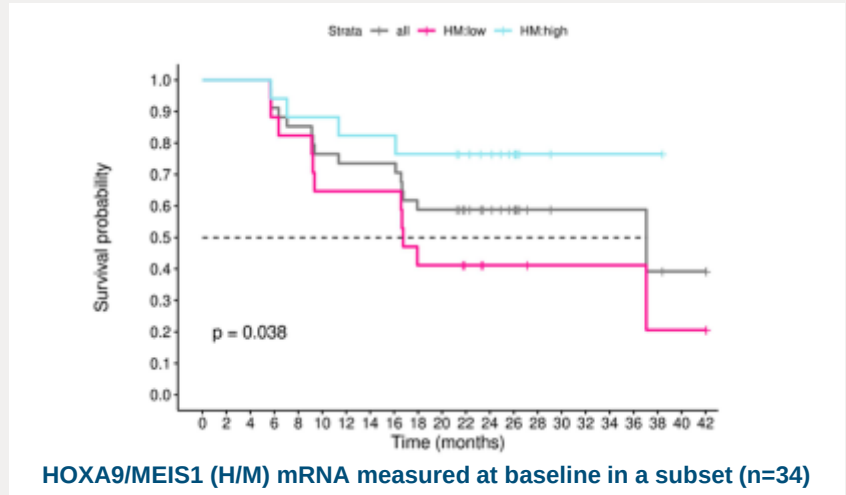
Entospletinib + 7+3 Shows Preferential Activity in Patients Newly Diagnosed with AML With Mutations that Drive High HOXA9/MEIS1 Expression

PHASE 1b/2







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

- 3/23 MLL-r patients achieved CR with entospletinib monotherapy
- HOX/MEIS high patients achieved superior CR/CRi rate and OS vs HOX/MEIS normal
- Entospletinib was well tolerated with 7+3 induction

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

Walker et al, 2020. Clin Cancer Res 26:5852-5859.
AML: Acute myeloid leukemia. CR/CRi: Complete response/complete response with incomplete hematologic recovery. ENTO: Entospletinib. FLT3: Fms like tyrosine kinase 3.
MLL-r: Mixed-lineage leukemia rearrangements. NPM1: Nucleophosmin 1. OS: Overall survival.

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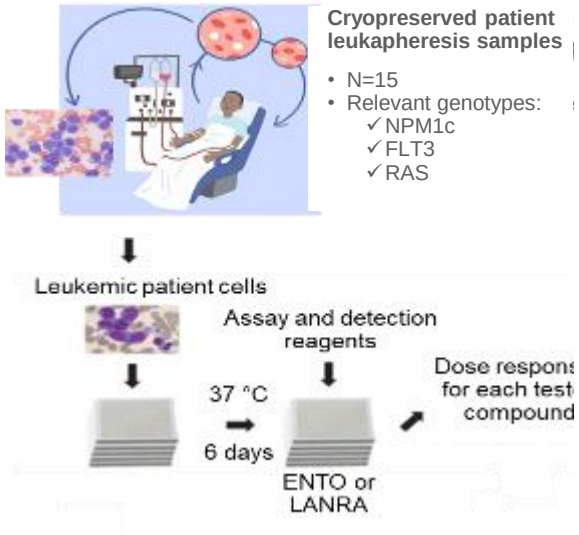
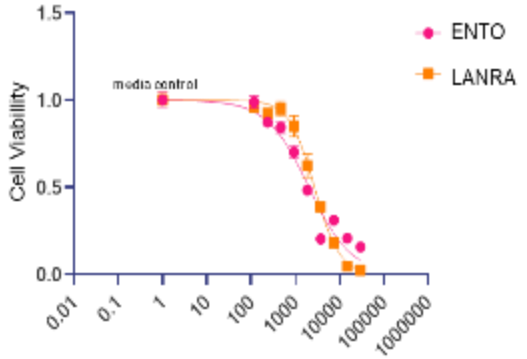
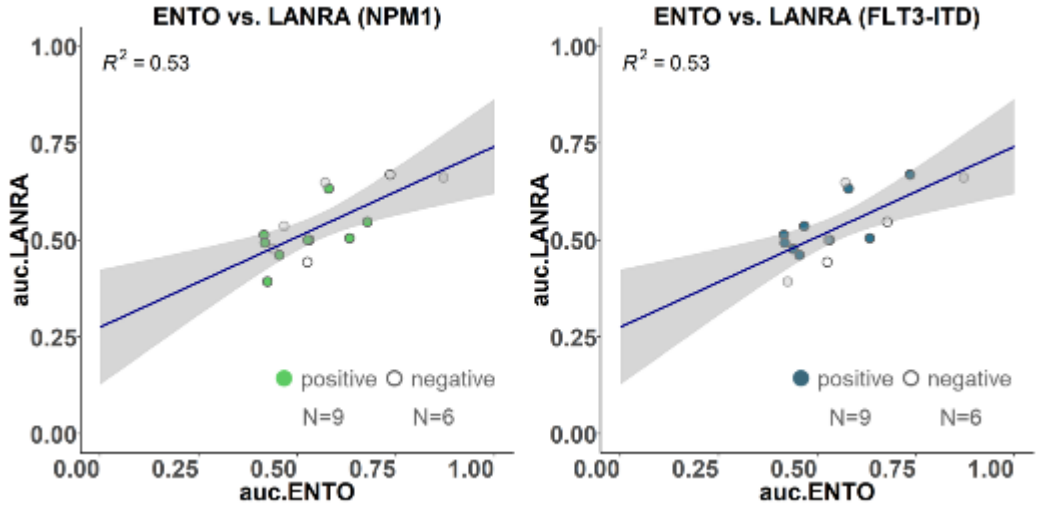
Lanraplenib Shows Preclinical Anti-Leukemic Activity Comparable to Entospletinib

Patient-derived AML cells were tested for sensitivity to lanraplenib or entospletinib in parallel





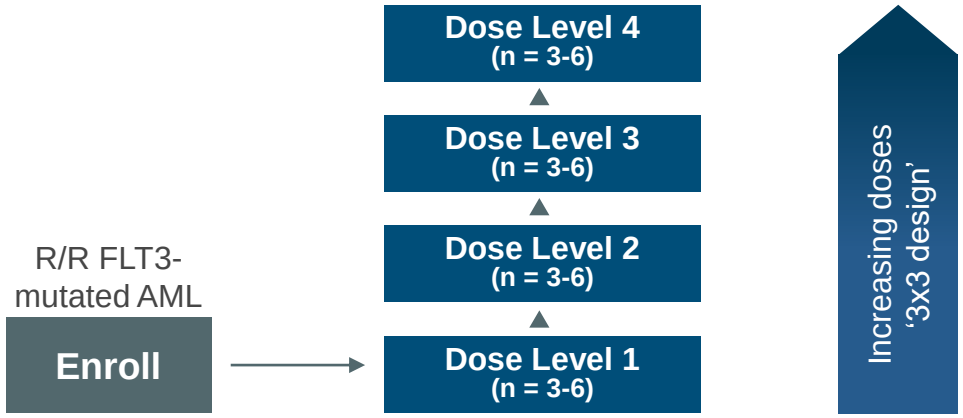
	IC_{50}	R^2	$LogIC_{50}$
ENTO	2033	0.9483	3.308
LANRA	2706	0.9902	3.432



Phase 1b/2 Trial of Lanraplenib + Gilteritinib in Relapsed/Refractory FLT3-Mutated AML

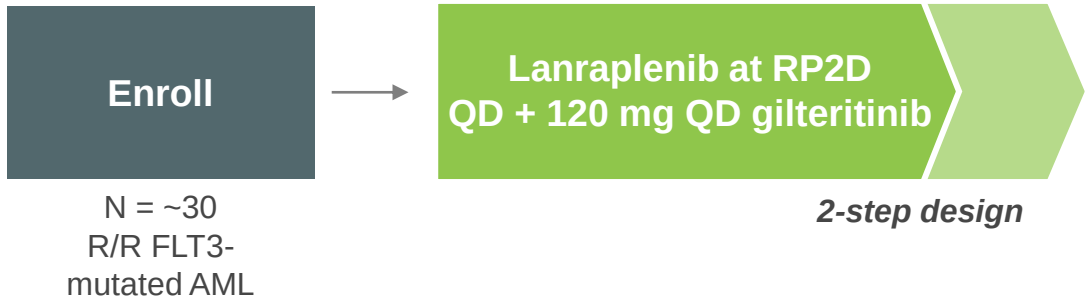
PHASE 1b/2

STAGE 1: DOSE ESCALATION



- Evaluate initial safety, PK, and anti-leukemic activity (cCR rate) in escalating doses of lanraplenib QD in combination with gilteritinib 120 mg QD

STAGE 2: EXPANSION COHORT

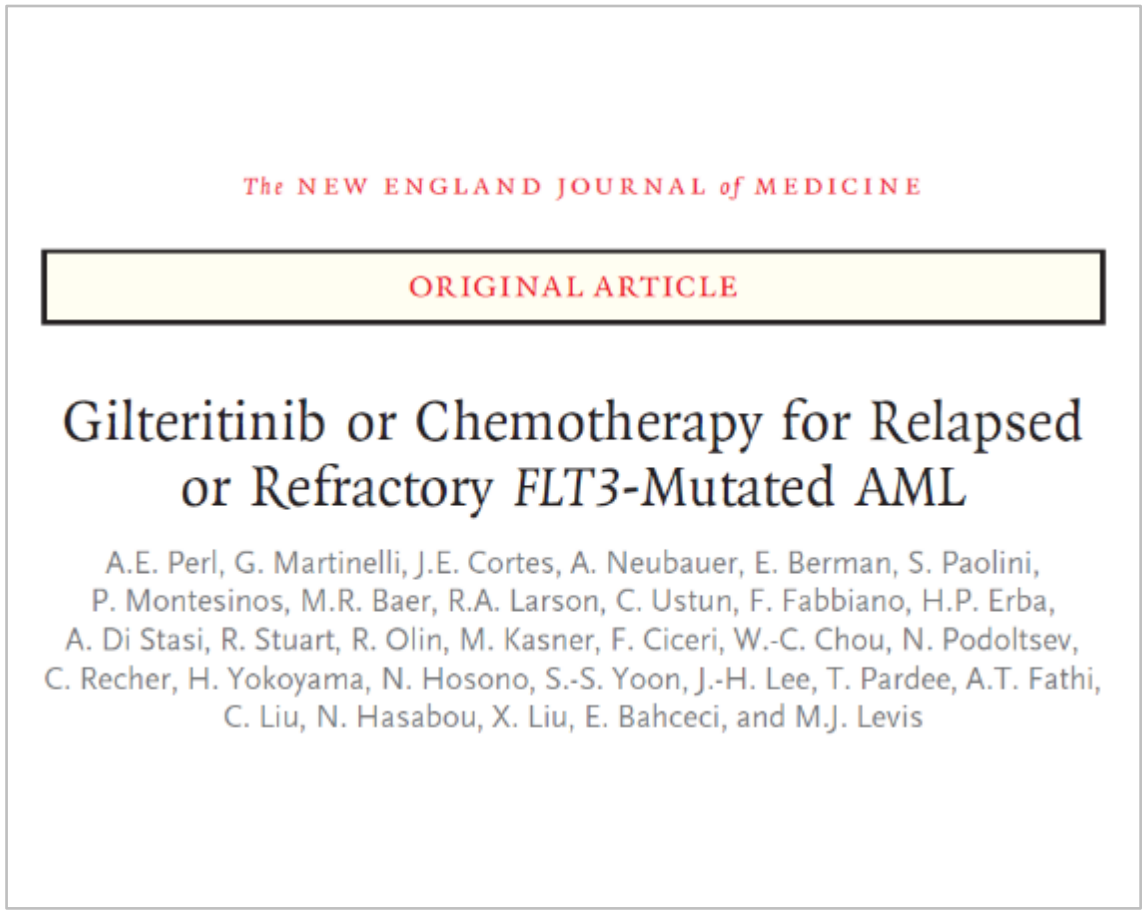


- Further evaluate safety and anti-leukemic activity (cCR rate and DoR)
- Inform Phase 3 trial design

Lanraplenib + gilteritinib clinical trial initiation expected in Q2 2022

AML: Acute myeloid leukemia. cCR: Complete clinical response. DoR: Duration of response. FLT3: Fms like tyrosine kinase 3. LANRA: Lanraplenib. PK: pharmacokinetics. QD: Quaque die (once a day). RP2D: Recommended Phase 2 dose. R/R: Relapsed/refractory.

Other AML Opportunities for SYK Inhibition: Investigational Combination with Gilteritinib in R/R Patients with FLT3 ITD/TKD



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Gilteritinib or Chemotherapy for Relapsed or Refractory *FLT3*-Mutated AML

A.E. Perl, G. Martinelli, J.E. Cortes, A. Neubauer, E. Berman, S. Paolini, P. Montesinos, M.R. Baer, R.A. Larson, C. Ustun, F. Fabbiano, H.P. Erba, A. Di Stasi, R. Stuart, R. Olin, M. Kasner, F. Ciceri, W.-C. Chou, N. Podoltsev, C. Recher, H. Yokoyama, N. Hosono, S.-S. Yoon, J.-H. Lee, T. Pardee, A.T. Fathi, C. Liu, N. Hasabou, X. Liu, E. Bahceci, and M.J. Levis

ADMIRAL Trial (gilteritinib approval)

- N = 371
- > 18 yo refractory to 1-2 cycles of 7+3 or relapsed after CR with 7+3 AND
- FLT3 ITD/TKD
- Enrolled at 107 sites/28 mo = 0.12 p/s/m

Endpoint	Chemo (N = 124)	Gilteritinib (N = 247)
CR	10.5%	21.1%
mOS*	5.6 mo	9.3 mo (HR 0.64)
mEFS	0.7 mo	2.8 mo (HR 0.79)

*Prior midostaurin (N = 37) OS HR = 0.70

Perl et al, 2019. NEJM 381:381:1728-1740.
AML: Acute myeloid leukemia. CR: Complete response. FLT3: Fms like tyrosine kinase 3. HR: Hazard ratio. ITD: Internal tandem duplication. mEFS: Median event-free survival. mOS: Median overall survival. R/R: Relapsed/refractory. SYK: Spleen tyrosine kinase. TKD: Tyrosine kinase domain.

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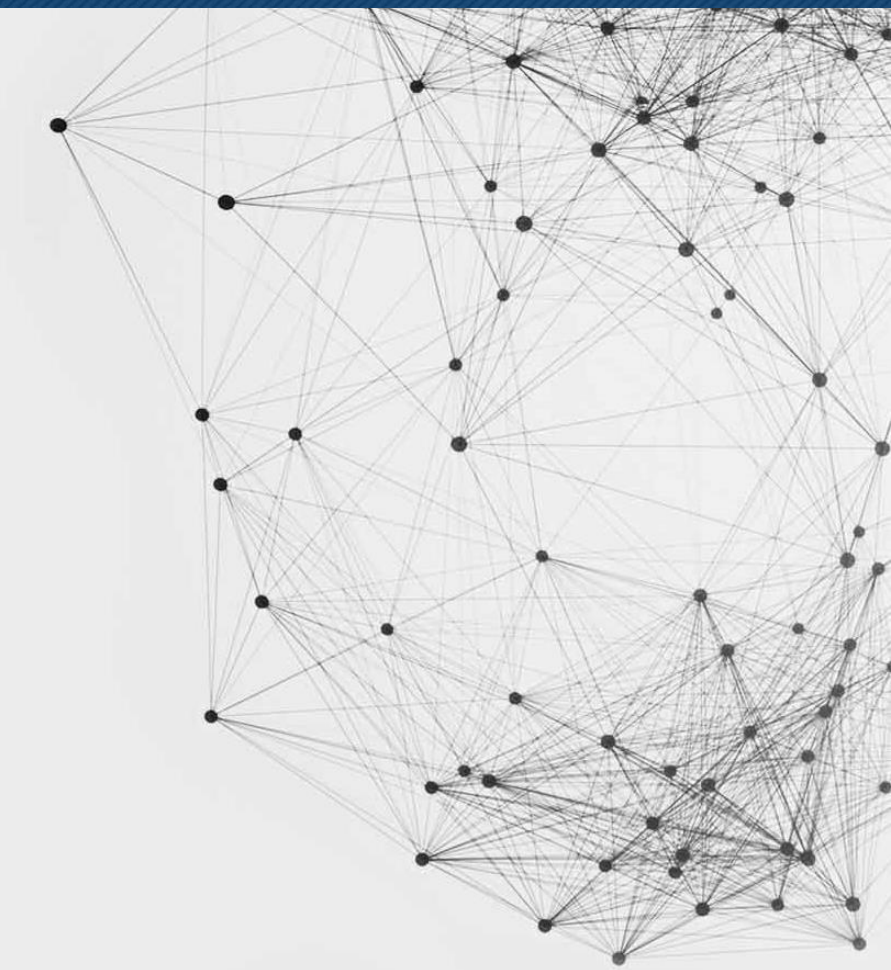

For additional information, please contact Marni Kottle at mkottle@kronsobio.com.