Fiscal Year 2023 Results and Corporate Overview

September 26, 2023

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; 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 our failure 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 may differ 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.

MEI Pharma

Corporate Overview

Two Oncology Candidates in Clinical Development with Near-term Data Readouts

- **Ongoing clinical studies of 2 assets with novel MOAs**
  - Cyclin-dependent Kinase 9 (CDK9) inhibition
  - Mitochondrial inhibition
- **Existing clinical and preclinical data sets support**
  - Clinical development plan
  - Combination regimens
- **Combinations directed at known resistance mechanisms to established SOC therapies**
  - Combinations with venetoclax and bevacizumab
  - Clear medical needs
  - Significant commercial opportunities
- **Engaged clinical investigators at leading institutions**

Near-term data readouts for both programs.

INVESTIGATIONAL AGENTS	THERAPEUTIC AREA	COMBINATION	PHASE 1/1B	PHASE 2	PHASE 3	CLINICAL DATA
Voruciclib Oral CDK9 Inhibitor	**Acute Myeloid Leukemia** Relapsed/refractory (2L+)	Monotherapy VENCLEXTA® (venetoclax)	Completed Enrolling			Early 2024
ME-344 Mitochondrial Inhibitor	**HER2-negative Breast Cancer***	AVASTIN® (Bevacizumab)	Completed			
	Colorectal Cancer Relapsed	AVASTIN® (Bevacizumab)	Enrolling			H1 2024

*Phase 0 window of opportunity study: investigator initiated, randomized, open label.

Voruciclib: A Potential First-in-Class Oral Cyclin-dependent Kinase 9 (CDK9) Inhibitor

Important Target; Encouraging Early Results

- Evidence of clinical activity as a single agent in R/R AML and B-cell malignancies
- Evidence of clinical activity in combination with venetoclax in R/R AML and B-cell
- No DLTs and MTD not reached as a single-agent at plasma concentrations sufficient to inhibit target
- Decrease in Mcl-1 and MYC observed in patient samples, supporting target inhibition
- Safety and preliminary efficacy data of the dose ascending combination with venetoclax in R/R AML in early 2024

KOLs Involved in Voruciclib Development Program

- Alexey Danilov, MD, PhD, co-Director, Lymphoma Program, City of Hope, and Voruciclib SAB Chair
- Mathew Davids, MD, Director, Clinical Research, Dana Farber Cancer Institute, Ph 1 study co-chair
- Yesid Alvarado-Valero, MD, MD Anderson Cancer Center, Ph 1 study co-chair
- Vijaya R. Bhatt, MD, Medical Director, Leukemia Program, University of Nebraska Medical Center, and NCCN AML Panel member

ME-344: A Potential First-in-Class Mitochondrial Inhibitor

Novel Target; Promising Opportunity

- New approach to treatment: inhibit energy production in tumor cells to induce an antitumor effect
- 42 patient, controlled window of opportunity clinical study demonstrated initial potential of ME-344 in combination with Avastin
- Generally well-tolerated and evidence of clinical activity as a single-agent in earlier clinical studies at dose currently being investigated in Phase 1b
- Safety and preliminary efficacy data from first 20 patient cohort of Phase 1b expected H1 2024

KOLs Involved in ME-344 Development Program

- Howard Hochster, MD, Distinguished Professor, and Director, GI Oncology, Rutgers Cancer Institute
- Patrick Boland, MD, GI Oncology, Rutgers Cancer Institute. Study Chair
- Heinz-Josef Lenz, MD, Professor Medicine and J. Terrence Lanni Chair, Gastrointestinal Cancer Research, Keck School of Medicine; co-director Center for Molecular Pathway and Drug Discovery, and Norris Center for Cancer Drug Development of USC
- Academic GI Cancer Consortium (AGICC) investigators

Large Commercial Opportunity in AML in Combination with Venclexta: Other B Cell Malignancies Also Present Opportunities

Venclexta generated ~$2B in 2022 WW sales; projected to grow to ~$3.4B by 2028

	AML	CLL	DH DLBCL
US Patient Population	1L Tx : ~11,300 (~40% unfit) R/R Tx : ~7,400	2L Tx : ~11,100 3L Tx : ~5,300	2L Tx: ~9,400 (20% DH) 3L Tx: ~4,100 (35% DH)
All Product Sales (Global)	1L: $2.6B by 2028 R/R: $845M by 2028	2L: $3.8B by 2028 3L: $2.3B by 2028	2L: $4.2B by 2028 3L: $2.9B by 2028
Venclexta Patient Share (US)	1L Unfit: 35% (48% by 2028)	2L: 19% 3L: 13%	2L: Ven not currently used but unmet need is high for improved outcomes

"Venetoclax-based combinations, which have become the standard of care for patients who are ineligible for intensive induction, and FLT3 inhibitors (mainly gilteritinib)**, will drive the AML market."**

Clarivate™

Source: Evaluate June 2023

Large Commercial Opportunity Exists Across Multiple Solid Tumors for ME-344 If POC is Successful in Combination with Avastin in Colorectal Cancer

Avastin and bevacizumab biosimilars generated ~$2B in 2022 WW sales; projected to grow to ~$3.3B by 2028

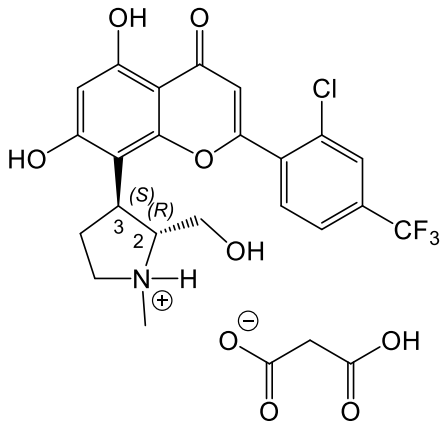
	CRC	HCC	Ovarian	Cervical	GBM
US Patient Population	2L Tx : ~13,000 3L Tx : ~4,800	1L Tx : ~11,800 2L Tx : ~4,500	1L Tx : ~17,000 2L Tx : ~11,300	1L Treatable: ~5,200	1L Treatable: ~15,000
All Product Sales (Global)	2L: $1.3B by 2028 3L: $889M by 2028	1L: $4.1B by 2028 2L: $500M by 2028	1L: $11.5B by 2028 2L: $1.1B by 2028	Sales of newer agents projected to reach $1B by 2028	Temodar peak sales of $1B in 2010 with subsequent LOE in 2013
Avastin Patient Share (US)	2L: 43% 3L: 30%	1L: 36%	1L: 21% (30%+ by 2028) 2L: 30%/23% in platinum refract/sensitive	Avastin remains SOC and continued use driven by CPI/chemo combo	Avastin still widely used in recurrent GBM due to lack of new Tx options

Source: Evaluate June 2023

Two Oncology Candidates in Clinical Development with Near-term Data Readouts

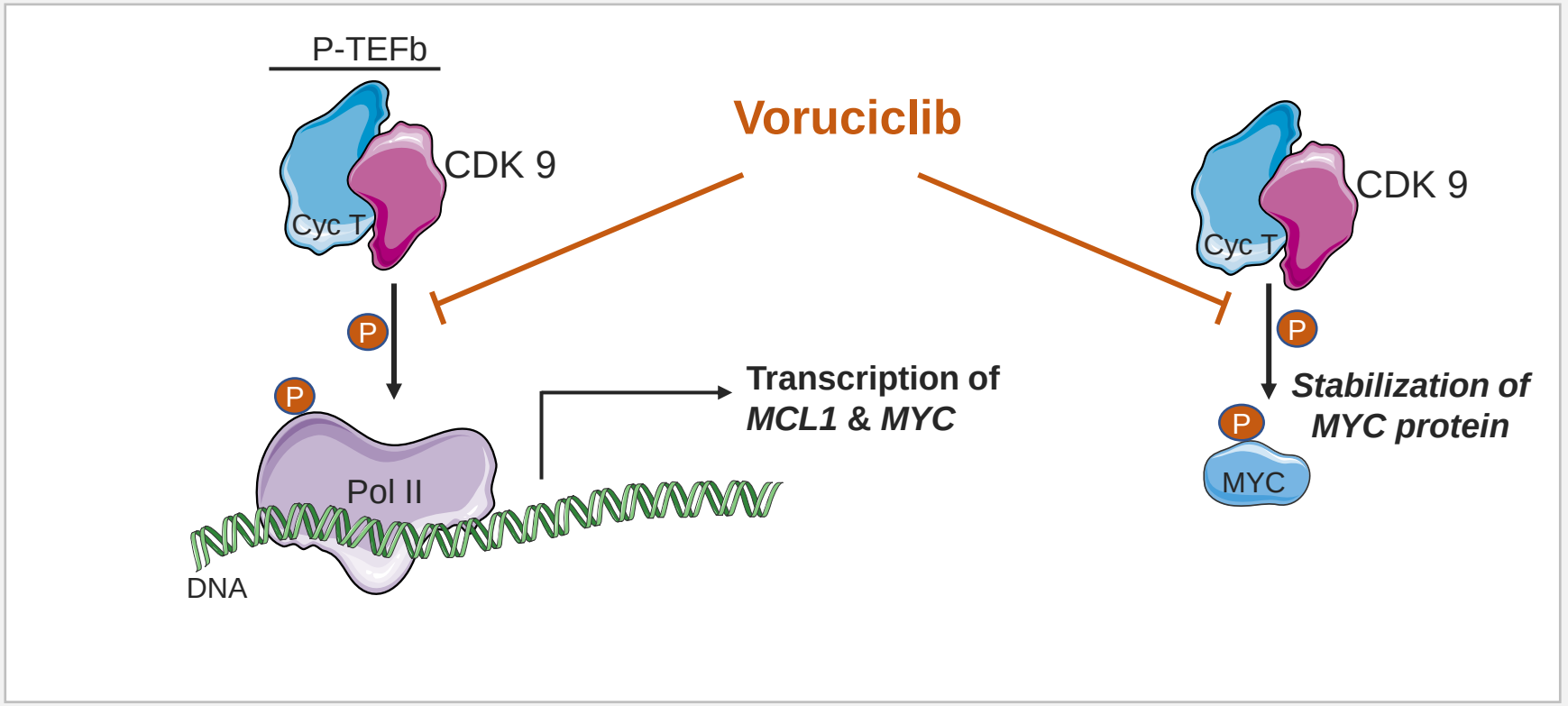
- Two Programs in Ongoing Clinical Studies
- Significant Commercial Opportunities
- Support for Mechanisms and Anti-Tumor Activity
- Engaged clinical investigators at leading institutions
- Combinations with Current SOC Therapies

Near-term data readouts for both programs



Voruciclib: A Selective Oral CDK9 Inhibitor Drug Candidate

Voruciclib Modulates Two Important CDK9 Interactions for MCL1 and MYC



Dey, et al. Sci Reports (2017) 7(1):8007. Li, et al. Transcription (2018) 9(2):88. Luedtke, et al. Signal Transduct Ther (2020) 5(1):17. Dollinger, et al. J Mol Biol (2021) 433(14):1666897. Wiley, et al. Cancer Research (2021) 81(13 supp):1962

Voruciclib Holds Favorable PK/PD Attributes

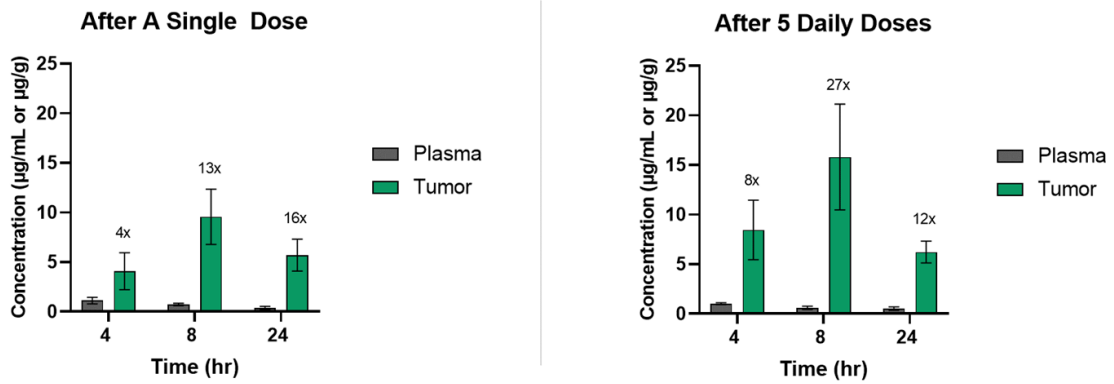
- **Oral administration**
- **Selective**
  - Higher specificity and longer residence time on target vs CDK 6, 4 &1
  - Greater selectivity against CDKs relative to other kinases
- **Potent**
  - IC_{50} from 0.2 to 1.7 µM in various cell lines
- **Concentrates in Tumor Over Plasma**

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. Nature Sci Rep. 2017; 7:18007

Voruciclib Concentrates in Tumor Tissue Over Plasma



HCT-116 CRC cell xenograft in SCID mice. The accumulation index in tumors after 5 days of repeat dosing was 1.45. Concentration in plasma in µg/ml and in tumors in µg/g for tumors. Voruciclib fold increase in tumors relative to plasma are indicated. Data on file.

Voruciclib is a Selective Oral CDK9 Inhibitor Targeting Cell Proliferation Regulation

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

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Focusing on Hematologic Malignancies in Indications Where Venetoclax is Used: Starting with AML

- Venetoclax-based therapies SOC in elderly/unfit AML patients
  - Ongoing studies to establish venetoclax as SOC in chemotherapy-eligible patients

Voruciclib + venetoclax has potential to:

- **Restore sensitivity to venetoclax**
- **Improve durability of response**

"Venetoclax-based combinations, which have become the standard of care for patients who are ineligible for intensive induction, and FLT3 inhibitors (mainly gilteritinib)**, will drive the AML"**

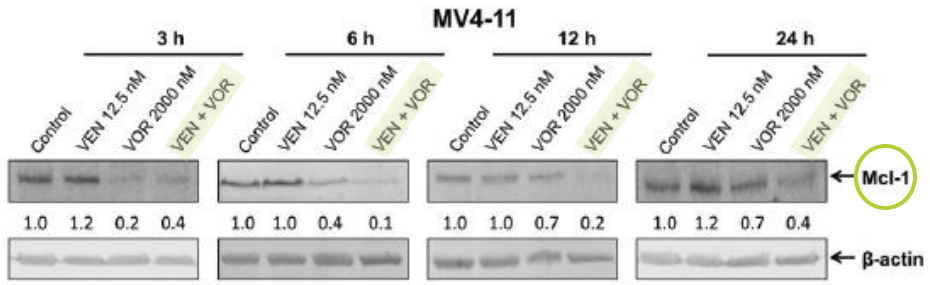
market.

Clarivate™

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

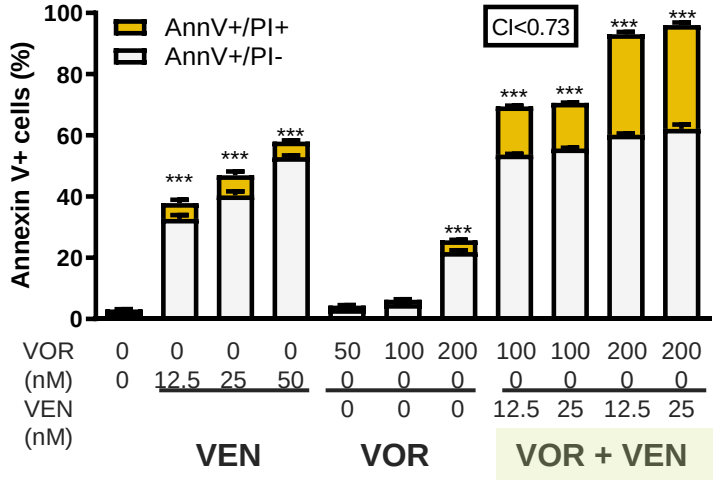
Suppresses Mcl-1 Level

a.



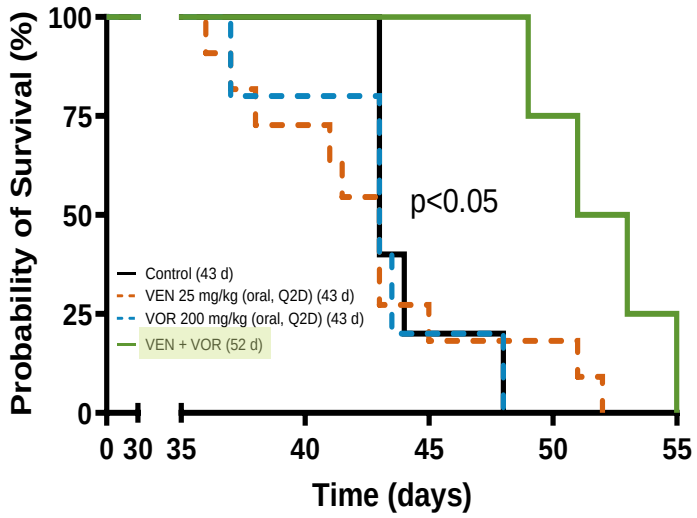
Increases Apoptosis

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]

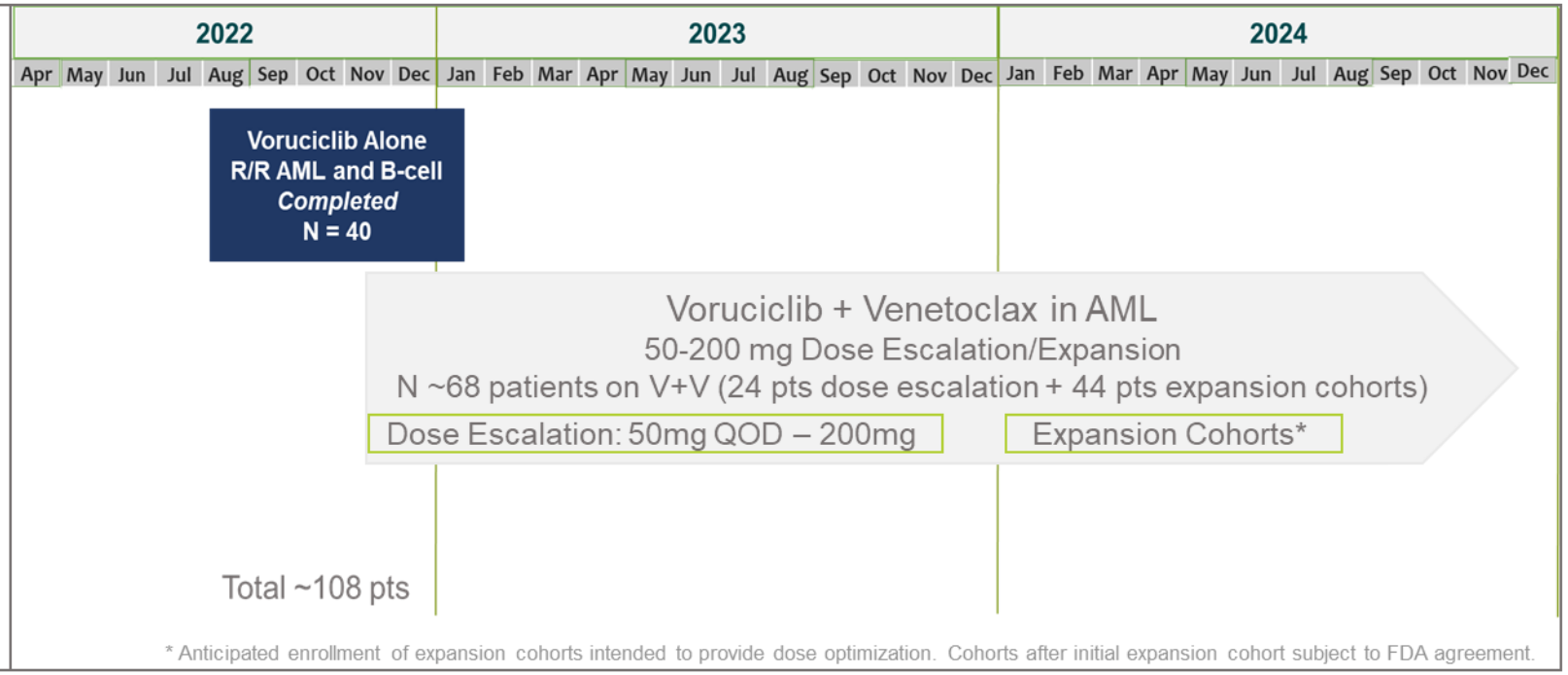
1. Lesnick et al. J Clin Oncol.2020;38(15_suppl):e20009; Paiva et al, PLoS ONE. 2015;10(11):e0143685; 3. Dey et al. Nature Sci Rep. 2017; 7:18007

Phase 1 Study: Generate Data in up to ~108 Patients to Demonstrate Safety & Evidence of Activity as a Single Agent and in Combination with Venetoclax, Plus Provide Biologic Correlates

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

Dose escalation with standard 3+3 design
- Single agent
- In combination with venetoclax

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



Enrolling: **Voruciclib + Venetoclax**

All V+V patients enrolled are heavily pretreated with a median of 2 prior therapies and have previously progressed after receiving treatment with venetoclax.

V^2 50 qod → 50 mg → 100 mg → 150 mg → 200 mg

Enrollment Complete

Initiation of first expansion cohort upon confirmation of dose that appears well-tolerated with evidence of biologic activity

Monotherapy Safety Results Suggest No Overlapping Toxicity with Venetoclax in Patients with AML or B-cell Malignancies

- Patient characteristics
  - 21 AML, 19 B-cell malignancies
  - Median prior therapies: 3 (range, 1-8)
- Administration schedule
  - Group I: Daily continuously (N = 16 pts)
  - Group II: 14 days on /14 days off (N = 24 pts)
- Group I: 2 DLTs of pneumonitis at 100 mg, confounded by differentiation syndrome and prior allogeneic HSCT
- Group II at 100, 150 and 200 mg: No DLTs
- Antitumor activity
  - 1 AML pt had MLFS
  - 5 of 10 pts with AML at 200 mg had stable disease
- Decreased MCL-1 and MYC by scRNA-Seq from 3 CLL and 2 AML serial samples

Voruciclib at doses up to 200 mg for 14 days in a 28-day cycle:

- Well-tolerated and no DLT
- No drug-related neutropenia
- No Grade 3+ drug related toxicity
- No discontinuation due to drug related toxicity

Promising Early Data in Dose Escalation Demonstrates Tolerability and Evidence of Activity of Voruciclib Plus Venetoclax Combination

- No DLTs observed
- PK analysis does not show drug-drug interaction
- Clinical activity observed at low doses
  - Reduced transfusions
  - Improved counts
  - Responses observed
  - 85% of patients continue therapy beyond DLT window

All V+V patients are heavily pretreated with a median of 2 prior therapies including venetoclax

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

Mcl-1:

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MYC:

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- **CDK9 inhibition leads to reduced transcription and stability of Myc**

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

Monotherapy Phase 1 Studies in Solid Tumors Evaluated Higher Doses and Demonstrated No Myelosuppression and Evidence of Activity

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

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

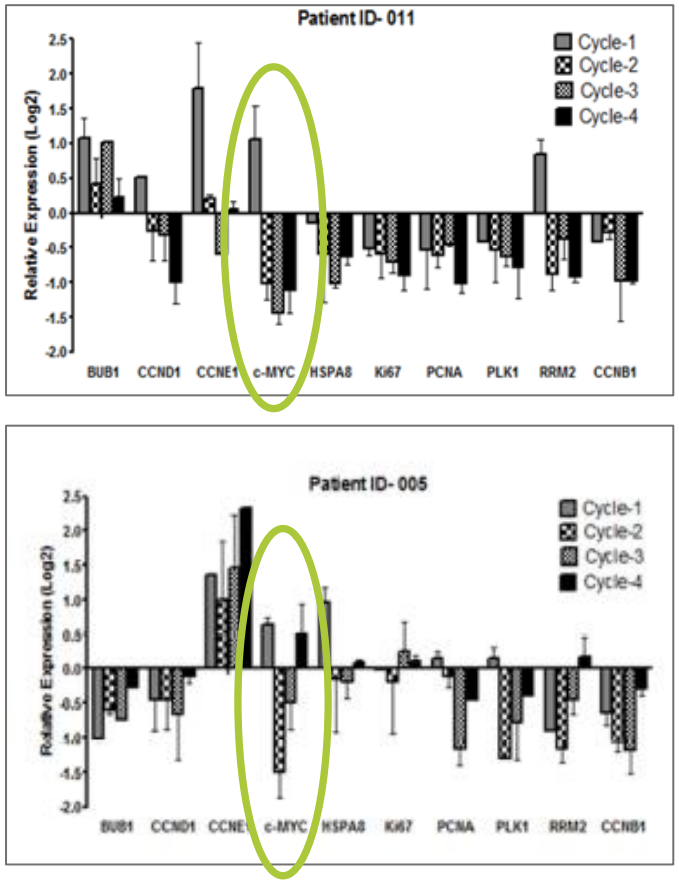
Daily continuously schedule (N = 39 pts)

- 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

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

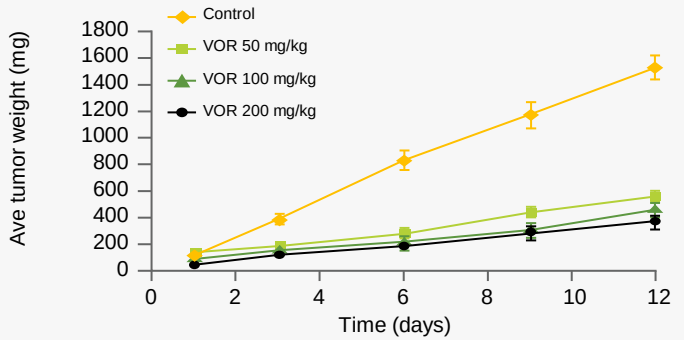
VOR inhibited proliferation of KRAS mutant cell lines from multiple indications

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

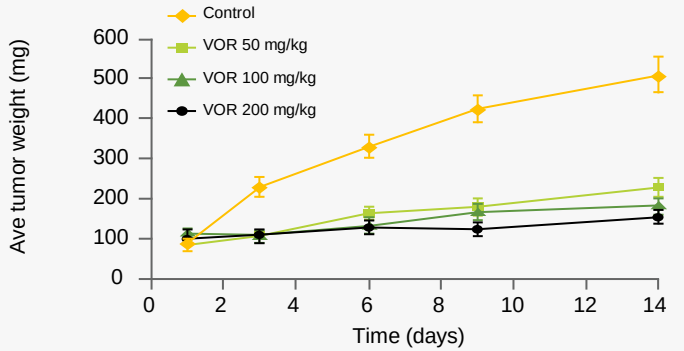
VOR inhibited proliferation of cell lines with various KRAS mutations

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

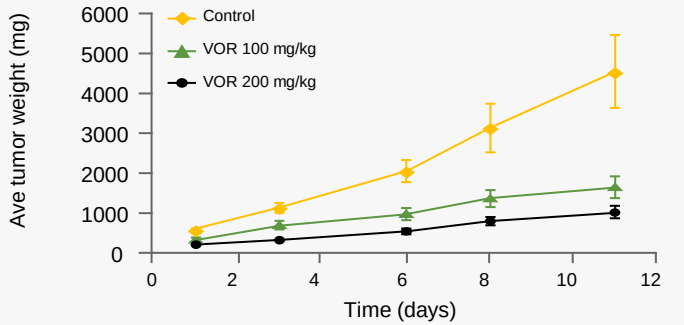
*Sotorasib more effective in STK11 mut, Keap1 WT, TP53 WT patients



HCT-116
(CRC, KRAS G13D)



SW-480
(CRC, KRAS G12V)

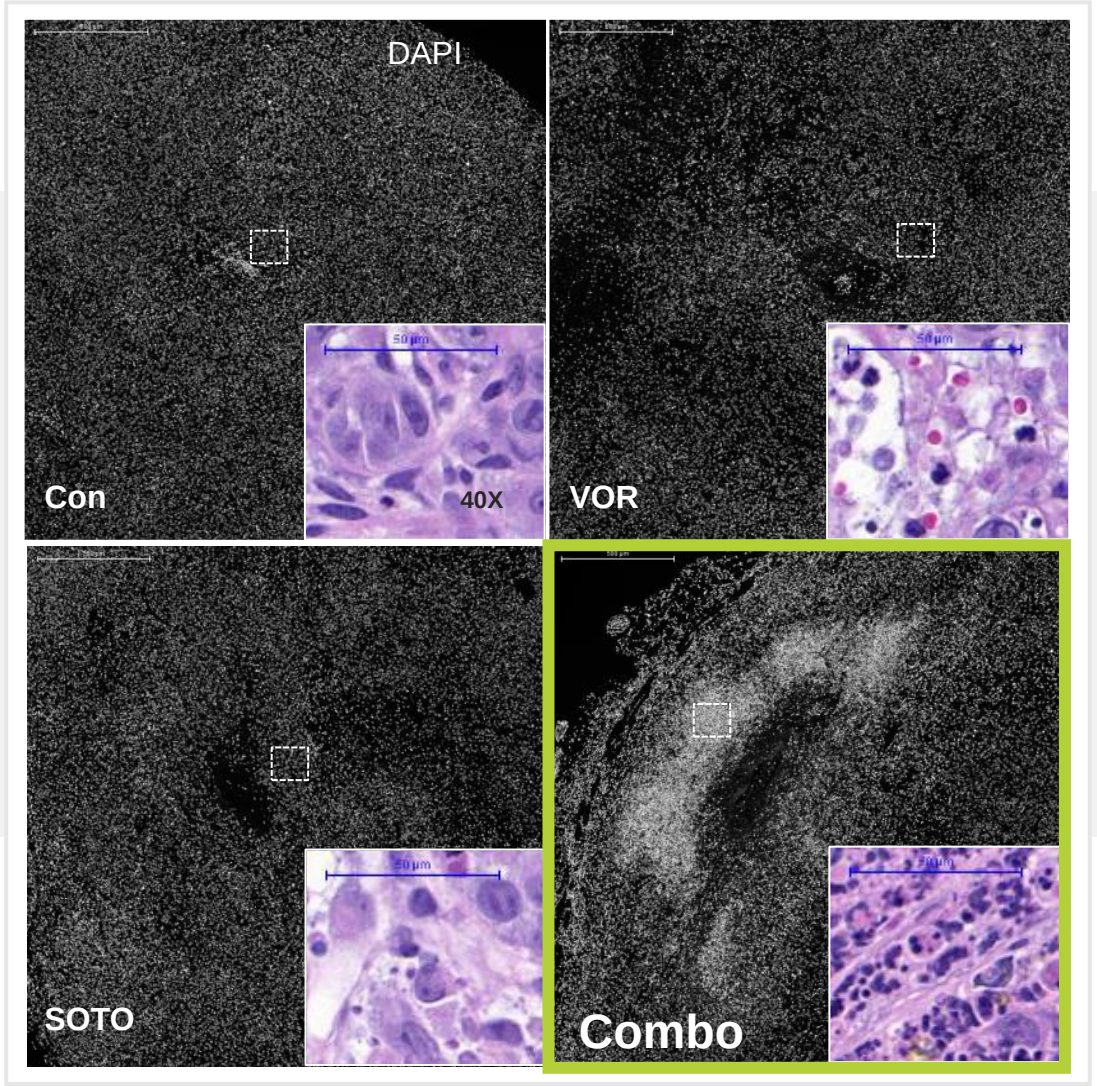


H460
(NSCLC, KRAS Q61H)

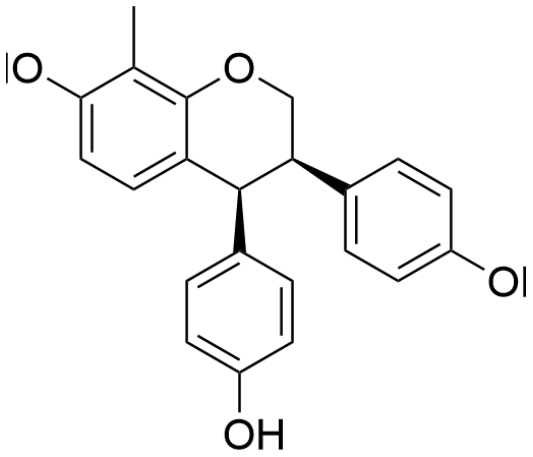
Wiley, SE. et al. AACR, 2021 & unpublished.

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: A Mitochondrial Inhibitor Drug Candidate

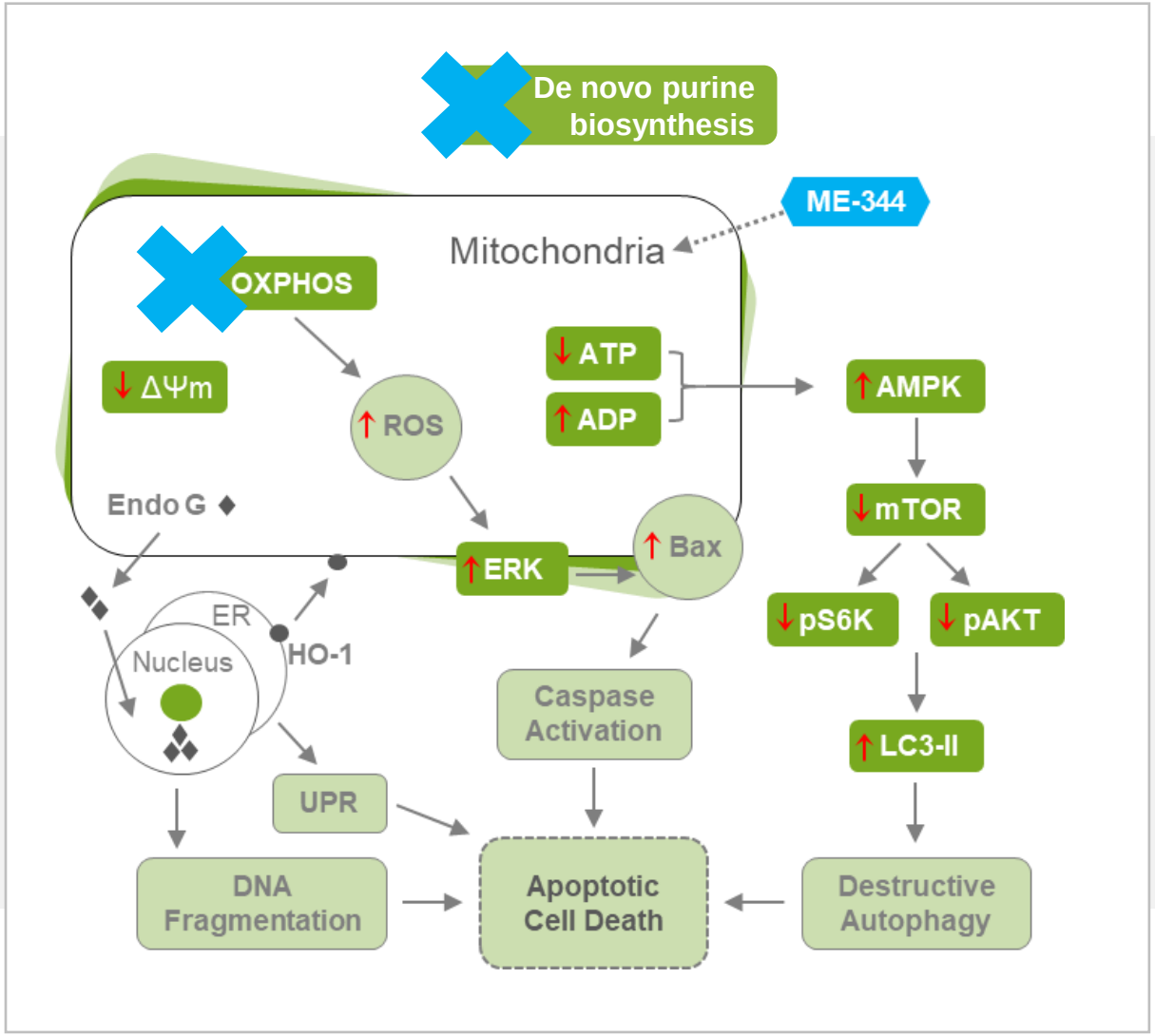
ME-344: A Novel Mitochondria Inhibitor with Dual Effect on OXPHOS and Purine Synthesis

Inhibition of OXPHOS

- Reduced mitochondrial membrane potential and OXPHOS lead to decreased ATP[1]
- Decreased ATP/ADP induces cell death through multiple signaling pathways

Reduction in Purine Biosynthesis

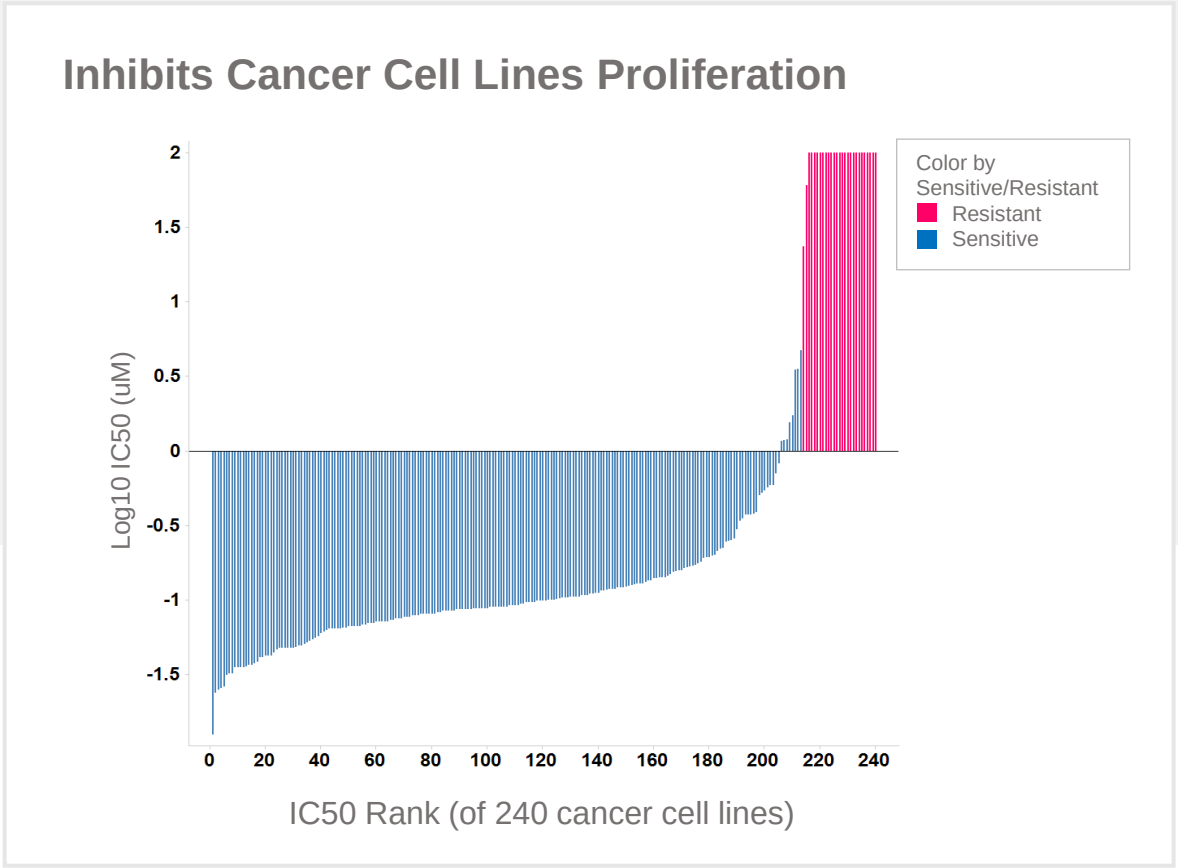
- ME-344 decreases de novo purine biosynthesis & increases the purine salvage pathway
- ME-344 can synergize with other agents that affect mitochondrial function (e.g., venetoclax)



[1] *Am J Cancer Res. 2015 Jan 15;5(2):689-701*

ME-344 as a Single Agent is an Inhibitor of Cancer Cell Proliferation in Pre-Clinical Models

ME-344 displays nM potency against cell lines from multiple solid tumors and AML



ME-344 Initial Studies in Solid Tumors

Ph 1 Study
Single Agent

Bendel, Cancer 2015

- 1.25 to 20 mg/kg weekly in 28-day cycles
- Refractory solid tumors
- 30 pts
- Maximum Tolerated Dose (MTD) = 10 mg/kg
- Dose Limiting Toxicity (DLT) = Gr 3 neuropathy at 15-20 mg/kg
- 1 Partial Response (PR) in small cell lung cancer and 10 stable disease (SD)
- Disease control rate = 37%

Ph 1-2 Study
with Topotecan

Diamond, Invest New Drugs 2017

- 10 mg/kg + topotecan 4 mg/m^2 Days 1, 8, 15
- R/R small cell lung cancer and ovarian cancer
- 46 pts
- Myelosuppression due to topotecan
- 1 PR in ovarian cancer and 21 SD
- Disease control rate = 49%

ME-344 Monotherapy Phase 1 Dose Escalation Study - Key Safety Findings

Treatment-Related Adverse Events in ≥10% of Patients (N= 30)

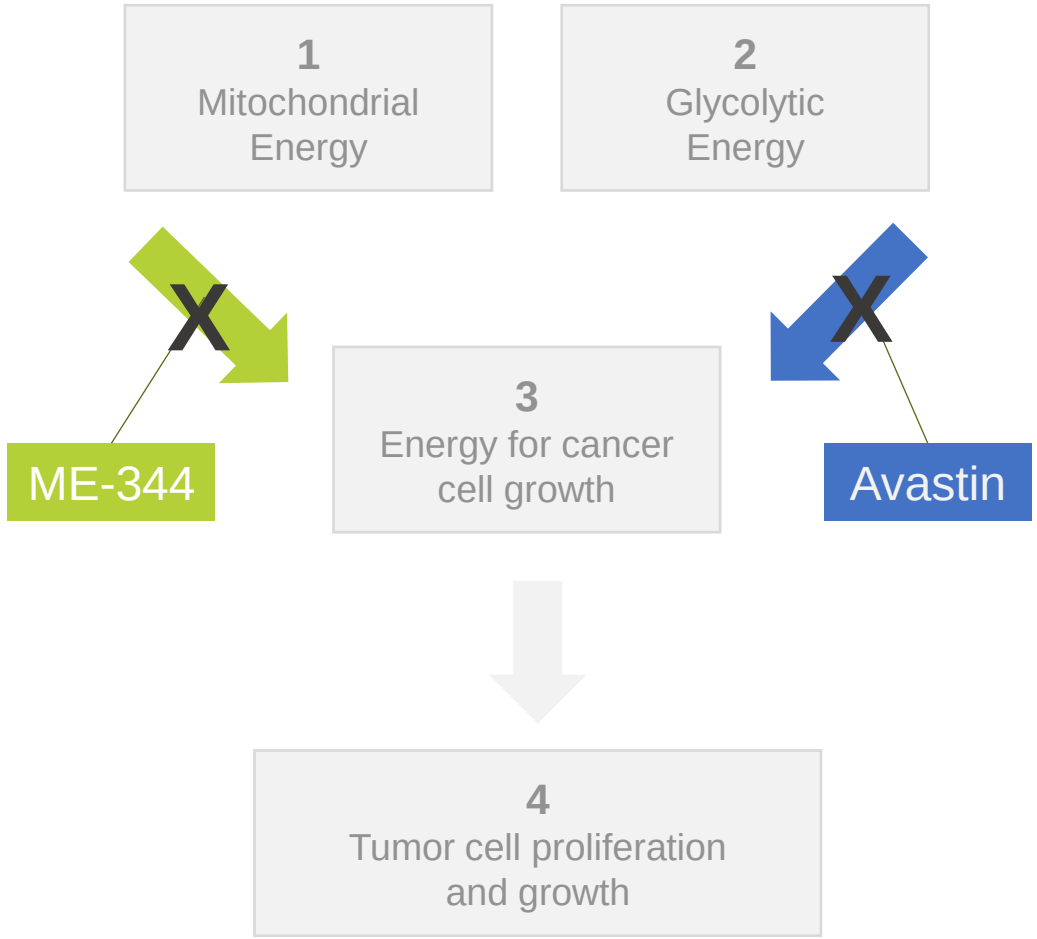
Adverse Event[a]	Grade 1	Grade 2	Grade 3	Total
Neuropathy[b]	1 (3%)	1 (3%)	4 (14%)	6 (20%)
Nausea	4 (13%)	2 (7%)	0	6 (20%)
Dizziness	3 (10%)	1 (3%)	2 (7%)	6 (20%)
Fatigue	2 (7%)	3 (10%)	0	5 (17%)
Vomiting	2 (7%)	2 (7%)	0	4 (13%)
Diarrhea	1 (3%)	2 (7%)	0	3 (10%)
Asthenia	1 (3%)	1 (3%)	1 (3%)	3 (10%)

[a] No grade 4 treatment-related adverse events reported.
[b] Includes peripheral neuropathy, peripheral motor neuropathy, and peripheral sensory neuropathy

Bendel et al. Cancer 2015

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

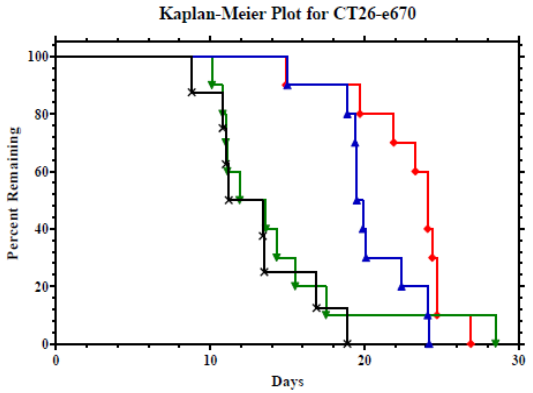
1. ME-344 blocks the production of ATP by inhibiting the OXPHOS pathway
2. Anti-angiogenic therapies reduce glycolysis
3. Cancer cells switch between mitochondria and glycolytic metabolic pathways to escape the blocking of either energy source
4. The potential to inhibit both mitochondrial energy production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



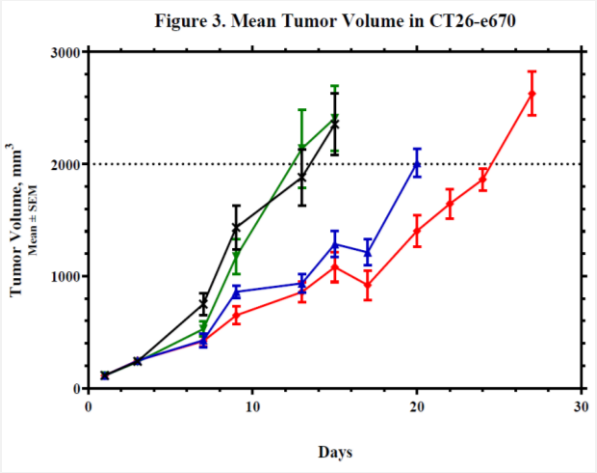
ME-344 Synergizes with Anti-angiogenic TKI in Various Solid Tumors Models

ME-344 + Regorafenib in CRC Model

Survival



Tumor Volume



G1:vehicle

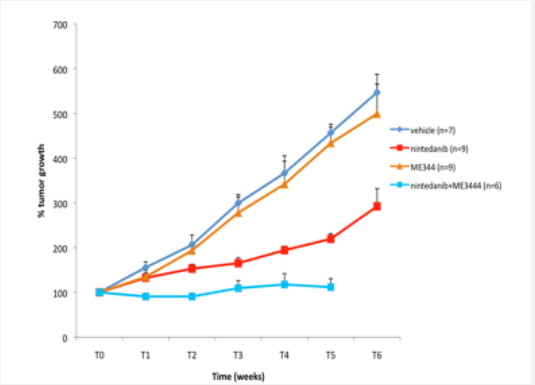
G2:Stivarga(10)po;(5/2) x3

G3:ME-344(50)ip;Days 1,3,5,8,10,12,15,17,19

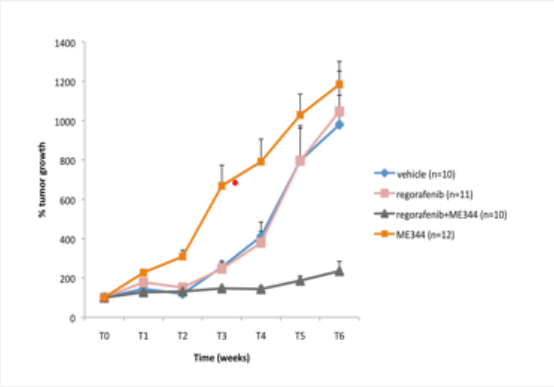
G4:Stivarga(10)po;(5/2) x3 / ME-344(50)ip;Days 1,3,5,8,10,12,15,17,19

ME-344 + TKI in PyMT Breast Cancer Model

Tumor Volume



Nintedanib + ME-344



Regorafenib + ME-344

MEI Data on file

Clinical Proof of Concept Study of ME-344 in Combination with Bevacizumab to Prevent Anti-Angiogenic Escape in Breast Cancer

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

Treatment-naïve HER2-negative breast cancer

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

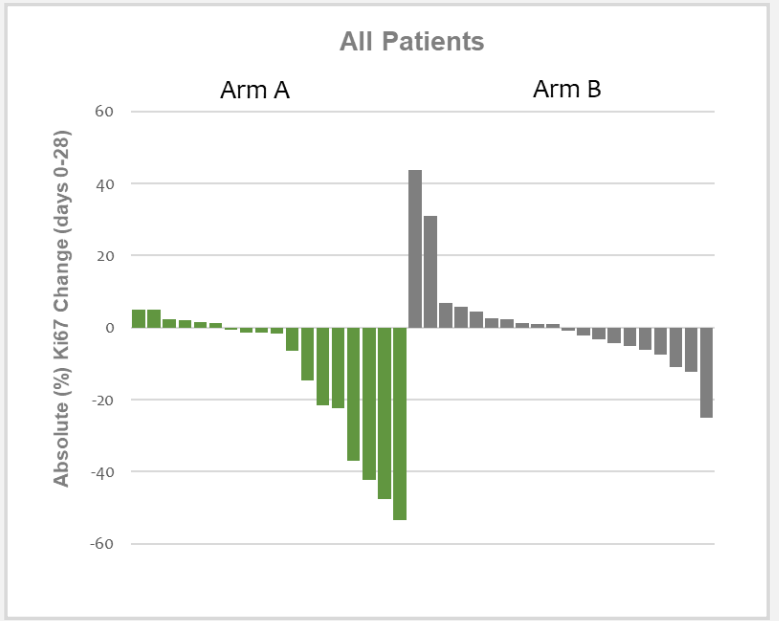
Sponsored by Spanish National Cancer Research Centre

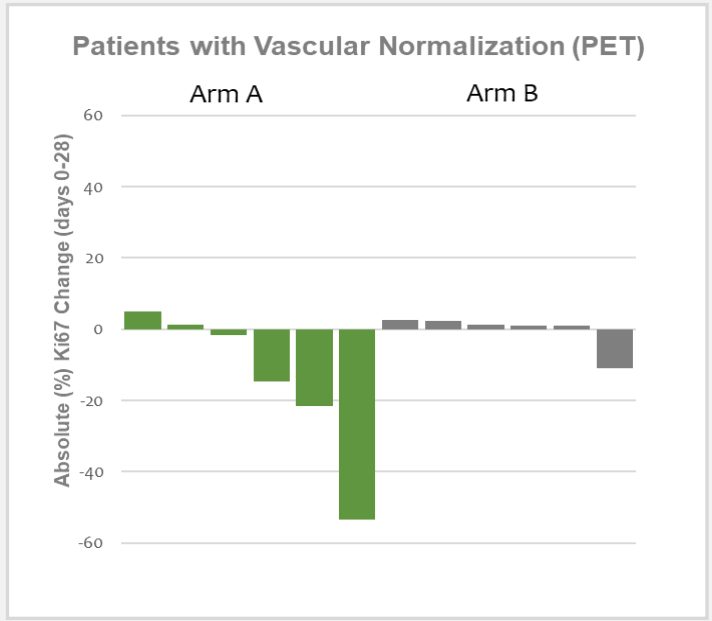
Analysis:
FDG-PET: days 1 and 28
Biopsy: days 1 and 28

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

ME-344 in Combination with Bevacizumab Shows Decrease in Ki67

ME-344 in combination with bevacizumab in Her2-negative breast cancer patients demonstrated biologic activity as evidenced by a decrease of proliferation biomarker Ki67





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

Phase 1b Study Intended to Show Clinical Proof-of-Concept of ME-344 in Combination with VEGF Inhibition in Recurrent Metastatic Colorectal Cancer

Relapsed/Refractory Colorectal Cancer

- Patients with progressive disease after failure of standard therapies and no available approved options

Primary Objective: PFS

Secondary Objectives: OS, safety

Aug. 2023

Cohort 1

ME-344 at 10 mg/kg Day 1, 8, 15

Bevacizumab 5 mg/kg Day 1, 15

28-day cycle

N = 20

(Open cohort 2 if 4-mo PFS in Cohort 1 > 20%)

Data Read Out H1 2024

If PFS at 4 months 20% or higher

Cohort 2

ME-344 at 10 mg/kg Day 1,15

Bevacizumab 5 mg/kg Day 1, 15

28-day cycle

N = 20

Up to ~60 Patients Total, Including 2 Expansion Cohorts

Treatment Until Disease Progression or Unacceptable Toxicity

Financial Overview

Financial Highlights

- As of June 30, 2023, MEI had $100.7 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, and through the reporting of clinical data readouts from the ongoing and planned voruciclib and ME-344 Phase 1 and Phase 1b clinical programs, respectively.

Two Oncology Candidates in Clinical Development with Near-term Data Readouts

- **Ongoing clinical studies of 2 assets with novel MOAs**
  - Cyclin-dependent Kinase 9 (CDK9) inhibition
  - Mitochondrial inhibition
- **Existing clinical and preclinical data sets support**
  - Clinical development plan
  - Combination regimens
- **Combinations directed at known resistance mechanisms to established SOC therapies**
  - Combinations with venetoclax and bevacizumab
  - Clear medical needs
  - Significant commercial opportunities
- **Engaged clinical investigators at leading institutions**

Near-term data readouts for both programs.

INVESTIGATIONAL AGENTS	THERAPEUTIC AREA	COMBINATION	PHASE 1/1B	PHASE 2	PHASE 3	CLINICAL DATA
Voruciclib Oral CDK9 Inhibitor	**Acute Myeloid Leukemia** Relapsed/refractory (2L+)	Monotherapy VENCLEXTA® (venetoclax)	Completed Enrolling			Early 2024
ME-344 Mitochondrial Inhibitor	**HER2-negative Breast Cancer***	AVASTIN® (Bevacizumab)	Completed			
	Colorectal Cancer Relapsed	AVASTIN® (Bevacizumab)	Enrolling			H1 2024

*Phase 0 window of opportunity study: investigator initiated, randomized, open label.

MEI Pharma

Q&A

Fiscal Year 2023 Results and Corporate Overview

September 26, 2023