MEI Pharma

Corporate Overview

Novel drug candidates to address known resistance mechanisms to standard-of-care cancer therapies

May 21, 2024

Forward Looking Statements

Certain information contained in this communication that are not historical in nature are "forward-looking statements" within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995 including, without limitation, statements regarding: the potential, safety, efficacy, and regulatory and clinical progress of our product candidates, including the anticipated timing for initiation of clinical trials and release of clinical trial data and our expectations surrounding potential regulatory submissions, approvals and timing thereof, our business strategy and plans and the sufficiency of our cash, cash equivalents and short-term investments to fund our operations. You should be aware that our actual results could differ materially from those contained in the forward-looking statements, which are based on management's current expectations and are subject to a number of risks and uncertainties, including, but not limited to, risk relating to our ability to successfully commercialize our product candidates; the availability or appropriateness of utilizing the FDA's accelerated approval pathway for our product candidates; final data from our pre-clinical studies and completed clinical trials potentially differing materially from reported interim data from ongoing studies and trials; costs and delays in the development and/ or FDA approval, or the failure to obtain such approval, of our product candidates; uncertainties or differences in interpretation in clinical trial results; uncertainty regarding the impact of rising inflation and the increase in interest rates as a result; potential economic downturn; activist investors; our inability to maintain or enter into, and the risks resulting from, our dependence upon collaboration or contractual arrangements necessary for the development, manufacture, commercialization, marketing, sales and distribution of any products; competitive factors; our inability to protect our patents or proprietary rights and obtain necessary rights to third party patents and intellectual property to operate our business; our inability to operate our business without infringing the patents and proprietary rights of others; general economic conditions; the failure of any products to gain market acceptance; our inability to obtain any additional required financing; technological changes; government regulation; changes in industry practice; and one-time events. We do not intend to update any of these factors or to publicly announce the results of any revisions to these forward-looking statements. Under U.S. law, a new drug cannot be marketed until it has been investigated in clinical studies and approved by the FDA as being safe and effective for the intended use.

Clinical-stage Oncology Company Evaluating Novel Drug Candidates to Address Known Resistance Mechanisms to Standard-of-care Cancer Therapies

Science Driven Development of Combination Therapies

- Two Novel Drug Candidates
- Clinical Data Support Combinations
- Significant Addressable Markets
- Near-term Value Drivers

Cash sufficient to fund operations for at least the next 12 months

Two Novel Oncology Drug Candidates

VORUCICLIB: Oral CDK9 Inhibitor

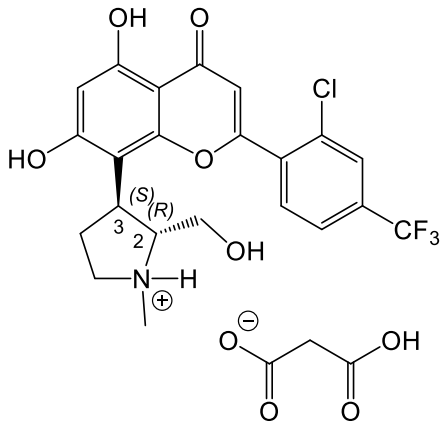
- **Plan to initiate Phase 2 arm in R/R AML 2025**
- Encouraging results in AML patients treated, including 42 in combination with venetoclax (Venclexta®)
- Mutation agnostic therapy: potential to address larger % of AML population than menin, FLT3 or IDH inhibitors

ME-344: Novel Inhibitor of OXPHOS

- **Clinical data support combination with bevacizumab (Avastin®) in metastatic colorectal cancer**
- Novel approach intended to address cancers where VEGF inhibition is SoC
- Readying new formulation for Phase 1

Investigational Agents	Therapeutic Area	Combination	Pre-IND	Phase 1/1b	Phase 1/2
Voruciclib Oral CDK9 Inhibitor	Acute Myeloid Leukemia Relapsed/refractory (2L+)	Single-agent VENCLEXTA® (venetoclax)	Completed	Ongoing	
	Solid Tumors*	Single-agent & Vemurafenib	Completed		
ME-344 OXPHOS Inhibition	HER2-negative Breast Cancer**	AVASTIN® (Bevacizumab)	Completed		
	Colorectal Cancer Relapsed	AVASTIN® (Bevacizumab)	Completed		
	Solid Tumors	VEGF Inhibitors (Bevacizumab & tyrosine kinase inhibitors)	New Formulation		

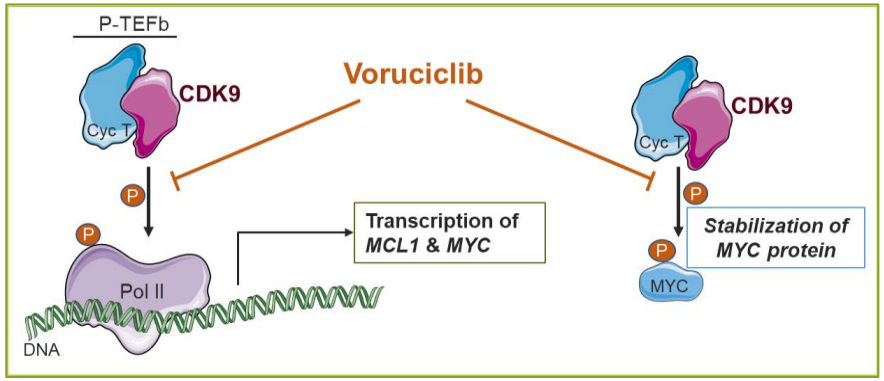
*Three Phase 1 Studies in a total of 77 patients. **Phase 0 window of opportunity study: investigator initiated; placebo controlled.



Voruciclib: A Selective Oral CDK9 Inhibitor Drug Candidate

Voruciclib: A Selective Oral CDK9 Inhibitor with Potential to Address Hematologic Malignancies and Solid Tumors

Voruciclib Inhibits CDK9 Leading to Decreased Mcl-1 and Myc Proteins



Myeloid leukemia 1 (MCL-1):
An antiapoptotic protein of the BCL-2 family that prevents apoptosis by binding to the pro-apoptotic BCL-2 proteins.

Myc proto-oncogene:
A transcription factor that promotes cell growth and proliferation and regulates cell metabolism.

Voruciclib is Differentiated

- **Oral administration**
  - Favorable PK: Half-life of 26-32 hours allows once a day dosing
- **Selective**
  - Higher specificity *and* longer residence time on target vs CDK 6, 4 &1
  - Greater selectivity against CDKs relative to other kinases
- **Potent**
  - IC50 from 0.2 to 1.7 µM in various cell lines

Voruciclib Displays Selectivity and Specificity to CDK9

CDK / Cyclin	K_i (nM)	Residence Time (s)
CDK9 / Cyc T2	0.63	105
CDK9 / Cyc T1	1.68	151
CDK6 / Cyc D1	2.92	3.5
CDK4 / Cyc D1	3.96	4.8
CDK1 / Cyc A2	9.10	55
CDK2 / Cyc A2	55.1	19

Dey, et al. Sci Reports (2017) 7(1):8007. Luedtke, et al. Signal Transduct Ther (2020) 5(1):17. Wiley, et al. Cancer Research (2021) 81(13 supp):1962

Both Mcl-1 and Myc Proteins Represent Important Therapeutic Targets for Hematologic Malignancies and Solid Tumors

MCL-1:

- Increase associated with poor prognosis in AML and several B-cell malignancies
- Upregulation is an established venetoclax resistance mechanism
- Venetoclax inhibits BCL2 but can lead to stabilization of Mcl-1

MYC:

- Over expressed in many cancers, including those with KRAS mutations
- CDK9 inhibition leads to reduced transcription and stability of MYC

Voruciclib inhibits MCL-1 *and* MYC via CDK9 inhibition

Voruciclib is a Novel *Oral* CDK9 Inhibitor in Development for R/R AML in Combination with Venetoclax

Voruciclib Program Overview

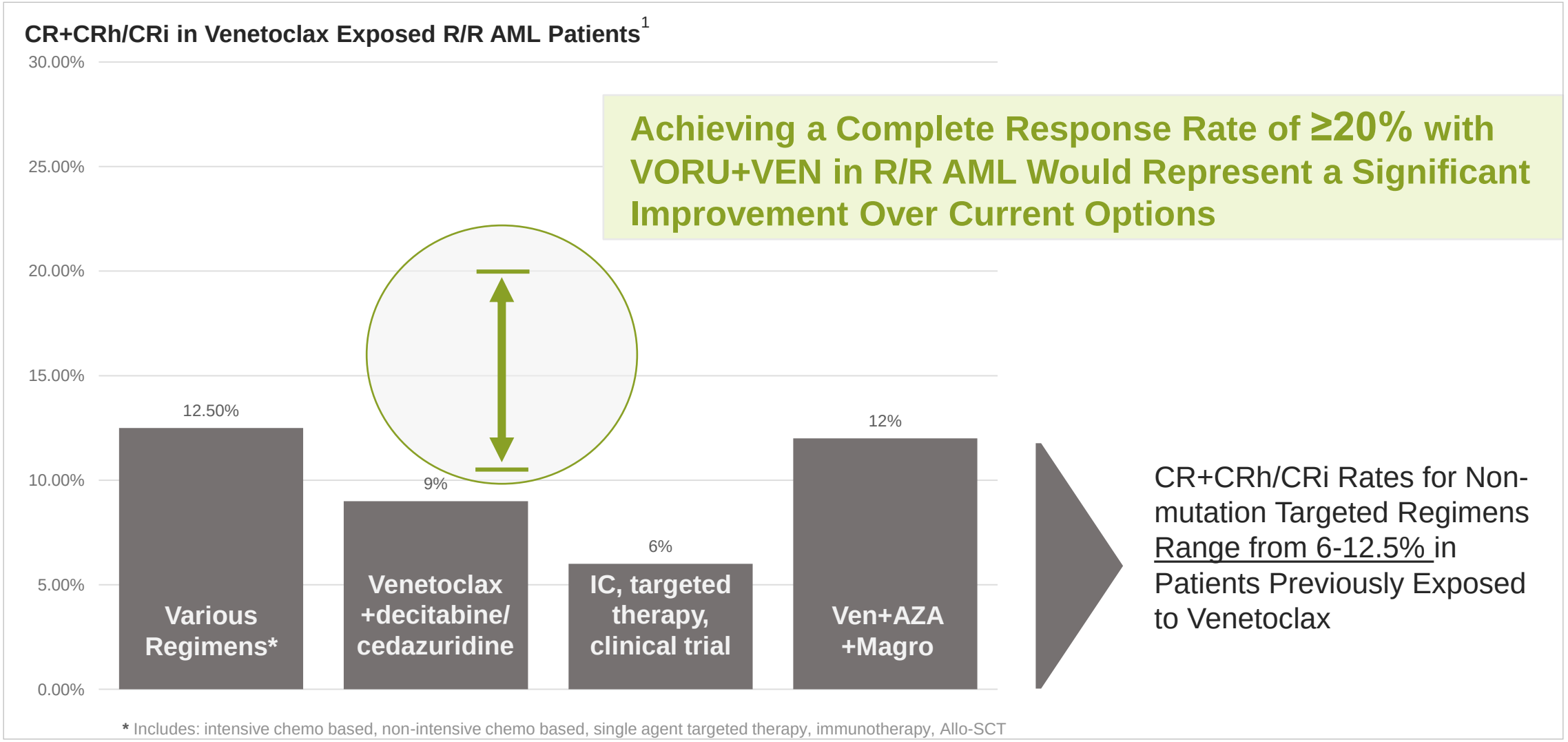
- Significant clinical experience: Over 150 patients treated to date, 82 with hematological malignancies and 77 with solid tumors
- Current focus on combination with venetoclax in R/R AML:
  - Addresses established venetoclax resistance mechanism
  - High Need: Poor outcomes with current salvage therapies after venetoclax failure
  - Phase 1 continues enrolling with additional combination readouts in H2 2024

Planned Dose Optimization in H2 2024 Followed by Ph 2 arm at RP2D in 2025, Creating Robust Ph 3 Read Package:

- Over 100 pts with r/r AML, including ~85 administered V+V, ~40 at RP2D

(Phase 2 study arm subject to FDA advice)

Poor Outcomes Post Venetoclax Failure in Patients with No Actionable Mutations



1. Maiti, A., et. al., Haematologica 2021: 106(3. Maiti, A., et. al., *Blood* 2023: 142 (Supplement 1): 4289. Zainaldin, C., et. al., *Leuk Lymphoma* 2022: 63(13):3245-3248. Daver, N., *Blood* (2022) 140 (Supplement 1): 141–144:

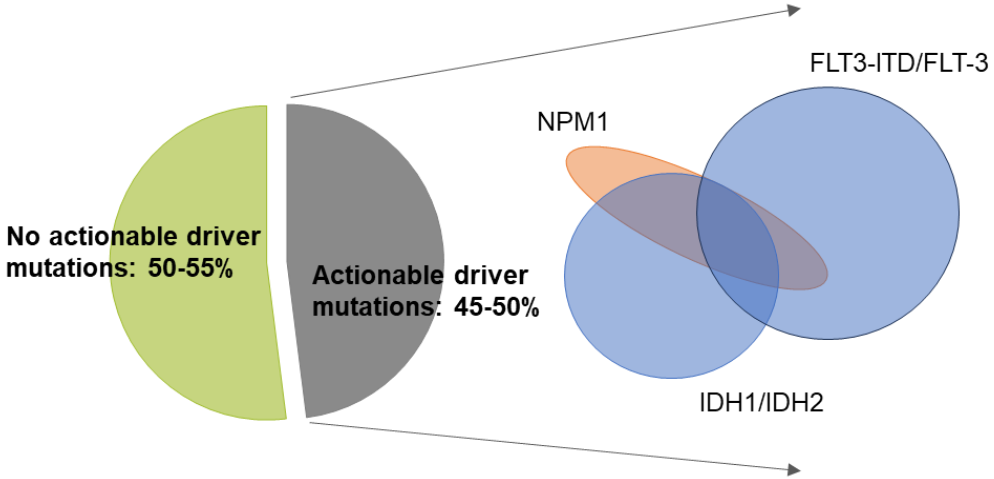
High Unmet Need Remains for ≥50% of AML Population With No Actionable Mutations

High Need For Mutation Agnostic Therapy:

50-55% of AML Patients Do Not Have Actionable Mutations[1,2]

In comparison, approximately 30%, 30% and 20% of AML patients have NPM1, FLT3 and IDH1/2 mutations, respectively[1].

There is significant overlap across mutation sub-populations[1]



In one study, approximately 80% of patients with an NPM1 mutation had a co-mutation in either FLT3, IDH1/2 or both[2]

Diagram is for illustrative purposes.

1. Miyamoto, K., et. al., Int J Mol Sci. 2020;21(14):5114. 2. Malani, D., Cancer Discov. 2022;12(2):388-401

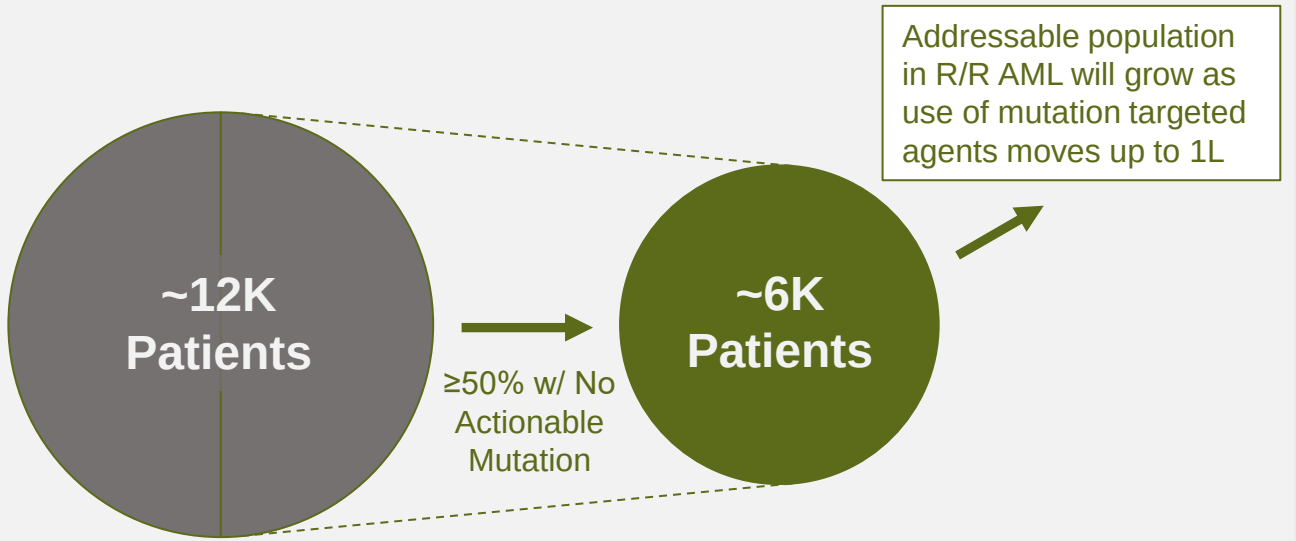
Large Potential Addressable Population with High Unmet Need for Mutation Agnostic Therapy

Use of Venetoclax Based Therapy Increasing Across AML Treatment in Various Combinations

- Venetoclax + HMA is entrenched as the dominant SOC in 1L unfit and expected to grow
- Ongoing studies expected to shift use of mutation targeted agents up to 1L in combination with Venetoclax + HMA backbone as triplet regimens
- Novel venetoclax combinations being studied in R/R AML

Venclexta sales were ~$2.3B in 2023 WW across all indications; projected to grow to ~$3.3B by 2028

~6K Addressable Patient Population for Mutation Agnostic Therapy in Patients with R/R AML



R/R AML Treatable Annually (U.S.)

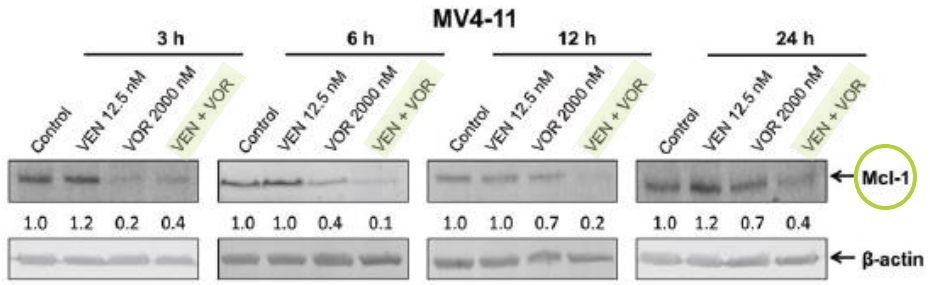
R/R AML Treatable w/o Actionable Mutations Annually (U.S.)

Sources: Evaluate, April 2024; Clarivate November 2023

Preclinical Studies Demonstrate Voruciclib Suppresses Mcl-1 and Synergizes with Venetoclax in AML Murine Xenograft Model

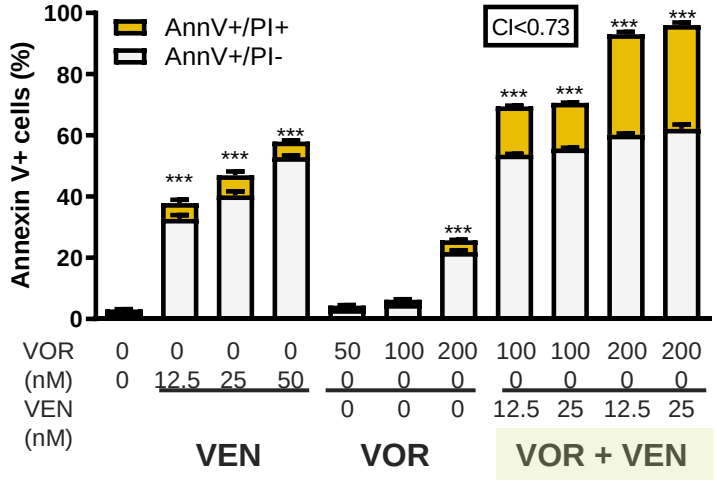
Suppresses Mcl-1 Level

a.



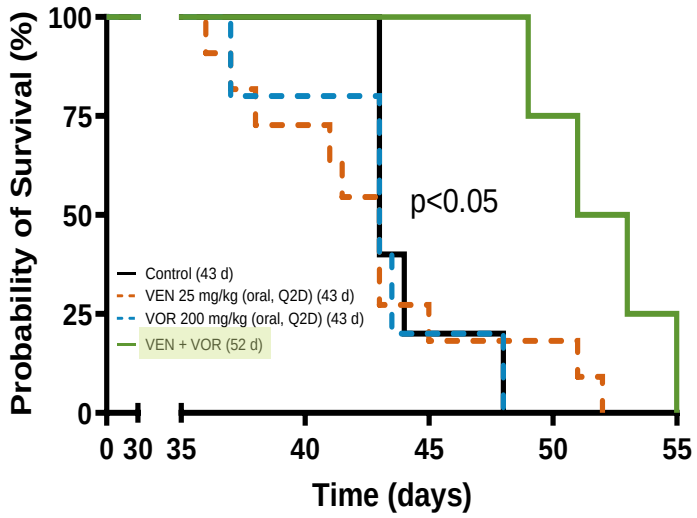
Increases Apoptosis

b.



Extends Survival in MV4-11 Tumor

c.



Voruciclib also synergizes with venetoclax in CLL patients[1,2] and in High Risk DLBCL Murine Models[3]

1. Lesnick et al. J Clin Oncol.2020;38(15_suppl):e20009; 2. Paiva et al, PLoS ONE. 2015;10(11):e0143685; 3. Dey et al. Nature Sci Rep. 2017; 7:18007

Ph 1/2 Study to Generate Data on ~106 Patients With R/R AML Administered Voruciclib Alone or with Venetoclax

Study Population

- Relapsed/Refractory AML
- Relapsed/Refractory B-cell malignancies

Dose escalation with standard 3+3 design

- Single agent
- Combination with venetoclax

Endpoints

- Safety and tolerability
- Pharmacokinetics
- Biologic correlative studies
  - BH3 profiling, MCL-1 expression
  - Molecular mutation analysis
- Preliminary activity

Ongoing Phase 1 Evaluation of Voruciclib + Venetoclax in R/R AML:
(N = ~66 pts in dose escalation and expansion cohorts)

	$IS_{2w,2w}$	$IS_{3w,1w}$	RP2D & Schedule
Dose Escalation	N=29 50 mg to 300 mg	N=12 150 mg to 300 mg	
Dose Expansion	N=12 300 mg	N=12 250 mg*	
Phase 2 Arm			N=20

* Final dose determination to be based on data from escalation cohort.

- $IS_{2w,2w}$: Intermittent dosing of voruciclib daily for 2 weeks in a 4-week cycle.
- $IS_{3w,1w}$: Intermittent dosing of voruciclib daily for 3 weeks in a 4-week cycle.
- RP2D: Recommended Phase 2 Dose

Plan to Initiate a Phase 2 Study Arm in 2025 After Determining Recommended Phase 2 Dose and Schedule
(Phase 2 study arm subject to FDA advice)

Single-Agent Voruciclib Well-Tolerated, Showed Clinical and Biomarker Activity in AML & CLL

Voruciclib up to 200mg for 14 days in a 28-day Cycle Demonstrated Anti-Leukemic Activity . . .

Single-agent activity (AML, n=21) at low dose studied in heavily pretreated patients:

- 1 Patient: MLFS (81 yo, 4 prior lines, adverse mutations & cytogenetics)
- 5 of 10 pts with AML at 200mg had stable disease
- 2 patients had differentiation syndrome demonstrative of biological activity

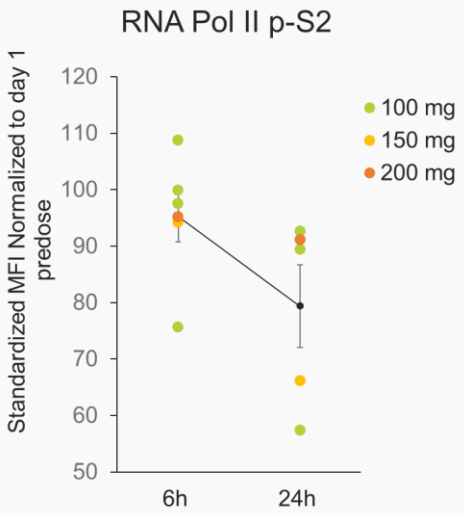
. . . and Was Well-tolerated on Intermittent Dosing

- No dose-limiting toxicity*
- No drug-related neutropenia
- No Grade 3+ drug related toxicity
- No discontinuation due to drug related toxicity

- Patient characteristics: 21 AML, 19 B-cell malignancies; Median prior therapies: 3 (range, 1-8)

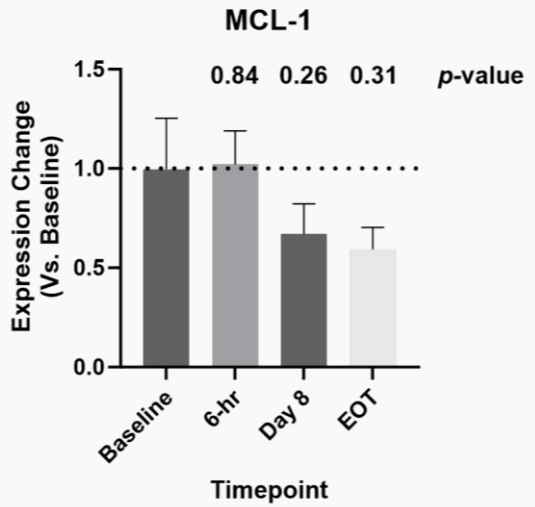
Dose escalation in Cohort 2 was stopped at 200 mg before reaching the MTD on this schedule to focus on evaluation of combination with venetoclax.

Single-Agent Correlative Studies Demonstrates on-Target Biologic Activity



AML

Analysis of patient AML blasts showed a decrease in RNA Pol II phosphorylation after voruciclib treatment. CDK9 directly phosphorylates RNA Pol II.



CLL

Longitudinal transcriptomic analysis of blood samples from CLL patients shows a trend towards reduced Mcl-1 mRNA.

Evidence of Activity and Tolerability in Dose Escalation Groups of Voruciclib Plus Venetoclax in R/R AML

- 29 patients in dose escalation cohorts ($IS_{2w,2w}$)
- Clinical activity observed:
  - Responses
  - Reduced transfusions
  - Improved blast counts
  - Mcl-1 decreases, including:
    - A greater decrease at a higher dose
    - Response related decreases
- No DLTs observed
- No significant increase in myelosuppression
- PK analysis does not show drug-drug interaction

All V+V patients are heavily pretreated with a median of 2 prior therapies (range 1-7) including venetoclax in 28/29 patients

Combination Arm Characteristics: Patients Typically Heavily Pretreated With a Median of 3 Prior Therapies and High Rate of Adverse Molecular and Cytogenetic Features

- **Median age 67 years (range 34-89)**
- **15 (52%) had ≥3 prior lines**
- **28/29 (97%) treated with venetoclax in earlier line of therapy**
- **21 (72%) with adverse 2017 ELN Risk Category**

	N = 29
Number of prior therapies	
Median (range)	3 (1-7)
≥3 prior	15 (52%)
Prior HSCT	9 (31%)
Prior venetoclax	28 (97%)
1st line	14 (50%)
≥2nd line	14 (50%)
Prior anthracyclines	18 (62%)

	Total (N=29)
2017 ELN Risk Category	
Favorable	3 (11%)
Intermediate	5 (17%)
Adverse	21 (72%)
Poor Cytogenetics	
Patients with adverse cytogenetics	14 (48%)
Adverse Molecular Mutations	
Patients with adverse mutations	19 (66%)
TP53	7 (24%)
ASLX1	7 (24%)
RUNX1	4 (14%)
GATA2	2 (7%)
Baseline Bone Marrow Blast	
Median (range)	45% (2-77%)

Voruciclib Dosed up to 300 mg Appeared Generally Well-Tolerated with No Apparent Dose Response to Adverse Events Reported

Ph 1 Study Treatment Emergent Adverse Events in ≥10% of Patients

	(50 mg QOD) (N=6)	(50 mg QD) (N=3)	(100 mg QD) (N=4)	(150 mg QD) (N=4)	(200 mg QD) (N=4)	(250 mg QD) (N=4)	(300 mg QD) (N=4)	Total (N=29)
Nausea	0	0	2 (50.0)	3 (75.0)	2 (50.0)	1 (25.0)	2 (50.0)	10 (34.5)
Platelet Count Decreased	0	1 (33.3)	1 (25.0)	3 (75.0)	1 (25.0)	1 (25.0)	1 (25.0)	8 (27.6)
Febrile Neutropenia	0	1 (33.3)	2 (50.0)	2 (50.0)	0	1 (25.0)	1 (25.0)	7 (24.1)
Anaemia	0	0	2 (50.0)	2 (50.0)	0	1 (25.0)	1 (25.0)	6 (20.7)
Hypokalaemia	0	0	2 (50.0)	1 (25.0)	2 (50.0)	1 (25.0)	0	6 (20.7)
Cough	2 (33.3)	0	1 (25.0)	1 (25.0)	1 (25.0)	0	0	5 (17.2)
Diarrhoea	1 (16.7)	0	1 (25.0)	1 (25.0)	1 (25.0)	1 (25.0)	0	5 (17.2)
Dyspnoea	2 (33.3)	0	1 (25.0)	1 (25.0)	0	1 (25.0)	0	5 (17.2)
Fatigue	0	0	0	3 (75.0)	1 (25.0)	0	1 (25.0)	5 (17.2)
Stomatitis	2 (33.3)	0	1 (25.0)	1 (25.0)	0	0	0	4 (13.8)
Vomiting	0	0	1 (25.0)	0	0	2 (50.0)	1 (25.0)	4 (13.8)
Anxiety	1 (16.7)	0	2 (50.0)	0	0	0	0	3 (10.3)
Corona Virus Infection	1 (16.7)	0	0	1 (25.0)	1 (25.0)	0	0	3 (10.3)
Hypotension	1 (16.7)	0	1 (25.0)	1 (25.0)	0	0	0	3 (10.3)

Responses and Blast Count Reductions Demonstrate Evidence of Anti-Leukemic Activity in Heavily Pretreated Patients After Venetoclax Failure

85% (17/20) of patients administered voruciclib at ≥100 mg achieved response or stable disease:

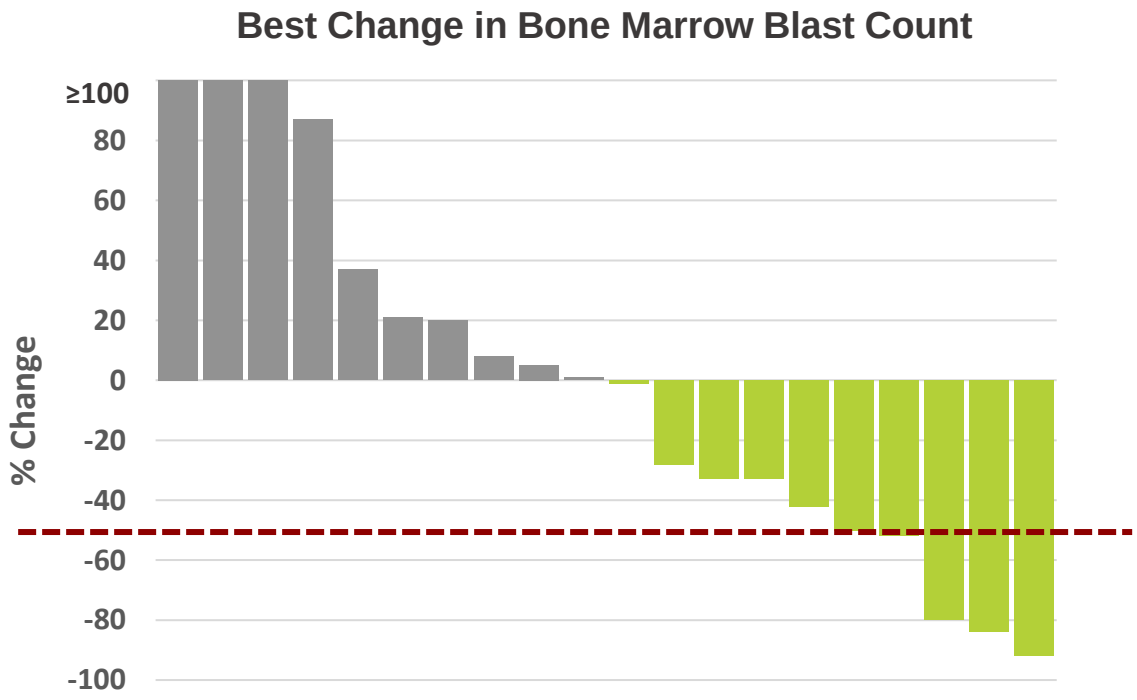
- 3 patients achieved a response
  - 2 patients achieved a complete response with incomplete hematologic recovery (CRi)
  - 1 patient achieved a morphologic leukemia-free state (MLFS)

  Each responding patient received venetoclax in an earlier line of treatment.

  Responses lasted 7 months in one patient, 5 months and ongoing in the second patient; the third patient was referred to stem cell transplant.

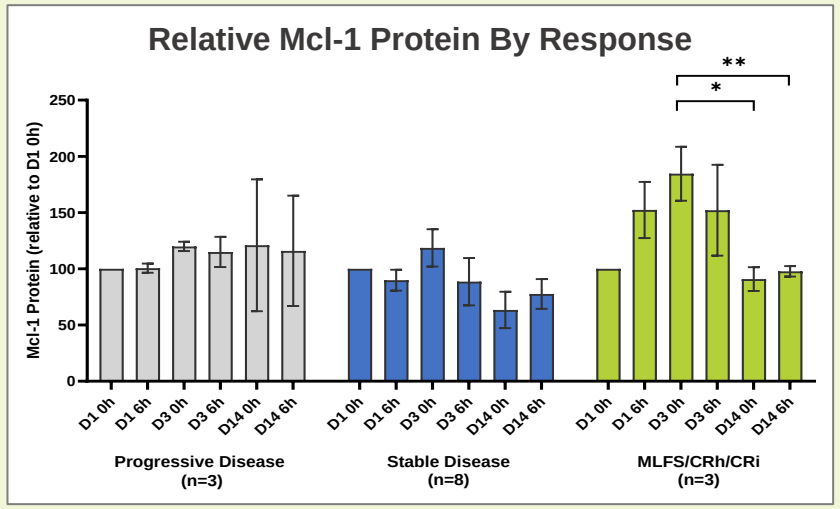
- 14 patients had stable disease, which lasted more than 90 days in 5 patients.

50% of Evaluable Patients with Pre/Post Bone Marrow Had a Decrease in Blast

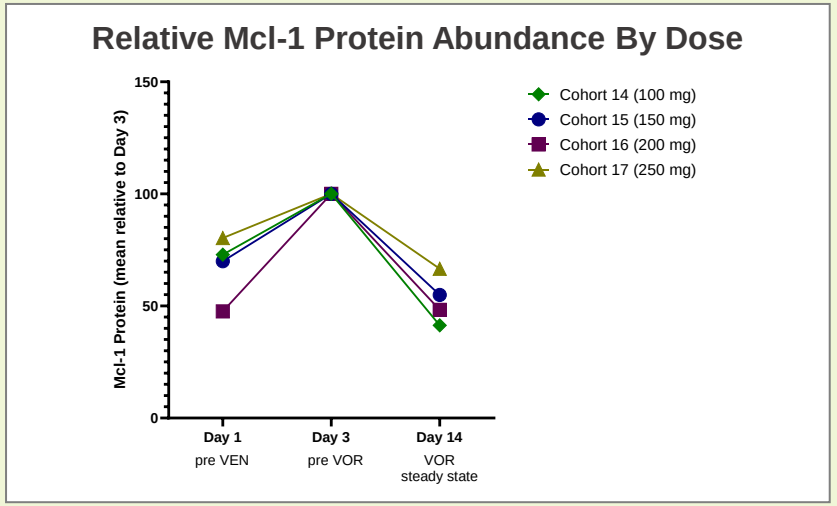


Decreases in Mcl-1 Demonstrate On-target Biological Activity

Greater Decrease in Mcl-1 Protein in Responders

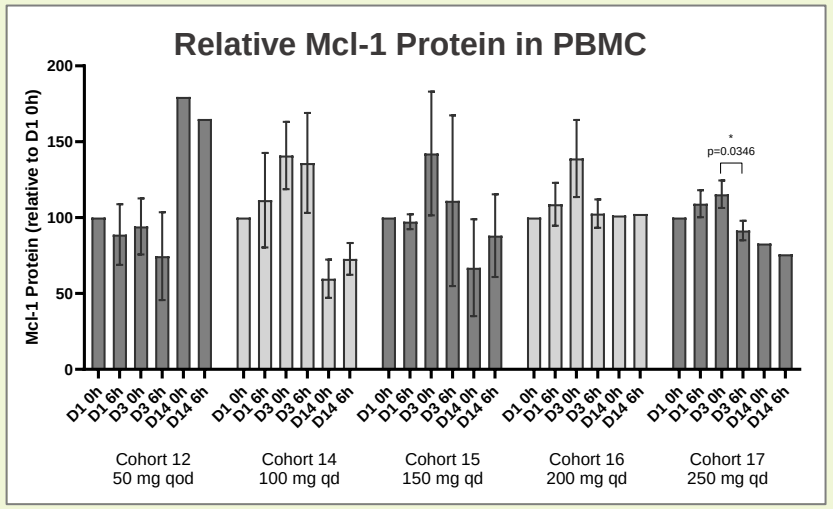


Mcl-1 Increases After Venetoclax Then Decreases with Voruciclib



Mcl-1 increases after venetoclax dosing (D1 0h – D3 0h) and decreases after 1st voruciclib dosing (D3 6h), continuing after 12 daily doses (D14 0h)

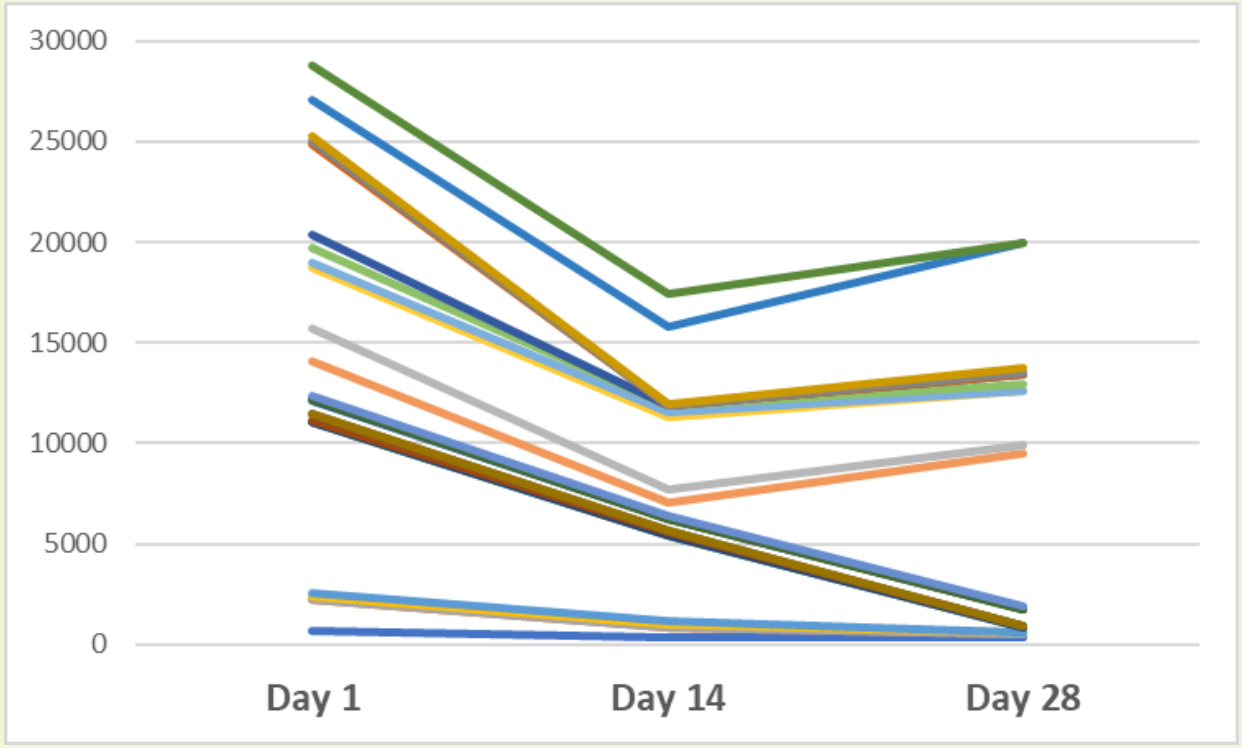
More Consistent Decrease in Mcl-1 at Higher Doses



- Preliminary analyses based on available patient samples
- Bars represents mean of Mcl-1 protein levels at various timepoints
- Mcl-1 protein expressed as mean fluorescence normalized to D1 0h or D3 0h

Peripheral Blast Counts Decrease on Voruciclib + Venetoclax Combination (Days 1 to 14) and Rebound with Venetoclax Alone (Days 15-28)

- 24 patients had serial blood tests for blasts with values >0
- 18/24 pts (75%) had decreased balsts on Day 14, at the end of voruciclib dosing
- 8/18 pts (44%) had increased blasts between Day 14 and 28, when off voruciclib but receiving venetoclax



Ongoing Phase 1 Study Evaluating Voruciclib administered on days 1 to 21 of the 28-Day Cycle to Extend Voruciclib Exposure

Near-term Data to Inform RP2D for Phase 2 Study Arm in R/R AML

Potential H2 2024 Updates:

- **$IS_{2w,2w}$ Dose Expansion** (N=12)
- **$IS_{3w,1w}$ Dose Escalation/Expansion** (N=12/12)

Phase 2 Study Arm* – Initiation and Data in 2025

RP2D and Schedule: N=~20

*Phase 2 study arm subject to FDA advice

Anticipated Total of ~40 Patients at RP2D & Schedule to Support Phase 3 Ready Program

Voruciclib Presents Novel Mechanism to Evaluate Treating a Range of Solid Tumors via MYC Inhibition

Voruciclib is a Selective Oral CDK9 Inhibitor Targeting Cell Proliferation Regulation

Opportunities to Address both Hematologic Malignancies and Solid Tumors

MCL-1:

- Increase associated with poor prognosis in AML and several B-cell malignancies
- Upregulation is an established venetoclax resistance mechanism
- Venetoclax inhibits BCL2 but can lead to stabilization of Mcl-1

MYC:

- **Over expressed in many cancers, including those with KRAS mutations**
- **CDK9 inhibition leads to reduced transcription and stability of Myc**

Voruciclib inhibits MCL-1 *and* MYC via CDK9 inhibition

Single-Agent Phase 1 Studies in Solid Tumors Demonstrated Reduction in MYC and was Generally Well-tolerated at Expected Therapeutic Doses

2 weeks on, 1 week off schedule (N = 29 pts)

- 75 to 850 mg
- MTD = 600 mg
- 41% disease control rate

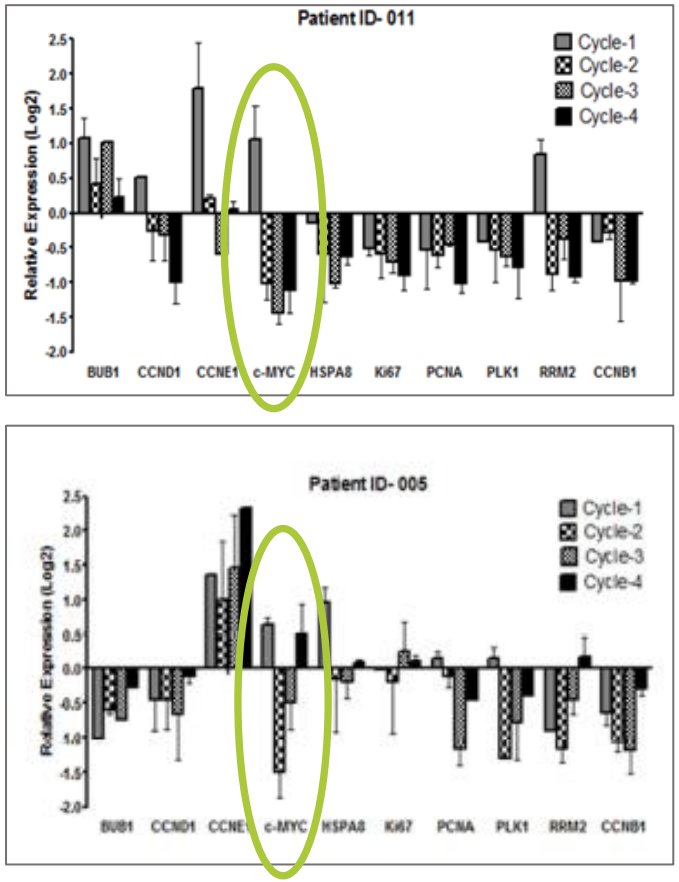
Daily continuously schedule (N = 39 pts)

- 75 to 500 mg
- MTD = 350 mg
- 31% disease control rate

Safety data

- No evidence of myelosuppression
- Most common AEs involved GI tract

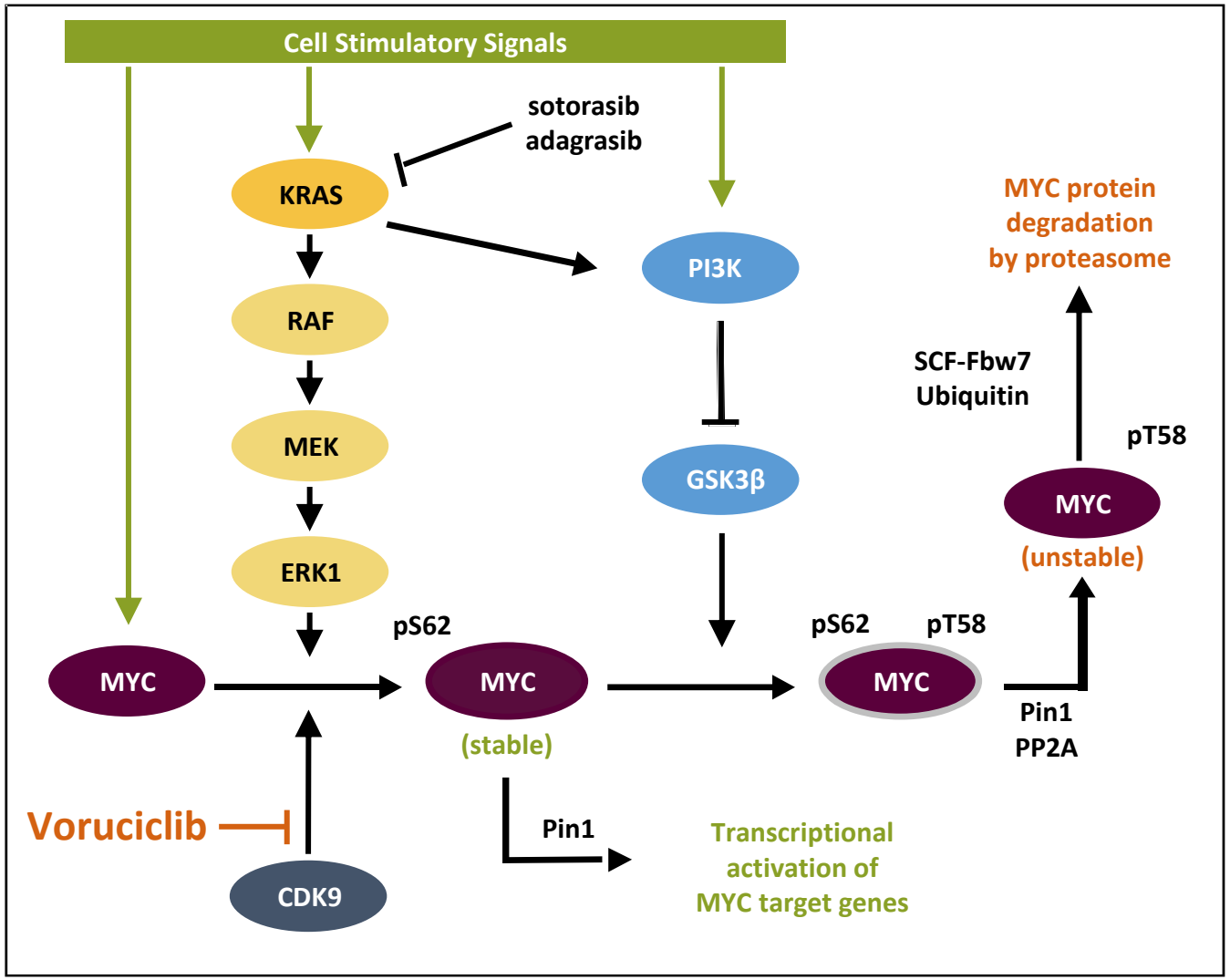
- 10 gene biomarkers evaluated in blood in daily dosing study
- c-MYC expression decreased in ~60% patients tested (n=25)



Gupta et al, ASCO 2012; Hao et al, ASCO 2012; MEI Data on file

CDK9 can influence MYC protein stability in KRAS mutant cancer cells

- Mutations in *KRAS* at G12, G13, and Q61 are oncogenic drivers in many cancers, including lung, colorectal, pancreatic, bone marrow, and endometrial carcinomas.
- *KRAS* mutations are frequently accompanied by stabilization of the MYC oncoprotein through increased *MYC* transcription and decreased protein degradation.
- MYC protein stability is mediated by phosphorylation of MYC on Ser 62 by ERK and CDK9 kinases.



Wiley, SE. et al. AACR, 2021.

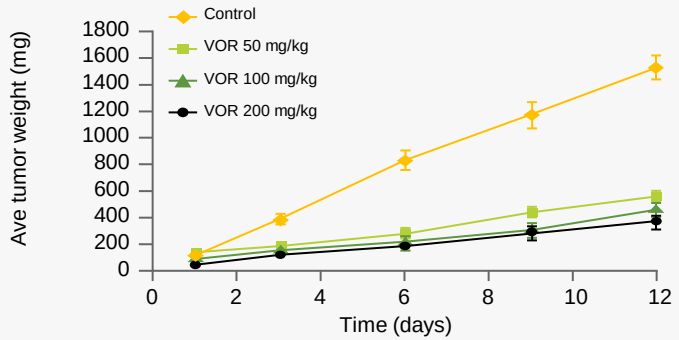
Voruciclib Inhibits *KRAS* Mutant Cell Lines *In Vitro* and *In Vivo* in Xenograft Mice

VOR inhibited proliferation of KRAS mutant cell lines from multiple indications

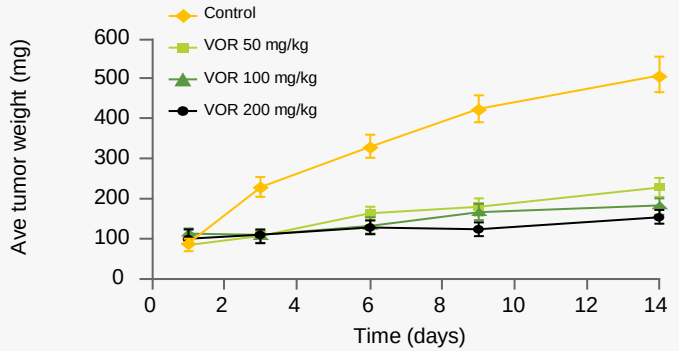
- CRC
- Esophageal
- Multiple Myeloma
- NSCLC
- Ovarian
- PDAC

VOR inhibited proliferation of cell lines with various KRAS mutations

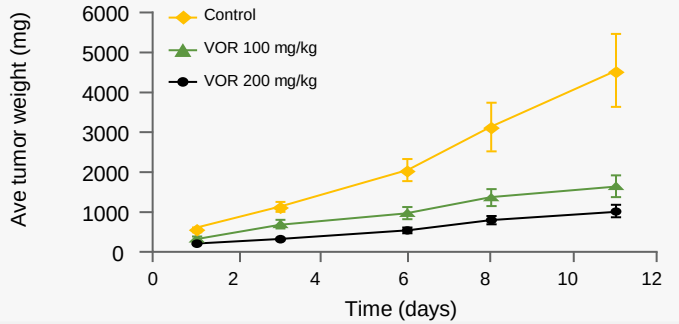
- G12C, G12D, G12A, G12V
- G13C, G13D
- Q61H



HCT-116
(CRC, KRAS G13D)



SW-480
(CRC, KRAS G12V)

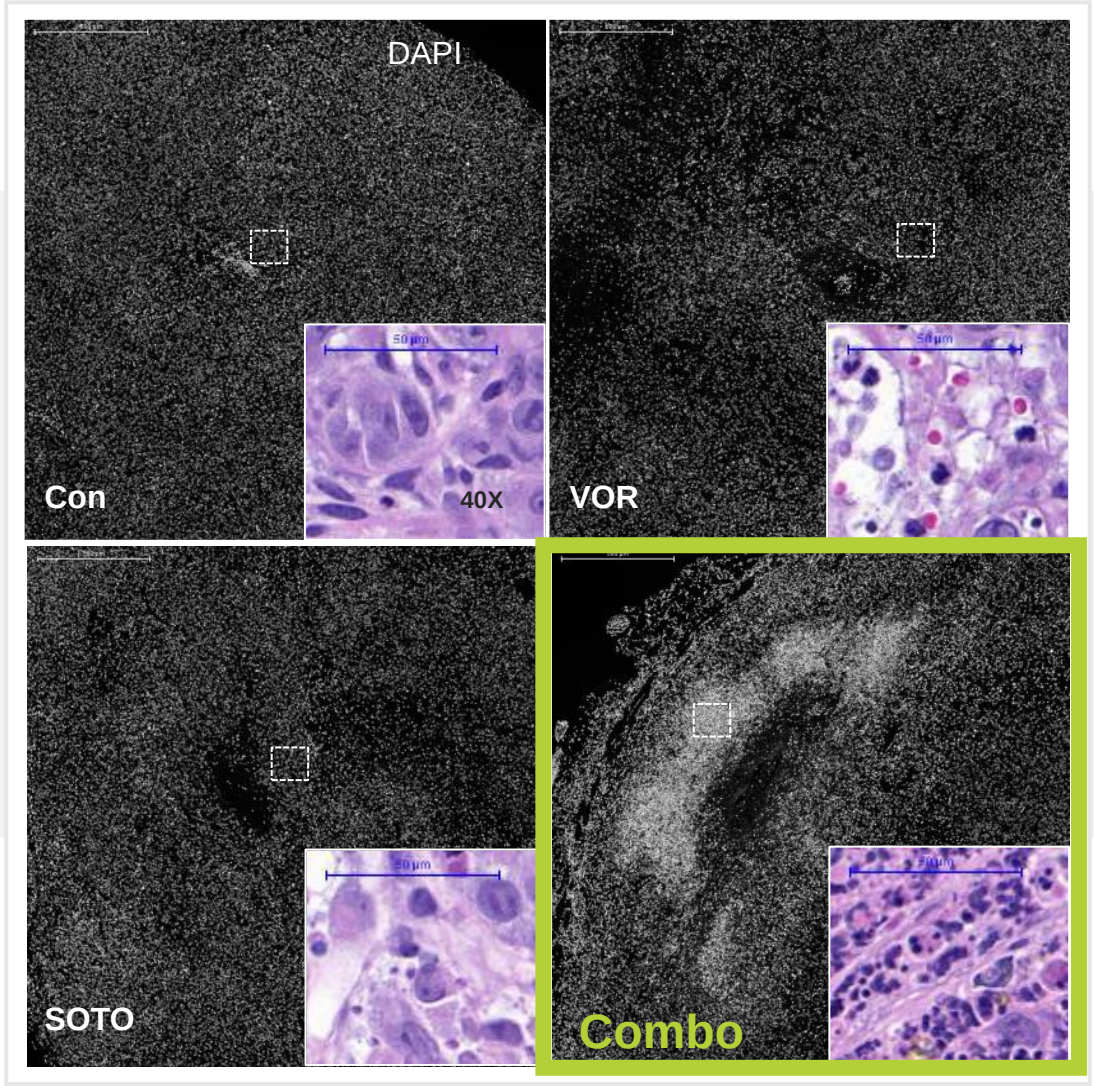


H460
(NSCLC, KRAS Q61H)

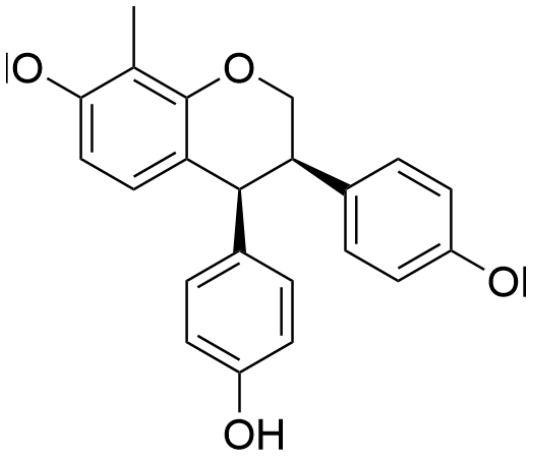
Wiley, SE. et al. AACR, 2021 & unpublished.

Combining Voruciclib with Sotorasib Results in Enhanced Cell Death in an *in vivo* MIA PaCa-2 Tumor Model

Cell death around each microinjection site measured by nuclear condensation and fragmentation



Wiley, SE. et al. AACR, 2021.



ME-344: An OXPHOS Inhibitor Drug Candidate

ME-344: A Novel Inhibitor of OXPHOS with Dual Effect on OXPHOS and Purine Synthesis

Potent Inhibitor of Proliferation

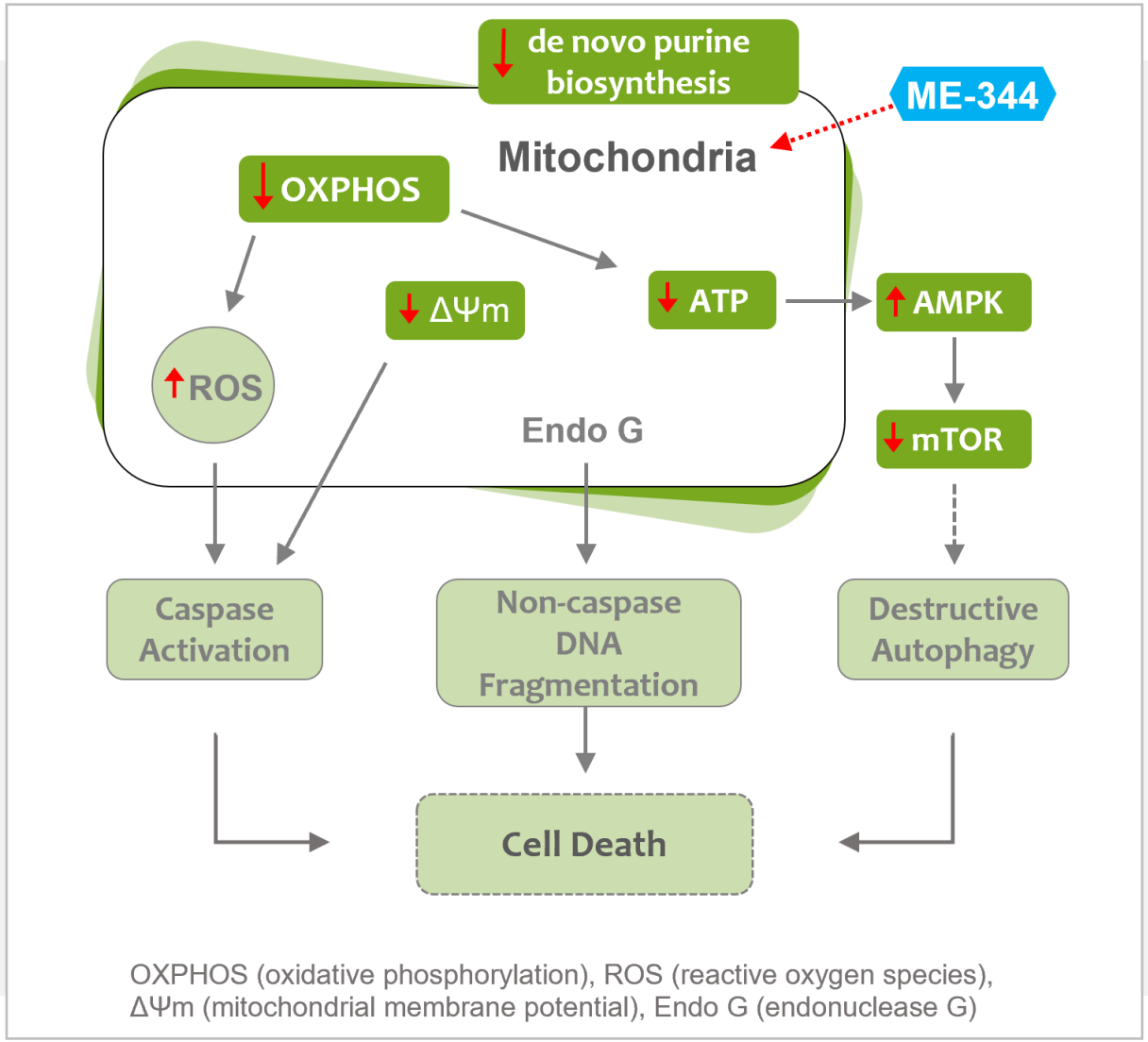
- nM potency against majority of 240 cell lines from solid tumors and hematologic malignancies[1]

Inhibition of OXPHOS leads to

- Reduced mitochondrial membrane potential, increased ROS, and decreased ATP[2,3]
- Induced cell death through multiple signaling pathways
  - Caspase and non-caspase mediated DNA fragmentation
  - Increased AMPK signaling leading to decreased mTOR signaling and destructive autophagy

Reduction in *de novo* Purine Biosynthesis

- Decreased *de novo* purine biosynthesis, an important pathway in cancer cells[4]



OXPHOS (oxidative phosphorylation), ROS (reactive oxygen species), ΔΨm (mitochondrial membrane potential), Endo G (endonuclease G)

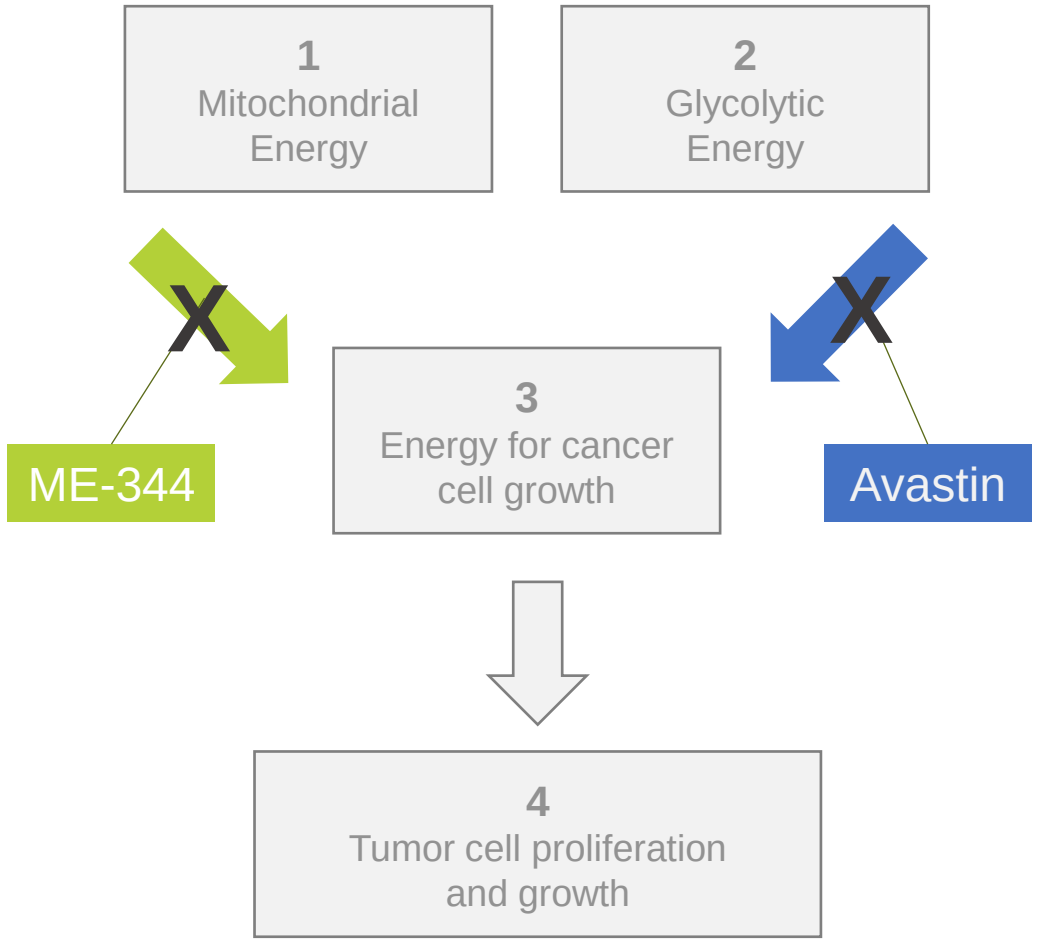
[1] MEI data on file, [2]Lim et al, Am J Cancer Res. 2015, [3]Manevich et al, J Pharm and Exper Ther. 2016, [4]Hurrish et al, Biochem Pharmacol 2024

ME-344 Program Overview: Inducing Synthetic Lethality in Solid Tumors in Combination with Anti-angiogenics

- Novel MOA with OXPHOS inhibition leading to ATP depletion in malignant cells
- Significant need in large addressable patient populations: anti-angiogenics like bevacizumab and TKIs used in multiple settings as SOC, including:
  - Colorectal, ovarian, renal cell, glioblastoma and pancreatic cancers
- Clinical data set supporting activity in combination with bevacizumab in a well-tolerated manner, including:
  - Phase 1b study in patients with mCRC *and* controlled Phase 0 study in HER2 negative breast cancer
- Current program focuses developing new formulation with potential to increase biological activity, patient convenience and commercial opportunity

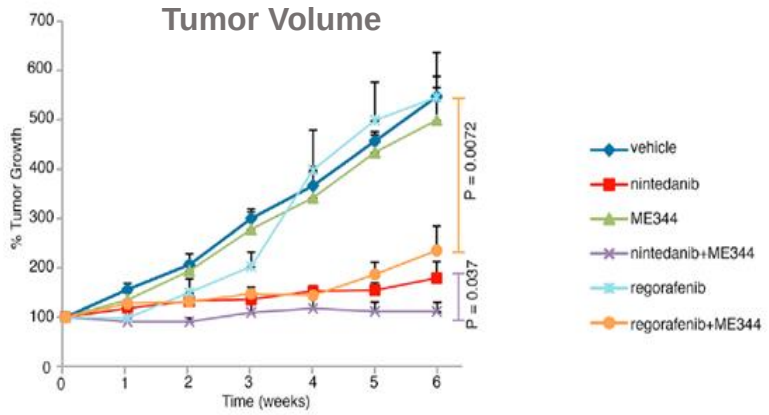
ME-344: A Novel Combination Approach with Anti-angiogenic Therapies Like Avastin® to Potentially Address Multiple Cancers

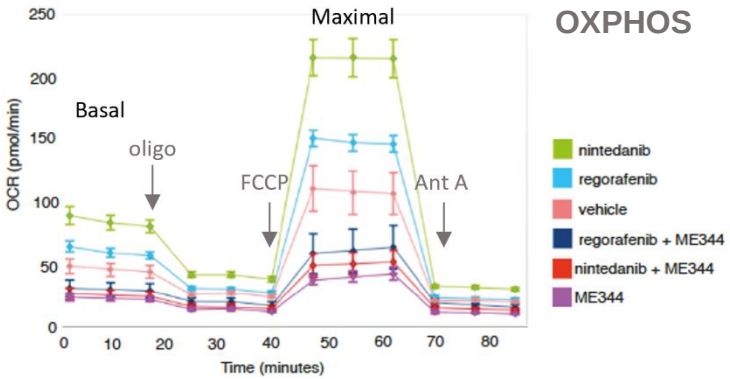
1. ME-344 blocks the production of ATP by inhibiting the OXPHOS pathway
2. Anti-angiogenic therapies reduce glycolysis
3. Cancer cells switch between mitochondria and glycolytic metabolic pathways to escape the blocking of either energy source
4. The potential to inhibit both mitochondrial energy production via ME-344 and glycolytic energy production via VEGF inhibition is intended to result in metabolic synthetic lethality of cancer cells



ME-344 Synergizes with Anti-angiogenics in Multiple Murine Solid Tumor Models Resulting in Tumor Growth Delay and Improved Survival

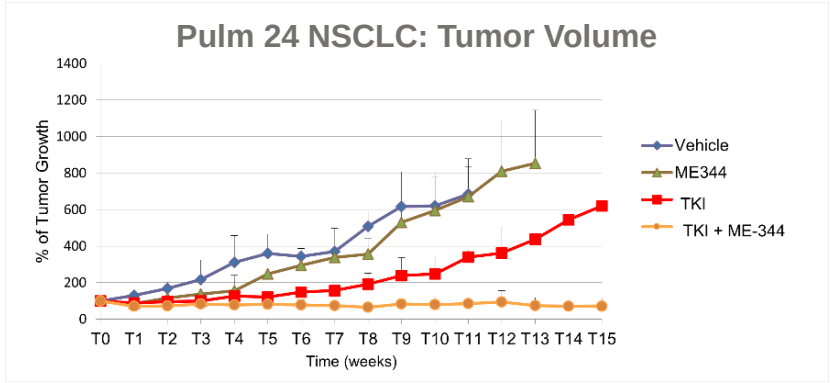
ME-344 + TKI in PyMT Breast Cancer Model[1]

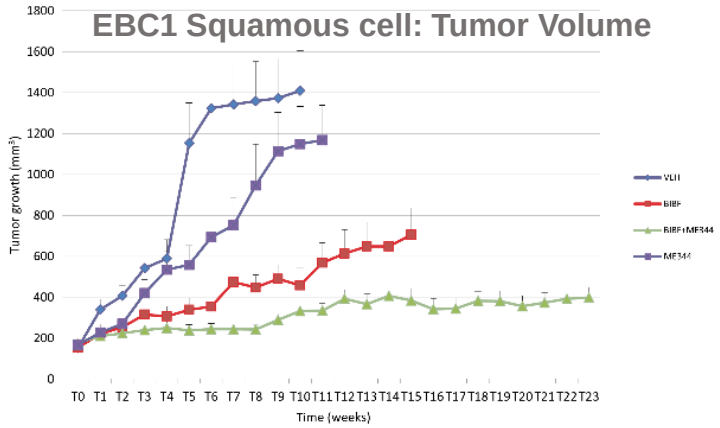




Reduced tumor growth and OXPHOS

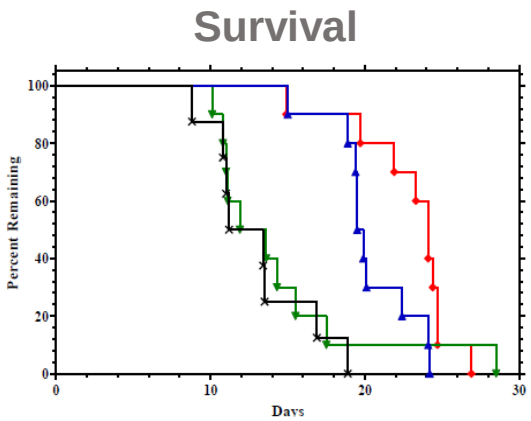
ME-344 + Nintedanib in Lung Cancer Models[2]





Reduced tumor growth

ME-344 + Regorafenib in CRC Model[2]



Tumor Growth Delay
Stivarga (60%), ME-344 (4%),
Stivarga + ME-344 (96%)

Reduced tumor growth, extended survival

MEIPharma

[1] Navarro et al, Cell Reports 2016, [2] MEI Data on file

Establishing Proof of Concept: ME-344 in Combination with Bevacizumab Demonstrates Ki67 Decrease Supporting Novel Therapeutic Strategy to Induce Synthetic Lethality in Tumors

Clinical Study Objectives

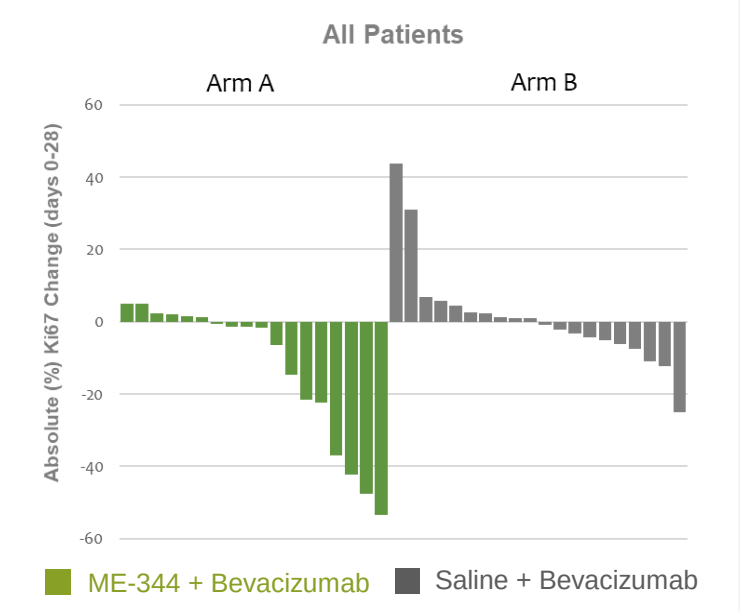
- Assess ability of bevacizumab to shift tumor reliance from glycolysis to mitochondrial metabolism
- Assess ability of ME-344 + Avastin to inhibit tumor proliferation compared to Avastin + placebo

Randomized, Controlled Phase 0 Study

Arm A **N = 21**	Bevacizumab 15 mg/kg day 1 ME-344 10mg/kg days 8, 15, 21
Arm B **N = 20**	Bevacizumab 15 mg/kg day 1 Saline days 8, 15, 21

Analysis:
FDG-PET: days 1 and 28
Biopsy: days 1 and 28

ME-344 in combination with bevacizumab in treatment-naïve HER2-negative breast cancer patients demonstrated biologic activity as evidenced by a decrease of proliferation biomarker Ki67.



Sponsored by Spanish National Cancer Research Centre

Qunitela-Fandino, Clin Cancer Res (2020) 26 (1): 35–45.

Clinical Support of Concept: Phase 1B Evaluating ME-344 Plus Bevacizumab in Patients with Relapsed Metastatic Colorectal Cancer

- Patients with mCRC disease after failure of standard therapies
- Primary objective: 16-week PFS
- Secondary objectives: ORR, OS, safety
- Continue therapy until disease progression or toxicity

- Cohort 1 (N=23)
  - ME-344 at 10 mg/kg Day 1,8, 15
  - Bevacizumab 5 mg/kg Day 1, 15

  28-day cycle
- Cohort 2 (not enrolled)
  - Option for second 20 patient Cohort 2 if PFS at 4-mo ≥20% in Cohort

- Cohort 1: Heavily pretreated patient population
  - Median of 4 prior therapies (range 1-8)
  - 100% of patients had prior chemotherapy
  - 100% of patients had prior bevacizumab
  - 39% had prior Lonsurf
- Tumor characteristics at diagnosis:
  - R & transverse colon: 26%
  - L colon: 43%
  - Rectum: 31%
- Liver metastases at enrollment: 70%

Phase 1b Further Supports ME-344 Novel Therapeutic Strategy to Induce Synthetic Lethality

Generally Well-tolerated

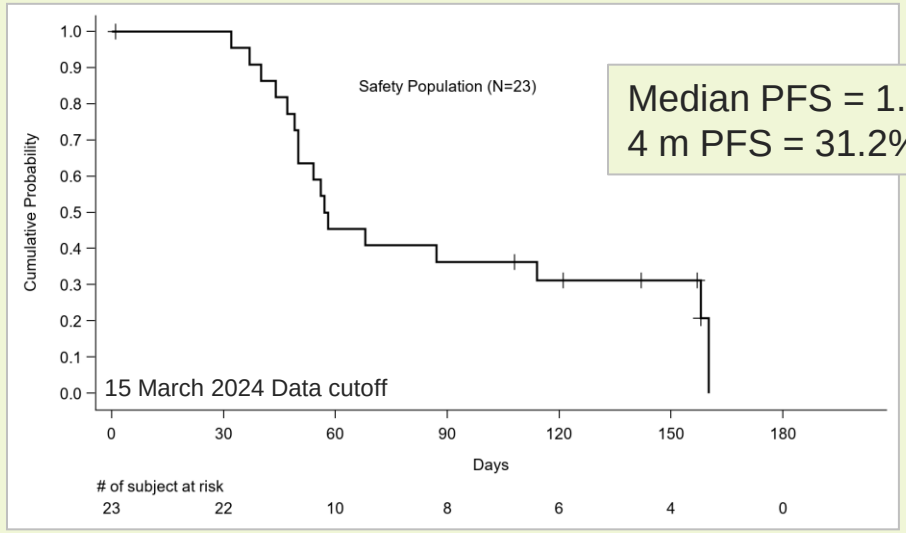
- No overlapping toxicities observed.
- 2 (9%) patients discontinued therapy due to AE: fatigue considered related to study drugs and sepsis considered unrelated.
- Most common (≥10%) drug-related adverse events (all grades/grade ≥3):
  - Fatigue in 8 (35%) / 3 (13%).
  - Abdominal pain in 3 (13%) / 2 (9%) patients.

Cohort 1 Exceeds Predetermined Non-progression Threshold

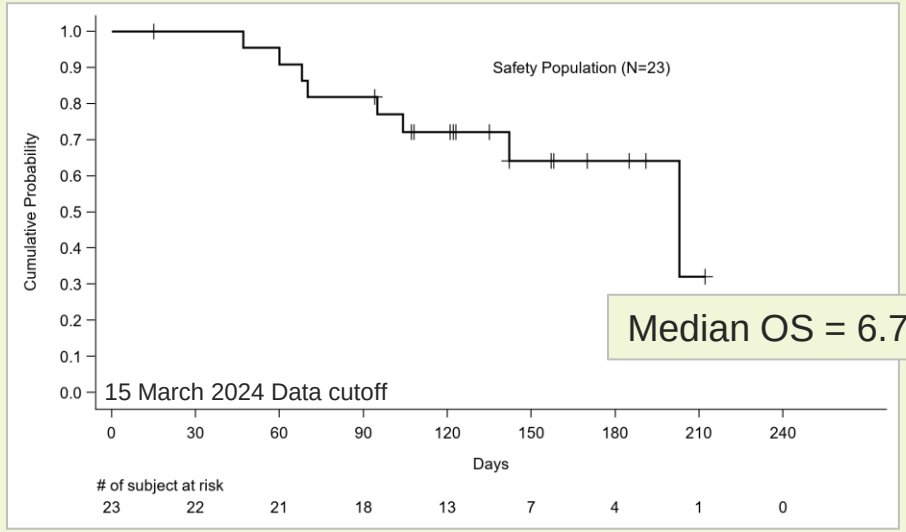
- 5 of 20 (25%) evaluable patients completed 16 weeks of therapy without evidence of disease progression, exceeding the 20% predetermined threshold as set forth in the Clinical Study Protocol.

Phase 1b: Encouraging Progression Free Survival and Overall Survival

	No. (%) of patients (N = 23)
Complete Response	0
Partial Response	0
Stable Disease	9 (39.1%)
Progressive Disease	11 (47.8%)
Not Evaluable for response	3 (13.0%)



Median PFS = 1.9 m (95% CI: 1.6-5.2)
4 m PFS = 31.2% (95% CI: 13.5-50.7)



Median OS = 6.7 m (95% CI: 3.4-NA)

All Adverse Events in ≥3 Patients

	All Subjects (N=23)					
N (%)	**Grade 1**	**Grade 2**	**Grade 3**	**Grade 4**	**Grade 5**	**All Grade**
Fatigue	4 (17.4)	2 (8.7)	3 (13.0)	0	0	9 (39.1)
Abdominal pain	3 (13.0)	3 (13.0)	2 (8.7)	0	0	8 (34.8)
Diarrhea	2 (8.7)	4 (17.4)	1 (4.3)	0	0	7 (30.4)
Constipation	4 (17.4)	1 (4.3)	0	0	0	5 (21.7)
Blood sodium decreased	2 (8.7)	2 (8.7)	0	0	0	4 (17.4)
Nausea	2 (8.7)	1 (4.3)	1 (4.3)	0	0	4 (17.4)
Vomiting	2 (8.7)	1 (4.3)	1 (4.3)	0	0	4 (17.4)
Blood bilirubin increased	1 (4.3)	0	2 (8.7)	0	0	3 (13.0)
Dehydration	0	0	3 (13.0)	0	0	3 (13.0)
Hypertension	0	1 (4.3)	2 (8.7)	0	0	3 (13.0)
Leukocytosis	0	0	3 (13.0)	0	0	3 (13.0)
Non-cardiac chest pain	3 (13.0)	0	0	0	0	3 (13.0)

ME-344 to Continue Advancing via Development of a New Formulation

- **ME-344 New Formulation with Potential to:**
  - Increase Biological Activity
  - Improve Patient Convenience
  - Enhance Commercial Opportunity

New Formulation Update Expected H1 2025

Financial Overview

Financial Highlights

- As of March 31, 2024, MEI had $56.6 million in cash, cash equivalents, and short-term investments with no outstanding debt.

- The Company believes its cash balance is sufficient to fund operations for at least the next 12 months

MEI Pharma

Corporate Overview

Novel drug candidates to address known resistance mechanisms to standard-of-care cancer therapies

May 21, 2024