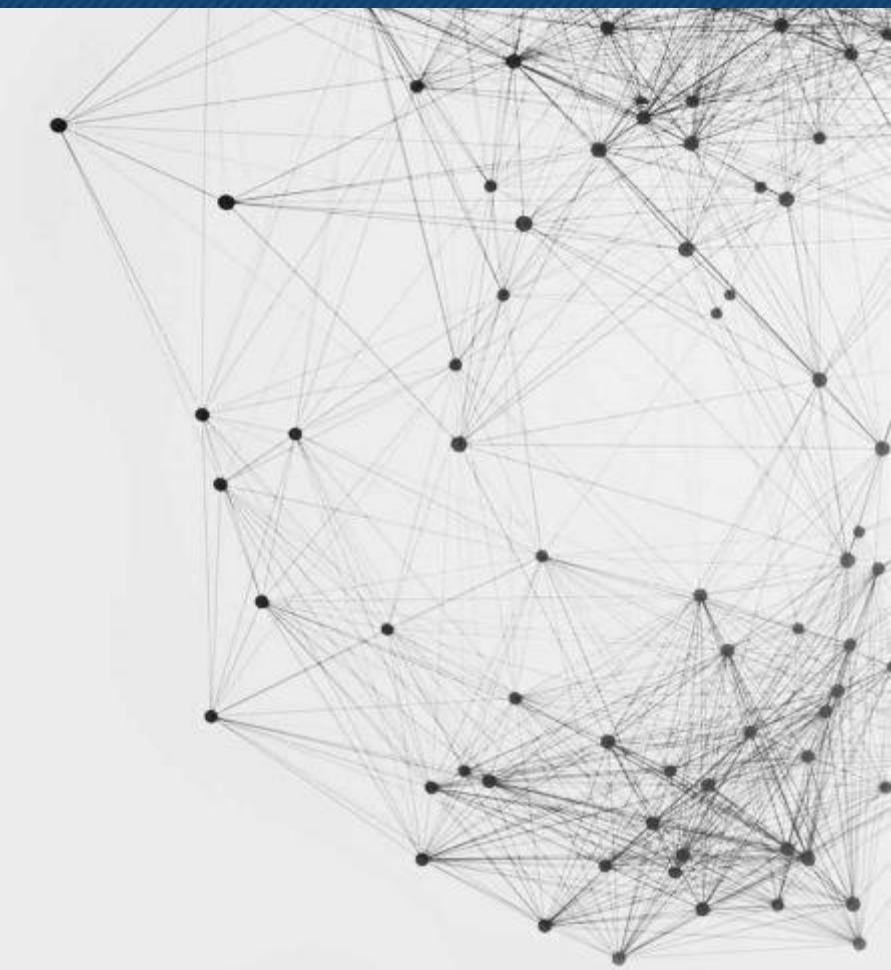
Corporate Overview

March 2024

KRONOS·BIO



01 **Introduction**

02 Our pipeline

- KB-0742 (CDK9 inhibitor)
- KB-9558 (p300 KAT inhibitor)

03 Our product engine

- TRN mapping
- TRN screening

04 Kronos Bio milestones and financials

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 are developing two internally discovered compounds, KB-0742, a Ph1/2 CDK9 inhibitor, and KB-9558, a p300 KAT inhibitor in IND-enabling studies

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

KRONOS·BIO

Leader in drugging transcription to address unmet needs in cancer

Leader in drugging transcription

Transcriptional deregulation is a hallmark of cancer

Focused pipeline of best-in-class molecules derived from our machine learning, systems biology, small molecule microarray, and medicinal chemistry capabilities

Large market potential

Transcriptional regulatory networks of focus are implicated in **>30% of all tumors**

KB-0742 has the potential to annually **address up to >150,000 U.S. patients**

Near-term value drivers

KB-0742 clinical data update expected **mid-year**; completion of dose escalation expected in **Q3 2024**; data from expansion phase expected **1H 2025**

Progressing p300 KAT inhibitor development candidate, KB-9558, for IRF4 TRN in multiple myeloma; completion of IND enabling studies expected **Q4 2024**

Strong strategic collaborations

Discovery collaboration with **Genentech worth up to $574M** in upfront and milestone payments to advance novel therapies against transcriptional targets in oncology

Multi-omic data collaboration with **Tempus** drives discovery and clinical development using real world evidence

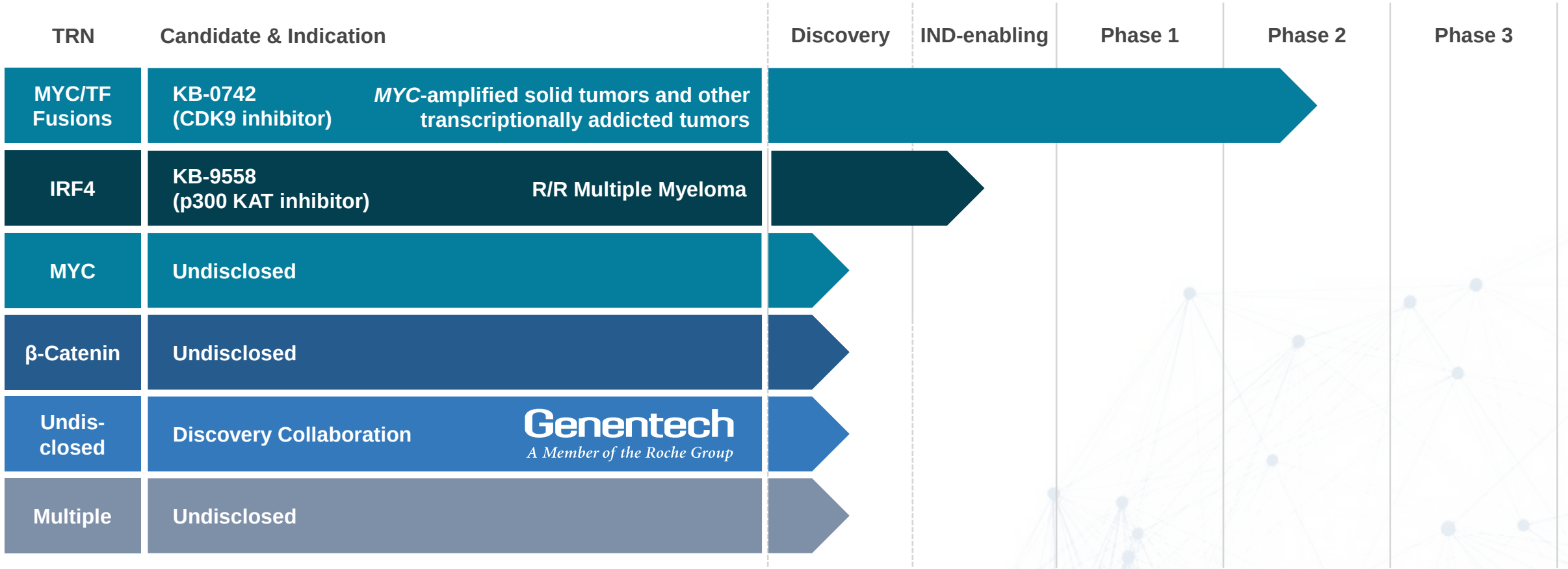
Well-funded

$175M in cash, cash equivalents and investments (as of December 31, 2023)

Expected cash runway into 2H 2026

IRF4: interferon regulatory factor 4. KAT: lysine acetyltransferase. TRN: transcription regulatory network.

Advancing both clinical and discovery programs across multiple oncogenic TRNs



KAT: lysine acetyltransferase. IRF4: interferon regulatory factor 4. TF: transcription factor. TRN: transcription regulatory network.

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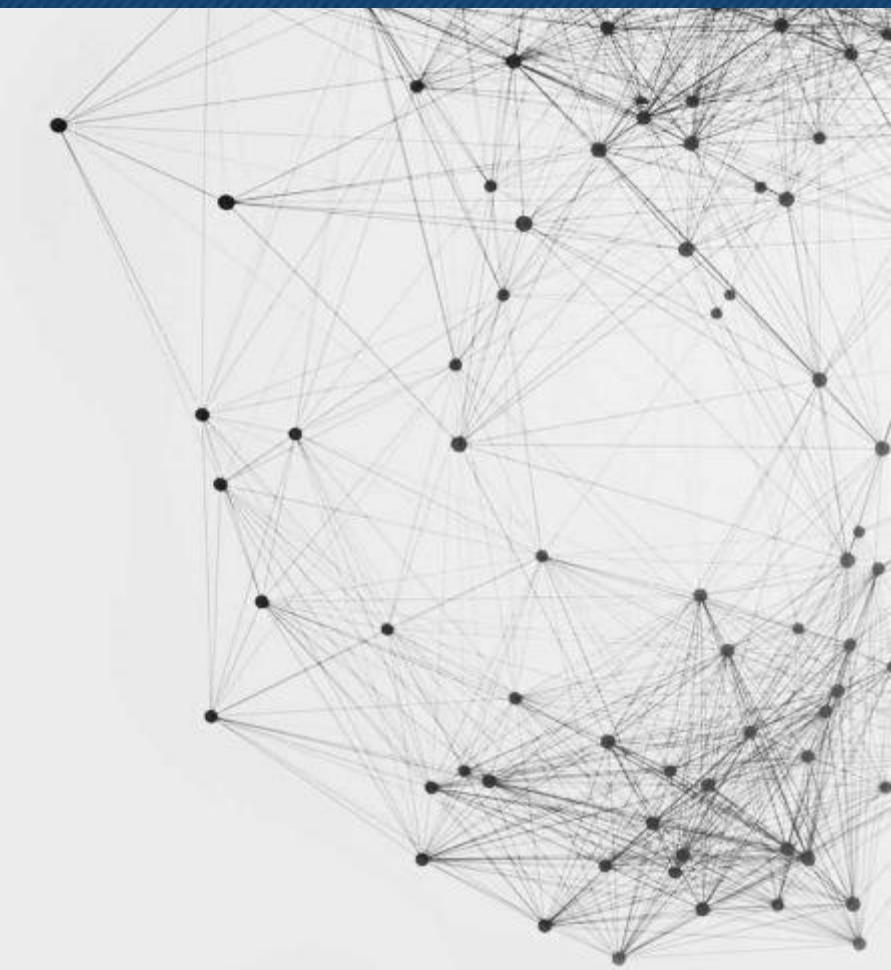

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KB-0742, an oral CDK9 inhibitor, in phase1/2 dose escalation and expansion trial for solid tumors

- **Preliminary efficacy established in TF-fusion driven tumor as presented at AACR-NCI-EORTC 2023**
  - At the 60mg dose (3 on/4 off), KB-0742 demonstrated on-mechanism single-agent anti-tumor activity in heavily pre-treated patients. Patient who achieved PR remained on treatment for 398 days
- **On-mechanism single agent activity expected across multiple histologies**
  - *MYC* amplification or overexpression present in ~85% of ovarian cancer[2], ~78% of TNBC[2], ~45% of NSCLC[2]
  - All of SCLC is dependent on oncogenic transcription factor programs, including MYC-family[3] and lineage specific[5] transcription factors
  - >30%[1] of sarcomas and >95%[4] of ACC patients present with TF-fusions
- **No grade 3/4 neutropenia has been observed at doses up to 80mg 3 days on/4 days off per week**
  - Continuing with dose escalation since MTD has not been defined
- **Differentiated product profile - highly selective, oral dosing, with 24 hour half-life**
- **Composition of matter patent until 2039**

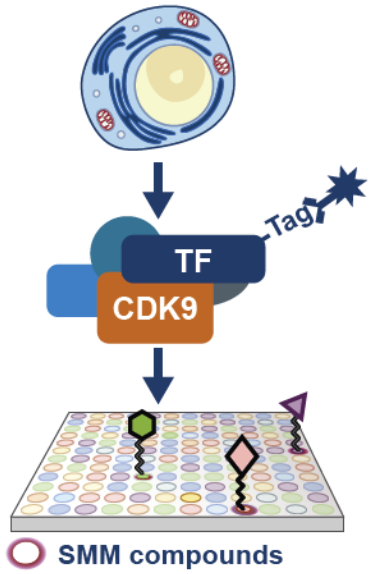
Data from 60mg dose expansion and 80mg dose escalation to be presented mid-2024

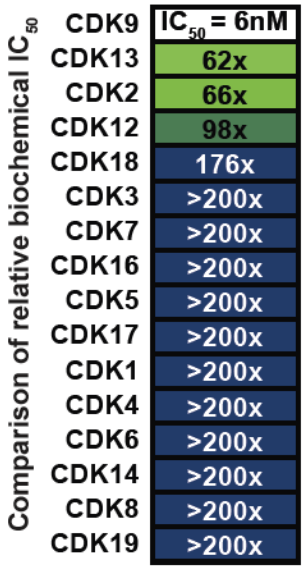
ACC: adenoid cystic carcinoma. MTD: maximum tolerable dose. NSCLC: non-small cell lung cancer. PR: partial response. SCLC: small cell lung cancer. TF: transcription factor. TNBC: triple negative breast cancer.

Source: 1. Huang et al (2017) ; 2. Tempus proprietary analysis; 3. Tlemsani et al. (2020) 4. Togashi et al. (2018) 5. Baine et al. (2020)

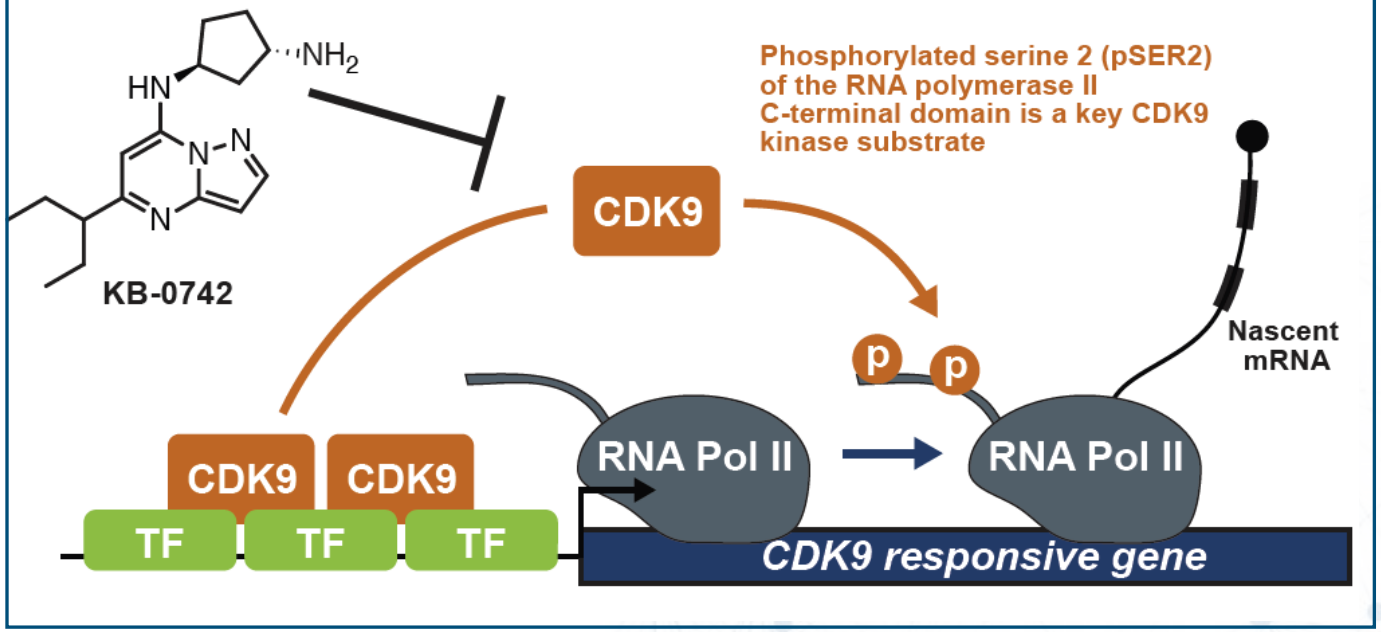
KB-0742 is a highly selective, orally bioavailable inhibitor of CDK9, a critical regulator of oncogene transcription

KB-0742 was optimized following a Small Molecule Microarray (SMM) screen against an oncogenic variant of the androgen receptor TF





CDK9 is a kinase that is recruited to DNA by TFs and drives transcription elongation

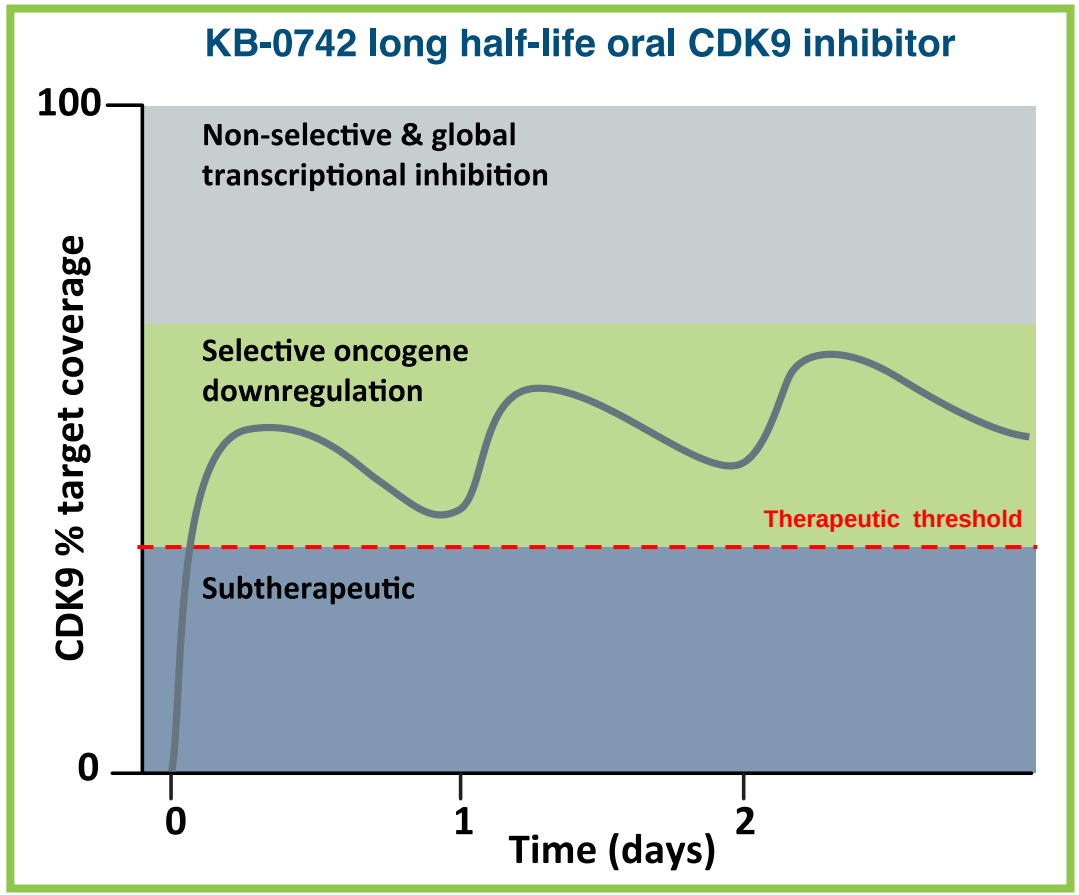


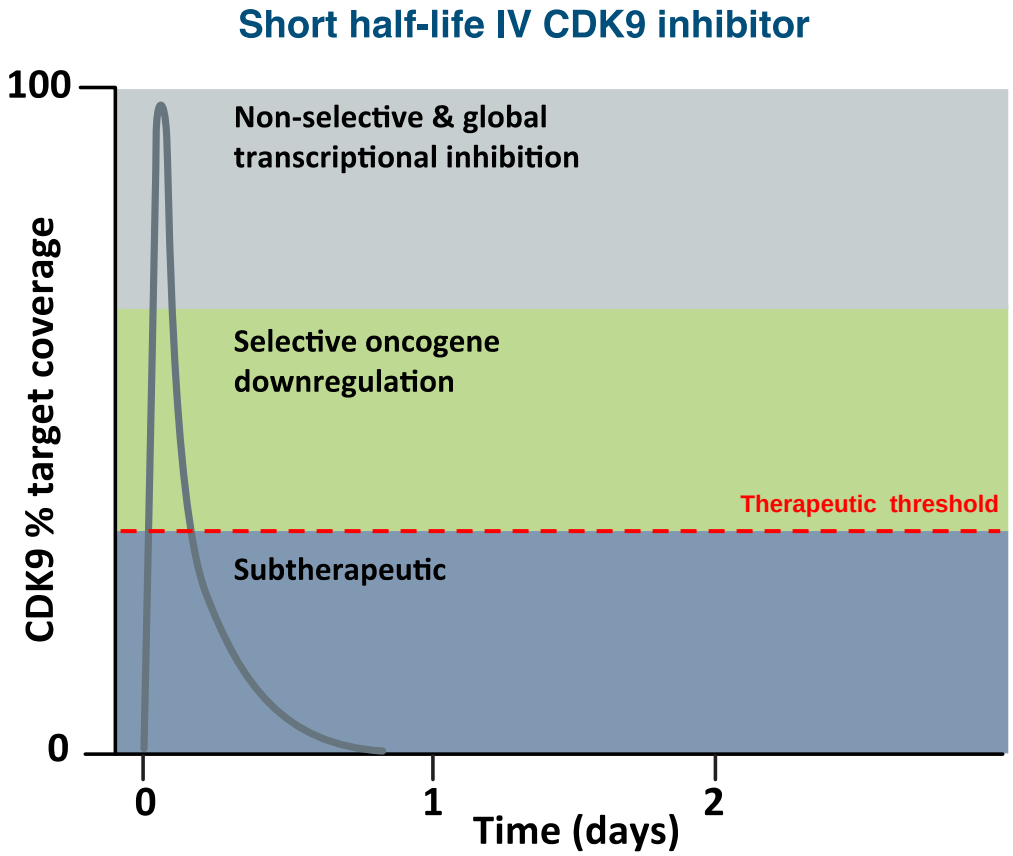
Oncogene transcription is disproportionately dependent on CDK9 as compared to transcription of essential housekeeping genes

SMM: small molecule microarray. TF: transcription factor.

Sources: Richters, A et al. Modulating Androgen Receptor-Driven Transcription in Prostate Cancer with Selective CDK9 Inhibitors. Cell Chem. Biol. 2020, 28, 1-14; Freeman, D. B et. al., Discovery of KB-0742, a Potent, Selective, Orally Bioavailable Small Molecule Inhibitor of CDK9 for MYC-Dependent Cancers, J. Med. Chem. 2023, 66, 23, 15629–15647

KB-0742's long plasma half-life and kinase selectivity provide a differentiated profile that avoids non-selective transcriptional inhibition



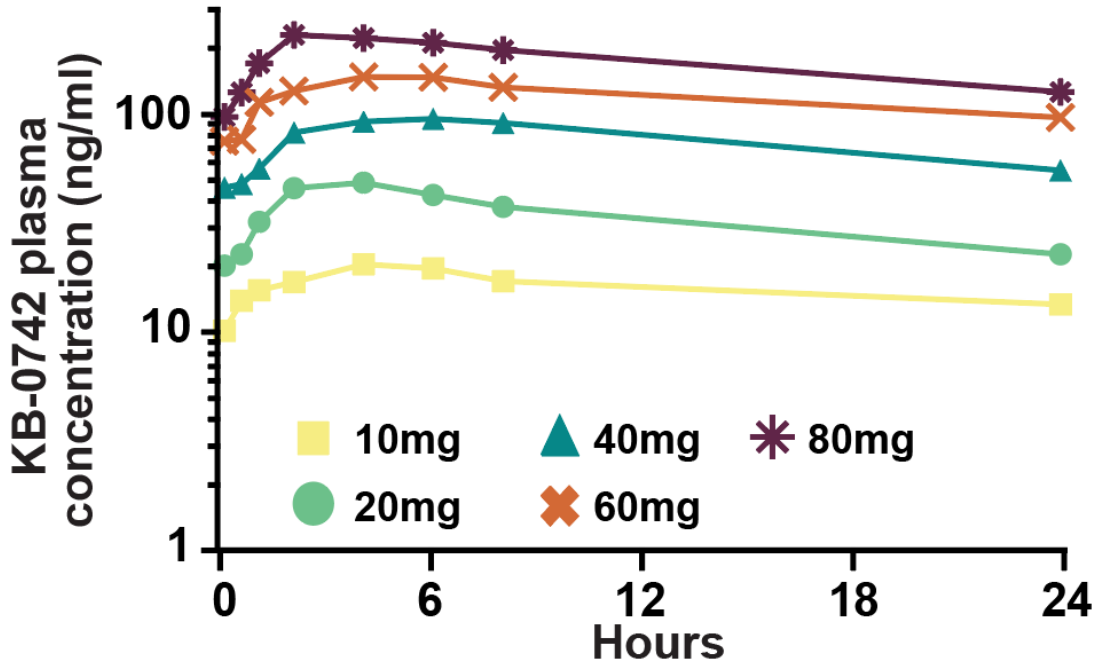


Figures for illustrative purposes only

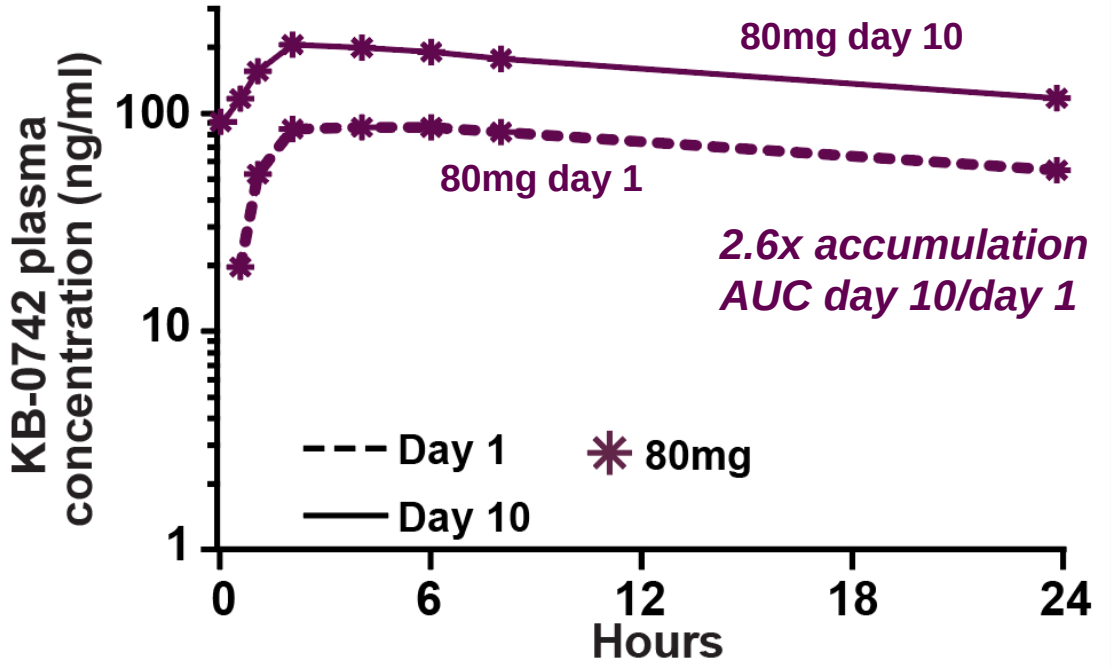
Our hypothesis: Time above therapeutic threshold drives efficacy while increased Cmax impacts safety/tolerability

KB-0742's pharmacokinetic profile is suitable for achieving sustained partial inhibition of CDK9

KB-0742 plasma concentrations at day 10 of dosing across the 10mg to 80mg cohorts



KB-0742 plasma half-life is approximately 24 hours, leading to accumulation



AUC: area under curve.

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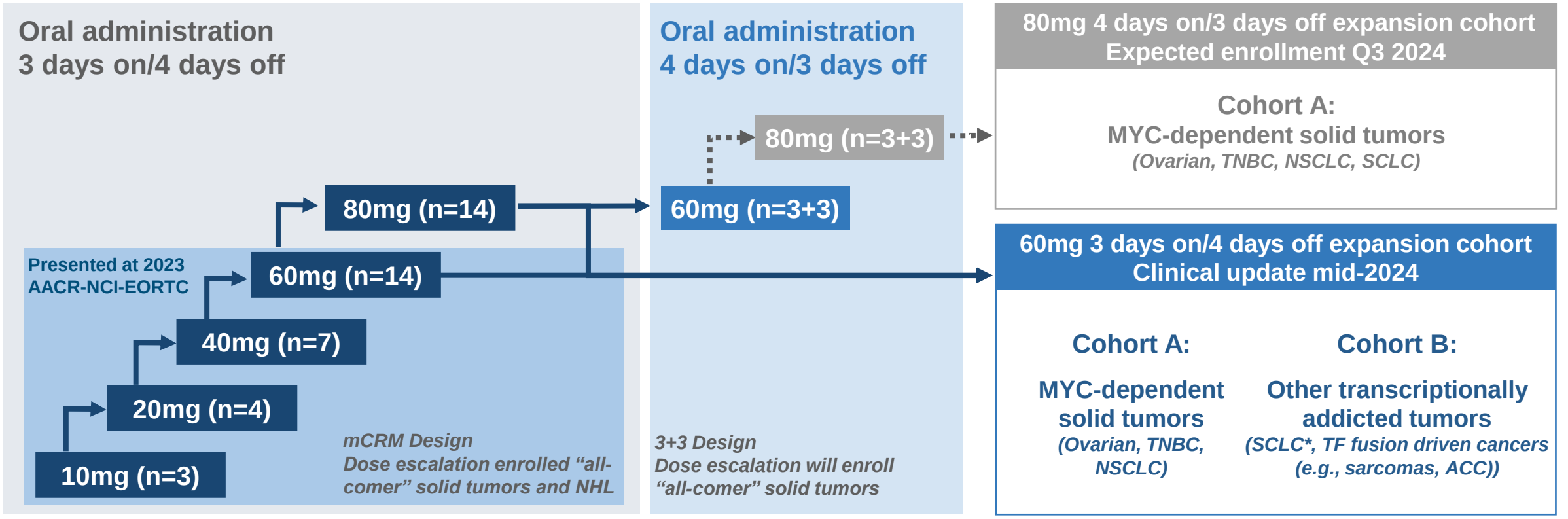
Source: A First-In-Human Study of CDK9 Inhibitor KB-0742 Demonstrates Evidence of Tolerability and Clinical Activity [Poster Presentation]. 2023 EORTC-NCI-AACR (Villalona-Calero, Miguel, et al.), Boston and 2023 CTOS, Dublin (Van Tine, Brian, et al.)

Positive data up to 60mg 3 days on/4 days off presented at AACR-NCI-EORTC 2023

Phase 1/2, first-in-human, open-label dose escalation and expansion study of KB-0742 in patients with relapsed or refractory solid tumors or NHL



ACC: adenoid cystic carcinoma. mCRM: modified continual reassessment method; NHL: Non-Hodgkin lymphoma; NSCLC: non-small cell lung cancer. SCLC: small cell lung cancer. TF: transcription factor. TNBC: triple negative breast cancer.

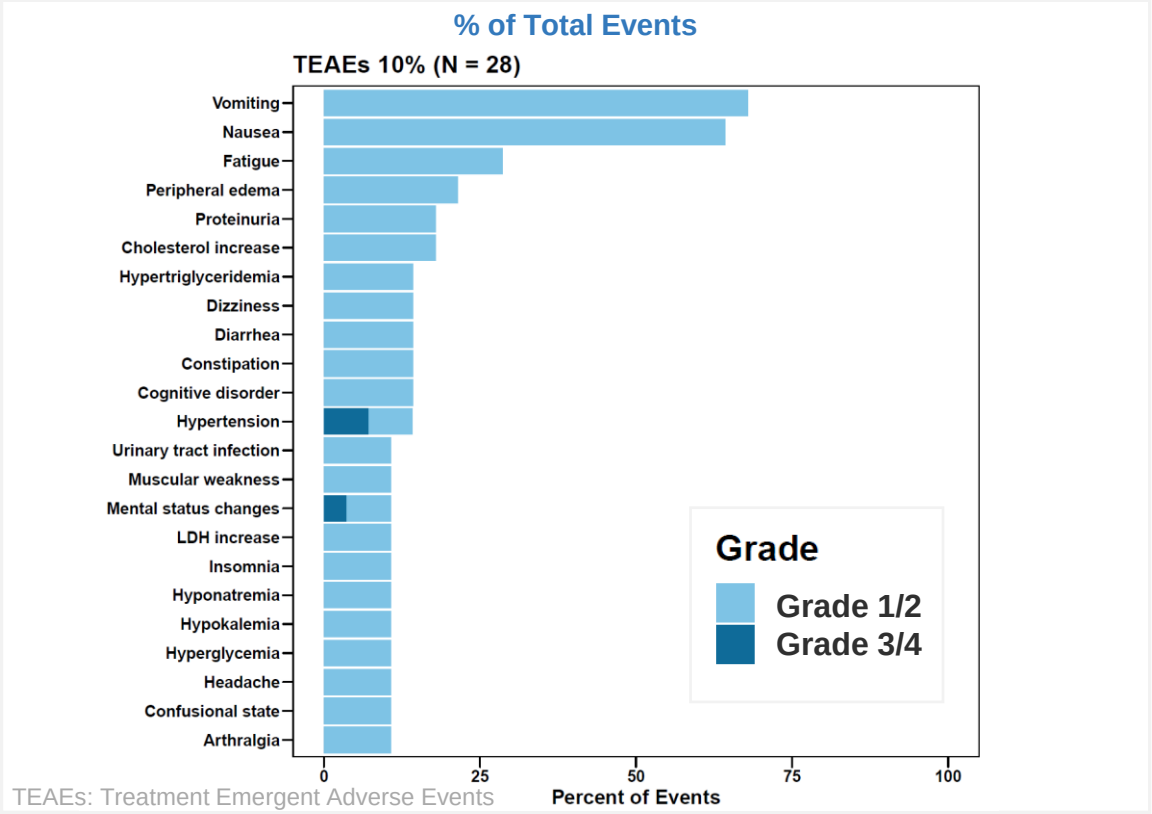
Ongoing enrollment

Future enrollment

**SCLC is also MYC-dependent*

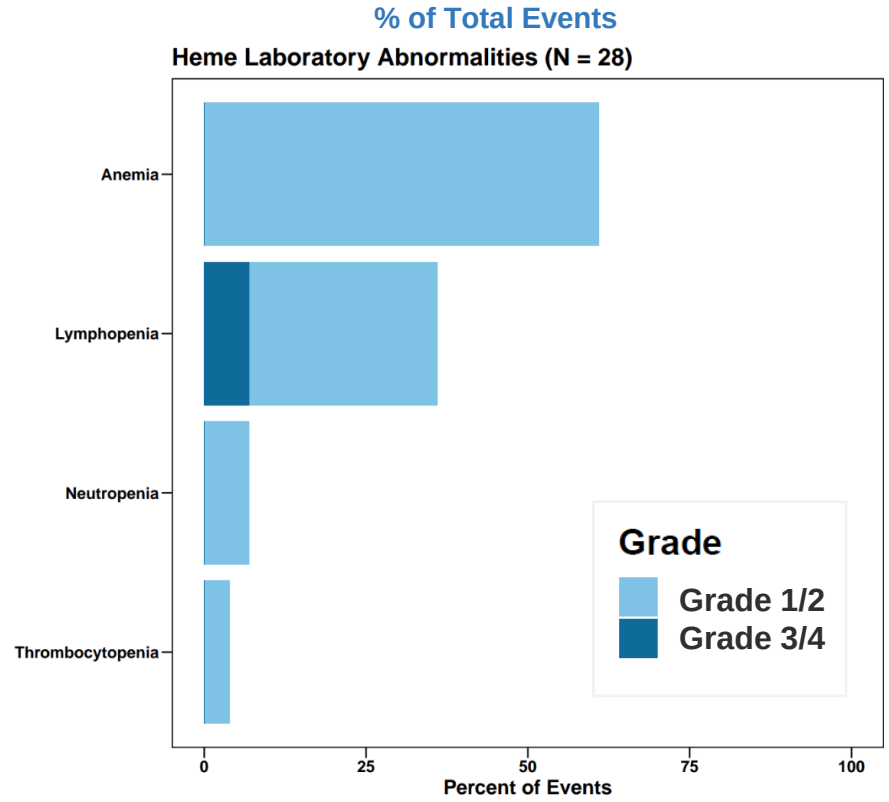
KB-0742 exhibits an acceptable safety profile with limited hematologic abnormalities at 60mg 3 days on/4 days off

Most common TEAES (occurring in ≥ 10% of patients)



TEAEs: Treatment Emergent Adverse Events

Hematologic laboratory abnormalities (based on NCI CTCAE grading)



Patients enrolled to date in the 80mg 3 on/4 off dose escalation cohort exhibit a similar safety profile

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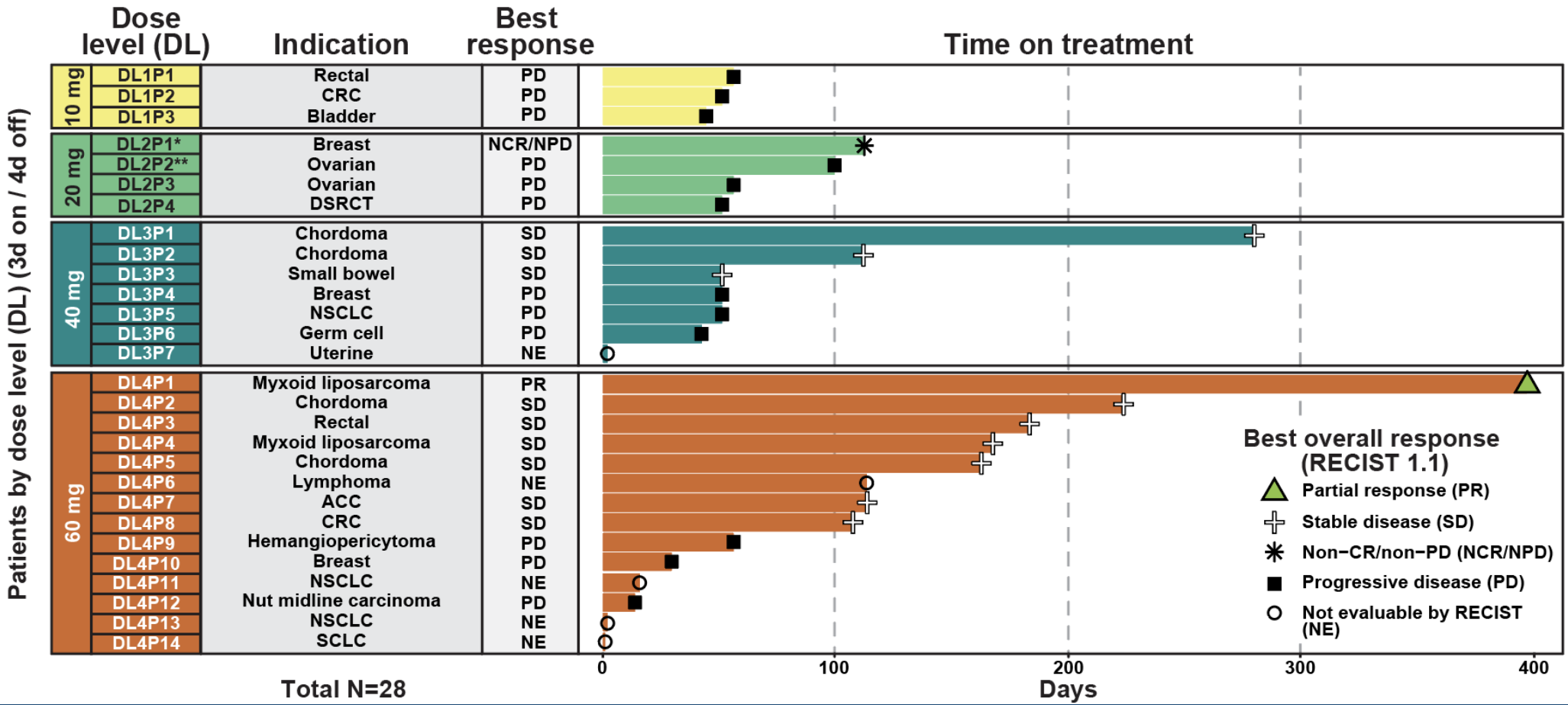
Source: A First-In-Human Study of CDK9 Inhibitor KB-0742 Demonstrates Evidence of Tolerability and Clinical Activity [Poster Presentation]. 2023 EORTC-NCI-AACR (Villalona-Calero, Miguel, et al.), Boston and 2023 CTOS, Dublin (Van Tine, Brian, et al.)

KB-0742 duration of treatment across dose levels
(as of September 1, 2023)

*Patient DL2P1 response NCR/NPD was due to the patient having evaluable but not measurable disease at baseline.

**Patient DL2P2 progressed at an earlier date and stayed on treatment post-progression.



At the 60mg dose, the median duration on study was 111 days

ACC: Adenoid cystic carcinoma; CRC: Colorectal cancer; DSRCT: Desmoplastic small round cell tumors; NSCLC: Non-small cell lung cancer. SCLC: small cell lung cancer.

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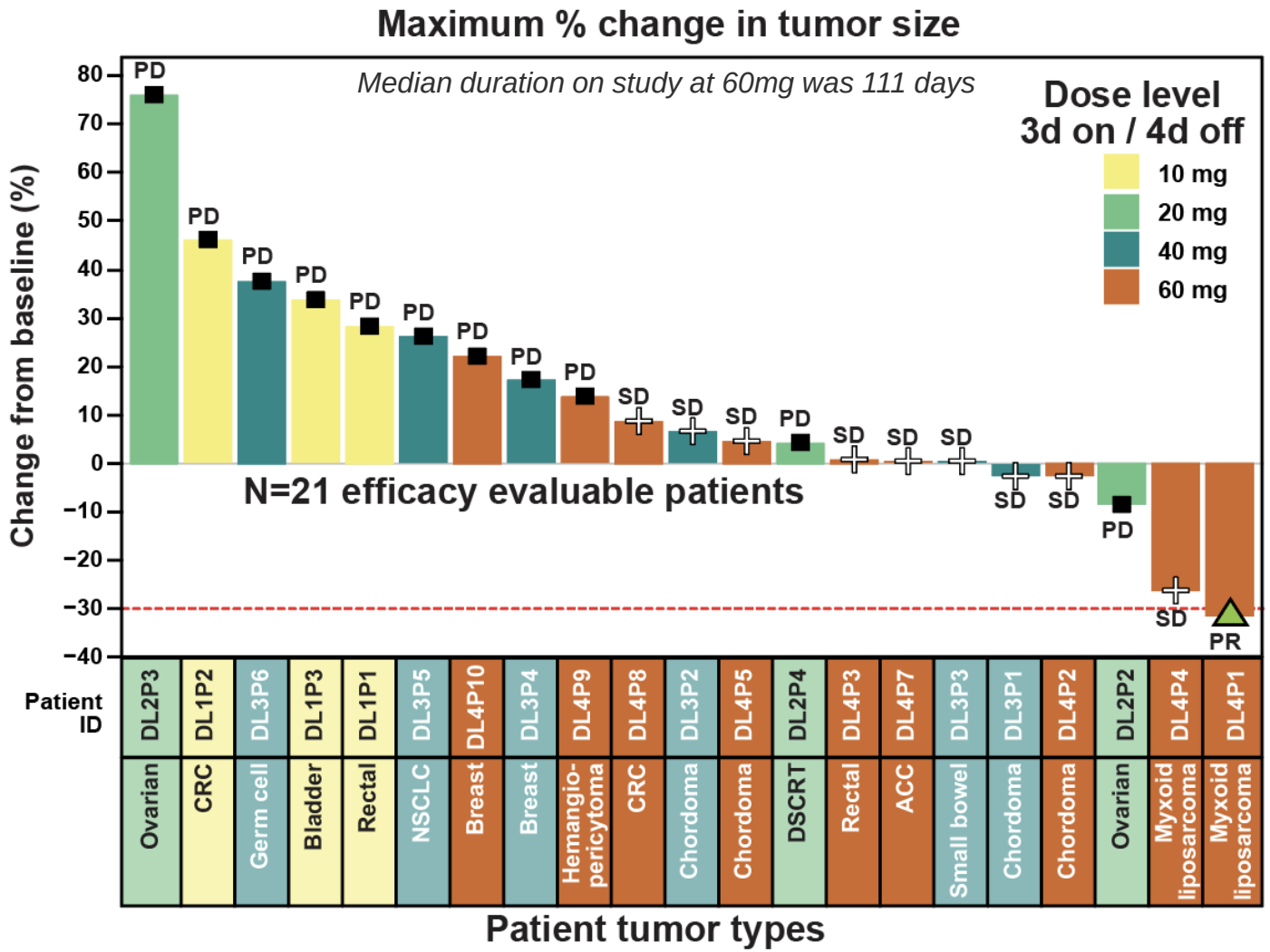
Source: A First-In-Human Study of CDK9 Inhibitor KB-0742 Demonstrates Evidence of Tolerability and Clinical Activity [Poster Presentation]. 2023 EORTC-NCI-AACR (Villalona-Calero, Miguel, et al.), Boston and 2023 CTOS, Dublin (Van Tine, Brian, et al.)

KB-0742 anti-tumor activity: objective regressions in two transcription factor (TF) fusion-driven tumor patients (as of September 1, 2023)



Tumors with TF fusions

Example tumor type	TF#1	TF#2
Ewing sarcoma	EWSR1	FLI1
	FUS	ERG
Myxoid liposarcoma	DDIT3	FUS
Adenoid cystic carcinoma	MYB	NFIB
Alveolar rhabdomyosarcoma	PAX3	FOXO1
	PAX7	FOXO1

- One partial response lasting 113 days in a 7th line myxoid liposarcoma patient. Second patient achieved 26% reduction in tumor diameters.
- 9 (43%) patients had stable disease (SD) as the best response.
- Overall disease control rate was 47.8% - defined as a CR (complete response), partial response (PR), or stable disease (SD).

ACC: Adenoid cystic carcinoma; CRC: Colorectal cancer; NSCLC: Non-small cell lung cancer.

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Source: A First-In-Human Study of CDK9 Inhibitor KB-0742 Demonstrates Evidence of Tolerability and Clinical Activity [Poster Presentation]. 2023 EORTC-NCI-AACR (Villalona-Calero, Miguel, et al.), Boston and 2023 CTOS, Dublin (Van Tine, Brian, et al.)

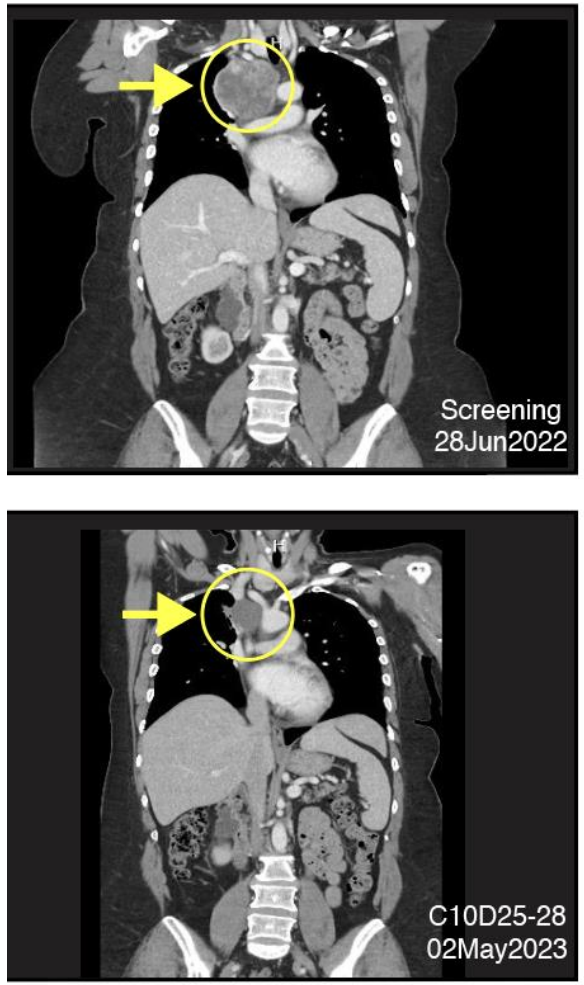
Patient DL4P1 with myxoid liposarcoma achieved a partial response (PR) at cycle 10 which lasted for 113 days

Patient characteristics and treatment history

- 50-year-old female
- Diagnosed with myxoid liposarcoma in May 2009
- Stage 4 at enrollment
- Six prior lines of therapy and best overall response included:
  - Adriamycin/Ifosfamide: April-September 2015 (PD)
  - Atezolizumab: July-September 2016 (PD)
  - Trabectedin: December 2016-January 2017 (PD)
  - NY-ESO-1C259 T: September 2017-June 2018 (SD)
  - Atezolizumab: November 2018-June 2019 (SD)
  - Ifosfamide: December 2021-January 2022 (SD)

KB-0742 treatment course

- KB-0742 treatment initiated in July 2022
- 60mg 3 on/4 off for 398 days on treatment
- PR achieved at cycle 10 lasting 113 days



Reference: https://ascopubs.org/doi/abs/10.1200/JCO.2018.36.15_suppl.3005

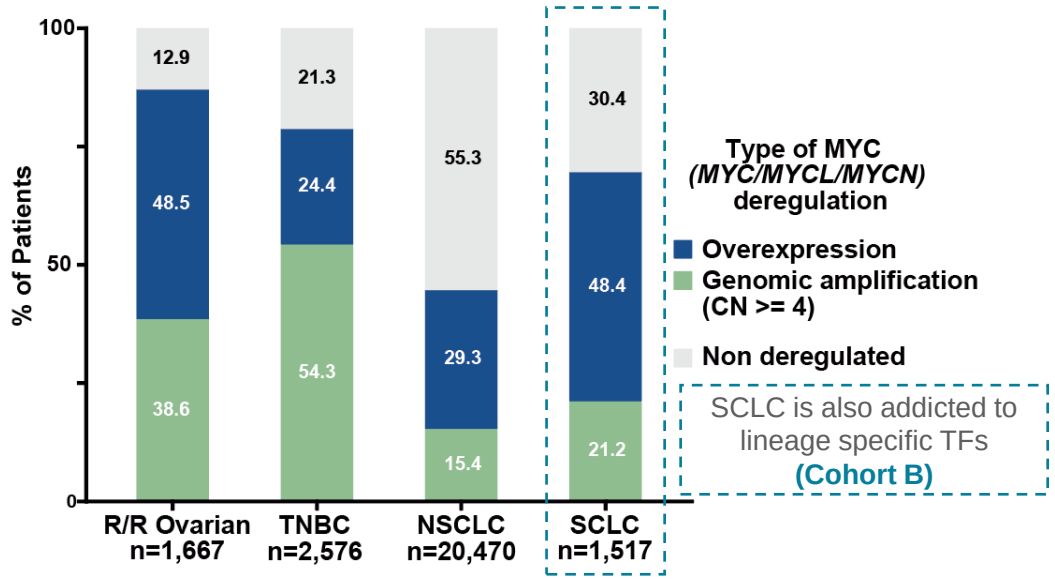
Brian Van Tine, M.D., Ph.D.

PR: partial response. SD: stable disease.

MYC-dependent (cohort A) and TF fusion-driven (cohort B) addicted tumor types included in expansion cohorts

Cohort A: Oncogenic TF = MYC

Tumors with high prevalence of MYC overexpression or amplification[1]



Est. Drug Treatable ≥2L U.S. Patients >100,000[2]

Cohort B:

Oncogenic TF = Fusion TF (TF#1 / TF#2)

Oncogenic TF = Lineage specific

Example tumor type	TF#1	TF#2
Ewing sarcoma	EWSR1	FLI1
	FUS	ERG
Myxoid liposarcoma	DDIT3	FUS
Adenoid cystic carcinoma	MYB	NFIB
Alveolar rhabdomyosarcoma	PAX3	FOXO1
	PAX7	FOXO1

Example tumor type	Example lineage TF
Small Cell Lung Cancer*	NEUROD1
	ASCL1
	POU2F3
	YAP1

Est. Drug Treatable U.S. Patients >50,000[2,3,4,5]

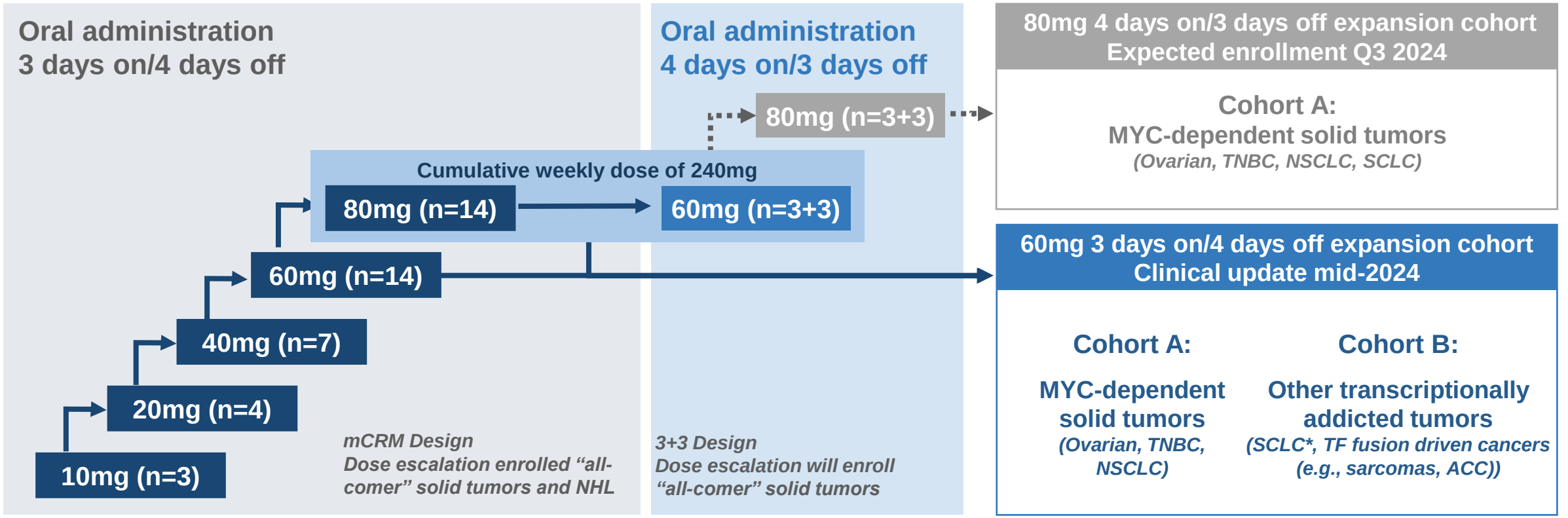
NSCLC: Non-small cell lung cancer. R/R: relapsed / refractory. SCLC: small cell lung cancer. TF: transcription factor. TNBC: triple negative breast cancer.

**SCLC is also MYC-dependent*

Source: 1. Villalona-Calero, Miguel, et al. EORTC-NCI-AACR 2023 [Poster]. 2. Decision Resources Group 2022, 3. Huang et al. Scientific Reports 2023; 4. Gage et al. Oncotarget (2019) 5. Togashi et al. Modern Pathology (2018).

Expansion data at 80mg 4 days on/3 days off dose and schedule expected 1H 2025

Phase 1/2, first-in-human, open-label dose escalation and expansion study of KB-0742 in patients with relapsed or refractory solid tumors or NHL



ACC: adenoid cystic carcinoma. mCRM: modified continual reassessment method; NHL: Non-Hodgkin lymphoma; NSCLC: non-small cell lung cancer. SCLC: small cell lung cancer. TF: transcription factor. TNBC: triple negative breast cancer.

Ongoing enrollment

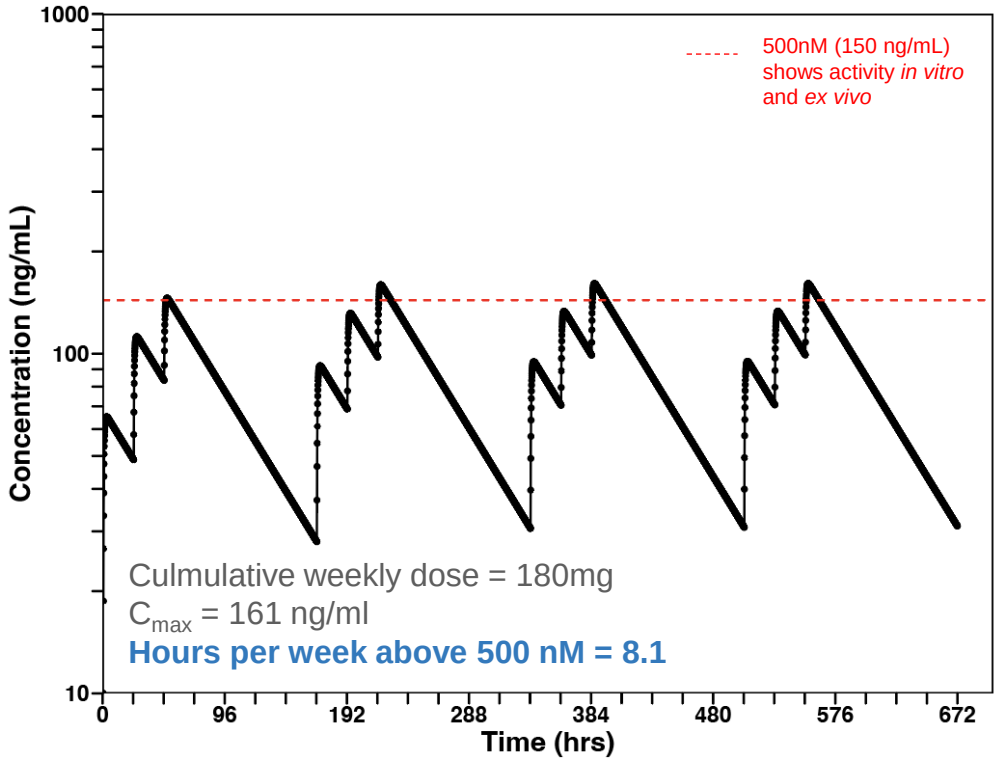
Future enrollment

**SCLC is also MYC-dependent*

80mg 4 on/3 off dose schedule results in ~10x time above efficacy threshold of established clinically active dose of 60mg 3 days on/4 days off

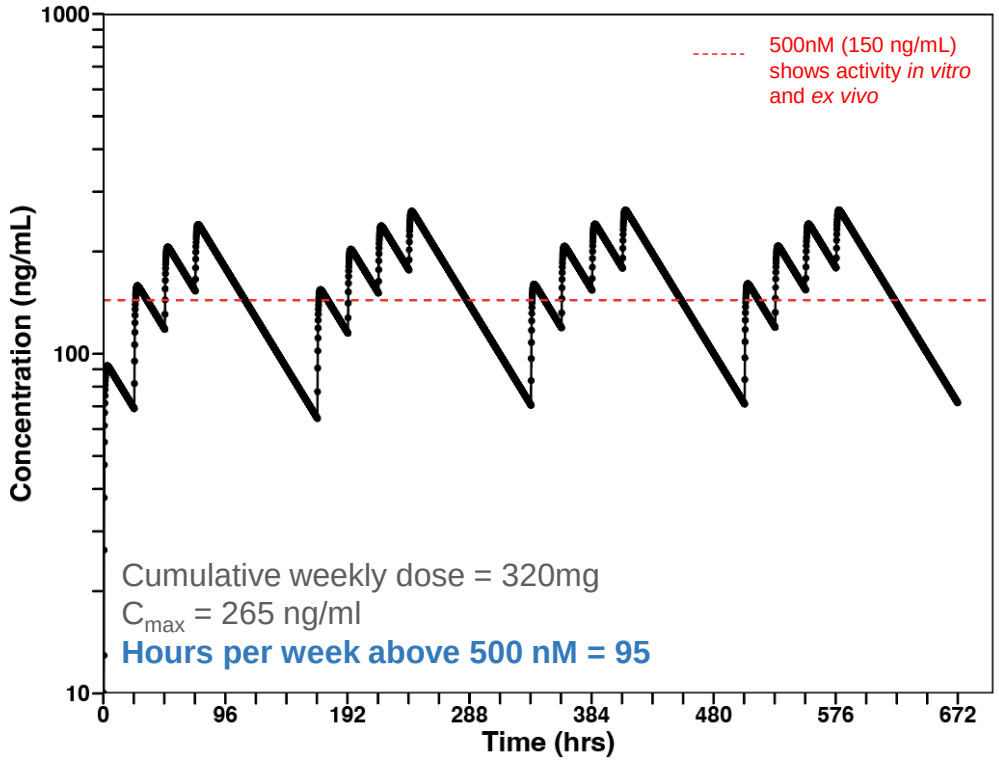
Dose at which we have currently seen activity

60mg (3 days on/4 days off) / week

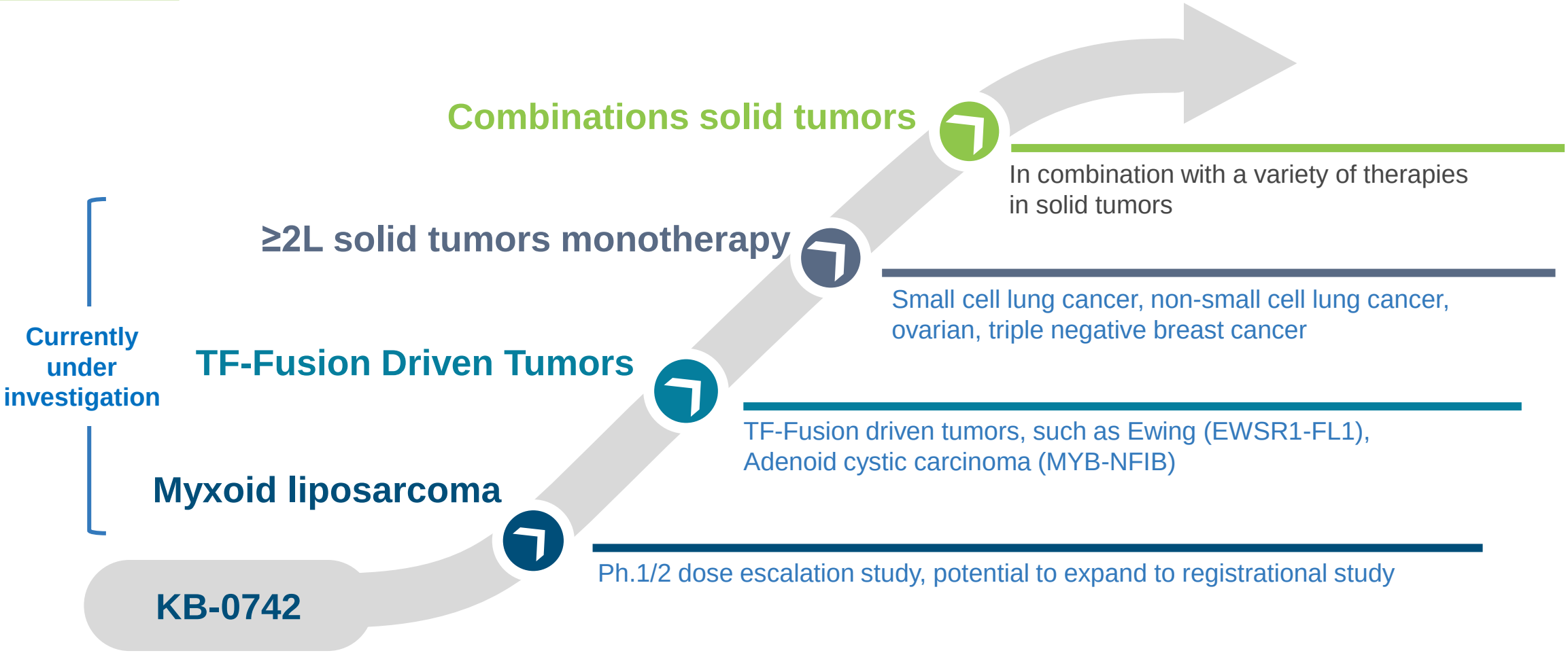


Dose at which we expect to enroll expansion cohort

80mg (4 days on/3 days off) / week



KB-0742 may have broad utility in transcriptionally addicted cancers, both as monotherapy and in combination



KB-0742 potentially addresses >30% of solid tumors

TF: transcription factor.

KB-0742 has the potential to show increased efficacy in transcriptionally addicted solid tumors with multiple upcoming data readouts

- Preliminary on-mechanism activity seen at 60mg 3 on/4 off dose
- Acceptable safety profile observed through 80mg 3 on/4 off dose

Update on clinical data to be presented in mid-2024

- Enrollment of expansion cohort at extended dosing schedule of 80mg 4 on/3 off expected to start in Q3 2024
- Potential to establish monotherapy activity to enable future monotherapy or combination studies across multiple solid tumor indications

KB-0742 expansion cohort data at 80mg 4 on/3 off dose and schedule expected 1H 2025

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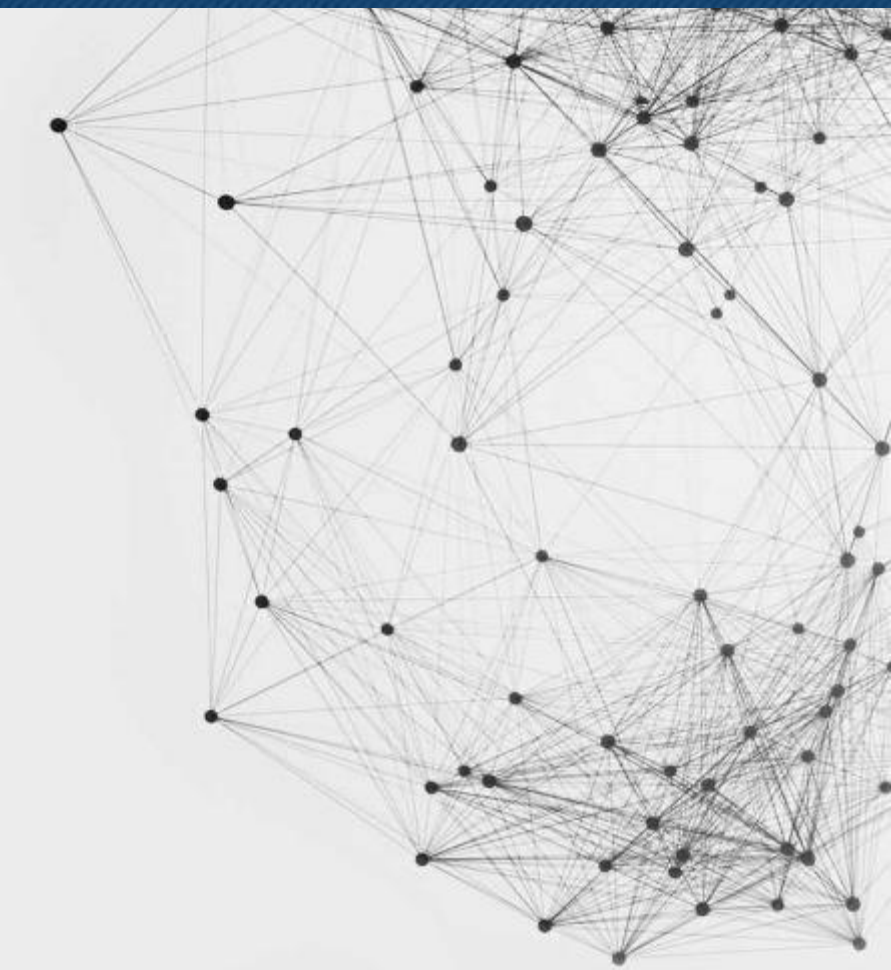

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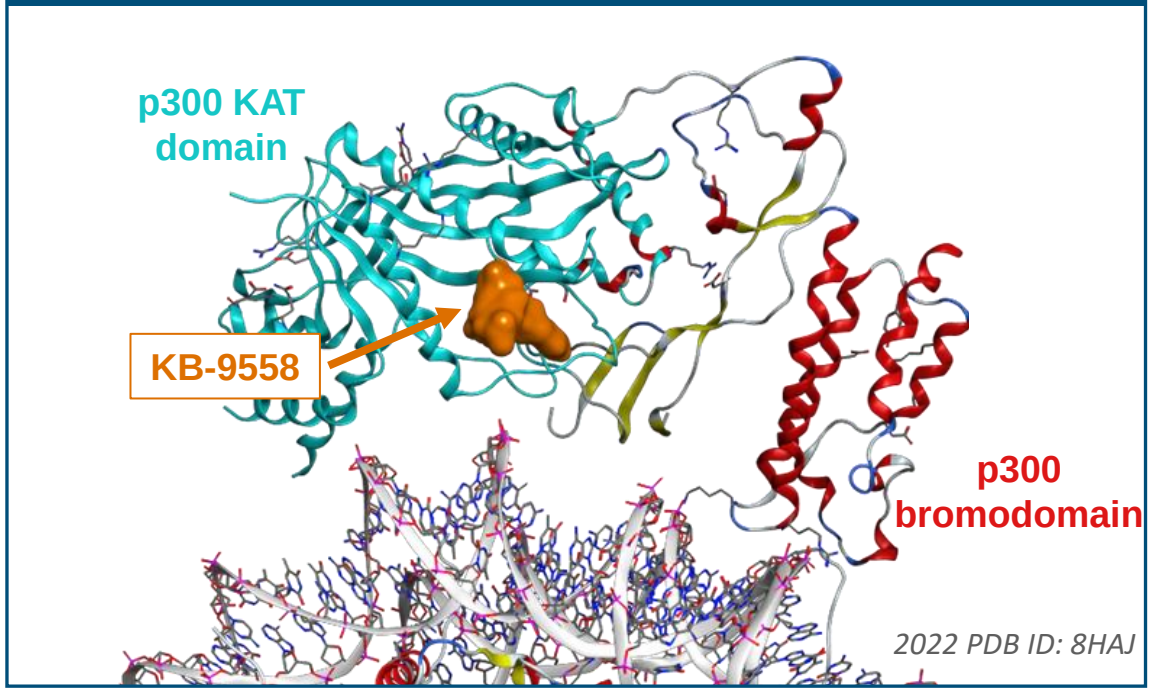
KB-9558 is a p300 KAT inhibitor that downregulates interferon regulatory factor 4 (IRF4) transcription regulatory network (TRN), a key driver of multiple myeloma

- Interferon regulatory factor 4 (IRF4) is a master TF deregulated in multiple myeloma
- p300 is a critical node of the IRF4 TRN, and KAT domain inhibition selectively targets IRF4 TRN
- KB-9558 is a development candidate currently in IND-enabling studies that potently and selectively inhibits the p300/CBP KAT domains
- KB-9558 exhibits single agent activity *in vivo* and in patient derived samples *ex vivo* (including relapsed/refractory patients)
- Strong potential clinical and market opportunity in relapsed/refractory multiple myeloma

KB-9558 binds to the KAT domain of p300

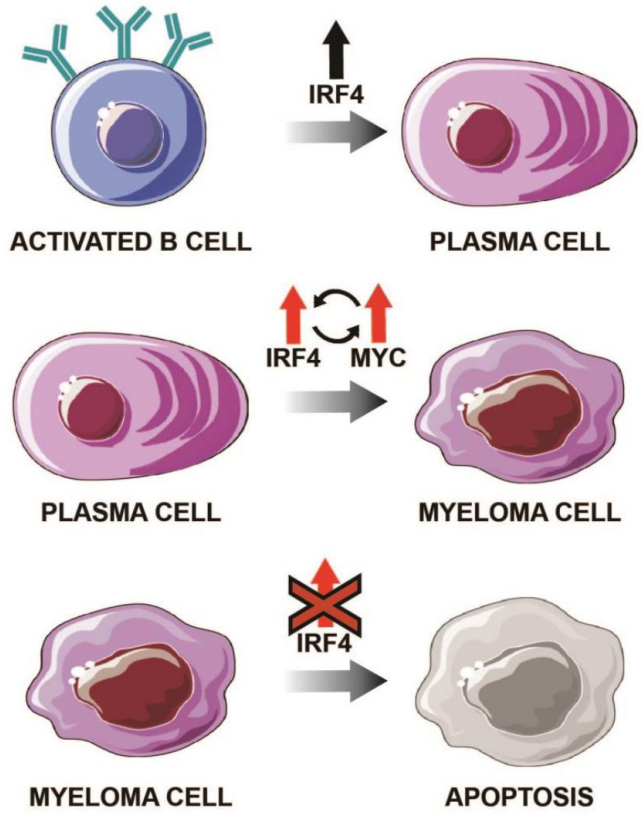


p300 KAT inhibitor more potently downregulates IRF4 compared to either p300 bromodomain inhibitors or IMiDs

CBP:CREB-binding protein. IMiD: immunomodulatory drugs. IRF4: interferon regulatory factor 4. KAT: lysine acetyltransferase. TRN: Transcription regulatory network.

IRF4 is the key target in multiple myeloma

Myeloma cells are addicted to high levels of IRF4 function



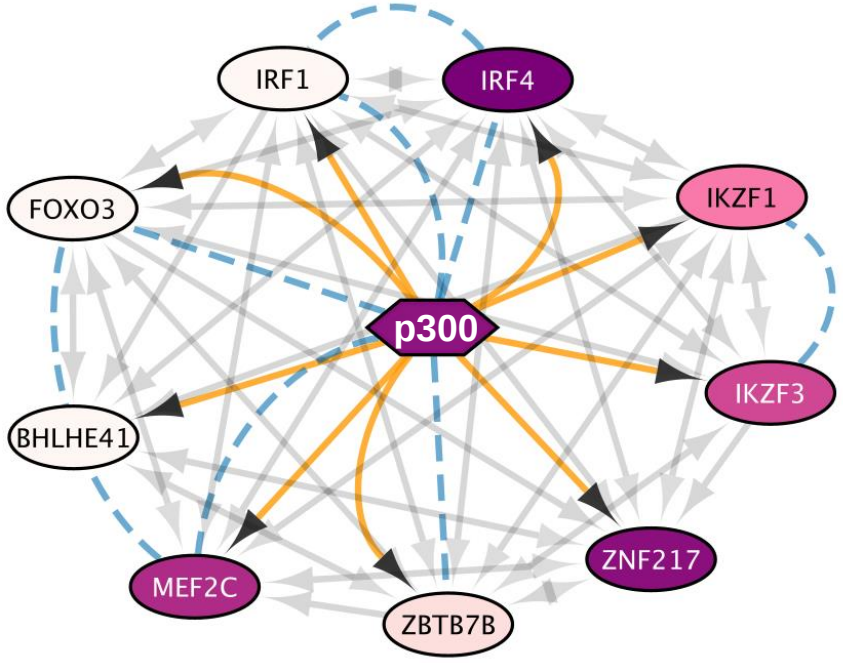
Agnarelli, 2018

- IRF4 is a master transcription factor that defines plasma cell identity
- IRF4 plays a similar role in multiple myeloma as AR does in prostate cancer
- In multiple myeloma, IRF4 is deregulated in an oncogenic feedback loop with MYC
- Targeting IRF4 is lethal to multiple myeloma cells regardless of therapy resistance and is orthogonal to other drivers such as IKAROS (target of IMiDs)

AR: androgen receptor. IMiDs; immunomodulatory drugs. IRF4: interferon regulatory factor 4.

Our TRN mapping identified p300 as a critical node of IRF4

p300 is central to the multiple myeloma (MM) TRN



% of Multiple Myeloma DepMap screens demonstrating a dependency

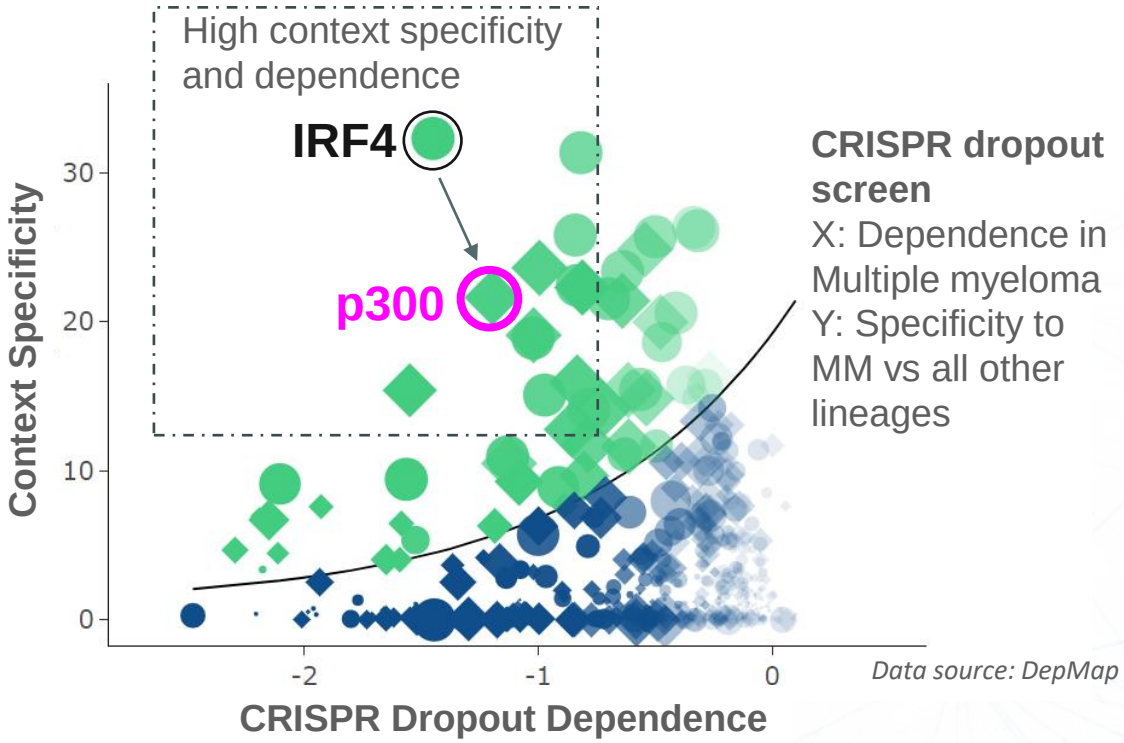
0% 100%

Regulatory

Physical

p300 Direct binding

p300 is the nearest druggable node to IRF4 in MM



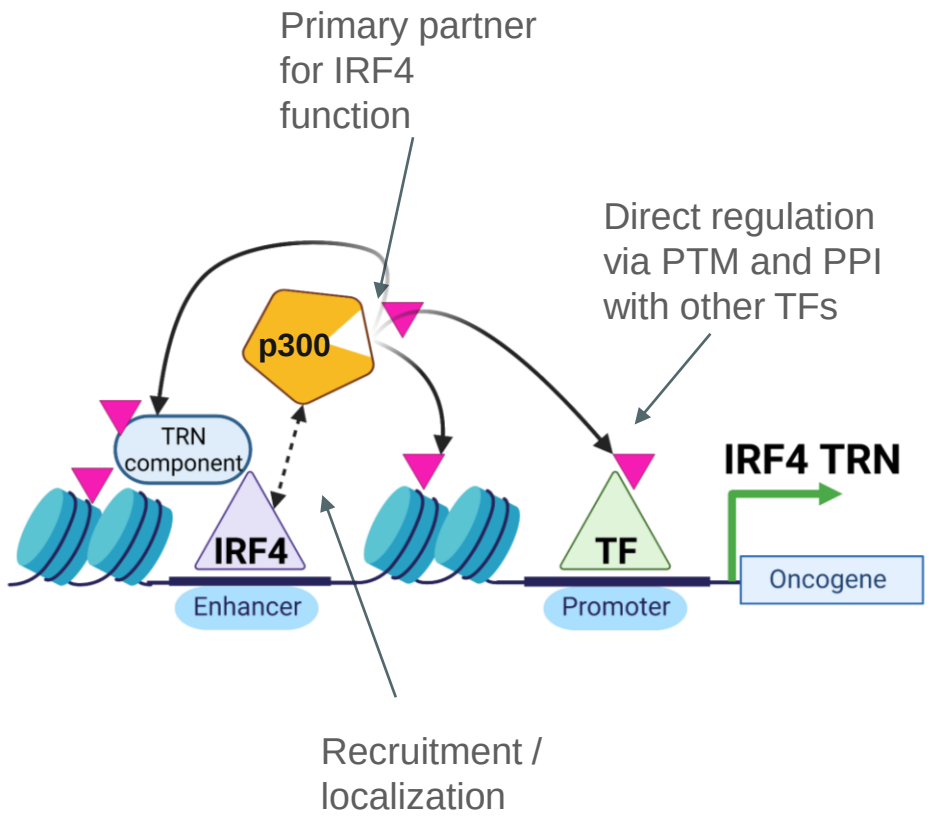
CRISPR dropout screen
X: Dependence in Multiple myeloma
Y: Specificity to MM vs all other lineages

TFs Cofactors Increasing effect size

IRF4: interferon regulatory factor 4. TF: transcription factor. TRN: transcription regulatory network.

p300 KAT inhibition selectively targets IRF4 activity in multiple myeloma

p300 is a critical node cofactor of IRF4

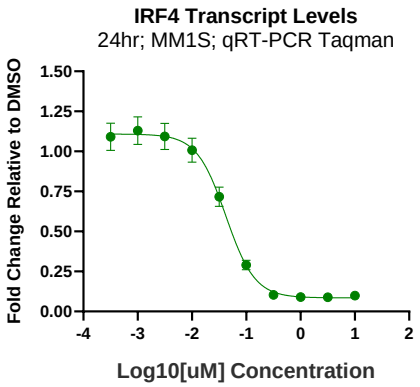


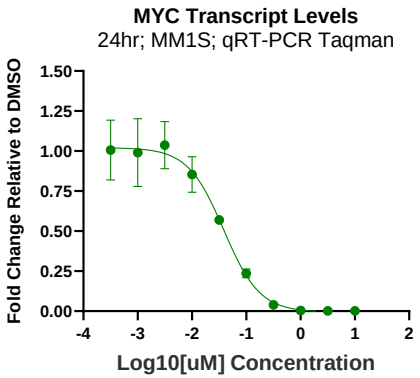
- p300 directly interacts with IRF4 and colocalizes across the genome
- p300 is recruited by IRF4 to regulate its target genes, including IRF4 itself
- p300 acetylates chromatin and other transcription factors at IRF4 binding sites
- Inhibition of p300 KAT domain leads to loss of IRF4 and its downstream activity

IRF4: interferon regulatory factor 4. KAT: lysine acetyltransferase. PTM: post-translational modifications. PPI: protein-protein interactions. TRN: transcription regulatory network.

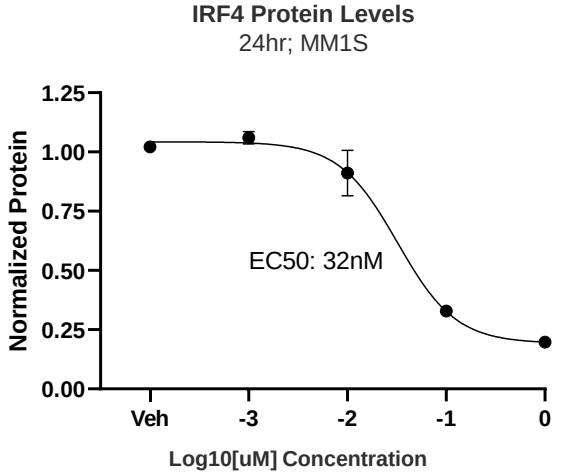
p300 KAT inhibition leads to IRF4 TRN suppression and apoptosis in multiple myeloma

p300 KAT inhibition leads to loss of IRF4 and MYC expression…



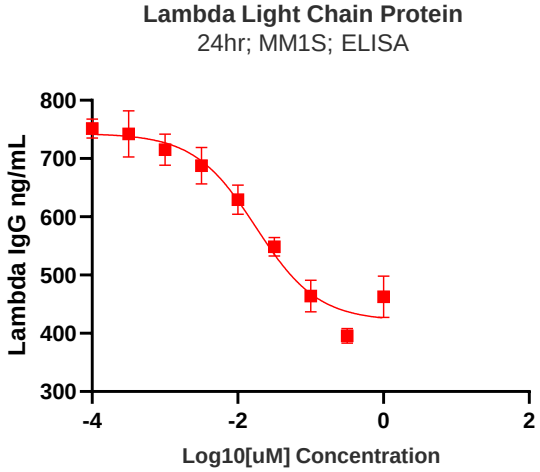


…and loss of IRF4 protein level…



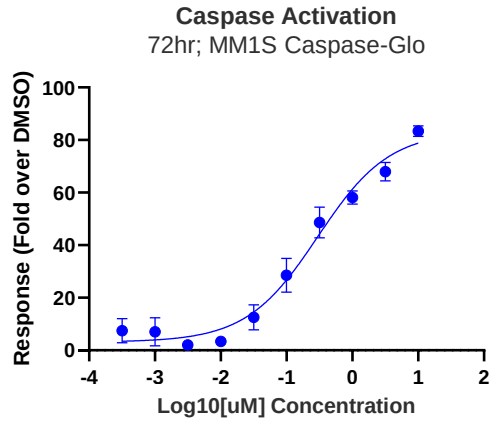
- Robust downregulation of IRF4 itself
- Collapses the oncogenic TRN

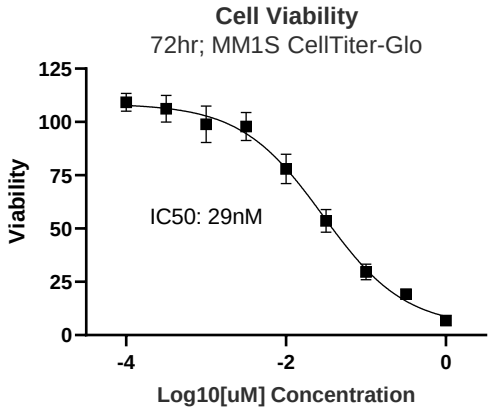
…causing loss of expression of IRF4 targets…



- Circulating IgG commonly assessed as a clinical biomarker
- Pairs with Kronos Bio established proximal and functional PD assays

…ending with cell death





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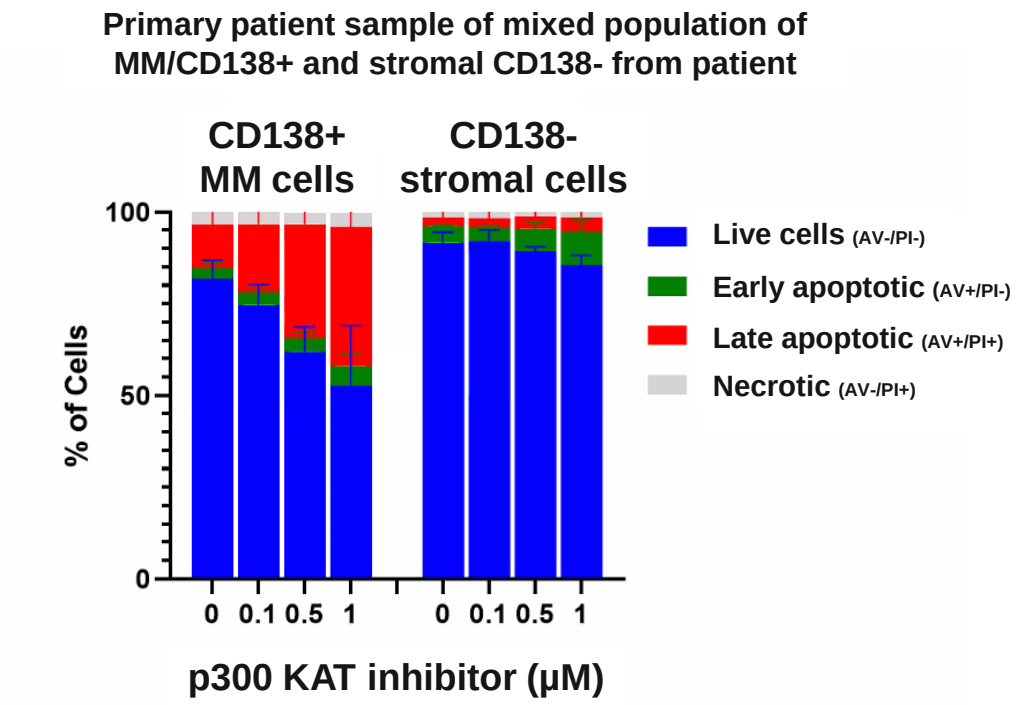
. IRF4: interferon regulatory factor 4. KAT: lysine acetyltransferase. TRN: Transcription regulatory network.

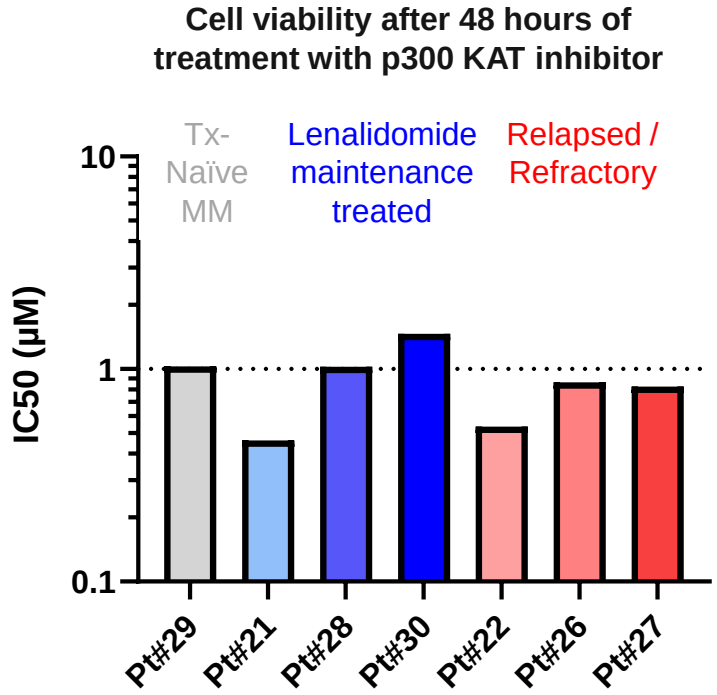
Our p300 KAT inhibitor shows selective activity on myeloma vs. stromal cells in relapsed/refractory patient samples *ex vivo*

In *ex vivo* patient samples, p300 KAT inhibitor shows anti-myeloma effects at doses that spare stroma cells



Assessed by flow cytometry using Annexin V (AV) / propidium iodide (PI)

p300 KAT inhibitor activity was similar across naïve, IMiD maintenance treated, and R/R MM patient lines



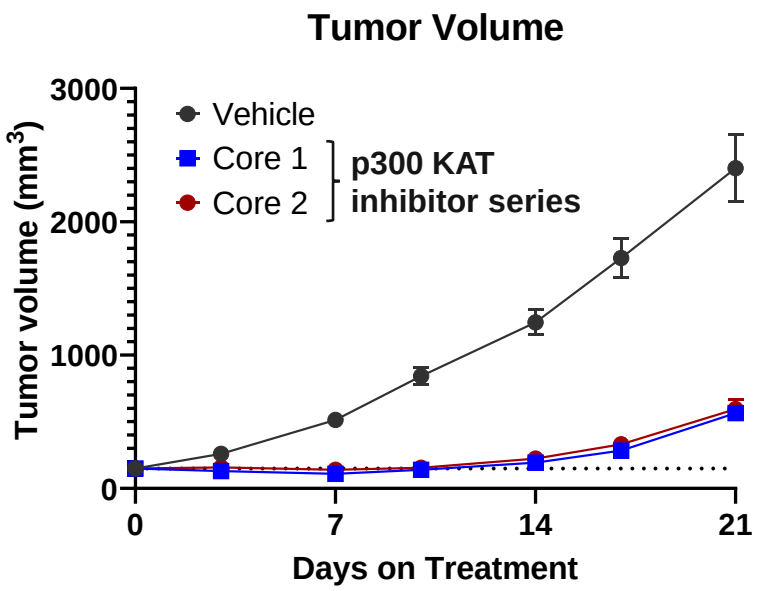
Assessed by flow cytometry

In collaboration with Mariateresa Fulciniti and Nikhil Munshi, Dana-Farber Cancer Institute

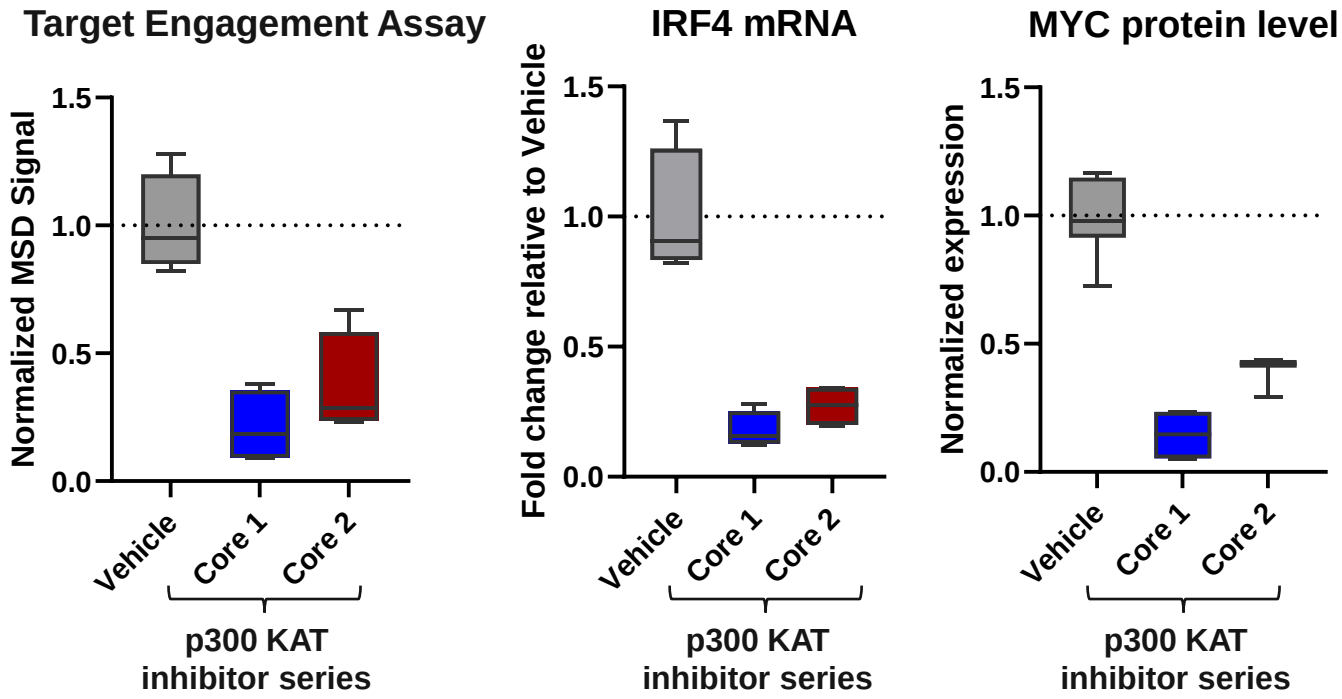
IMiD: immunomodulatory drugs. KAT: lysine acetyltransferase. MM: multiple myeloma. R/R: relapsed / refractory.

Our p300 KAT inhibitor series exhibits strong anti-tumor activity

p300 KAT inhibitor shows tumor reduction with >80% TGI in MM1S

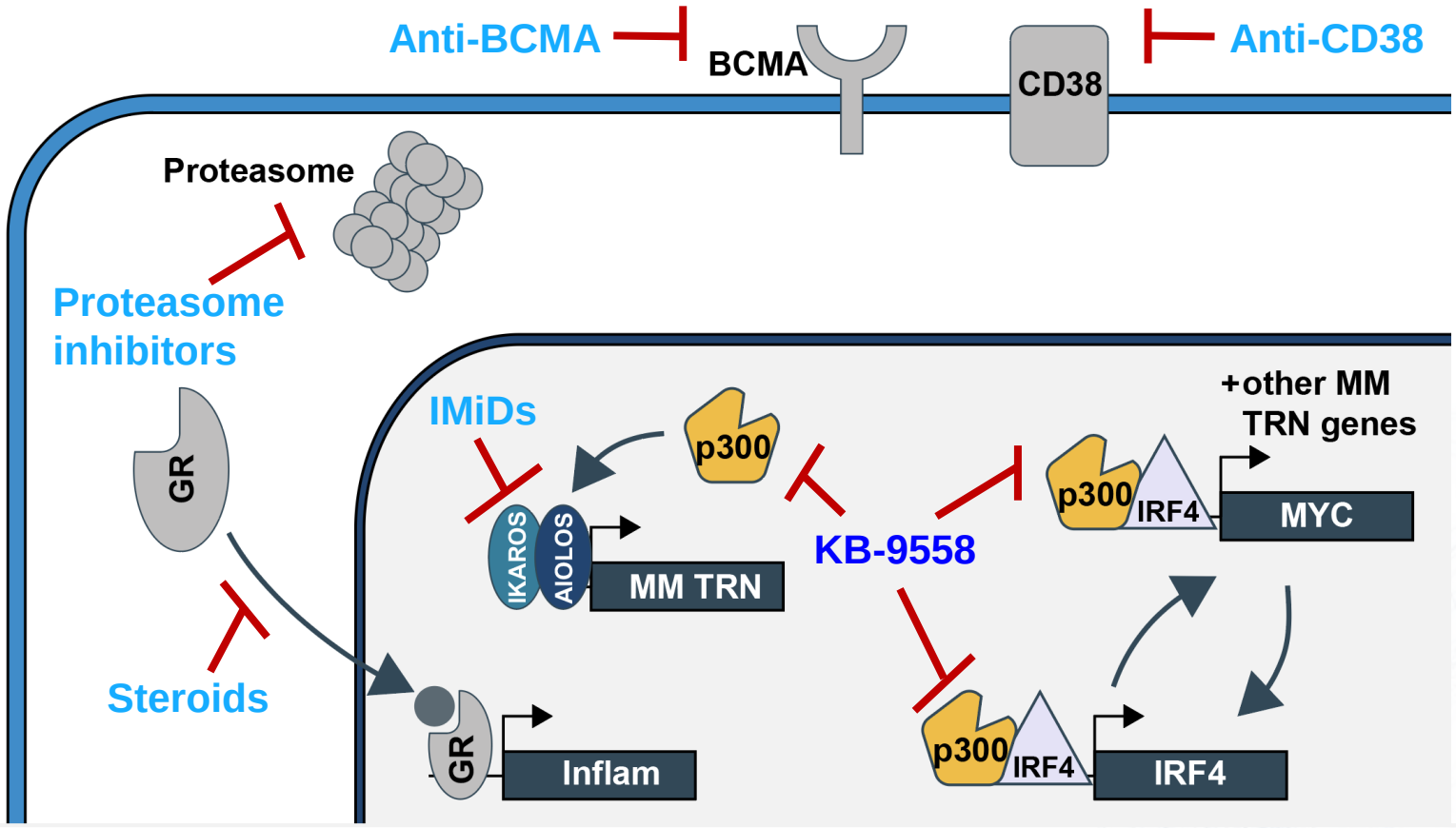


Anti-tumor activities of p300 KAT inhibitors linked to modulation of multiple pharmacodynamic markers



KAT: lysine acetyltransferase. MM1S:A commonly used cell line to evaluate novel therapies for multiple myeloma; TGI: tumor growth inhibition.

KB-9558 inhibits multiple myeloma drivers that are distinct and orthogonal to existing therapeutic targets



KB-9558 has the potential to be used either as single agent or in combination

BCMA: B-cell maturation antigen. CD38: cluster of differentiation 38. IMiD: immunomodulatory drugs.

KB-9558 p300 KAT inhibitor of IRF4 TRN is well positioned for relapsed/refractory multiple myeloma

- Orally bioavailable, highly selective, p300 KAT inhibitor candidate for IRF4/MYC suppression
  - Targeting IRF4 is lethal to multiple myeloma cells regardless of therapy resistance
- Potential to address the high unmet need in relapse or refractory multiple myeloma with a novel mechanism of action
- Opportunity for monotherapy in patients refractory to existing therapies and combinations with approved therapies in earlier lines of treatment
- Additional opportunities in other indications

KB-9558 IND-enabling studies expected to be completed in 2024

IRF4: interferon regulatory factor 4. KAT: lysine acetyltransferase; TRN: transcription regulatory network

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01 Introduction

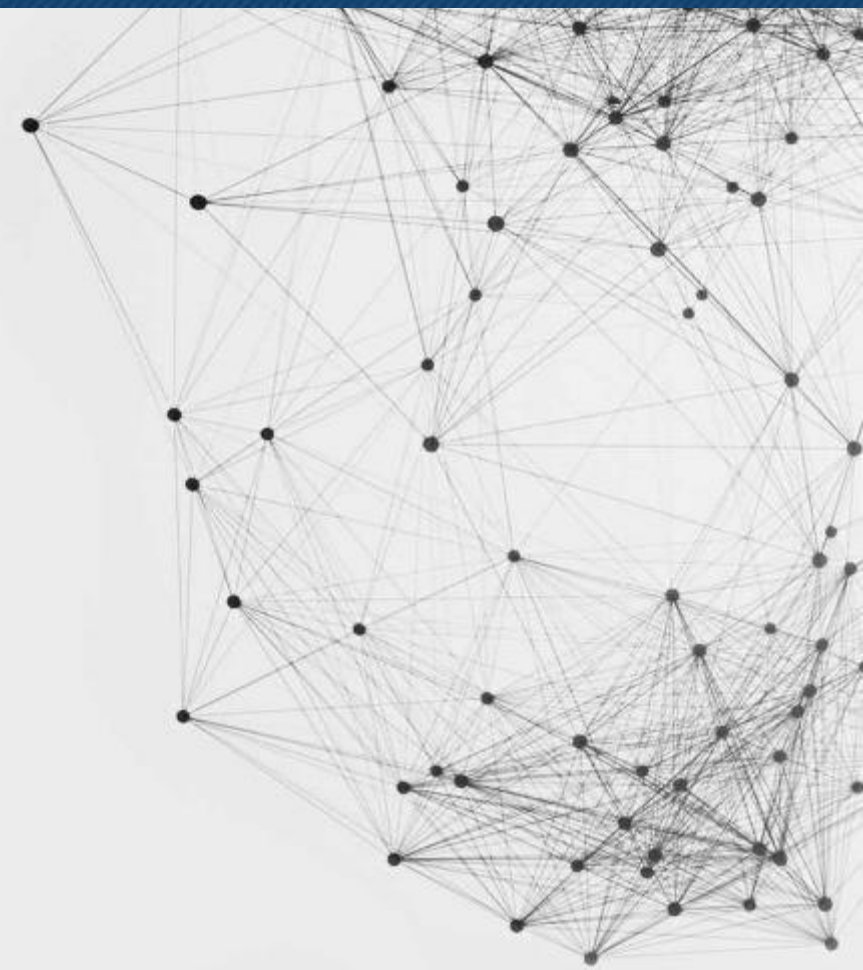
02 Our pipeline

- KB-0742 (CDK9 inhibitor)
- KB-9558 (p300 KAT inhibitor)

03 **Our product engine**

- **TRN mapping**
- **TRN screening**

04 Kronos Bio milestones and financials



Significant potential:
Only 7 of the 100+ TFs implicated in driving cancer have been drugged

APPROVED*

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

IN CLINICAL DEVELOPMENT

- IKZF2
- MYB
- MYC
- NOTCH
- PPAR
- NUT
- STAT5
- STAT3
- YAP1/TEAD
- TP53
- CTNNB1

- FDA NDA approval of compound drugging this TRN
- Approvals in oncology indications only
- Information validated in January 2024

UNDRUGGED

ASCL1	GATA2/3	NEUROD1	RUNX2
BCL6	GLI1	NF1	SMADs
C19ORF11	GLI2	NFKB	SNAI2
CEBPa/b	HAND1/2	NR2F2	SOX2
CRX	IRF4	NRL	T
E2F	ID1	OLIG2	TAL1
ERG	ID2	PAX3	TAL2
EWS	JUN	PAX8	TCF3
ETV1	MAF	PHOX2A	TCF4
FLI	MAZ	PHOX2B	TCF7L2
FOS	MECOM	POU2AF1	TP63
FOXL2	MEF2C	PRDM1	TWIST1
FOXOs	MITF	PRRX1	XBP1
FOXP3	MYCL/N	RUNX1	

TF: transcription factor.

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The Kronos Bio product engine addresses the two major challenges to drugging transcription

Challenges to drugging transcription

TFs function as massive transcriptional regulatory networks, which complicates:

- Knowing the optimal target(s) within the network
- Providing readouts of TF function

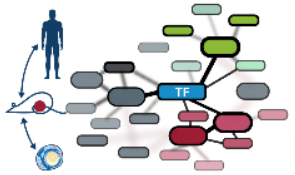
TFs are difficult targets for traditional small molecule screens because TFs:

- Have intrinsically disordered protein structure in isolation
- Only adopt a defined structure in their native complexes

Kronos Bio's Approach

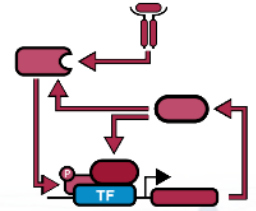
MAP TRN

Integrative networks shaped by real world evidence

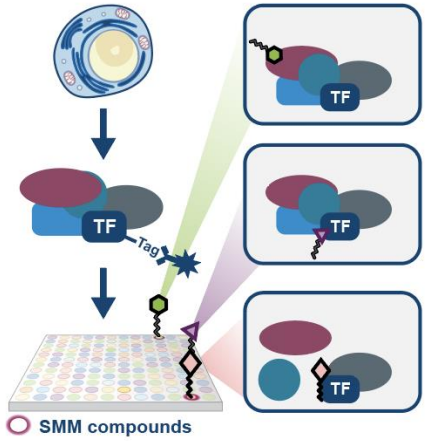


DEFINE DEPENDENCIES

Target and patient selection driven by causal networks

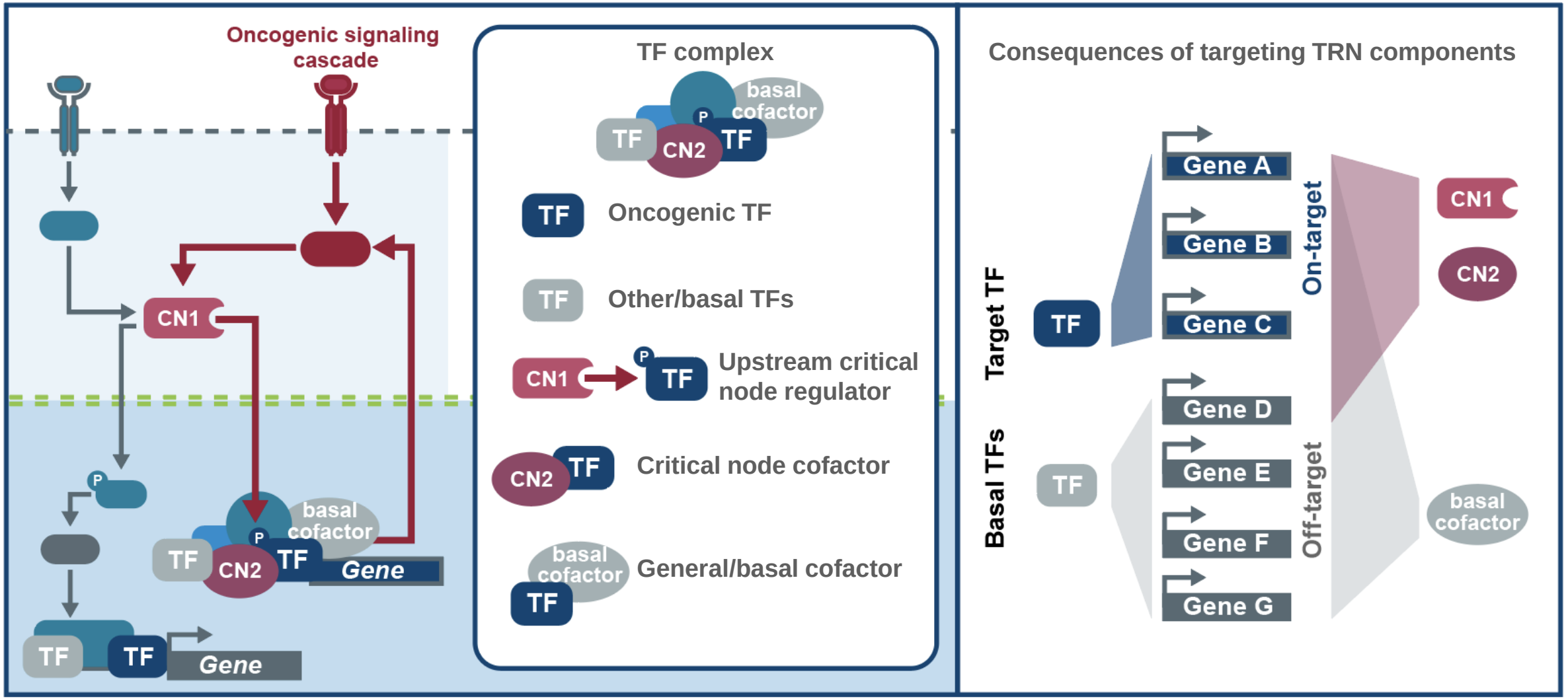


SMALL MOLECULE MICROARRAY



TF: transcription factor. TRN: transcriptional regulatory network.

TRN maps identify optimal critical nodes to selectively target oncogenic TF activity



CN: critical node; TF: transcription factor. TRN: transcriptional regulatory network.

Product engine identifies and optimizes compounds that target TRN components and mechanisms

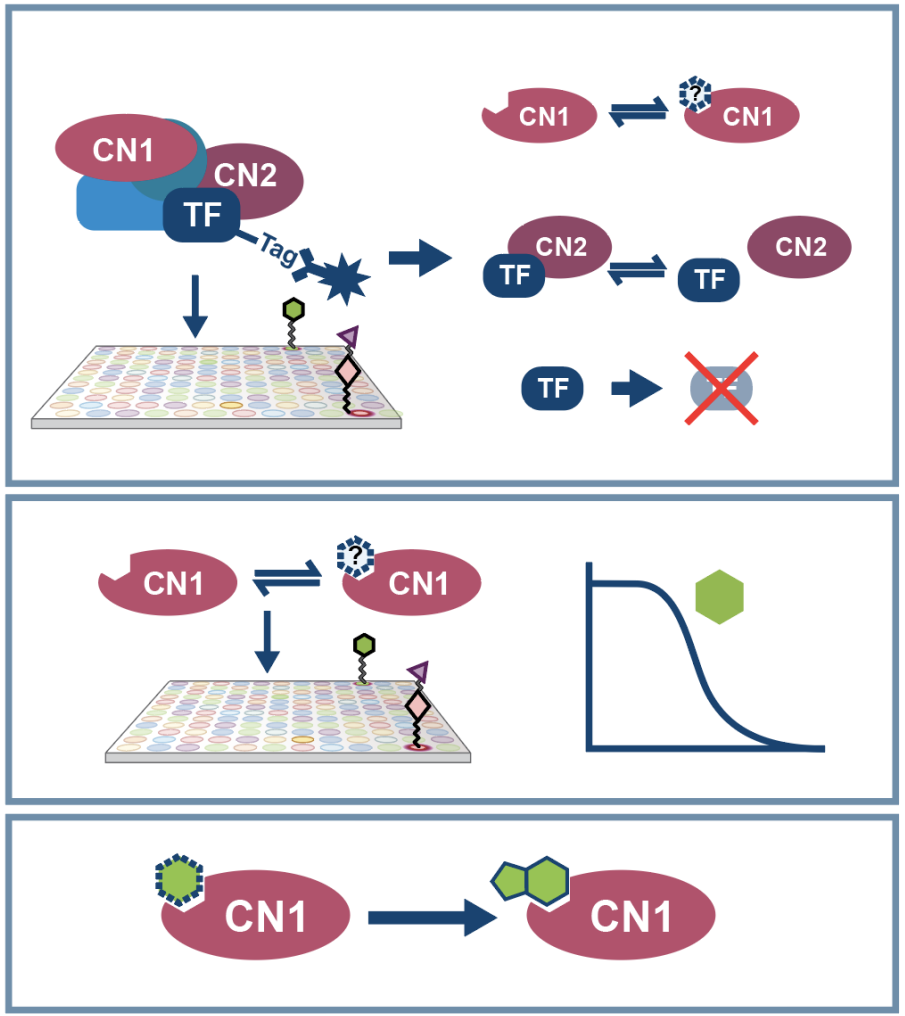
Path 1: Identify TF complex binders in lysate using SMM

- Screen for binders of TF complex in lysates using Small Molecule Microarray (SMM)
- Triage hits for multiple targets and mechanisms

Path 2: Direct *in vitro* screen and optimization

- Directly screen druggable critical nodes in vitro using SMM or traditional HTS
- Validate and optimize in vitro

Path 3: Leverage and advance existing chemical matter



CN: critical node. HTS: high-throughput screening. TF: transcription factor. TRN: transcriptional regulatory network.

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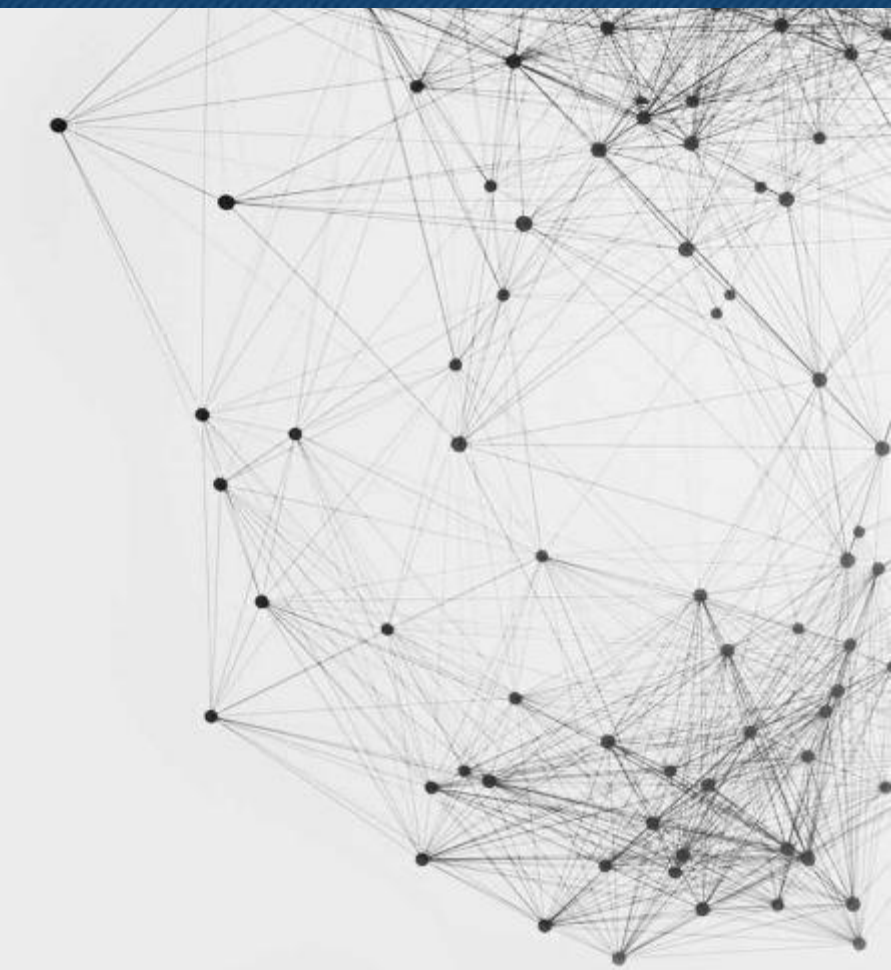

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04 **Kronos Bio milestones and financials**

Kronos Bio milestones and financials

Upcoming Catalysts

KB-0742

Mid-2024 Phase 1/2 trial clinical data update

Q3 2024 Enroll 80mg 4 on/3 off expansion cohort

1H 2025 Topline safety and efficacy from expansion cohort

KB-9558

Q4 2024 Completion of IND-enabling studies

1H 2025 Commence a first-in-human study in multiple myeloma, pending completion of IND-enabling studies

Strong Financial Position

- Approx. $175 million in cash, cash equivalents and investments (as of December 31, 2023)
- Cash runway projected into 2H 2026
- Approx. 58.9 million shares outstanding (common, as of December 31, 2023)

Corporate Partnerships

- Platform discovery collaboration with Genentech to advance novel therapies against transcriptional targets in oncology
- Ongoing collaboration with Tempus provides access to real-world and multi-omics data

Genentech
A Member of the Roche Group

TEMPUS

For additional information, please contact:

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Margaux Bennett
Vice President, Corporate Development and Investor Relations, Kronos Bio
mbennett@kronosbio.com

Thank You