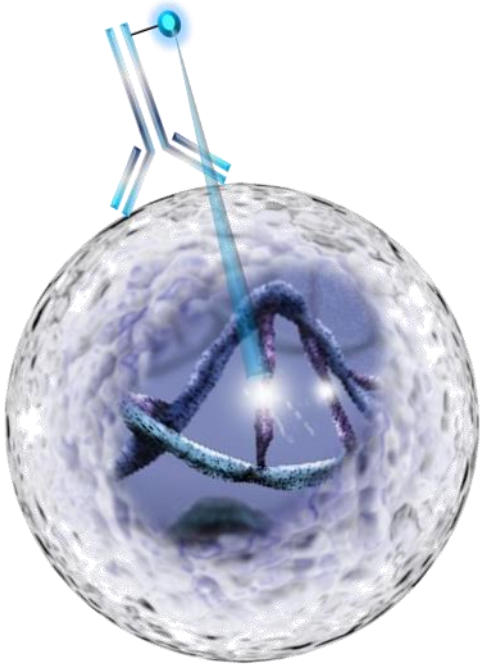
Actinium Pharmaceuticals, Inc.



Leveraging the strengths of

Antibody Radiation Conjugates

to build the

Best in Class CD33 Program

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Today's Featured Speakers

UCLA Health
David Geffen School of Medicine

Dr. Gary Schiller

Professor, Hematology-Oncology
Director, Hematologic Malignancy/Stem Cell Transplant Program

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

Dr. Hagop Kantarjian

Professor & Chair
Department of Leukemia

Dr. Tapan M. Kadia

Assistant Professor
Department of Leukemia

Actinium Pharmaceuticals, Inc.

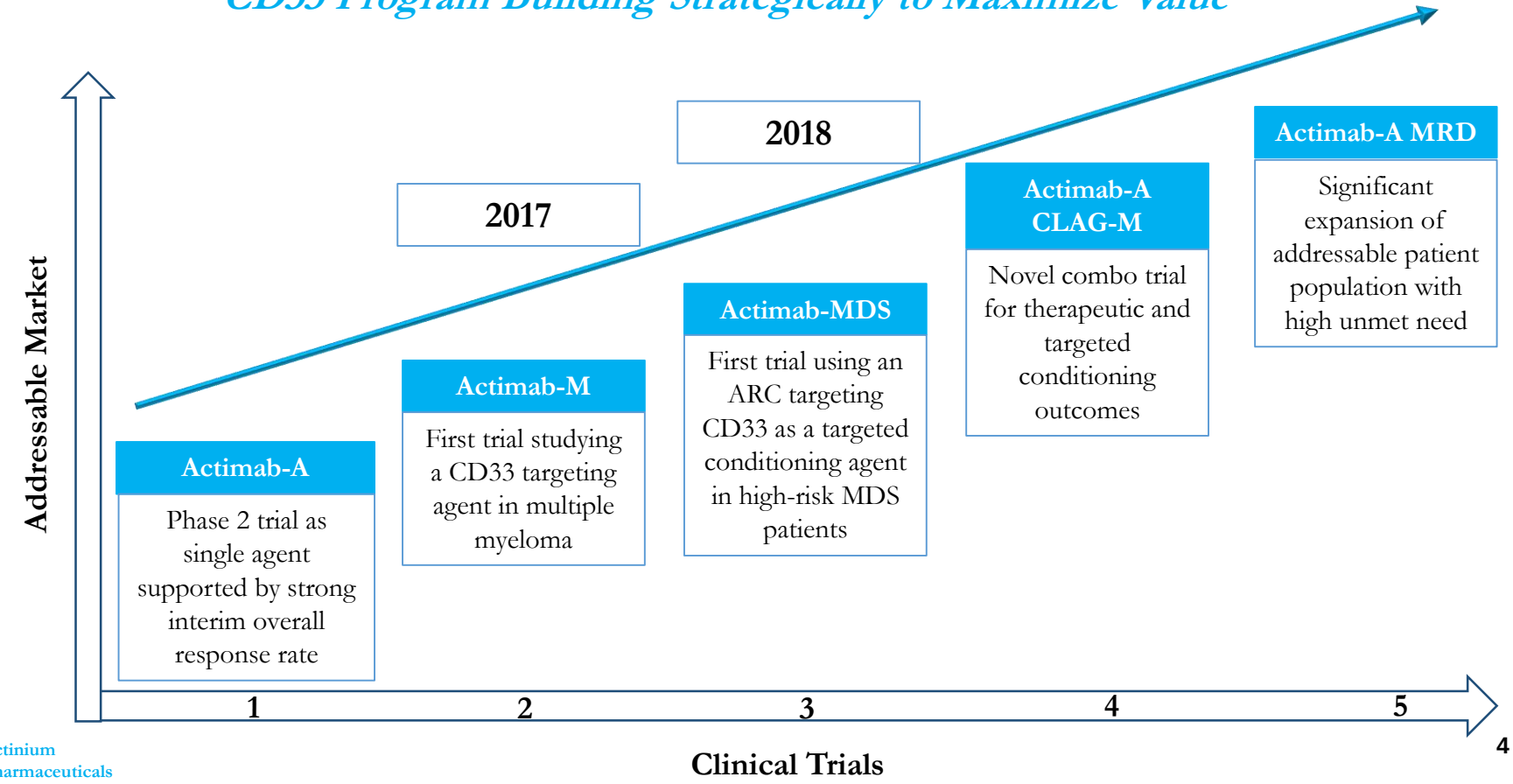
Sandesh Seth
Chairman & CEO

Dr. Mark Berger
Chief Medical Officer

Progressing and Expanding Actimab CD33 Program

- Only multi-disease, multi-indication CD33 program - 5 trials in 3 diseases with Ac-225 - Lintuzumab
- Expansion enabled by highly differentiated Antibody Radiation Conjugate (ARC) technology
- Broadening of program driven by interest from and in collaboration with key opinion leaders

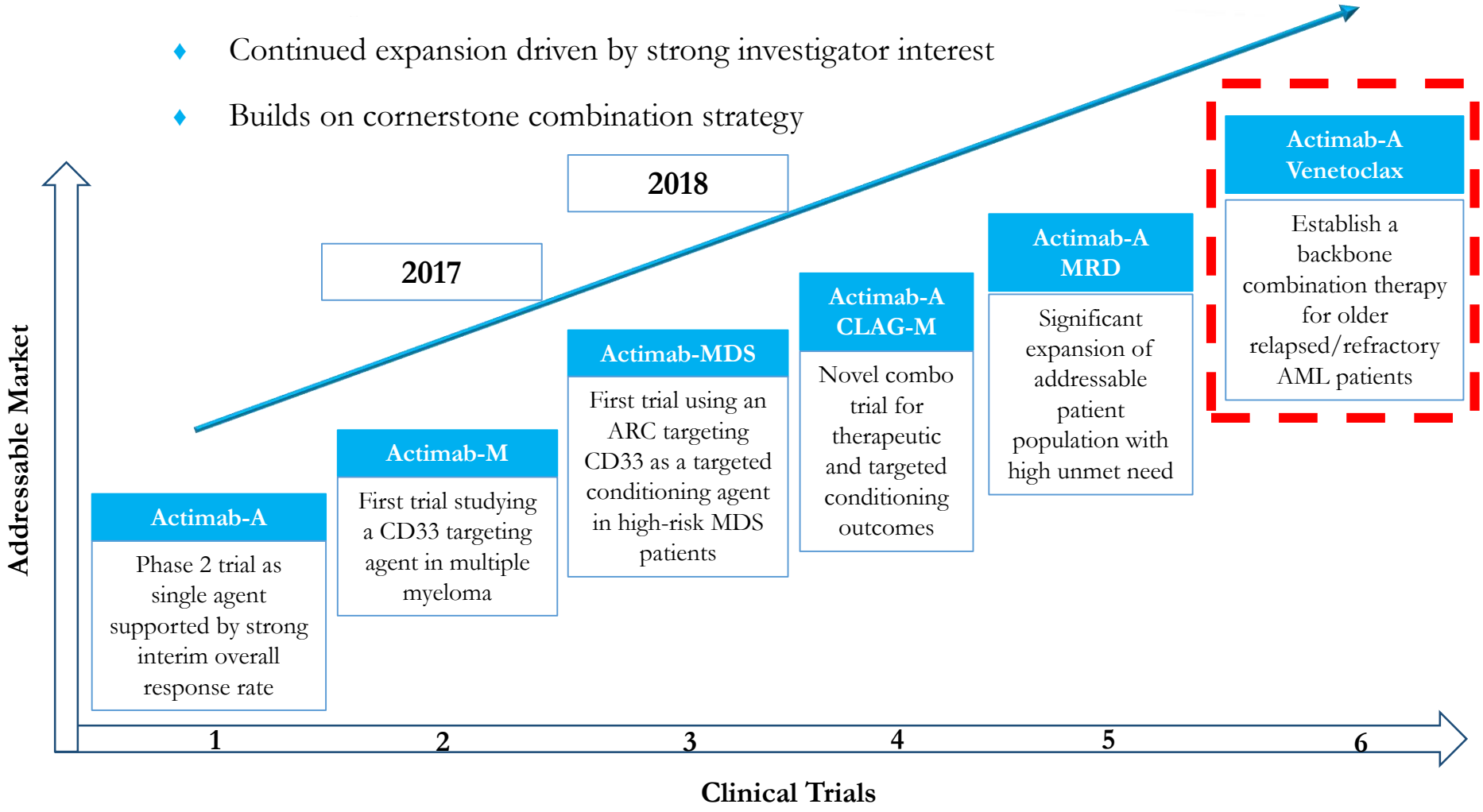
CD33 Program Building Strategically to Maximize Value



Continued Momentum to Advance Actimab CD33 Footprint

Latest initiative leverages targeted radiation via a potentially highly synergistic combination for patients with high unmet needs

- Continued expansion driven by strong investigator interest
- Builds on cornerstone combination strategy



II. CD33 Program Update

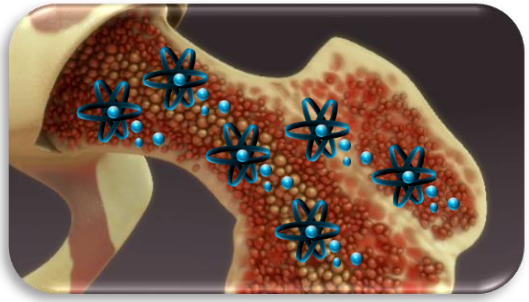
Actimab CD33 Program – Key Learnings

Significant clinical experience informs novel CD33 program strategy with several first in class trials and indications that build on strengths of ARC modality

CD33 Program Clinical Experience	4 Clinical Trials	100+ Patients	Therapeutic Effects at Multiple Doses

- ARC mechanism of action implies broad potential
- Single infusion, outpatient administration
- Strong myelosuppressive ability
- Minimal extramedullary toxicities

Targeted Conditioning



Therapeutic

AWE Technology Powers CD33 Program

Mechanistic benefits of Antibody Radiation Conjugate enables a broader development approach with multiple trials in targeted conditioning and therapeutic indications

ARC

Actinium-225

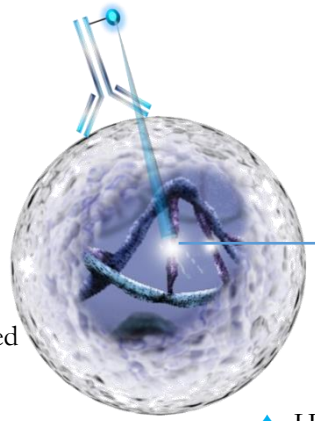
Range: .06 mm[1]

Energy: 6 MeV[1]

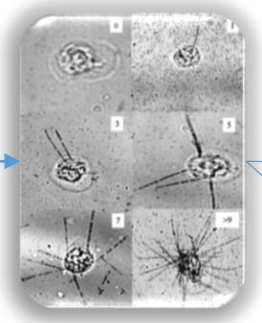
+

Lintuzumab

Extensively studied CD33 mAb

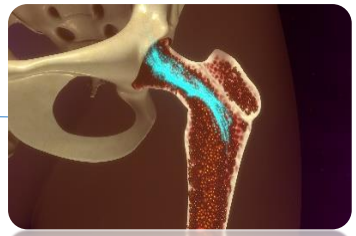


Alpha Particles



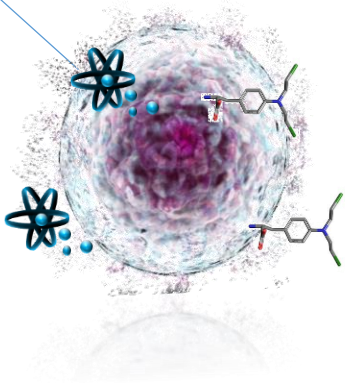
- High linear energy transfer[2] = potency
- Short path length = safety
- No known resistance mechanism

High Dose - Targeted Conditioning



- Actimab-MDS
- Actimab-A + CLAG-M

Low Dose – Therapeutics/Combinations



- Actimab-A + Venetoclax
- Actimab-A MRD
- Actimab-A + CLAG-M
- Actimab-M

AWE Technology Program Highlights

- 75 issued and pending patents
- Antibody conjugates with DOTA chelators
- Actinium-225 labeling, manufacture in cyclotron
- Validating partnership with Astellas

astellas

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1) Sgouros et al. Radiobiology and Dosimetry of a-Particle Emitters for Targeted Radionuclide Therapy. The Journal of Nuclear Medicine 2010; 51:311–328
2) Neti et al. Log Normal Distribution of Cellular Updake of Radioactivity. Implications for Biologic Responses to Radiopharmaceuticals. J Nucl Med. 2006; 47:1049-1058. Autoradiographs of cells within a population exposed to media containing 67 kBq/mL of 210Po-citrate

Clinical Evidence Supporting Actimab-MDS Trial

Clinical data demonstrating combination of broad myelosuppression coupled with low extramedullary toxicity supports development as a safer targeted conditioning agent prior to a bone marrow transplant

	Dose	Patients with prior MDS	Myelosuppression	Extramedullary Toxicities
Phase 2 Trial[1] Ac-225 - Lintuzumab	**4.0 µCi/kg[2]**	**5 (38%)**	**100% of patients had Grade 4 laboratory values**	**Minimal Grade 3-5 events**

- Many patients treated with Ac-225-Lintuzumab had previously progressed from MDS to AML
- Ac-225-Lintuzumab generated responses in these difficult to treat, heavily pre-treated patients
- Generally well tolerated
- Myelosuppression an issue

Minimal extramedullary toxicities

No occurrence of Veno-occlusive disease of any grade

Related/Possibly Related Non-Hematologic Adverse Events Grade 3/4/5 (N=13)	
Epistaxis	1
Fatigue	1
Febrile Neutropenia	1
Pneumonia	2
Pneumonia fungal	2
Sepsis	1
Septic Shock	2

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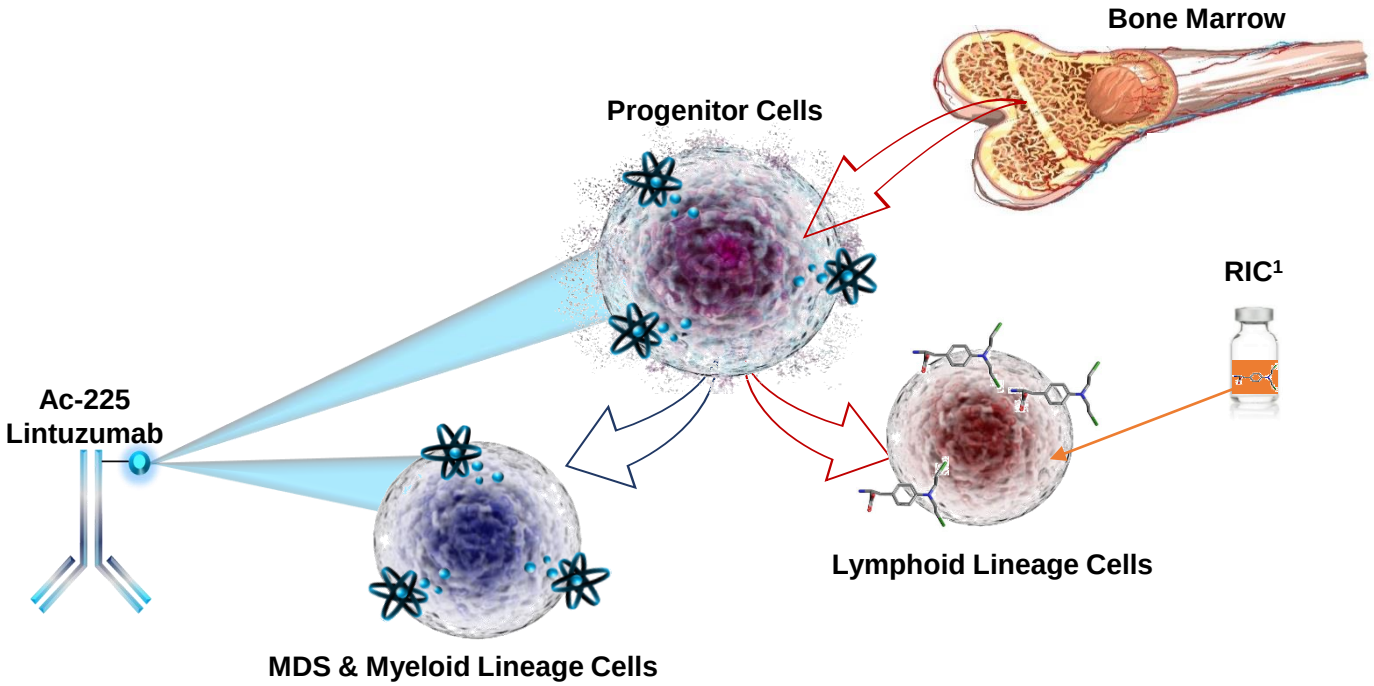
1) Finn et al. Phase 2 Study of Ac-225-Lintuzumab in Older Patients with Previously Untreated AML Unfit for Intensive Chemotherapy. ASH 2017 Poster
2) 2.0 µCi/kg/fraction of Ac-225-Lintuzumab on days 1 and 8

Value Proposition of the Actimab-MDS Trial

Actimab-MDS trial combines Ac-225-Lintuzumab with Reduced Intensity Conditioning to achieve safer, more effective myeloablation with potentially superior outcomes

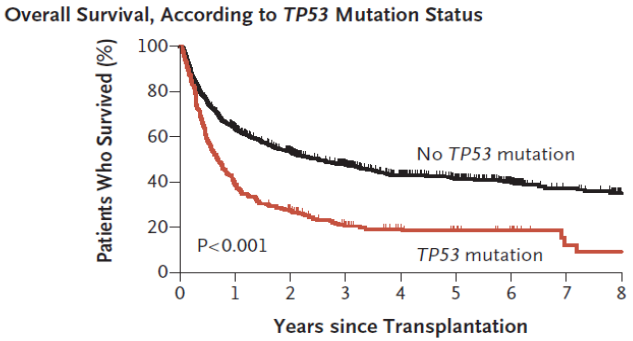
- Dr. Roboz (Cornell) conceptualized Actimab-MDS trial based on clinical experience with Actimab-A
  - Ac-225-Lintuzumab to eliminate MDS and myeloid lineage cells
  - Reduced Intensity Conditioning or RIC to deplete lymphoid lineage cells



1) Reduced Intensity Conditioning comprised of Melphalan and Fludarabine

Actimab-MDS Development Pathway Proposed to FDA

Targeted Conditioning PoC Trial for High-Risk MDS Patients with TP53 Mutation



Proposed Development Pathway:

- Phase 2, Open-label, 60-80 Patients
- Pivotal trial to follow Phase 2

Proposed Patient Population:

- Diagnosis of MDS, prior treatment with HMAs is allowed
- TP53 mutated
- Age 18 and above

Gail J. Roboz, M.D

Principal Investigator

Weill Cornell Medicine

NewYork-Presbyterian

Phase 2 MDS Trial Consortium

DANA-FARBER CANCER INSTITUTE

Cleveland Clinic

JOHNS HOPKINS MEDICINE

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center

MOFFITT CANCER CENTER

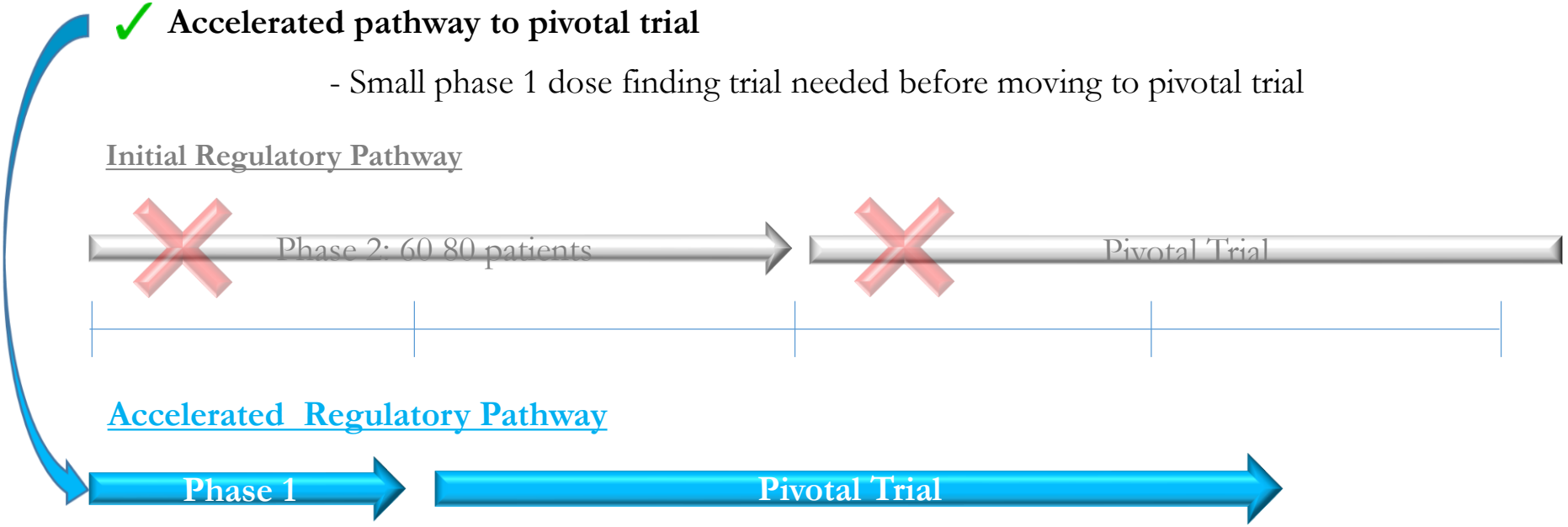
1) Lindsley, Saveer, Mar, et al, Prognostic Mutations in Myelodysplastic Syndrome after Stem-Cell Transplantation, The New England Journal of Medicine 376:536-47, 2017

Actimab-MDS Regulatory and Clinical Update

Positive FDA interactions have yielded an accelerated pathway to a pivotal trial in a larger addressable patient population

✓ **Accelerated pathway to pivotal trial**

- Small phase 1 dose finding trial needed before moving to pivotal trial



✓ **Broader patient population**

- All patients with complex cytogenetics, not limited to TP53+
- Up to 50% of MDS patients have complex cytogenetics vs. 20% with TP53 mutation[1]

1) Detlef Haase. Cytogenetic features in myelodysplastic syndromes. Ann Hematol. 2008 Jul; 87(7) 515-526

Actimab-A Clinical Update

Full Phase 2 data to be presented at ASH 2018 Annual Meeting

- Difficult to treat patient population with poor prognoses
  - Age 60+ who are unfit for intensive chemotherapy
  - Median age of 75 years old
  - Initial data presented at ASH 2017, 6 (67%) patients had prior hematologic disease - 5 (MDS) & 1 (tCML)

Actimab-A Phase 2 Trial

✓ Trial enrollment complete, strong evidence to support next stage of development

✓ As a result of the trial there is significant investigator interest to study Actimab-A in a wide range of patient populations and in combination

✓ Based on solid Phase 2 results and strong clinical experience with Actimab-A, a exciting development pathway has been established with KOL's

1) Detlef Haase. Cytogenetic features in myelodysplastic syndromes. Ann Hematol. 2008 Jul; 87(7) 515-526

III. Actimab-A in Combination with Venetoclax

Dr. Gary Schiller

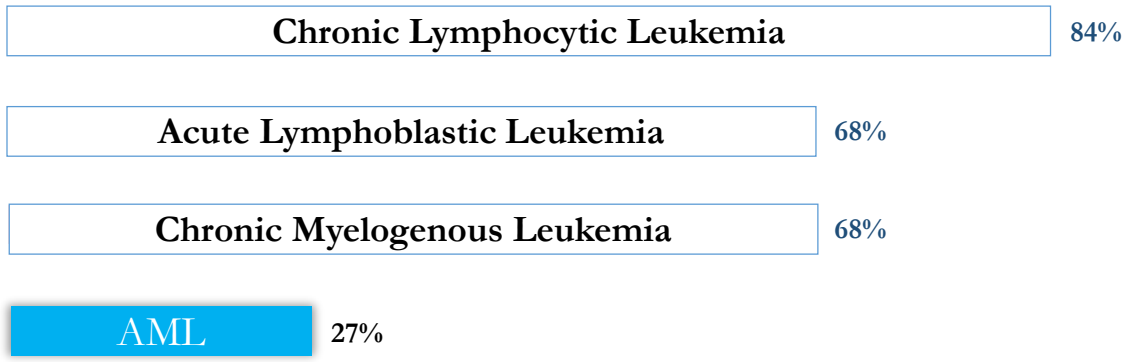
UCLA Health
David Geffen School of Medicine

- Professor, Hematology – Oncology at UCLA
- Director, Hematologic Malignancy/Stem Cell Transplant Program
- Over 20 years of clinical experience
- Over 85 peer-reviewed publications
- Research efforts focused on clinical trials of new drugs, therapies and bone marrow transplant for patients with leukemia, lymphoma and multiple myeloma
- MD Keck School of Medicine of USC
- Internship and Residency in Internal Medicine at UCLA School of Medicine
- Fellowship in Hematology-Oncology at UCLA School of Medicine

Dismal Prognosis for Older AML Patients

2018 Est. New Cases (US)	Average Age of AML Patient[2]	% of Older Patients Treated with Intensive Therapy[4]	% of Older Patients Achieving Durable Complete Remission[2]	Relapse Rate[3]	Median Survival[3]
~19,000	68	<30%	<10%	>90%	5-10 months

5-year OS shows need for better AML treatment



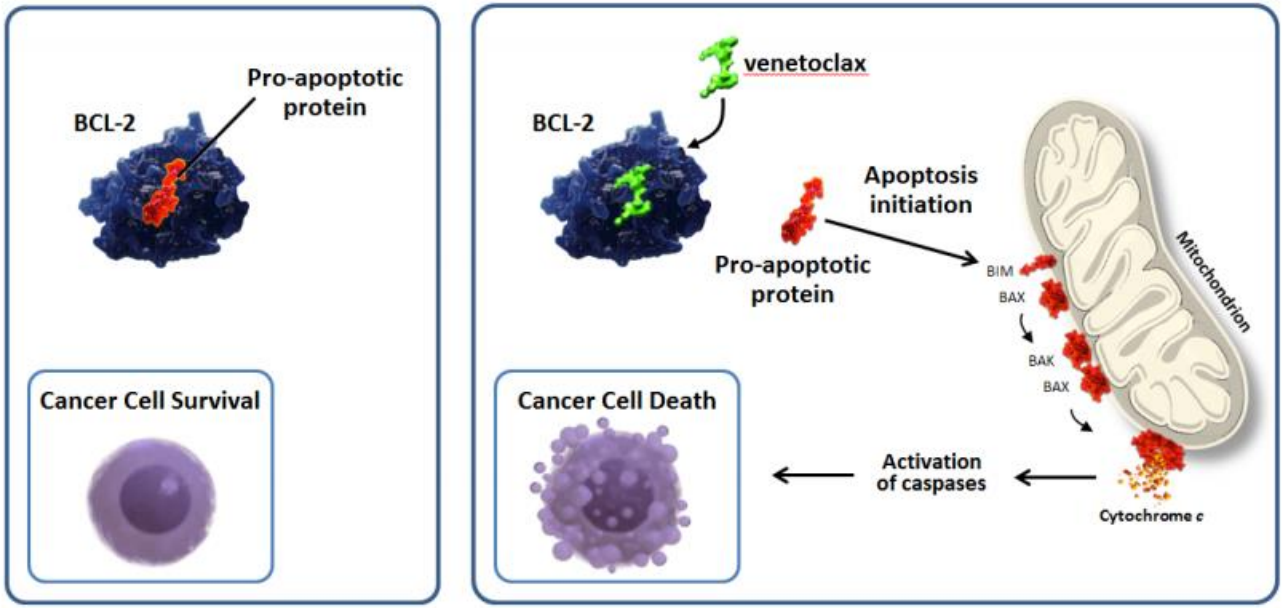
AML has among the lowest 5-year survival rate of blood cancers

Most AML patients will relapse

1) WHO, SEER AML Factsheet
2) Medeiros, Big data analysis of treatment patterns and outcomes among elderly acute myeloid leukemia patients in the United States. Ann Hematol (2015)
3) Median survival in older patients unfit for intensive therapy. Döhner H, et al. Acute myeloid leukemia. N Engl J Med. 2015;373(12):1136-1152.
4) Texas Oncology (2018). Acute Myeloid Leukemia Consolidation.

Introduction to Venetoclax, BCL-2 Inhibitor

- B-Cell Lymphoma 2 (BCL-2) is one of several proteins encoded by the BCL2 gene family that regulates apoptosis or programed cell death
- BCL-2 is overexpressed in certain cancers and enables cancer cells to evade apoptosis
- Venetoclax binds to BCL-2 family proteins and frees apoptotic proteins to initiate programmed death of cancer cells
- Venetoclax does not bind to MCL-1, a member of the BCL-2 family



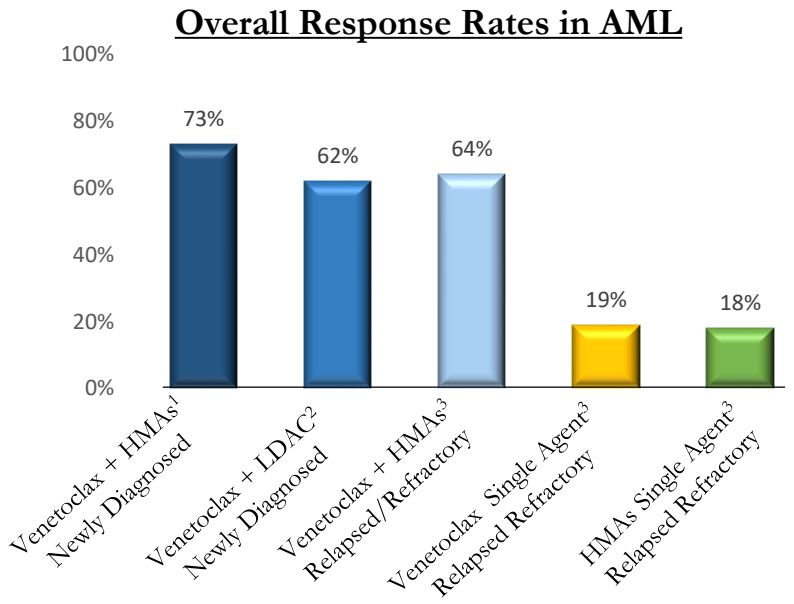
BCL-2 overexpression allows cancer cells to evade apoptosis by sequestering pro-apoptotic proteins.

Venetoclax binds selectively to BCL-2, freeing pro-apoptotic proteins that initiate programmed cell death (apoptosis). [1]

1) Roche https://www.roche.com/dam/jcr:cd8b9047-a812-4bc5-9473-ed74fccc46bb/en/ASH_2016_Reema_Mewar_FINAL.pdf

Venetoclax Development in AML

- Approved in chronic lymphocytic leukemia (CLL) or small lymphocytic lymphoma (SLL) 17p deletion
- Being evaluated in patients with NHL, MM, AML and other diseases
- Modest single agent activity in AML – synergistic with HMA's and LDAC
- sNDA filed for patient with AML based on two studies highlighted below



Significant unmet needs remain in AML despite new therapeutic agents as treatment is not expected to be curative

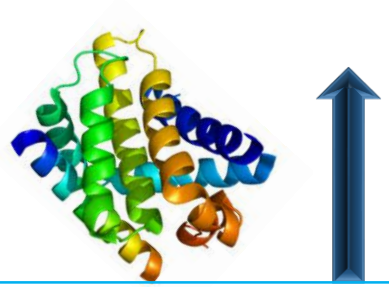
1) Dinardo CD, et al. Durable response with venetoclax in combination with decitabine or azacitadine in elderly patients with acute myeloid leukemia (AML). Presented at: ASCO Annual Meeting; 2018 USA. Abstract #701
2) Wei AH, et al. Updated safety and efficacy results of phase 1/2 study of venetoclax plus low-dose cytarabine in treatment-naïve acute myeloid leukemia patients aged ≥65 years and unfit for standard induction therapy. Madrid, Spain. Abstract #S473.
3) Aldoss et al. Efficacy of the combination of venetoclax and hypomethylating agents in relapsed/refractory acute myeloid leukemia. Haematologica 2018.1888094.

Unmet Needs Remain in AML with Venetoclax

Need a Viable Solution for Patients that Relapse, Stop Responding or Discontinue use of Venetoclax

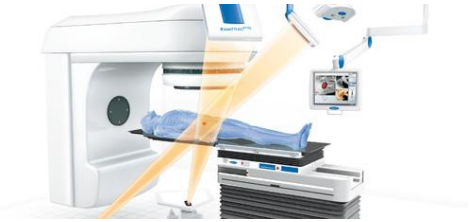
- 6-month Leukemia Free Survival of 52% in relapsed or refractory AML patients (n=33)[1]
- 54% of patients had Serious Adverse Events (SAE's)
- 13 of 21 responders stopped Venetoclax treatment most due to disease progression, BMT or sepsis
- Patients likely to be heavily pretreated with chemotherapy but have not received radiation for disease
- Despite 50% of patients achieving MRD$^-$ status, 1-year Overall Survival is 53%

MCL1 becomes upregulated in R/R AML patients[2]



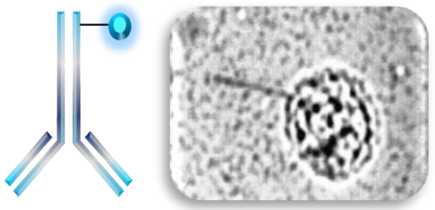
Promotes drug and apoptosis resistance

Radiation can reduce MCL1 levels[2]



External beam radiation not feasible in AML – diffuse disease

Actimab-A can deliver targeted radiation for AML

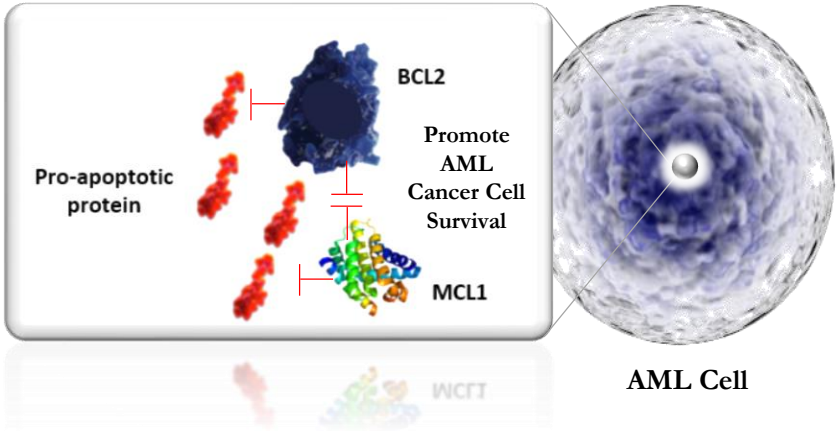


Strong potential synergy to improve outcomes

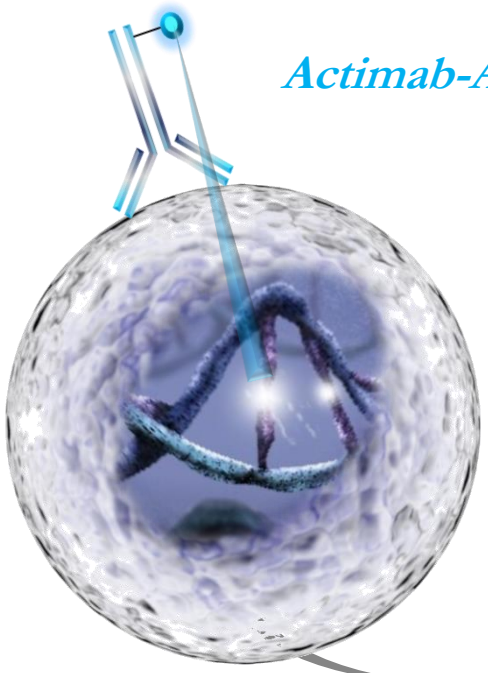
1) Aldoss et al. Efficacy of the combination of venetoclax and hypomethylating agents in relapsed/refractory acute myeloid leukemia. Haematologica 2018.1888094
2) Li et al.. MicroRNA-30 inhibits antiapoptotic factor Mcl-1 in mouse and human hematopoietic cells after radiation exposure. Apoptosis (2016) 21:708–720
3) Autoradiographs of cells within a population exposed to media containing 67 kBq/mL of 210Po-citrate

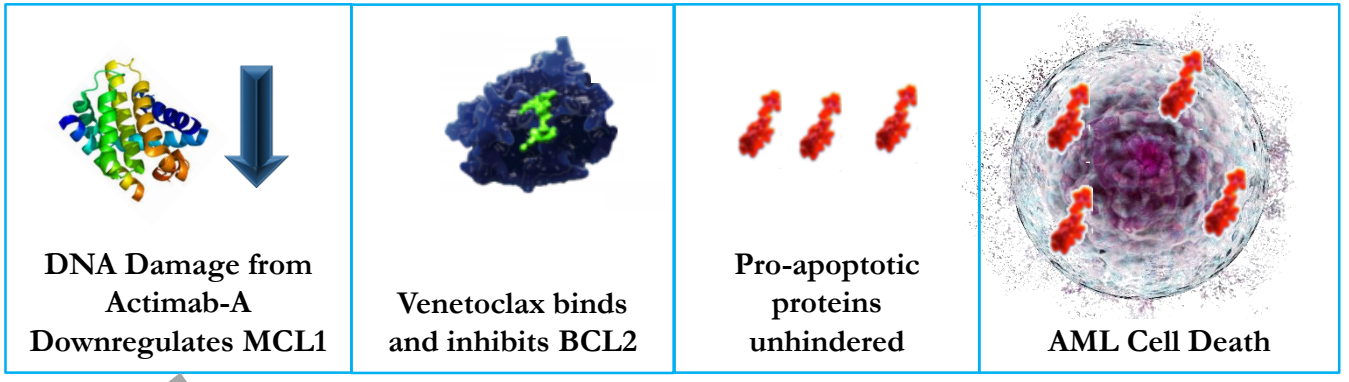
Actimab-A's Potential Synergy Via MCL-1 Downregulation

- MCL1 is up-regulated in refractory AML cells[1]
- MCL1 and BCL2 prevent apoptotic proteins from signaling cells to die
- MCL1 is an anti-apoptotic protein that is a mediator for resistance to Venetoclax[2]
- Venetoclax does not bind to MCL-1



Actimab-A will downregulate MCL1 making AML cells more susceptible to apoptosis via Venetoclax and other agents

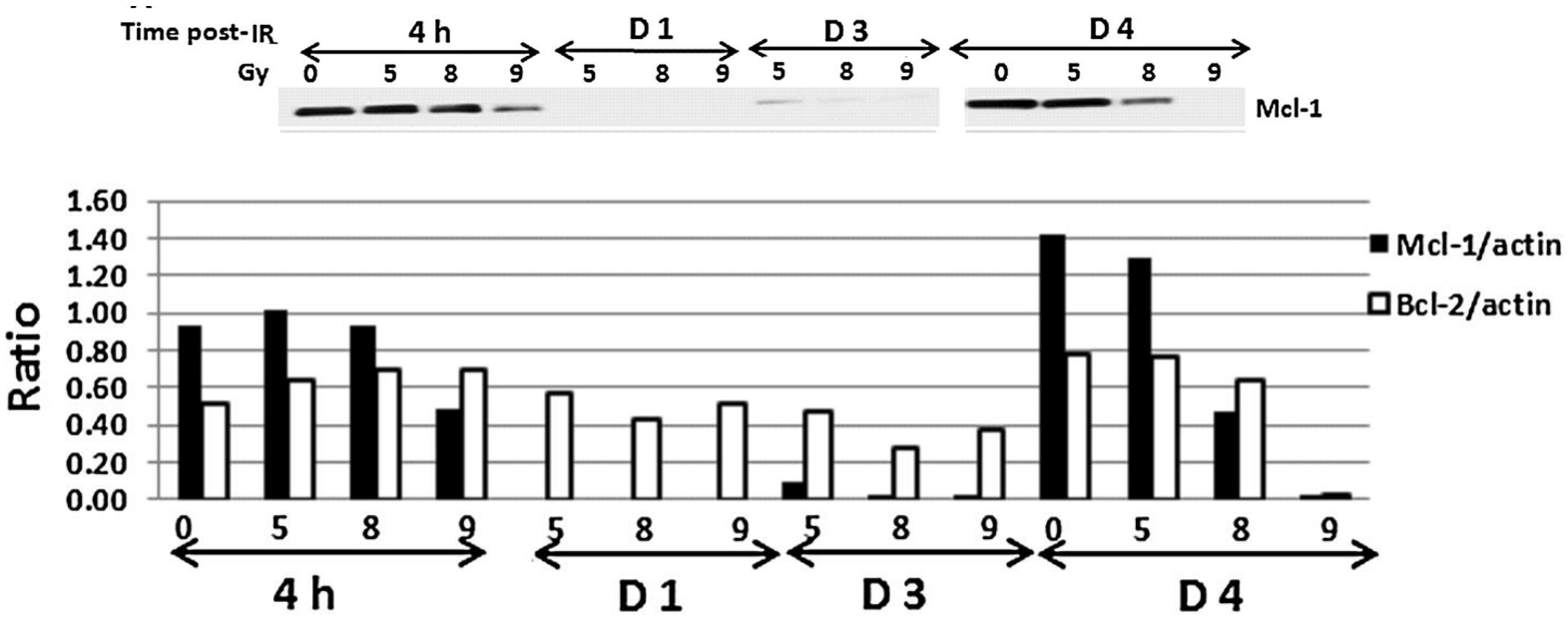




1) Li et al.. MicroRNA-30 inhibits antiapoptotic factor Mcl-1 in mouse and human hematopoietic cells after radiation exposure. Apoptosis (2016) 21:708–720
2) Tahir et al. Potential mechanisms of resistance to Venetoclax and strategies to circumvent it. BMC Cancer (2017) 17-399

Radiation is Effective at Depleting MCL-1

- MCL-1 was highly suppressed by external radiation in human hematopoietic progenitor cells[1]
- External radiation is not feasible in a diffuse disease such as AML
- Actimab-A has the potential to reduce MCL-1 through targeted internalized radiation
- MCL-1 depletion will result in increased sensitivity to Venetoclax and the release of pro-apoptotic proteins that can cause cell death

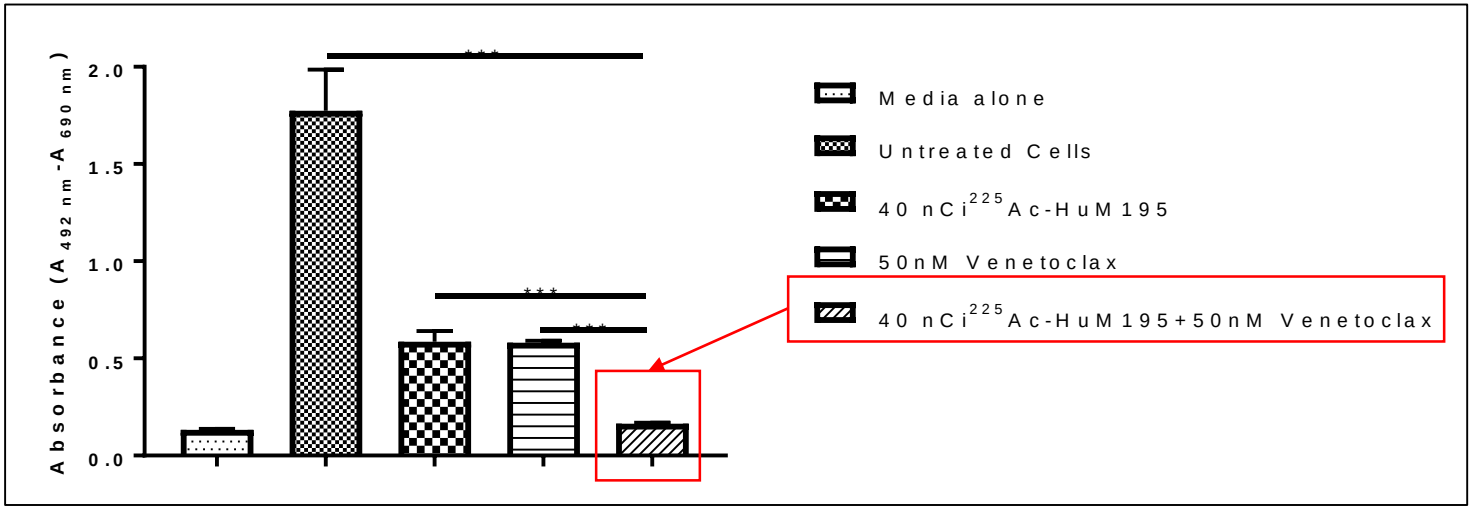


1) Li et al.. MicroRNA-30 inhibits antiapoptotic factor Mcl-1 in mouse and human hematopoietic cells after radiation exposure. Apoptosis (2016) 21:708–720

Demonstrated Synergy with Actimab-A and Venetoclax

- Single-agent cell killing ability comparable between Actimab-A and Venetoclax
- Actimab-A and Venetoclax resulted in greater cell killing than Venetoclax or Actimab-A alone
- Data implies potential for Actimab-A to be used in synergistic combination

Combination Efficacy of Actimab-A + Venetoclax on AML cell lines[1]



1) Company generated data on AML cell lines

Strong Rationale for Actimab-A + Venetoclax Trial

Continued unmet medical need and preclinical data supporting synergy provide strong rational for a Actimab-A plus Venetoclax clinical trial

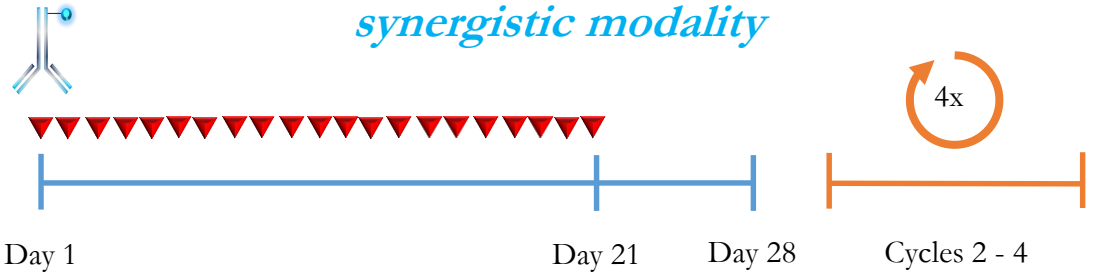
- Preclinical data shows clear synergy that may result in higher response rates and improved clinical outcomes of an Actimab-A, Venetoclax combination approach
- Actimab-A's minimal extramedullary toxicities could increase attractiveness of this combination

Study Objective

Improve response rates by combining Actimab-A and Venetoclax to introduce radiation mechanism for patients not exposed to a potentially synergistic modality

Study Endpoints

- **Determine MTD of Actimab-A**
- **Overall Response Rate (CR+CRi+CRp)**
- **Evaluate Overall Survival**



Venetoclax

Actimab-A

Trial Design

- 3+3 trial design, 9 – 18 patients
- Single infusion of Actimab-A in combo with Venetoclax
- Up to 4 cycles

IV. Actimab-A in Combination with Venetoclax

Dr. Hagop Kantarjian

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

- Professor and Chair of the Department of Leukemia at The University of Texas MD Anderson Cancer Center
- Samsung Distinguished Leukemia Chair in Cancer Medicine
- Research and collaborations have led to over 20 drugs approved in leukemia
- Author of over 1,800 peer-reviewed publications
- Has made significant contributions that improved prognosis and survival in patients in diseases such as CML, ALL, MDS and leukemia

Dr. Tapan Kadia

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

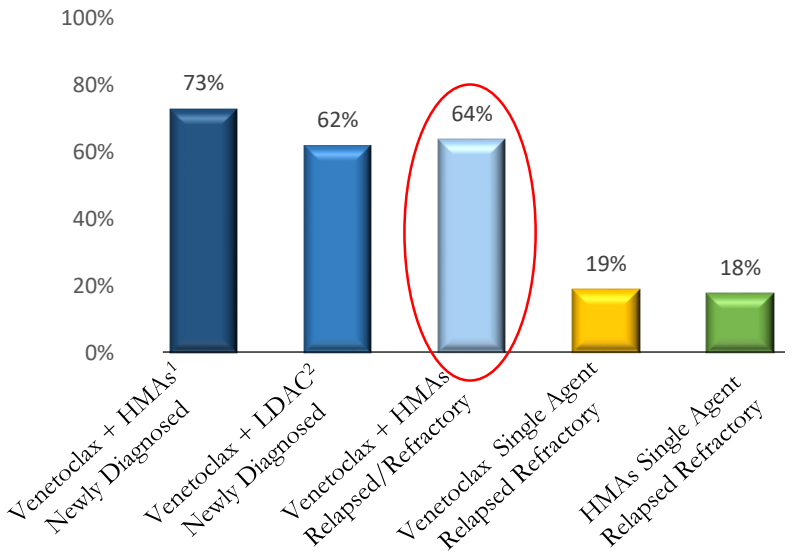
- Associate Professor in the Department of Leukemia at The University of Texas MD Anderson Cancer Center
- Focused on developmental therapeutics in acute leukemia for individualized frontline therapies, biologically rational targeted therapy and maintenance strategies
- Author of over 160 peer-reviewed publications and numerous abstracts
- M.D. from Robert Wood Johnson Medical School
- Chief Residency, Baylor College of Medicine
- Clinical Residency at Baylor College of Medicine
- Clinical Internship at Baylor College of Medicine

Clinical History of Venetoclax in R/R AML

Promising regimen but not a silver bullet due to limited duration of responses and lack of curative outcomes

- It is hypothesized that Venetoclax makes AML blast cells more sensitive to HMAs by reducing apoptosis escape mechanism
- Patient relapses and limited response duration support the need for further improvement

Overall Response Rates in AML



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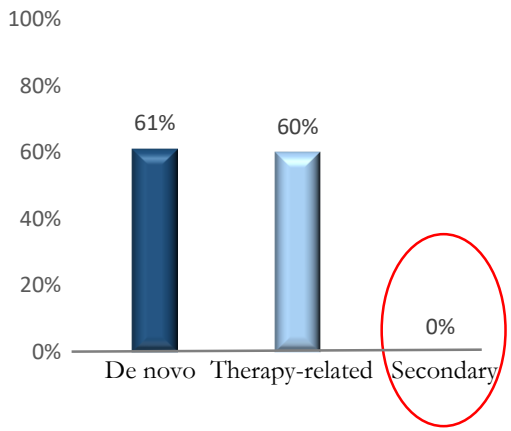
3) Aldoss et al. Efficacy of the combination of venetoclax and hypomethylating agents in relapsed/refractory acute myeloid leukemia. Haematologica 2018.1888094.

Limitations of Venetoclax & HMA's in R/R AML

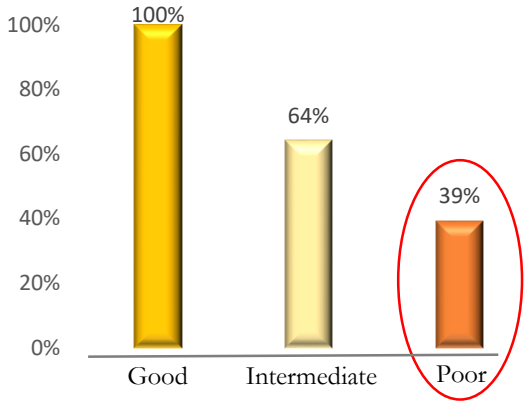
Actimab-A has the potential to improve outcomes for patient populations where Venetoclax + HMA's and other therapies are suboptimal

- Limited responses seen in certain patients based on[1]:
  - Type of AML
  - Risk factors
  - Cytogenetics

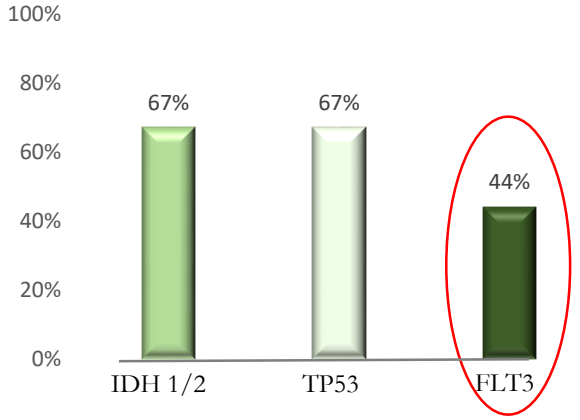
Response Rate by AML Type



Response Rate by AML Risk



Response Rate by Cytogenetics



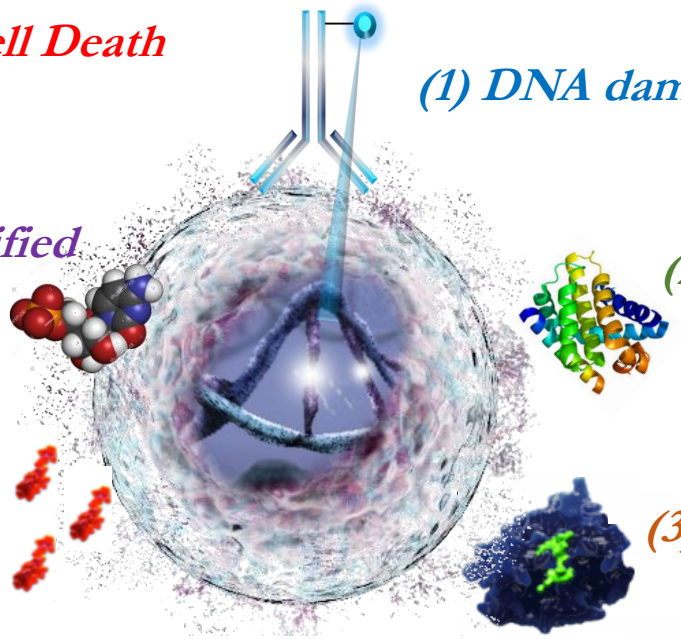
1) Aldoss et al. Efficacy of the combination of venetoclax and hypomethylating agents in relapsed/refractory acute myeloid leukemia. Haematologica 2018.1888094.

Actimab-A's Potential in R/R AML

- Actimab-A has produced responses in patients with high-risk disease
- Ac-225 not reliant on cytogenetics – applicable to patients with FLT3 mutations who have poorer responses
- Actimab-A produced responses in secondary AML patients who have been heavily pre-treated

Secondary AML	Antecedent Hematologic Disease	CRp	CRi	TF	Unk
Yes	MDS	♦			
Yes	MDS		♦		
Yes	MDS			X	
Yes	MDS			X	
Yes	Atyp CML			X	
No	None		♦		
Yes	MDS				X
No	None	♦			
Yes	tAML		♦		
No	None	♦			
Yes	CMML		♦		
Yes	CMML		♦		
No	None		♦		

Actimab-A's Potential Synergy Via MCL-1 Downregulation



(1) DNA damage from Actimab-A

CD33 directed Ac-225 payload causes double stranded DNA breaks via linear energy transfer

(2) MCL1 downregulation

Radiation damage to DNA by Ac-225 causes reduction in MCL1 protein. Reduction in MCL1 shown to reduce a cancer cell's ability to resist BCL-2 inhibitors like Venetoclax

(3) Venetoclax binds to BCL2

Less resistance to pro-apoptotic proteins released after Venetoclax binds to BCL2 increases programed cell death

(4) Pro-apoptotic proteins unhindered

Pro-apoptotic proteins are not resisted by MCL1 or BCL2 allowing them to trigger programmed cell death of AML cells

(5) HMAs cell killing amplified

AML cells become highly sensitive to HMAs as a result of resistance mechanisms being depleted or eliminated

(6) AML Cell Death

AML cell is killed as a result of triple attack of Actimab-A, Venetoclax and HMAs

1) Li et al.. MicroRNA-30 inhibits antiapoptotic factor Mcl-1 in mouse and human hematopoietic cells after radiation exposure. Apoptosis (2016) 21:708–720
2) Tahir et al. Potential mechanisms of resistance to Venetoclax and strategies to circumvent it. BMC Cancer (2017) 17-399

Actimab-A + Venetoclax + HMA Trial Highlights

Multiple synergistic mechanisms have the potential to improve outcomes for patients with limited viable treatment options

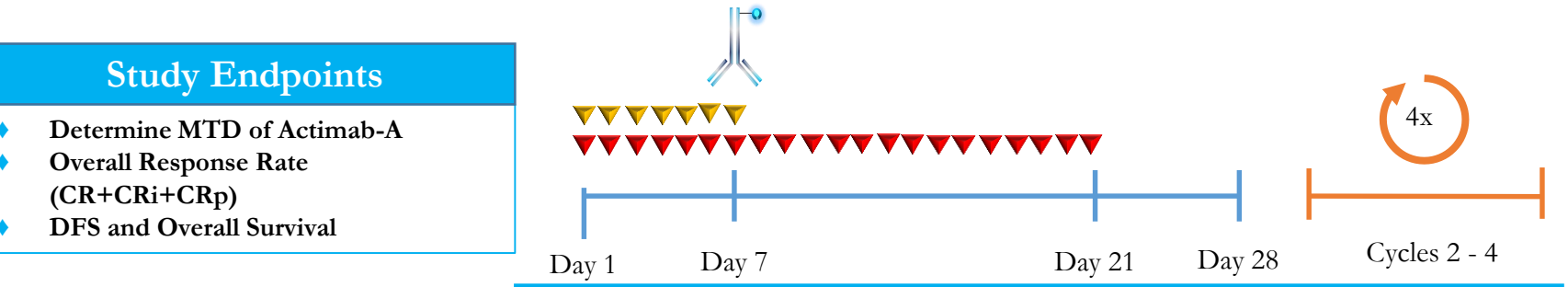
- Strong mechanistic rationale for adding targeted radiation via Actimab-A to Venetoclax + HMAs
- Minimal toxicity profile of Actimab-A makes combination feasible in difficult to treat patients

Study Objective

Determine if addition of Actimab-A can improve Disease Free Survival and Overall Survival

Study Endpoints

- **Determine MTD of Actimab-A**
- **Overall Response Rate (CR+CRi+CRp)**
- **DFS and Overall Survival**



Venetoclax HMA Actimab-A

Trial Design

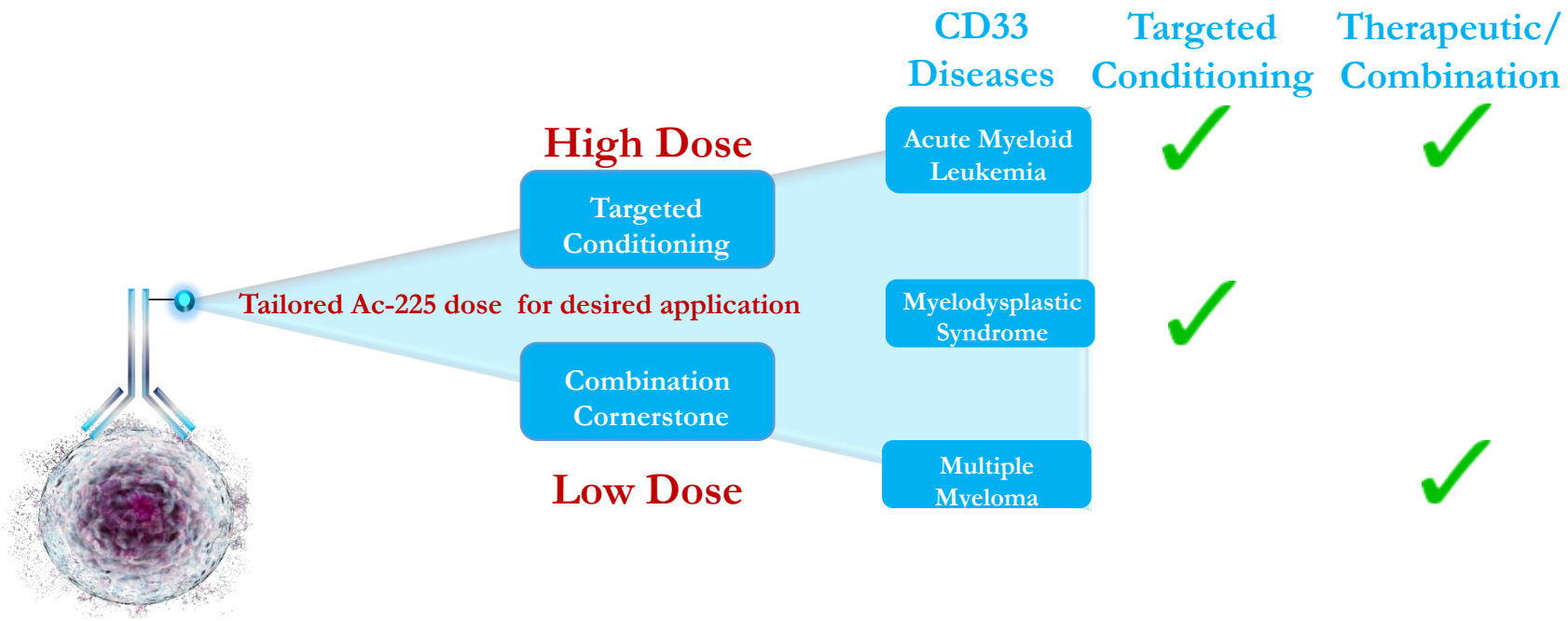
- 3+3 trial design, 9 – 18 patients
- Single infusion of Actimab-A in combo with Venetoclax
- Up to 4 cycles

V. CD33 Program Outlook and Summary

Actimab CD33 Program Development Strategy

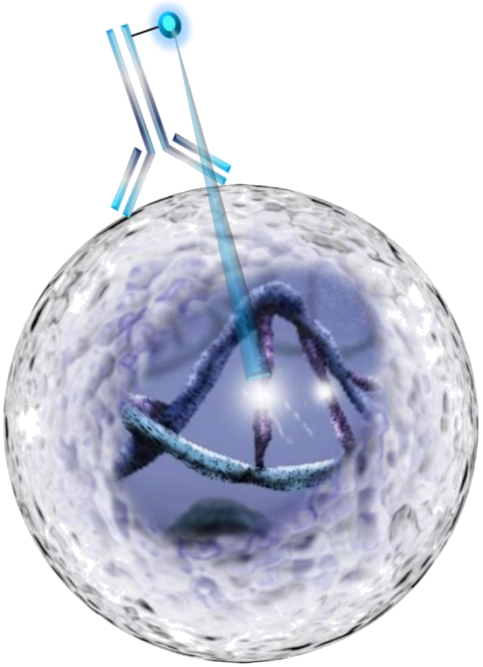
Effectively exploit broad potential of the ARC as a superior targeted conditioning agent and in therapeutic combinations in populations with unmet medical needs to create the industry leading CD33 program in terms of market potential

- ARC approach introduces unique modality and mechanism of action
- Combinations with Actimab-A open up numerous therapeutic possibilities in CD33 expressing diseases
- Potential in targeted conditioning across CD33 expressing diseases



Actimab CD33 Program Combination Approach

Leverage novel radiation mechanism to create synergies with other therapeutic modalities for patients with significant unmet needs



Potent Tumor Cell Death

Multi-faceted tumor cell killing

Mechanistic anti-tumor synergy

Disruptive therapy neoantigen release

Chemotherapy

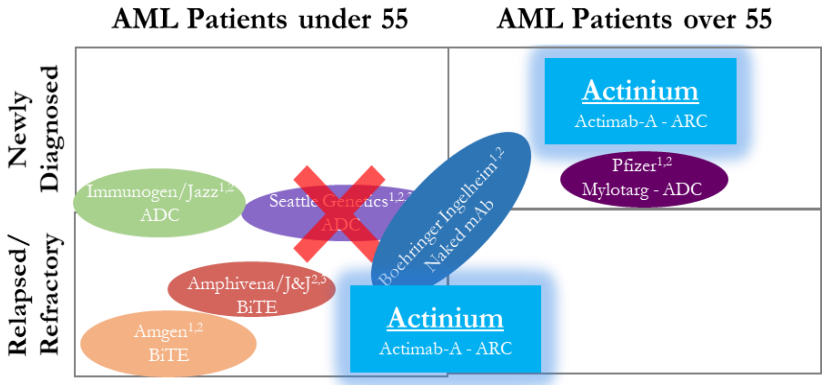
Targeted Therapies

Immunotherapy

Actimab-A Combo Trials

- CLAG-M
- Venetoclax
- TBA

Building the Industry Leading CD33 Program



- CD33 development still focused on AML
- ARC approach enable development beyond AML
- Only CD33 ARC in clinical development

Largest Addressable Patient Population in Aggregate Among CD33 Programs
Three Diseases. Therapeutic Combinations. Targeted Conditioning.

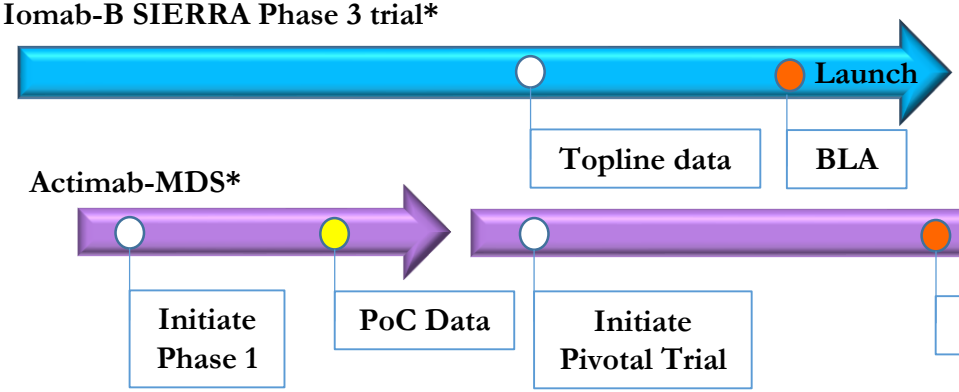
Disease	BMT	Therapeutics				Patient Population
	Targeted Conditioning	Newly Diagnosed	Consolidation	Relapsed/Refractory		Addressable US, EU Market
AML	Actimab-A CLAG-M	Actimab-A Ven*	Actimab-A MRD	Actimab-A CLAG-M	Actimab-A Ven	69,300
MDS	Actimab-MDS					37,500[1]
Multiple Myeloma				Actimab-M (penta refractory)		47,400[2]
						Total 154,200[3]

1) Includes complex and high-risk MDS patients
2) Based on 25% of Multiple Myeloma patients being CD33+
3) Company estimates
* Planned future study

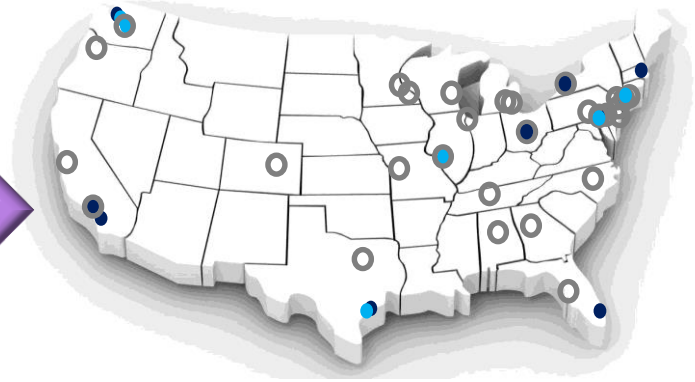
CD33 Developments Strengthen Company Outlook

Increasingly attractive and viable opportunity to develop a multi-product commercial opportunity in targeted conditioning in the 2020-2022 period

Iomab-B SIERRA Phase 3 trial*

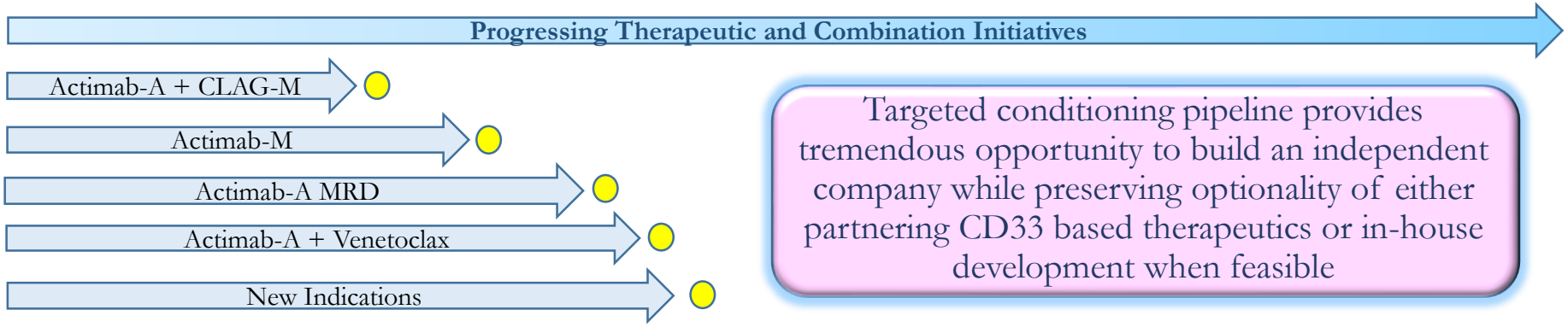


Actimab-MDS*



Iomab-ACT

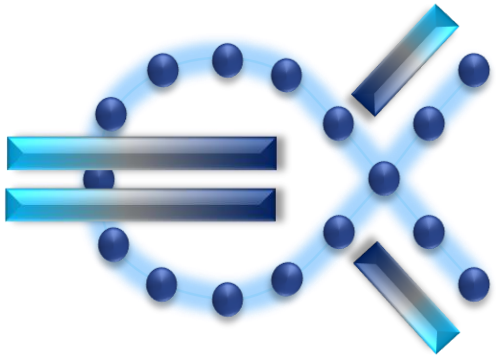
Targeted Conditioning Pipeline Enables CD33 Therapeutic Optionality



Targeted conditioning pipeline provides tremendous opportunity to build an independent company while preserving optionality of either partnering CD33 based therapeutics or in-house development when feasible

* Image is for illustrative purposes only and is not to scale

Thank-You



Actinium Pharmaceuticals, Inc.