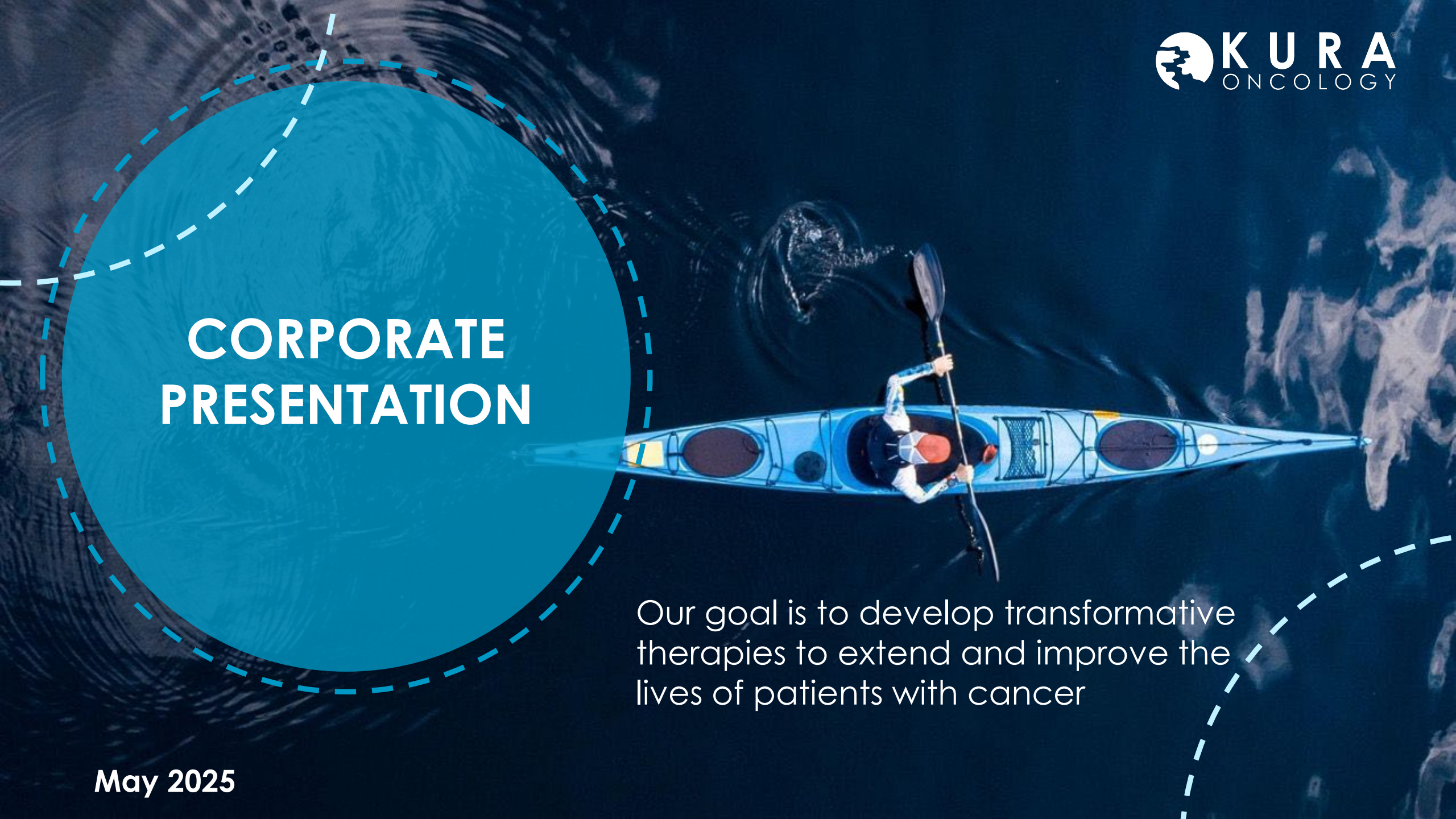
CORPORATE PRESENTATION



Our goal is to develop transformative therapies to extend and improve the lives of patients with cancer

May 2025

FORWARD-LOOKING STATEMENTS

This presentation contains forward-looking statements. Such statements include, but are not limited to, statements regarding our research, preclinical and clinical development activities, plans and projected timelines for ziftomenib, KO-2806 and tipifarnib, plans regarding regulatory filings, our expectations regarding the relative benefits of our product candidates versus competitive therapies, our expectations regarding the therapeutic and commercial potential of our product candidates, and our expectations regarding our collaboration with Kyowa Kirin. The words "believe," "may," "should," "will," "estimate," "promise," "plan", "continue," "anticipate," "intend," "expect," "potential" and similar expressions (including the negative thereof) are intended to identify forward-looking statements. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. Risks that contribute to the uncertain nature of the forward-looking statements include: our preclinical studies and clinical trials may not be successful; the U.S. Food and Drug Administration (FDA) may not agree with our interpretation of the data from clinical trials of our product candidates; we may decide, or the FDA may require us, to conduct additional clinical trials or to modify our ongoing clinical trials; we may experience delays in the commencement, enrollment, completion or analysis of clinical testing for our product candidates, or in the reporting of data from such clinical testing, or significant issues regarding the adequacy of our clinical trial designs or the execution of our clinical trials may arise, which could result in increased costs and delays, or limit our ability to obtain regulatory approval; our product candidates may not receive regulatory approval or be successfully commercialized; unexpected adverse side effects or inadequate therapeutic efficacy of our product candidates could delay or prevent regulatory approval or commercialization; we may not be able to obtain additional financing; and our collaboration with Kyowa Kirin may not be successful. Additional risks and uncertainties may emerge from time to time, and it is not possible for Kura's management to predict all risk factors and uncertainties.

All forward-looking statements contained in this presentation speak only as of the date on which they were made. Other risks and uncertainties affecting us are described more fully in our filings with the Securities and Exchange Commission. We undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

This presentation also contains statistical, preclinical and clinical data obtained from and prepared by third parties. The recipient is cautioned not to give undue weight to such disclosures. Neither the Company nor any other person makes any representation as to the accuracy or completeness of such data or undertakes any obligation to update such data after the date of this presentation.

KURA IS ADVANCING A ROBUST PIPELINE OF THERAPEUTIC PRODUCT CANDIDATES

Ziftomenib: Potentially Best-in-Class Menin Inhibitor for AML

Relapsed/refractory (R/R) and frontline acute myeloid leukemia (AML) market opportunity could exceed $7B per year in the U.S.

Positive topline results from KOMET-001 study in R/R *NPM1*-m AML; NDA submitted in Q1 2025; full data accepted for presentation at ASCO and EHA

Kyowa Kirin collaboration funds expansive AML development program through 1L U.S. commercialization

Farnesyl Transferase Inhibitors (FTIs) in Large Solid Tumor Indications

FTIs may overcome innate and adaptive resistance to PI3Kα inhibitors, KRAS inhibitors and tyrosine kinase inhibitors (TKIs) in certain indications

Target indications include HNSCC, lung, colorectal, pancreatic and renal cell carcinomas

Clinical data for KO-2806 and tipifarnib in combination expected in 2H 2025

Additional Therapeutic Opportunities for Menin Inhibitors

First patients dosed in Phase 1 study of ziftomenib + imatinib in gastrointestinal stromal tumors (GIST); additional potential $1B opportunity

Encouraging preclinical data for menin inhibitors in type 2 diabetes; development candidate nomination anticipated mid-2025

EXPERIENCED LEADERSHIP TEAM AND BOARD OF DIRECTORS

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MAXIMIZE VALUE OF ROBUST THERAPEUTIC PIPELINE

PROGRAM	CLINICAL TRIAL	RESEARCH	DOSE ESCALATION	DOSE OPTIMIZATION	REGISTRATION-ENABLING	REGULATORY SUBMISSION
Ziftomenib MENIN INHIBITOR	komet-001 Monotherapy	*NPM1*-mutant acute myeloid leukemia (AML)[1]				
		KMT2A-rearranged acute lymphoblastic leukemia (ALL)				
		Non-*NPM1*-mutant / Non-*KMT2A*-rearranged AML				
	komet-007 Combinations with venetoclax/azacitidine, cytarabine + daunorubicin (7+3)	*NPM1*-mutant AML				
		KMT2A-rearranged AML				
	komet-008 Combinations with gilteritinib, FLAG-IDA, LDAC	*NPM1*-mutant AML				
		KMT2A-rearranged AML				
	komet-015 Combination with imatinib	Advanced GIST				
	komet-017 Combinations with venetoclax/azacitidine, cytarabine + daunorubicin (7+3)	*NPM1*-mutant AML[2]				
		KMT2A-rearranged AML[2]				
KO-2806 & Tipifarnib FARNESYL TRANSFERASE INHIBITORS (FTIs)	FIT-001 Monotherapy, combinations with cabozantinib and adagrasib	Solid tumors				
		Renal cell carcinoma (RCC)				
		$KRAS^{G12C}$-mutant non-small cell lung cancer (NSCLC), Colorectal cancer (CRC), Pancreatic ductal adenocarcinoma (PDAC)				
	KURRENT-HN Combination with alpelisib	PIK3CA-dependent head and neck squamous cell carcinoma (HNSCC)[3]				
Next-Gen Menin Inhibitor		Diabetes				

1 Topline results reported on February 5, 2025; patients remain on study in survival follow-up. NDA submitted in 1Q 2025 for the treatment of adult patients with relapsed or refractory AML with an NPM1 mutation.
2 KOMET-017 program to advance directly to Registration-Enabling trials, leveraging data from Phase 1 combination trials.
3 Enrollment is complete.

ANTICIPATED UPCOMING MILESTONES: SEVERAL EXPECTED 2025 DATA READ-OUTS ACROSS MULTIPLE PROGRAMS

Ziftomenib	Report topline results from KOMET-001 Phase 2 registration-directed trial in R/R *NPM1*-m AML	✓
	FDA feedback on KOMET-017 registration-enabling protocol in 1L *NPM1*-m and *KMT2A*-r intensive and non-intensive AML	✓
	NDA submission for ziftomenib in R/R *NPM1*-m AML	✓
	Initiate KOMET-015 Phase 1 trial of ziftomenib in combination with imatinib in patients with advanced GIST	✓
	Present full data from KOMET-001 Phase 2 registration-directed trial in R/R *NPM1*-m AML	2Q 2025
	Present preliminary clinical data from KOMET-007 Phase 1b trial in 1L intensive AML	2Q 2025
	Initiate KOMET-017 Phase 3 registration-enabling trials in 1L *NPM1*-m and *KMT2A*-r intensive and non-intensive AML	2H 2025
	Present preliminary clinical data from Phase 1b expansion of KOMET-007 in 1L non-intensive AML	2H 2025
KO-2806 / tipifarnib	Initiate one or more expansion cohorts in combination with cabozantinib in RCC	2H 2025
	Present preliminary clinical data from FIT-001 trial for KO-2806 as monotherapy and combo with cabozantinib in RCC	2H 2025
	Present clinical data from the KURRENT-HN trial of tipifarnib in combo with alpelisib in *PIK3CA*-dependent HNSCC	2H 2025
Next-gen Menin	Nominate a development candidate for next-generation menin inhibitor program for diabetes	Mid-2025

FINANCIAL HIGHLIGHTS (NASDAQ: KURA)

Cash, Cash Equivalents and Marketable Securities

$703.2M

in pro forma cash, cash equivalents and short-term investments as of March 31, 2025*

Anticipated Significant Near-Term Milestones

$375M

in potential near-term milestones, including launch of ziftomenib in the monotherapy R/R setting

Shares Outstanding

80.8M
COMMON STOCK

24.5M options, RSUs, PSUs, warrants & pre-funded warrants as of March 31, 2025

Kura anticipates collaboration plus cash balance as of March 31, 2025 to fund ziftomenib AML program to potential commercialization in frontline combinations

MENIN AML GIST DIABETES

ZIFTOMENIB

POTENT, SELECTIVE MENIN INHIBITOR

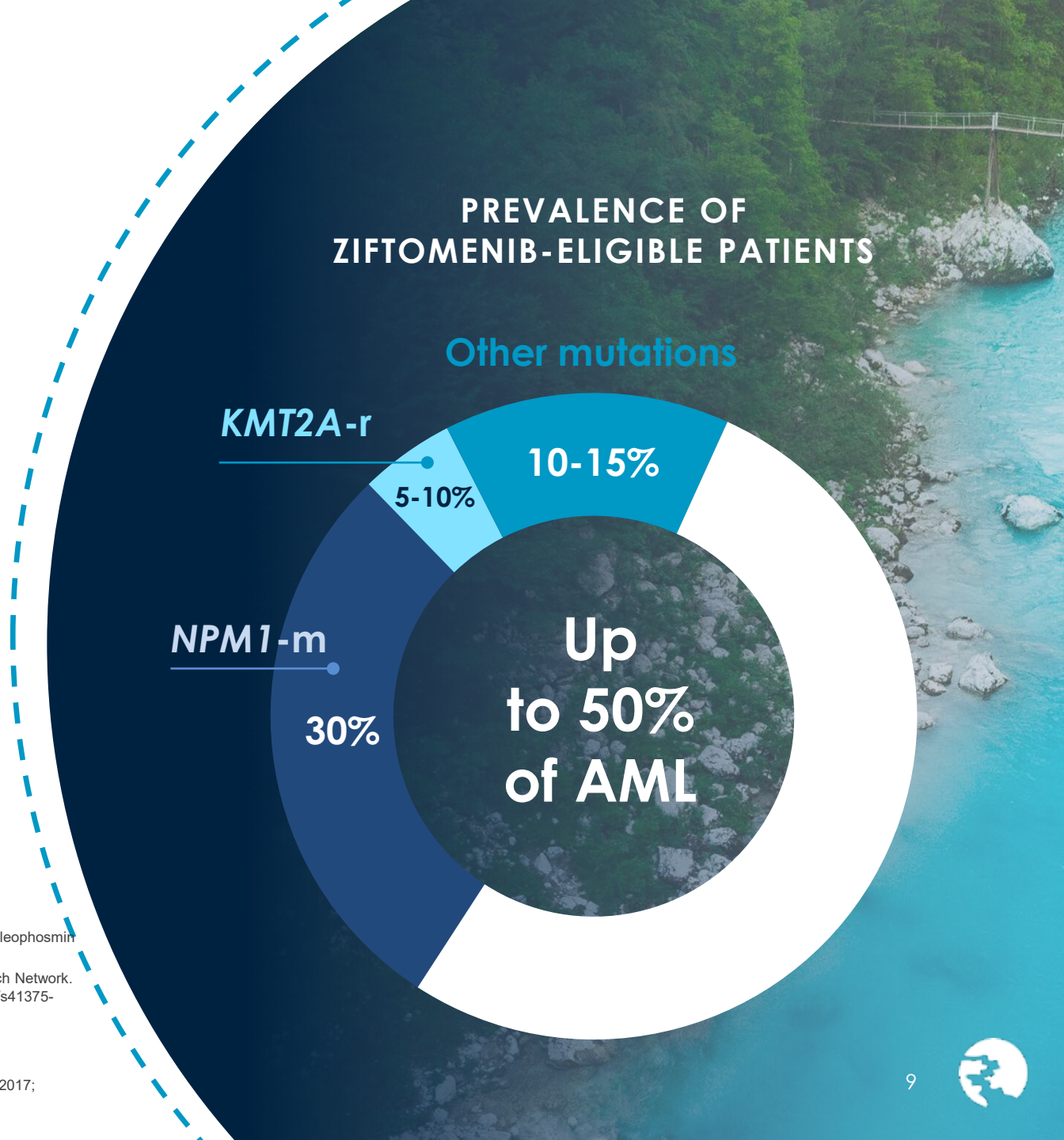
Targeted Investigational Menin Inhibitor for Newly Diagnosed and Relapsed/Refractory Acute Myeloid Leukemia (AML)

UP TO 50% OF AML PATIENTS MAY BENEFIT FROM MENIN INHIBITOR THERAPY

AML is characterized by significant genetic heterogeneity due to driver mutations, including *NPM1*m, *FLT3*m, *IDH1/2*m and *KMT2A*r [1-2]

Up to 50% of AML cases may be menin-dependent, including those driven by *NPM1*m and *KMT2A*r[3-7]

NPM1m are observed in 30% to 35% of cases and are an important upstream driver mutation that uses the menin pathway[8,9]



AML, acute myeloid leukemia; *KMT2A*r, lysine methyltransferase 2A rearrangement; *NPM1*-m, mutated nucleophosmin 1; *NPM1*m, nucleophosmin 1 mutation; *FLT3*m, FMS-like tyrosine kinase 3 mutation; *IDH1/2m*, mutations in isocitrate dehydrogenases types 1 and 2.

1. Papaemmanuil E *et al. N Engl J Med.* 2016;374(23):2209-2221. doi:10.1056/NEJMoa1516192 **2.** The Cancer Genome Atlas Research Network. *N Engl J Med.* 2013;368(22):2059-2074. doi:10.1056/NEJMoa1301689 **3.** Issa GC *et al. Leukemia.* 2021;35(9):2482-2495. doi:10.1038/s41375-021-01309-y **4.** Candoni A, Coppola G. *Hematol Rep.* 2024;16(2):244-254. doi:10.3390/hematolrep16020024 **5.** Bertrums EJM *et al. Haematologica.* 2023;108(8):2044-2058. doi:10.3324/haematol.2022.281653 **6.** National Cancer Institute. Accessed October 16, 2024. https://seer.cancer.gov/seertools/hemelymph/ 51f6cf59e3e27c3994bd547d/ **7.** National Cancer Institute. Accessed October 16, 2024. https://seer.cancer.gov/seertools/hemelymph/5a7e288d1ef557f9c8636d31/ **8.** Burrows F *et al.* Poster presented at: AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics: Discovery, Biology, and Clinical Applications; October 26-30, 2017; Philadelphia, PA. **9.** Falini B, Dillon R. *Blood Cancer Discov.* 2024;5(1):8-20. doi:10.1158/2643-3230.BCD-23-0144

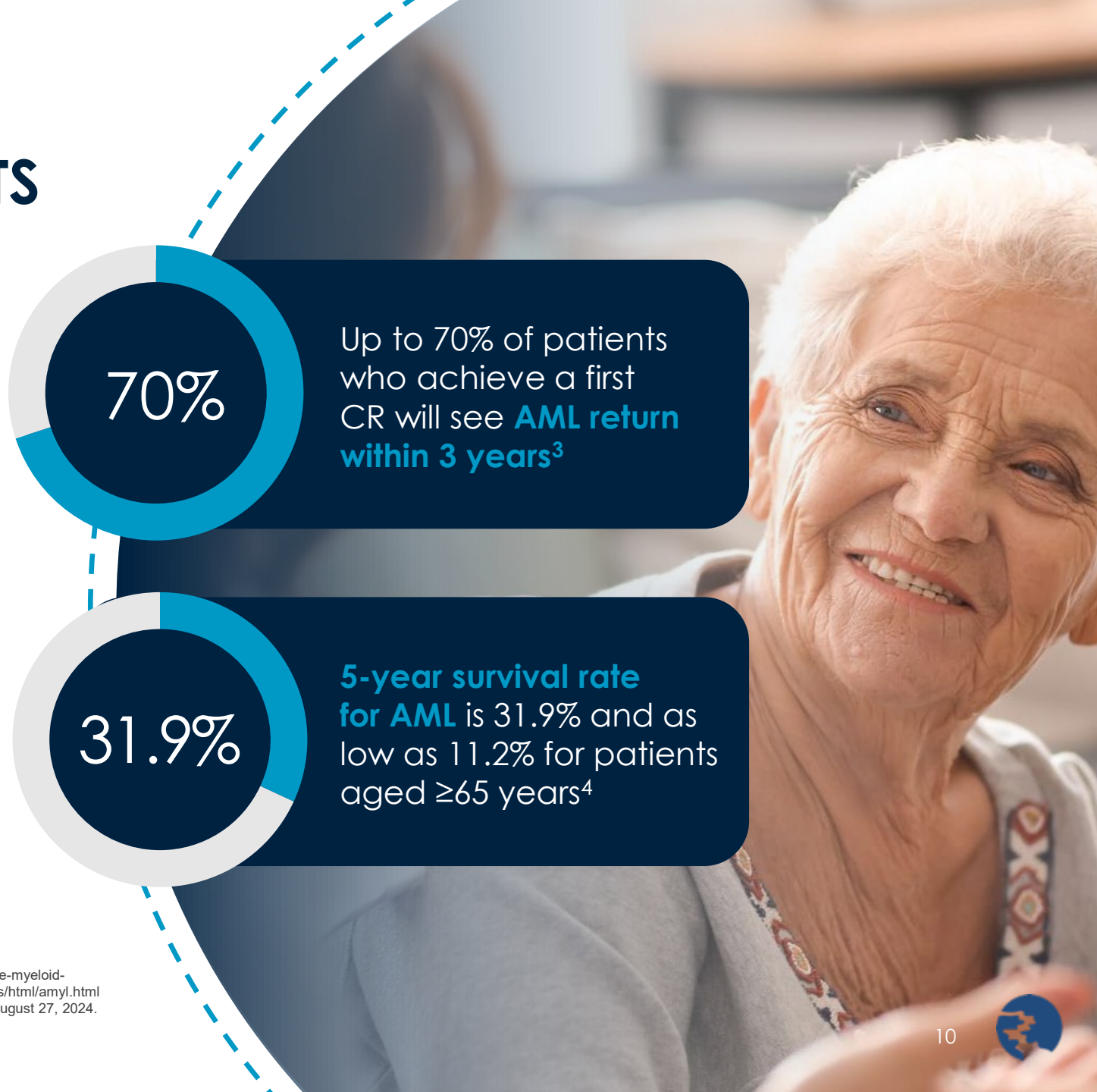
SIGNIFICANT UNMET NEED REMAINS FOR AML PATIENTS

Acute Myeloid Leukemia (AML)

An estimated 20,800 new cases of AML diagnosed each year in the United States[1]

Median age at diagnosis is 69 years; majority of diagnoses made in patients aged 65 to 74 years.[2]

Current FDA approved therapies include combination chemotherapy regimens such as 7+3, venetoclax and hypomethylating agents (HMAs) and FLT3 inhibitors like midostaurin or quizartinib



AML, acute myeloid leukemia; CR, complete response.

1. American Cancer Society. Updated June 5, 2024. Accessed August 27, 2024. https://www.cancer.org/cancer/types/acute-myeloid-leukemia/about/key-statistics.html **2.** National Cancer Institute. Accessed August 27, 2024. https://seer.cancer.gov/statfacts/html/amyl.html **3.** Kumar CC. *Genes Cancer*. 2011;2(2):95-107. doi:10.1177/1947601911408076 **4.** National Cancer Institute. Accessed August 27, 2024. https://seer.cancer.gov/statfacts/html/amyl.html

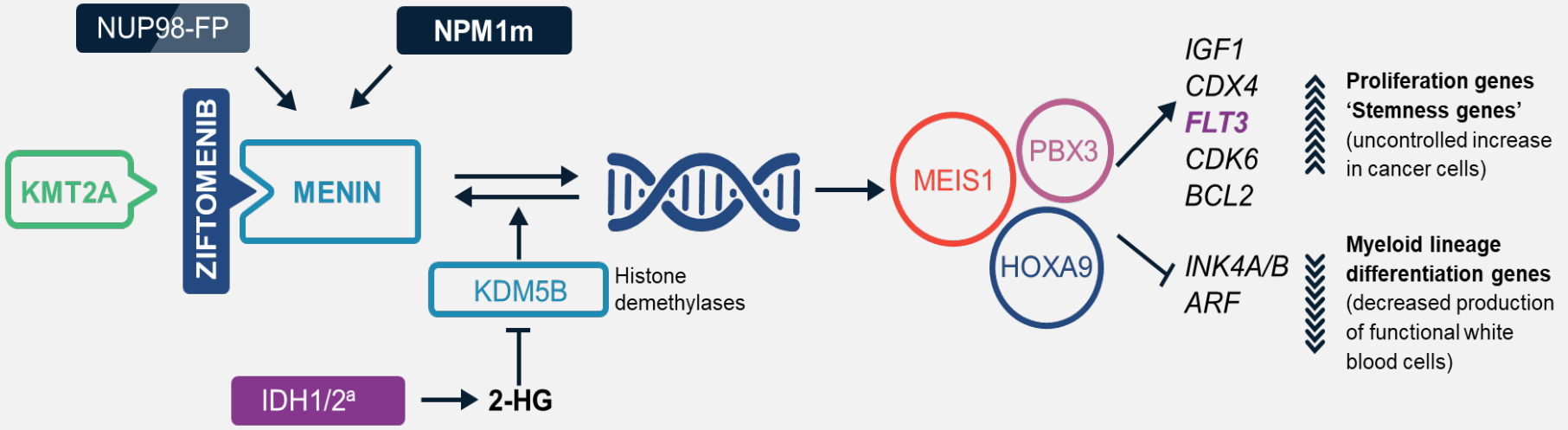
ZIFTOMENIB TARGETS THE MENIN PATHWAY, A FOUNDATIONAL TARGET IN AML

In ~35–40% of AML, leukemogenesis is driven by either NPM1 mutations or KMT2A rearrangements,[1,2] which cause AML by blocking differentiation of blasts[3]

KMT2A (MLL) and NPM1 sit upstream from major AML targets (*i.e.*, FLT3, BCL2 and IDH1/2)[4]

Inhibiting the menin-KMT2A complex downregulates HOXA9/MEIS1, leading to differentiation of leukemic blasts[5]

Ziftomenib Mechanism of Action[3, 6-13]



1. Papaemmanuil et al. *N Engl J Med* 2016; 375: 900-1; 2. Issa GC et al. *Leukemia* 2021;3:2482-95; 3. Collins and Hess. *Curr Opin Hematol* 2016;23(4)354-61; 4. Matthews AH et al. *Cancers* (Basel) 2022 Nov 29;14(23):5906. 5. Thomas. *Oncol Ther* 2024;12(1):57-72; 6. Lu et al. *Cancer Cell* 2016;30(1):92–107; 7. Ferreira et al. *Oncogene* 2016;35(23):3079-82; 8. Jeong et al. *Nat Genet* 2014;46(1):17-23; 9. Wang et al. *Blood* 2005;106(1):254–64; 10. Chowdhury et al. *EMBO Rep* 2011;12(5):463-9; 11. Schmidt et al. *Leukemia* 2019;33(7):1608-19; 12. Xu et al. *Cancer Cell* 2016;30(6):863-78; 13. Brunetti et al. *Cancer Cell* 2018; 34(3):499–512.

INVESTIGATING ZIFTOMENIB ACROSS THE AML CONTINUUM IN UP TO 50% OF PATIENTS

for Whom Menin-KMT2A Pathway is a Disease Driver

1L TREATABLE

Intensive (IC) or Non-Intensive (NIC) Tx

Transplant/ No Transplant

Post-Transplant Maintenance

komet ACUTE LEUKEMIAS

KOMET-007
1L Zifto + Ven/Aza
1L Zifto + 7+3

KOMET-017-IC
1L Zifto + 7+3
1L Placebo + 7+3

KOMET-017-NIC
1L Zifto + Ven/Aza
1L Placebo + Ven/Aza

RELAPSED / REFRACTORY

IC or NIC Therapy or Tolerable Therapy

Transplant/ No Transplant

Targeted Therapy if FLT3-m, ~~IDH1/2m~~ and/or NPM1m

Non-Intensive Therapy/ Palliative Care

komet ACUTE LEUKEMIAS

KOMET-001
R/R *NPM1*-m AML

KOMET-007
R/R Zifto + Ven/Aza
R/R Zifto + Ven Only

KOMET-008
R/R Zifto + FLAG-IDA
R/R Zifto + LDAC
R/R Zifto + gilteritinib

Investigator-/Company-Sponsored Studies
Post-HSCT Maintenance

*IC = patients eligible for induction chemotherapy; NIC = patients eligible for non-intensive chemotherapy

SIGNIFICANT UNMET NEED FOR NEWLY DIAGNOSED AML PATIENTS IN FIT AND UNFIT PATIENT POPULATIONS

1L FIT AML

Many patients who are eligible to receive intensive chemotherapy (IC) are treated with an expectation of receiving stem cell treatment (SCT)[1]

Potential opportunity for patients to receive induction, consolidation, then post-consolidation treatment presents a significant opportunity for ziftomenib

Ziftomenib in combination with 7+3 in *NPM1*-m and *KMT2A*-r AML patients, and in combination with 7+3 and quizartinib in *FLT3* co-mutated patients, may address up to 50% of the 1L fit AML market[2-6]

1L UNFIT AML

Venetoclax/azacitidine (ven/aza) is the standard of care for patients unfit for IC treatment, with SCT as a limited option for elderly patients[1]

Patients benefit from the standard-of-care options, but toxicities often limit the long-term treatment of ven/aza[7-9]

Addition of a menin inhibitor in targeted patients may deepen responses, extending duration of benefit and treatment

Treatment duration between 12-24 months may lead to a significant market opportunity in 1L Fit and 1L Unfit AML

7+3 = cytarabine / daunorubicin 1. CancerImpact AML Treatment Architecture, 2023; 2. Issa GC et al. Leukemia. 2021;35(9):2482-2495. doi:10.1038/s41375-021-01309-y 3. Candoni A, Coppola G. Hematol Rep. 2024;16(2):244-25 4. doi:10.3390/hematolrep16020024 5. Bertrums EJM et al. Haematologica. 2023;108(8):2044-2058. doi:10.3324/haematol.2022.281653 5. National Cancer Institute. Accessed October 16, 2024. https://seer.cancer.gov/seertools/hemelymph/ 51f6cf59e3e27c3994bd547d/ 6. National Cancer Institute. Accessed October 16, 2024. https://seer.cancer.gov/seertools/hemelymph/5a7e288d1ef557f9c8636d31/ 7. Pollyea, D. A., DiNardo, C. D., Fathi, A. T., Konopleva, M., Letai, A., & Brunner, A. M. (2020). Management of toxicities associated with targeted therapies for acute myeloid leukemia: A focus on venetoclax-based regimens. Hematology American Society of Hematology Education Program, 2020(1), 57-65. 8. Aldoss, I., Yang, D., Pillai, R., Aribi, A., Mei, M., Nguyen, D., & Marcucci, G. (2023). Long-term outcomes after venetoclax-based therapy in patients with acute myeloid leukemia. Blood, 142(Supplement 1), 4225. 9. Zeidan, A. M., & Daver, N. G. (2023). Managing venetoclax-associated toxicity in AML. OncLive.

komet-007
ACUTE LEUKEMIAS
KURA KO-MEN-007

PHASE 1 COMBINATION TRIAL OF ZIFTOMENIB IN PATIENTS WITH NEWLY DIAGNOSED AML

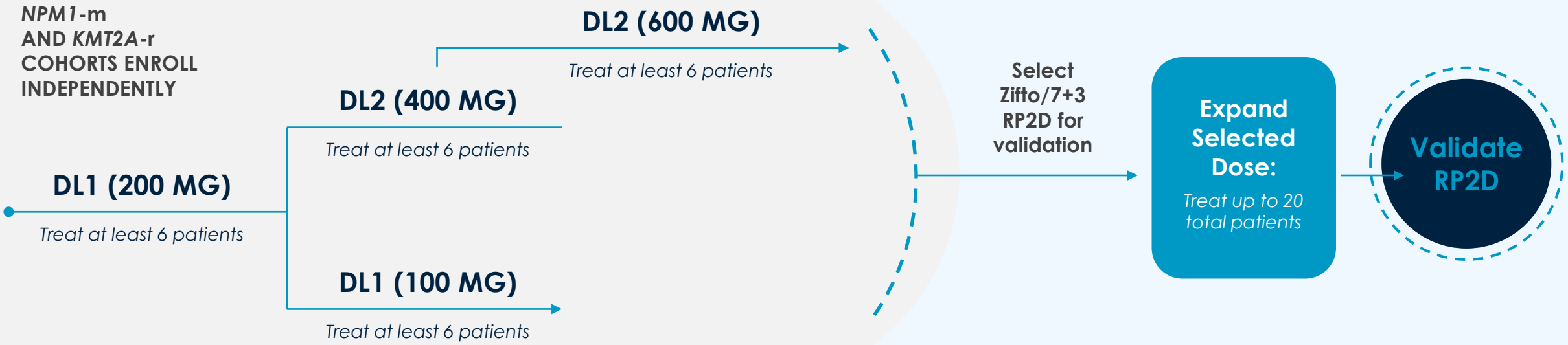
Ziftomenib/cytarabine/daunorubicin (7+3) combination

Ziftomenib dosing begins on Cycle 1 Day 8 and is continuous thereafter

Cytarabine administered on Cycle 1 Day 1-7; administration of an additional cycle based on C1 bone marrow biopsy results

Daunorubicin administered on Cycle 1 Day 1-3; administration of an additional cycle based on C1 bone marrow biopsy results

Dose escalation conducted in patients with adverse risk*



*Age ≥ 60 years and/or treatment-related AML and/or adverse risk cytogenetics per ELN
DL = ziftomenib dose level; zifto = ziftomenib; 7+3 = cytarabine/daunorubicin; RP2D = recommended Phase 2 dose; 1L = first-line; IC = intensive chemotherapy

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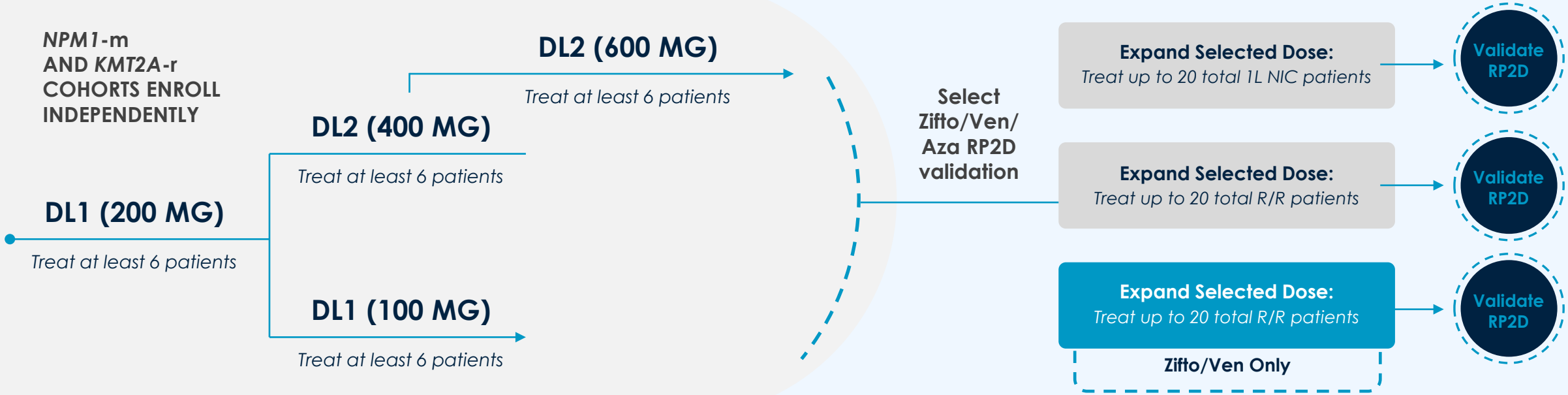
PHASE 1 COMBINATION TRIAL OF ZIFTOMENIB IN PATIENTS WITH NEWLY DIAGNOSED OR R/R AML

Ziftomenib/venetoclax/azacitidine (Ven/Aza) combination

Ziftomenib dosing begins on Cycle 1 Day 8 and is continuous thereafter

Venetoclax administered per label in 28-day cycles with adjustments to cycle length based on Cycle 1 bone marrow biopsy results

Azacitidine administered per label on Cycle 1 Day 1-7 with additional cycles based on bone marrow biopsy results



DL = ziftomenib dose level; zifto = ziftomenib; ven = venetoclax; ven = venetoclax; RP2D = recommended Phase 2 dose; NIC = non-intensive chemotherapy; 1L = first-line; R/R = relapsed/refractory

BASELINE CHARACTERISTICS & DISPOSITION

1L AML (N=51)

		NPM1-m				*KMT2A*-r			
	All Patients (N=51)	**200 mg (n=8)**	**400 mg (n=7)**	**600 mg (n=9)**	**Total (n=24)**	**200 mg (n=10)**	**400 mg (n=9)**	**600 mg (n=8)**	**Total (n=27)**
Median age, years (range)	**60 (18–74)**	65 (43–74)	66 (55–68)	66 (60–68)	**66 (43–74)**	53 (31–73)	51 (28–60)	40 (18–67)	**50 (18–73)**
Female, n (%)	31 (61)	4 (50)	4 (57)	4 (44)	12 (50)	7 (70)	6 (67)	6 (75)	19 (70)
Race, n (%)									
White	33 (65)	7 (88)	6 (86)	4 (44)	17 (71)	8 (80)	4 (44)	4 (50)	16 (59)
Non-White	18 (35)	1 (13)	1 (14)	5 (56)	7 (29)	2 (20)	5 (56)	4 (50)	11 (41)
ECOG PS 0, n (%)	16 (31)	4 (50)	4 (57)	4 (44)	12 (50)	0	2 (22)	2 (25)	4 (15)
1	18 (35)	3 (38)	1 (14)	4 (44)	8 (33)	3 (30)	2 (22)	5 (63)	10 (37)
2	7 (14)	1 (13)	1 (14)	1 (11)	3 (13)	3 (30)	1 (11)	0	4 (15)
Co-mutations, n (%)	17 (33)	2 (25)	1 (14)	5 (56)	8 (33)	4 (40)	2 (22)	3 (38)	9 (33)
FLT3	3 (6)	0	0	0	0	1 (10)	0	2 (25)	3 (11)
IDH1/2	7 (13)	2 (25)	0	5 (56)	7 (29)	0	0	0	0
Therapy-related AML, n (%)	11 (22)	1 (13)	1 (14)	1 (11)	3 (13)	3 (30)	2 (22)	3 (38)	8 (30)
Patients on study*, n (%)	**45 (88)**	8 (100)	7 (100)	9 (100)	**24 (100)**	6 (60)	8 (89)	7 (88)	**21 (78)**
Median follow-up, weeks (range)	25 (2–66)	46 (35–66)	31 (29–34)	21 (17–24)	31 (17–63)	33 (2–43)	25 (15–31)	10 (4–17)	19 (2–43)

* Patients on-treatment or in long-term follow-up.

Zeidan et al. ASH 2024 #214. Data cutoff: Oct 1, 2024.
ECOG PS, Eastern Cooperative Oncology Group performance status

komet-007
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KURA KO-MEN-007

SAFETY AND TOLERABILITY OF ZIFTOMENIB WITH 7+3 IN 1L AML (N=51)

TEAEs in ≥ 30% of All Patients

Safety profile of ziftomenib in combination with intensive chemotherapy similar to that reported for newly diagnosed AML patients treated with 7+3 alone[1]

Rate of TEAEs was consistent across escalating doses of ziftomenib

		NPM1-m				*KMT2A*-r			
TEAEs, n (%)	All Patients (N=51)	200 mg (n=8)	400 mg (n=7)	600 mg (n=9)	Total (n=24)	200 mg (n=10)	400 mg (n=9)	600 mg (n=8)	Total (n=27)
Any Grade	**48 (94)**	8 (100)	6 (86)	8 (89)	22 (92)	10 (100)	9 (100)	7 (88)	26 (96)
Febrile neutropenia	**34 (67)**	5 (63)	4 (57)	8 (89)	17 (71)	8 (80)	4 (44)	5 (63)	17 (63)
Diarrhea	**27 (53)**	4 (50)	4 (57)	4 (44)	12 (50)	6 (60)	7 (78)	2 (25)	15 (56)
Platelet count decreased	**22 (43)**	7 (88)	4 (57)	4 (44)	15 (63)	3 (30)	2 (22)	2 (25)	7 (26)
Anemia	**19 (37)**	4 (50)	2 (29)	4 (44)	10 (42)	4 (40)	3 (33)	2 (25)	9 (33)
Nausea	**19 (37)**	4 (50)	3 (43)	3 (33)	10 (42)	4 (40)	2 (22)	3 (38)	9 (33)
Neutrophil count decreased	**18 (35)**	6 (75)	3 (43)	3 (33)	12 (50)	3 (30)	2 (22)	1 (13)	6 (22)
Constipation	**18 (35)**	5 (63)	2 (29)	2 (22)	9 (38)	5 (50)	2 (22)	2 (25)	9 (33)

[1]Lin et al. Blood Adv 2021 Mar 23;5(6):1719-1728 (NCT01696084).
Zeidan et al. ASH 2024 #214. Data cutoff: Oct 1, 2024.
TEAE, treatment-emergent adverse event.

komet-007
ACUTE LEUKEMIAS
KURA KO-MEN-007

SAFETY AND TOLERABILITY OF ZIFTOMENIB WITH 7+3 IN 1L AML (N=51)

Grade ≥3 TEAEs in ≥ 10% of All Patients

- One case (2%) of Gr3 differentiation syndrome (*NPM1*-m 600 mg); successfully managed and patient remained on treatment
- No ziftomenib-associated QTc prolongation
- No dose-limiting toxicities (DLTs) at any dose level

		NPM1-m				*KMT2A*-r			
TEAEs, n (%)	**All Patients (N=51)**	**200 mg (n=8)**	**400 mg (n=7)**	**600 mg (n=9)**	**Total (n=24)**	**200 mg (n=10)**	**400 mg (n=9)**	**600 mg (n=8)**	**Total (n=27)**
Grade ≥3	**46 (90)**	8 (100)	6 (86)	8 (89)	**22 (92)**	10 (100)	8 (89)	6 (75)	**24 (89)**
Febrile neutropenia	**30 (59)**	5 (63)	4 (57)	8 (89)	**17 (71)**	7 (70)	3 (33)	3 (38)	**13 (48)**
Platelet count decreased	**21 (41)**	7 (88)	4 (57)	3 (33)	**14 (58)**	3 (30)	2 (22)	2 (25)	**7 (26)**
Anemia	**18 (35)**	4 (50)	2 (29)	3 (33)	**9 (38)**	4 (40)	3 (33)	2 (25)	**9 (33)**
Neutrophil count decreased	**18 (35)**	6 (75)	3 (43)	3 (33)	**12 (50)**	3 (30)	2 (22)	1 (13)	**6 (22)**
White blood cell count decreased	**13 (26)**	3 (38)	2 (29)	2 (22)	**7 (29)**	2 (20)	3 (33)	1 (13)	**6 (22)**
Sepsis	**7 (14)**	2 (25)	0	2 (22)	**4 (17)**	1 (10)	1 (11)	1 (13)	**3 (11)**
Pneumonia	**6 (12)**	1 (13)	2 (29)	0	**3 (13)**	2 (20)	0	1 (13)	**3 (11)**

[1]Lin et al. Blood Adv 2021 Mar 23;5(6):1719-1728 (NCT01696084).
Zeidan et al. ASH 2024 #214. Data cutoff: Oct 1, 2024.
TEAE, treatment-emergent adverse event.

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CLINICAL ACTIVITY IN ALL RESPONSE-EVALUABLE* 1L PATIENTS (N=46)

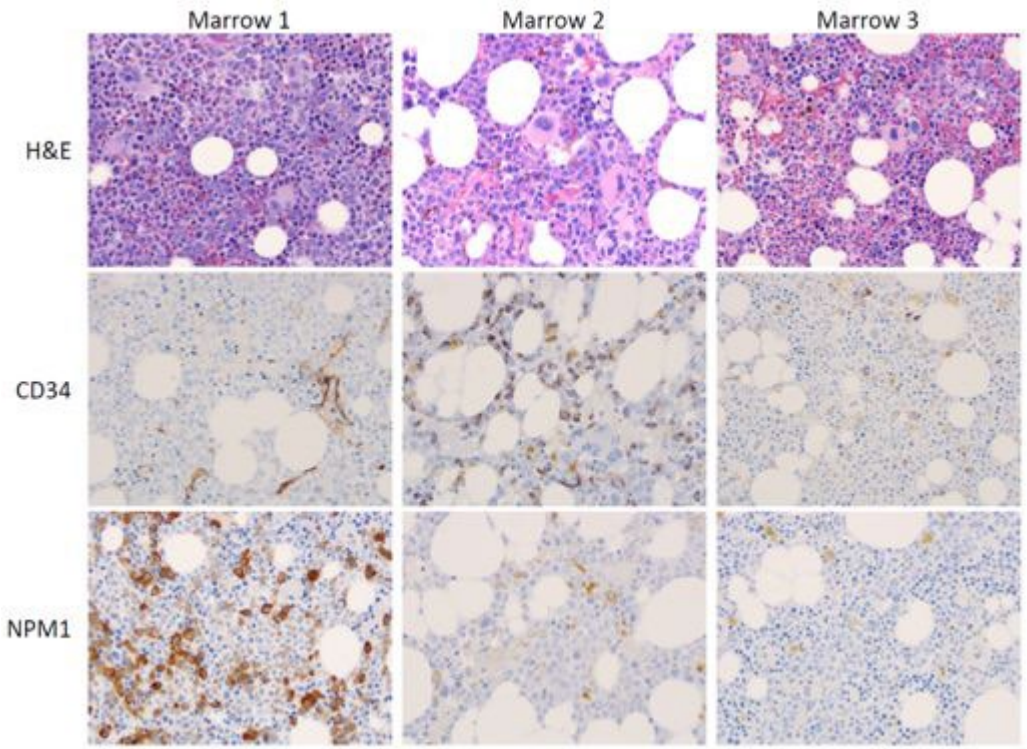
Historically, only 33% of 7+3 treated newly diagnosed, adverse-risk AML patients achieve CRc, with a median overall survival of ~6 months[1-2]

		NPM1-m				*KMT2A*-r			
	All Patients (N=46)	**200 mg (n=8)**	**400 mg (n=7)**	**600 mg (n=8)**	**Total (n=23)**	**200 mg (n=10)**	**400 mg (n=9)**	**600 mg (n=4)**	**Total (n=23)**
CRc	**42 (91)**	8 (100)	7 (100)	8 (100)	**23 (100)**	9 (90)	6 (67)	4 (100)	**19 (83)**
ORR	**42 (91)**	8 (100)	7 (100)	8 (100)	**23 (100)**	9 (90)	6 (67)	4 (100)	**19 (83)**
CR	**42 (91)**	8 (100)	7 (100)	8 (100)	**23 (100)**	9 (90)	6 (67)	4 (100)	**19 (83)**
CRh	0	0	0	0	0	0	0	0	0
CRi	0	0	0	0	0	0	0	0	0
MLFS	0	0	0	0	0	0	0	0	0
PR	0	0	0	0	0	0	0	0	0
NR	3(7)	0	0	0	0	0	3 (33)	0	3 (13)
NE	1(@)	0	0	0	0	1 (10)	0	0	1 (4)
MRD negativity, n/N**	**28/37 (76)**	8/8 (100)	4/6 (67)	4/7 (57)	**16/21 (76)**	5/8 (63)	5/6 (83)	2/2 (100)	**12/16 (75)**

* Patients who have ≥ 1 response assessment or who had died.
** Among CRc responders tested for MRD per local assay (NGS, RT-qPCR, FISH, flow cytometry).

1. Lin et al. *Blood Adv* 2021 Mar 23;5(6):1719-1728. 2. Lancet et al. *Blood* 2014 May 22;123(21):3239-46.
Zeidan et al. ASH 2024 #214. Data cutoff: Oct 1, 2024.
Per ELN 2022: CR, complete remission; CRc, composite complete remission; CRh, complete remission with partial hematological recovery; CRi, complete remission with incomplete hematological recovery; FISH, fluorescence in situ hybridization; MLFS, morphologic leukemia-free state; MRD, measurable residual disease; NE, not evaluable; NGS, next-generation sequencing; NR, no response; PR, partial remission; RT-qPCR, quantitative reverse transcription polymerase chain reaction.

60-YR-OLD FEMALE WITH NEWLY DIAGNOSED *NPM1*-m AML TREATED WITH ZIFTOMENIB AND 7+3



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Screening marrow: blasts 8%, $CD34^{-}$ $NPM1^{+}$ (IHC), NPM1 PCR 38%

Day 21 marrow: 30% morphologic blasts, but now $CD34^{+}$ $NPM1^{-}$ (IHC), NPM1 PCR 0.08%

Days 22–24: Platelet count recovering, decision to hold off salvage therapy and repeat marrow at Day 35

Day 35 marrow: 1% blasts, $NPM1^{-}$ (IHC), NPM1 PCR 0.05%

KEY CONSIDERATIONS:

- Distinguish refractory disease from differentiating / regenerating blasts
- Allow time for count recovery and re-assess bone marrow, especially when clinical picture suggests otherwise (*e.g.*, change in blast immunophenotype, recovering counts, high CR rate)

Source: 2024 ASH Investor Presentation

PRELIMINARY CLINICAL OUTCOMES IN 1L *NPM1*-m AML PATIENTS

For *NPM1*-m, after a median follow-up of 31 weeks (range 17–63):

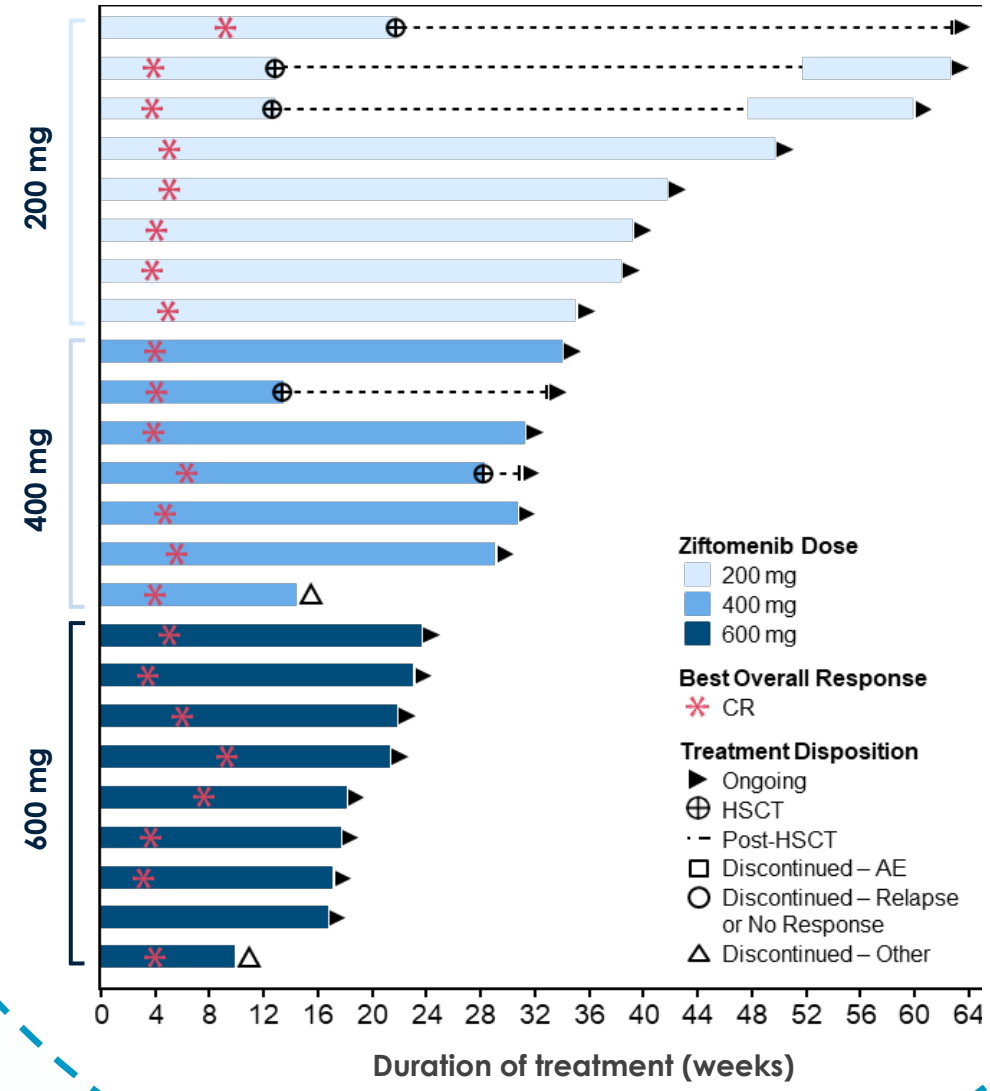
Median duration of CR **was not reached**

Median OS **was not reached**

5 *NPM1*-m patients received HSCT (200 mg n=3, 400 mg n=2); 2 went onto ziftomenib maintenance

No discontinuations due to AE or relapse

100% (24/24) patients remained alive



Zeidan et al. ASH 2024 #214. Data cutoff: Oct 1, 2024.
AE, adverse event; CR, complete remission; HSCT, hematopoietic stem cell transplant.

PRELIMINARY CLINICAL OUTCOMES IN 1L *KMT2A*-r AML PATIENTS

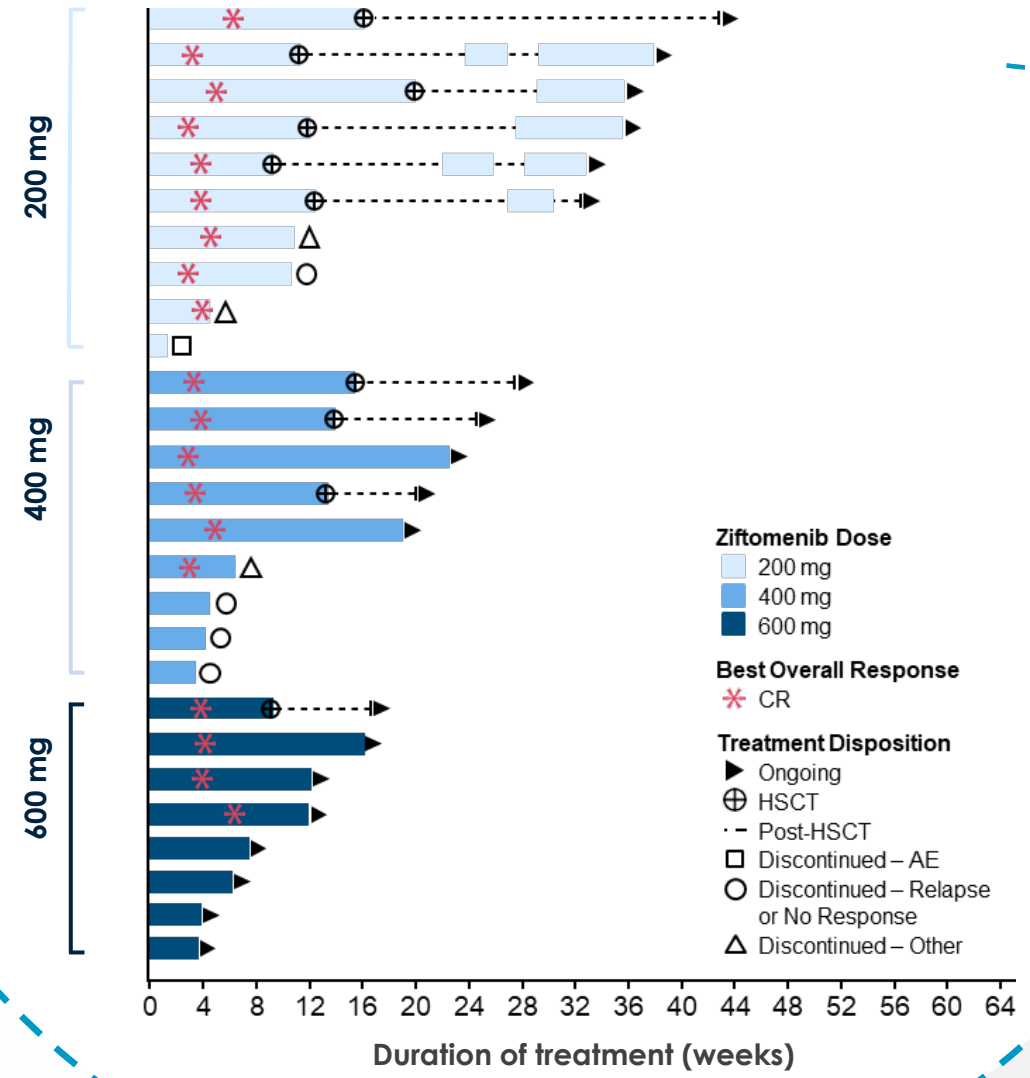
For *KMT2A*-r, after a median follow-up of 19 weeks (range 2–43):

Median duration of CR **was not reached**

Median OS **was not reached**

10 *KMT2A*-r patients received HSCT (200 mg n=6, 400 mg n=3, 600 mg n=1); 5 went onto ziftomenib maintenance

96% (26/27) patients remained alive



Zeidan et al. ASH 2024 #214. Data cutoff: Oct 1, 2024.
AE, adverse event; CR, complete remission; HSCT, hematopoietic stem cell transplant.

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ABSOLUTE NEUTROPHIL COUNT (ANC) AND PLATELET RECOVERY IN CRc RESPONDERS

Higher ziftomenib doses did not impact or delay neutrophil and platelet count recovery

	NPM1-m + *KMT2A*-r		
Median (range)	**200 mg (n=17)**	**400 mg (n=13)**	**600 mg (n=12)**
Days to ANC ≥ 0.5x10^9/L, Cycle 1	32 (20–40)	27 (20–40)	28 (19–38)
Days to ANC ≥ 1.0x10^9/L, Cycle 1	33 (21–62)	28 (20–40)	28 (20–48)
Days to Platelets ≥ 50x10^9/L, Cycle 1	28 (15–62)	26 (15–40)	26 (18–48)
Days to Platelets ≥ 100x10^9/L, Cycle 1	32 (20–62)	26 (18–40)	28 (20–48)

[1]Lin et al. Blood Adv 2021 Mar 23;5(6):1719-1728 (NCT01696084).
Zeidan et al. ASH 2024 #214. Data cutoff: Oct 1, 2024.
TEAE, treatment-emergent adverse event.

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CONCLUSIONS FROM ONGOING KOMET-007 STUDY

Ziftomenib combined with 7+3 was well tolerated across all dose levels

No DLTs or ziftomenib-associated QTc prolongation were reported

On-target DS occurred in 2% (n=1, Gr3), successfully managed and patient remained on treatment

Higher ziftomenib doses did not impact or delay ANC and platelet count recovery

Robust clinical activity was demonstrated in both *NPM1*-m and *KMT2A*-r subtypes

CR: 100% for *NPM1*-m, 83% for *KMT2A*-r patients

MRD negativity: 76% for *NPM1*-m, 75% for *KMT2A*-r patients

100% (24/24) of *NPM1*-m and 96% (26/27) *KMT2A*-r patients remained alive at data cutoff

We believe these data support advancement of ziftomenib with 7+3 in all 1L *NPM1*-m and *KMT2A*-r patients

Given encouraging safety, tolerability and clinical activity, the ongoing Phase 1b dose expansion is investigating 600 mg ziftomenib-based combinations in all newly diagnosed *NPM1*-m and *KMT2A*-r AML patients

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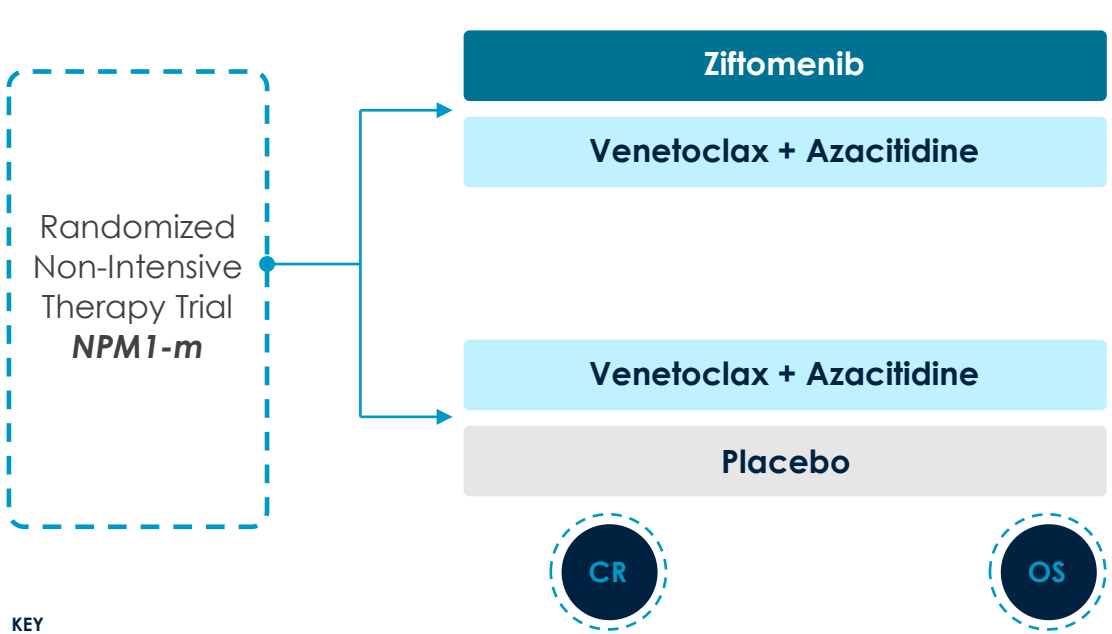
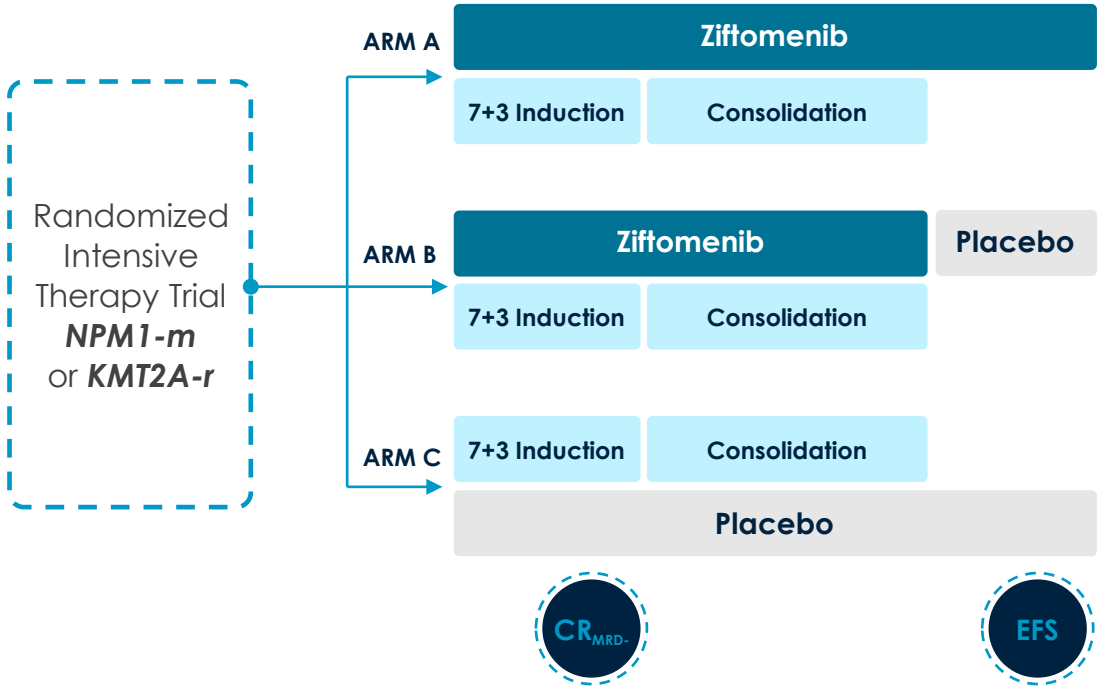
KOMET-017
TWO PHASE 3 TRIALS UNDER A SINGLE PROTOCOL

Provides Treatment Options to the Broadest Frontline AML Patient Pool

KOMET-017-NIC
(NON-INTENSIVE CHEMOTHERAPY)



KOMET-017-IC
(INTENSIVE CHEMOTHERAPY)



7+3 means seven days of cytarabine and 3 days of daunorubicin
CR, complete response; OS, overall survival; CR MRD-, complete response with minimal residual disease; EFS, event-free survival

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NON-INTENSIVE CHEMOTHERAPY (NIC) TRIAL

Randomized, Double-Blind, Placebo-Controlled Study: Venetoclax + Azacitidine + Ziftomenib or Placebo

Patient Population

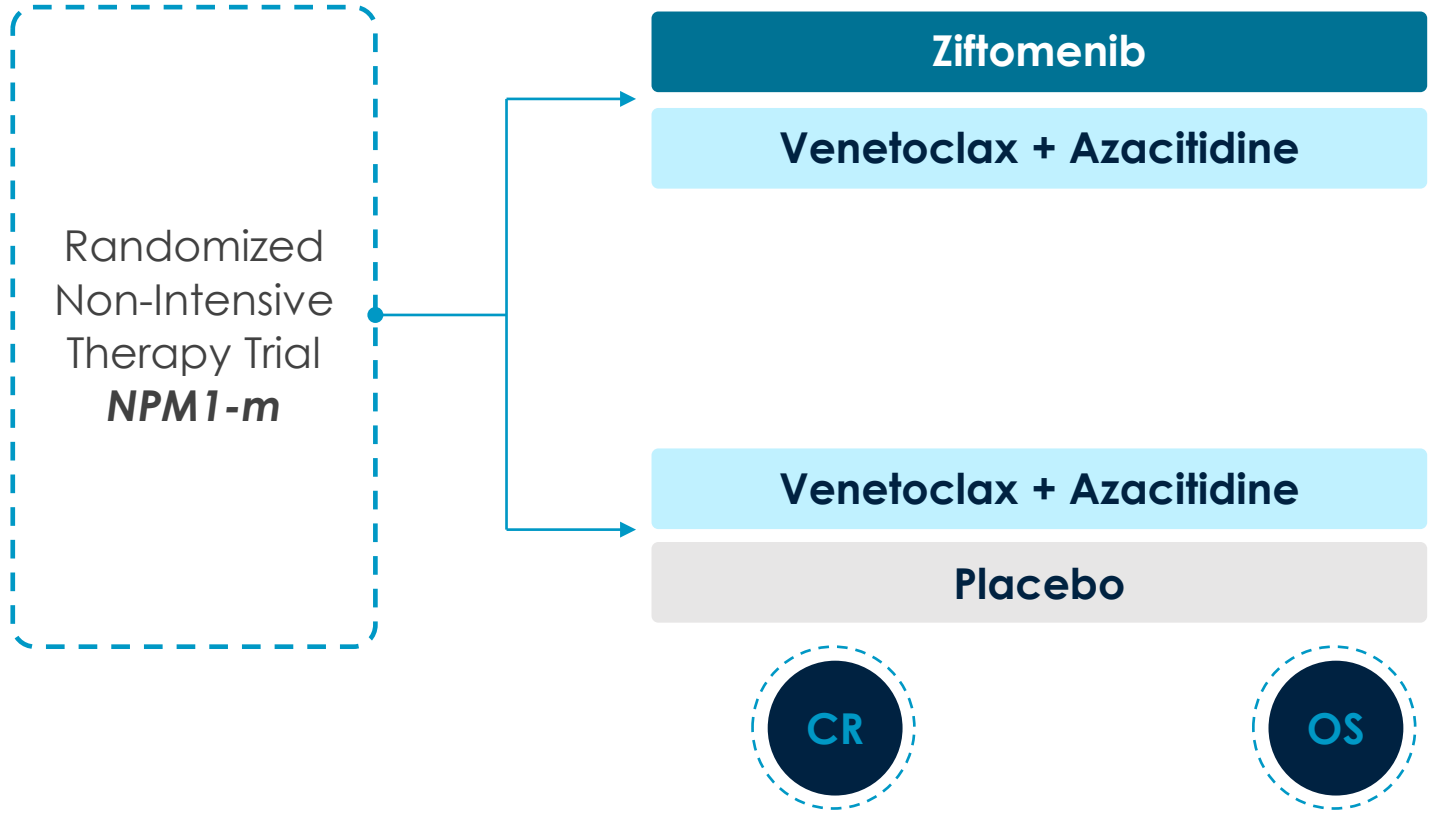
AML with NPM1 mutation

Age 75+ or < 75 with medical comorbidities

Dual Primary Endpoints

Complete Response

Overall Survival



KEY

Ziftomenib | SOC Backbone | Placebo

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NON-INTENSIVE CHEMOTHERAPY (NIC) TRIAL

FDA Aligned Around Modified Venetoclax Dosing Regimen

- Known myelosuppression with venetoclax + azacitidine from indicated dose and schedule has evolved in current clinical practice
- Standard practice has moved towards minimizing venetoclax exposure upon response
- Proposed dose modifications for use of venetoclax are based on long-term follow-up exposure data from VIALE-A trial[1] and real-world evidence (RWE) cohort results published by Othman *et al* 2024.[2]
- FDA agreement to allow modified dosing to incorporate real-world practice
    - Cycles 1 and 2: venetoclax days 1-28 per label
    - Cycles 3 and beyond: days 1-21
    - Further dose reductions for toxicity per label

1. Pratz KW, Jonas BA, Pullarkat V, *et al.* Long-term follow-up of VIALE-A: Venetoclax and azacitidine in chemotherapy-ineligible untreated acute myeloid leukemia. *Am J Hematol* 2024;99(4):615-624. **2.** Othman J, Lam HPJ, Leong S, *et al.* Real-world outcomes of newly diagnosed AML treated with venetoclax and azacitidine or low-dose cytarabine in the UK NHS. *Blood Neoplasia.* 2024;1(3):2950-3280.

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INTENSIVE CHEMOTHERAPY (IC) TRIAL

Randomized, Double-Blind, Placebo-Controlled Study: 7+3/Consolidation + Ziftomenib or Placebo

PROPOSED 3-ARM DESIGN TO CAPTURE MAINTENANCE INDICATION IN THE LABEL FOR NEWLY DIAGNOSED AML

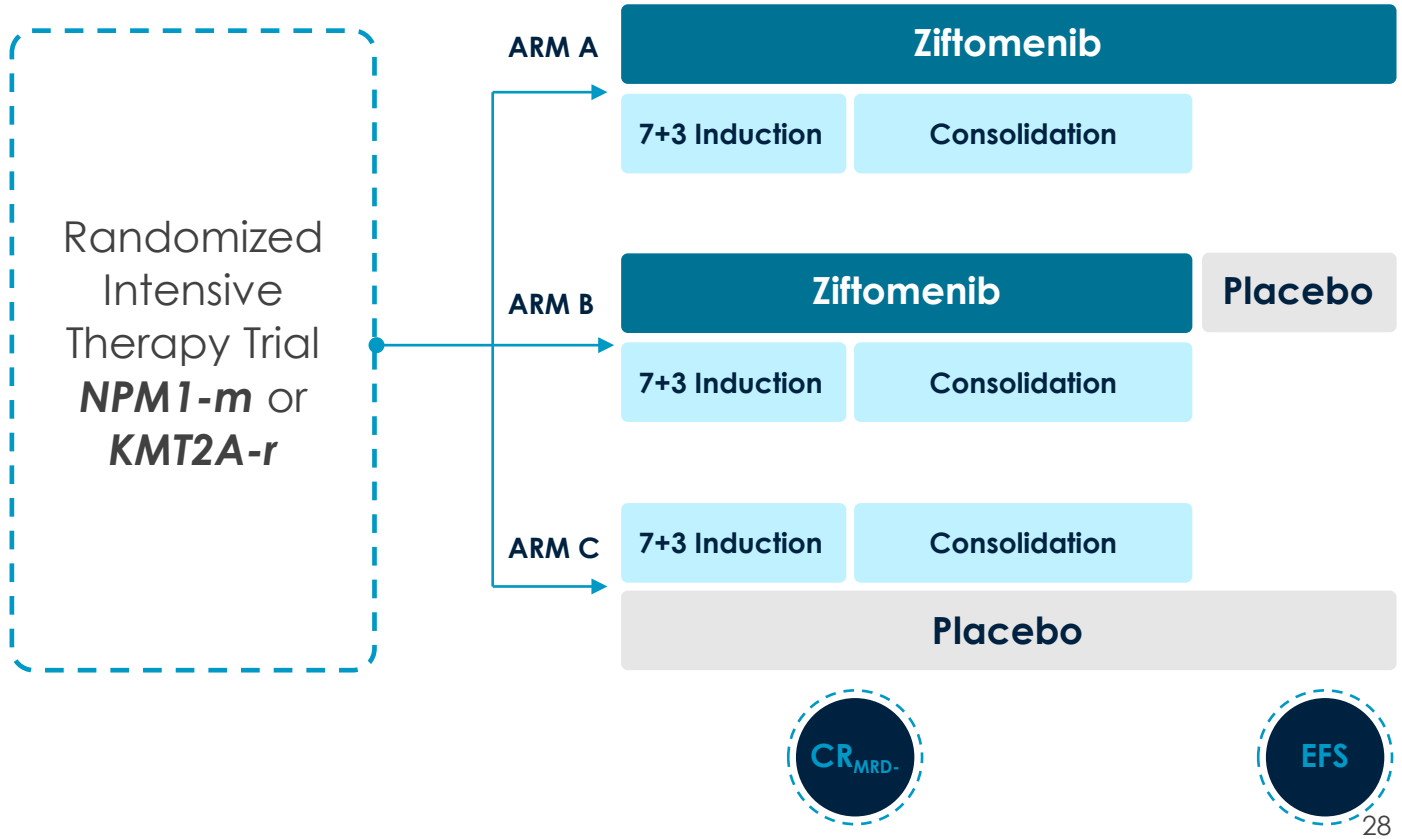
Primary Endpoints

CR_{MRD-}

as potential accelerated approval endpoint with accompanying supportive/external evidence on correlation of CR MRD-[1] and EFS/OS

EFS

powered to demonstrate additional benefit of post-consolidation maintenance



KEY

Ziftomenib | SOC Backbone | Placebo

[1] CR MRD- endpoint is only applicable to NPM1-m population in KOMET-017-IC trial

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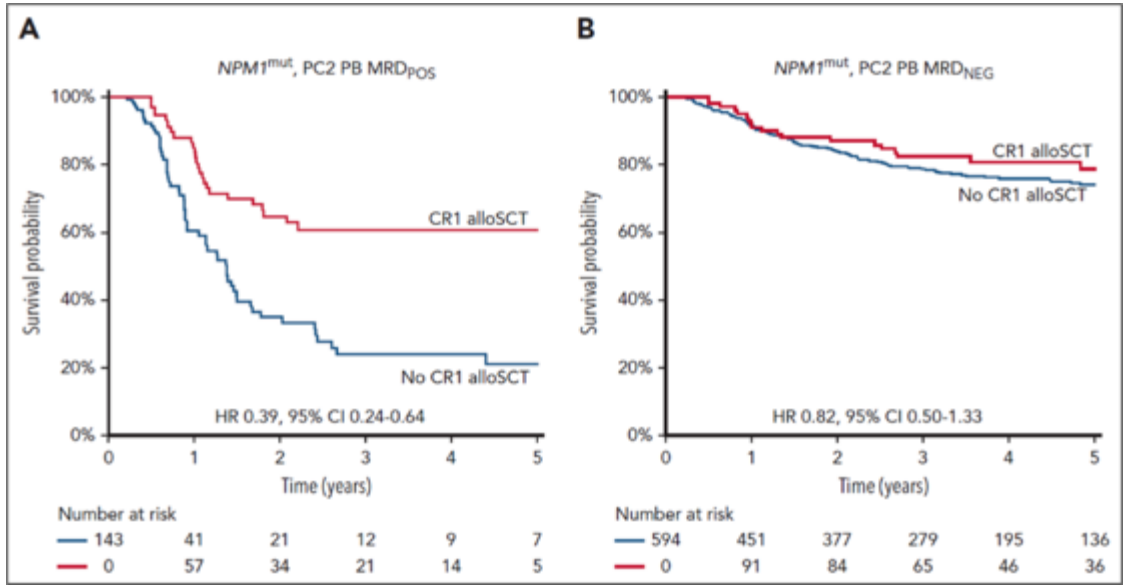
INTENSIVE CHEMOTHERAPY (IC) TRIAL

Importance of the CR_{MRD-} Endpoint

- Published data suggest CR MRD- may correlate better with long-term survival than morphologic CR alone
- Literature highlights significant impact of *NPM1*-m MRD negativity on long-term survival and the implication on consolidation therapy choices
- Higher rates of CR MRD negativity with ziftomenib have potential to diminish the need for allogeneic stem cell transplantation
- Utilizing this endpoint in KOMET-017-IC:
  - *Provides potential to pave the way in the field to establish this new surrogate endpoint*



Othman J, *et al.* Postinduction molecular MRD identifies patients with NPM1 AML who benefit from allogeneic transplant in first remission. *Blood* (2024) 143 (19): 1931–1936. Figure 2.

ZIFTOMENIB MARKET POTENTIAL IN NEWLY DIAGNOSED AML

High Unmet Medical Need

~70% of patients who achieve a first CR will relapse within 3 years[2]

~11% 5-year survival rate for AML patients aged ≥ 65 years[3]

Large Population & Potential for Sustained Benefit

~20,800 Newly diagnosed cases of AML each year in the U.S.[1]

12-24 months Potential for benefit / risk to support sustained treatment

Expansive Market Opportunity

$36-40k /month Analog pricing, including for recently approved product

>$7B/yr Annual U.S. market opportunity in 1L AML

Combination of encouraging clinical activity and safety in a once-daily oral medication could unlock a large market opportunity

AML, acute myeloid leukemia; CR, complete response.

1. American Cancer Society. Updated June 5, 2024. Accessed August 27, 2024. https://www.cancer.org/cancer/types/acute-myeloid-leukemia/about/key-statistics.html 2. Kumar CC. Genes Cancer. 2011;2(2):95-107. doi:10.1177/1947601911408076 3. National Cancer Institute. Accessed August 27, 2024. https://seer.cancer.gov/statisticsnetwork/explorer/application.

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PHASE 1/2 STUDY OF ZIFTOMENIB IN RELAPSED/ REFRACTORY AML

PHASE 1A Dose Escalation	PHASE 1B Validation Cohorts	PHASE 1B Validation Cohorts	PHASE 1B Expansion
Completed	*Completed*	*Completed*	*Enrollment Completed*
50 mg QD → 100 mg QD → 1000 mg QD	Cohort 1: 200 mg QD Cohort 2: 600 mg QD	Expansion of 600 mg QD	600 mg QD
NPM1-m, *KMT2A*-r, Other	*NPM1*-m or *KMT2A*-r	*NPM1*-m	*NPM1*-m

OBJECTIVES

• Safety and tolerability • Pharmacokinetics • Early evidence of antitumor activity	• Safety and tolerability • Pharmacokinetics • Clinical activity	Continue enrollment of Phase 1b validation cohort(s) consistent with FDA's Project Optimus • Safety and tolerability • Pharmacokinetics • Clinical activity	Primary endpoint: • CR/CRh Secondary endpoint: • Duration of CR/CRh • Transfusion independence • CR/CRh MRD negativity • Adverse events

CR, complete remission; CRh, complete remission with partial hematological recovery; FDA, United States Food and Drug Administration; MRD, measurable residual disease; R/R, relapsed/refractory; RP2D, recommended phase 2 dose.

ZIFTOMENIB DEMONSTRATES OPTIMAL PHARMACEUTICAL PROPERTIES

Ziftomenib demonstrates a dose-dependent increase in exposure up to RP2D at 600 mg

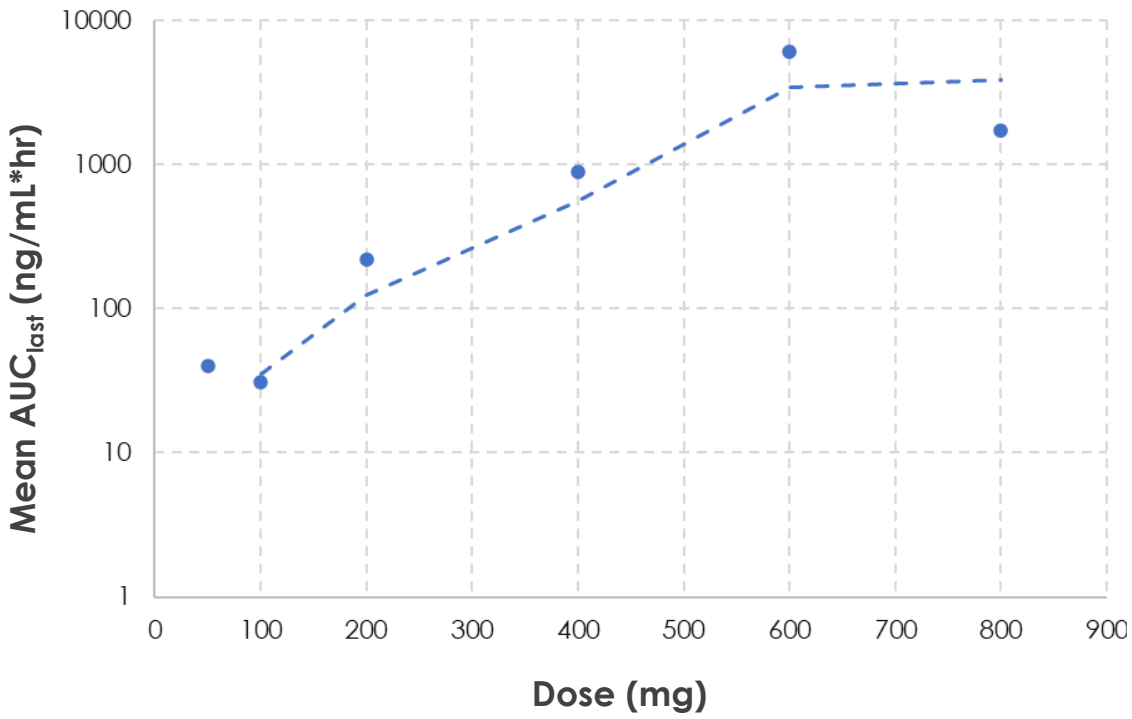
Ziftomenib is not a clinically meaningful CYP3A4 substrate

- No dose adjustment of ziftomenib needed when administered with a CYP3A4 inhibitor (*e.g.*, azoles)

Ziftomenib is not a clinically meaningful CYP3A4 inhibitor[1]

- No dose adjustment needed for CYP3A4 substrates (*e.g.*, venetoclax)

No drug-induced QTc prolongation observed



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[1] FDA Guidance (2020): Clinical Drug Interaction Studies – Cytochrome P450 Enzyme- and Transporter-Mediated Drug Interactions Guidance for Industry

ZIFTOMENIB DEMONSTRATES ENCOURAGING SAFETY PROFILE IN PHASE 1

Differentiation syndrome (DS) appears manageable in *NPM1*-m monotherapy patients with mitigation strategy

- 20% rate of mild to moderate DS

Rates of DS in *KMT2A*-r monotherapy patients were 42.9% at 200 mg and 38.9% at 600 mg; potential to mitigate in combination

DS is an on-target adverse event and represents evidence of clinical activity

No reports of drug-induced QTc prolongation

Maintained count recovery suggests no drug-induced myelosuppression

ZIFTOMENIB DEMONSTRATES MEANINGFUL CLINICAL ACTIVITY AS MONOTHERAPY

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40% of *NPM1*-m patients achieved a CR during course of study

Best Overall Response	600 mg
NPM1-m Phase 1a + 1b	**(n=20)**
CR	**7 (35.0)**
CR/CRh	7 (35.0)
CRc	8 (40.0)
MRD negativity	4 (50.0)[1]
ORR	9 (45.0)
KMT2A-r Phase 1a + 1b	**(n=18)**
CR/CRh	2 (11.1)
CRc	3 (16.7)
MRD negativity	3 (100.0)
ORR	3 (16.7)

DIFFERENTIATED CR RATES VS. SOC IN HEAVILY PRETREATED PATIENTS

	MUTATION	CR %	mDOR	MEDIAN PRIORS
Ziftomenib 600mg QD	***NPM1***	**35%**	**7.7** mo*	**3**
	FLT3	**33%**	-	
	IDH1/2	**50%**	-	
Gilteritinib	***FLT3***	14.2%	14.8 mo	1
Enasidenib	***IDH2***	19%	8.2 mo	2
Ivosidenib	***IDH1***	25%	10.1 mo	2

*Median DoR (Duration of Response) for CRc without censoring at HSCT
Source: USPI's

[1] MRD was assessed for 6/8 CRc patients; 4 of those 6 patients (67%) tested were MRD negative
CRc includes CR, CRh, CRi, CRp; ORR includes CR, CRh, CRi, CRp, MLFS
Wang *et al*. Lancet Oncol. 2024 Oct;25(10):1310-1324.

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KURA KO-MEN-001

CONCLUSIONS FROM KOMET-001 PHASE 1 CLINICAL TRIAL

Ziftomenib displays an encouraging safety and tolerability profile

Reported events most often consistent with features and manifestations of underlying disease

No evidence of drug-induced QTc prolongation

Differentiation syndrome, an on-target effect, manageable with mitigation strategy

Clinical activity of ziftomenib monotherapy is optimal at the 600 mg daily dose

Positive *NPM1*-m benefit/risk balance with pronounced activity and 35% CR rate (n=20)

High levels of ziftomenib tissue penetration drive clearance of extramedullary disease

Emergence of resistance mutations observed at a much lower rate relative to certain competitor

Monotherapy data supportive of combination strategies

No predicted adverse drug-drug interactions

Optimization of *KMT2A*-r benefit/risk planned via combination strategies to maximize time on treatment

Oral, QD dosing allows for convenient administration and combination with standards of care

Wang et al. Lancet Oncol. 2024 Oct;25(10):1310-1324

ZIFTOMENIB MARKET POTENTIAL IN R/R AML

High Unmet Need in R/R *NPM1*-m AML

20%
~50%

20% are primary refractory; ~50% will relapse who achieved an initial CR[1-4]

<10%

Fewer than 10% of all patients with R/R AML are alive at 5 years[5]

Potential for Sustained Treatment

~6 mo
Duration of Treatment

Potential for safe and well-tolerated targeted Tx to support sustained treatment

$36-40k /month

Analog pricing, including for recently approved product

Attractive Market Opportunity

$350-400M/yr

U.S. market opportunity in R/R *NPM1*-m AML

Combination of encouraging clinical activity and safety in a once-daily oral medication supports an attractive R/R opportunity

AML, acute myeloid leukemia; CR, complete response.

1. Bertoli S, et al. Blood. 2018;132(suppl 1):2802. 2. Hubmann M, et al. Haematologica. 2014;99(8):1317-1325. 3. SEER Cancer Stat Facts: Acute Myeloid Leukemia. National Cancer Institute. Bethesda, MD. Accessed March 14, 2023. https://seer.cancer.gov/statfacts/html/amyl.html. 4. Issa G, et al. Blood Adv. 2023;7(6):933-942. 5. DeWolf et. al, Blood 2020; 136 (9) 1023-1032.

KYOWA KIRIN STRATEGIC COLLABORATION

MAXIMIZING THE VALUE OF ZIFTOMENIB IN AML

STRATEGIC COLLABORATION WITH KYOWA KIRIN POSITIONS KURA TO UNLOCK THE FULL VALUE OF ZIFTOMENIB AND PIPELINE

- Complementary expertise and vision to capitalize on the full potential of ziftomenib in AML
- Kura retains leadership and key strategic rights to ziftomenib in the U.S. to preserve strategic flexibility
- Enables broad development and commercialization, including 1L fit/unfit, combos with targeted therapies and maintenance setting
- Along with current cash, collaboration has potential to fully fund ziftomenib AML program through to commercialization of 1L combinations – multi-$B opportunities
- Kura maintains rights to its programs while accelerating associated opportunities

SUMMARY OF COLLABORATION TERMS

SCOPE	Broad development & commercialization of ziftomenib in acute leukemias, including both fit and unfit 1L indications, post-transplant maintenance setting and combinations with targeted therapies
UPFRONT PAYMENT	Kura received $330M upon closing
NEAR-TERM MILESTONES	$420M, including milestone payments upon NDA filing and first commercial sale in the monotherapy R/R setting
TOTAL MILESTONES	$1.2B in total development, regulatory and commercial milestone payments
DEVELOPMENT	• Kura leads global development and U.S. regulatory* • Kyowa Kirin leads regulatory ex-U.S.
COMMERCIAL RIGHTS	• 50/50 US co-commercialization and profit/loss share • Kura books all U.S. revenue • Kyowa Kirin leads commercialization outside U.S.
OPT-IN RIGHT FOR SOLID TUMORS	Kyowa Kirin has ability to opt-in to development and commercialization of ziftomenib in gastrointestinal stromal tumors (GIST) and other solid tumors; triggers up to $228M in upfront and milestone payments (in addition to $1.2B above)
EX-U.S. ROYALTIES	Tiered double-digit royalties

* Kura funds the development costs until the end of 2028, and from 2029 onwards, both companies will share the costs at a 50:50 ratio

ZIFTOMENIB

MENIN INHIBITOR IN GIST

Targeted Investigational Menin Inhibitor in Combination with Imatinib for Treatment of Gastrointestinal Stromal Tumors (GIST)

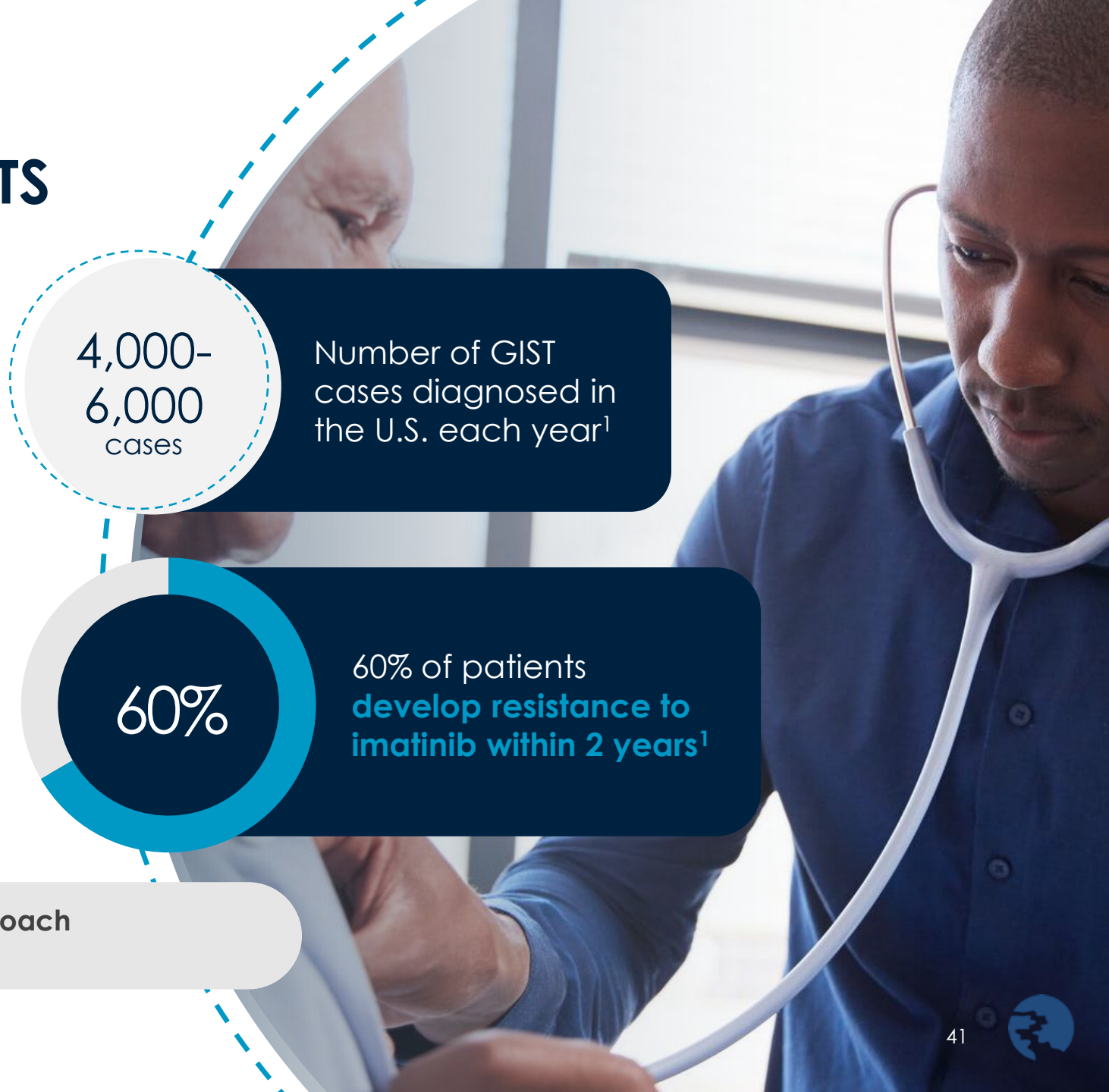
SIGNIFICANT UNMET NEED REMAINS FOR GIST PATIENTS

Gastrointestinal Stromal Tumor (GIST)

Tumors can start anywhere in the GI tract, but they occur most often in the stomach (about 60%) or the small intestine (about 35%)

Current FDA-approved therapies include imatinib, sunitinib, regorafenib and ripretinib

Until now, all approaches have targeted KIT inhibition via tyrosine kinase inhibitors (TKIs)



Menin inhibition is a potentially paradigm-altering approach to the treatment of GIST

1. https://www.cancer.org/cancer/types/gastrointestinal-stromal-tumor/about/key-statistics.html. American Cancer Society; 2. Gramza, A.W., Corless, C.L. and Heinrich, M.C., *Clin Cancer Res* (2009) 15 (24): 7510–7518.

A UNIQUE OPPORTUNITY FOR ZIFTOMENIB IN GIST

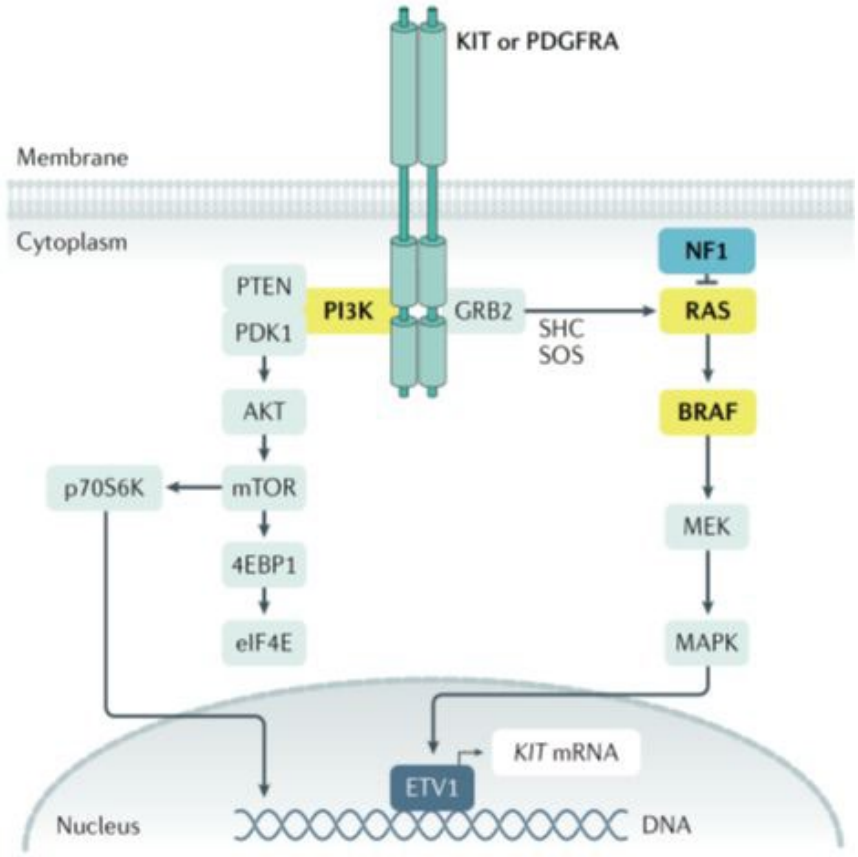
komet-015
GI STROMAL TUMORS
KURA KO-MEN-015

Ziftomenib + imatinib combination treatment showed robust antitumor activity in GIST PDX models representing the full GIST treatment continuum

Ziftomenib + imatinib exerts antitumor activity by a synthetic lethal mechanism through which ziftomenib epigenetically targets a vulnerability of GIST tumors, which is actively induced by TKI treatments

First patients dosed in proof-of-concept study of ziftomenib + imatinib in patients with advanced GIST after imatinib failure in 1H 2025

SIGNALING PATHWAYS OF KIT-MUTATED GIST[1]



[1] 1 Blay JY et al. Primer 2021, modified

ZIFTOMENIB OFFERS POTENTIAL TO SHIFT THE TREATMENT PARADIGM IN GIST

komet-015
GI STROMAL TUMORS
KURA KO-MEN-015

Ziftomenib may overcome resistance to imatinib

1L	2L	3L	4L
ORR ~ 60% PFS ~ 19 months	ORR ~ 7% PFS ~ 6 months	ORR ~ 5% PFS ~ 4.8 months	ORR ~ 9% PFS ~ 6.3 months
Imatinib	**Sunitinib**	**Regorafenib**	**Ripretinib**
1L Market Opportunity: 2,000 Patients Per Year [2]	2L Market Opportunity: 1,400 Patients Per Year [2]	3L Market Opportunity: 1,300 Patients Per Year [2]	4L Market Opportunity: 1,100 Patients Per Year [2]

Ziftomenib may prevent resistance to imatinib

60% of patients develop resistance to imatinib within 2 years[1]

1 FDA Guidance (2020): Clinical Drug Interaction Studies – Cytochrome P450 Enzyme- and Transporter-Mediated Drug Interactions Guidance for Industry

2 Estimated New Annual Treatable Patients Per Year – Epi: K. Søreide et al. / Cancer Epidemiology 40 (2016) 39–46; Kelly et al. J Hematol Oncol (2021) 14:2; 1L Imatinib: Assumes mid-point of annual GIST incidence (5K) * (35% metastatic + 7.5% (from locally advanced) + 7.5% (localized)) = 2,500 * .85% KIT; Assume 95% drug treatment rate in 1L ~2k; 2L: 1L to 2L progression having developed resistance to imatinib varies (60-75%), utilize 75% here ~1500 patients * 95% drug treatment rate. Note from 2L to 3L to 4L progression and treatment assumed at 90%

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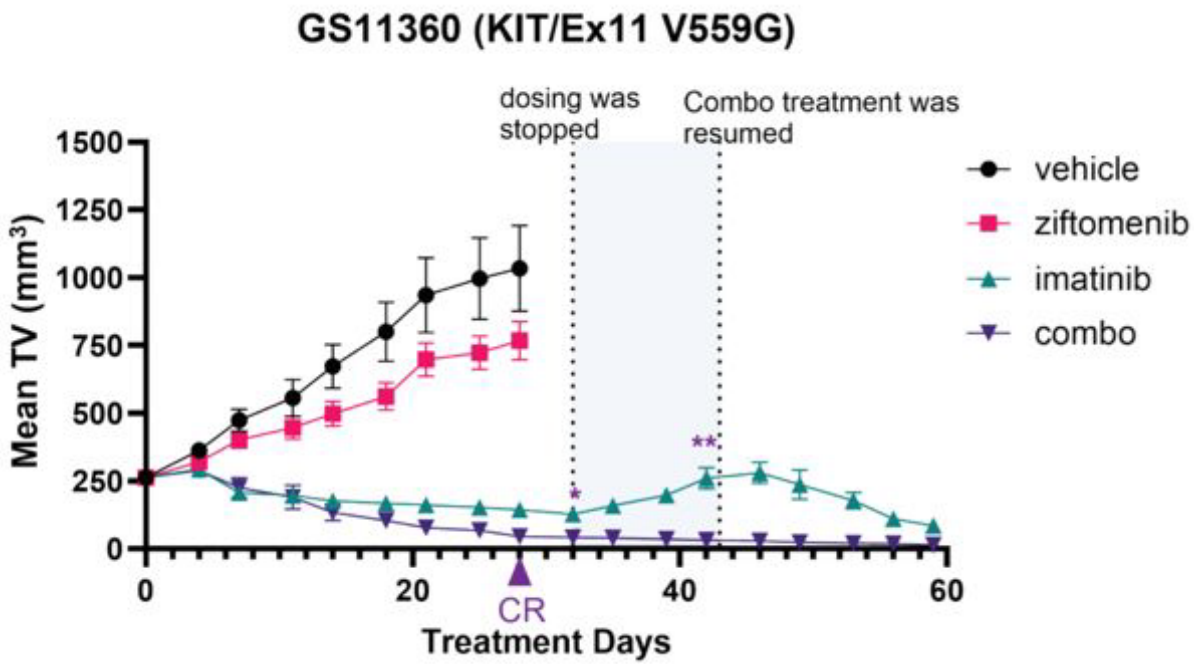
ZIFTOMENIB + IMATINIB COMBINATION DISPLAYS DURABLE ANTITUMOR ACTIVITY IN 1L GIST PDX MODEL

Imatinib monotherapy was tumoristatic, but the ziftomenib + imatinib combination induced deep regressions in all animals, including some CRs

Cessation of dosing resulted in rapid regrowth in imatinib-treated tumors, but tumors treated with the combination continued to regress

The relapsed imatinib-treated tumors regressed when exposed to the ziftomenib-imatinib combination



Data were presented as Mean ± SEM; t-test;
*P<0.05,**P<0.01,***P<0.001,****P<0.0001, not significant (ns)

McCloskey et al. ENA 2024 #227

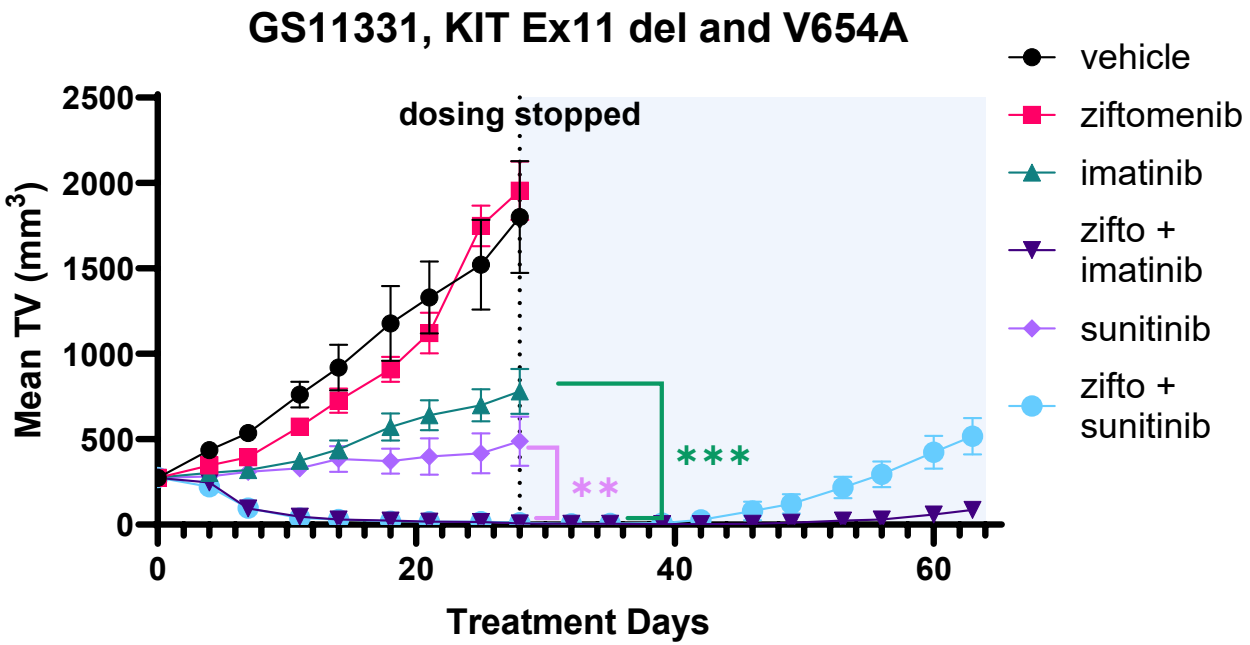
komet-015
GI STROMAL TUMORS
KURA KO-MEN-015

ZIFTOMENIB AND TKIs SYNERGIZE IN IMATINIB-RESISTANT PDX MODELS

Robust and durable responses seen in Ex13 GIST PDX model with combo

Both TKIs synergized with ziftomenib, inducing deep regressions in all animals, including some complete responses

Full suppression of tumor growth was maintained for up to four weeks after dosing stop



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*P<0.05,**P<0.01,***P<0.001,****P<0.0001, not significant (ns)

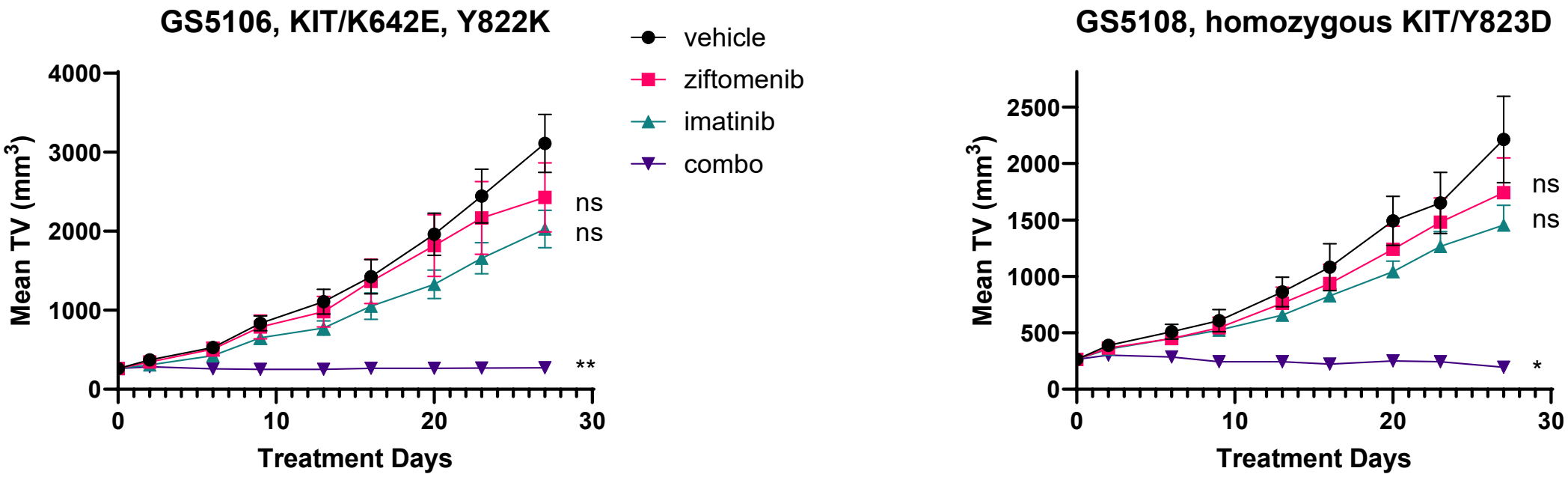
McCloskey et al. ENA 2024 #227

komet-015
GI STROMAL TUMORS
KURA KO-MEN-015

ZIFTOMENIB AND TKIs SYNERGIZE IN IMATINIB-RESISTANT PDX MODELS

Ziftomenib-imatinib combination antitumor activity is independent of KIT mutation type

SYNTHETIC LETHAL ACTIVITY OBSERVED IN IMATINIB-RESISTANT EX17 GIST PDX MODELS



Data were presented as Mean ± SEM; t-test; *P<0.05,**P<0.01, not significant (ns)

ZIFTOMENIB MARKET POTENTIAL IN GIST

komet-015
GI STROMAL TUMORS
KURA KO-MEN-015

Meaningful Unmet Need

~4-6K

Newly diagnosed cases of GIST each year in the U.S.[1]

~60%

Patients who **develop resistance** to imatinib **within 2 years**[2]

Potential for Sustained Benefit

24+ months

Potential for benefit / risk to support sustained treatment

$35k /month

Analog pricing, including for approved KIT inhibitors

Attractive Market Opportunity

$500M -$1B peak

Potential for benefit / risk to support sustained treatment

Ability to combine with imatinib in a once-daily oral medication could unlock a significant market opportunity

1. https://www.cancer.org/cancer/types/gastrointestinal-stromal-tumor/about/key-statistics.html. American Cancer Society;
2. Gramza, A.W., Corless, C.L. and Heinrich, M.C., Clin Cancer Res (2009) 15 (24): 7510–7518.

MENIN AML GIST DIABETES

MENIN INHIBITORS

FOR TREATMENT OF DIABETES

Targeted Investigational Menin Inhibitor for Treatment of Diabetes

DIABETES REPRESENTS A DISEASE EPIDEMIC

~1.2M New cases of diabetes each year in the U.S.[1]

8th Diabetes is the eighth leading cause of death in the U.S.

PREVALENCE

38.4 million adults (11.6%) in the United States have diabetes

- 29.7 million adults have diagnosed diabetes
- 8.7 million adults have undiagnosed diabetes

97.6 million adults in the U.S. have prediabetes

DIABETES AND PANCREATIC BETA-CELL FUNCTION

Beta cells are found in the pancreas and are responsible for the synthesis and secretion of insulin. Insulin is a hormone that helps the body use glucose for energy and helps control blood glucose levels.

A decline in beta-cell function and/or mass has been defined as a key contributing factor to disease progression in type 2 diabetes.

Loss of functional beta-cell mass is a core component of the natural history in type 2 diabetes (mediated by metabolic dysfunction).

American Diabetes Association. Statistics About Diabetes: https://diabetes.org/about-diabetes/statistics/about-diabetes

MENIN INHIBITORS OFFER UNIQUE ATTRIBUTES FOR DIABETES

Impact both insulin deficiency and insulin resistance

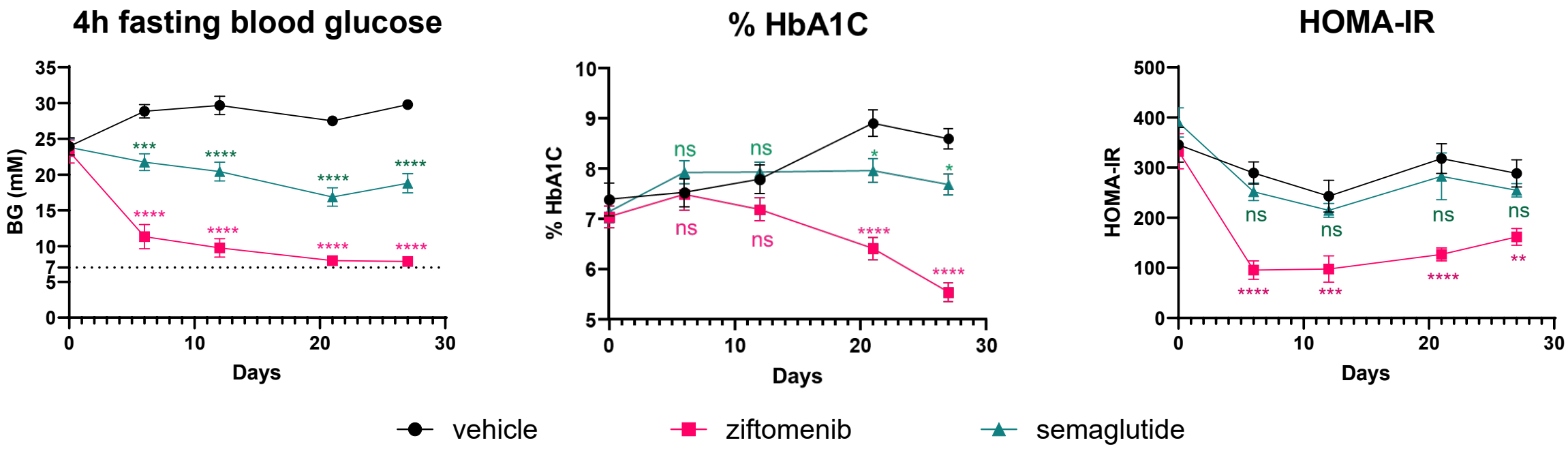
Potential for a first-in-class regenerative medicine that can **restore β-cell mass** and **improve insulin resistance**

Proof-of-concept studies with ziftomenib showed:

- Effects on fasting blood glucose, fasting insulin, and c-peptide levels maintained for a month after dosing cessation in a diabetic mouse model
- Selectivity for β-cells in islets is unique to the menin inhibitor class and may offer safety advantages over e.g., DYRK1 inhibitors
- Unpredicted effects of ziftomenib on insulin resistance suggest combination opportunities with T2D SOC, e.g., metformin, semaglutide

Nomination of next generation development candidate expected in mid-2025

ZIFTOMENIB REDUCES BLOOD GLUCOSE AND HbA1C LEVELS AND IMPROVES INSULIN SENSITIVITY IN ZDF RAT MODEL OF TYPE 2 DIABETES

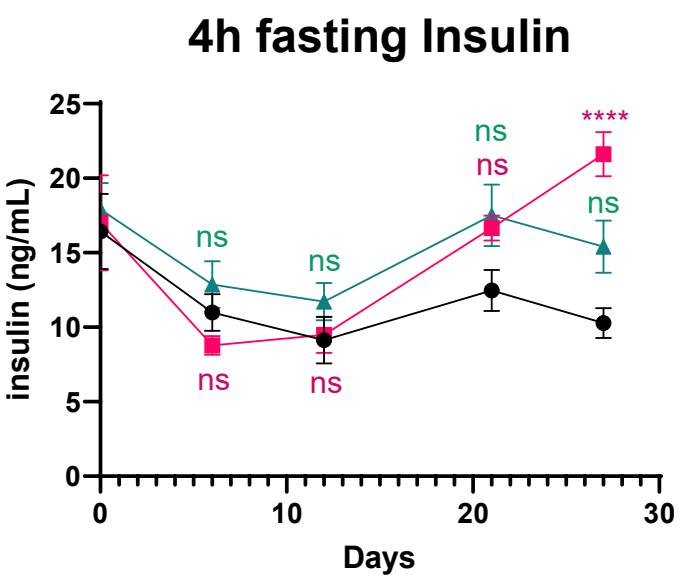


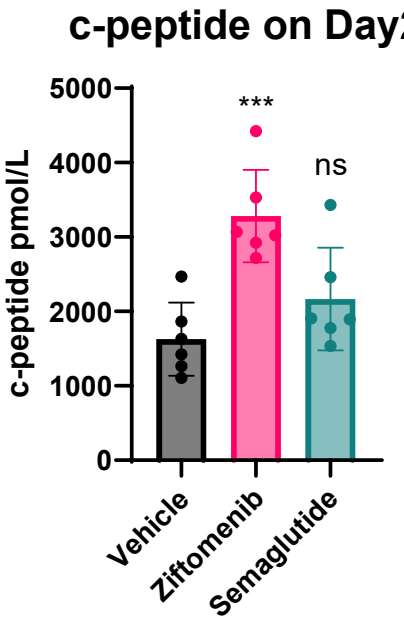
ns, not significant; *, p<0.05; **, p<0.01; ***, p<0.005; ****, p<0.001

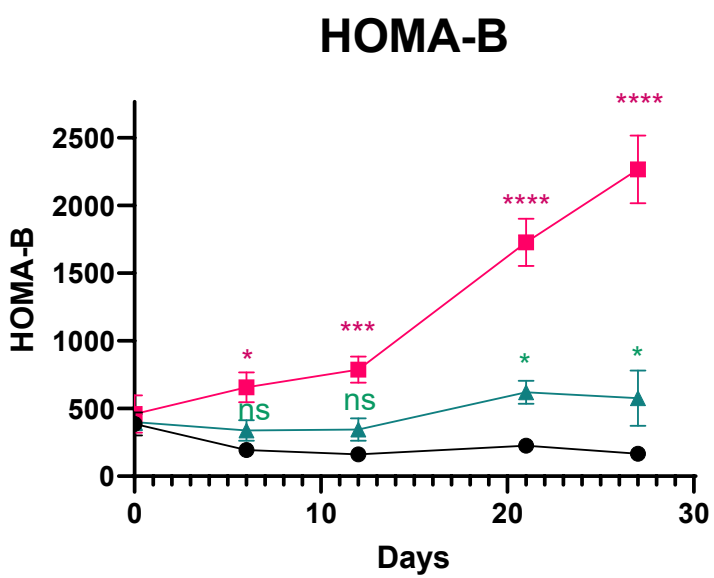
McCloskey et al. ADA 2024 #845-P
ZDF: Zucker Diabetic Fatty

ZIFTOMENIB STIMULATES INSULIN PRODUCTION

Ziftomenib significantly increased serum insulin levels and serum c-peptide levels, indicating significant improvement to steady-state β-cell function







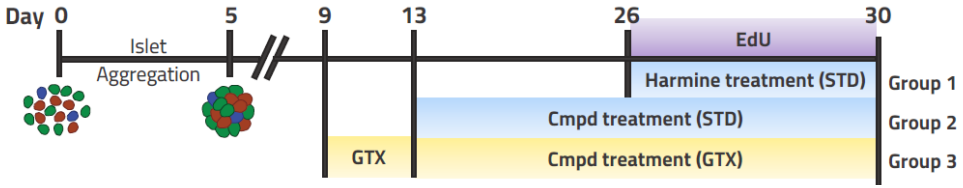
vehicle ziftomenib semaglutide

ns, not significant; *, p<0.05; **, p<0.01; ***, p<0.005; ****, p<0.001

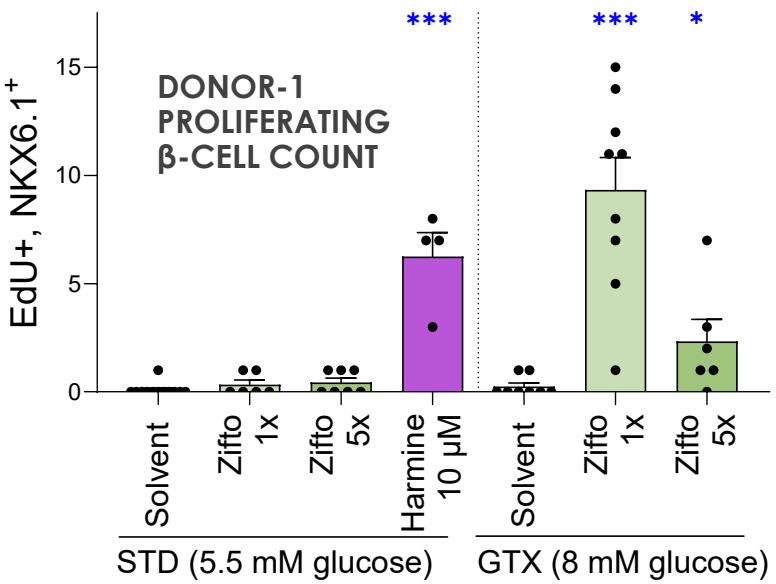
McCloskey et al. ADA 2024 #845-P
ZDF: Zucker Diabetic Fatty

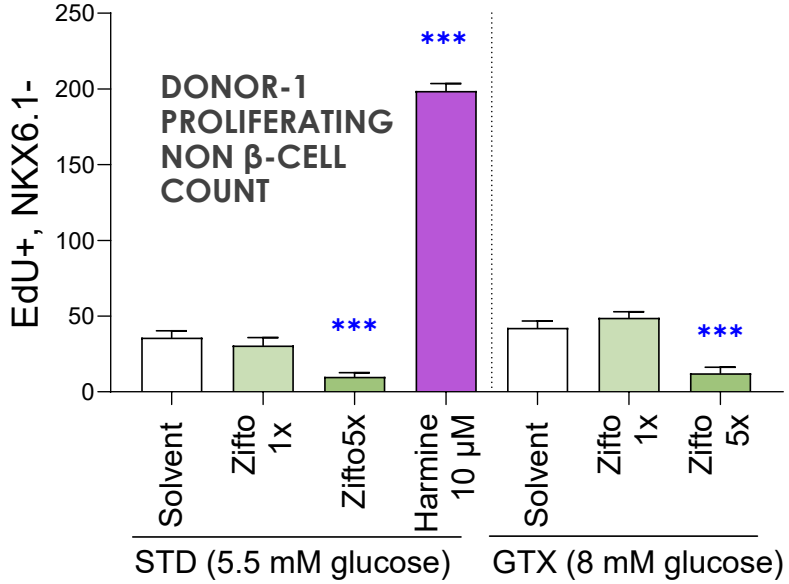
ZIFTOMENIB STIMULATES β-CELL PROLIFERATION WITH MINIMAL EFFECTS ON NON-β-CELLS IN HUMAN PANCREATIC ISLET MICROTISSUES

Ziftomenib stimulated the proliferation of β-cells specifically in donor samples



- Ziftomenib: 1x (nM) or 5x (nM)
- Harmine: 10 µM (effects are expected to be the same either under STD or GTX condition)
- Proliferating β-cells: NKX6.2+, EdU+
- Proliferating non-β-cells: NKX6.2-, EdU+





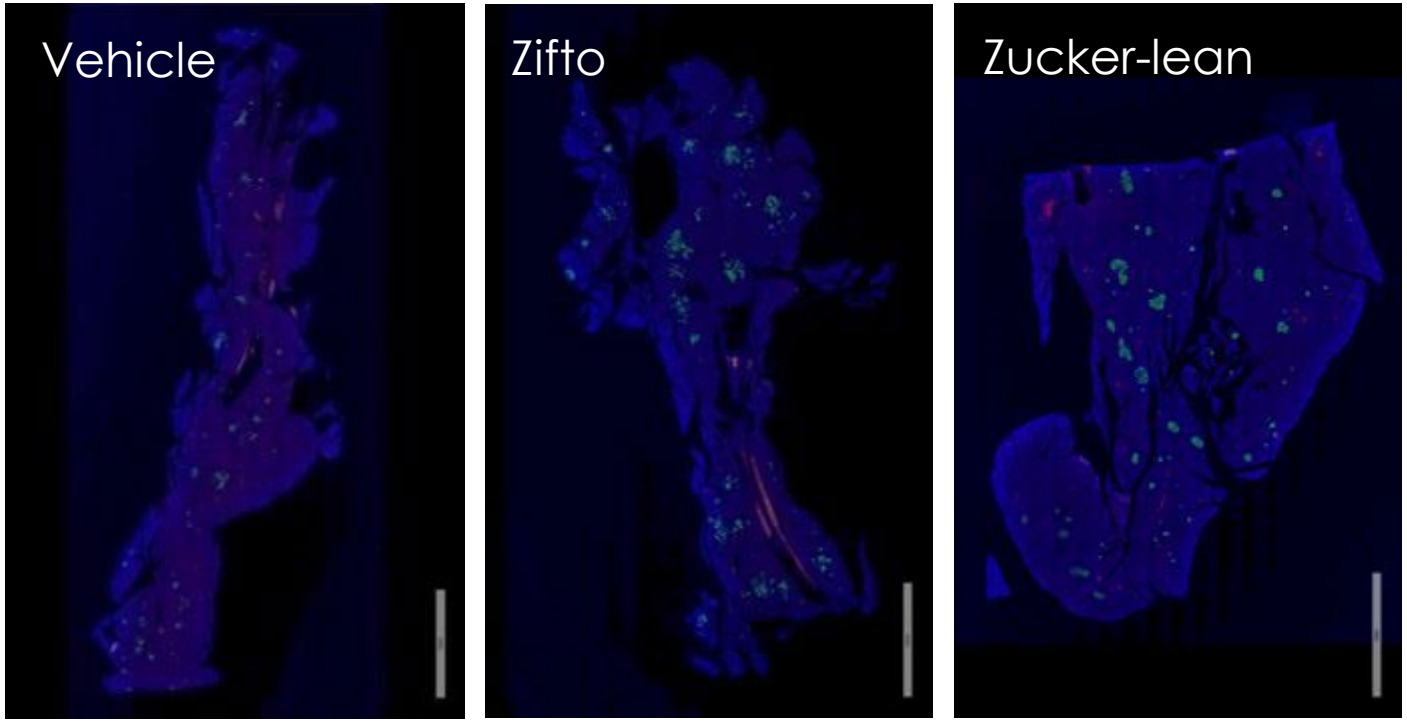
*, p<0.05; ***, p<0.005

GTX, glucotoxic conditions; STD, standard conditions

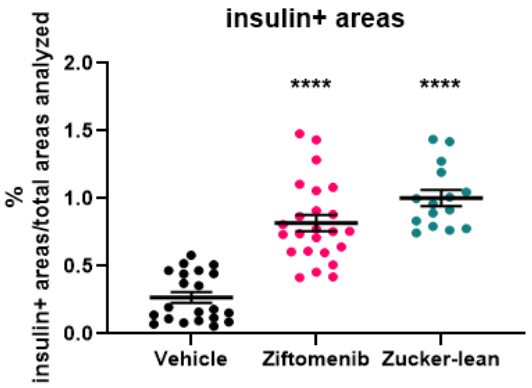
ZIFTOMENIB TREATMENT RESTORED β-CELL MASS IN ZDF RAT PANCREAS

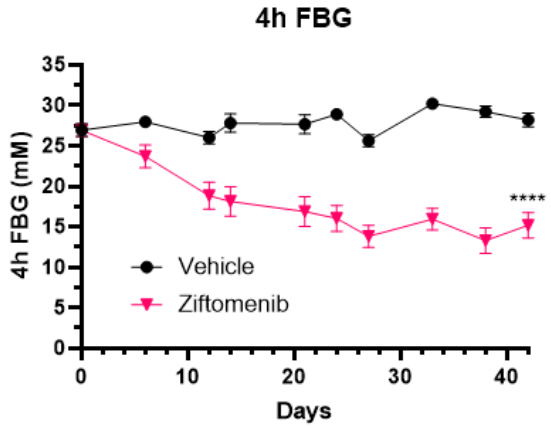
INSULIN+ AREAS EXPANDED AFTER 42 DAYS OF ZIFTOMENIB TREATMENT

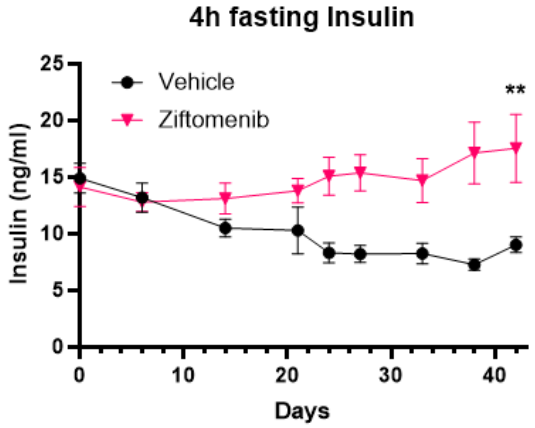
TAILS OF PANCREAS, DAPI + INSULIN



ns; not significant; *, p<0.05; **; p<0.01, ***, p<0.005; ****, p<0.001







MENIN INHIBITION FOR DIABETES DEVELOPMENT PLAN

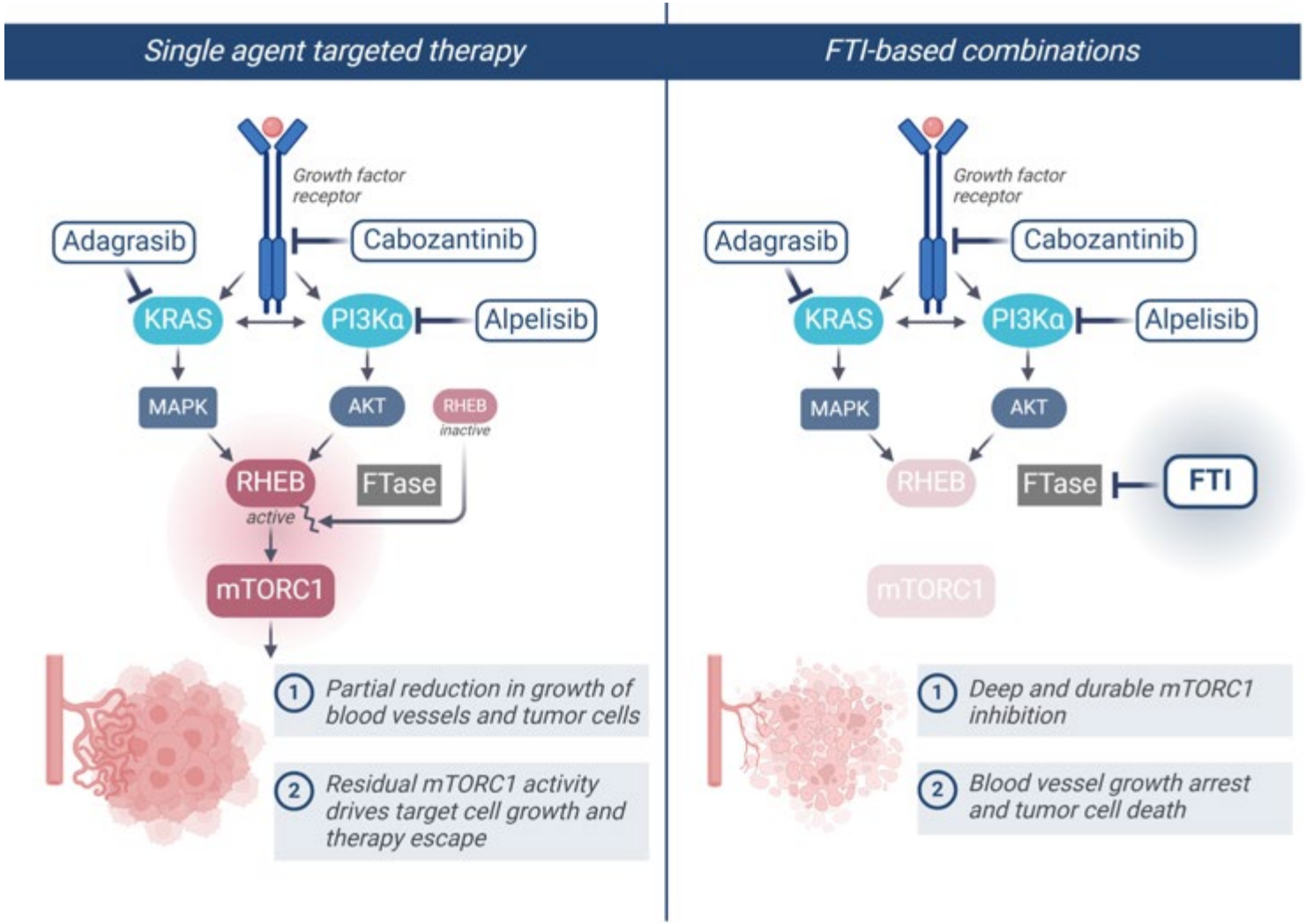
- REPORT PRECLINICAL DATA HIGHLIGHTING POTENTIAL FOR MENIN INHIBITORS IN DIABETES ✓
- NOMINATE NEXT-GEN MENIN INHIBITOR DEVELOPMENT CANDIDATE FOR EVALUATION IN DIABETES
- INITIATE IND-ENABLING STUDIES FOR DEVELOPMENT CANDIDATE
- FILE IND APPLICATION FOR DEVELOPMENT CANDIDATE
- INITIATE FIRST-IN-HUMAN STUDIES

KO-2806

A POTENT, SELECTIVE COMPANION INHIBITOR TO TARGETED THERAPIES

Class-leading drug candidate to address innate and adaptive resistance to various classes of targeted therapies

DESPITE ADVANCES, RESISTANCE REMAINS A CHALLENGE FOR MANY CLASSES OF TARGETED THERAPIES IN CANCER



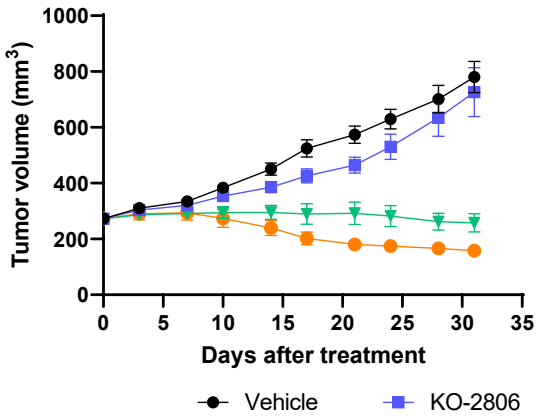
- Persistent mTORC1 activity seems to be a frequent and consistent non-genetic driver of inherent and adaptive resistance to multiple classes of targeted therapies (*e.g.*, KRAS inhibitors, PI3Kα inhibitors, TKIs in RCC)

- This mTORC1 vulnerability is targetable with a farnesyl transferase inhibitor, which blocks mTORC1 activation via de-farnesylation of RHEB while sparing mTORC2 inhibition and its associated toxicities.

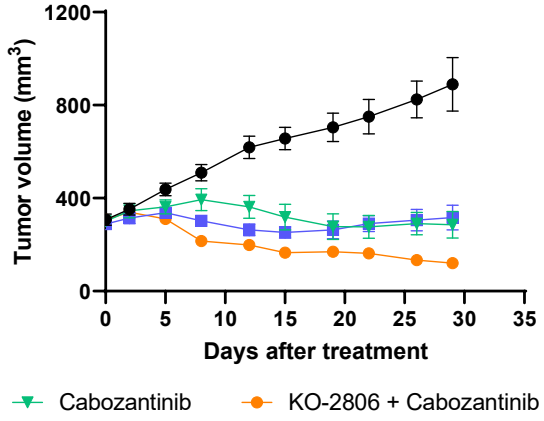
KO-2806 POTENTIATES THE ANTITUMOR ACTIVITY OF CABOZANTINIB IN ccRCC MODELS

The anti-angiogenic activity of cabozantinib is enhanced by addition of KO-2806

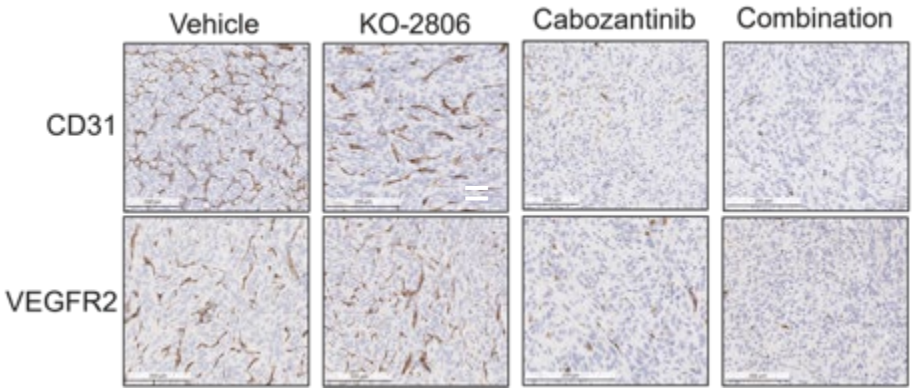
786-O CDX

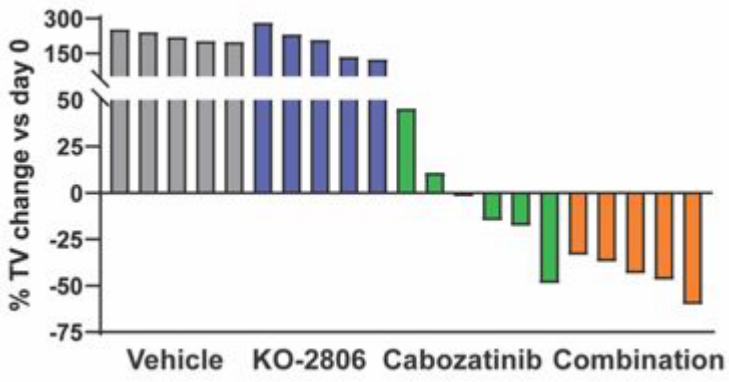


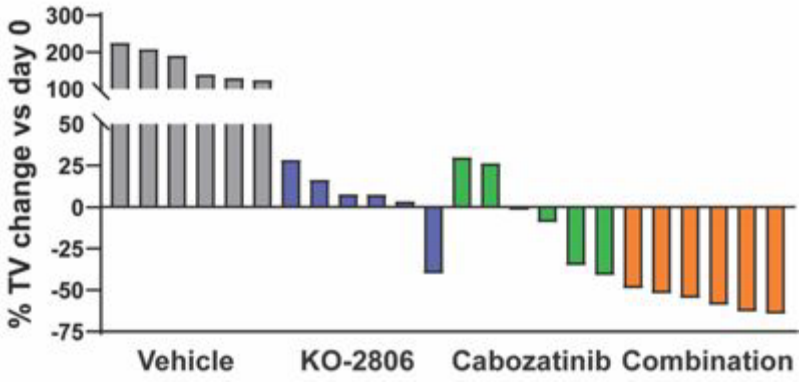
KI-12-0073 PDX



ANGIOGENIC MARKERS IN 786-O CDX TUMORS



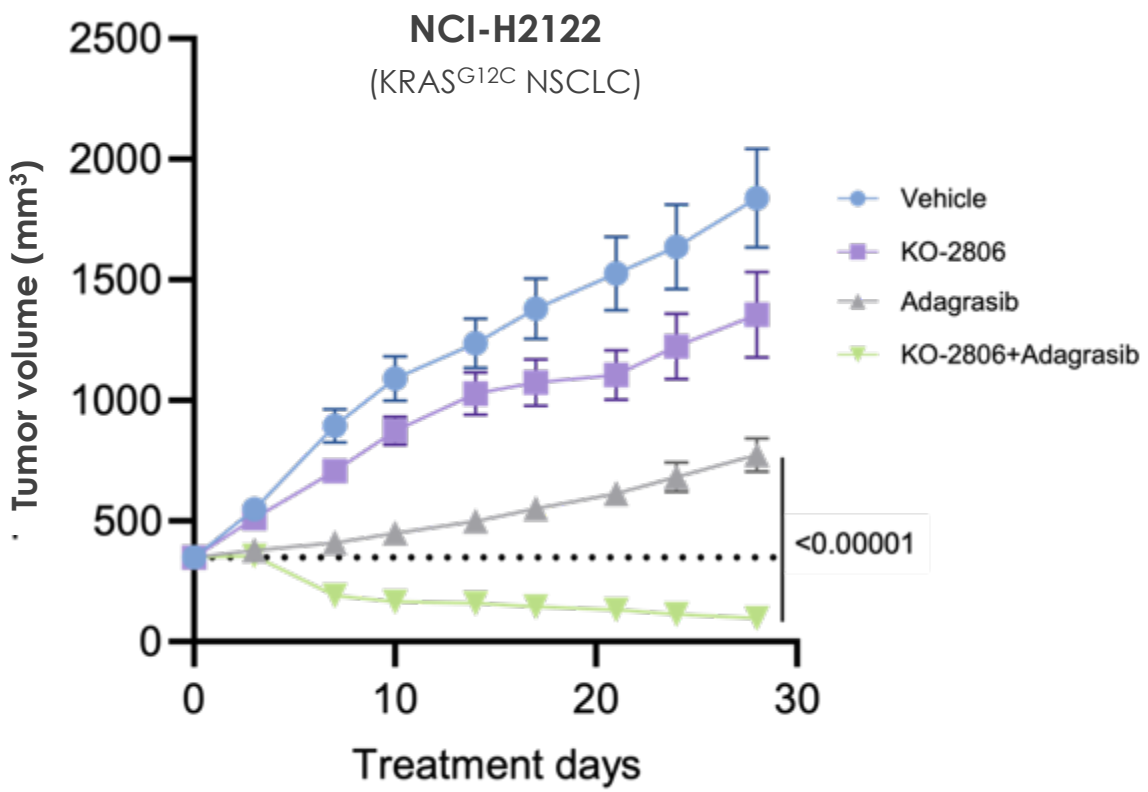


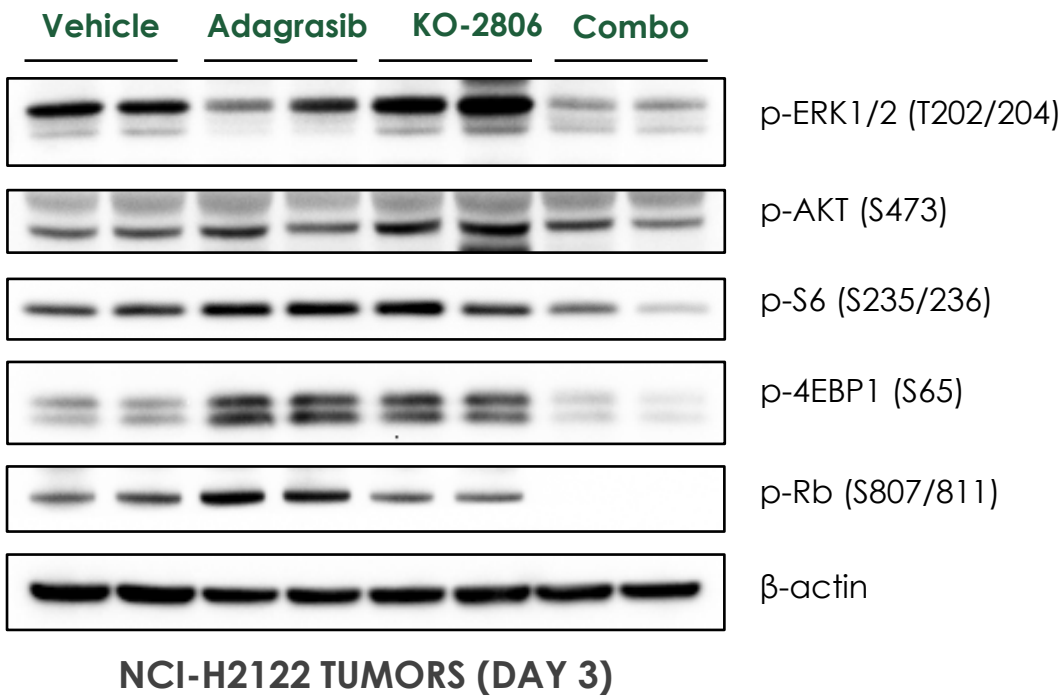


Gatchalian J. et al, AACR-NCI-EORTC 2023 Abstract #34634

ADDITION OF KO-2806 ENHANCES THE ANTITUMOR EFFICACY OF A KRAS^{G12C} INHIBITOR IN NSCLC MODELS

Combination of KO-2806 with adagrasib causes tumor regressions through suppression of mTOR signaling in KRAS^{G12C} NSCLC xenografts

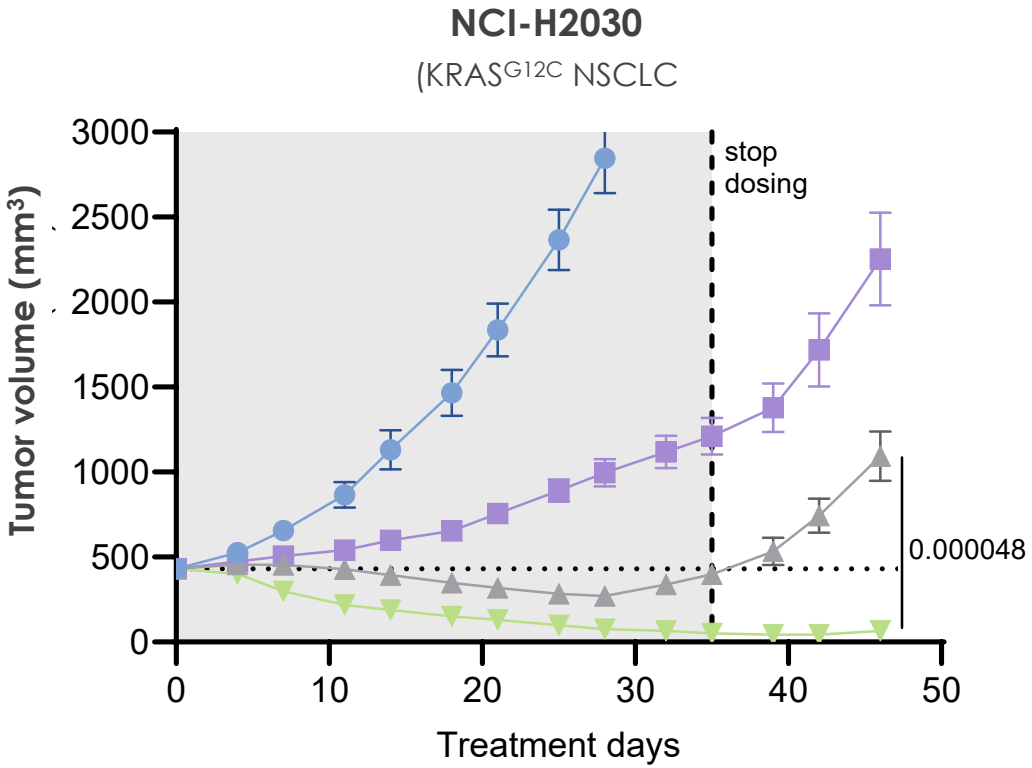


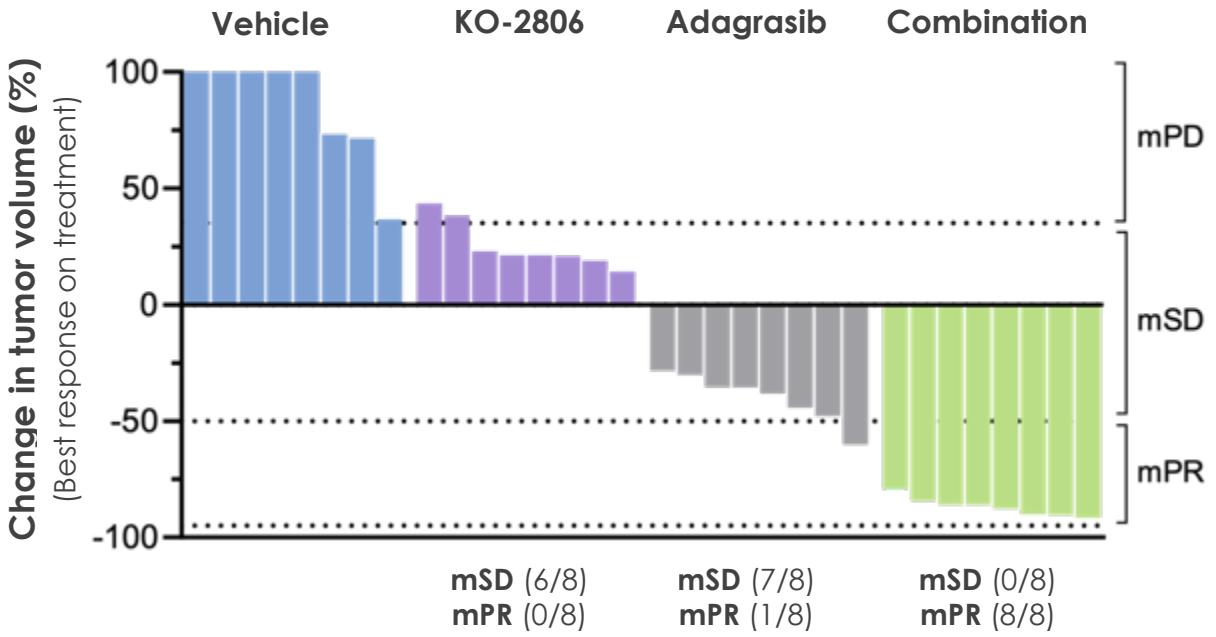


NCI-H2122 TUMORS (DAY 3)

Gatchalian J. et al, AACR-NCI-EORTC 2023 Abstract #34634

COMBINATION WITH KO-2806 ENHANCES DEPTH AND DURATION OF RESPONSE COMPARED TO ADAGRASIB ALONE

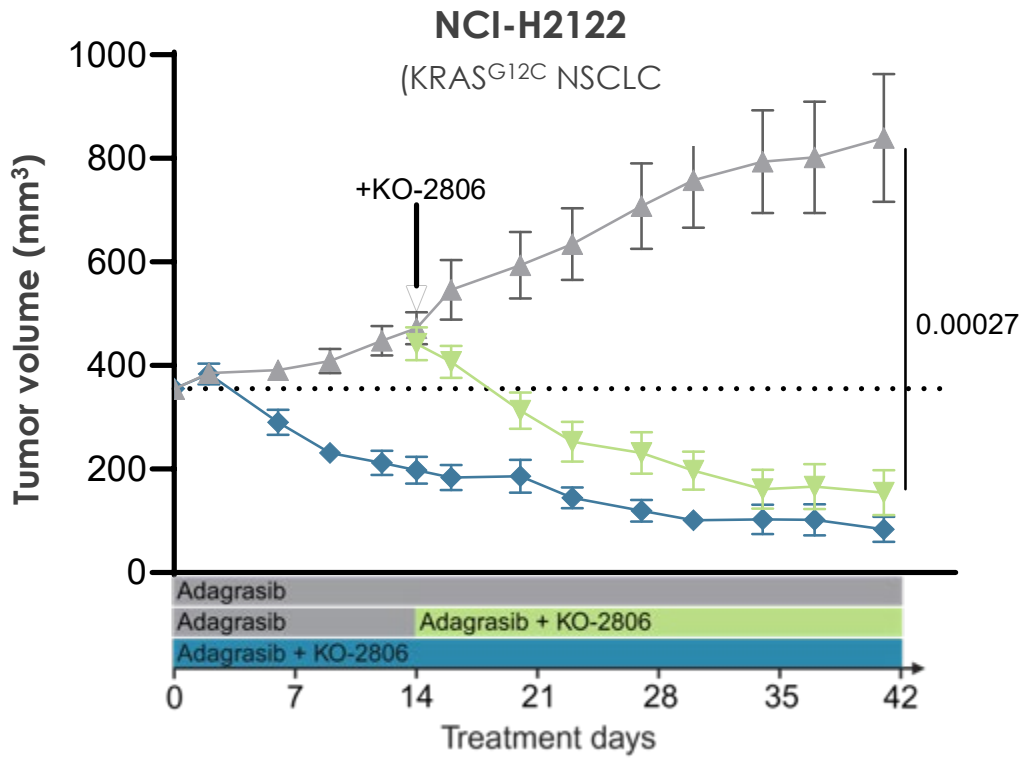


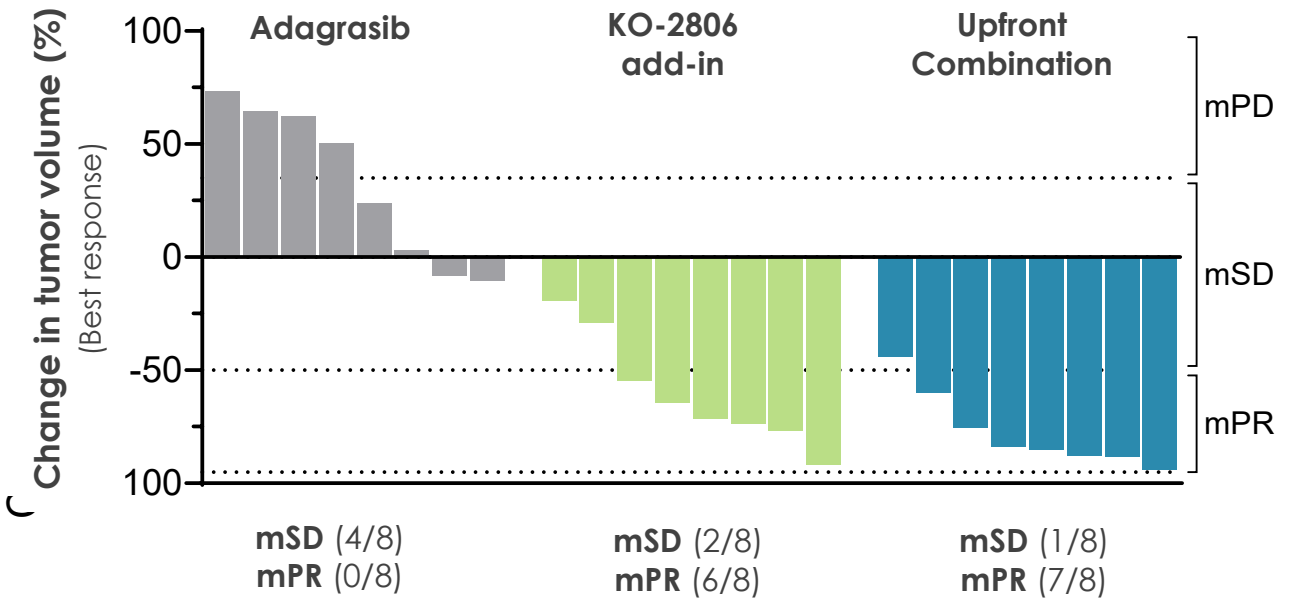


Patel H. *et al.* EORTC-NCI-AACR 2023 Abstract #34968

TUMORS PROGRESSING ON KRAS^{G12C} INHIBITOR, ADAGRASIB, ARE RE-SENSITIZED BY THE ADDITION OF KO-2806

Tumor regressions in the KO-2806 add-in group were comparable to the upfront combination of KO-2806 with adagrasib





Patel H *et al*. EORTC-NCI-AACR 2024 Abstract #388

FIT-001 PHASE 1 FIRST-IN-HUMAN CLINICAL TRIAL OF KO-2806 IN PATIENTS WITH ADVANCED SOLID TUMORS

FIT-001
KURA KO-2806-001

PART 1A (MONOTHERAPY) DOSE ESCALATION → **PART 1A (COMBINATIONS) DOSE ESCALATION** → **PART 1B (COMBINATIONS) DOSE EXPANSION**

OBJECTIVES

PRIMARY

Evaluate safety and tolerability of KO-2806 (dose escalation)

Determine the MTD/HPDD and/or the OBAD of KO-2806 (dose escalation)

Define RP2D of KO-2806 (dose expansion)

Evaluate the antitumor activity of KO-2806 in combination with cabozantinib in RCC and adagrasib in $KRAS^{G12C}$-mutant NSCLC (dose expansion)

SECONDARY

Evaluate the safety and tolerability of KO-2806 (dose expansion)

Evaluate the preliminary antitumor activity of KO-2806 (dose escalation and dose expansion)

Characterize the PK of KO-2806 when administered as monotherapy, and the PK of KO-2806 and the combination agents when administered in combination therapy (dose escalation and expansion)

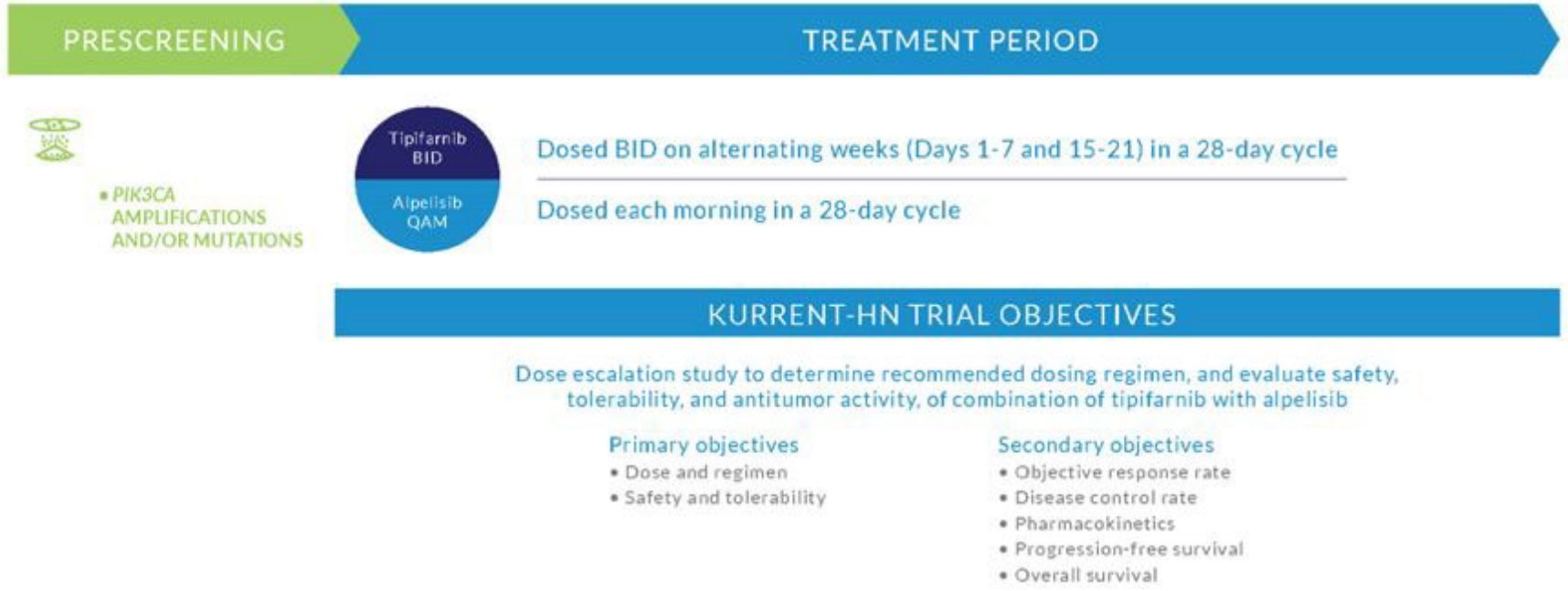
Preliminary clinical data for KO-2806 as a monotherapy and combo with cabozantinib in RCC expected in 2H 2025

MTD/HPDD = maximum tolerated dose/highest protocol-defined dose; OBAD = optimal biologically active dose

PHASE 1/2 COMBINATION TRIAL OF TIPIFARNIB AND ALPELISIB IN PATIENTS WITH HNSCC

Clinical collaboration to evaluate the first-generation FTI, tipifarnib, in combination with alpelisib for the treatment of patients with HNSCC whose tumors have *PIK3CA* mutations

Under the collaboration, Kura sponsors the trial and supplies tipifarnib and Novartis supplies alpelisib



Clinical data for Phase 1 dose-escalation trial of tipifarnib and alpelisib in patients with recurrent/metastatic *PIK3CA*-dependent HNSCC expected in 2H 2025

DURABLE CLINICAL RESPONSE OBSERVED IN PATIENT WITH PI3Kα-DEPENDENT HNSCC

35yo, male, nonsmoker, HPV16 positive

SCC of tonsil Stage III cT4N2M0; PD-L1 CPS = 60

Prior Treatments
- CDDP/rad for 1 mo (Nov-Dec2019), BOR UNK
- Cemiplimab/ISA101b (Jun-Nov2021), BOR PD

PIK3CA R88Q mutation (44%) and HRAS OE (3+ staining in 100% of tumor cells) by IHC from May 2021 biopsy

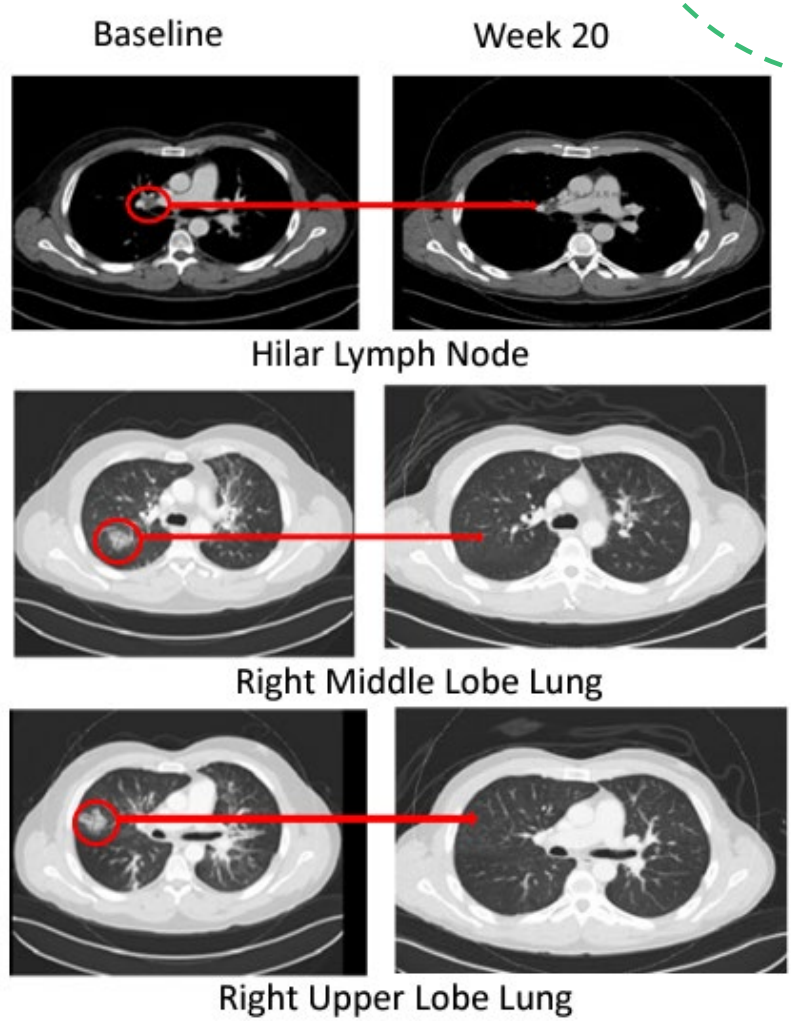
DL1 tipifarnib, DL2 alpelisib; completed 6 cycles

G1/2 TRAE, G3 lipase elevation; presented clinical benefit and improvement in respiratory symptoms

81% reduction in target lesions after 1 cycle of treatment

84% reduction in target lesions after 3 cycles (BOR)

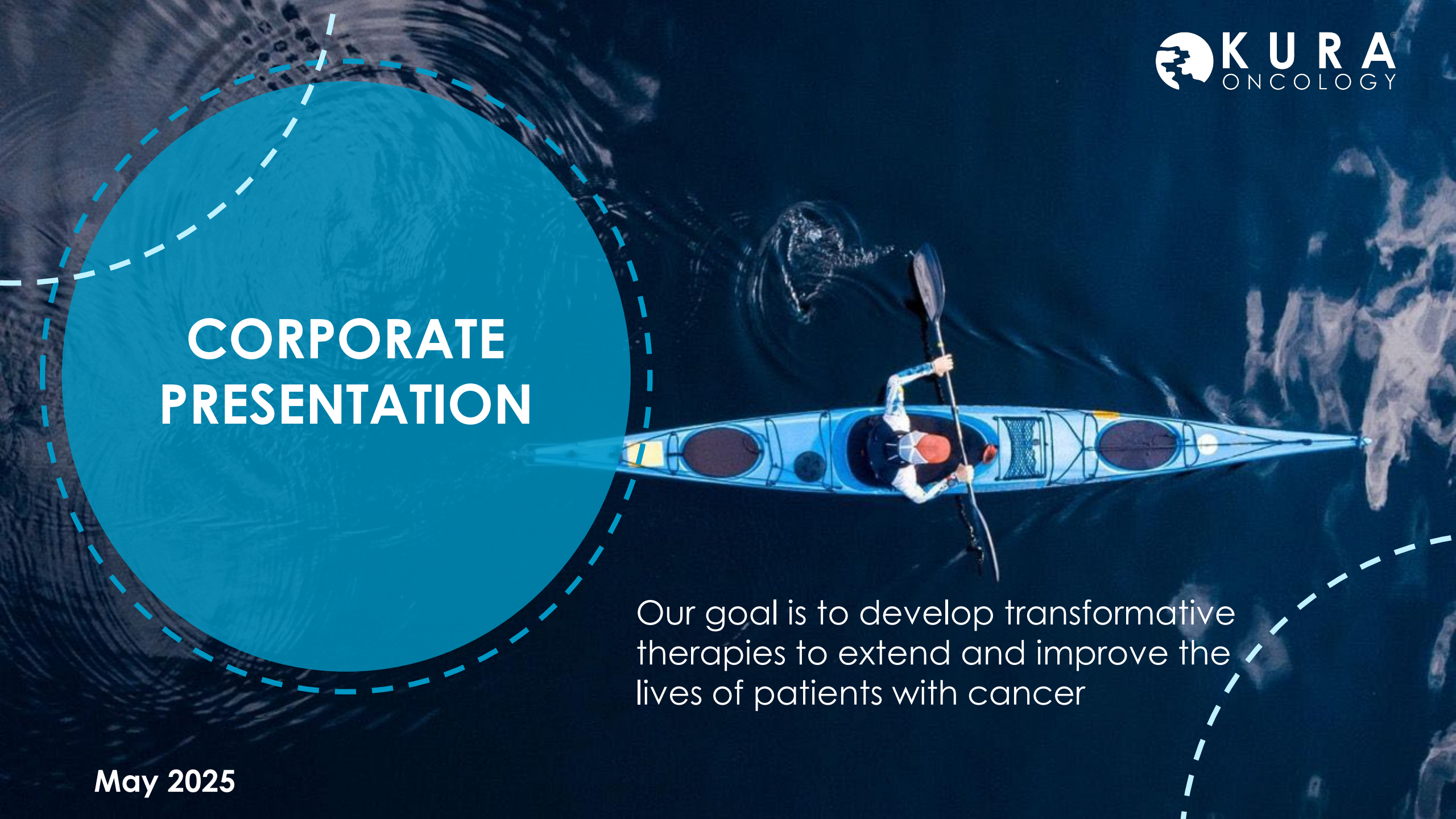
Continued on-study for > 27 weeks maintaining QoL



Soifer H et al, ENA 2022 PB041; Data cut as of 14Sep2022; Preliminary raw data

KURA ONCOLOGY

CORPORATE PRESENTATION



Our goal is to develop transformative therapies to extend and improve the lives of patients with cancer

May 2025