



Company Update

November 8, 2022

Today's Participants



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



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 Corp. Development



Marni Kottle
Senior Vice President,
Corp. Comms and
Investors Relations

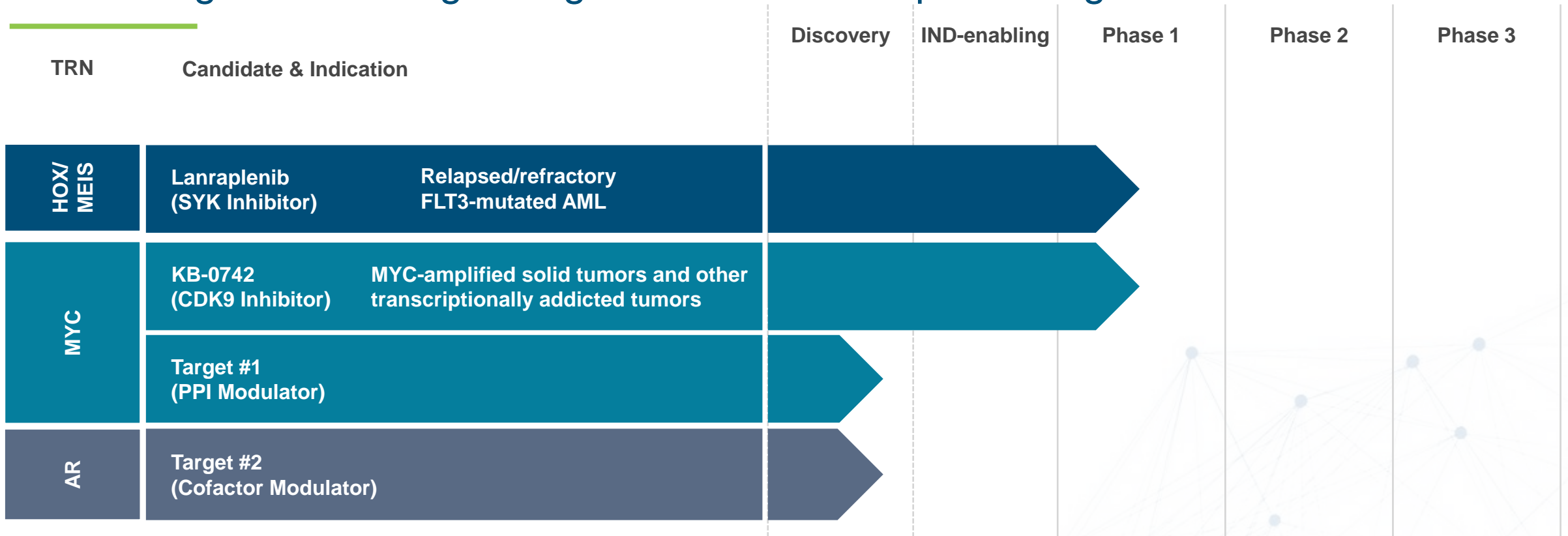
Forward Looking Statements

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 product development plans and timelines, the potential benefits of the Company’s product candidates, market size and opportunity, the Company’s strategy, intellectual property matters, regulatory matters, and the sufficiency of the Company’s resources and the Company’s future financial position. 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, those related to clinical trial enrollment, results of preclinical studies and early clinical trials are not necessarily predictive of future results, the development of the Company’s business, trends in the industry, the legal and regulatory framework for the industry and future expenditures. These and other risks are described in greater detail in the Company’s filings with the Securities and Exchange Commission (SEC), including under the heading “Risk Factors” in its Quarterly Report on Form 10-Q for the quarter ended September 30, 2022, filed with the SEC on November 8, 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.

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Advancing Clinical-Stage Programs Across Multiple Oncogenic TRNs



Additional programs from mapping and screening the MYC, AR, MYB, IRF4 and other TRNs

Potential Value Catalysts

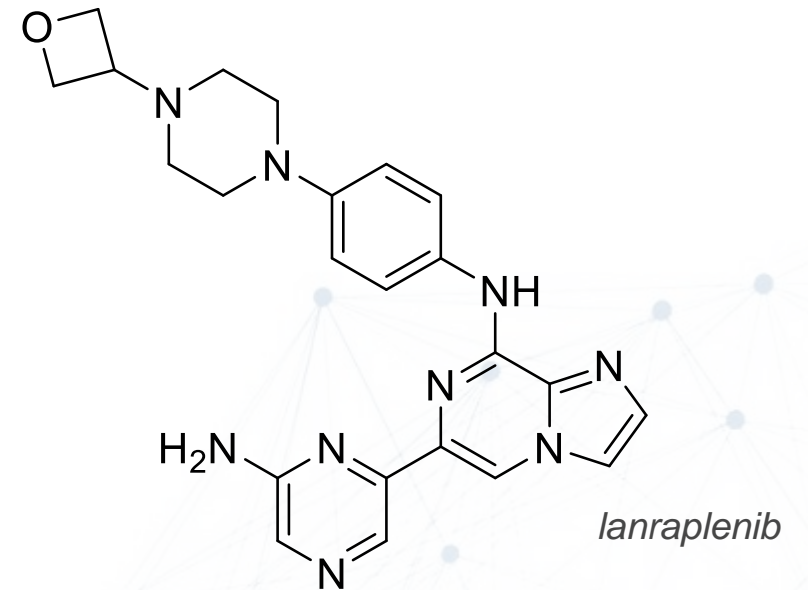
Program	2022	2023	2024
Clinical Programs			
Lanraplenib <i>SYK Inhibitor</i> R/R FLT3-mutated AML in combination with gilteritinib			RP2D and Initial Data
KB-0742 <i>CDK9 Inhibitor</i> MYC-amplified and transcriptionally addicted tumors	PK/ PD and safety data; RP2D	Initial Phase 2 Efficacy Data from Expansion Cohorts	

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

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

Lanraplenib Profile is Well-Suited as Part of a Chronically Administered Regimen

- Lanraplenib has been tested in more than 250 clinical trial participants and demonstrated pharmacokinetic (PK), pharmacodynamic (PD) properties consistent with once daily dosing, no food restrictions and PPI compatibility
- Lanraplenib's single agent safety profile in these studies has not demonstrated any clinically significant toxicities
- These properties support patient compliance in regimens that are dosed to progression



Lanraplenib: Addressing Distinct Unmet Needs in Relapsed/Refractory AML

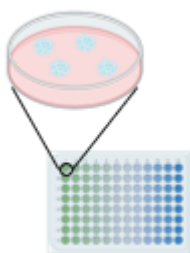
	Kronos Bio Phase 1b/2 Trial	Potential Future Combination Opportunities	
	LANRA + gilteritinib (FLT3)	LANRA + Menin (NPM1/MLLr)	LANRA+ IDH1/2i (IDH co-mutated with NPM1/FLT3/MLLr)
Addressable population (U.S.)	~5,000	~5,000	~1,200 – 1,500
Dose/schedule	Once Daily	Once Daily	Once Daily
Drug-drug Interactions	None	None	None
Duration of treatment	Progression	Progression	Progression

Lanraplenib could provide continuous disease suppression in combination with targeted agents, initially in relapsed/refractory AML

Lanraplenib + Gilteritinib has Greater than Additive Anti-Leukemic Activity in Preclinical Study



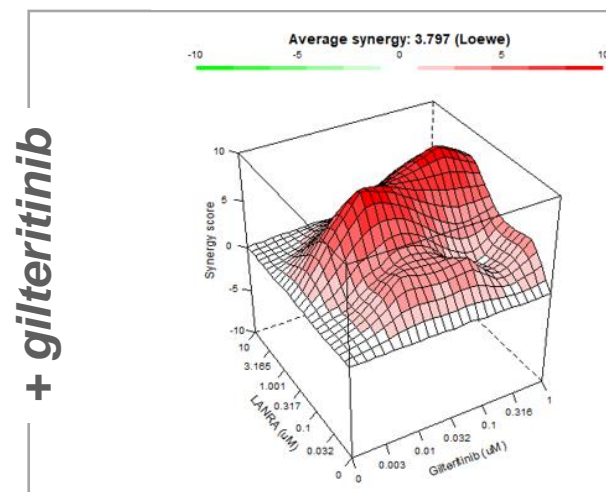
AML cells from patients are injected into mice for expansion in bone marrow niche



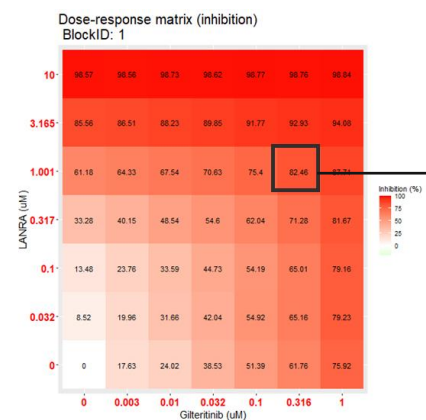
LANRA + gilteritinib

Culture cells ex vivo for 5 days with LANRA + gilteritinib and assess viability

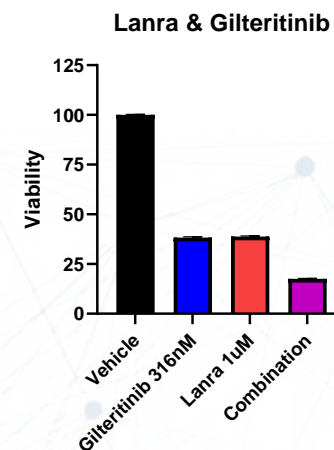
Synergistic effect topology



Viability inhibition matrix



Effect from indicated coordinate



Data presented at EHA, 2022. McKeown, Michael, et al. SYK Inhibition Drives Deep Responses in a Biomarker Guided Subset of AML Alone and in Rational Combinations (EHA poster presentation)

Gilteritinib is Important Advance for FLT3-mt AML; Significant Need Remains

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.

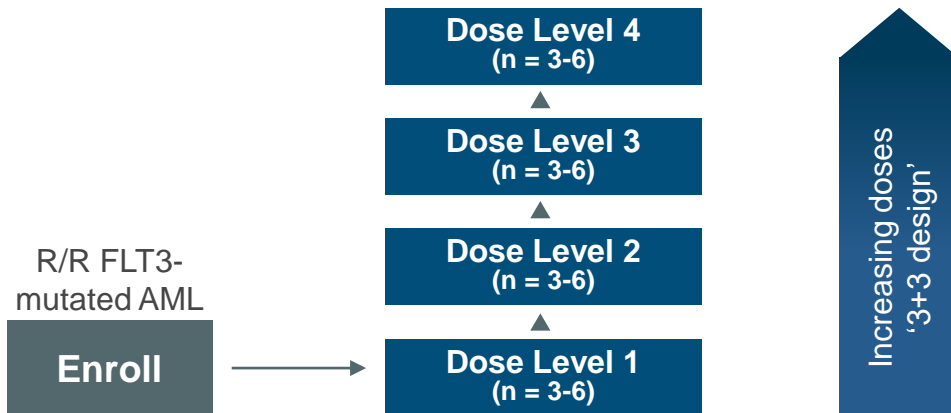
AML: Acute myeloid leukemia. CR: Complete response. FLT3: Fms like tyrosine kinase 3. HR: Hazard ratio. ITD: Internal tandem duplication. mEFS: Median event-free survival. mOS: Median overall survival.

R/R: Relapsed/refractory. SYK: Spleen tyrosine kinase. TKD: Tyrosine kinase domain.

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

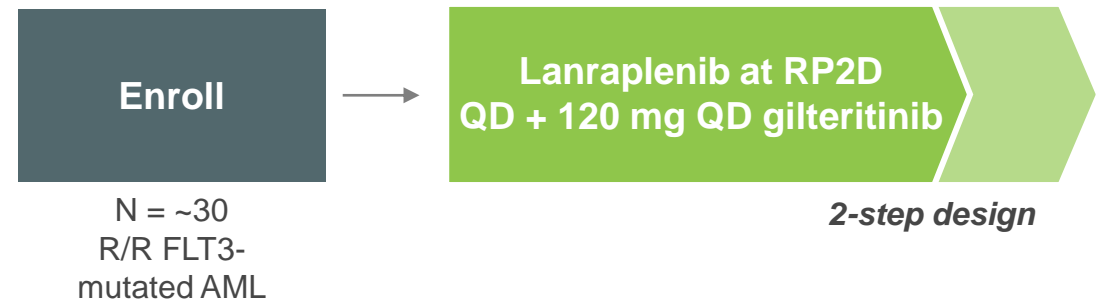
PHASE 1b/2

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



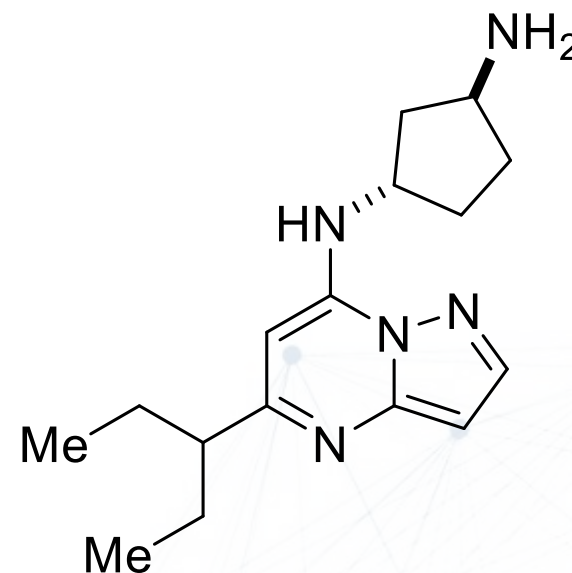
- Further evaluate safety and anti-leukemic activity (cCR rate and DoR)
- Inform Phase 3 trial design

Lanraplenib + gilteritinib clinical trial is ongoing

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.

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
- Preliminary PK analysis indicates that KB-0742 exhibited a dose-proportional increase in plasma exposure and half-life of ~24 hours
- Phase 1/2 trial ongoing with RP2D and additional data expected in Q4 2022

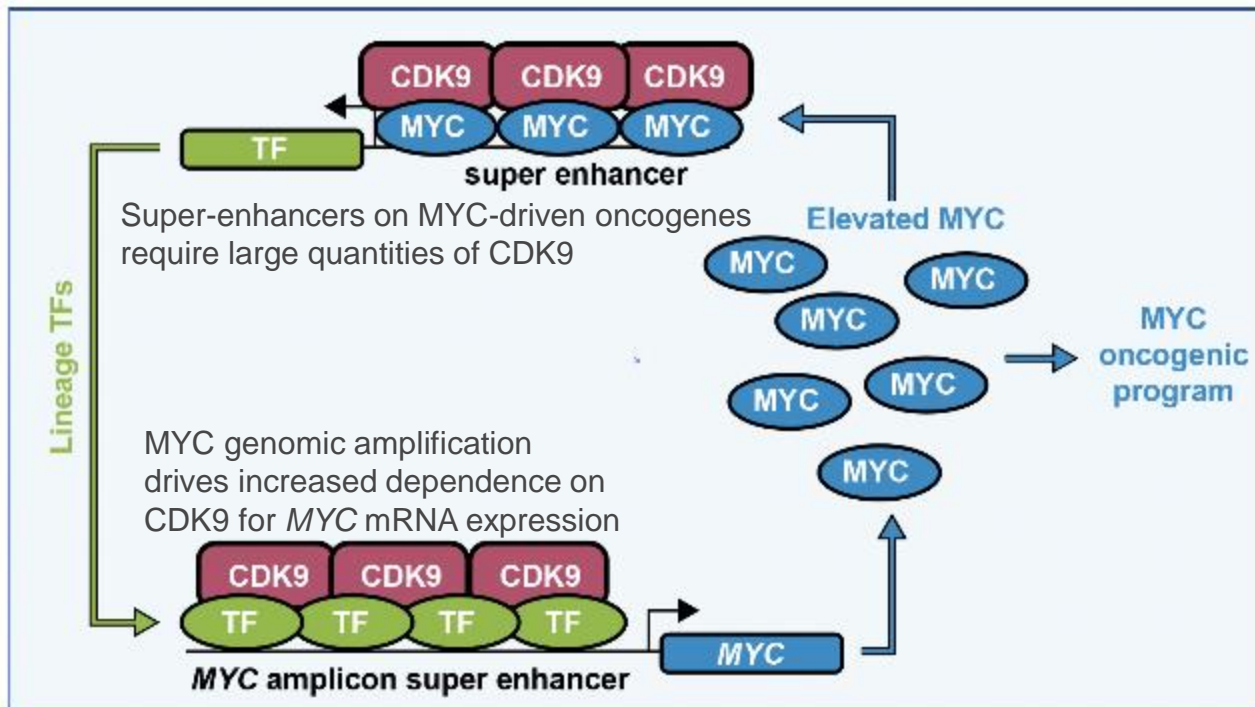


KB-0742

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.

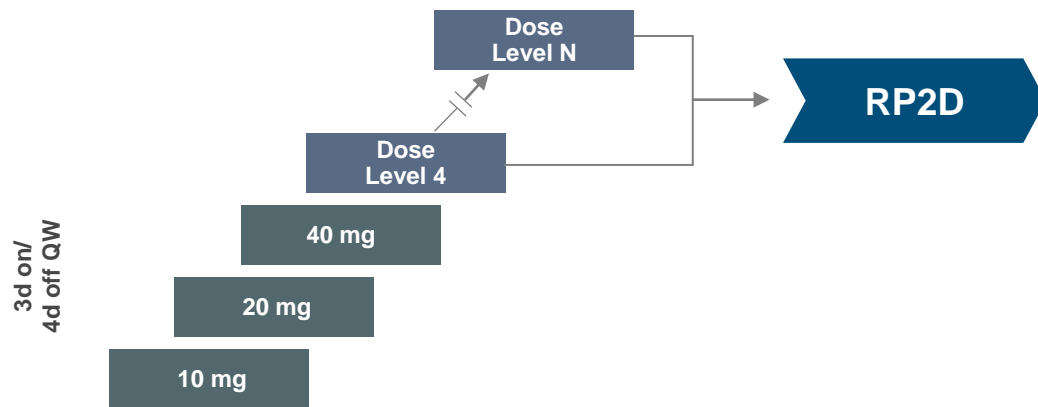
- CDK9 phosphorylates RNA pol II, allowing transcription to proceed driving mRNA expression of MYC itself and its target genes
- Tumors that are addicted to MYC have heightened sensitivity to CDK9 inhibition
- Intermittent, partial inhibition of CDK9 can be active without causing unacceptable toxicity

CDK9 is an attractive target in transcriptionally addicted cancers

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

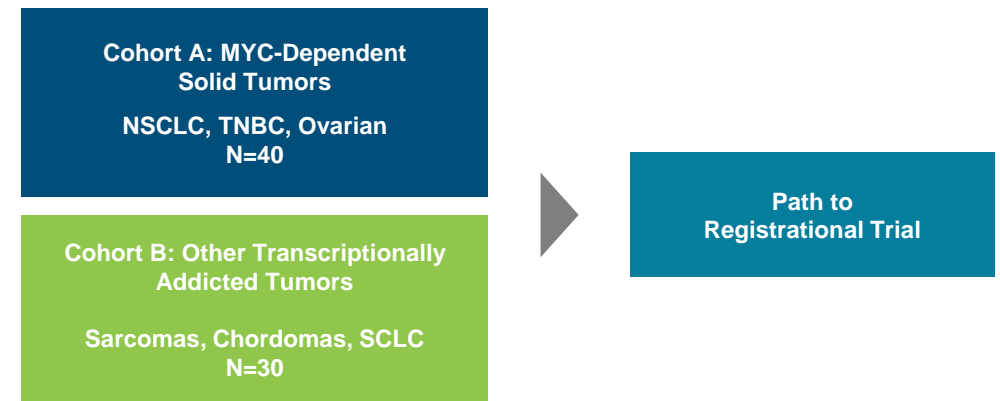
PHASE 1/2

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



- **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. SCLC: small cell lung cancer. PD: pharmacodynamics. PK: pharmacokinetics. QW: weekly. TNBC: triple-negative breast cancer. PBMC: peripheral blood mononuclear cells. RP2D: recommended Phase 2 dose

Kronos Bio has Financial Resources and Experienced Management Team

Strong Financial Position

- Approx. \$270.3 million in cash, cash equivalents and investments (unaudited, as of Sept. 30, 2022)
- Cash runway into Q2 2025
- Approx. 56.9 million shares outstanding (common, as of Nov. 2, 2022)

Experienced Corporate Development Team

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



Q&A





Thank you

