



NASDAQ: FTSV
Forty Seven

Helping Patients Defeat Their Cancer

Corporate Overview

June 2019



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.

We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy and financial needs. These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including, among other things: the success, cost and timing of our product development activities and clinical trials; our expectations about the timing of achieving regulatory approval and the cost of our development programs; our ability to obtain funding for our operations, including funding necessary to complete further development and commercialization of our product candidates; the commercialization of our product candidates, if approved; our plans to research, develop and commercialize our product candidates; our ability to attract collaborators with development, regulatory and commercialization expertise; our expectations regarding our ability to obtain and maintain intellectual property protection for our product candidates; future agreements with third parties in connection with the commercialization of our product candidates; our ability to maintain, expand, protect and enforce our intellectual property portfolio; our ability to operate our business without infringing, misappropriating or otherwise violating the intellectual property rights of third parties; the size and growth potential of the markets for our product candidates, and our ability to serve those markets; the rate and degree of market acceptance of our product candidates; regulatory developments in the United States and foreign countries; our ability to contract with third-party suppliers and manufacturers and their ability to perform adequately; the success of competing therapies that are or may become available; and our ability to attract and retain key scientific or management personnel. These risks are not exhaustive. New risk factors emerge from time to time and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance or achievements. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation.

In addition, statements that “we believe” and similar statements reflect our beliefs and opinions on the relevant subject. These statements are based upon information available to us as of the date of this presentation, and while we believe such information forms a reasonable basis for such statements, such information may be limited or incomplete, and our statements should not be read to indicate that we have conducted an exhaustive inquiry into, or review of, all potentially available relevant information. These statements are inherently uncertain and investors are cautioned not to unduly rely upon these statements.

More information about the risks and uncertainties faced by Forty Seven is contained under the caption “Risk Factors” included in the company’s periodic filings with the Securities and Exchange Commission at www.sec.gov. Forty Seven disclaims any intention or obligation to update or revise any forward-looking statements, whether as a result of new information, future events or otherwise.

Building a Leading Immuno-Oncology Company Focused on Macrophage Checkpoint Therapies

- Founded in 2015 by Irv Weissman and colleagues at Stanford University, identifying the CD47-SIRP α pathway as a novel macrophage immune checkpoint
- Developing a pipeline of macrophage-directed therapies
- Developed 5F9, our leading commercial CD47 targeting antibody that has been well-tolerated, has demonstrated clinical activity in monotherapy and in combination therapy with rituximab
- Advancing novel SIRP α and cKIT targeting antibodies towards IND
- IPO in July 2018 and added to NASDAQ Biotechnology Index (NBI) effective December 24, 2018
- Announced AstraZeneca/Acerta Pharma collaboration & Genentech collaboration expansion in May 2019
- Phase 1b AML/MDS Results 5F9 + azacitidine: ASCO Poster & EHA Oral Presentation
- Phase 1b/2 NHL Results 5F9 + rituximab: EHA Oral Presentation
- Cash through 1H2020
- Leveraging our scientific insights and pharmaceutical drug development expertise to develop novel therapies that activate the immune system to **help patients defeat their cancer**

Phase 1b NHL data published in the New England Journal of Medicine

The NEW ENGLAND
JOURNAL of MEDICINE

Original Article

CD47 Blockade by Hu5F9-G4 and Rituximab in Non-Hodgkin’s Lymphoma

Ranjana Advani, M.D., Ian Flinn, M.D., Ph.D., Leslie Popplewell, M.D., Andres Forero, M.D., Nancy L. Bartlett, M.D., Nilanjan Ghosh, M.D., Ph.D., Justin Kline, M.D., Mark Roschewski, M.D., Ann LaCasce, M.D., Graham P. Collins, M.D., Thu Tran, B.S., Judith Lynn, M.B.A., B.A., James Y. Chen, M.D., Ph.D., Jens-Peter Volkmer, M.D., Balaji Agoram, Ph.D., Jie Huang, Sc.D., Ravindra Majeti, M.D., Ph.D., Irving L. Weissman, M.D., Chris H. Takimoto, M.D., Ph.D., Mark P. Chao, M.D., Ph.D., and Sonali M. Smith, M.D.

From Stanford University, Stanford (R.A., T.T., R.M., I.L.W.), City of Hope, Duarte (L.P.), and Forty Seven, Menlo Park (J.L., J.Y.C., J.-P.V., B.A., J.H., R.M., I.L.W., C.H.T., M.P.C.) — all in California; Sarah Cannon Research Institute—Tennessee Oncology, Nashville (I.F.); University of Alabama at Birmingham, Birmingham (A.F.); Washington University in St. Louis, St. Louis (N.L.B.); Levine Cancer Institute—Atrium Health, Charlotte, NC (N.G.); University of Chicago, Chicago (J.K., S.M.S.); National Cancer Institute, Rockville, MD (M.R.); Dana–Farber Cancer Institute, Boston (A.L.); and University of Oxford, Oxford, United Kingdom (G.P.C.).

- First clinical publication of a CD47 targeting agent – November 1, 2018

Scientific Advisory Board



James Allison, Ph.D.

Chair, Department of Immunology, Director, Parker Institute for Cancer Research, and Executive Director, Immunotherapy Platform at the University of Texas MD Anderson Cancer Center; Winner, 2018 Nobel Prize in Physiology or Medicine



Ronald Levy, M.D.

Professor of Medicine at Stanford University School of Medicine



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



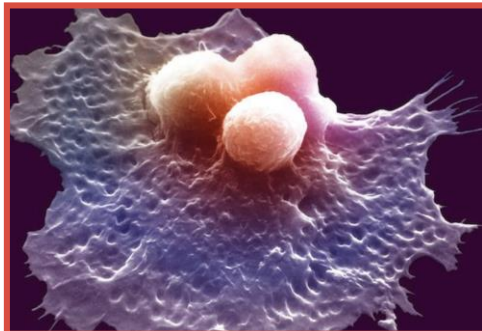
Louis Weiner, M.D.

Director, Georgetown Lombardi Comprehensive Cancer Center and Professor and Chair, Department of Oncology, at Georgetown University Medical Center

Highly Experienced Leadership Team

Management Team:			
Mark McCamish, M.D., Ph.D. <i>President & Chief Executive Officer</i>	   		
Chris Takimoto M.D., Ph.D. <i>Chief Medical Officer</i>	   		
Ann Rhoads, M.B.A. <i>Chief Financial Officer</i>	 		
Craig Gibbs, Ph.D., M.B.A. <i>Chief Business Officer</i>	  		
Norm Kruse, J.D., Ph.D. <i>Chief Patent Counsel</i>	   		
Kyle Elrod <i>SVP of Corporate Planning & Operations</i>	   		
Mark Chao, M.D., Ph.D. <i>VP of Clinical Development</i>			
Jens-Peter Volkmer, M.D. <i>VP of Research & Early Development</i>	 		
Mukul Agarwal, M.S., M.B.A. <i>VP of Corporate Development</i>	    		
Aimee Murphy <i>VP of Clinical Operations</i>	 		
Qinghai Zhao, Ph.D. <i>VP of Technical Development & Manufacturing</i>	   		
Board of Directors:			
Mark McCamish, M.D., Ph.D.	Forty Seven, Inc.	Dennis Henner, Ph.D.	Blackstone Life Sciences (formerly Clarus)
Kristine Ball, C.P.A.	Menlo Therapeutics	Ravi Majeti, M.D., Ph.D.	Stanford School of Medicine
Jeff Bird, M.D., Ph.D.	Sutter Hill Ventures	Irving Weissman, M.D.	Stanford School of Medicine
Ian Clark	Former Genentech CEO		

Targeting Macrophages Leverages the Innate Immune System in the Fight Against Cancer



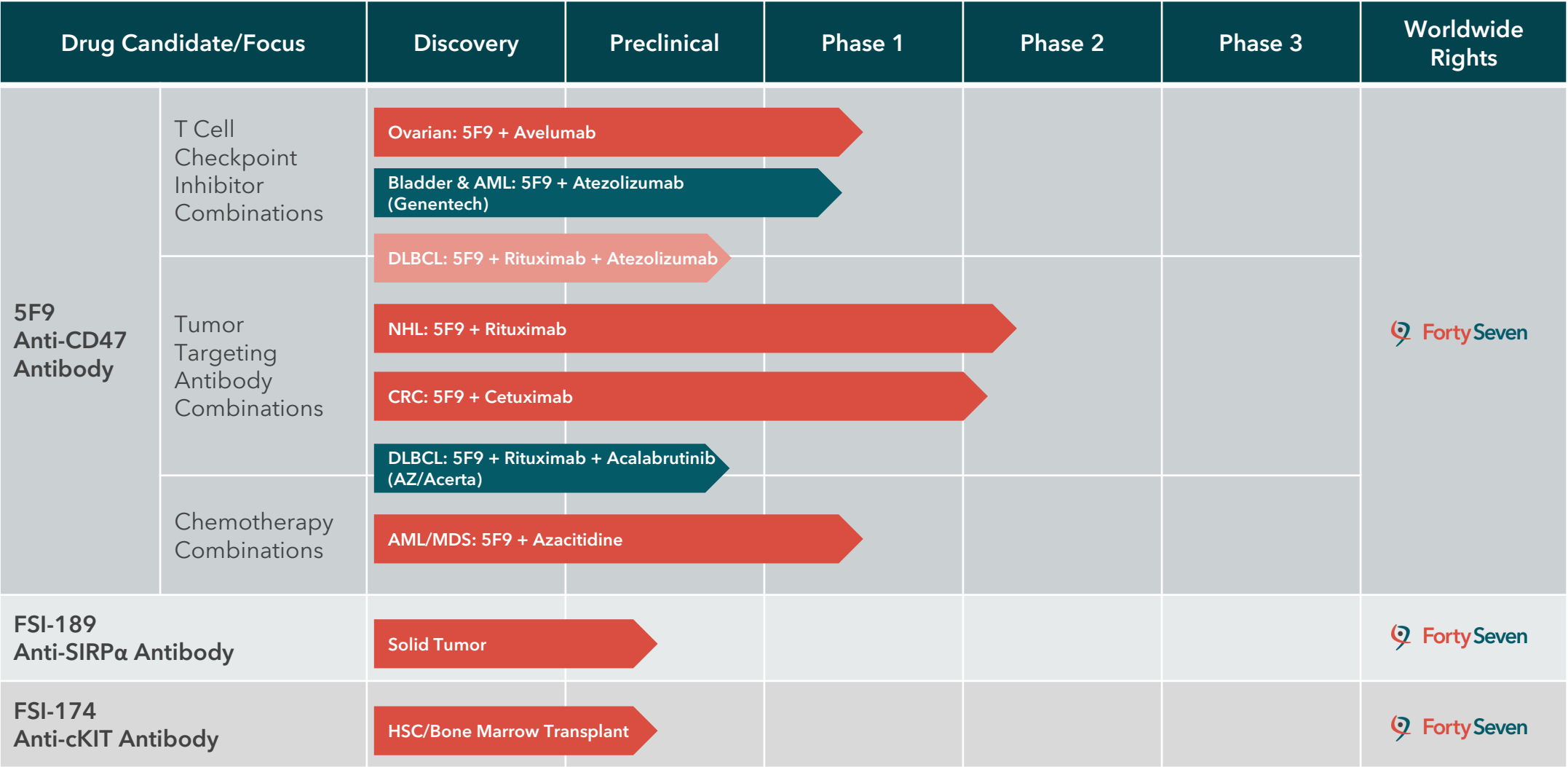
Macrophages are the primary first responders:

- Innate immune cell-type abundant in most tumors
- Phagocytose cells displaying abnormal “eat me” signals, including cancer cells, virally infected cells, and dead or dying cells
- Recruit, activate, and present cancer cell antigens to T cells

	T cells	Macrophages
Immune System Targeted	Adaptive immune system	Innate immune system
Percentage of Tumor Infiltrating Immune Cells	10-20% ¹	20-40% ¹
Cell-Surface Checkpoints and their Receptors	PD-1/PD-L1, CTLA-4	CD47/SIRPα
Applicability to Tumor Targets	Target limited	Not target limited
Dependency	Requires antigen presentation by innate immune cells	Works independently and can recruit adaptive immune cells

¹ Gentles and Alizadeh, Nature Medicine 2015.

Our Pipeline



 Forty Seven Conducting Trial

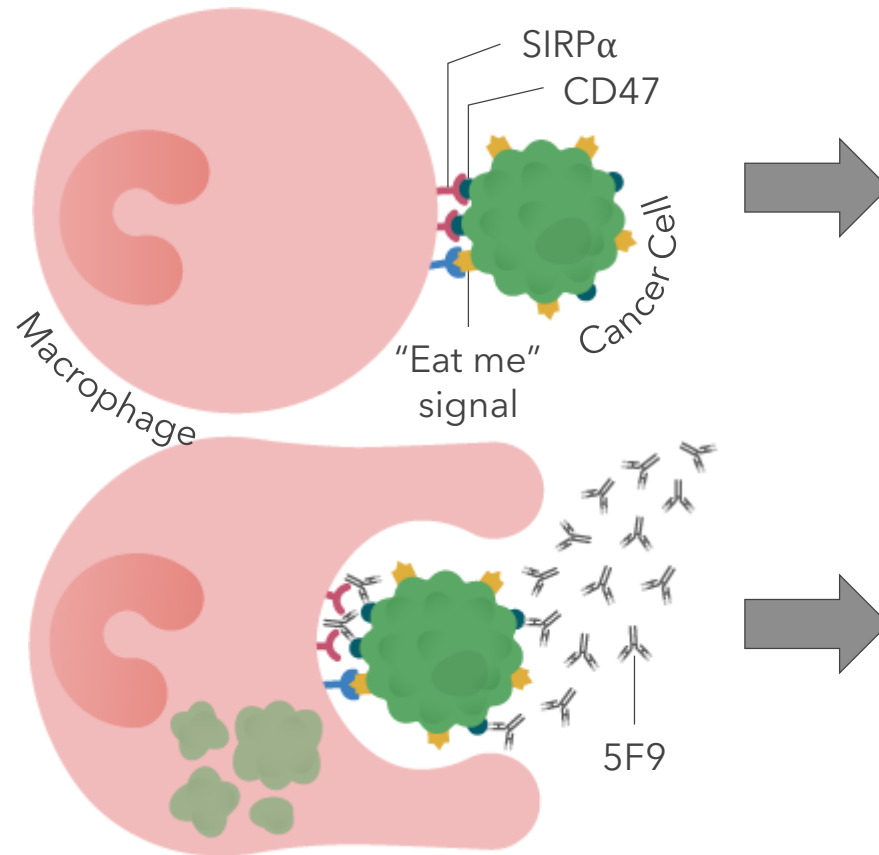
 Planned Forty Seven Trial

 Collaborator Conducting Trial

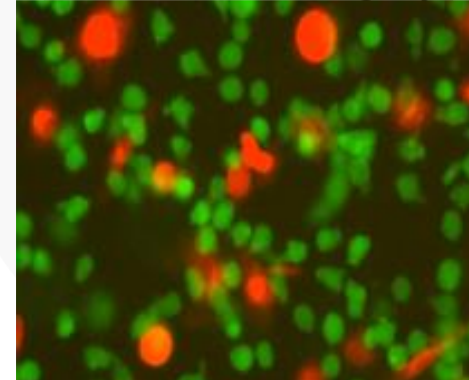
5F9: Anti-CD47 Antibody



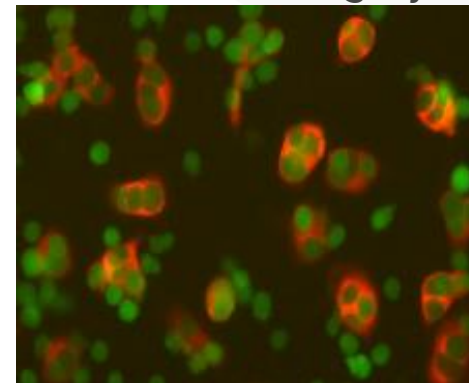
5F9 is a Novel Macrophage Immune Checkpoint Inhibitor Targeting CD47



Control mAb: No Phagocytosis



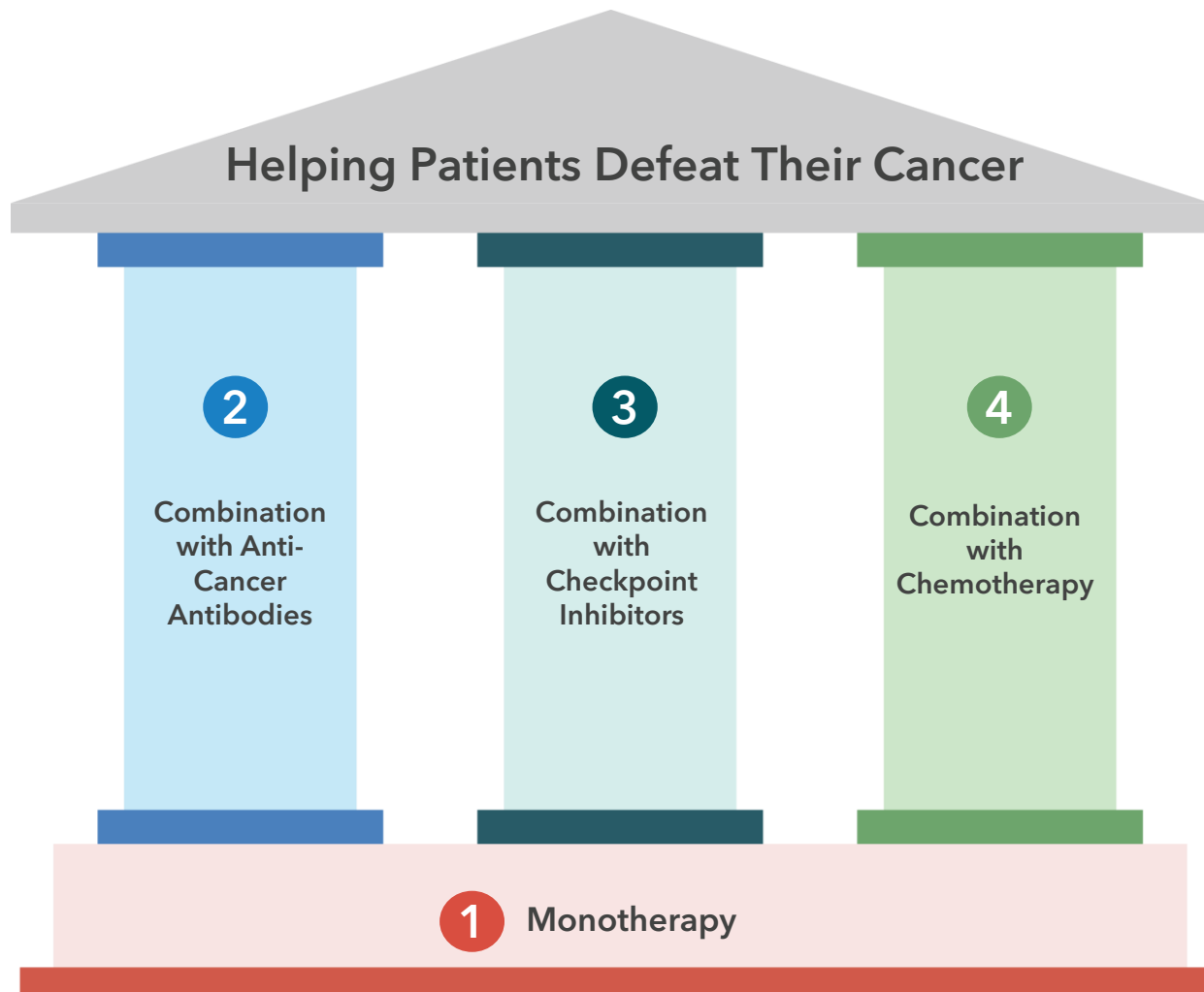
Anti-CD47 mAb: Phagocytosis



Macrophages
Cancer cells

- 5F9 enables macrophages to phagocytose cancer cells by blocking the binding of the "don't eat me" signal CD47 to its receptor SIRPα
- Normal cells are not phagocytosed as they do not express "eat me" signals, except for aged red blood cells
- Additional external "eat me" signals can be provided by cancer-specific antibodies

5F9 Has Applications in Four Treatment Modalities










1 Monotherapy:
Proof-of-concept, facilitates phagocytosis and elimination of tumor cells. Provides foundation for combination therapy

2 In Combination with Anti-Cancer Antibodies:
Synergizes with tumor targeting antibodies in a process called antibody dependent cellular phagocytosis (ADCP)

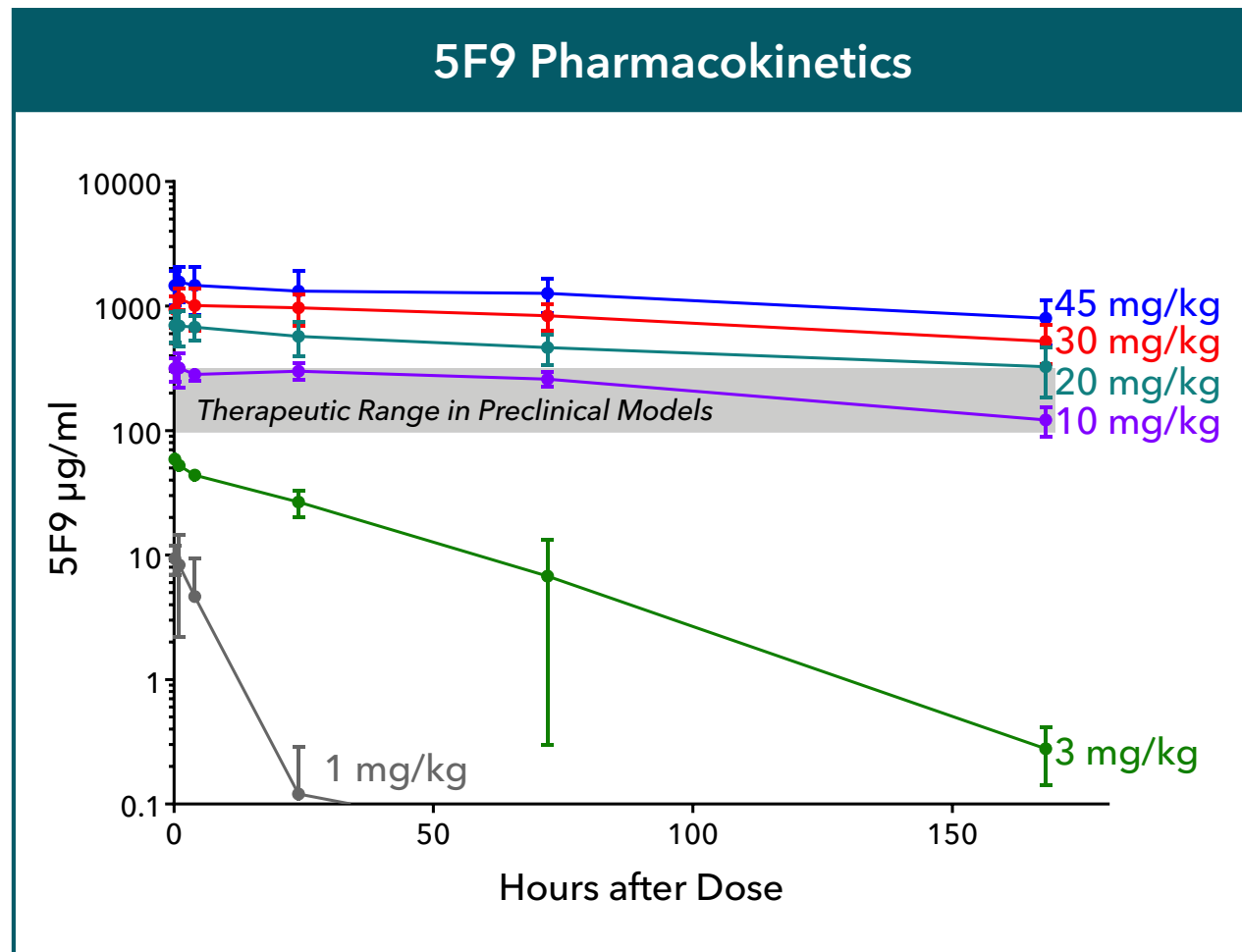
3 In Combination with Checkpoint Inhibitors:
Enhances T cell activation by cross-presentation of cancer cell antigens and amplifies the efficacy of checkpoint inhibitors

4 In Combination with Chemotherapy
Induction of pro-phagocytic signals on tumor cells by chemotherapy facilitates synergistic phagocytosis

Clinical Trials Currently Ongoing or Planned

	Mono-therapy	Combination with T Cell Checkpoint Inhibitors			Combination with Tumor Targeting Antibodies		Combination with Chemotherapy	
Indications	Solid Tumor & Acute Myeloid Leukemia	Ovarian Cancer	Bladder Cancer & Acute Myeloid Leukemia	Non-Hodgkin's Lymphoma	Colorectal Cancer	Non-Hodgkin's Lymphoma	Diffuse Large B-cell Lymphoma	Acute Myeloid Leukemia/ Myelodysplastic Syndrome
Study Stage	Phase 1	Phase 1b	Phase 1b	Phase 1b	Phase 1b/2	Phase 1b/2	Phase 1b	Phase 1b
Therapy	5F9	5F9 + Avelumab	5F9+ Atezolizumab	5F9 + Rituximab + Atezolizumab	5F9 + Cetuximab	5F9 + Rituximab	5F9 + Rituximab + Acalabrutinib	5F9 + Azacitidine
Patient Group	Advanced solid tumors including ovarian cancer and R/R AML	Safety: Advanced solid tumors Expansion: platinum-refractory ovarian cancer (checkpoint naïve)	Bladder cancer and AML	DLBCL	Phase 1b: Advanced solid tumors Phase 2: KRAS WT & mutant advanced CRC	Phase 1b: Relapsed/refractory B-cell NHL Phase 2: R/R indolent lymphoma and R/R diffuse large B-cell lymphoma	R/R diffuse large B-cell lymphoma	Treatment-naïve/unfit for induction chemotherapy
Primary Objective	Safety & tolerability, recommended dose	Safety & tolerability, efficacy	Safety & tolerability, efficacy	Safety & tolerability, efficacy	Safety & tolerability, recommended dose, and efficacy (Phase 2)	Safety & tolerability, recommended dose, and efficacy (Phase 2)	Safety & tolerability, efficacy	Safety & tolerability, efficacy
Status	Enrollment completed	Dosing up to 45 mg/kg plus full dose of avelumab	Anticipated 1H 2019 start	Anticipated 1H 2020 start	Dosing up to 45 mg/kg 5F9 plus full dose cetuximab	Dosing up to 45 mg/kg 5F9 plus full dose rituximab	Anticipated 2H 2019 start	Dosing at 30 mg/kg weekly plus full dose of azacitidine
Clinical Collaborators	--							

5F9 Achieves Target Levels at Clinically Feasible Doses



Forty Seven, Inc. unpublished

Key Points:

- 5F9 can overcome the CD47 antigen sink at 10 mg/kg or higher
- At saturating doses antibody half-life is ~2 weeks
- Free plasma drug levels exceed preclinical activity thresholds (>100 to 250 µg/ml)
- Anti-5F9 antibodies were observed in 15/190 (7.7%) of patients across all studies with no PK or clinical consequences

5F9 Monotherapy is Safe and Well-Tolerated

Solid Tumor Summary (n = 73)			
Adverse Event (AE) Term Patients treated at 10 (3 pts), 20 (39 pts), 30 (25 patients), or 45 (6 patients) mg/kg weekly	AE Grade		
	Any	3	4
Anemia	36 (49%)	8 (11%)	0
Hemagglutination	22 (30%)	1 (1%)	0
Hyperbilirubinemia/Blood bilirubin increased	11 (15%)	3 (4%)	0
Thrombocytopenia	9 (12%)	0	0
Neutropenia	2 (3%)	0	0
Lymphopenia/Lymphocyte count decreased	12 (16%)	7 (10%)	3 (4%)
Fatigue	36 (49%)	0	0
Headache	33 (45%)	1 (1%)	0
Chills	28 (38%)	0	0
Pyrexia	26 (36%)	0	0
Infusion-related reaction	16 (22%)	4 (5%)	0
Nausea	13 (18%)	0	0
Photopsia	7 (10%)	0	0
Back pain	7 (10%)	1 (1%)	0
Myalgia	7 (10%)	0	0
AST elevation	4 (5%)	1 (1%)	1 (1%)
ALT elevation	4 (5%)	0	1 (1%)

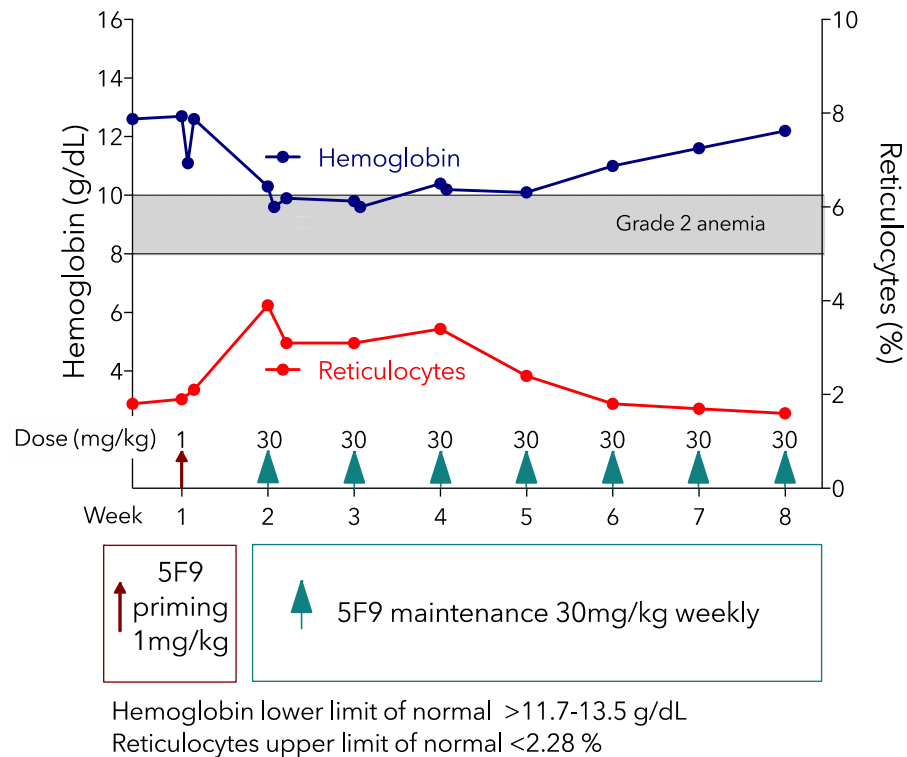
Key Points:

- Expected red blood cell findings are easy to manage using a priming dose regimen*
- Well tolerated at high and extended exposures
- 5F9 AE profile comparable as monotherapy or in combination
- MTD not reached with dose escalation up to 45 mg/kg and >290 patients treated as monotherapy or in combination

* Dose-regimen proprietary to Forty Seven, Inc.

Anemia is Mitigated with a Proprietary Prime and Maintenance Dosing Regimen

Anemia with Compensatory Reticulocytosis



Key Points:

- Proprietary priming dose results in an early, temporary decline in hemoglobin levels corresponding to mild to moderate anemia
- Hemoglobin levels return to baseline even with continued treatment with 5F9 at significantly higher doses (up to 45mg/kg)
- Mild to moderate anemia during the first two weeks of starting therapy
- Associated with a temporary and a reversible reticulocytosis that resolves during the dosing period

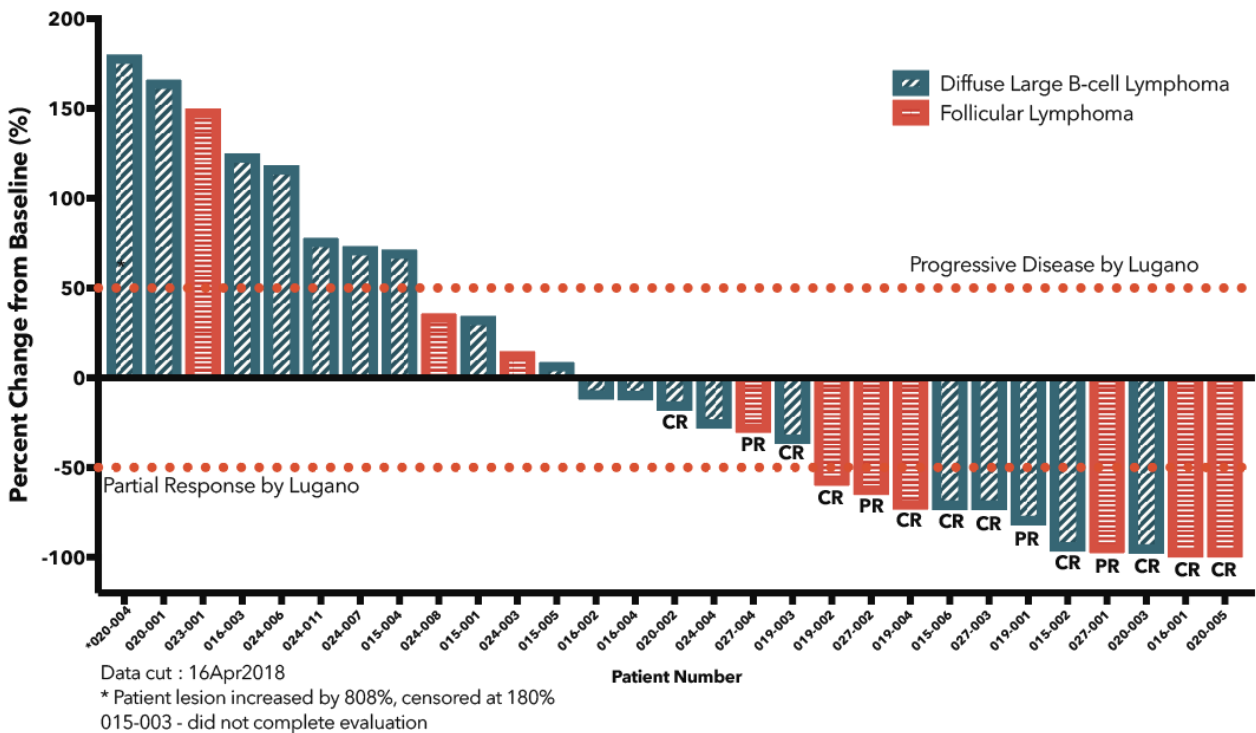
Antitumor Activity Observed with Rituximab Combination in Relapsed or Refractory NHL

Updated data from Feb 2019 data cut

Response	All Patients n=75	DLBCL n=46	Indolent (FL+MZL) n=29
Objective Response Rate (ORR)	49% (37)	39% (18)	66% (19)
Partial Response (PR)	28% (21)	20% (9)	41% (12)
Complete Response (CR)	21% (16)	20% (9)	24% (7)

Data from April 2018 data cut

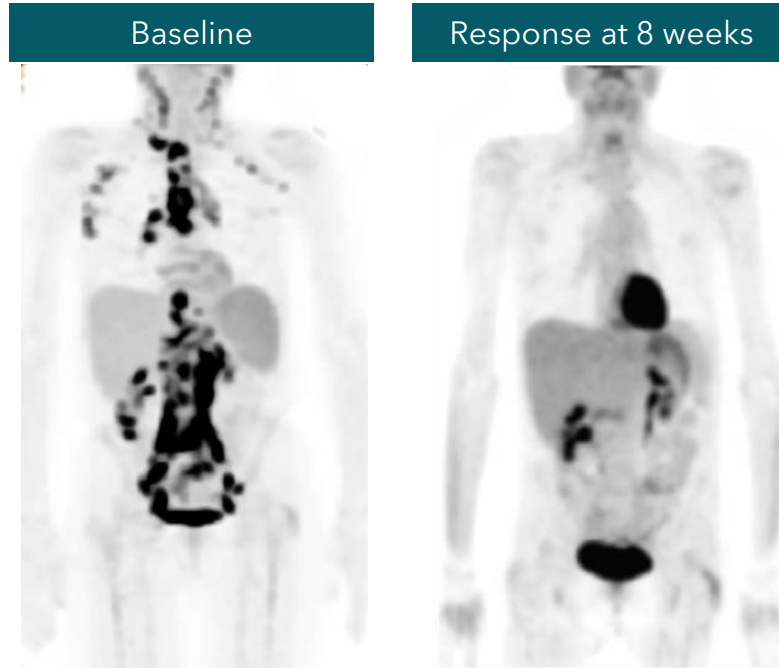
Study 003 (NHL) - Best Percent Change in Target Lesion



- Clinical activity is observed in rituximab-refractory patients
- Phase 1b data was published in the New England Journal of Medicine in November 2018
- Updated data (left) accepted for oral presentation at EHA conference in June 2019

Therapy Eliminates Disease in Refractory Disease Patients

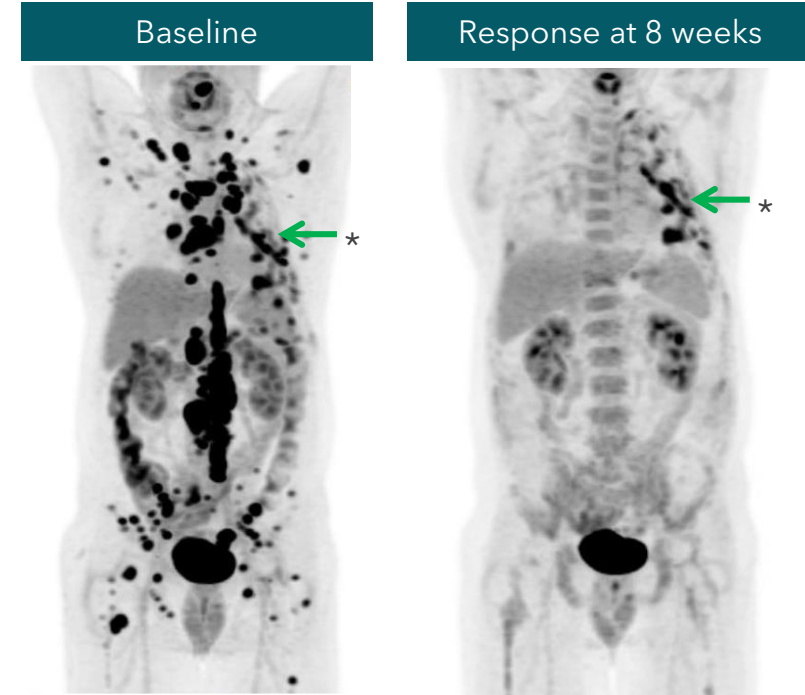
Patient 20-003: FL (CR)



PET scan

- 66F with FL
- Ten prior therapies, bulky disease
- Complete response at 8 weeks

Patient 27-003: DLBCL (CR)



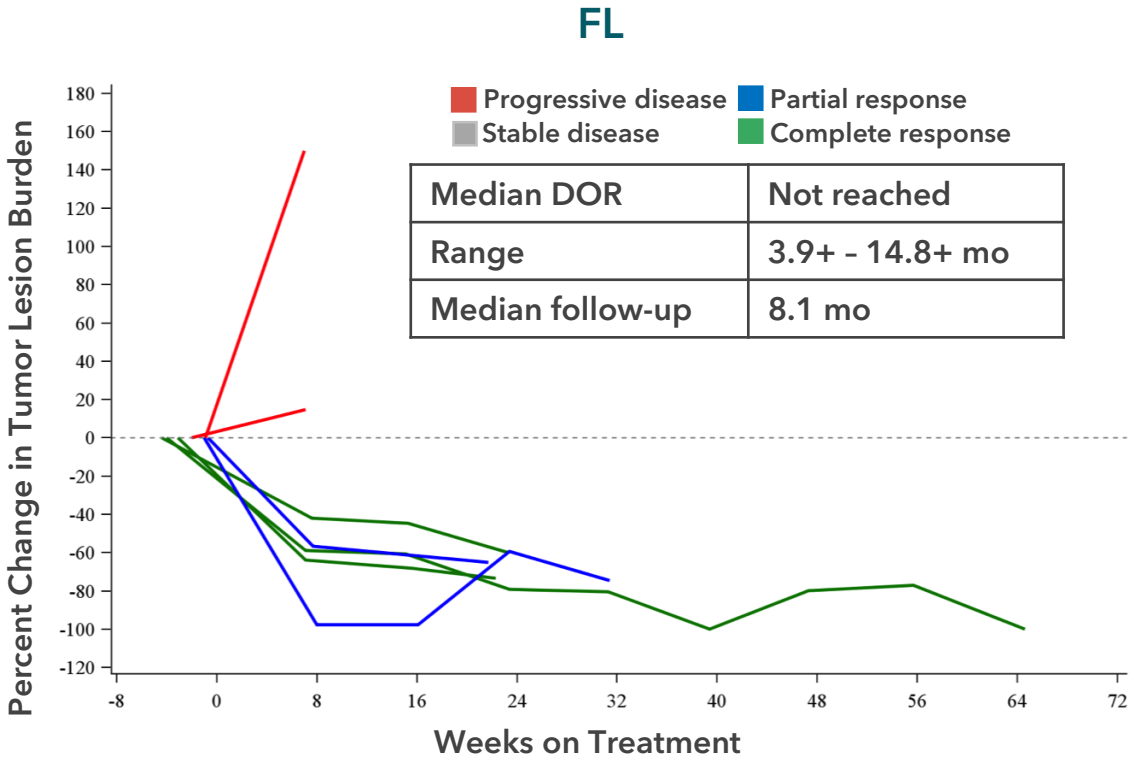
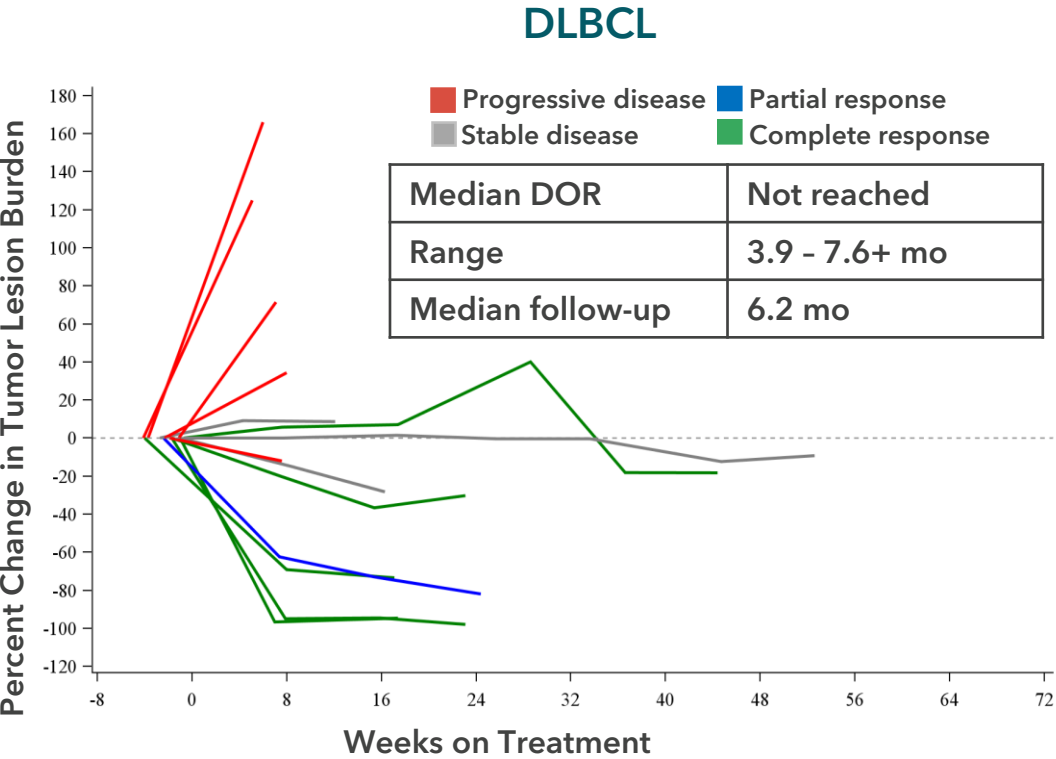
* Hypermetabolic calcified left pleural thickening from prior surgery and not lymphoma

PET scan

Advani et al., ASCO oral presentation 2018

- 56M with primary refractory DLBCL
- Two prior lines of therapy, bulky disease
- Complete response at 8 weeks

Durable Responses Observed in Phase 1b DLBCL and FL Patients¹



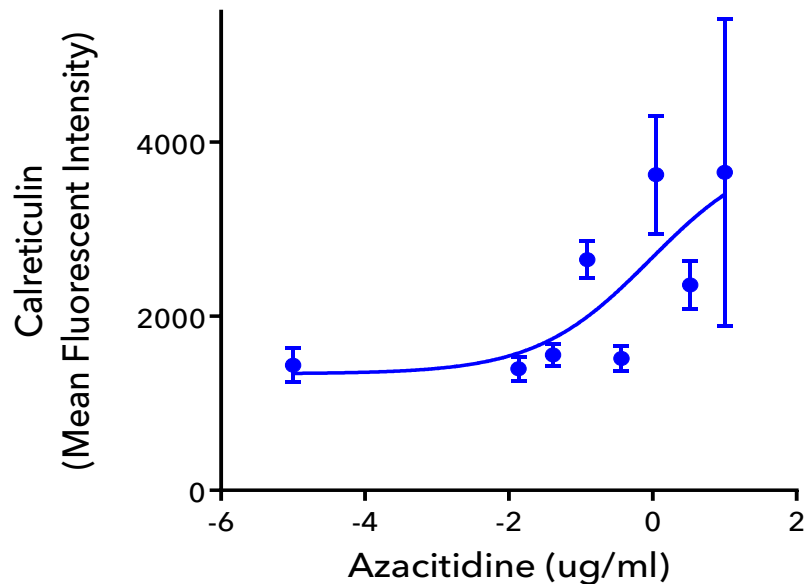
Advani et al., ASCO oral presentation 2018

- As of February 2019 no median duration of response reached for either DLBCL or FL with median follow up of 12 months (DLBCL) and 18 months (FL)
- Durable responses have been observed in patients for >20 months

¹ These plots show data from 22 Phase 1b patients as of April 16, 2018. The plots exclude 8 Phase 2 patients whose responses are included on the previous slide. Of these Phase 2 patients, 3 had objective responses and 5 had progressive disease.

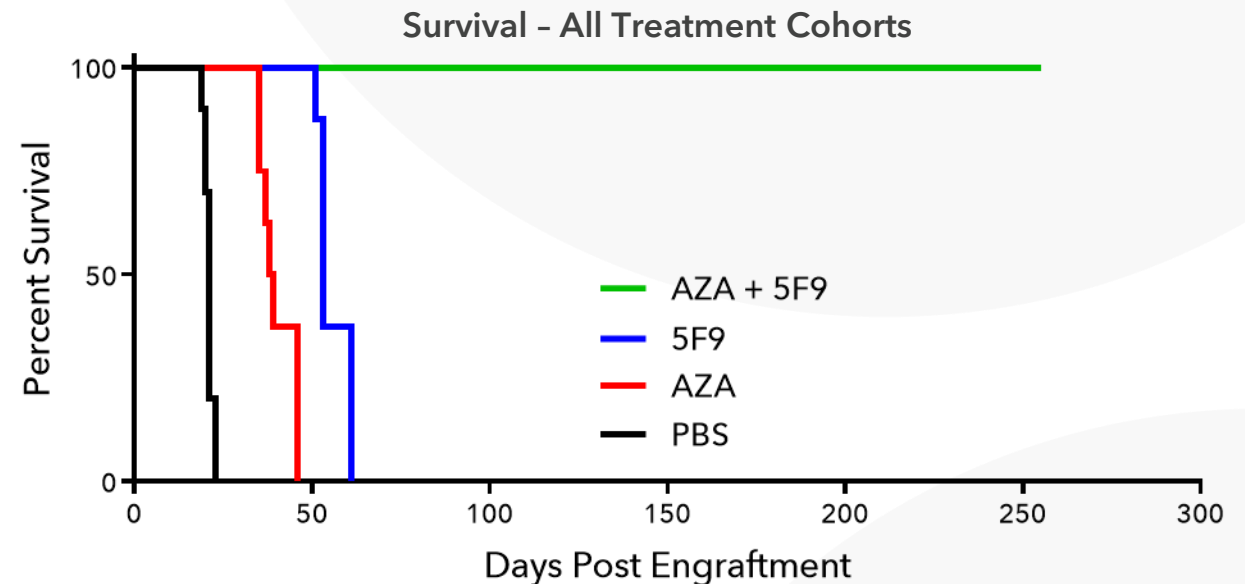
Clinical Monotherapy Activity and Preclinical Combination Data Provide the Foundation for 5F9 + Azacitidine Combination in AML

Azacitidine Increases Expression of the “Eat Me” Signal Calreticulin on AML Cancer Cells



- Azacitidine increases the expression of the pro-phagocytic “eat me” signal calreticulin on the surface of AML cells

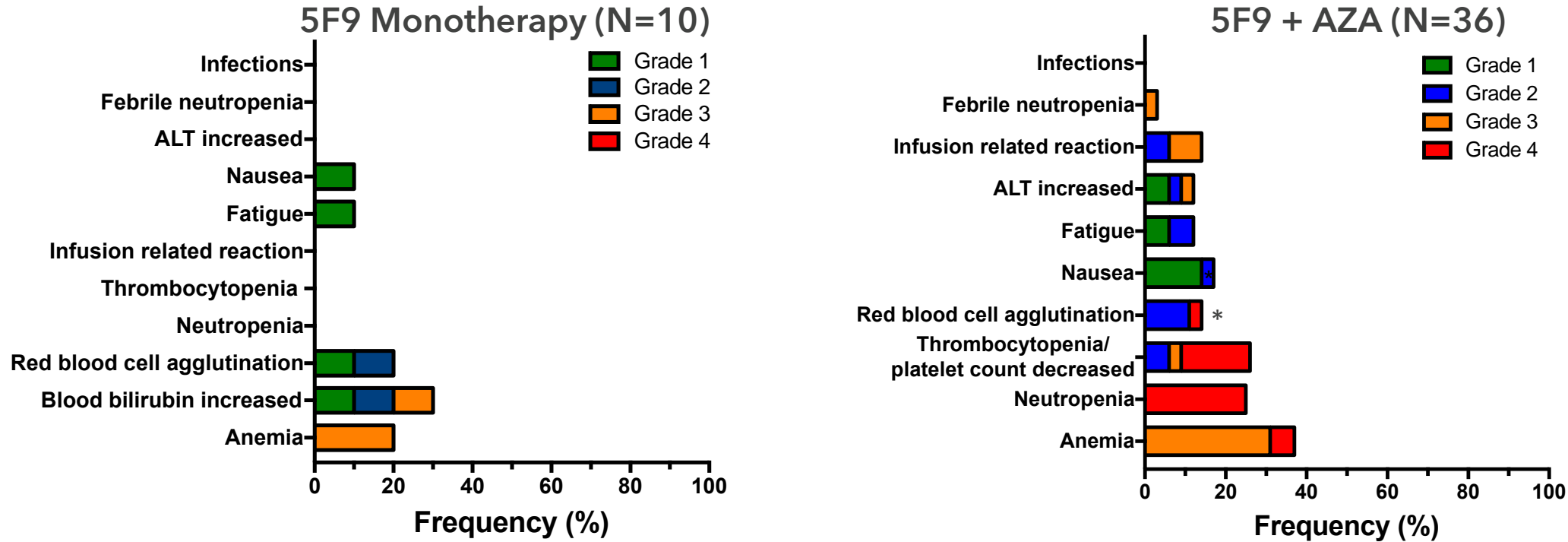
Combination of 5F9 with Azacitidine Enhances Elimination of AML and Prolongs Survival in Xenograft Mouse Model



- Combination of 5F9 and azacitidine (AZA) but not either therapy alone does promote clearance of AML in an aggressive human AML xenograft mouse model
- None of the mice treated with 5F9 - AZA combination had any evidence for relapse when the study was terminated at day 255

5F9 Alone or in Combination with Azacitidine is Well Tolerated

Treatment-related AEs to 5F9 and/or AZA



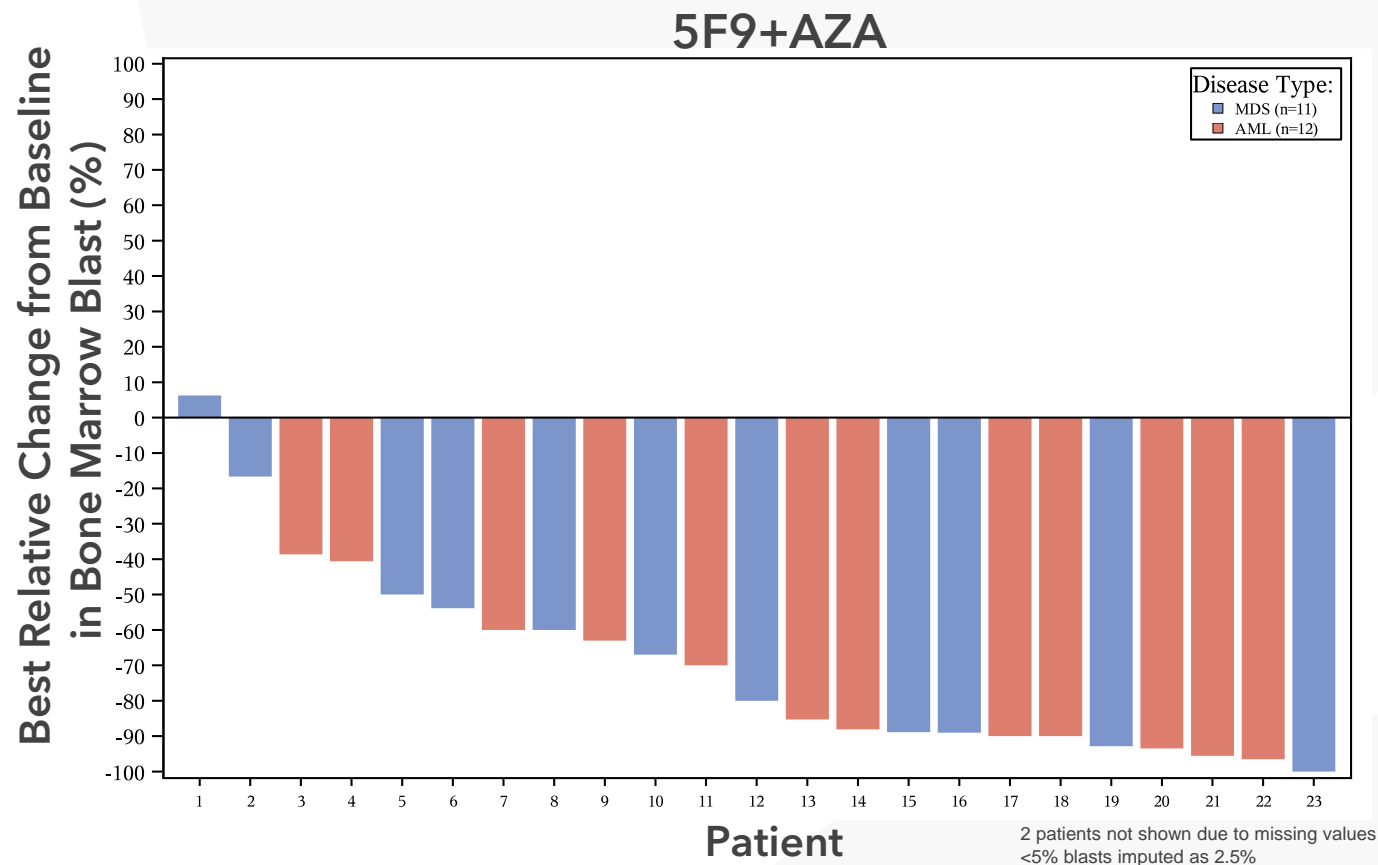
TRAEs > 10% (mono), ≥ 10% (combo), AEs of interest, dose limiting toxicities* (DLTs) regardless of frequency are shown; All patients dosed with 5F9 are shown

- No MTD reached with 5F9 alone or in combo; 5F9+AZA profile consistent with AZA monotherapy
- Treatment discontinuation due to AE occurred in only 1/46 (2%) of patients
 - 1 DLT observed with 5F9+AZA: G4 hemagglutination: patient with IRR symptoms, with peripheral smear agglutination on first dose, resolved after 24 hours, neurologic work-up was negative, patient discontinued therapy
- No significant cytopenias, infections, or autoimmune AEs occurred (most patients cytopenic at baseline)
- No study deaths observed within the first 60 days of treatment for 5F9+AZA

Anti-Leukemic Activity is Observed with 5F9 Monotherapy and in Combination with AZA in AML and MDS

Best Overall Response	R/R AML/MDS 5F9 mono N=10	1L AML 5F9+AZA N=14	1L MDS 5F9+AZA N=11
ORR	1 (10%)	9 (64%)	11 (100%)
CR	0	5 (36%)	6 (55%)
CRi	0	2 (14%)	-
PR	0	0	0
MLFS/ marrow CR	1 (10%)	2 (14%)	4 (36%) 2 with marrow CR+HI
Hematologic improvement (HI)	-	-	1 (9%)
SD	7 (70%)	5 (36%)	0
PD	2 (20%)	0	0

Response assessments per 2017 AML ELN criteria and 2006 IWG MDS criteria; Patients with at least one post-treatment response assessment are shown
“-” not applicable

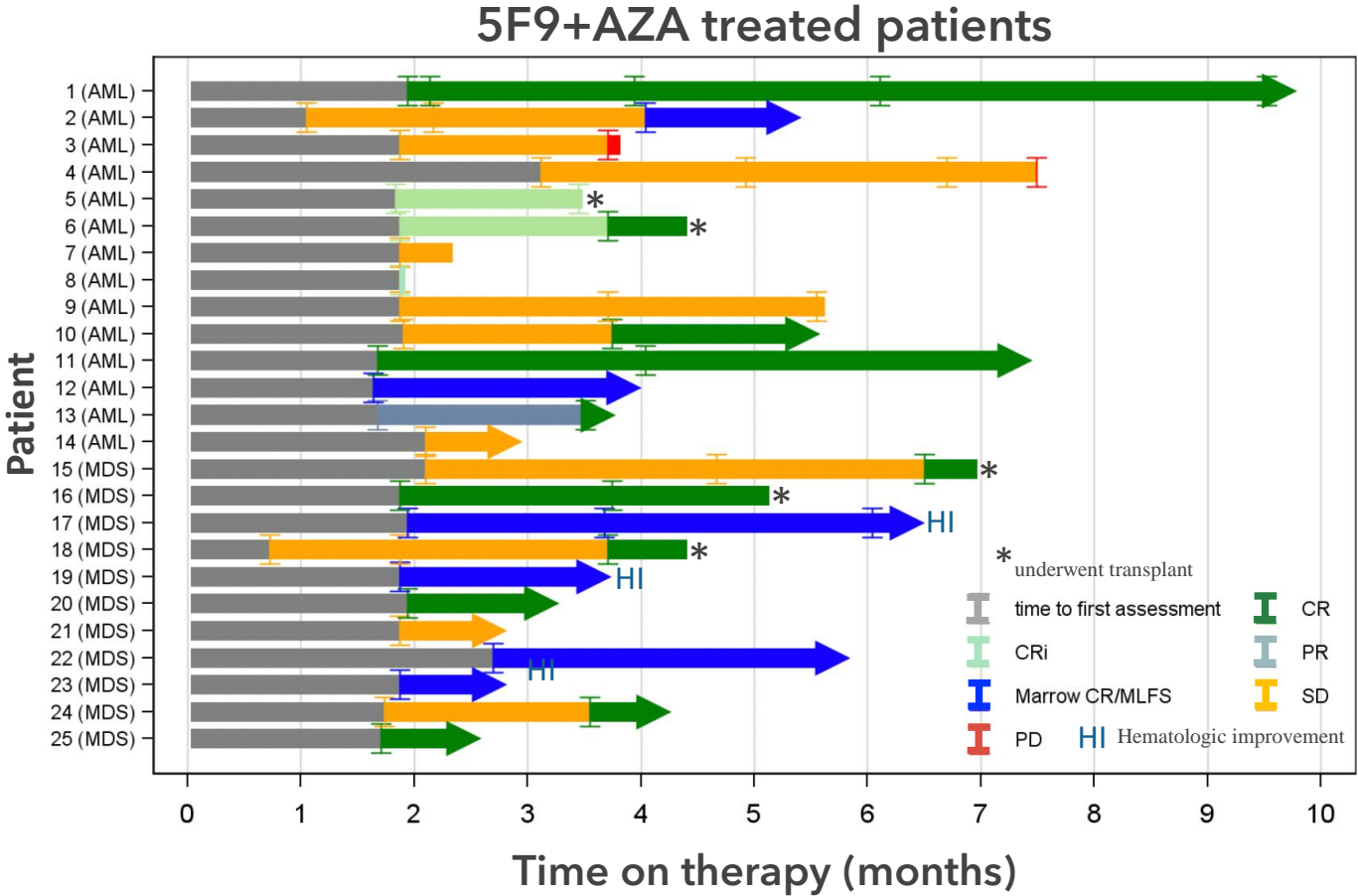


- 5F9 monotherapy has an ORR of 10% in r/r AML/MDS
- 5F9+AZA has an ORR of 100% in MDS, 64% in AML which compares favorably to AZA monotherapy ORR
Median time to response is more rapid (1.9 months) than AZA alone

Deep and Potential Durable Responses Seen in 5F9 + AZA Treated Patients

Parameter	1L AML N=14	1L MDS N=11
RBC transfusion independence	9/14 (64%)	-
Complete cytogenetic response in responders*	2/7 (29%)	3/7 (43%)
MRD negativity in responders	3/9 (33%)	2/10 (20%)
Median duration of response (months)	NR (0.03+ - 8.3+)	NR (0.5+ - 4.3+)
Median follow-up [range] (months)	3.8 (1.9 - 10.3)	3.7 (2.5 - 6.8)

Minimal residual disease (MRD) was evaluated by multiparameter flow cytometry
Hematologic improvement (HI-E, HI-P, HI-N) defined per 2006 IWG MDS criteria
Cytogenetic response defined per 2003 and 2006 IWG criteria; NE: not reached
*Cytogenetic responses shown for all responding patients with abnormal cytogenetics at baseline
“-” not applicable



- No responding patient has relapsed or progressed on 5F9 + AZA
- Multiple patients have improved responses over time
- MRD negativity has been observed (time to MRD negativity ranged from 1.7 to 6.1 months)
- 5/20 (25%) of responding patients have successfully received an allogeneic stem cell transplant
- The longest patient in response is in CR 9+ months on therapy and ongoing

Catalyst Events Expected in 2019 – 2020

	Indication (Study Stage)	Therapy	Presented	Projected				
			2018	Q1 2019	Q2 2019	Q3 2019	Q4 2019	2020
Monotherapy	Solid Tumor (Phase 1)	5F9	ASCO: Safety + Ovarian Initial Efficacy					
	Acute Myeloid Leukemia (Phase 1)	5F9	EHA: Monotherapy Safety					
Combination with Cancer- Specific Antibodies	Non-Hodgkin's Lymphoma (Phase 1b/2)	5F9 + rituximab	ASCO: Phase 1b Safety + Efficacy			EHA: Phase 2 Efficacy (DLBCL & Indolent Lymphoma)		
	Colorectal Cancer (Phase 1b/2)	5F9 + cetuximab					Phase 1b Safety + Phase 2 Efficacy	
Combination with Checkpoint Inhibitors	Ovarian Cancer (Phase 1b)	5F9 + avelumab					Phase 1b Safety + Efficacy	
	Bladder Cancer (Phase 1b)	5F9+ atezolizumab						Phase 1b Safety + Efficacy
	Acute Myeloid Leukemia (Phase 1b)	5F9+ atezolizumab						Phase 1b Safety + Efficacy
Combination with Chemotherapy	Acute Myeloid Leukemia/ Myelodysplastic Syndrome (Phase 1b)	5F9+ azacitidine				ASCO & EHA: Phase 1b Safety + Efficacy		

Our Intellectual Property Rights Covering CD47 and Other Immunomodulatory Compounds

- We have a license to over 100 issued patents worldwide including 25 US patents covering CD47 related inventions including 5F9
- 5F9 is protected by multiple patent positions
 - Composition of matter: drug product and formulations
 - Methods of use: monotherapy and combinations
 - Methods of use: proprietary prime → maintenance dosing
- As of December 2018, we have a license to 102 issued and 124 pending patents worldwide, including 26 US issued patents
- In July 2018, we announced a settlement and license agreement with Synthon Biopharmaceuticals
 - Forty Seven to withdraw ongoing oppositions and challenges in the USPTO and EPO against patents licensed (from SSB) by Synthon
 - Provides a non-exclusive, worldwide sub-license to commercialize anti-CD47 antibodies including 5F9, to treat cancer in combination with certain other antibodies, such as cetuximab and rituximab
- In August 2018, the European Patent Office (EPO) Opposition Division ruled in favor of Forty Seven, rejecting challenges to our licensed European patent (EP '512)
 - EPO decision reaffirms Forty Seven's patent protection for the use of 5F9 in Europe and generally covers the use of CD47 antibodies (not just 5F9) to treat cancer by targeting cancer cells for phagocytosis



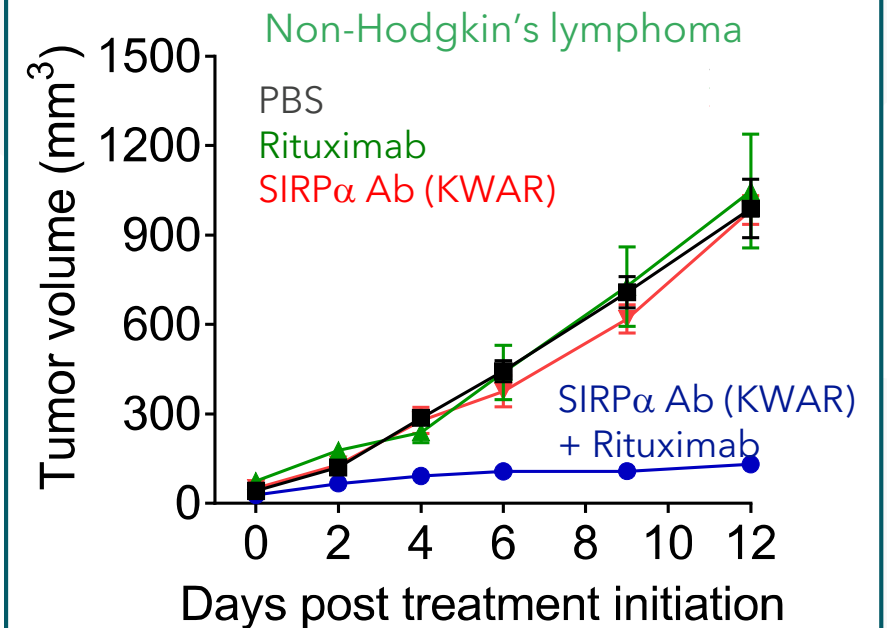
FSI-189: Anti-SIRP α Antibody Program



FSI-189 Anti-SIRP α Antibody Program: Potential Next Generation Antibody in Oncology and Non-Oncology Indications

Target	<ul style="list-style-type: none"> ○ SIRPα, CD172a
MOA	<ul style="list-style-type: none"> ○ Blockade of CD47-SIRPα macrophage immune checkpoint ○ Enhanced target cell phagocytosis <u>in combination with targeted antibodies</u>
Indication	<ul style="list-style-type: none"> ○ Oncology ○ Non-oncology indications: stem cell transplantation in conjunction with cKIT antibody, infectious disease, cardiovascular disease
Addressed Need	<ul style="list-style-type: none"> ○ Smaller antigen sink, potential for lower dose ○ Potential for improved dosing convenience ○ Lower cost of goods ○ Lack of RBC binding, reduced potential for anemia
Development Status	<ul style="list-style-type: none"> ○ Preclinical POC established ○ Lead candidate selected ○ Cell line development ongoing ○ IND anticipated Q1 2020
IP	<ul style="list-style-type: none"> ○ Composition of matter patent filed
Competition	<ul style="list-style-type: none"> ○ Two anti-SIRPα mAb's (Celgene CC-95251, OSE / Boehringer Ingelheim OSE-172) anticipated to enter Phase 1 Q1 2019

Combination of SIRP α Antibody with Rituximab Enhances Phagocytic Potency and Prolongs Survival in Mouse Model



Ring et al., PNAS 2017

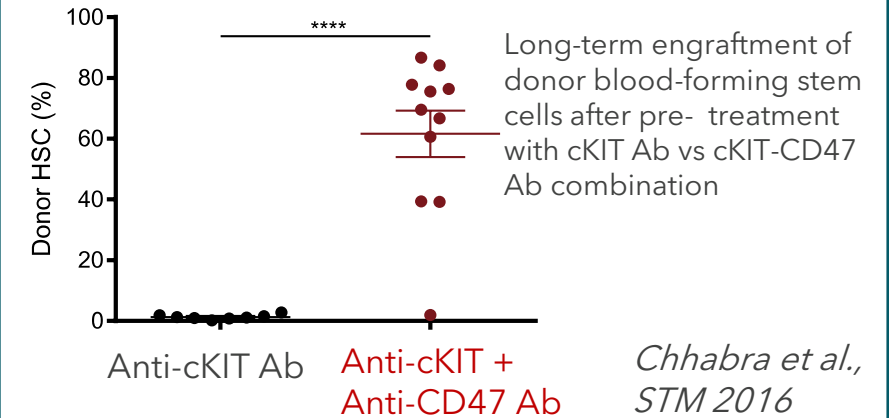
FSI-174: Anti-cKIT Antibody Program



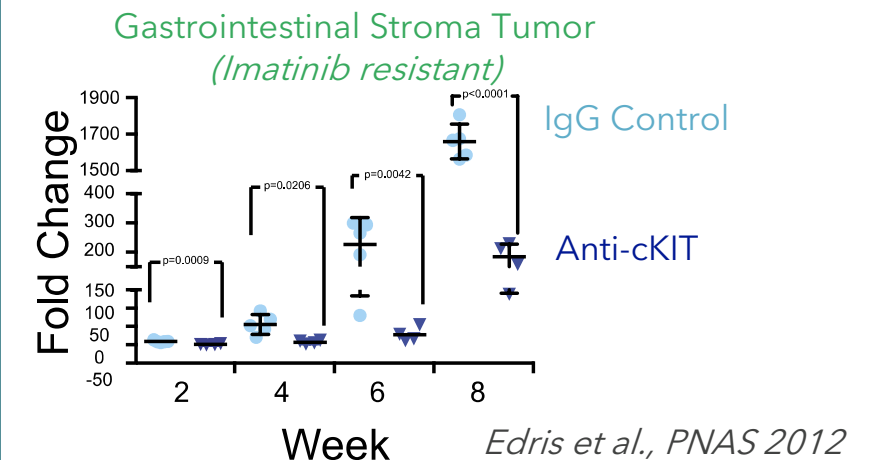
FSI-174 Anti-cKIT Antibody Program

Target	<ul style="list-style-type: none"> cKIT, CD117, stem cell growth factor receptor
MOA	<ul style="list-style-type: none"> Blockade of stem cell factor signaling Depletion of cKIT expressing cells
Indication	<ul style="list-style-type: none"> Hematopoietic stem cell (HSC) and bone marrow transplantation <ul style="list-style-type: none"> Genetic disorders Leukemia & lymphoma Autoimmune diseases Organ transplantation Oncology: cKIT expressing cancers, e.g. leukemia, melanoma, renal cell cancer, gastrointestinal stroma tumor
Addressed Need	<ul style="list-style-type: none"> Improved conditioning regimens (chemo and radiation free) <ul style="list-style-type: none"> Potential for lower incidence of morbidity and mortality Expanded patient populations and indications
Development Status	<ul style="list-style-type: none"> Preclinical POC established for both indications Cell line development initiated June 2018 IND enabling studies in 2019
IP	<ul style="list-style-type: none"> Methods patent for cKIT Ab and cKIT + CD47 Ab filed Antibody compositions for Anti-cKIT and Anti-CD47 Abs
Competition	<ul style="list-style-type: none"> Stanford sponsored trial in SCID patients with AMG191 (cKIT Ab with dead Fc) cKIT antibody drug conjugate in preclinical development by Magenta Therapeutics

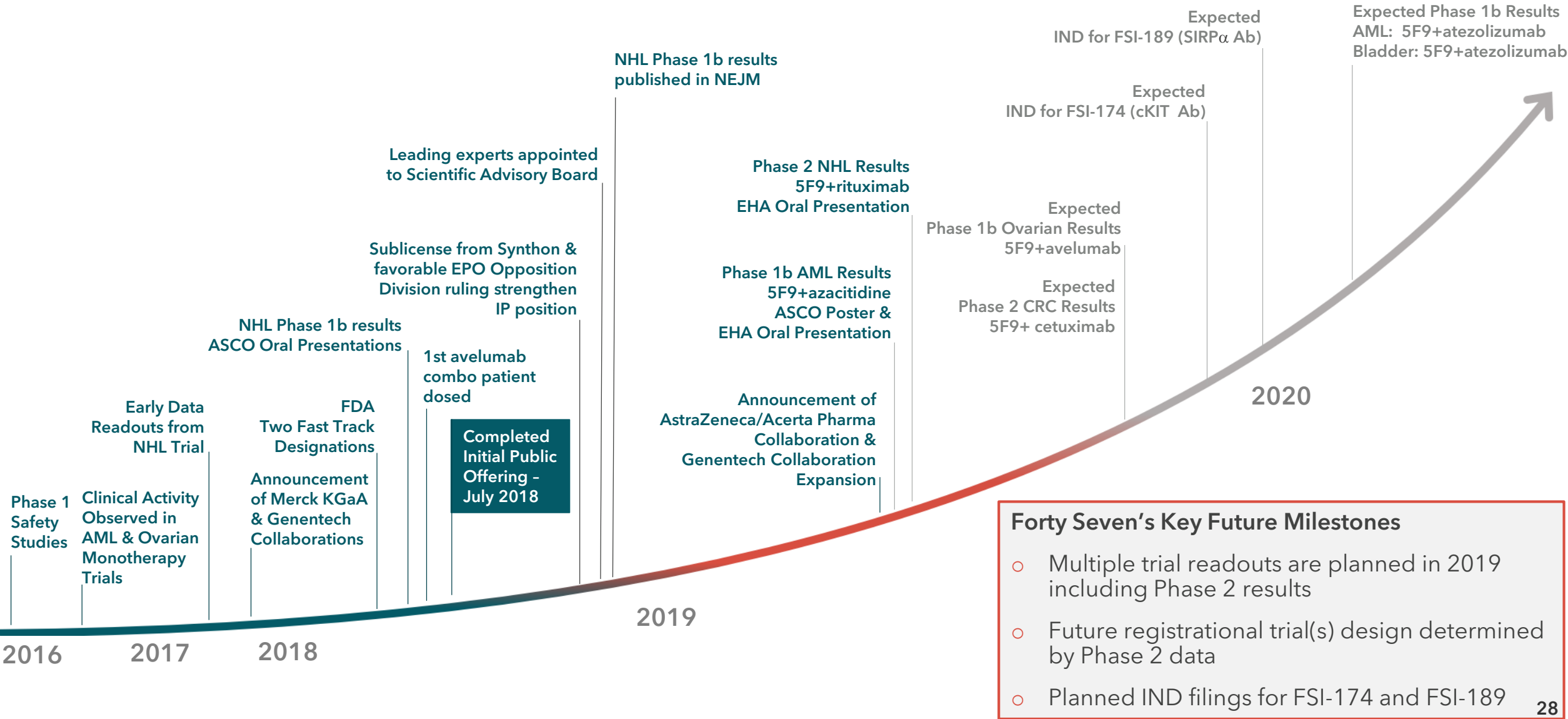
Combination of cKIT and CD47 Antibodies Enables Transplantation of Blood-Forming Stem Cells in Mouse Model



cKIT Antibody Inhibits Tumor Growth in Mouse Model



Forty Seven and 5F9 Development Progress and Future Plans



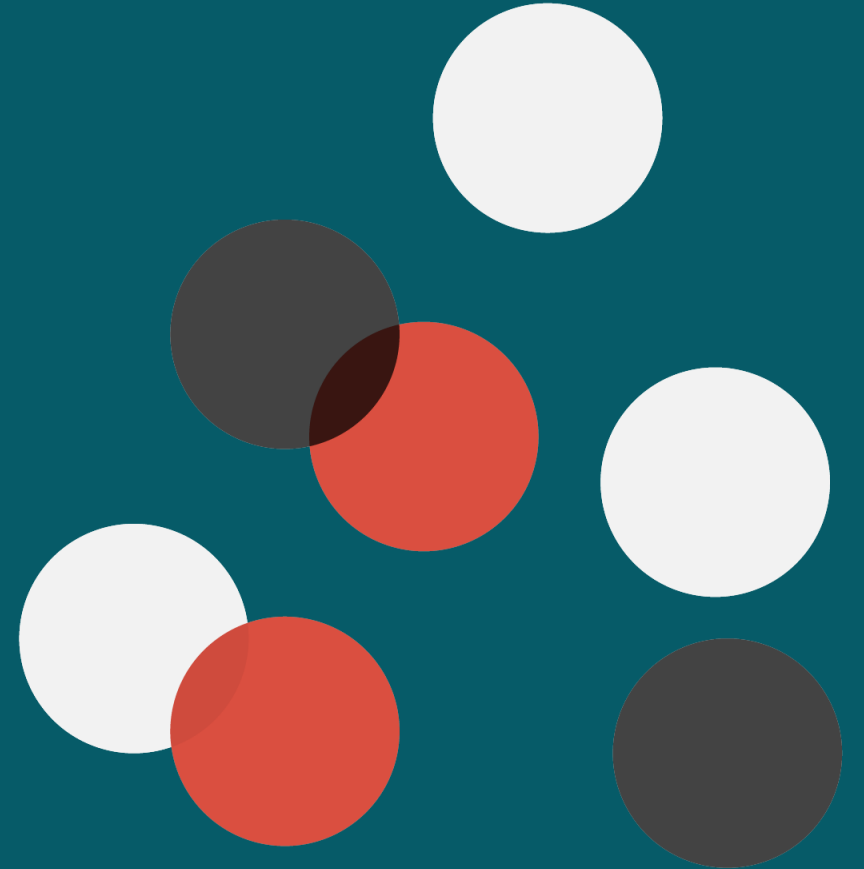
Forty Seven's Key Future Milestones

- Multiple trial readouts are planned in 2019 including Phase 2 results
- Future registrational trial(s) design determined by Phase 2 data
- Planned IND filings for FSI-174 and FSI-189

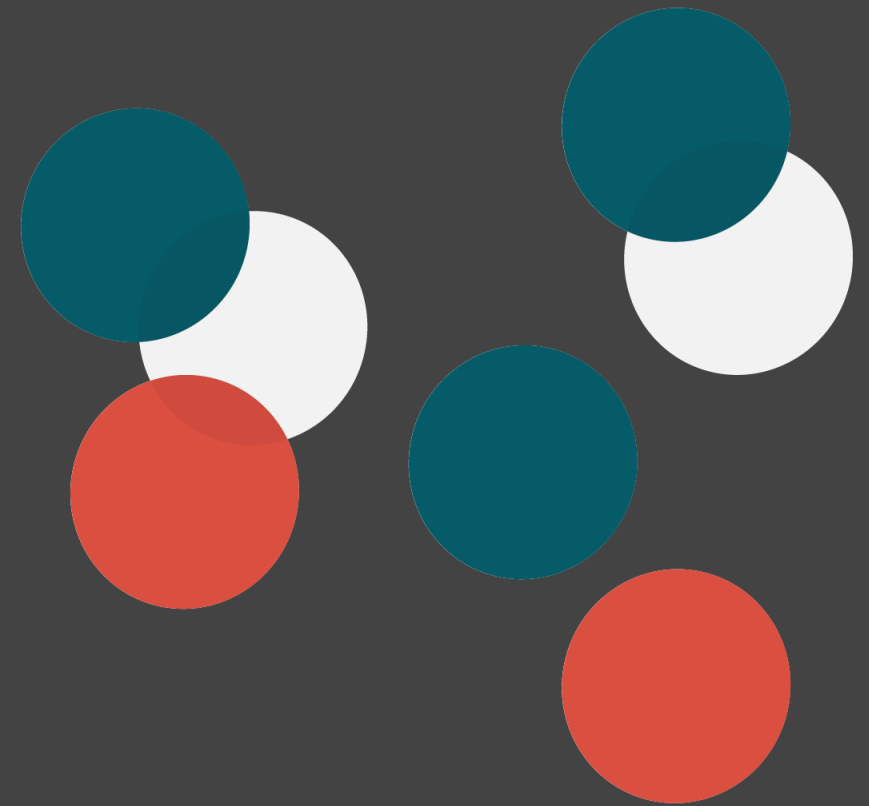


NASDAQ: FTSV
Forty Seven

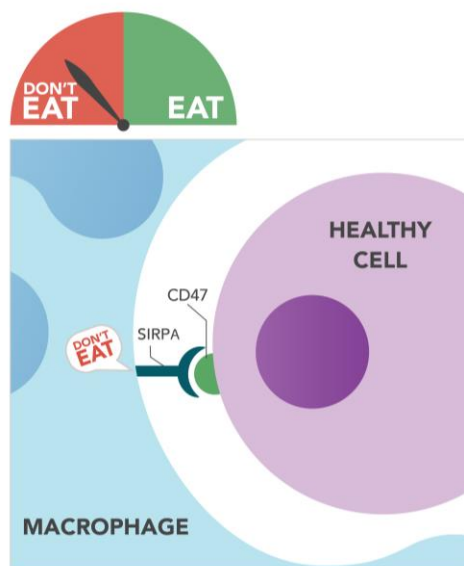
Helping Patients Defeat Their Cancer
BOLD | HUMBLE | INQUISITIVE | PASSION TO ACTION



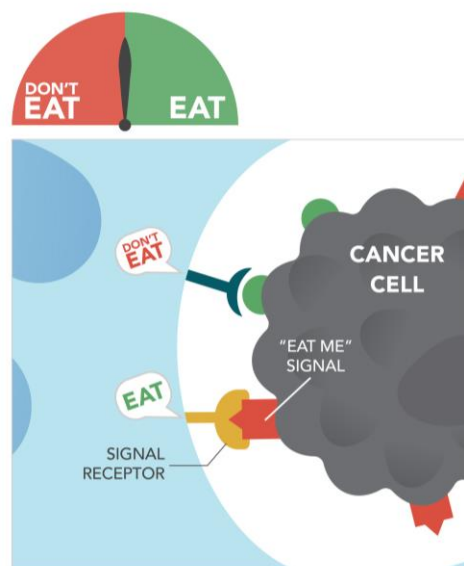
Back-Up Slides



Anti-Cancer efficacy of 5F9 involves tipping the balance between "eat me" and "don't eat me" signals



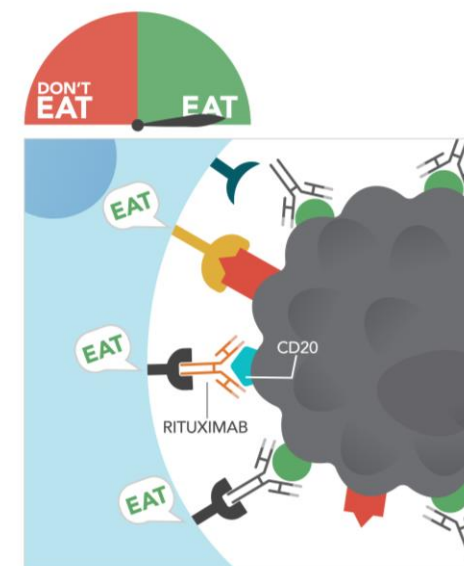
Macrophage with
Healthy Cell



Macrophage with
Cancer Cell



Macrophage with
Cancer Cell and
5F9



Macrophage with
Cancer Cell and
5F9 + rituximab

Forty Seven Differentiated from Competitors in Clinical Development



Compound	5F9	CC-90002	TTI-621	TTI-622	ALX148	SRF231	IBI188	AO-176	TG-1801 (NI-1701)	TJC4
Molecule	mAb	mAb	WT SIRP α fusion protein	WT SIRP α fusion protein	High affinity SIRP α fusion protein	mAb	mAb	mAb	Bi-specific Ab CD47/CD19	mAb
Class	IgG4	IgG4	IgG1	IgG4	Inactive Fc	IgG4	IgG4	IgG2	IgG1	--
Clinical Start Date	August 2014 first-in-clinic	March 2015	January 2016	May 2018	February 2017	March 2018	January 2019	February 2019	February 2019	IND approved, Est. Start 2Q 2019
Study Stage	Phase 2	Phase 1b	Phase 1a/b	Phase 1a/b	Phase 1	Phase 1 ² <i>Deprioritized</i>	Phase 1 (China)	Phase 1	Phase 1	IND
Clinical Trials	8	1	2	1	1	1	1	1	1	--
Partner(s)	AstraZeneca/Acerta, Genentech, Merck KGaA, Lilly	N/A	N/A	N/A	N/A	N/A	N/A	N/A	TG Therapeutics	N/A



- Most advanced program
 - First-in-clinic with initial trial started in August 2014
 - 8 trials ongoing with >290 patients dosed for up to 2 years
 - 4 pharma collaborations
 - Comprehensive intellectual property
 - Efficient manufacturing process; relationship with Lonza
- 5F9 has the IgG4 subclass
 - Allows for safe dosing by avoiding toxicity to normal tissues caused by antibody-dependent cell-mediated cytotoxicity and complement dependent cytotoxicity⁴
- Proprietary dosing regimen
 - Mitigates transient anemia and enables high maintenance dose levels

1. Formerly Alexo Therapeutics
2. Surface Oncology reported 2 hematologic DLTs (Dec 2018) and a decision not to open expansion cohorts. The program was deprioritized.
3. Formerly Tioma, formerly Vasculox
4. Davies and Sutton, Immunology Reviews, 2015

Poster Presentations at the American Society of Hematology (ASH) Annual Meeting, December 2018

RBC-Specific CD47 Pruning Confers Protection and Underlies the Transient Anemia in Patients Treated with Anti-CD47 Antibody 5F9

- 5F9 priming dose not only triggered clearance of a subset of aged RBCs, but also resulted in a near complete loss of CD47 on RBCs
- CD47 loss only occurred on RBCs but not WBCs and AML cancer cells
- Similar phenomenon exhibited in mouse models
- CD47 pruning is Fc-independent
- Provides fundamental insight into the mechanism underlying how anti-CD47 Abs are tolerated without impairing therapeutic efficacy
- Loss of CD47 on RBCs after the priming dose suggests that the potential risk of CD47 Ab-mediated RBC agglutination reduced during maintenance dosing

**Abstract number 2327 - ASH Annual Meeting, December 2018*

Combination Treatment with 5F9 and Azacitidine Enhances Phagocytic Elimination of Acute Myeloid Leukemia

- Azacitidine can increase the “eat me” signal calreticulin on AML cancer cells
- Combination of 5F9 and azacitidine enhances phagocytic clearance of AML cells by human macrophages in vitro
- Combination of 5F9 and azacitidine enhances phagocytic clearance of AML cells in vivo and prolongs survival compared to single agent treatment
- A clinical trial with this combination in patients with AML is currently ongoing (NCT03248479)

** Abstract number 2729 - ASH Annual Meeting, December 2018*