
Forty Seven
Helping Patients Defeat Their Cancer
Webcast to Begin Shortly


Forty Seven
Helping Patients Defeat Their Cancer
Forty Seven ASH Investor Event
December 9, 2019

Forward Looking Statements

This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation, including statements regarding our future financial condition, results of operations, business strategy and plans, and objectives of management for future operations, as well as statements regarding industry trends, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as “anticipate,” “believe,” “continue,” “could,” “estimate,” “expect,” “intend,” “may,” “plan,” “potentially” “predict,” “should,” “will” or the negative of these terms or other similar expressions.

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Speakers at Today's Event

Chris Takimoto, M.D., Ph.D.
Chief Medical Officer, Forty Seven, Inc.

David Sallman, M.D.
Assistant Member, Malignant Hematology Department,
H. Lee Moffitt Cancer Center and Research Institute

Mark Chao, M.D., Ph.D.
Co-Founder and VP Clinical Development, Forty Seven, Inc.

Leslie Kean, M.D., Ph.D.
Director, Stem Cell Transplantation Program, Division of Hematology/Oncology,
Boston Children's Hospital

Jens-Peter Volkmer, M.D.
Co-Founder and VP Research & Early Development, Forty Seven, Inc.

Today's Agenda

Welcome and Program Overview	Chris Takimoto, M.D., Ph.D.
Review of Updated Phase 1b Results for Magrolimab in Combination With Azacitidine in MDS and AML Patients	David Sallman, M.D.
Treatment Landscape, Clinical and Registration Strategy in MDS	Mark Chao, M.D., Ph.D.
Q&A	
Overview of Stem Cell Transplantation and cKIT Program (FSI-174)	Leslie Kean, M.D., Ph.D.
FSI-174 Development Plan	Jens-Peter Volkmer, M.D.
Q&A	

Our Foundation

Forty Seven is built on a culture of scientific rigor and passion for helping people to live fuller, healthier lives. This is seen in our actions and every decision we make.

Highly Experienced Management Team and Advisors

Management Team

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Craig Gibbs, Ph.D., M.B.A. *Chief Business Officer*	Tobira Therapeutics; Gilead; Genentech
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Kyle Elrod *SVP of Corporate Planning & Operations*	virobay; life technologies; Celera; Axys
Mark Chao, M.D., Ph.D. *VP of Clinical Development*	Stanford School of Medicine
Jens-Peter Volkmer, M.D. *VP of Research & Early Development*	Stanford School of Medicine; Heinrich Heine Universität Düsseldorf
Mukul Agarwal, M.S., M.B.A. *VP of Corporate Development*	revance; Anacor Pharmaceuticals; Allergan; MedImmune; gsk GlaxoSmithKline
Aimee Murphy *VP of Clinical Operations*	Aduro Biotech; Cerus
Qinghai Zhao, Ph.D. *VP of Technical Development & Manufacturing*	AnaptysBio; NGMBio; Teva Pharmaceutical Industries Ltd.; Human Genome Sciences

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 **Padmanee Sharma, M.D., Ph.D.**	Professor, Department of Genitourinary Medical Oncology and Department of Immunology, Division of Cancer Medicine at the University of Texas MD Anderson Cancer Center, Scientific Director, Immunotherapy Platform and Co-Director of the Parker Institute for Cancer Immunotherapy at MD Anderson Cancer Center
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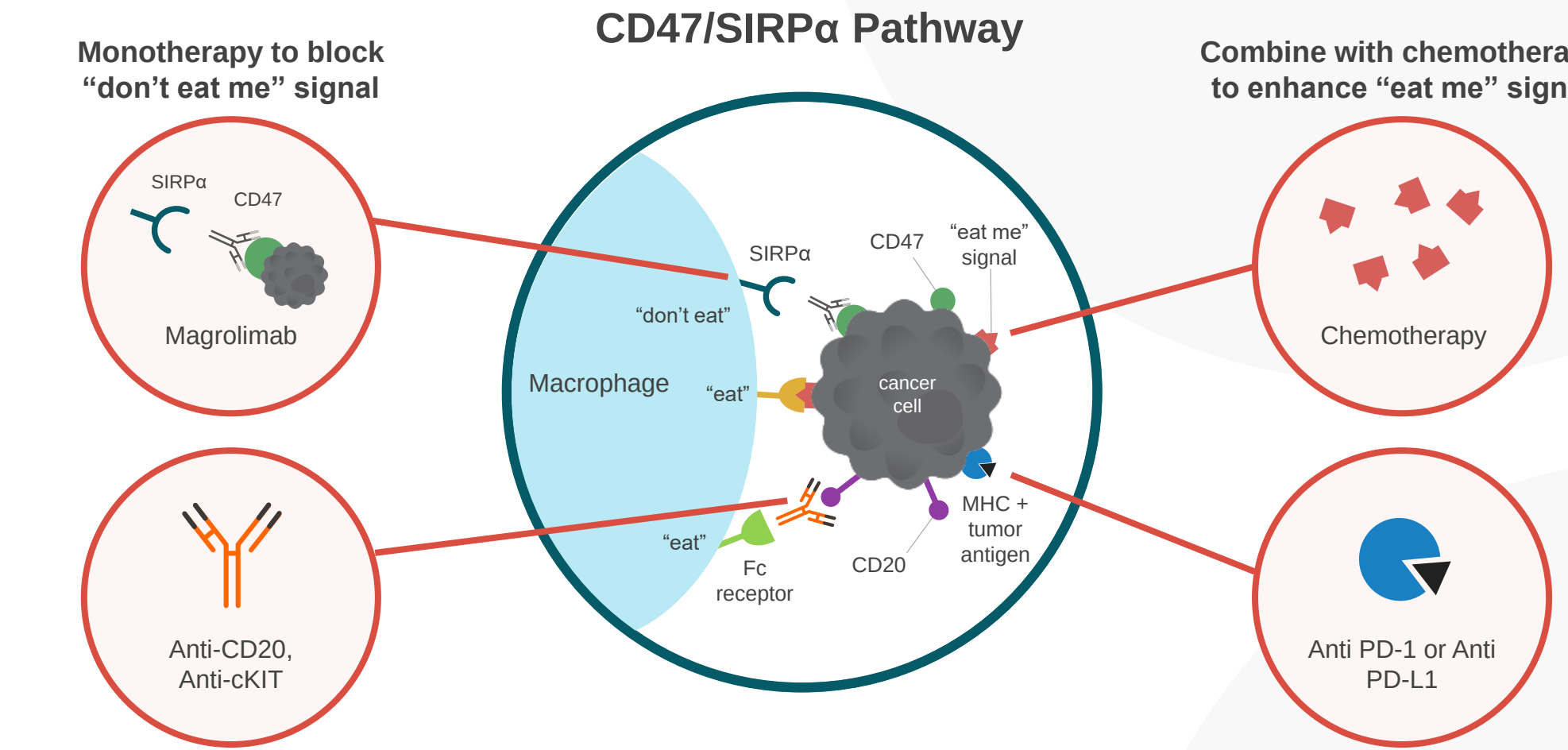
CD47/SIRPα Pathway Offers Multiple Opportunities to Engage Macrophages

Target cells overexpress CD47 to evade destruction by macrophages



Broad Pipeline Targeting CD47/SIRPα Pathway

MAGROLIMAB*

Discovery	Preclinical	Phase 1	Phase 2	Registrational Trial	Clinical Collaborators
Myelodysplastic Syndrome (MDS): *Magrolimab + Azacitidine*					CIRM; Leukemia & Lymphoma Society
Diffuse Large B-Cell Lymphoma (DLBCL): *Magrolimab + Rituximab*					Leukemia & Lymphoma Society
Acute Myeloid Leukemia (AML): *Magrolimab + Azacitidine*					CIRM
AML: *Magrolimab + Atezolizumab*					Roche Genentech
DLBCL: *Magrolimab + Rituximab + Atezolizumab*					Roche Genentech
DLBCL: *Magrolimab + Rituximab + Acalabrutinib*					Acerta Pharma; AstraZeneca
DLBCL: *Magrolimab + Rituximab + Gem/Ox***					
Bladder: *Magrolimab + Atezolizumab*					Roche Genentech
Colorectal: *Magrolimab + Cetuximab*					CIRM; Lilly
Ovarian: *Magrolimab + Avelumab*					Merck

ADDITIONAL PIPELINE PROGRAMS

Discovery	Preclinical	Phase 1	Phase 2	Registrational Trial	Clinical Collaborators
FSI-174: *Anti-cKIT Antibody for HSC Transplantation*					bluebirdbio
FSI-189: *Anti-SIRPα Antibody for Oncology/Non-Oncology*					

** Ono Pharmaceutical has rights to all magrolimab programs in Japan, Taiwan, South Korea and other ASEAN countries | **Expansion arm of ongoing NHL: magrolimab + rituximab trial*

Updated Phase 1b Results for Magrolimab in Combination with Azacitidine in MDS and AML Patients

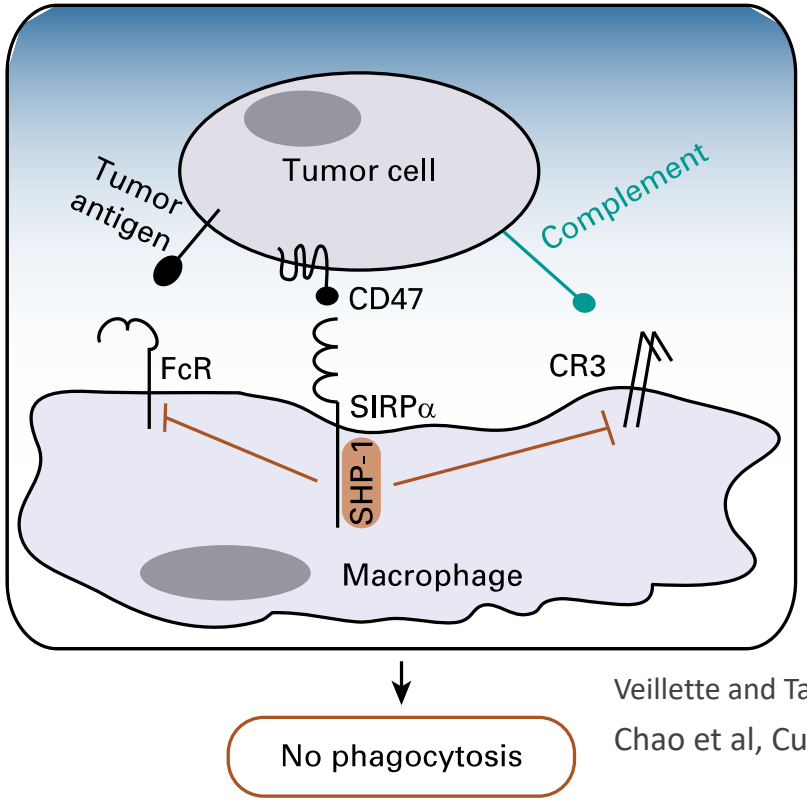
David Sallman, M.D

The First-in-Class Anti-CD47 Antibody Magrolimab in Combination with Azacitidine is Effective in MDS and AML Patients: Updated Ongoing 1b Results

David A Sallman[1], Adam Asch[2], Monzr Al-Malki[3], Daniel Lee[4], Guillermo Garcia-Manero[5], William Donnellan[6], Daniel Pollyea[7], Suman Kambhampati[8], Guido Marcucci[3], Rami Komrokji[1], Joanna Van Elk[9], Ming Lin[9], Jens-Peter Volkmer[9], Roy Maute[9], Chris Takimoto[9], Mark Chao[9], Paresh Vyas[10], Naval Daver[5]

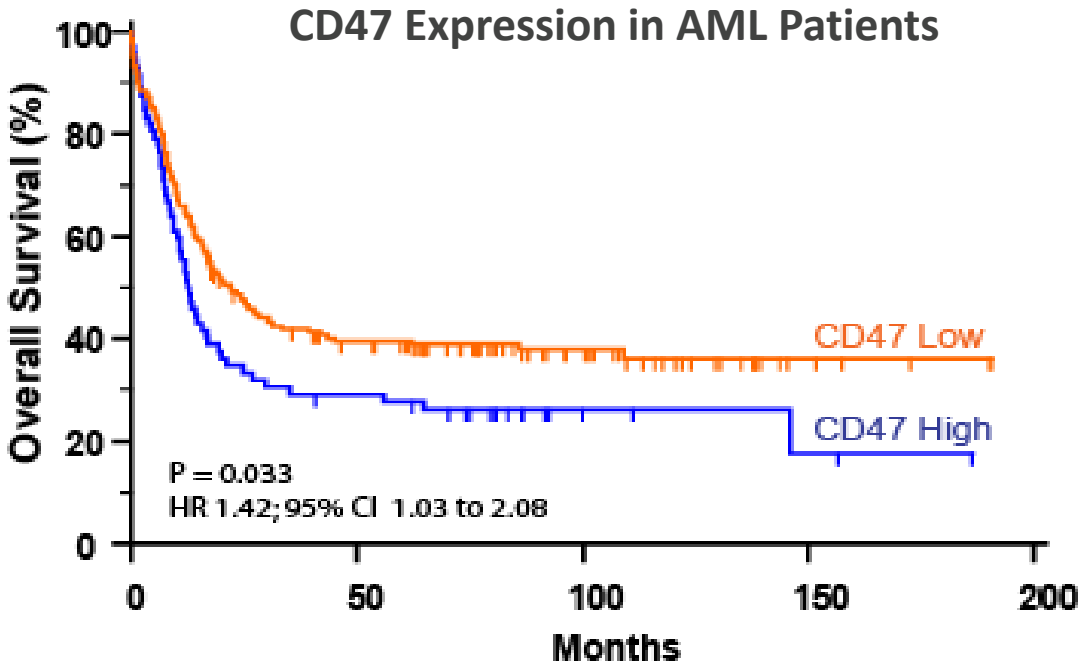
[1]Moffitt Cancer Center, Tampa, FL; [2]University of Oklahoma, Oklahoma City, OK; [3]City of Hope, Duarte, CA; [4]Columbia University, New York, NY; [5]MD Anderson Cancer Center, Houston, TX; [6]Sarah Cannon Research Institute, Nashville, TN; [7]University of Colorado, Denver, CO; [8]Healthcare Midwest, Kansas City, MO; [9]Forty Seven, Inc., Menlo Park, CA; [10]University of Oxford, Oxford, UK

CD47 is a Major Macrophage Immune Checkpoint and "Do Not Eat Me" Signal in Myeloid Malignancies including MDS and AML



Veillette and Tang, JCO 2019

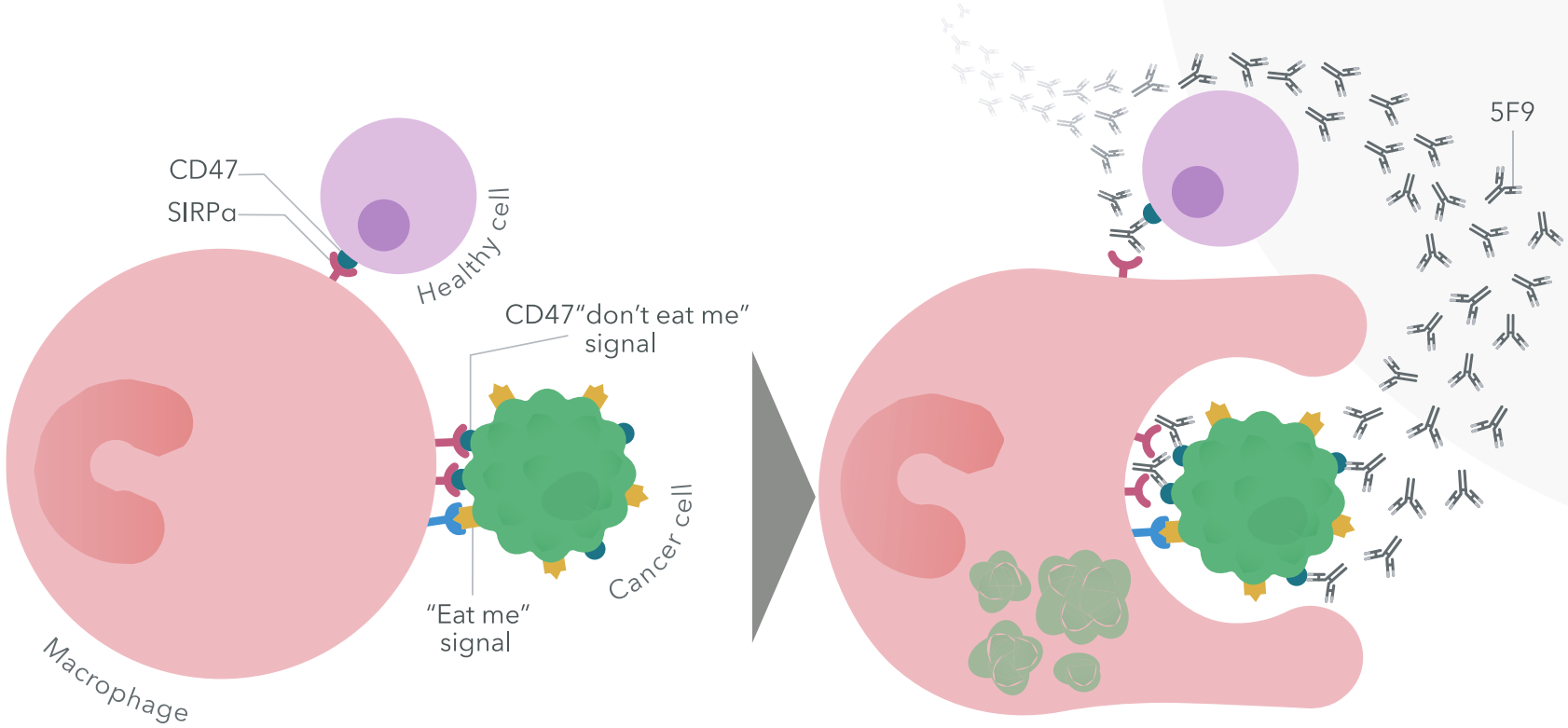
Chao et al, Current Opin Immunol 2012



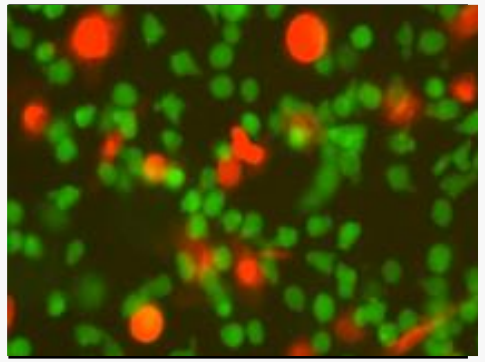
Majeti, Chao et al., Cell 2009

- CD47 is a "do not eat me" signal on cancers that enables macrophage immune evasion
- Increased CD47 expression predicts worse prognosis in AML patients

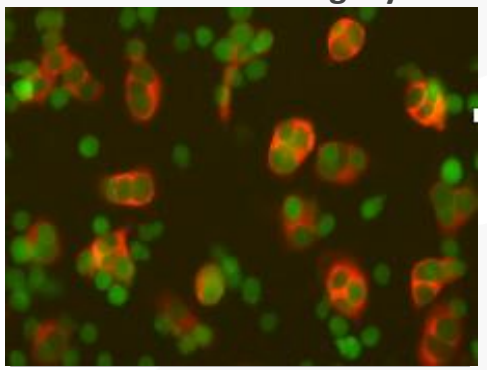
Magrolimab is a First-in-class Macrophage Immune Checkpoint Inhibitor Targeting CD47



Control mAb: No Phagocytosis



Anti-CD47 mAb: Phagocytosis



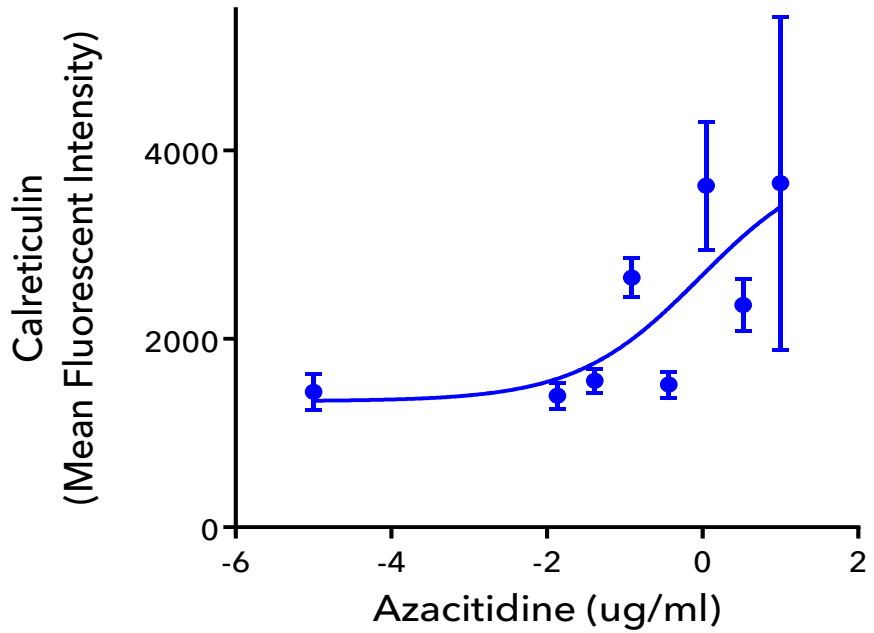
Macrophages Cancer cells

- Magrolimab is an IgG4 anti-CD47 monoclonal antibody being investigated in multiple cancers
- Magrolimab was well tolerated in a UK Phase 1 trial in r/r AML with no MTD reached (Vyas et al., EHA abs 2018)

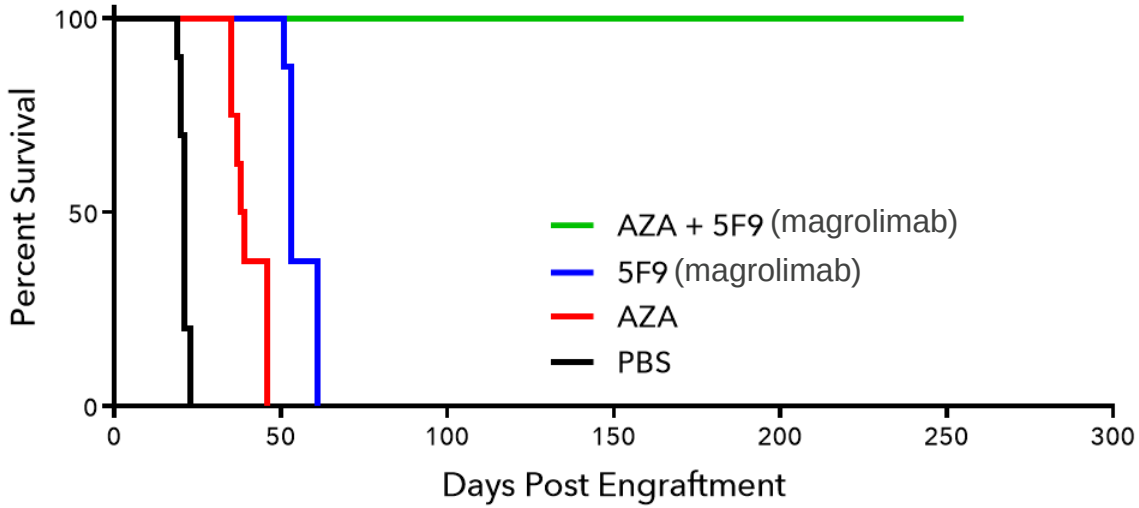
Magrolimab Synergizes with Azacitidine to Induce Remissions in AML Xenograft Models

- Azacitidine (AZA) induces pro-phagocytic “eat me” signals like calreticulin on cancer cells
- Increased eat me signals induced by azacitidine synergizes with CD47 blockade of the “don’t eat me” signal leading to enhanced phagocytosis

Calreticulin expression in AML



AML xenograft model



Feng et al., ASH abs 2018

5F9005 Study Design: Magrolimab in Combination with Azacitidine in MDS and AML

Untreated AML ineligible for induction chemotherapy or untreated MDS intermediate to very high risk by IPSS-R

Magrolimab + AZA Combo Safety Evaluation (N=6)

Magro: 1, 30 mg/kg* weekly
AZA: 75 mg/m^2 D1-7

Expansion

Magro: 1, 30 mg/kg* weekly
AZA: 75 mg/m^2 D1-7

*Dose ramp up from 1 to 30 mg/kg by week 2, then 30 mg/kg maintenance dosing

Primary objectives

1) Safety of magrolimab alone or with AZA
2) Efficacy of magrolimab + AZA in untreated AML/MDS

Secondary objectives

1) PK, PD and immunogenicity of 5F9
2) Additional measures of efficacy (DOR, PFS, OS)

Exploratory objectives

1) To assess CD47 receptor occupancy, markers of immune cell activity, and molecular profiling in AML/MDS

- A magrolimab priming dose (1 mg/kg) and dose ramp up was utilized to mitigate on target anemia
- Data from the Expansion Cohort is presented

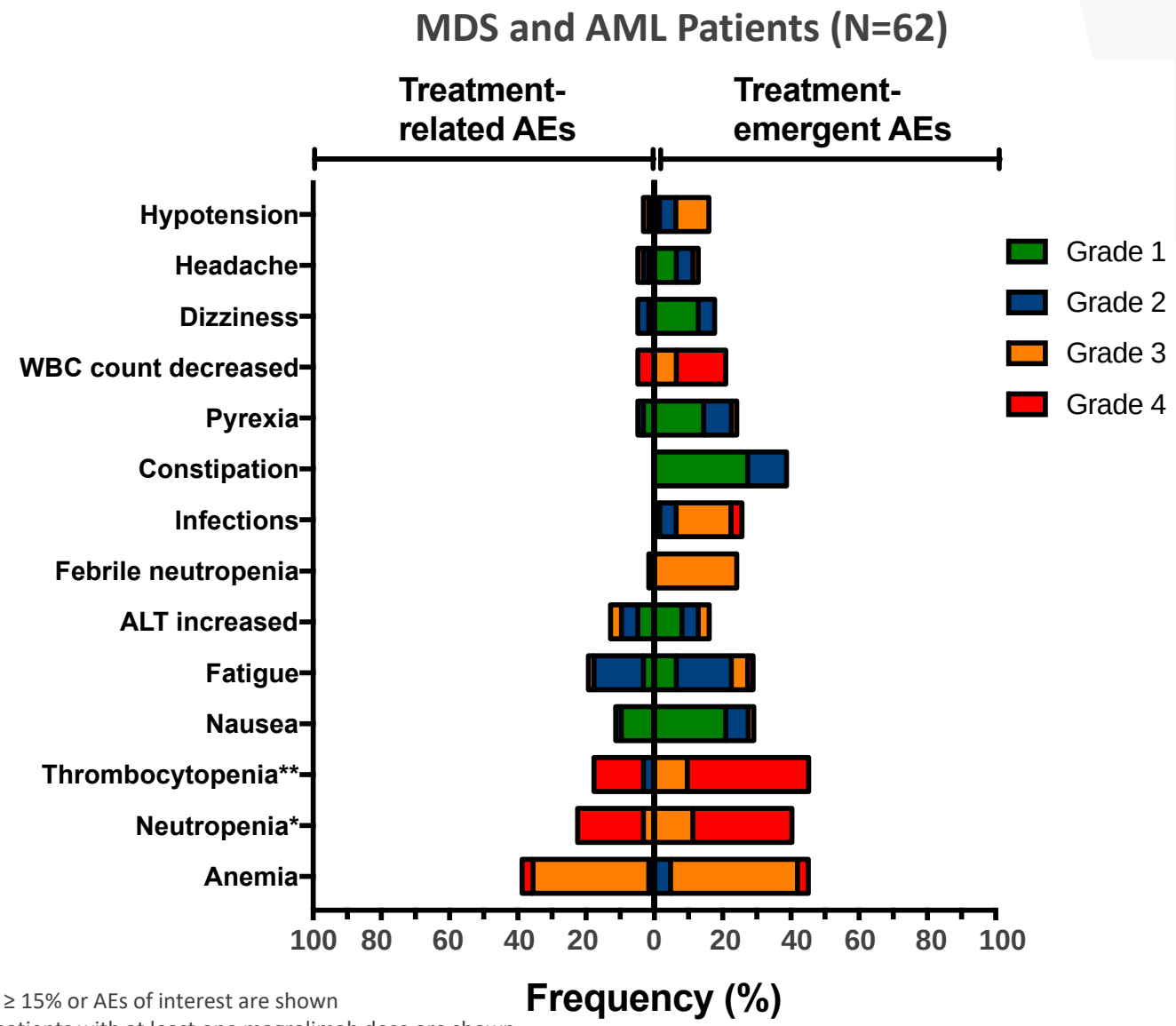
Patient Characteristics (N=62): Magrolimab + AZA in Untreated (1L) MDS/AML

Characteristic	1L MDS 5F9+AZA (N=35)	1L AML 5F9+AZA (N=27)
Median age (range)	70 (47 – 80)	74 (60 – 89)
ECOG Performance Status: 0 1 2	13 (37%) 21 (60%) 1 (3%)	9 (33%) 16 (59%) 2 (7%)
Cytogenetic Risk: Favorable Intermediate Poor Unknown/missing	0 10 (29%) 23 (66%) 2 (6%)	0 2 (7%) 18 (67%) 7 (26%)
WHO AML classification: MRC		19 (70%)
Recurrent abnormalities Therapy-related NOS	-	2 (7%) 1 (4%) 5 (19%)
WHO MDS classification: RS and single/multi-lineage dysplasia Multilineage dysplasia Excess blasts Unclassifiable/unknown/missing	 3 (9%) 6 (17%) 19 (54%) 7 (20%)	-
IPSS-R (MDS): Intermediate High Very High Unknown/missing	11 (31%) 18 (51%) 5 (14%) 1 (3%)	-
Therapy-related MDS	11 (31%)	
Unknown/missing	1 (3%)	-
Harboring a *TP53* mutation	4 (11%)	11 (41%)

- 66-67% of MDS and AML patients are poor cytogenetic risk
- 70% of AML patients have underlying myelodysplasia (MRC)
- 41% of AML patients are *TP53* mutant
- 31% of MDS patients are therapy-related
- The majority of MDS patients were high or very high risk by IPSS-R

IPSS-R: revised international prognostic scoring system
MRC: myelodysplasia-related changes
NOS: not otherwise specified
WHO: world health organization
"-" not applicable; All patients enrolled on study are shown

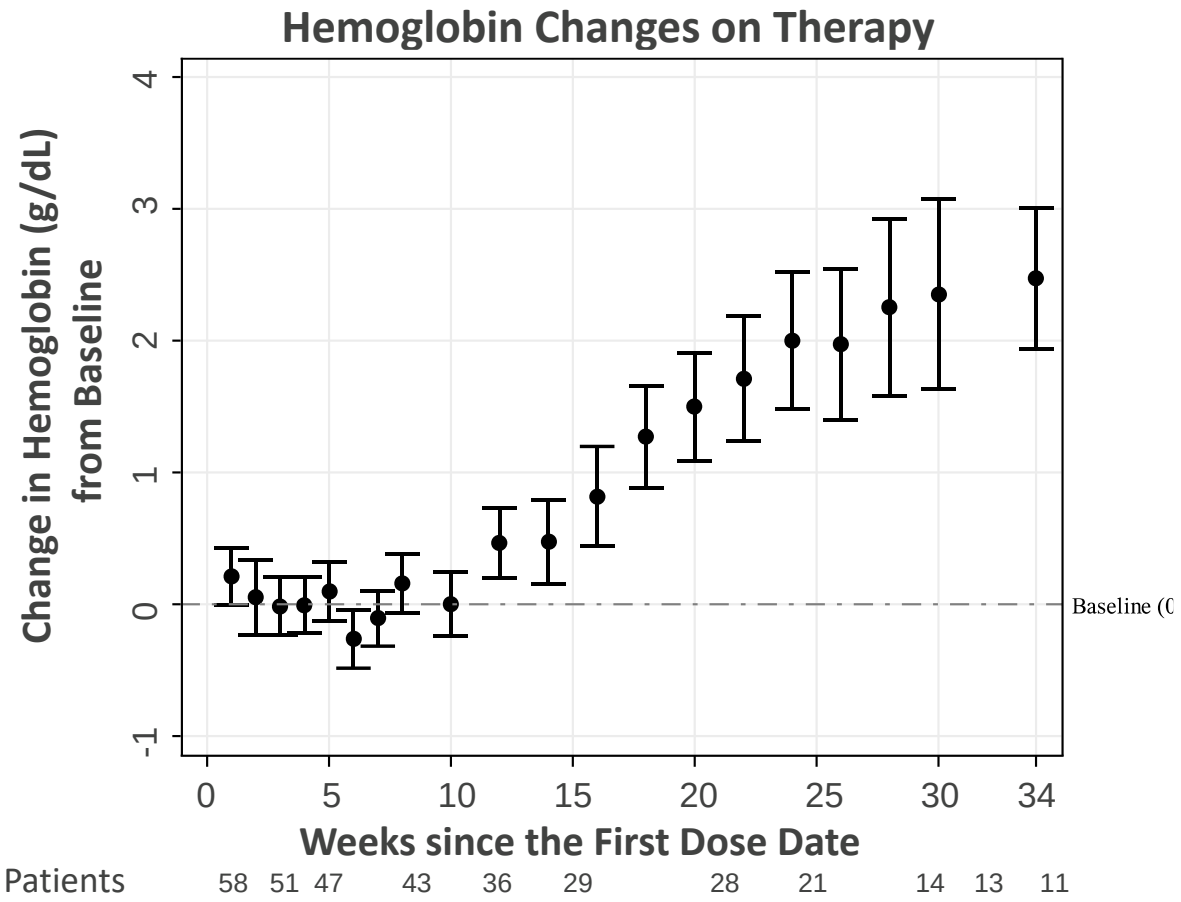
Magrolimab in Combination with Azacitidine is Well Tolerated

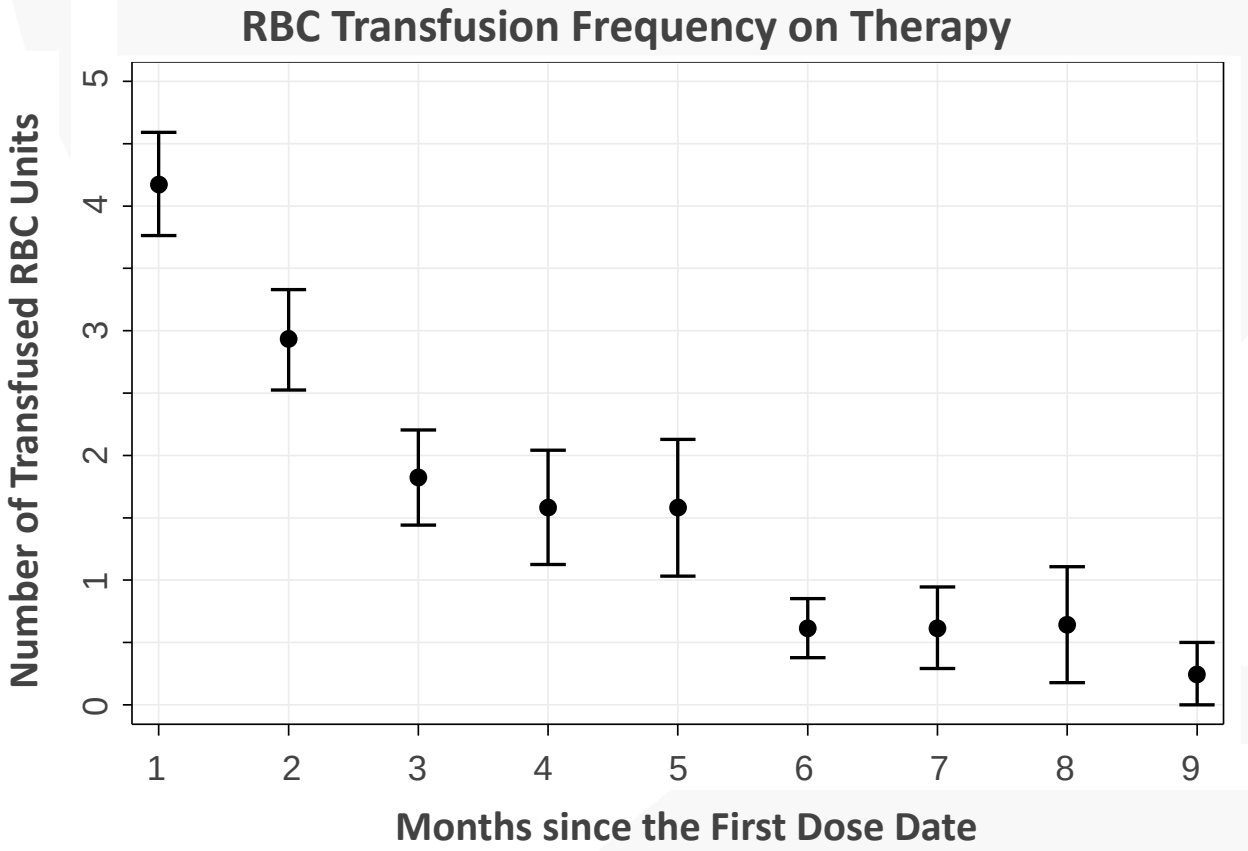


AEs ≥ 15% or AEs of interest are shown
All patients with at least one magrolimab dose are shown
*Includes neutropenia and neutrophil count decreased
**Includes thrombocytopenia and platelet count decreased

- No MTD was reached; magrolimab+AZA profile consistent with AZA monotherapy
- No significant cytopenias, infections, or autoimmune AEs were observed (most patients cytopenic at baseline)
- No deaths were observed in the first 60 days on therapy
- Treatment discontinuation due to AE occurred in only 1 of 62 (1.6%) of all patients treated with magrolimab + AZA

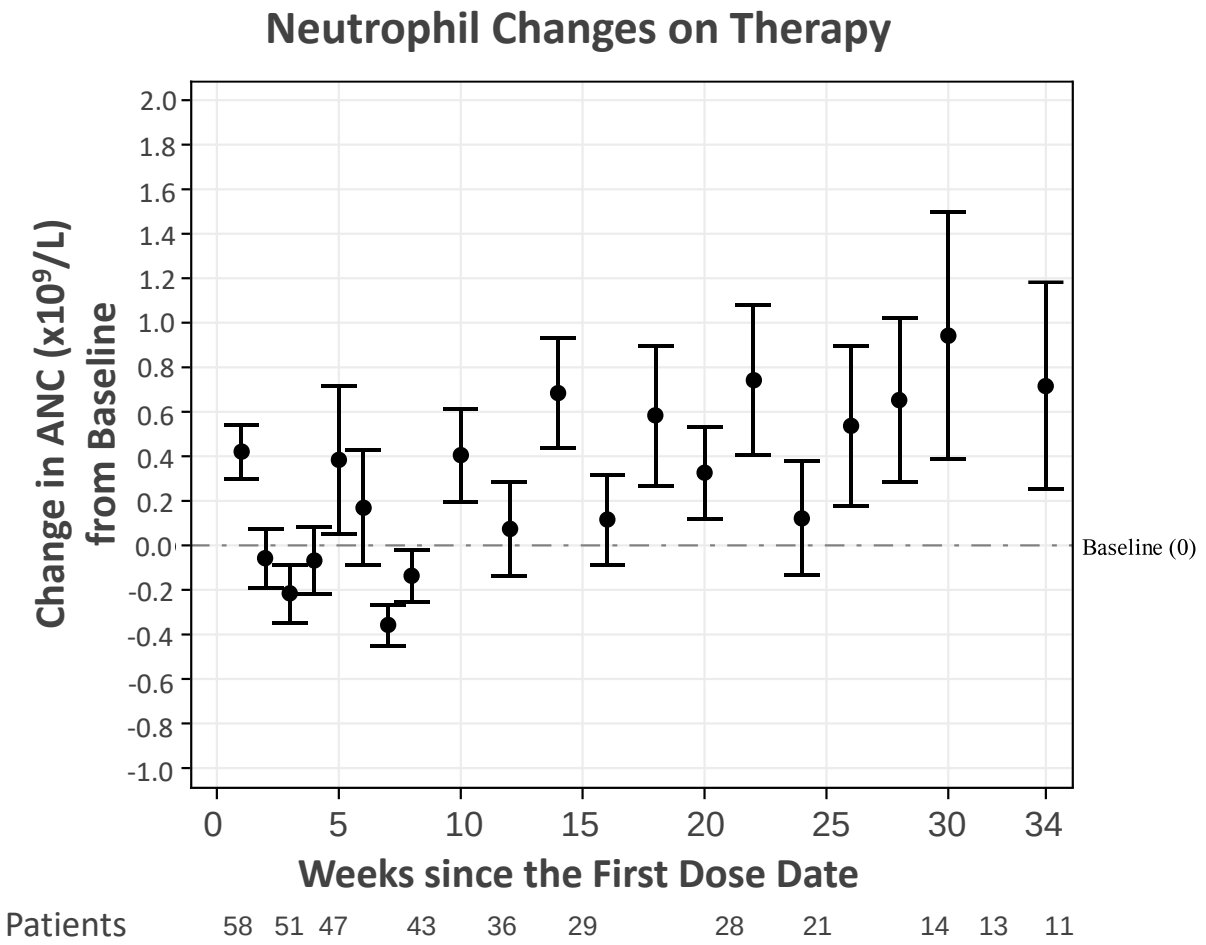
On Target Anemia is a Pharmacodynamic Effect and is Mitigated with a Magrolimab Priming and Maintenance Dosing Regimen

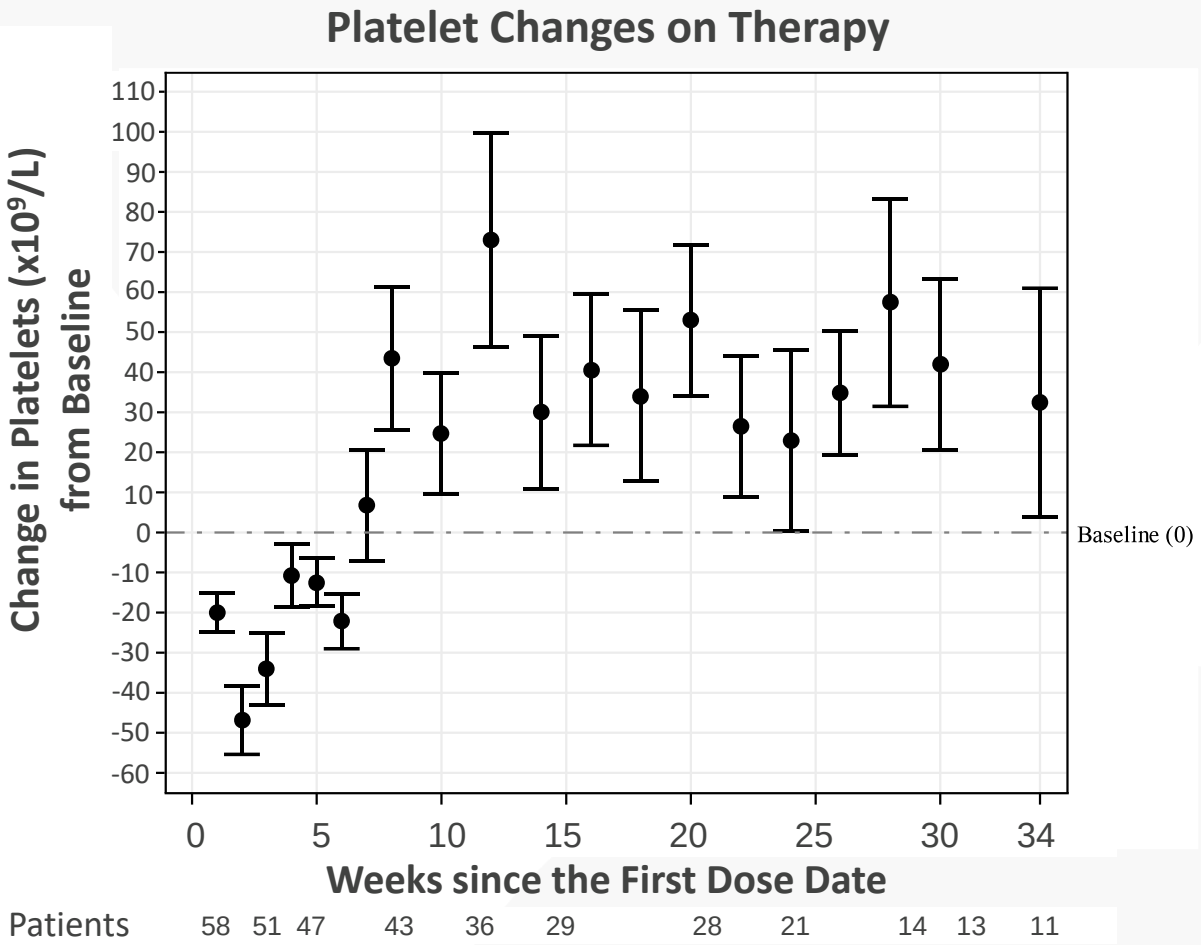




- An initial priming dose mitigates on target anemia by CD47 blockade, resulting in a transient mild hemoglobin drop on the first dose (mean of 0.4 g/dL) , which returns to baseline
- The majority of patients have had significant hemoglobin improvement and decrease in transfusion frequency with therapy

Neutrophil and Platelet Improvement is Seen on Magrolimab + AZA Therapy



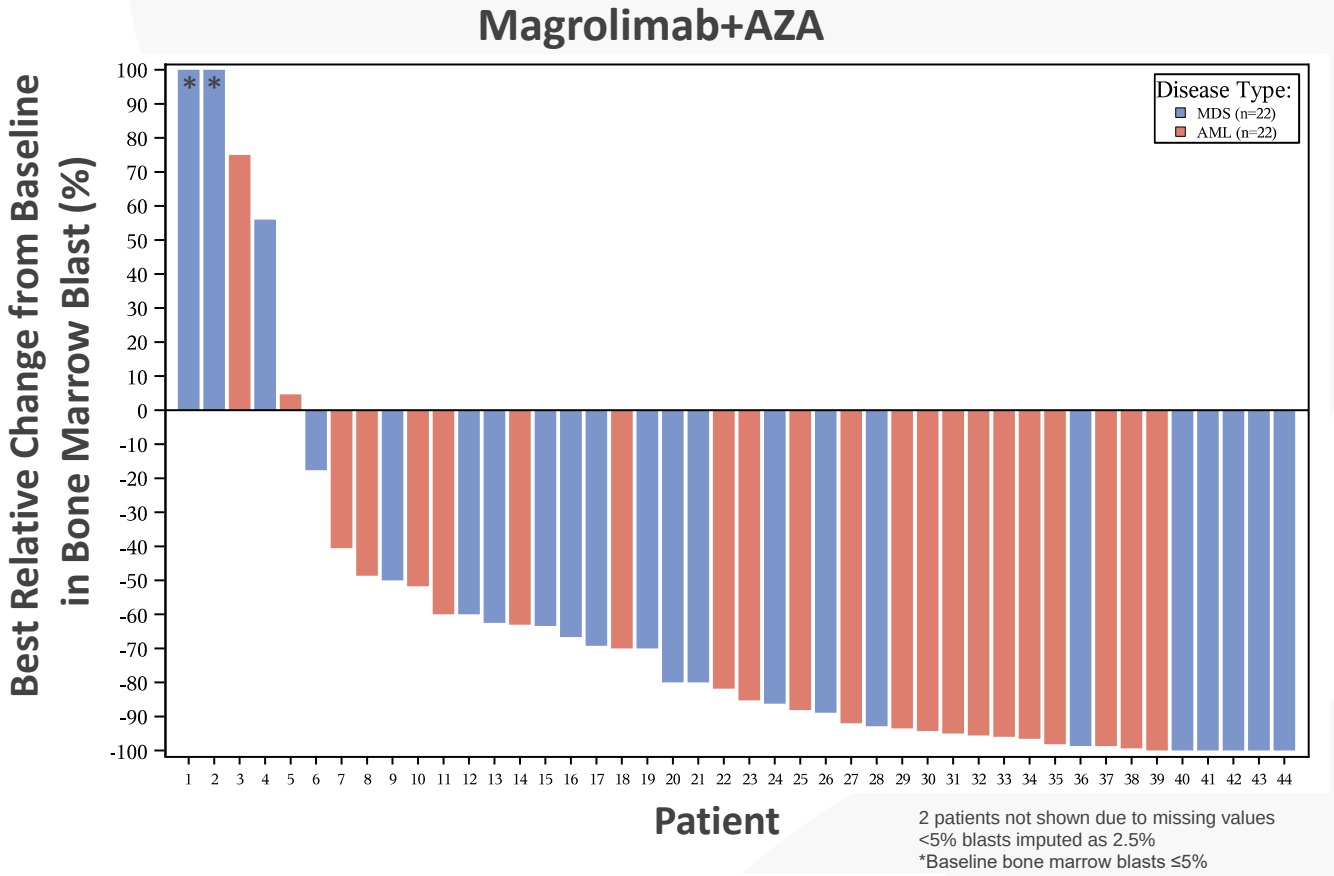


- Magrolimab + AZA does not induce significant neutropenia or thrombocytopenia
- The majority of patients improve their neutrophil and platelet count while on therapy

Anti-Leukemic Activity is Observed with Magrolimab + AZA in MDS and AML

Best Overall Response	1L MDS N=24	1L AML N=22
ORR	22 (92%)	14 (64%)
CR	12 (50%)	9 (41%)
CRi	-	3 (14%)
PR	0	1 (5%)
MLFS/ marrow CR	8 (33%) 4 with marrow CR + HI	1 (5%)
Hematologic improvement (HI)	2 (8%)	-
SD	2 (8%)	7 (32%)
PD	0	1 (5%)

Response assessments per 2006 IWG MDS criteria and 2017 AML ELN criteria;
Patients with at least one post-treatment response assessment are shown, all other patients are on therapy and are too early for first response assessment, except for 2 MDS patients not evaluable (withdrawal of consent) and 3 AML (1 AE, 2 early withdrawal)
"-" not applicable

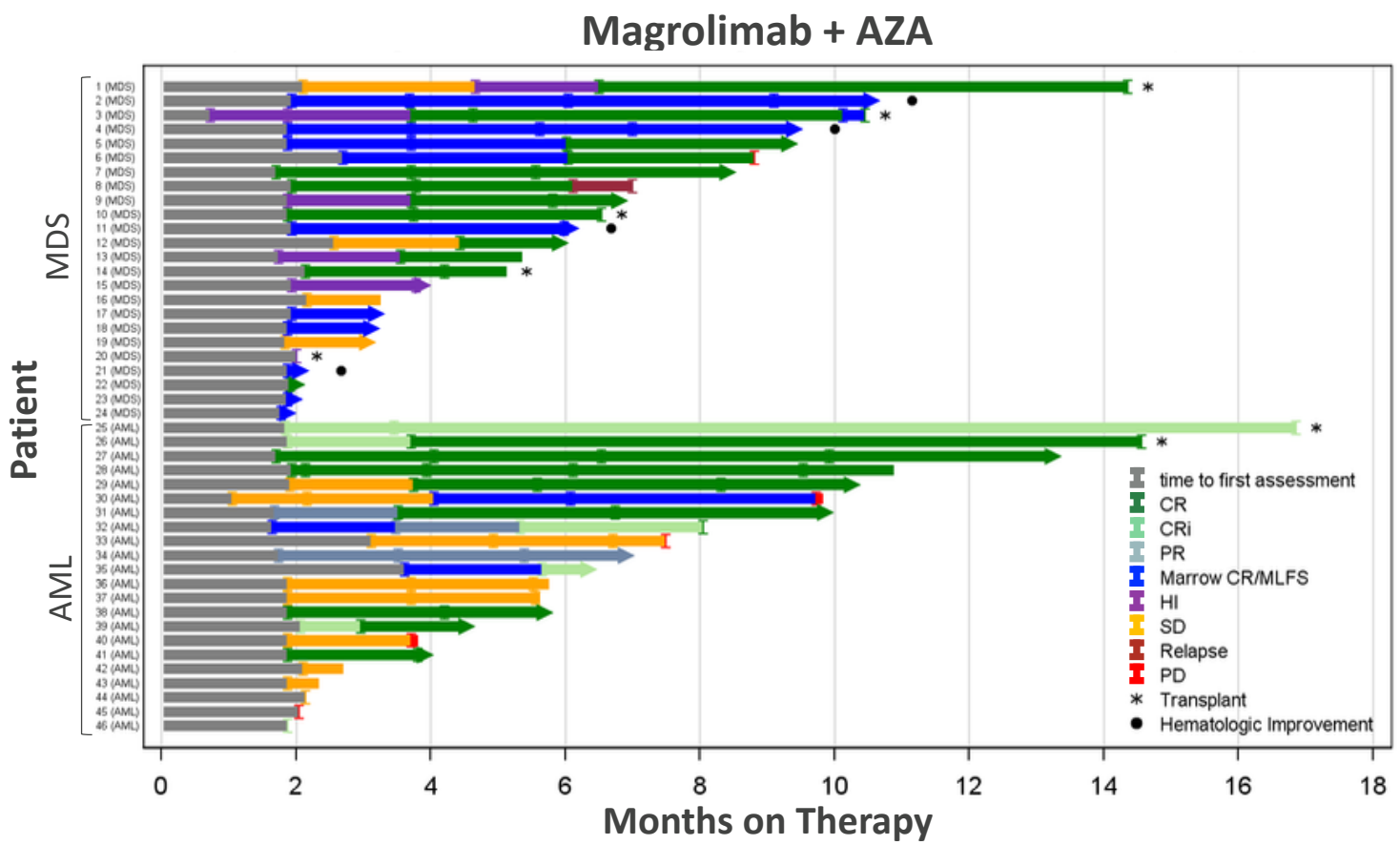


- Magrolimab + AZA induces a 92% ORR (50% CR) in MDS and 64% ORR (55% CR/CRi) in AML
- Median time to response is 1.9 months, more rapid than AZA alone
- Magrolimab + AZA efficacy compares favorably to AZA monotherapy

Deep and Durable Responses are Seen in Magrolimab + AZA Treated Patients

Parameter	1L MDS N=24	1L AML N=22
RBC transfusion independence[1]	4/9 (44%)	8/11 (73%)
Complete cytogenetic response in responders[2]	5/19 (26%)	6/10 (60%)
MRD negativity in responders	5/22 (23%)	8/14 (57%)
Median duration of response (months)	Not reached (0.03+ – 9.76+)	Not reached (0.03+ – 15.1+)
Median follow-up [range] (months)	6.4 [2.0 – 14.4]	8.8 [1.9 – 16.9]

Minimal residual disease (MRD) was evaluated by multiparameter flow cytometry
Cytogenetic response defined per 2003 and 2006 IWG criteria;
[1]Patients shown for those who were RBC transfusion dependent at baseline and achieved RBC transfusion independence at any time on study
[2]Responses shown for all responding patients with abnormal cytogenetics at baseline



Ongoing response post-transplant is shown

- Complete cytogenetic responses and MRD negativity is observed in MDS and AML patients
- No median duration of response has been reached for MDS or AML [median follow-up of 6.4 and 8.8 months, respectively]
- 15% of patients (7/46) received an allogeneic stem cell transplant and continue in response
- Median overall survival has not been reached in either MDS or AML patients

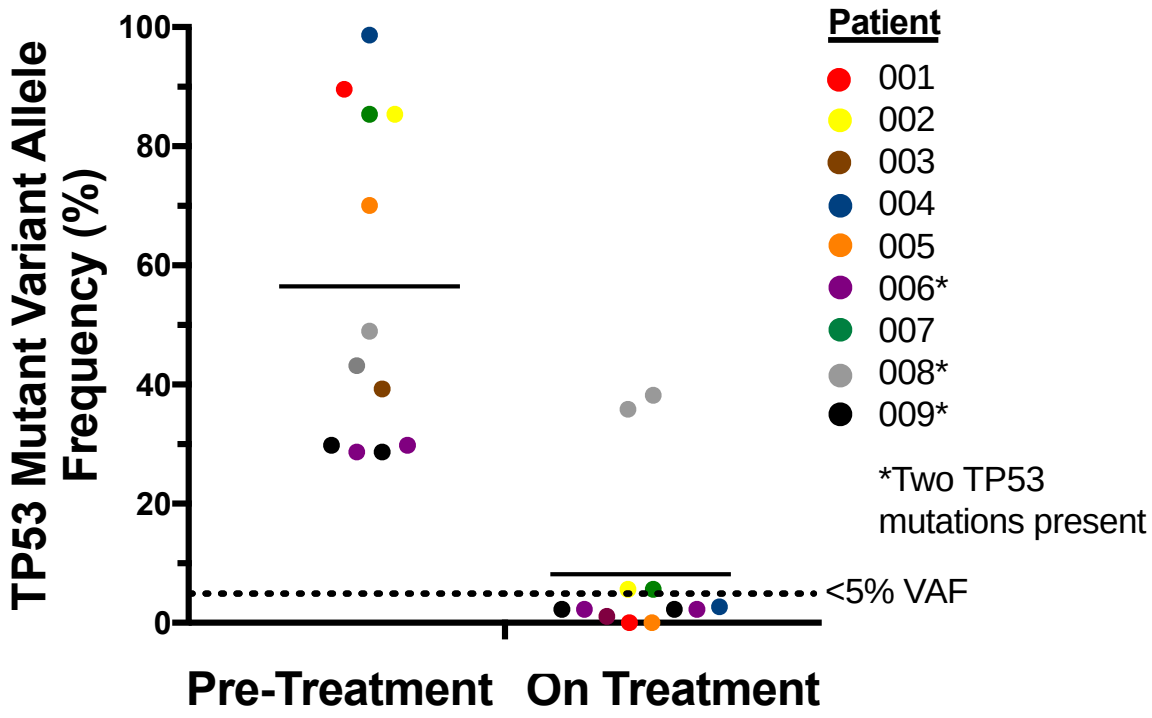
Magrolimab + AZA Eliminates Disease in AML Patients with *TP53* Mutation

Efficacy in *TP53* Mutant AML Patients

Best Overall Response	AML *TP53* Mutant (N=9)
ORR	7 (78%)
CR	4 (44%)
CRi	3 (33%)
Complete cytogenetic response in responders*	4/6 (67%)
MRD negative of responders	4/7 (57%)
Median duration of response (months)	Not reached (0.03+ – 15.1+)
Median overall survival (months)	Not reached (3.8+ – 16.9+)
Median follow-up [range] (months)	6.9 [1.9 – 16.9]

*For patients with abnormal cytogenetics at baseline

TP53 Mutation Burden on Treatment

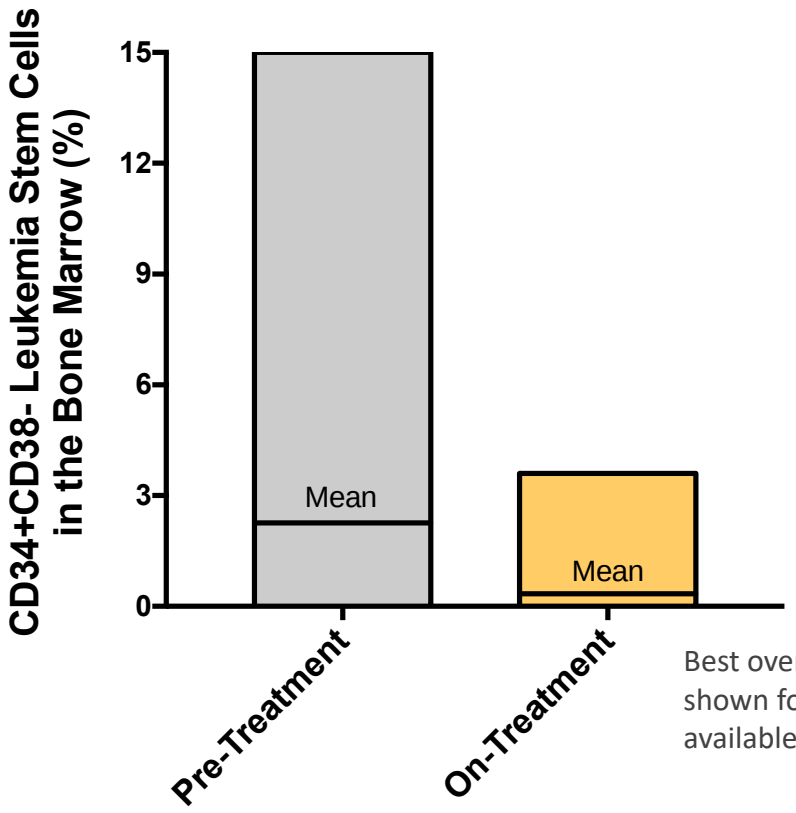


Best overall reduction is shown
NGS data shown

- Magrolimab + AZA has a high response rate and MRD negativity in *TP53* mutant AML patients
- *TP53* mutational burden is dramatically reduced in AML patients on therapy
- Median duration and survival has not been reached, which compares favorably to current therapies
  - [Venetoclax+AZA: ORR 47%, DOR 5.6mo, OS 7.2 mo (DiNardo et al., Blood 2019)]

Magrolimab + AZA Depletes Leukemia Stem Cells (LSCs) in Patients

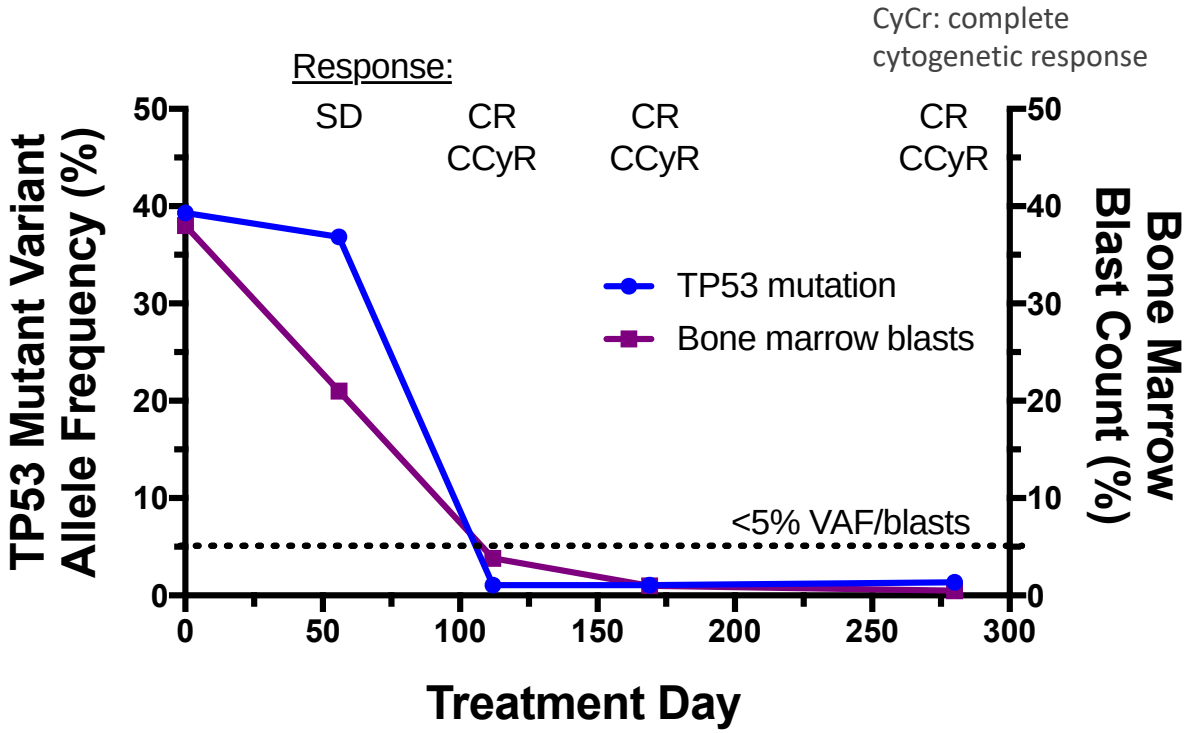
Magrolimab + AZA Impact on LSCs in MDS/AML Patients



Best overall response is shown for all patients available for analysis (N=29)

- CD34+CD38- putative LSCs were eliminated in 40% of responding MDS/AML patients

TP53 Mutant AML Patient

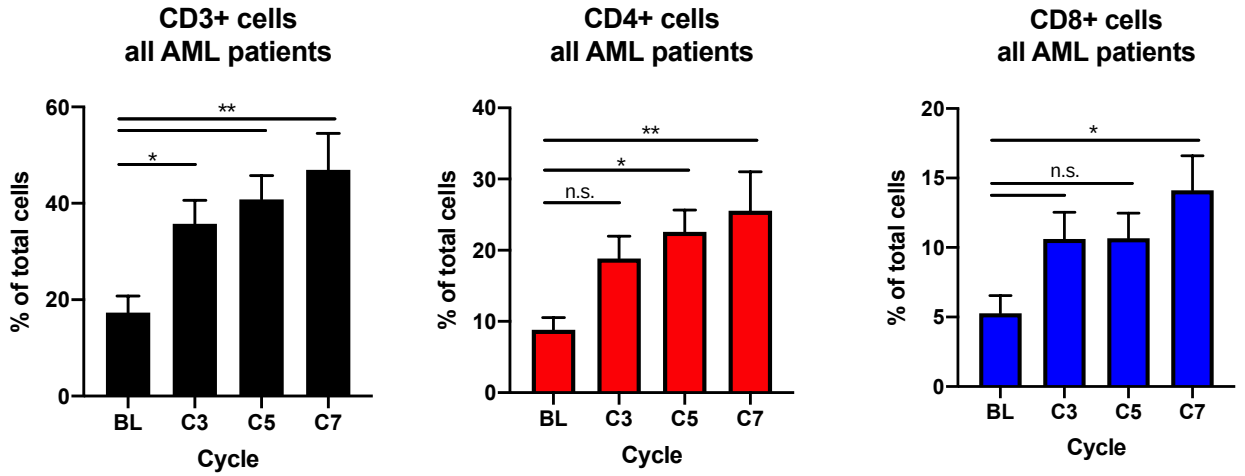


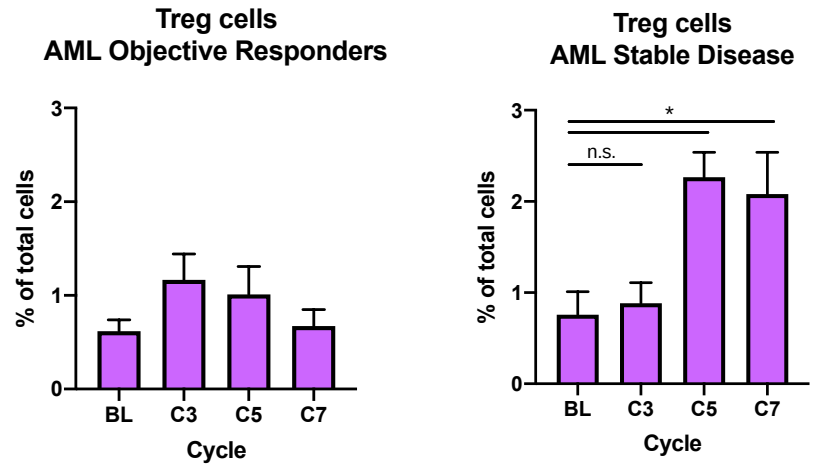
- 65 year old woman with therapy-related AML, complex karyotype and *TP53* mutation
- Achieved CR, complete cytogenetic response (CCyR) and elimination of LSCs at month 5 with ongoing response >280 days

Increased T Cell Infiltration in AML Patients Treated With Magrolimab Azacitidine Combination

Data produced in collaboration with the Immunotherapy Platform at MD Anderson Cancer Center

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center





- In AML patients, an increase in total T cells, CD4+ cells, and CD8+ cells is observed in the bone marrow
- Stable disease patients show significantly elevated Treg levels
- These data suggest an adaptive immune response and the opportunity for combination with checkpoint inhibitors
- Additional analyses are ongoing

Conclusions

- Magrolimab is a first-in-class antibody targeting the macrophage checkpoint CD47
- Magrolimab is well tolerated with AZA with improvement in cytopenias on therapy
- Encouraging efficacy is observed with Magrolimab + AZA in untreated MDS and AML
  - MDS: (ORR 92%, CR 50%), median DOR not reached (6.4 mo median follow-up)
  - AML: (ORR 64%, CR/CRi 55%), median DOR not reached (8.8 mo median follow-up)
- Efficacy is observed particularly in *TP53* mutant AML patients (78% CR/Cri), a poor prognosis population
- Magrolimab + AZA eliminates putative LSCs in responding patients
- Expansion cohorts are ongoing in MDS and AML (NCT03248479) with registrational studies in progress for MDS

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CALIFORNIA'S STEM CELL AGENCY

Trial Sponsor: Forty Seven, Inc.

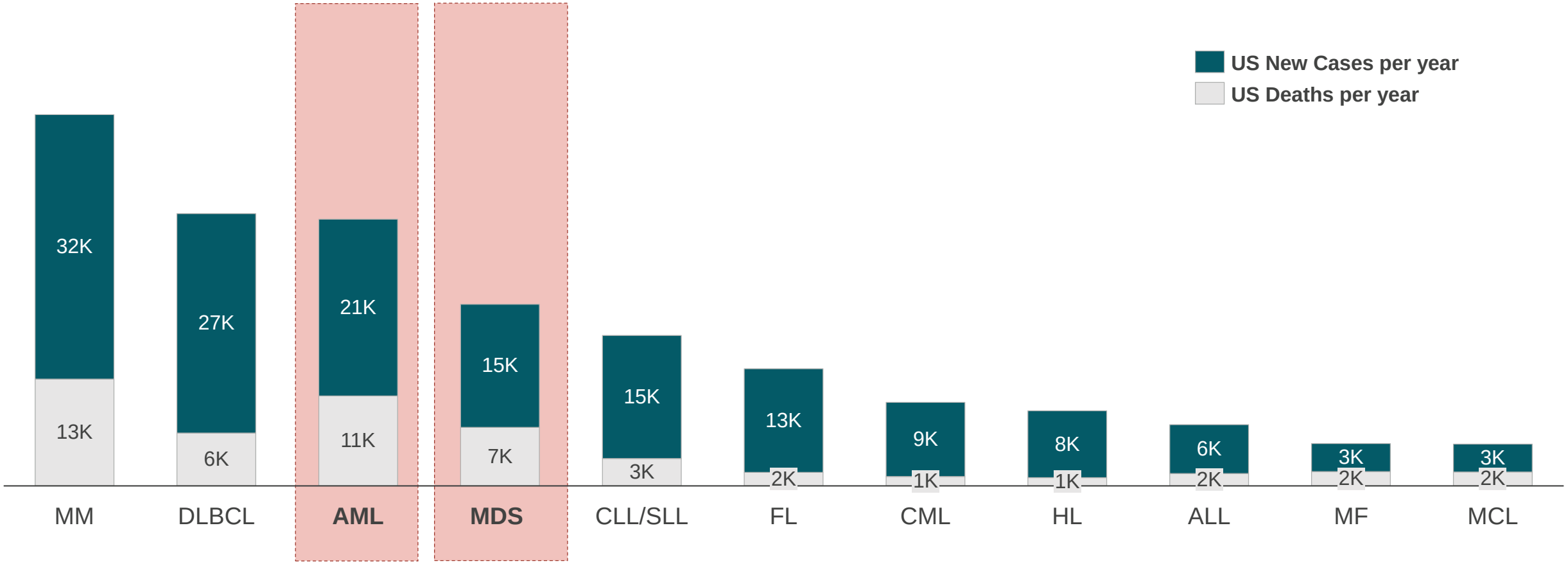
Special thanks to the patients, families and medical staff that participated on this trial

Treatment Landscape, Clinical and Registration Strategy in MDS

Mark Chao, M.D., Ph.D.

High Burden of Disease For MDS and AML

Annual Incidence and Mortality by Hematological Malignancy Type in US[1-5]



Note: MDS incidence is underreported to registries (SEER) 14.7K and broader analyses of medical claims reveals >2x higher incidence

[1] National Cancer Institute Surveillance, Epidemiology, and End Results (SEER) Cancer Stat Facts,

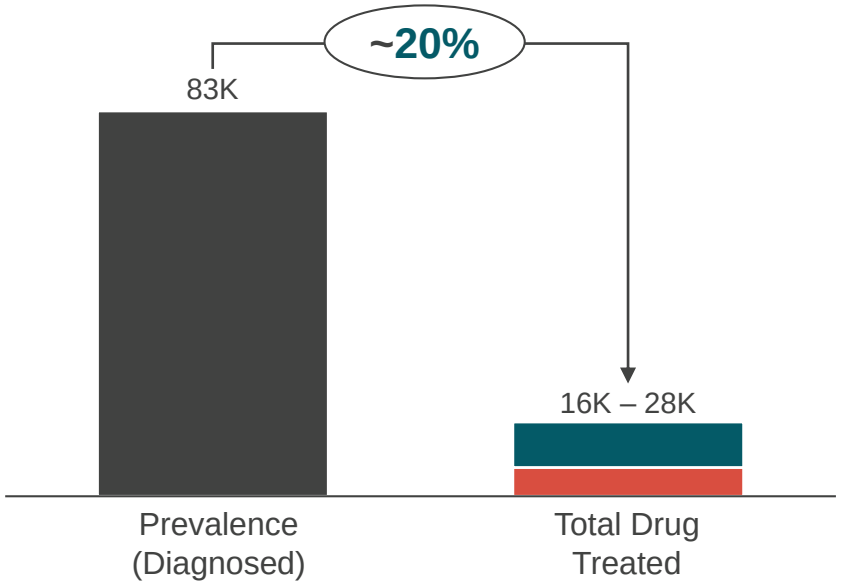
[2] CancerMPact® Patient Metrics (Kantar Health). Available from www.cancermpact.com. Accessed July 2019

[3] CDC Wonder https://wonder.cdc.gov, [5] Cancer statistics www.cancer.net

There is a High Unmet Need in MDS

Low Treatment Rates

US MDS Epidemiology 2018[1-3]



Limited Treatment Options

< 5%	~20%	~75%
Allogeneic Stem Cell Transplant	**Active Therapies** *Vidaza, Dacogen, Revlimid (Low Risk del5q)*	**Supportive Care** or **Watchful Waiting**

or or

No new therapies approved in over 13 years[5]

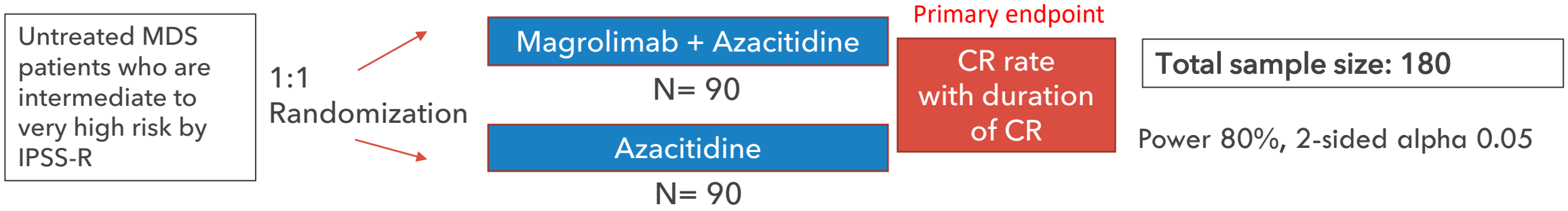
1L Higher Risk MDS US Estimated Market Size = $1B - $2B[4]

Source: [1.] CancerMPact® Patient Metrics (Kantar Health). Available from www.cancermpact.com. Accessed July 2019, [2] US MDS prevalence = 50 – 170K based on National Cancer Institute Surveillance, Epidemiology, and End Results – SEER (www.seer.cancer.gov) and Zeidan et al Blood Reviews 2019, [3] Decision Resource Group MDS Report 2019, [4] Estimated Market Size = Total Drug Treated MDS Patients (16K - 28K) x % of Higher Risk MDS (40%) x Average Branded Immuno-Oncology Drug Price ($158K); Average List Price of 7 approved I/O Agents (AnalySource June 2019), [5]Dacogen was last approved therapy for MDS; Package insert (https://www.accessdata.fda.gov/drugsatfda_docs/label/2018/021790s021lbl.pdf) provides original approval date of 2006.

Registration Plan Provides Two Distinct Opportunities for Accelerated Approval

- Following recent interactions with FDA under SPA process:
  - Amending the protocol of the ongoing Phase 1b trial to evaluate every two week dosing in approximately 90 patients
  - Plan to initiate ENHANCE in 1H20, a randomized Phase 3 trial to support potential full and ex-U.S. approval
    - Will enroll approximately 180 patients randomized 1:1 to receive magrolimab and azacitidine or azacitidine alone
    - Adaptive trial design to potentially include additional patients evaluated for a longer period to establish overall survival benefit in support of full approval
    - Given decision to design ENHANCE as RCT with well-validated endpoints, SPA process has concluded
  - Phase 1b trial and ENHANCE will share same primary endpoint: CR with duration of response
- Clinical and CMC-enabling activities allow for a BLA filing as early as 4Q 2021
- Will also continue to enroll AML patients with TP53 mutant disease in ongoing Phase 1b trial

ENHANCE: A Randomized Phase 3 Trial in 1L Higher Risk MDS



Primary Endpoint: CR with durability of CR

Key Secondary Endpoint*
Overall survival

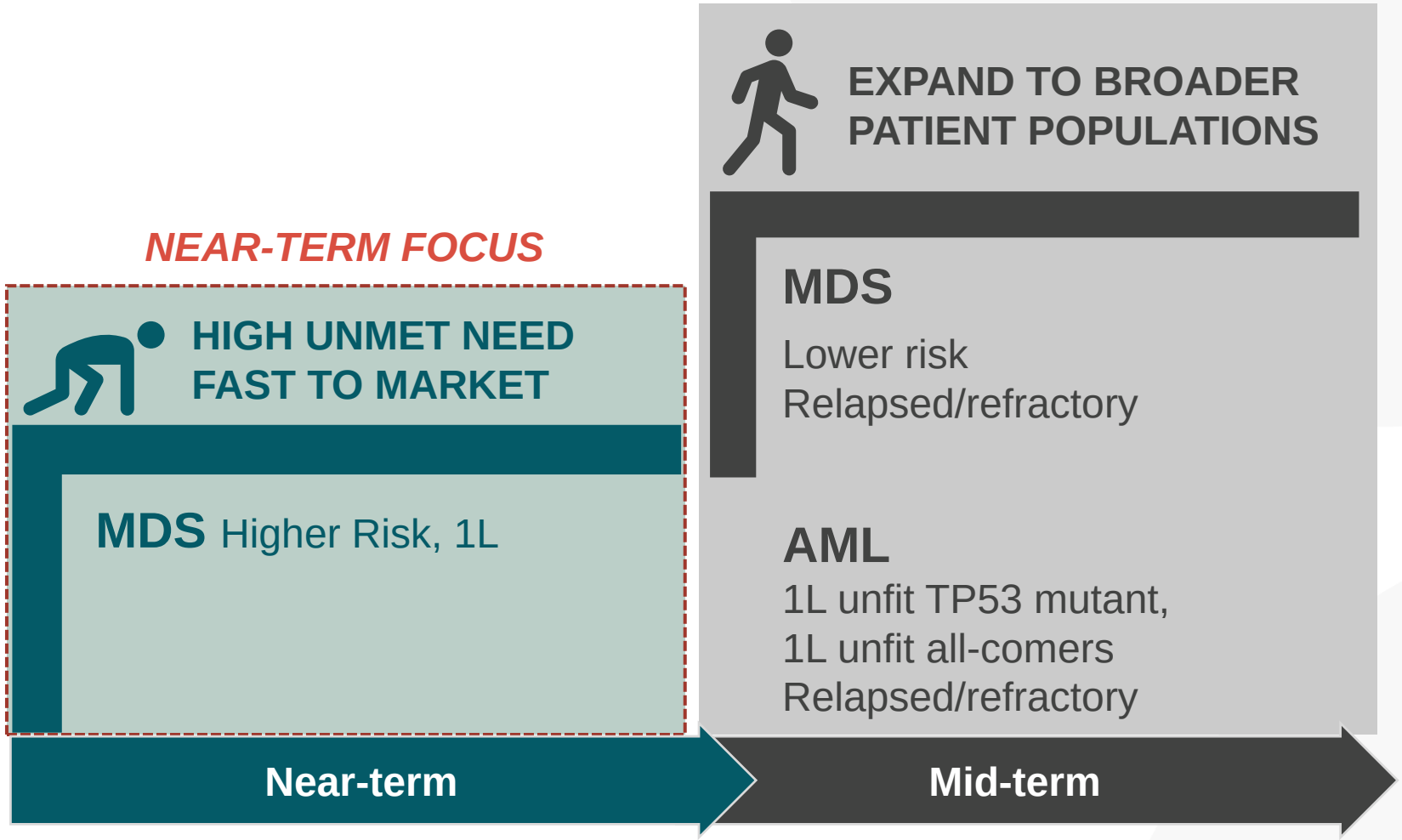
Primary analysis: CR rate/duration of CR at 180 patients

Pre-specified modification of sample size:
Based on the ongoing Phase 1b study and emerging data, the trial sample size can be modified to evaluate an overall survival endpoint for full approval and ex-US approval.

*Additional secondary endpoints are included

Magrolimab: Development Strategy for MDS and AML

Demonstrate clinical value for high unmet needs, and expand into broader patient populations



Questions

Overview of Stem Cell Transplantation and cKIT Program (FSI-174)

Leslie Kean, M.D., Ph.D.

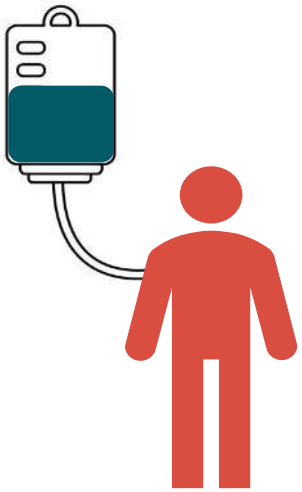
Hematopoietic Stem Cell Transplantation

Transplantation

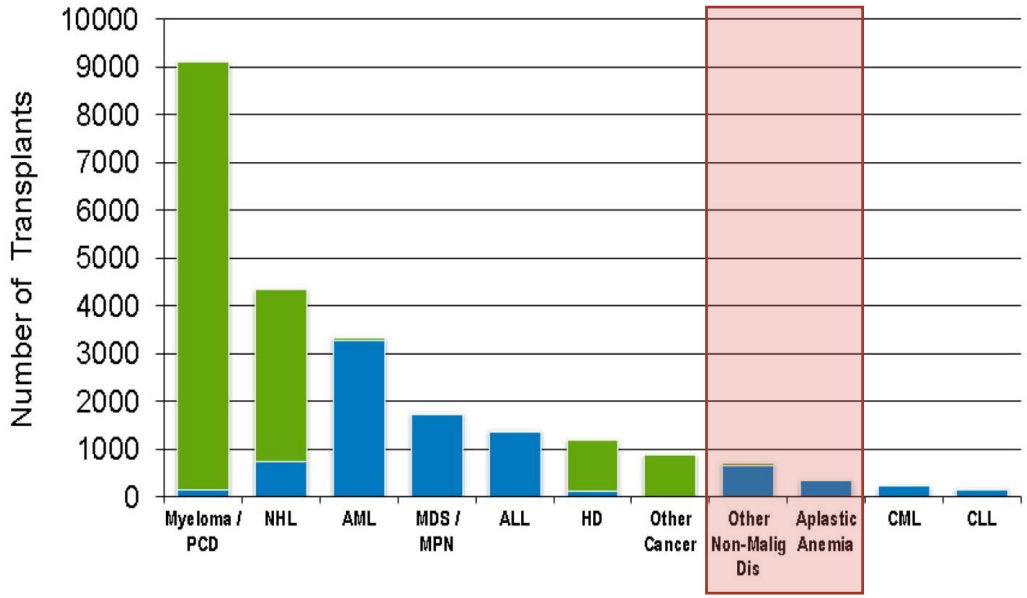
Goal: Replace a patient's HSCs with **autologous** stem cells (patient's own cells with or without gene therapy correction) or **allogeneic** stem cells (from a healthy donor).

Successful transplantation may:

- Cure genetic blood diseases (in combination with gene therapy)
- Cure autoimmune diseases by generating a new, healthy immune cell pool
- Enable organ transplantation without need for chronic immune suppression



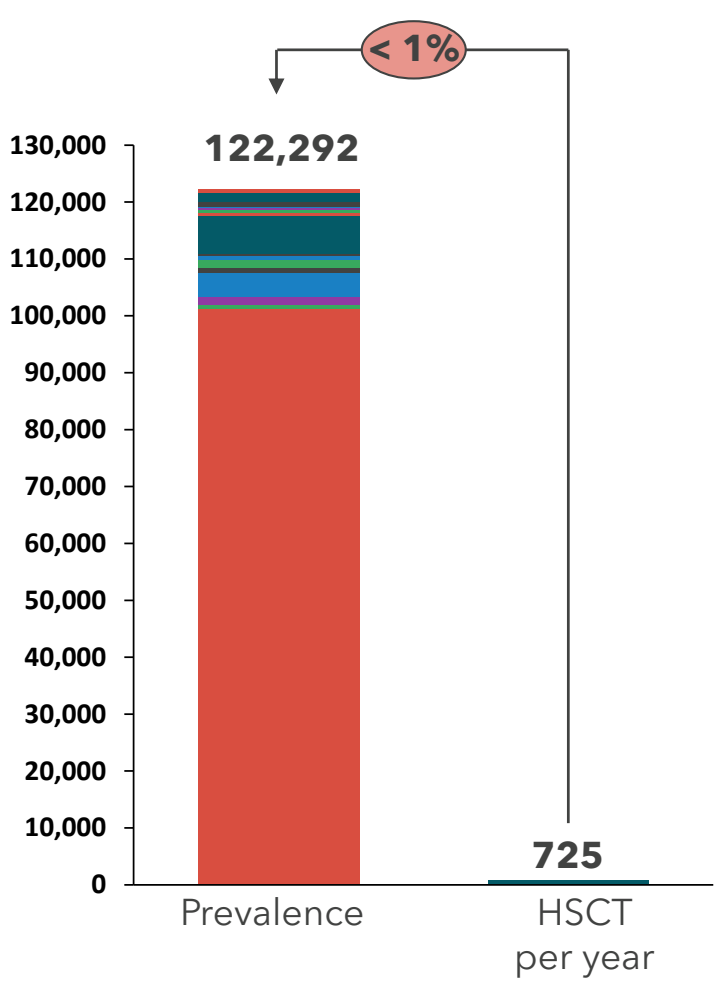
Current Use of HSCT



D'Souza A, Fretham C. Current Uses and Outcomes of Hematopoietic Cell Transplantation (HCT): CIBMTR Summary Slides, 2018

Unmet Medical Need and Potential to Expand HSC Transplantation

Non-Malignant Diseases That Could be Treated with Hematopoietic Stem Cell Transplantation



- Kostmann's syndrome
- Shwachman-Diamond syndrome
- Fanconi Anemia
- Osteopetrosis
- Metachromatic leukodystrophy
- X-linked adrenoleukodystrophy
- Mucopolysaccharidoses
- Gaucher disease
- Wiskott-Aldrich syndrome
- Chronic granulomatous disease
- Leukocyte adhesion deficiency
- Hemophagocytic lymphohistiocytosis
- X-linked hyper igM syndrome
- X-linked lymphoproliferative dis.
- X-linked agammaglobulinemia
- Common variable deficiency
- SCID
- B-Thalassemia
- Sickle cell disease

Fewer than 1% of patients in the US that could benefit from hematopoietic stem cell transplantation (HSCT) receive currently a HSCT due to the risk affiliated with the current conditioning regimens

Our Approach to Overcome Barriers to Transplantation

Current Transplantation Barriers

Approaches have not improved in half a century

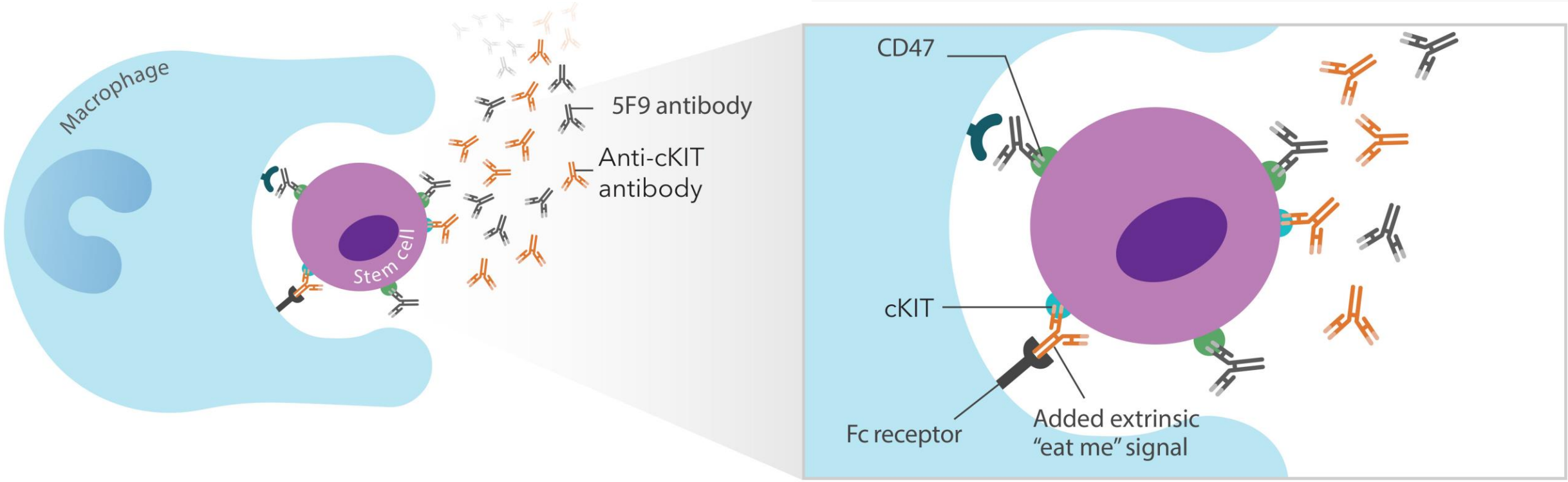
- Severe damage by toxic radiation and chemo conditioning
  - Impaired brain development in children
  - Infertility
  - Development of secondary malignancies and MDS
  - Too toxic for elderly patients and patients with pre-existing conditions
- Graft vs host disease
- Requirement for (life-long) immune suppression
- Severe life-threatening infections
- Shortage of matched bone marrow/organ donors

The Forty-Seven Solution

Science-driven approach to overcome risks and limitations of an outdated and toxic regimen

- All antibody-based regimen (radiation- and chemo-free)
- Selective and short-term antibody-mediated immune suppression to facilitate fast immune recovery
- Prevention of graft vs host disease by transplantation of purified hematopoietic stem cells (HSCs)
- Facilitation of immune tolerance for transplanted tissues and organs obviates need for immune suppression
- Co-transplantation approach (HSCs + organ) enables transplantation from non-matched organ donors and extends donor pool

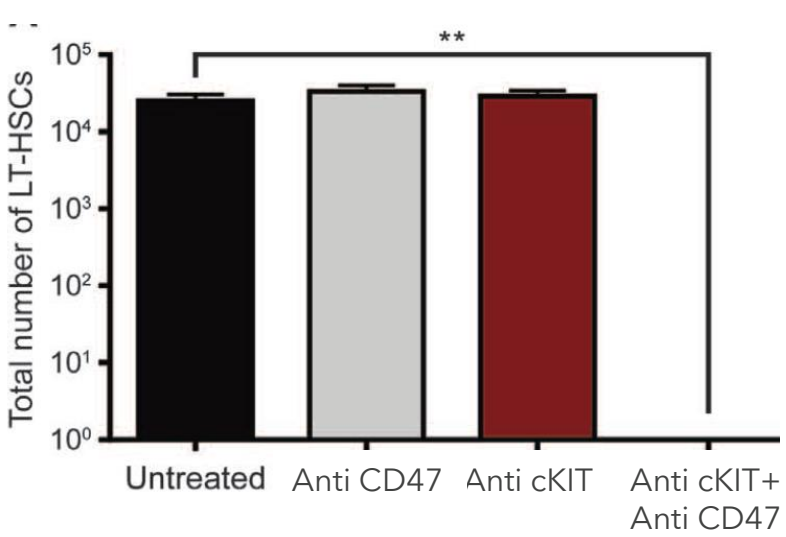
Mechanism of cKIT-CD47 Antibody Based Conditioning Regimen

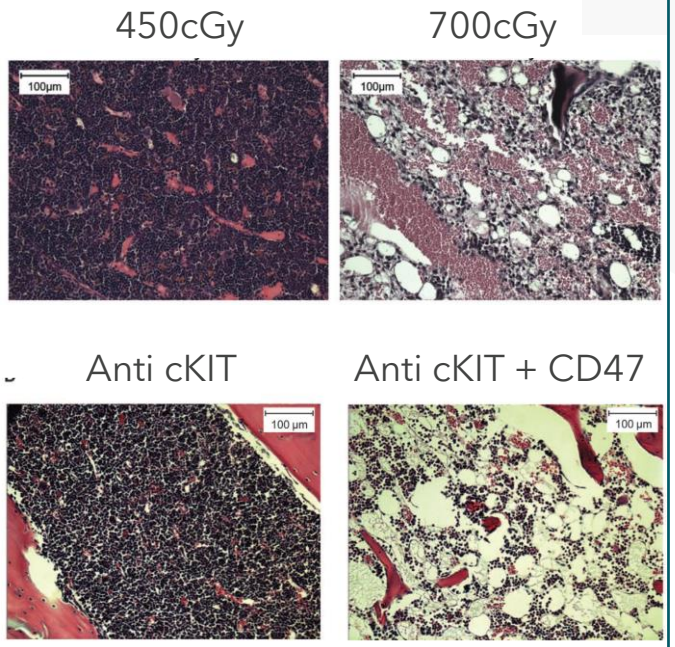


- cKIT is expressed on hematopoietic stem cells
- CD47 is a ”don’t eat me” signal that inhibits macrophage phagocytosis
- Combination of anti-CD47 antibody with targeted antibody - cKIT Ab - enhances phagocytosis of targeted - hematopoietic stem cells - by macrophages

Preclinical Proof of Concept for cKIT-CD47 Antibody-Based Conditioning Regimen in Autologous HSC Transplantation in Mouse Model

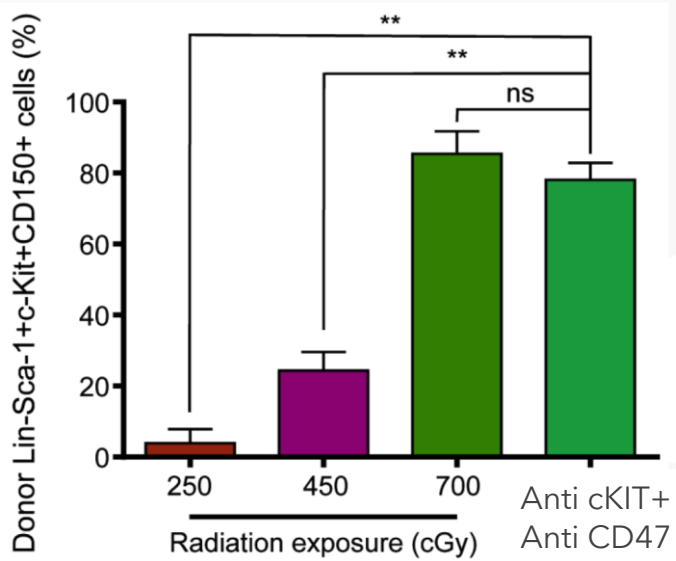
HSC Depletion in Bone Marrow 7 Days After Conditioning





Chhabra, Weissman, Shizuru, STM 2016

HSC Chimerism 24 Weeks After Transplantation



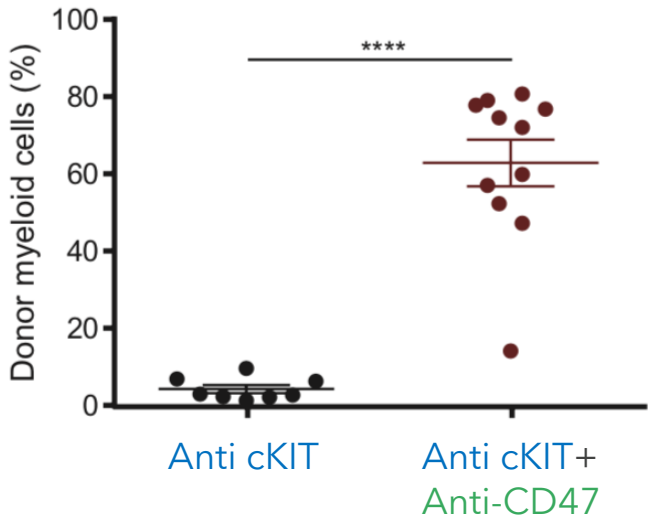
Chhabra, Weissman, Shizuru, STM 2016

Conditioning with a combination of cKIT + CD47 antibodies (but not single cKIT antibody) depletes endogenous hematopoietic stem cells (HSCs) from the bone marrow and enables HSC transplantation comparable to high dose radiation

Antibody Conditioning Approach for Autologous and Allogeneic Transplantation for Patients with Non-Oncological Diseases in Mouse Model

Autologous Transplantation

Antibodies utilized: cKIT Ab + CD47 Ab

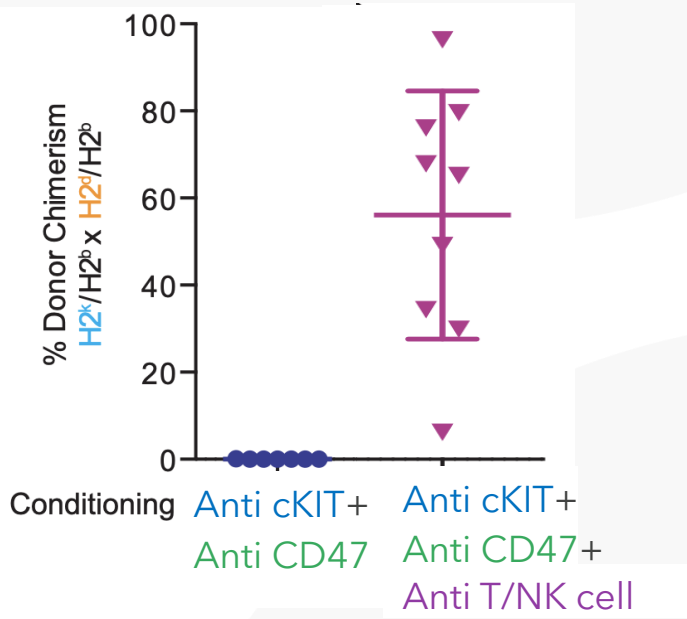


Chhabra, Weissman, Shizuru, STM 2016

Transplantation between congenic mice that differ in one gene CD45.1 vs CD45.2 → model for gene therapy of monogenic blood disorders

Allogeneic Transplantation

Antibodies utilized: cKIT Ab + CD47 Ab + T/NK cell Ab



George, Weissman, Cell Stem Cell 2019

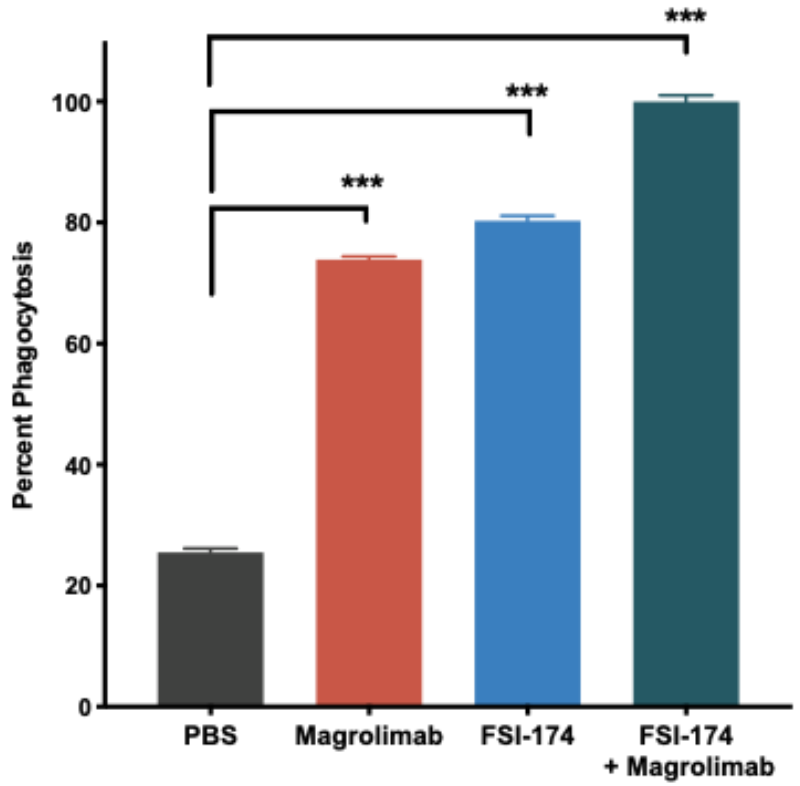
Transplantation between mice that differ in multiple genes → model for allogeneic hematopoietic stem cell transplantation

FSI-174:
A Humanized Anti-cKIT Antibody

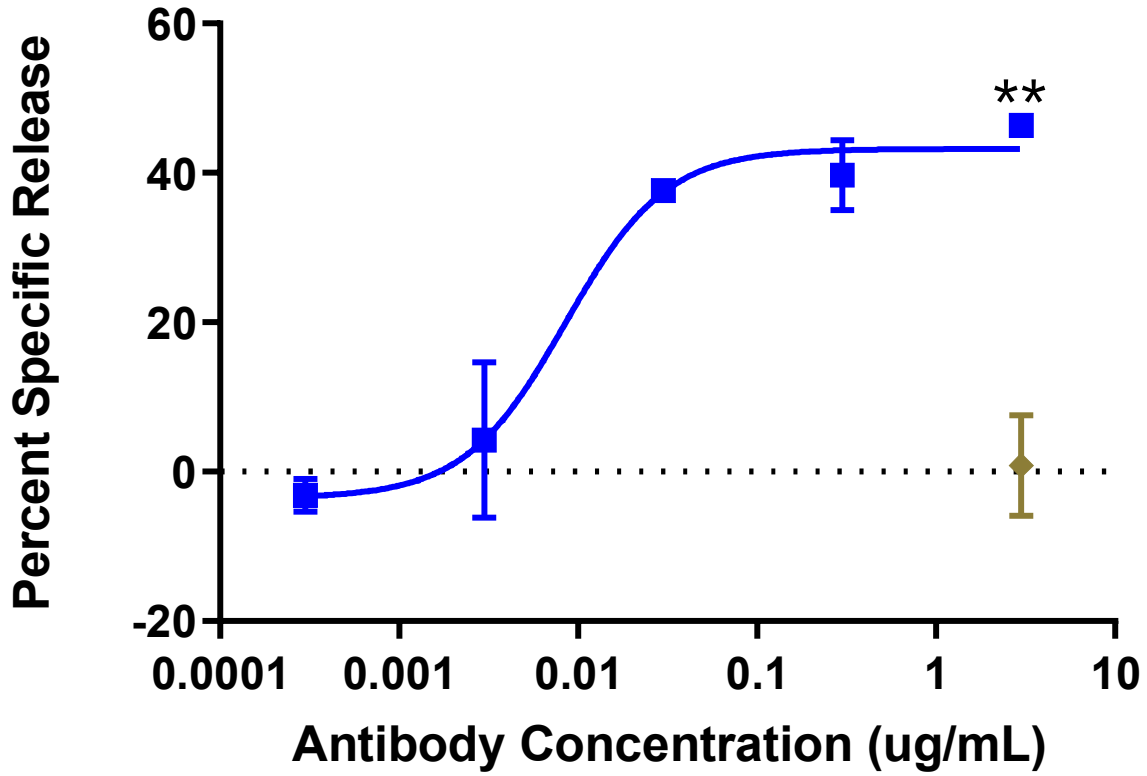
- FSI-174 binds both human and monkey cKIT with high affinities (KD = 20.1 pM and 31.9 pM, respectively)
- FSI-174 blocks stem cell factor (SCF) binding
- FSI-174 has an active IgG1, which induces:
  - Antibody dependent cell phagocytosis
  - Antibody dependent cell cytotoxicity
  - Complement dependent cytotoxicity

FSI-174 is a Novel Humanized cKIT Antibody

Antibody Dependent Cell Phagocytosis



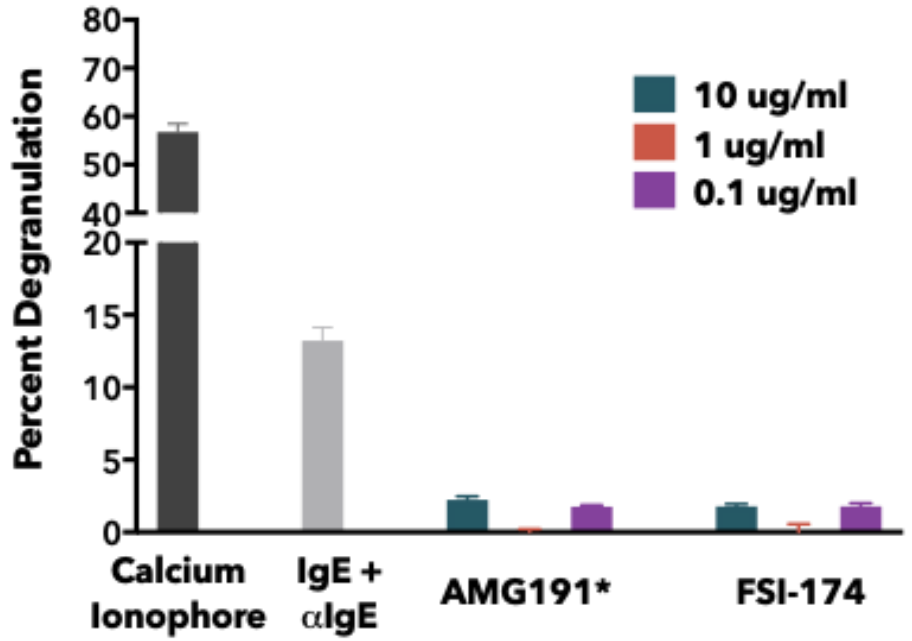
Antibody Dependent Cell Cytotoxicity



- FSI-174 has an active IgG1, which enables macrophage mediated phagocytosis and NK-cell mediated cytotoxicity

FSI-174 is Safe in Preclinical Studies - In Vitro and in Non-Human Primates

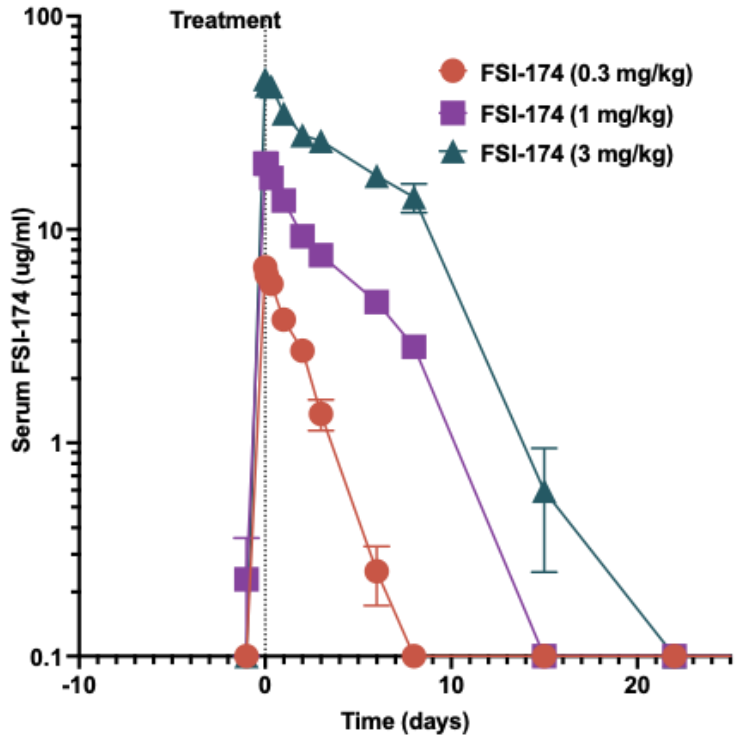
Human Mast Cell Degranulation Assay



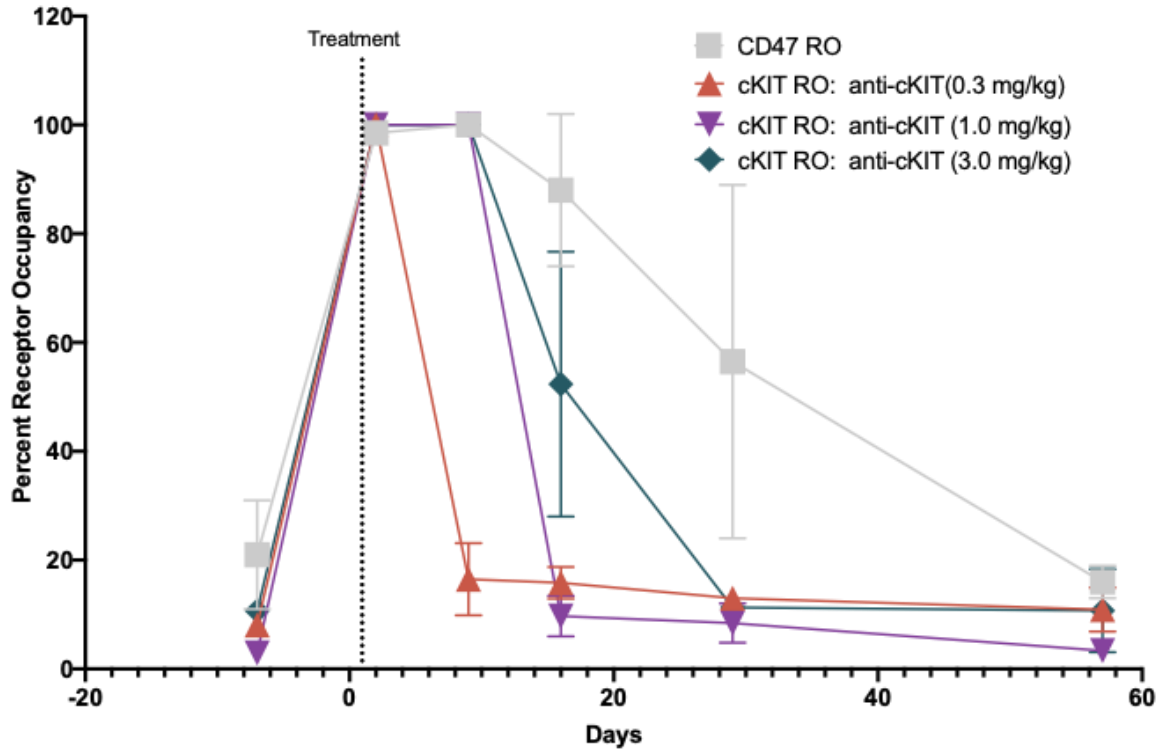
- FSI-174 has been administered to NHPs up to 50 mg/kg/week for three weeks
- FSI-174 is well tolerated
- No Evidence of mast cell degranulation
- No Lymphopenia
- **No observed adverse effect level at the highest dose (50 mg/kg)**

FSI-174 Pharmacokinetics and Pharmacodynamics in Non-Human Primates

Pharmacokinetics



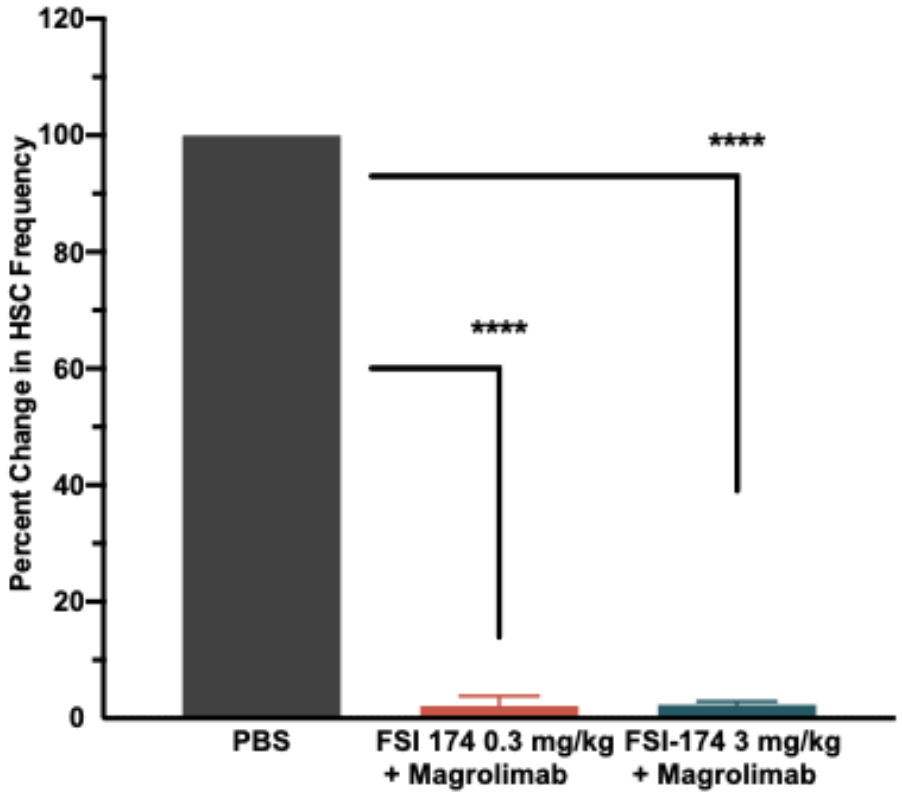
Pharmacodynamics



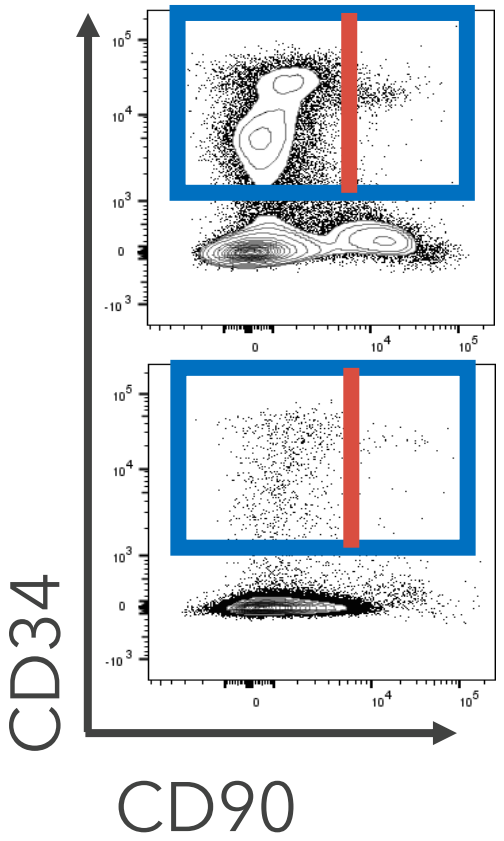
- All doses achieve measurable serum levels above the desired threshold of 0.1 ug/ml
- Dose 0.3mg/kg drops after 6 days below minimal measurable serum level, dose 1 mg/kg after 13 days and dose 3 mg/kg after 19 days
- 100% cKIT receptor occupancy on HSCs is achieved with all dose levels

FSI-174 Depletes Hematopoietic Stem Cells (HSCs) in Non-Human Primates

HSC Depletion



Bone Marrow Cytograms



- Combination of FSI-174 and magrolimab depletes hematopoietic stem cells (HSC) in non-human primates
- This shows that conditioning for HSC transplantation with antibodies instead of chemo or radiation is feasible

FSI-174 Development Plan

Jens-Peter Volkmer, M.D.

Development Approach For All Antibody Based Conditioning Regimen

First Stage
cKIT Single Antibody First-In-Human Trial

Antibodies:
- anti-cKIT

First-In-Human Trial to establish safety, PK and PD of cKIT Ab

Second Stage
Autologous Transplantation of Gene Corrected Cells

Antibodies:
- anti-cKIT
- anti-CD47

Indications:
Initially target diseases that have the potential to be corrected with transplantation of autologous gene-modified blood forming stem cells

Third Stage
Allogeneic Transplantation

Antibodies:
- anti-cKIT
- anti-CD47
- anti-T/NK cell

Indications:
- Polygenic non-malignant blood disorders
- Monogenic blood disorders for which gene therapies do not exist

Summary

- Hematopoietic stem cell transplantation can be curative for a number of malignant and non-malignant diseases; however, its use has been limited by toxicities from conditioning approaches
- Forty Seven is developing a new antibody-based conditioning approach targeting cKIT to provide a pro-phagocytic signal and blocking the anti-phagocytic (“don’t eat me”) signal CD47
- Forty Seven has developed a novel anti-human cKIT antibody: FSI-174
- FSI-174 has been shown to be safe in a NHP toxicology study
- FSI-174 has been shown to deplete HSCs in NHPs when combined with magrolimab without depletion of mature immune cells
- FSI-174 will be tested in a first-in-human study to establish dose and safety starting in Q1 2020
- FSI-174 will be tested subsequently in combination with magrolimab for depletion of hematopoietic stem cells to initially target diseases that have the potential to be corrected with transplantation of autologous gene-modified blood-forming stem cells– in collaboration with bluebird bio

Our Foundation

Forty Seven is built on a culture of scientific rigor and passion for helping people to live fuller, healthier lives. This is seen in our actions and every decision we make.

Questions

NASDAQ: FTSV

Forty Seven

Helping Patients Defeat Their Cancer

BOLD | HUMBLE | INQUISITIVE | PASSION TO ACTION