Corporate presentation

August 2024

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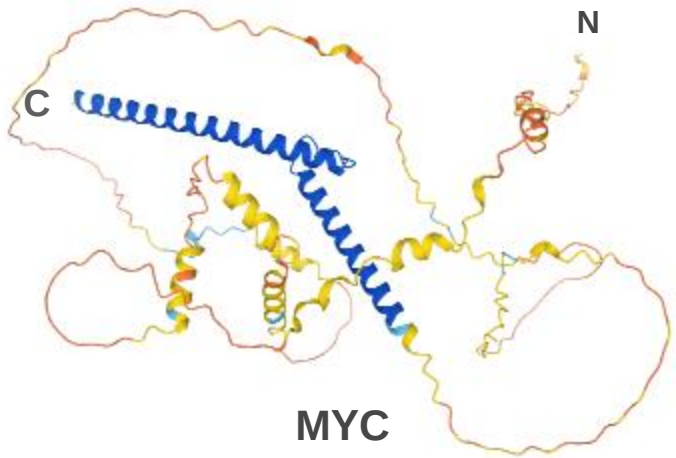
Leader in drugging transcription to address unmet needs in cancer

Our proprietary discovery engine **decodes complex transcription factor (TF) regulatory networks** to identify druggable cofactors in a **tumor-specific context**

Most TFs are intrinsically disordered



Alphafold - P01108

Only 7 of the 100+ TFs implicated in cancer have been successfully targeted

APPROVED*

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

IN CLINICAL DEVELOPMENT

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

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

UNDRUGGED

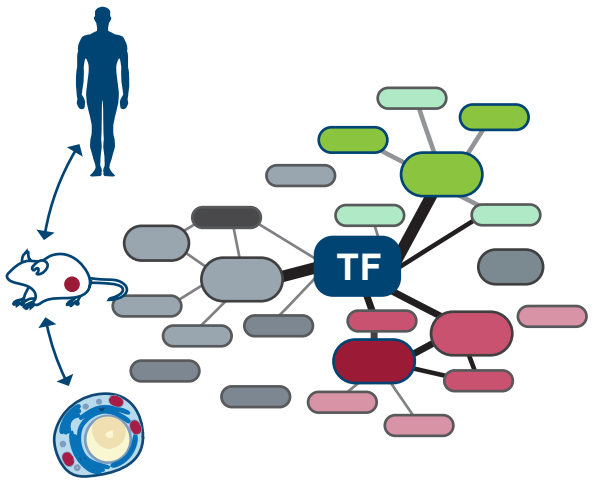
ASCL1	GATA2/3	NEUROD1	RUNX2
BCL6	GLI1	NF1	SMADs
C19ORF11	GLI2	NFKB	SNAI2
CEBPa/b	HAND1/2	NR2F2	SOX2
CRX	IRF4	NRL	T
E2F	ID1	OLIG2	TAL1
ERG	ID2	PAX3	TAL2
EWS	JUN	PAX8	TCF3
ETV1	MAF	PHOX2A	TCF4
FLI	MAZ	PHOX2B	TCF7L2
FOS	MECOM	POU2AF1	TP63
FOXL2	MEF2C	PRDM1	TWIST1
FOXOs	MITF	PRRX1	XBP1
FOXP3	MYCL/N	RUNX1	

The Kronos Bio approach to drugging transcription

We target transcription factor regulatory networks

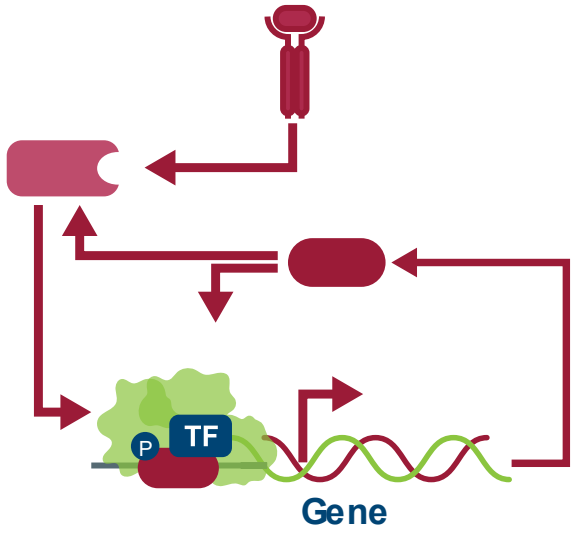
MAP TRN 01

Integrative networks shaped by real world evidence



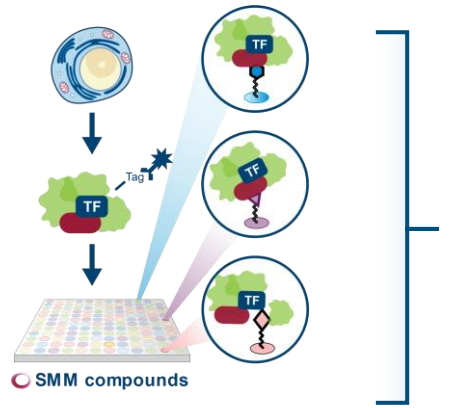
DEFINE DEPENDENCIES 02

Target and patient selection driven by causal networks

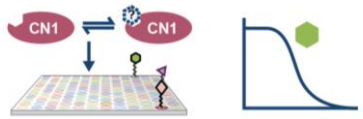


IDENTIFY MODULATORS 03

Screen for small molecules that target prioritized TF cofactors



Screen in molecules nuclear lysates with Small Molecule Microarray (SMM)

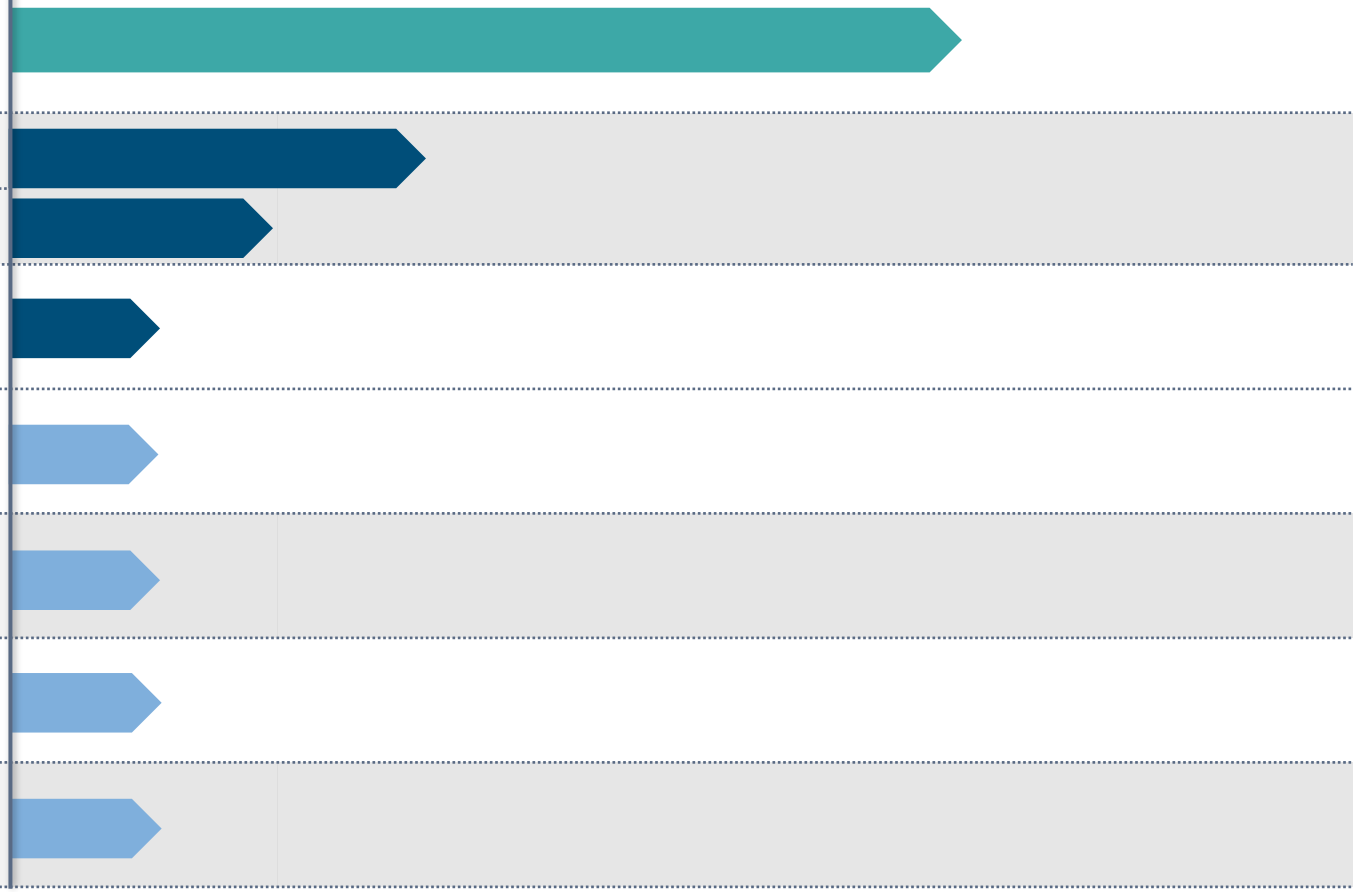


Screen *in vitro* or with HTS

TF: transcription factor. TRN: transcriptional regulatory network.

Advancing both clinical and discovery programs across multiple oncogenic TRNs

TRN	Candidate	Indication	Discovery	IND-enabling	Phase 1	Phase 2	Phase 3
MYC	Istisociclib (KB-0742) (CDK9 inhibitor)	Platinum-resistant high-grade serous ovarian cancer					
IRF4	KB-9558 (p300 KAT inhibitor)	R/R multiple myeloma					
		HPV-driven tumors					
	Undisclosed p300 KAT inhibitor	Autoimmune indications					
MYC	Undisclosed						
β-Catenin	Undisclosed						
Undis-closed	Discovery collaboration	Genentech A Member of the Roche Group					
Multiple	Undisclosed						



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KAT: lysine acetyltransferase. IRF4: interferon regulatory factor 4. R/R: Relapsed/ Refractory. TF: transcription factor. TRN: transcription regulatory network.

KB-0742, an oral CDK9 inhibitor

Dose expansion in platinum resistant high grade serous ovarian cancer ongoing

There is high unmet need in ovarian cancer

~22,000 new cases of ovarian cancer each year in the US alone[1]

85% of patients will experience progression after platinum therapy[2]

<50% 5-year survival[3]

Current treatment outcomes in platinum-resistant ovarian cancer

~15% overall response rates[2]

~3.5mo progression free survival[2]

1. OCRA.org
2. St. Laurent & Liu 2023
3. Phung et al., 2023

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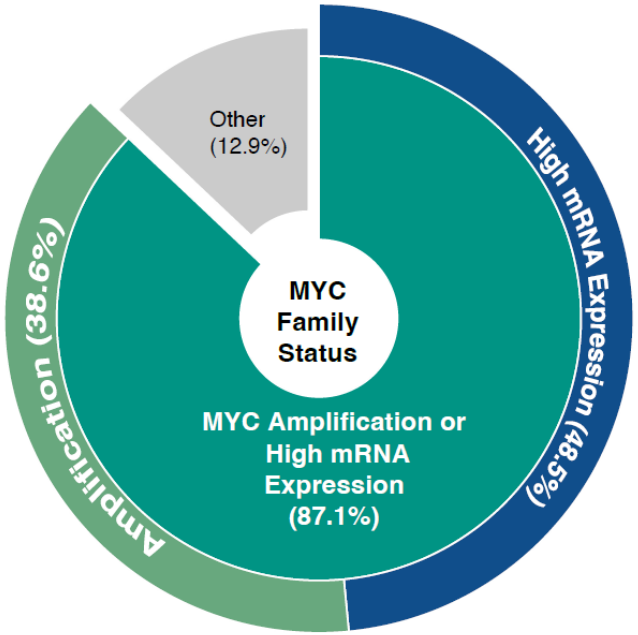
Majority of ovarian cancer patients exhibit deregulated MYC or deficiencies in homologous recombination

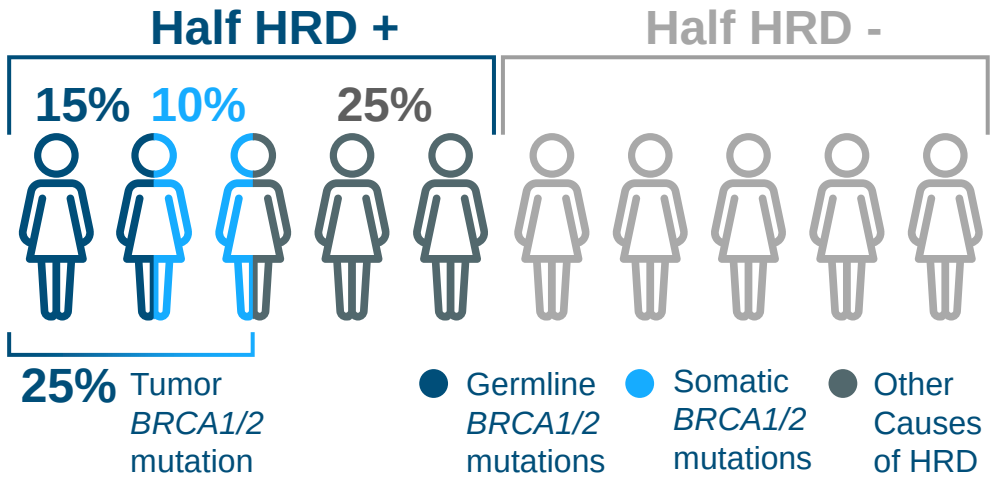
~85% of ovarian cancer patients have deregulated MYC driven transcription[1]



% of Patients
N=1,667 R/R Ovarian patients in Tempus RWD

Type of MYC
(*MYC/MYCL/MYCN*) deregulation

50% of ovarian cancer patients are homologous recombination deficient (HRD+)[2]



Myriad Genetics

Therapies that undermine the dependency on MYC or that capitalize on the HRD+ genotype may be beneficial in treating these high unmet need platinum-resistant ovarian cancer patients

HRD: homologous recombination deficient

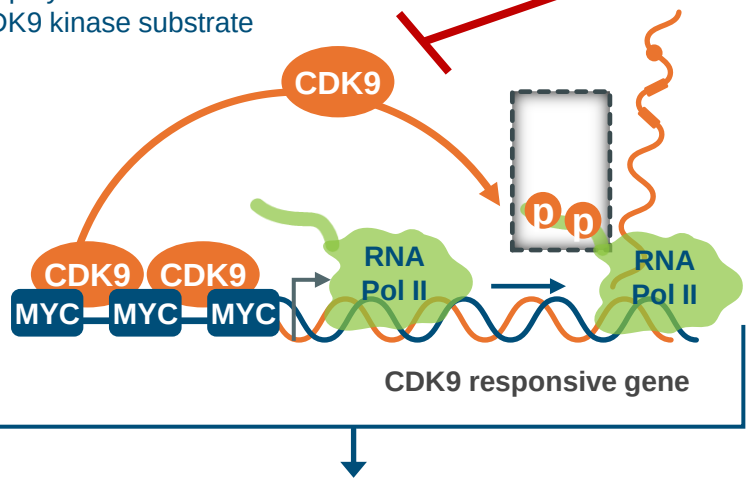
1.) Villalona-Calero, Miguel, et al. EORTC-NCI-AACR 2023 [Poster] 2.) TCGA research Nature. ; 474(7353): 609–615. doi:10.1038/nature10166

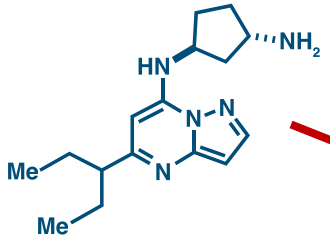
KB-0742 may drive anti-tumor response in ovarian cancer by impairing the MYC transcriptional program and inducing transcriptional and replication stress

Inhibition of CDK9 leads to…

…anti-tumor effects in tumors with de-regulated MYC

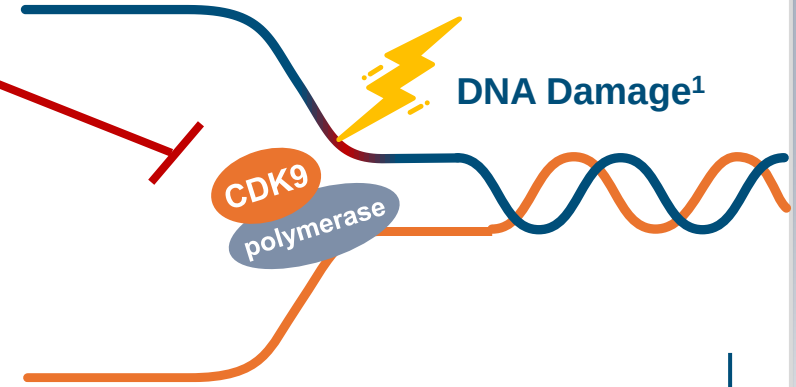
Phosphorylated serine 2 (pSER2) of the RNA polymerase II C-terminal domain is a key CDK9 kinase substrate



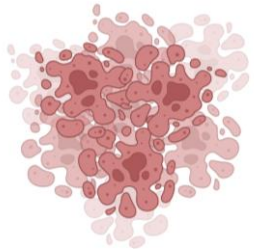


KB-0742[2,3]

…synthetic lethality in homologous recombination deficient (HRD+) cells



…by impairing transcription elongation, collapsing the MYC transcriptional network…



Cell death

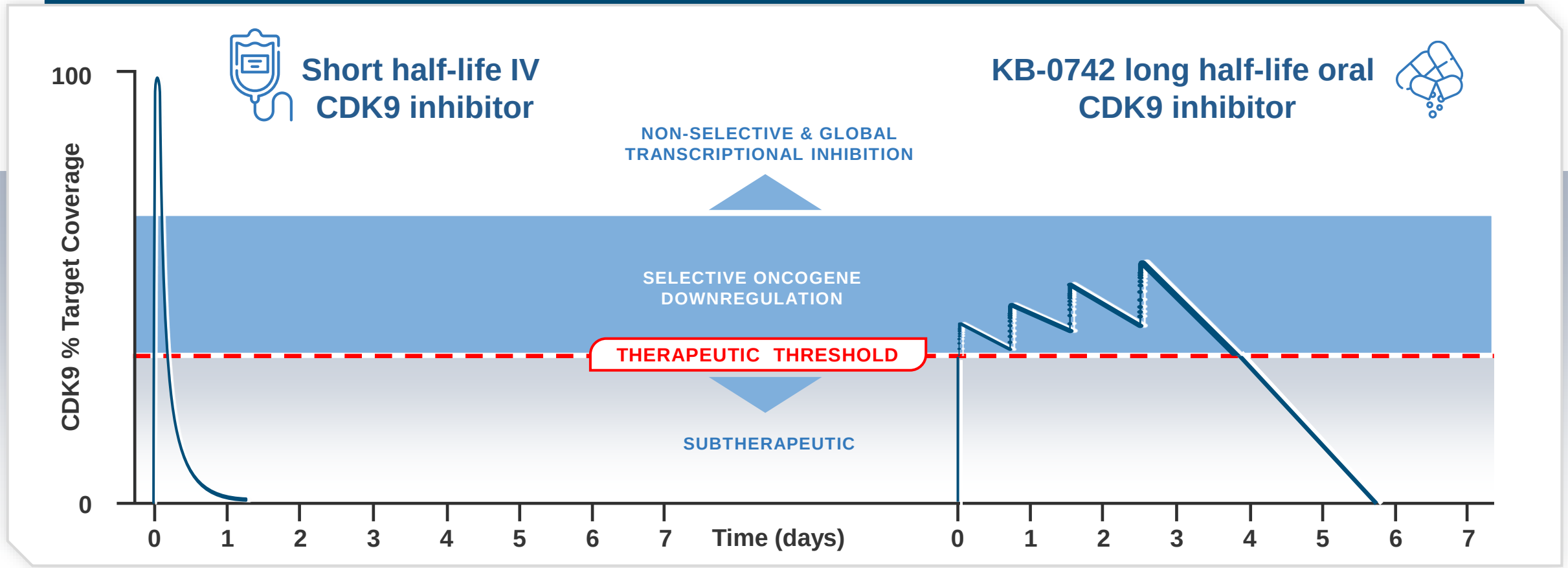
…by stalling RNA and DNA polymerases which induces DNA double stranded breaks...

HRD: homologous recombination deficient. TF: transcription factor.

1.) Yu and Cortez 2010 2.) Richters, A et al. Modulating Androgen Receptor-Driven Transcription in Prostate Cancer with Selective CDK9 Inhibitors. Cell Chem. Biol. 2020, 28, 1-14; 3.) 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, 15629–15647µ

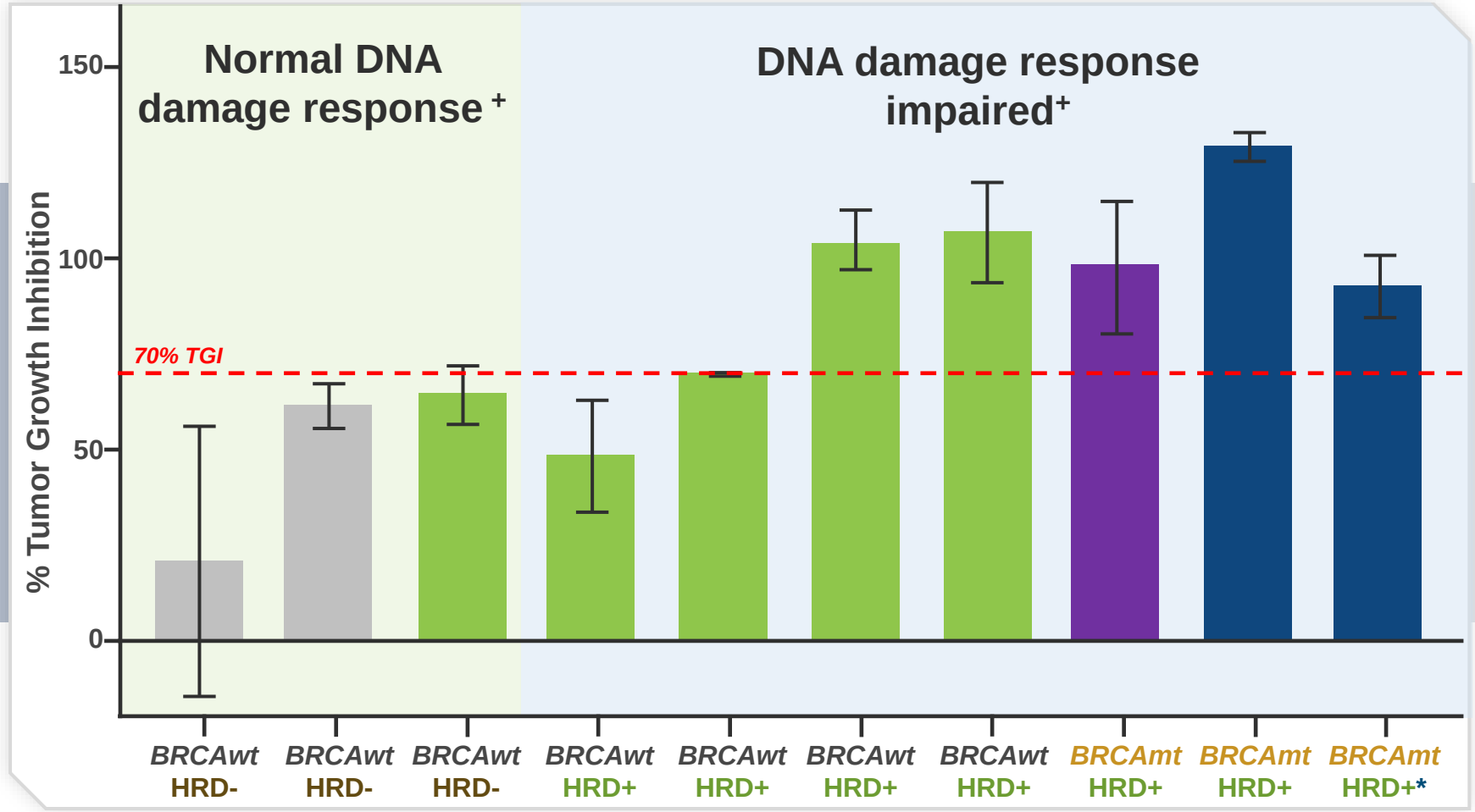
KB-0742's long plasma half-life, kinase selectivity, and oral dosing provide a differentiated profile that avoids global transcriptional inhibition

OUR HYPOTHESIS:
Time above therapeutic threshold drives efficacy while increased C_{max} impacts safety/tolerability



Figures for illustrative purposes only

MYC deregulated and HRD+ ovarian cancer patient derived xenograft (PDX) models show increased tumor growth inhibition in response to KB-0742



Amp
MYCL Exp
MYCN/MYCL Exp
Other

Summary of Results

→ **8** out of **10** tested models exhibited **>50% TGI with KB-0742**

→ **6** out **7** HR-deficient models exhibited **>70% TGI (-- red line) with KB-0742**

***Known treatment history:** taxol, carboplatin, doxil, and gemcitabine

Dosing regimen:
60mg/kg 3-days on/4-days off

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+ Normal DNA damage response = HRD-; DNA damage response impaired = HRD+

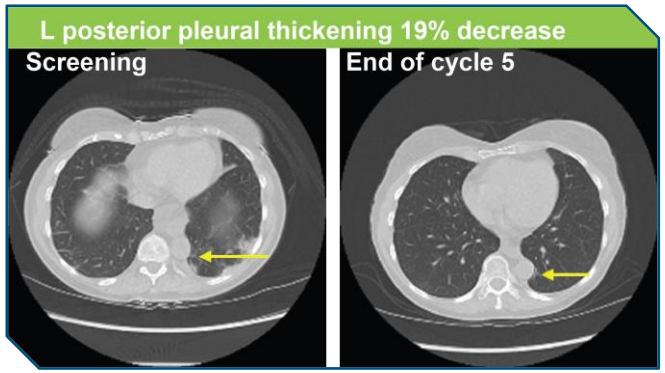
Clinical benefit observed with KB-0742 at 60mg 3 days on/4 days off in a platinum-resistant HGSOC patient

- Platinum-resistant patient (<6mo on platinum therapy)
- High-Grade Serous Ovarian Cancer
- MYC amplified or overexpressed
  MYCL1 amplified
- HRD- by Myriad
- HRD+ by Tempus

(Based on HR pathway deficiency)

MYCL+ platinum resistant patient shows target lesion reduction and long-term stable disease >300 days

- 75-year-old female Stage 3b high-grade serous ovarian cancer in September 2022
- Genetic profiling:
  - Foundation Tissue: PIK3CA, SOX2, TP53, EWSR, FGF12 amplified, KDM5A, *MYCL1* amplified, *PRKC1* amplified, *TERC* amplified, MSS, TMB2.
  - Invitae: ATM, MSH6+; negative for BRCA1/2.
  - Tempus: ATM, MSH6+, *MYC* amplified (4+ copies) and overexpressed (cMYC H score > 50)
- Prior lines: Paclitaxel+carboplatin, pembrolizumab: (OCT 22- JAN 23); DS-6000A (FEB 23-JUN 23; SD = best response)



Data as of April 4, 2024

KB-0742 treatment
On study drug through C10 and ongoing; stable disease (SD) for **>320 days and continuing therapy as of July 8, 2024**

HGSOC: high-grade serous ovarian cancer. HRD: homologous recombination deficient.

In 103 patients treated in the 60mg and 80mg 3 days on/4 days off cohorts, KB-0742 demonstrated manageable safety and tolerability

Event	60mg (N=82)		80mg (N=21)		60mg and 80mg (N=103)	
	All grades	Grade 3	All grade	Grade 3	All Grade	Grade 3
Any TEAE n(%)	**81 (98.8)**	**31 (37.8)**	**21 (100)**	**9 (42.9)**	**102 (99.0)**	**40 (38.8)**
Nausea	57 (69.5)	2 (2.4)	15 (71.4)	0	72 (69.9)	2 (1.9)
Vomiting	45 (54.9)	2 (2.4)	9 (42.9)	0	54 (52.4)	2 (1.9)
Fatigue	24 (29.3)	0	6 (28.6)	0	30 (29.1)	0
Constipation	21 (25.6)	0	3 (14.3)	0	24 (23.3)	0
Anaemia	18 (22.0)	3 (3.7)	4 (19.0)	1 (4.8)	22 (21.4)	4 (3.9)
Diarrhoea*	16 (19.5)	1 (1.2)	5 (23.8)	0	21 (20.4)	1 (1.0)
Hyponatraemia	14 (17.1)	1 (1.2)	5 (23.8)	1 (4.8)	19 (18.4)	2 (1.9)

Event	60mg (N=82)		80mg (N=21)		60mg and 80mg (N=103)	
Hematologic TEAE* n(%)	All grade	Grade 3**	All grade	Grade 3**	All grade	Grade 3**
Anaemia	18 (22.0)	3 (3.7)	4 (19.0)	1 (4.8)	22 (21.4)	4 (3.9)
Lymphopenia	12 (14.6)	5 (6.1)	1 (4.8)	0	13 (12.6)	5 (4.9)
Neutropenia	2 (2.4)	0	0	0	2 (1.9)	0
Thrombocytopenia	5 (6.1)	1 (1.2)	2 (9.5)	0	7 (6.8)	1 (1.0)

No observed grade 3/4 neutropenia

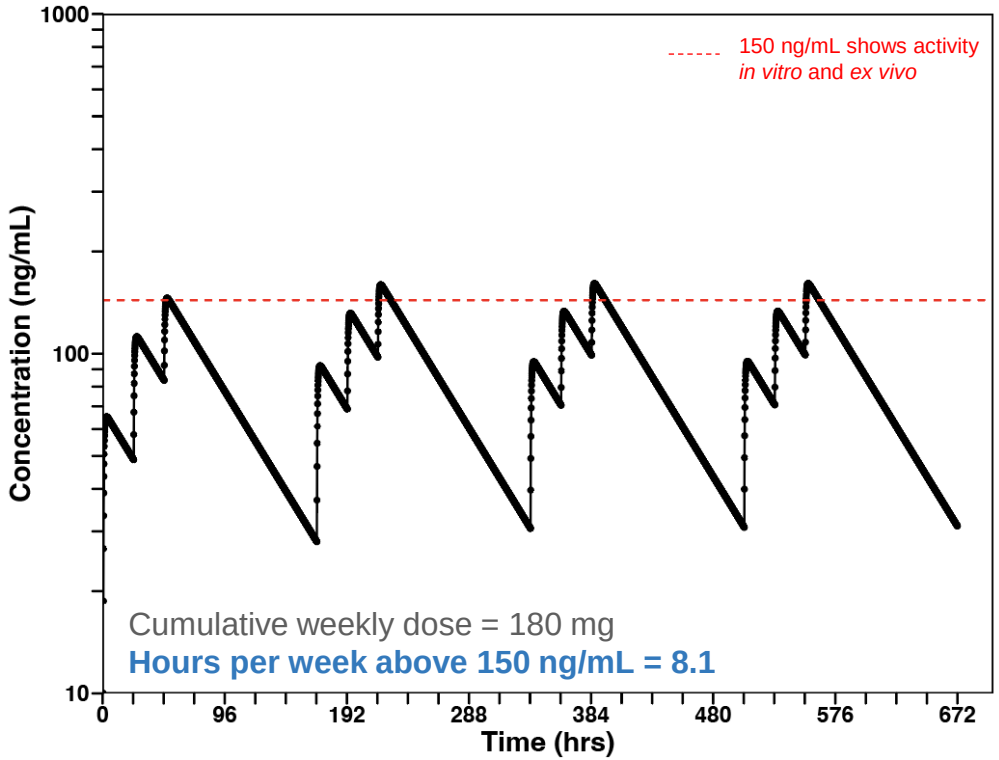
Less than 10% of patients discontinued due to AEs

*TEAE Diarrhoea Grade 4, reported in one patient, administered KB-0742 80 mg 3 days on/4 days off
Note: TEAE = Treatment Emergent Adverse Event; MedDRA v23.1

Pharmacokinetic of 80mg dose given 4 days on/3 days shows a 1.8-fold increase in 7-day AUC; safety profile consistent with previous experience

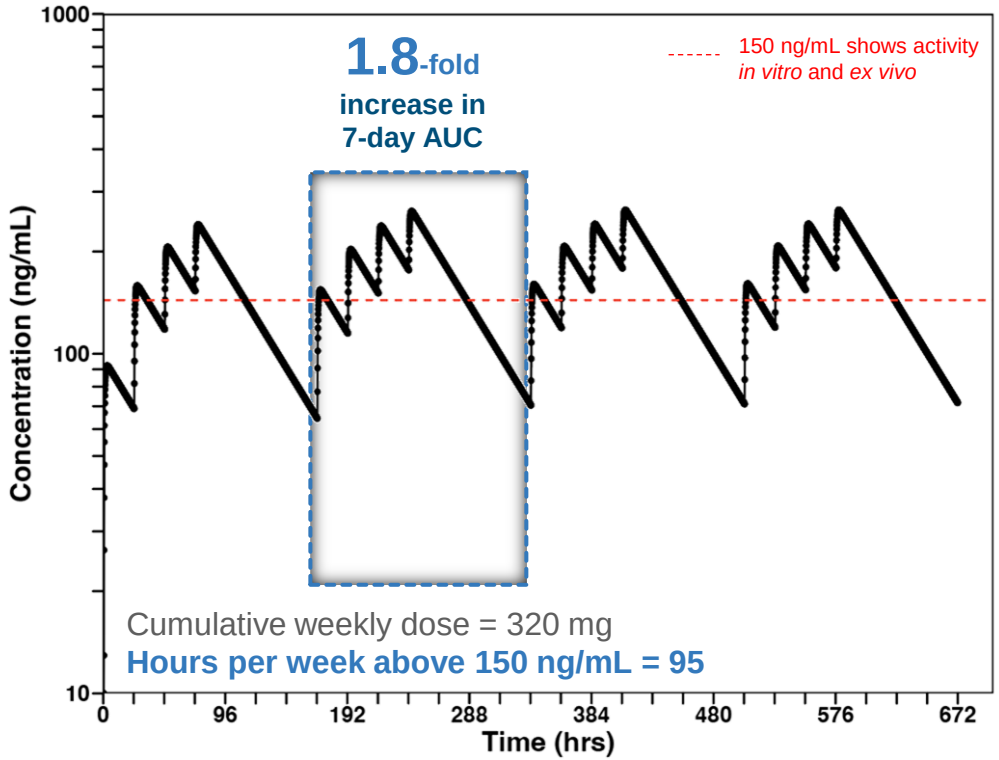
Dose/schedule at which we have seen activity

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



Dose/schedule at which we are enrolling platinum-resistant HGSOC expansion cohort

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



HGSOC: high-grade serous ovarian cancer

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KB-0742 is expected to show efficacy in platinum-resistant high-grade serous ovarian cancer

- Preliminary on-mechanism activity seen at 60mg 3 days on/4 days off dose
- Acceptable safety profile observed through 80mg 4 days on/3 days off dose
- July 2024, first patient enrolled in platinum-resistant, high-grade serous ovarian cancer cohort at 80mg 4 days on/3 days off
- Potential to establish monotherapy activity to enable future monotherapy or combination studies across multiple solid tumor indications

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

KB-9558, a p300 lysine acetyltransferase inhibitor

IND-enabling studies in multiple myeloma ongoing

Despite recent transformational therapeutics for multiple myeloma, patients progress and could benefit from oral therapy

Limitations of CAR-T and bi-specific therapies:

- **Access limited primarily to academic centers:** Majority of multiple myeloma is treated in the community[9]
- **Significant risk of adverse events:** Hospitalization required due to adverse events; CAR-T requires 30 day stay post transfusion (~80% experience CRS)[10,11]
- **Patients eventually progress:** As many as 60% patients (≥2 class refractory) progress on CAR-T after 2 year of therapy, and ~50% progress on bispecific therapies by 1 year[1-8,11-16]

We set about to develop an oral therapy that inhibits interferon regulatory factor 4 (IRF4)

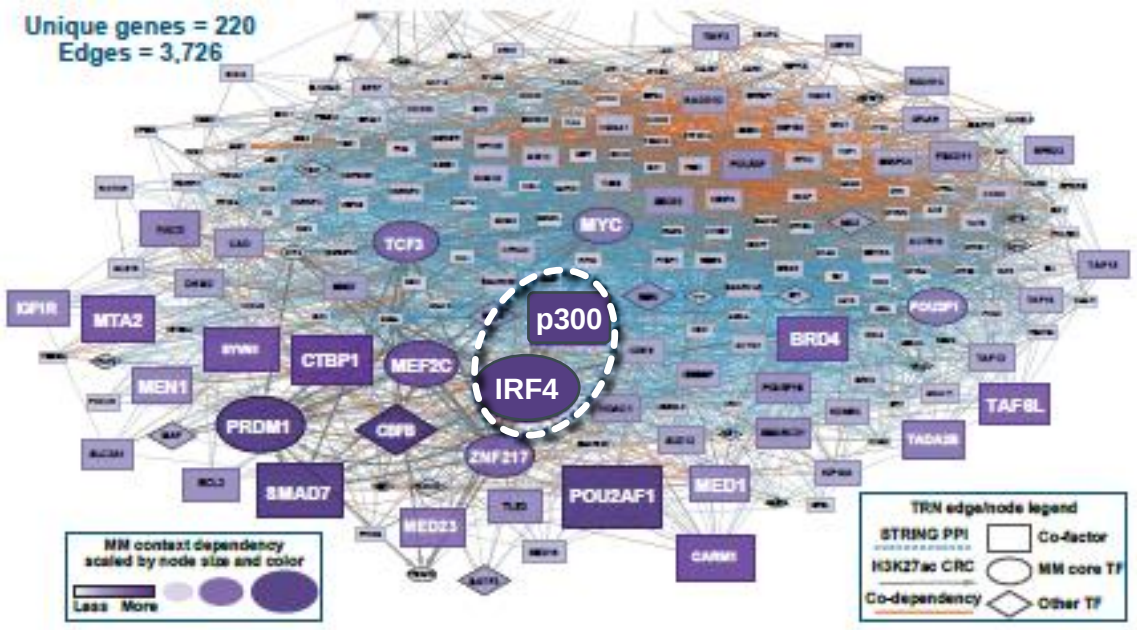
IRF4 is a master TF deregulated in multiple myeloma

CRS: Cytokine Release Syndrome.

1. Mikhael J. Clin Lymphoma Myeloma Leuk. 2020;20(1):1-7; 2. Sevcikova S, et al. Blood Rev. 2019;36:32-39; 3. Gandhi UH, et al. Leukemia. 2019;33(9):2266-2275; 4. Hari P, et al. J Ger Oncol. 2018;9:138-144; 5. Willenbacher W, et al. Int J Mol Sci. 2018;19:E2087; 6. Yong K, et al. Br J Haematol. 2016;175:252-264; 7. Corre J, et al. Leukemia. 2018;32(12):2636-2647; 8. Nijhof IS, et al. Drugs. 2018;78:19-37. 9. Braunlin et al. Leukemia & Lymphoma, 62:2, 377-386 2021. 10. Munshi et al., 2021 N Engl J Med 384;8 11. CARVYKTI prescribing information. 12. CARVYKTI prescribing info CARTITUDE-1. 13. ABCEMA prescribing information. 14. Swan D. et al., Cancers 2023, 15, 1819. 15. Bahlis N. et al., Nat Med. 2023; 29(10): 2570–2576. 16. Liu L, Krishnan A. Haematologica 2024;109(3):718-724

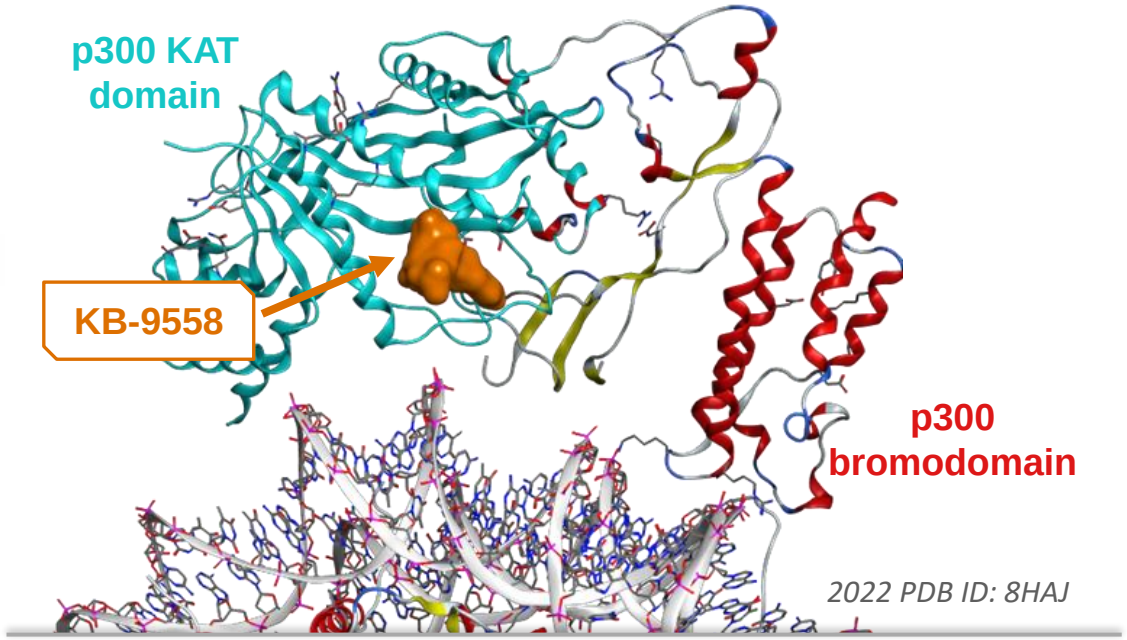
Discovery of KB-9558, an inhibitor of the lysine acetyltransferase domain of p300

p300 is the nearest druggable node to IRF4 in multiple myeloma



Multiple myeloma transcription regulatory network

Medicinal chemistry campaign resulted in identification of KB-9558, a p300 KAT inhibitor



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

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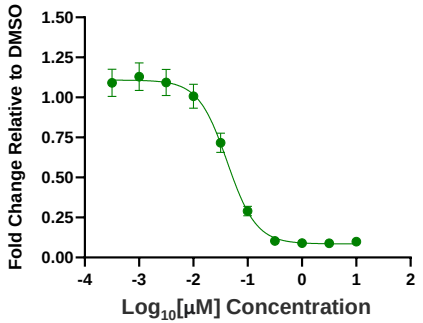
p300 KAT inhibition leads to IRF4 TRN suppression & apoptosis in multiple myeloma

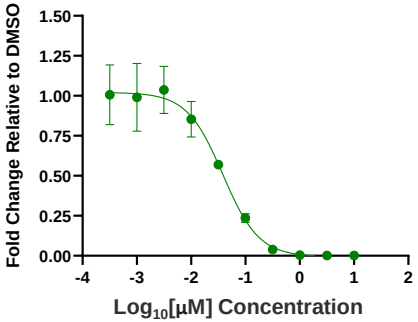
In vitro *In vivo*

p300 KAT inhibition leads to loss of IRF4 and MYC expression… → **…and loss of IRF4 protein level…** → **…causing loss of expression of IRF4 targets…** → **…ending with cell death…** → **…which results in TGI as monotherapy and in combination**

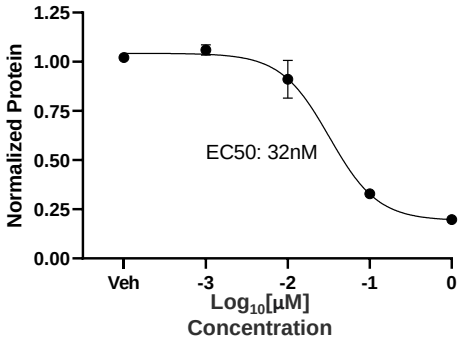
IRF4 Transcript Levels
24hr; MM1S; qRT-PCR Taqman



MYC Transcript Levels
24hr; MM1S; qRT-PCR Taqman

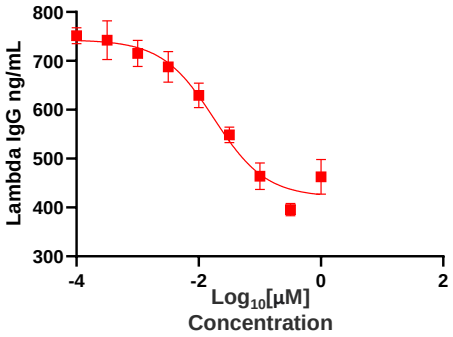


IRF4 Protein Levels
24hr; MM1S



→ Collapses the oncogenic IRF4 TRN

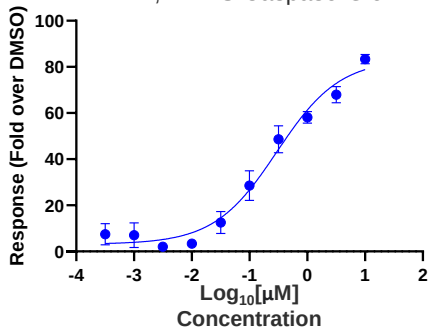
Lambda Light Chain Protein
24hr; MM1S; ELISA



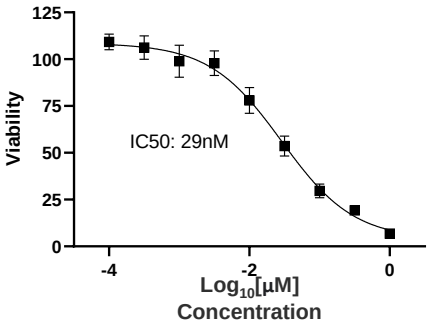
→ Circulating IgG commonly assessed as a clinical biomarker

→ Pairs with Kronos Bio established proximal and functional PD assays

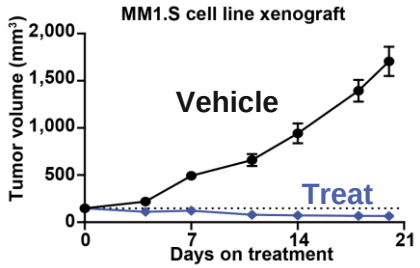
Caspase Activation
72hr; MM1S Caspase-Glo



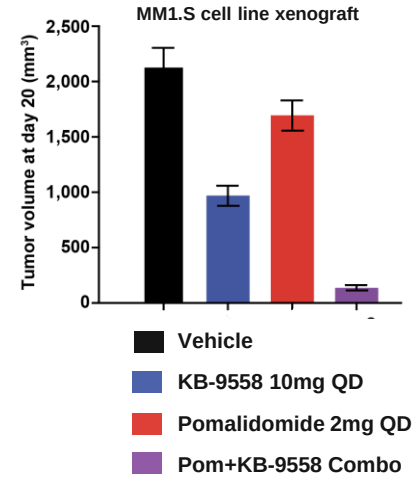
Cell Viability
72hr; MM1S CellTiter-Glo



MM1.S cell line xenograft



MM1.S cell line xenograft

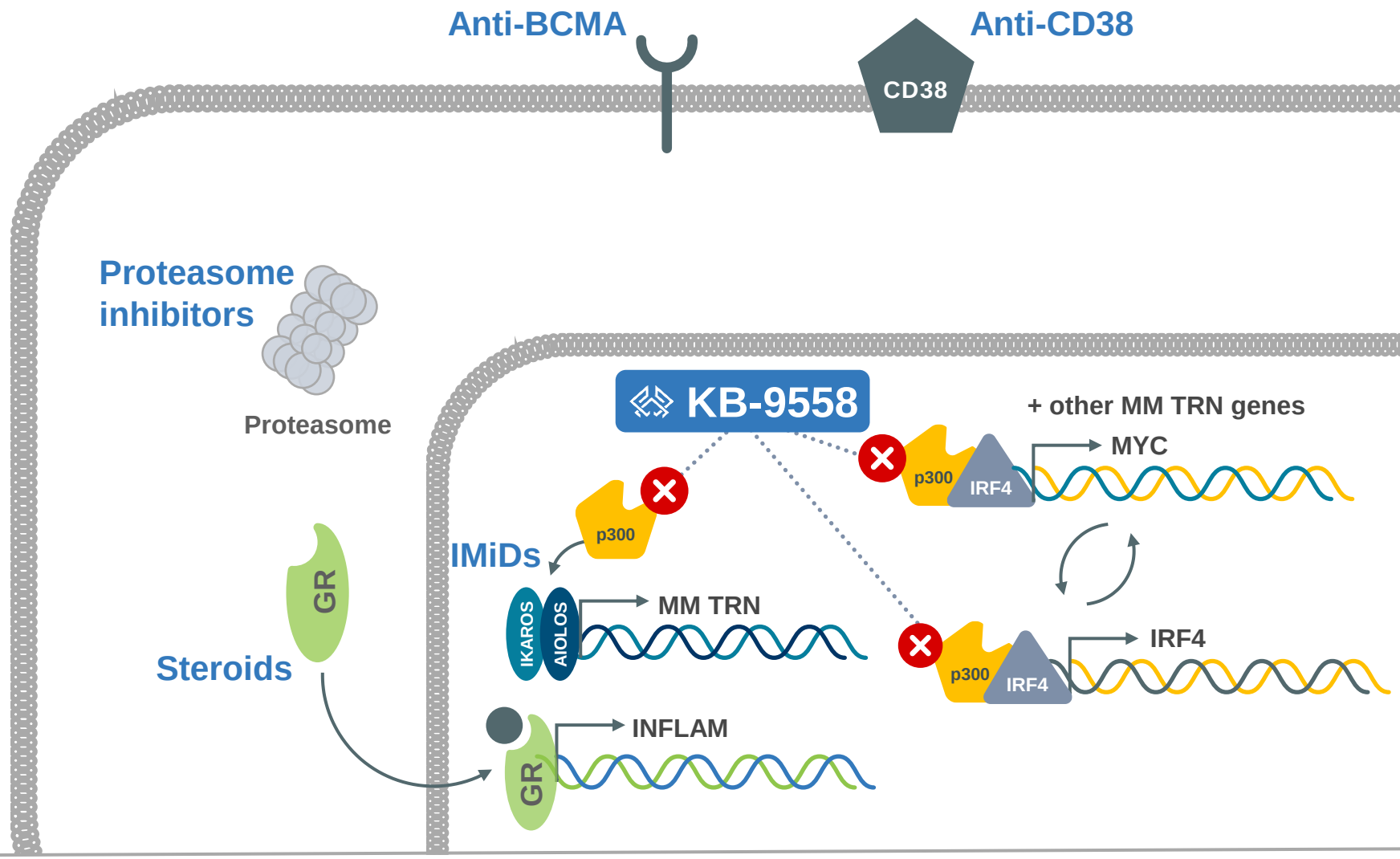


IRF4: interferon regulatory factor 4. KAT: lysine acetyltransferase. TGI: Tumor growth inhibition. TRN: Transcription regulatory network.

KRONOS·BIO

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 in multiple lines of therapy



BCMA: B-cell maturation antigen. CD38: cluster of differentiation 38. GR: glucocorticoid receptor. IMiD: immunomodulatory drugs. TRN: transcription regulatory network.

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

- KB-9558, by inhibiting the KAT domain of p300, collapses the IRF4 transcription regulatory network, a key driver of multiple myeloma
- Preclinical studies with KB-9558 show tumor growth inhibition in multiple myeloma both as monotherapy and in combination with pomalidomide
- As a small molecule with a novel mechanism, KB-9558 has the potential to benefit multiple myeloma patients either as a monotherapy or in an all-oral combination regimen

First patient in Phase 1 dose escalation study of KB-9558 expected to enroll 1H 2025

Milestones and financials

Kronos Bio milestones and financials

Upcoming Catalysts

KB-0742

1H 2025 Topline safety and efficacy data from platinum-resistant HGSOC expansion cohort

KB-9558

Q4 2024 Complete IND-enabling studies

1H 2025 Initiate a first-in-human study in multiple myeloma

Strong Financial Position

- Approx. $136.6 million in cash, cash equivalents and investments (as of June 30, 2024)
- Cash runway projected into 2H 2026
- Approx. 60.11 million shares outstanding (common, as of June 30, 2024)

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

HGSOC: high-grade serous ovarian cancer

Thank you