

ALX ΦNCOLOGY

March 13, 2023

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This presentation contains forward-looking statements that involve substantial risks and uncertainties. Forward-looking statements include statements regarding future results of operations and financial position, business strategy, product candidates, planned preclinical studies and clinical trials, results of clinical trials, research and development costs, regulatory approvals, timing and likelihood of success, plans and objectives of management for future operations, as well as statements regarding industry trends. Such forward-looking statements are based on ALX Oncology's beliefs and assumptions and on information currently available to it on the date of this presentation. Forward-looking statements may involve known and unknown risks, uncertainties and other factors that may cause ALX Oncology's actual results, performance or achievements to be materially different from those expressed or implied by the forward-looking statements. These and other risks are described more fully in ALX Oncology's filings with the Securities and Exchange Commission ("SEC"), including ALX Oncology's Annual Report on Form 10-K and other documents ALX Oncology files with the SEC from time to time. Except to the extent required by law, ALX Oncology undertakes no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

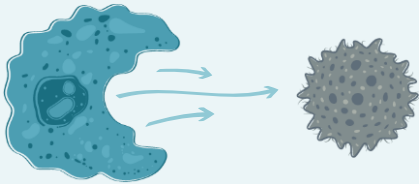
This presentation concerns product candidates that are under clinical investigation, and which have not yet been approved for marketing by the U.S. Food and Drug Administration. These product candidates are currently limited by federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

This presentation also contains estimates and other statistical data made by independent parties and by ALX Oncology relating to market size and growth and other industry data. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of ALX Oncology's future performance and the future performance of the markets in which it operates are necessarily subject to a high degree of uncertainty and risk.

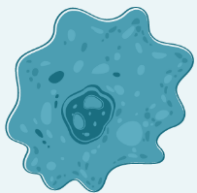
ALX ONCOLOGY ADVANCING A HIGHLY DIFFERENTIATED IMMUNO-ONCOLOGY PIPELINE LED BY EVORPACEPT, A CD47 INNATE IMMUNE SYSTEM CHECKPOINT INHIBITOR

Evorpaccept: designed to maximize a patient's immune response

Exclusively inhibits CD47, a key mechanism tumors use to evade the immune system

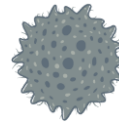


Acts on CD47 as a checkpoint inhibitor, not as a tumor associated antigen target.



Activates immune system without eliminating healthy cells that express CD47.

Demonstrated activity in both solid & liquid tumors



- Three ongoing randomized Phase 2 studies underway in solid tumors.



- Two studies underway in combination with antibody drug conjugates (ADCs).
- Hematology studies ongoing.

Potential to be a best-in-class cornerstone treatment for a broad range of cancers

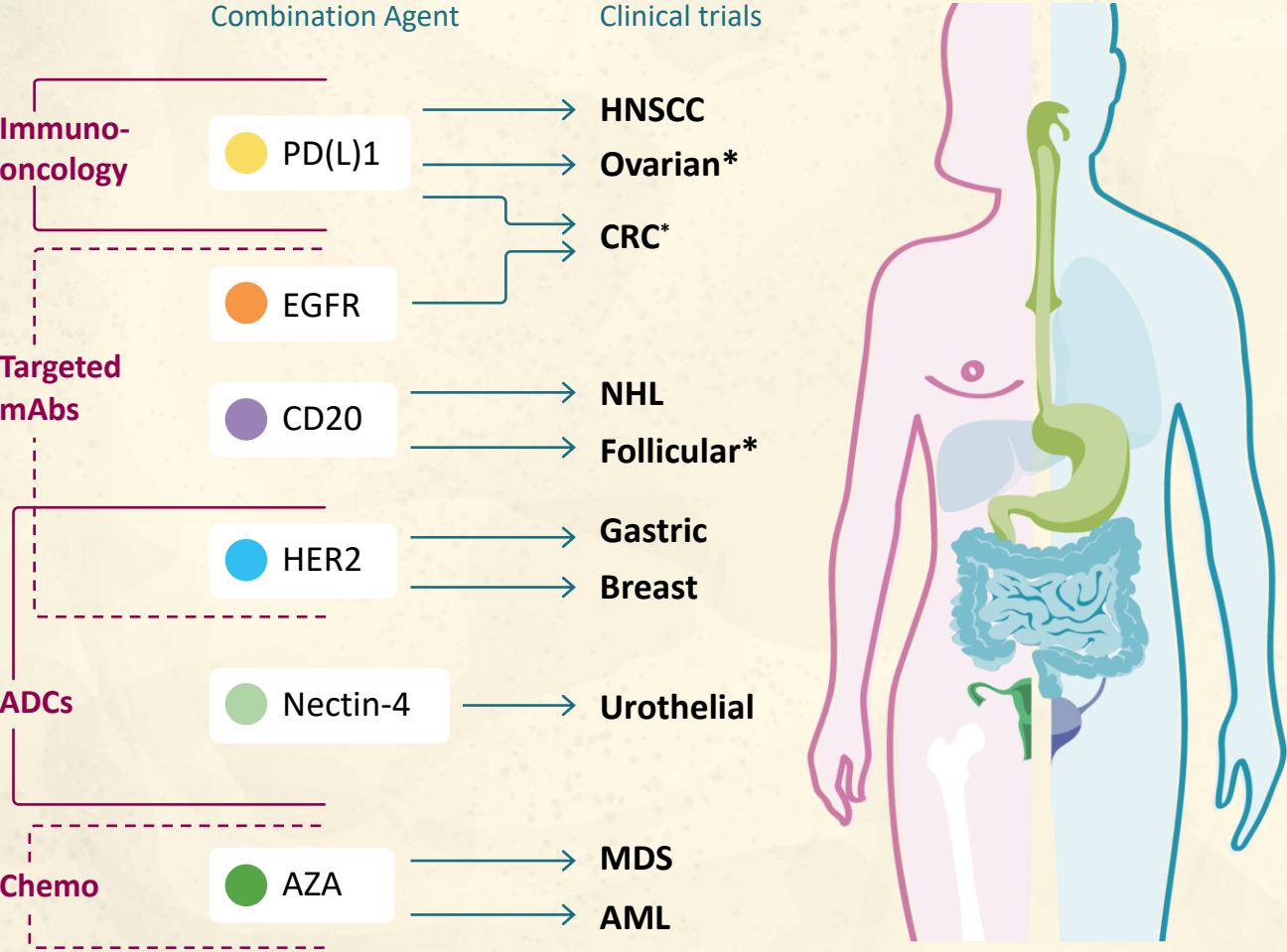
Continuing to build a pipeline based on expertise in protein engineering and oncology

- 2023 IND planned for ALTA-002*.
- Early preclinical development of ADC platform.

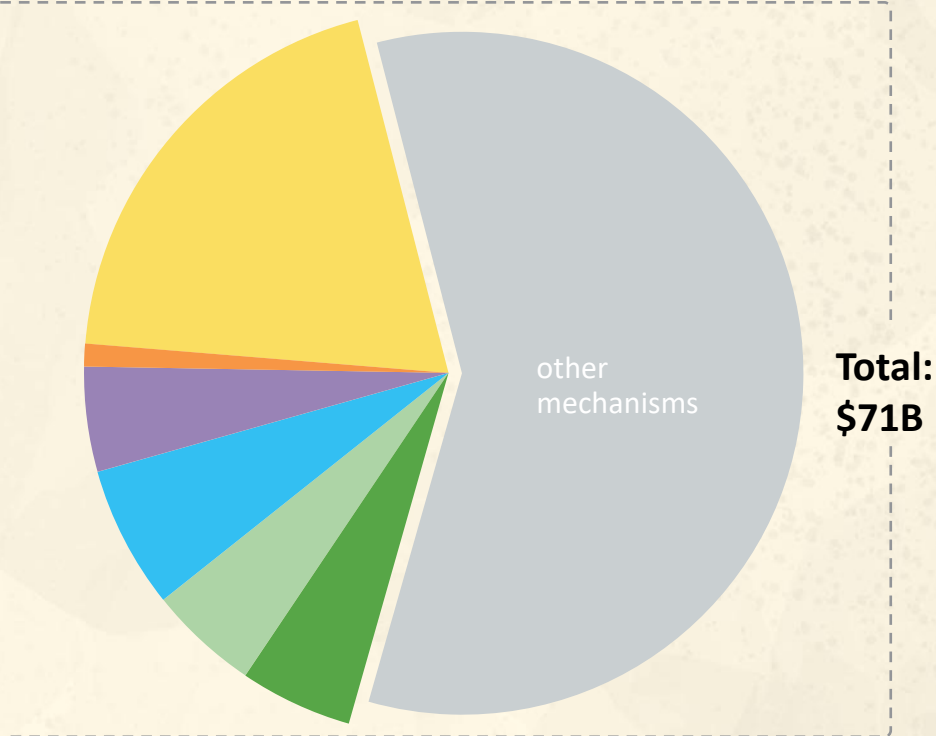
Strong financial position

- Cash, cash equivalents and investments of \$282.9M as of December 31, 2022.
- \$100M loan facility available.
- Expected cash runway through mid-2025.

EVORPACEPT IS DESIGNED TO IMPROVE THE ACTIVITY OF MANY CANCER THERAPIES RESULTING IN BROAD POTENTIAL UTILITY



U.S. oncology spending by mechanism



Evorpaccept combination agents represent nearly half of US cancer drug sales⁽¹⁾

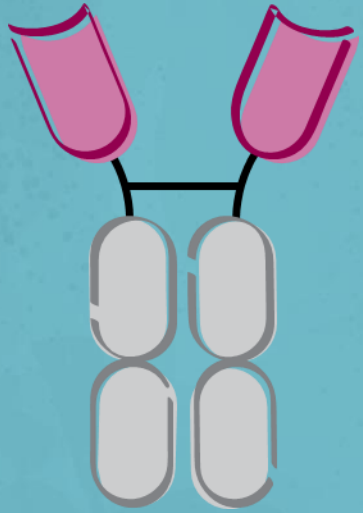
*Investigator sponsored trial. ADCs = antibody drug conjugates, AZA = azacitidine, HNSCC = head and neck squamous cell carcinoma, CRC = colorectal cancer, NHL = non-Hodgkin lymphoma, MDS = myelodysplastic syndromes, AML = acute myeloid leukemia
(1) 2021 IQVIA Global oncology trends report

PURSUING A ROBUST DEVELOPMENT PLAN

Indication		Evorpaccept Combination Agent	Discovery	IND Enabling	Phase 1	Phase 2	Phase 3	Fast Track	Collaboration Partner
Evorpaccept Combination Studies	SOLID TUMORS	HNSCC Head And Neck Squamous Cell Carcinoma	Keytruda (ASPEN-03)						MERCK
			Keytruda + 5FU + Platinum (ASPEN-04)						MERCK
		GC Gastric/Gastroesophageal Junction Cancer	Herceptin (ASPEN-01)						
			Herceptin + Cyramza + Paclitaxel (ASPEN-06)						<i>Lilly</i>
		Urothelial Cancer	Padcev (ASPEN-07)						
		Breast Cancer	Zanidatamab						zymeworks
	HEMATOLOGY		Enhertu (I-SPY)						QL HC Quantum Leap Healthcare Collaborative
		MDS Myelodysplastic Syndromes	Azacitidine (ASPEN-02)						
		AML Acute Myeloid Leukemia	Azacitidine + Venclexta (ASPEN-05)						
		NHL Non-Hodgkin's Lymphoma	Rituximab (ASPEN-01)						
ALTA 002*		Advanced Cancer							TALLAC THERAPEUTICS

EVORPACEPT INVESTIGATOR SPONSORED TRIALS (ISTs)

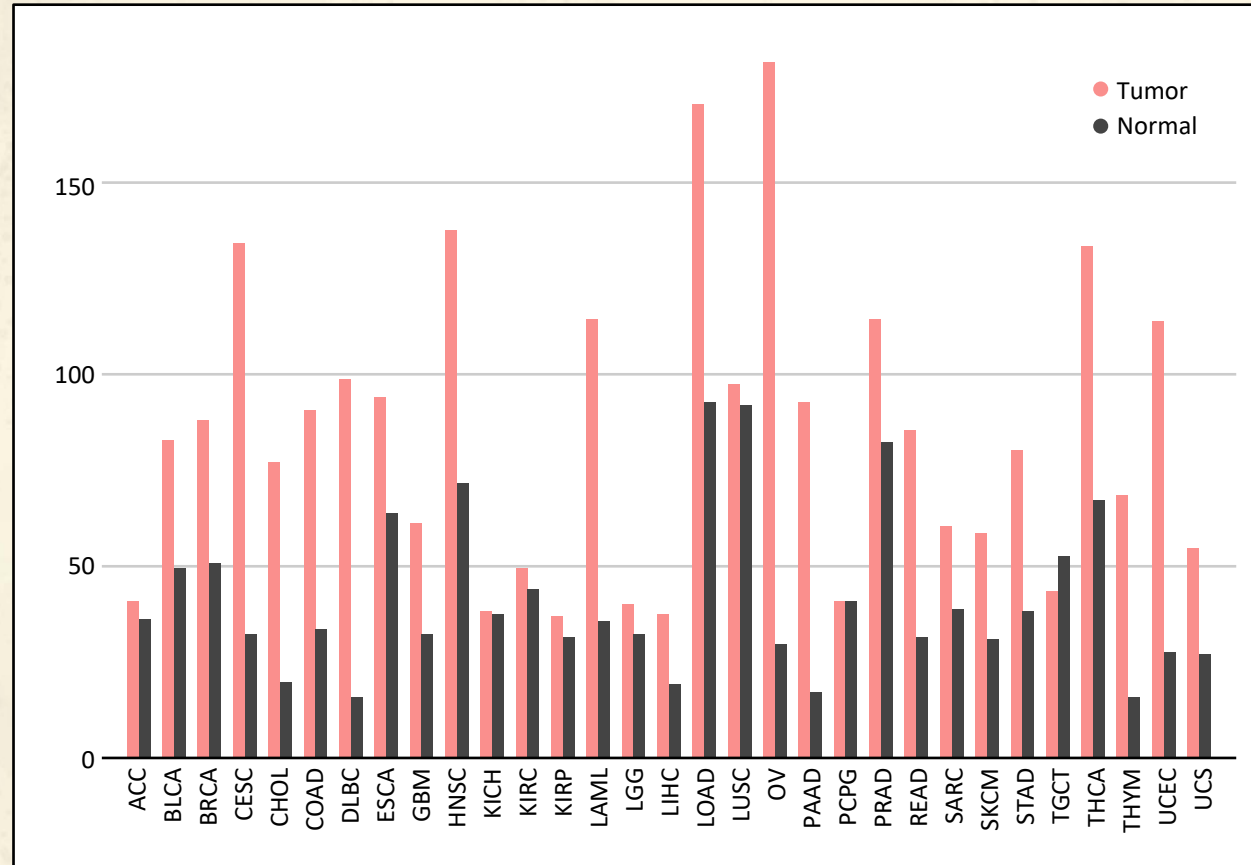
Indication		Evorpacept Combination Agent	Phase	Institution	
Evorpacept Combination Studies	SOLID TUMORS	Ovarian Cancer	Keytruda + Liposomal Doxorubicin	Ph2	The University of Pittsburgh Medical Center Hillman Cancer Center - <i>Planned</i>
		mCRC Metastatic Colorectal Cancer	Keytruda + Erbitux	Ph1b	The Academic GI Cancer Consortium
	HEMATOLOGY	NHL Non-Hodgkin’s Lymphoma	Rituximab + Lenalidomide	Ph1/2	The University of Texas M.D. Anderson Cancer Center



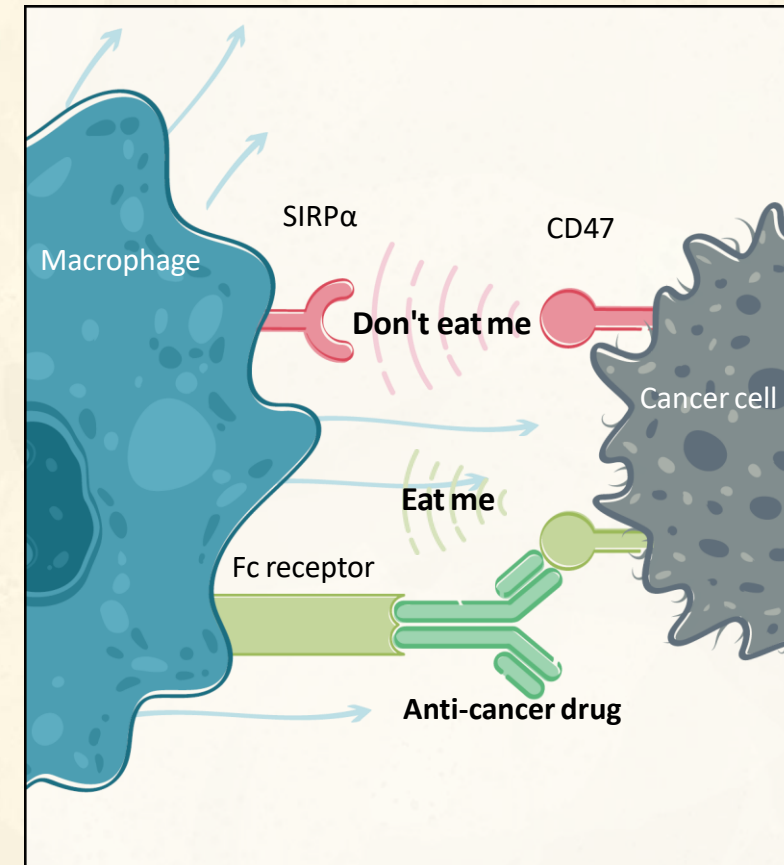
**EVORPACEPT
(ALX148)**

CD47 IS AN INNATE IMMUNE SYSTEM CHECKPOINT THAT CANCER CELLS USE TO MINIMIZE ACTIVITY OF ANTI-CANCER REGIMENS

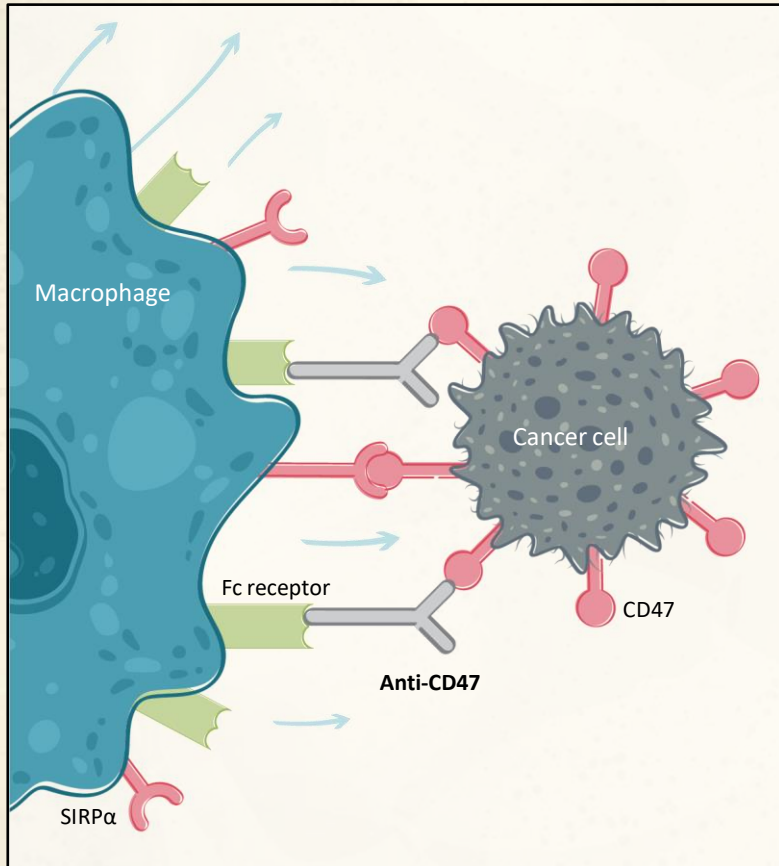
CD47 is expressed across many tumor types



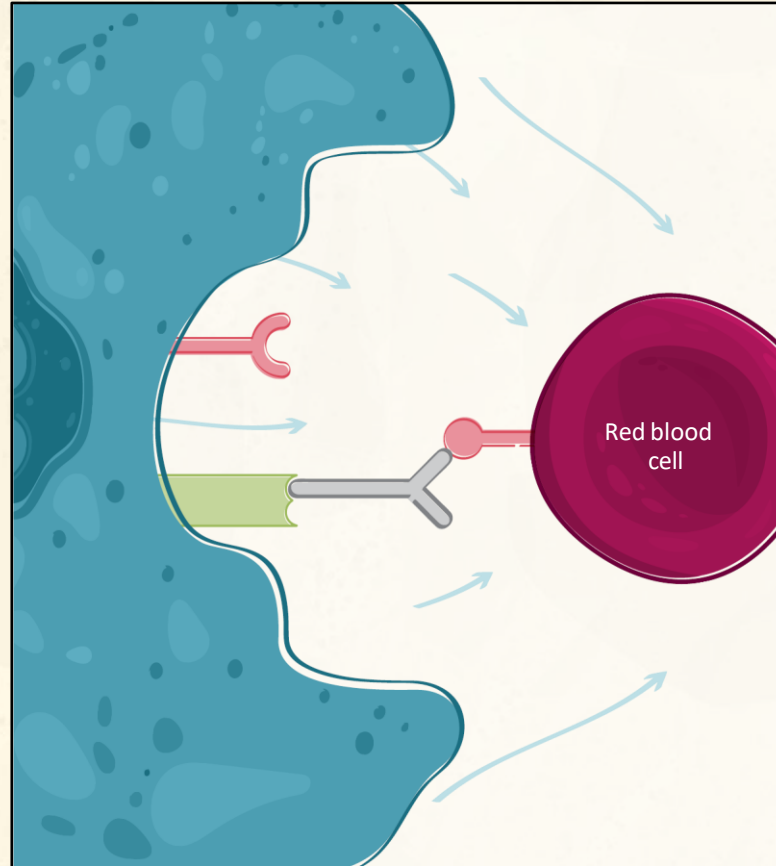
Anti-cancer drugs must overcome CD47 checkpoint to be effective



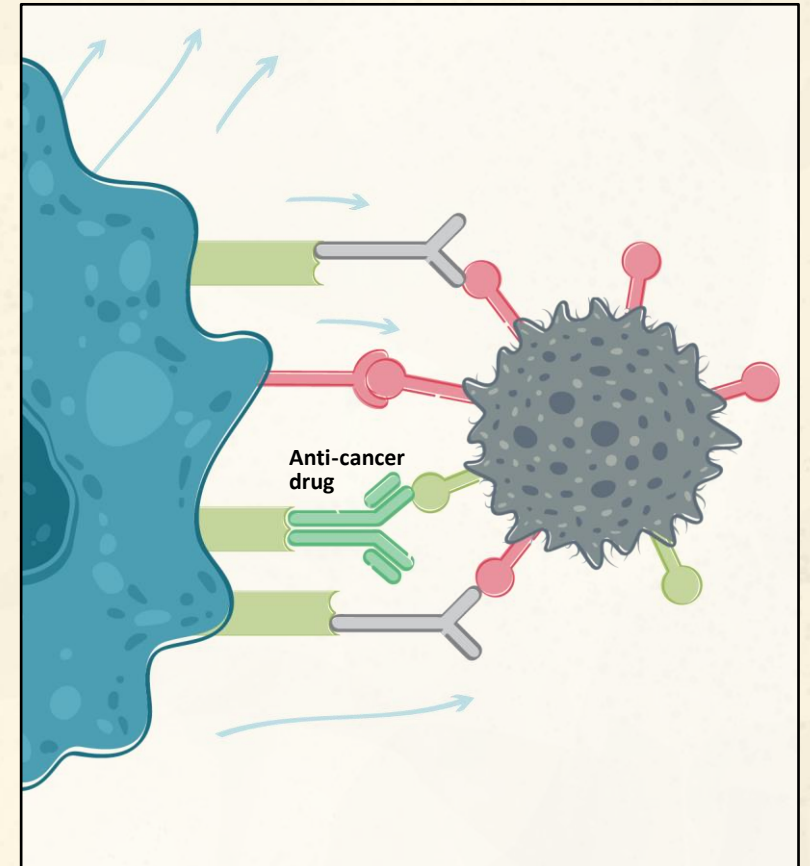
OTHER AGENTS TARGET CD47 AS A TUMOR ASSOCIATED ANTIGEN



Anti-CD47 with active Fc directly targets cancer cells

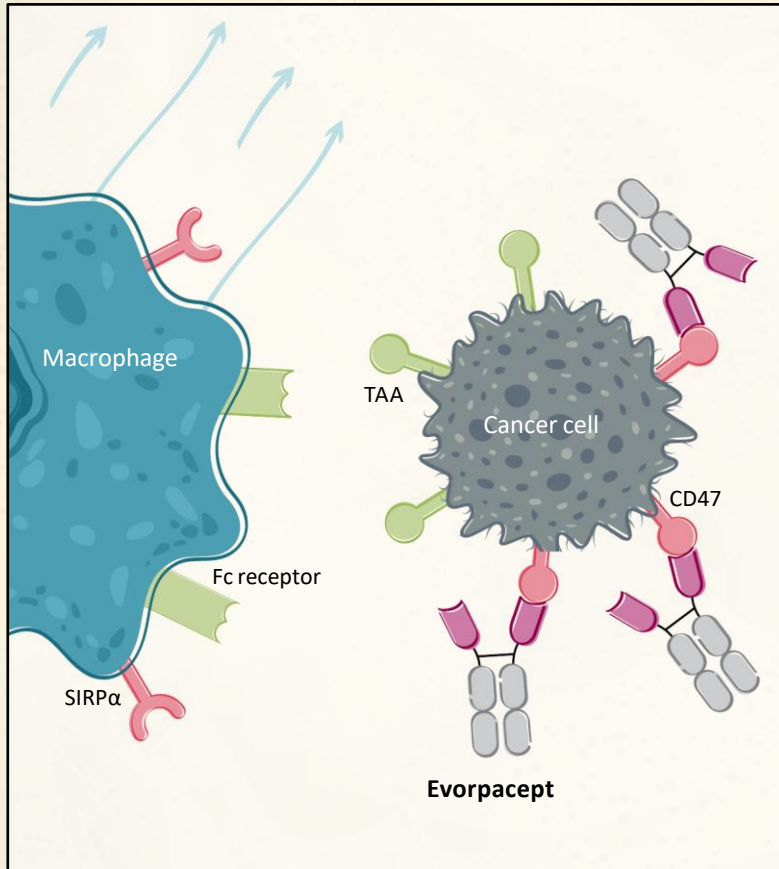


But also targets normal cells, causing toxicity

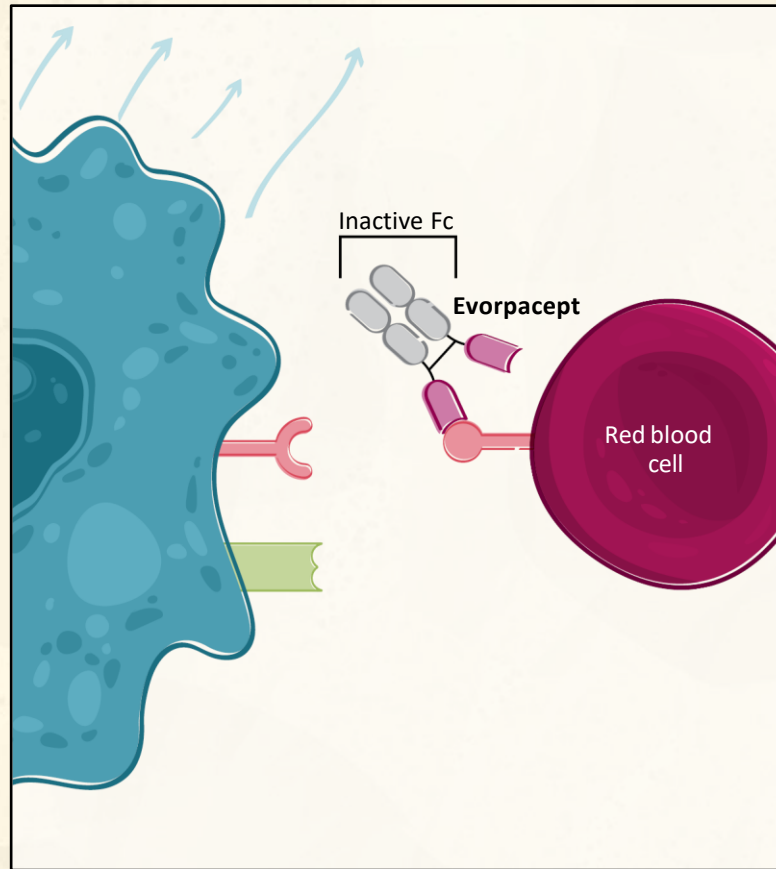


Dose limitations prevent full blockade of CD47 and active Fc competes with cancer therapies

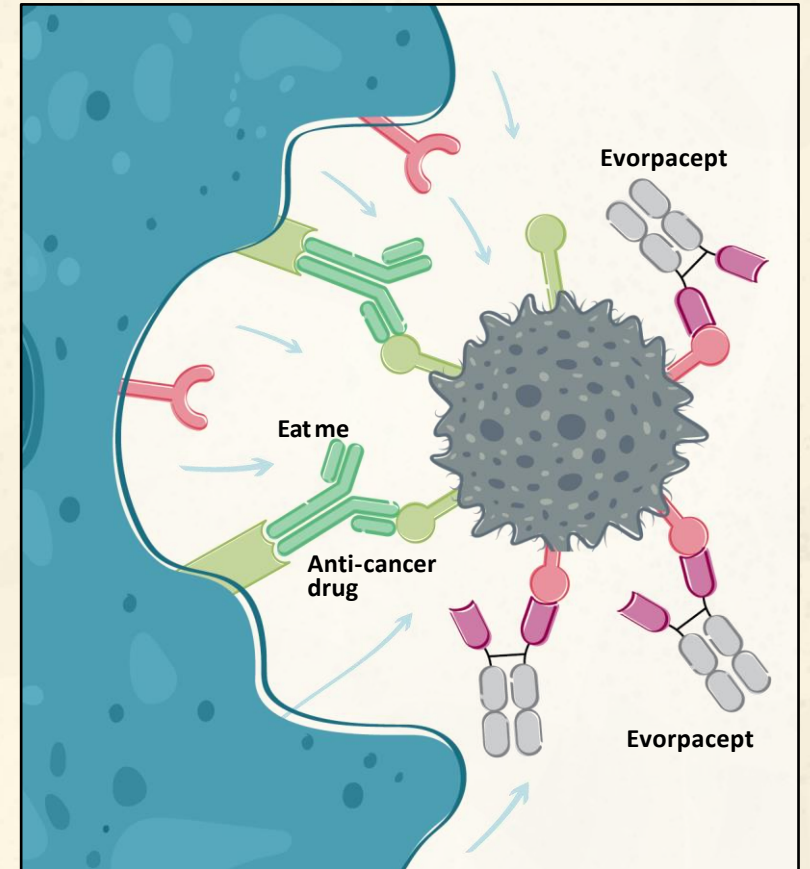
EVORPACEPT TARGETS THE CD47 CHECKPOINT



Anti-CD47 with inactive Fc binds and blocks CD47-SIRPα interaction

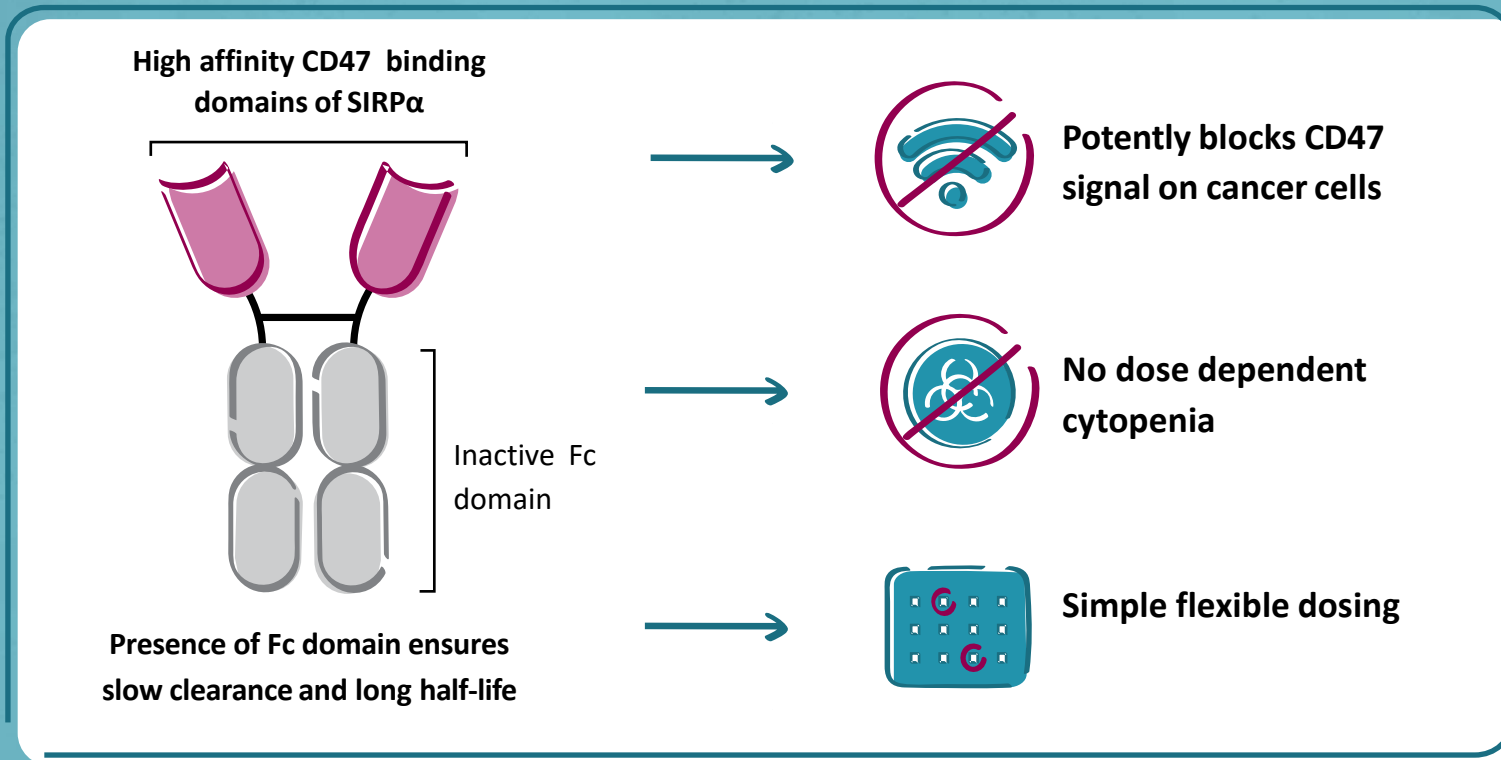


Inactive Fc spares normal cells, minimizing toxicity



Maximizing the activity of cancer therapies

EVORPACEPT: DESIGNED TO BE BEST-IN-CLASS CD47 CHECKPOINT INHIBITOR



- ~Half the molecular weight of an antibody
- Increases solid tumor penetration
- Standard antibody manufacturing process

**Designed to maximize
a patient's immune
response**

EVORPACEPT IS A HIGHLY DIFFERENTIATED CD47 BLOCKER



Name	evorpaccept	magrolimab	TTI-621	TTI-622	Lemzoparlimab
Molecule Structure	High-affinity SIRPα-Fc fusion protein	CD47 mAb	Wild Type SIRPα-Fc fusion protein	Wild Type SIRPα-Fc fusion protein	CD47 mAb
Affinity	0.1 nM	8 nM	500 nM ¹	500 nM ¹	0.5 nM
Fc Effector Function	None	Medium (IgG4)	High (IgG1)	Medium (IgG4)	Medium (IgG4)
Hematologic toxicity signal	No	Yes	Yes	Yes	Yes
Solid Tumor Proof of Principle	✓ (gastric, HNSCC) ²	✗ (CRC, ovarian) ^{3,4}	✗	✗	✗
Heme-Onc Initial Signs of Activity	✓ (NHL, MDS, AML)	✓ (NHL, MDS, AML)	✓ (NHL, T cell lymphomas)	✓ (NHL, T cell lymphomas)	✓ (NHL, MDS)

EVORPACEPT DEMONSTRATED A CONSISTENT TOLERABILITY PROFILE IN ASPEN-01

Over 300 patients dosed in evorpaccept trials with no dose dependent cytopenias

Treatment related adverse events	evorpaccept + Herceptin + Cyramza + chemo (N=18)		evorpaccept + Keytruda + chemo (N=13)		evorpaccept + Keytruda (N=52)		evorpaccept + azacitidine (N=22)	
	Total n (%)	≥Grade 3	Total n (%)	≥Grade 3	Total n (%)	≥Grade 3	Total n (%)	≥Grade 3
Fatigue	2 (11.1%)	-	1 (7.7%)	-	6 (11.5%)	-	-	-
Rash / dermatitis acneiform	4 (22.2%)	-	-	-	5 (9.6%)	-	-	-
AST increased	-	-	-	-	9 (17.3%)	-	-	-
Platelets decreased	-	-	-	-	4 (7.7%)	2 (3.8%)	-	-
ALT increased	-	-	-	-	7 (13.5%)	1 (1.9%)	-	-
Pruritus	2 (11.1%)	-	-	-	5 (9.6%)	-	-	-
Pyrexia	-	-	-	-	3 (5.8%)	-	-	-
Decreased appetite	-	-	-	-	2 (3.8%)	-	-	-
Anemia	1 (5.6%)	-	1 (7.7%)	1 (7.7%)	5 (9.6%)	1 (1.9%)	-	-
Infusion reaction	-	-	-	-	4 (7.7%)	-	4 (18.2%)	-
Neutropenia / neutrophil count decrease	-	-	1 (7.7%)	-	2 (3.8%)	1 (1.9%)	3 (13.6%)	2 (9.1%)
Nausea	-	-	-	-	2 (3.8%)	-	2 (9.1%)	-
Alkaline phosphatase incr	-	-	-	-	3 (5.8%)	-	-	-
Arthralgia	-	-	-	-	3 (5.8%)	-	-	-
WBC decreased	-	-	-	-	3 (5.8%)	-	-	-
Myalgia	-	-	-	-	2 (3.8%)	-	-	-
Diarrhea	3 (16.7%)	-	-	-	-	-	-	-
Urticaria	3 (16.7%)	-	-	-	-	-	-	-
Lymphocyte count decreased	1 (5.6%)	1 (5.6%)	-	-	-	-	-	-
Headache	1 (5.6%)	-	-	-	-	-	-	-
Stomatitis	1 (5.6%)	-	-	-	-	-	-	-
Back pain	1 (5.6%)	-	-	-	-	-	-	-
Vision blurred	1 (5.6%)	-	-	-	-	-	-	-
Abdominal pain / abdominal pain upper	1 (5.6%)	-	-	-	-	-	-	-
Hypersensitivity	-	-	1 (7.7%)	1 (7.7%)	-	-	-	-
Pneumonitis	-	-	1 (7.7%)	-	-	-	-	-
Constipation	-	-	-	-	-	-	3 (13.6%)	-
Vomiting	-	-	-	-	-	-	2 (9.1%)	-

Tolerability profile enables broad combination potential

EVORPACEPT'S INITIAL CLINICAL ACTIVITY IS MAGNIFIED IN SURVIVAL-BASED ENDPOINTS ACROSS SOLID TUMOR TYPES IN MULTIPLE TRIALS (ASPEN-01 COHORTS)

Population	≥2L HER2+ GC		1L HNSCC		≥2L HNSCC (CPI-Naïve)	
Combination (N-evaluable)	evorpacept + Herceptin + Cyramza + paclitaxel (N=18)		evorpacept + Keytruda + 5FU + platinum (N=13)		evorpacept + Keytruda (N=10)	
ORR	evorpacept 72%	benchmark ¹ 28%	evorpacept 39%	benchmark ² 36%	evorpacept 40%	benchmark ³ 15%
mPFS (months)	17.1	4.4	5.6	4.9	4.6	2.1
mOS (months)	17.1	9.6	NR	13.0	24.5	8.4
OS rate at 12 months	79%	40%	88%	53%	80%	37%
Benchmark regimen	Cyramza + paclitaxel		Keytruda + 5FU + platinum		single agent Keytruda	

Data as of September 1, 2021. NR = not reached, ORR = Objective Response Rate, mPFS = median progression free survival, mOS = median overall survival. CPI = checkpoint inhibitor.

¹Wilke, Lancet Oncology, 2014; ²Burtneess, Lancet, 2019; ³Cohen, Lancet, 2018.

Data as of February 1, 2022. NC = not calculable, (95% CI)

≥2L HER2+ GC: mOS not reached (CI: 9.84-NC) with median follow up of 18.7 months (CI: 9.28-21.7)

1L HNSCC: mOS not reached (CI: 5.99-NC) with median follow up of 15.8 months (CI: 5.0-17.8)

≥2L HNSCC (CPI-Naïve): mOS of 24.6 months (CI: 3.13-NC) with median follow-up of 35.3 months (CI: 27.0-41.0)

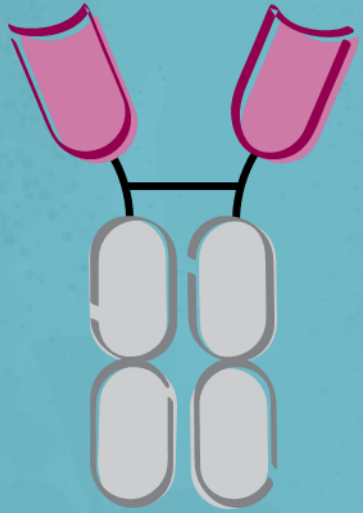
EARLY DATA SHOWS EVORPACEPT COMBINATIONS HAVE ACHIEVED COMPLETE RESPONSES IN AGGRESSIVE HEMATOLOGIC MALIGNANCIES

ASPEN-02

Population	Previously untreated higher risk myelodysplastic syndromes (MDS) with TP53 mutation		Relapsed / refractory MDS
Combination	Evorpacept + azacitidine	Magrolimab + azacitidine ¹	Evorpacept + azacitidine
N-evaluable	5	25	9
CR	2	10	-
mCR	1 with HI	5	5*
SD	1		2

ASPEN-01

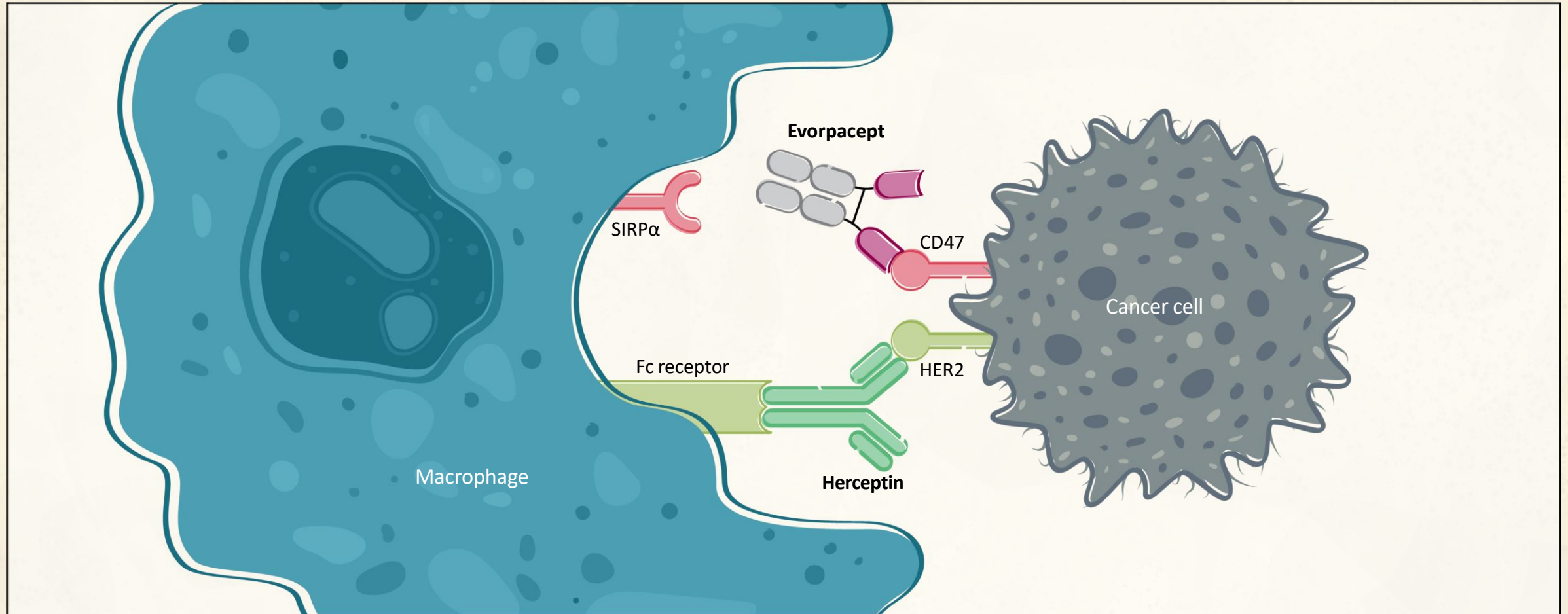
Population	≥2L aggressive non-Hodgkin's lymphoma	
Combination	Evorpacept + Rituximab ²	Magrolimab + Rituximab ³
N-evaluable	21	38
ORR (%)	8 (38%)	11 (29%)
CR (%)	1 (5%)	2 (5%)
PR (%)	7 (33%)	9 (24%)



**ASPEN-06:
EVORPACEPT (ALX148)
IN HER2+ GASTRIC/GEJ CANCER**

GC TRIAL: EVORPACEPT + HERCEPTIN MECHANISM OF ACTION

evorpaccept
in
GASTRIC



Evorpaccept increases antibody dependent cellular phagocytosis in combination with Herceptin

CLINICAL ACTIVITY OF HISTORICAL BENCHMARKS IN PATIENTS WITH HER2 POSITIVE GASTRIC CANCER

Population	N	ORR	DOR (m) [95% CI]	PFS (m) [95% CI]	OS (m) [95% CI]	OS rate at 12 m
≥2L Gastric ramucirumab/paclitaxel RAINBOW ¹	330	28%	4.4 [IQR 2.8–7.5]	4.4 [4.2-5.3]	9.6 [8.5-10.8]	40%
≥2L Gastric trastuzumab/ram/paclitaxel ²	50	52%	5.1 [3.3-6.9]	7.4 [6.5-8.3]	13.6 [9.6-17.5]	-
2L Gastric trastuzumab-deruxtecan DESTINY 02 ³	79	38%	8.1 [4.1-NE]	5.5 [4.2-7.3]	-	-
≥3L Gastric trastuzumab-deruxtecan DESTINY 01 ⁴	126	41%	11.3 [5.6-NE]	5.6 [4.3-6.9]	12.5 [9.6-14.3]	52%

ASPEN-01 PHASE 1B GC TRIALS: PATIENT BASELINE CHARACTERISTICS

		evorpaccept + Herceptin ≥2L GC (N=20)	evorpaccept + Herceptin + Cyramza/chemo ≥2L GC (N=18)
Median age, years (range)		58 (45-79)	67.5 (36-83)
Sex, n	M	15	13
	F	5	5
Race, n	Asian	13	15
	White	6	3
	Other	1	-
ECOG PS, n	0	7	8
	1	13	10
Progressed upon prior anti-HER2 therapy, n (%)		19 (95)	17 (94)
Progressed upon ≥2 prior anti-HER2 therapy n (%)		9 (45)	2 (11)
Progressed upon prior CPI therapy, n (%)		9 (45)	2 (11)
Visceral distant metastasis, n (%)		17 (85)	15 (83)

ASPEN-01 PHASE 1B ≥ 2 LINE GC TRIAL: EVORPACEPT + HERCEPTIN + CYRAMZA + PACLITAXEL

Phase 1b higher dose + chemo trial:



Patients:

R/R HER2 positive GC, 2L or greater;
Progressed on prior Herceptin and
fluoropyrimidine or platinum.



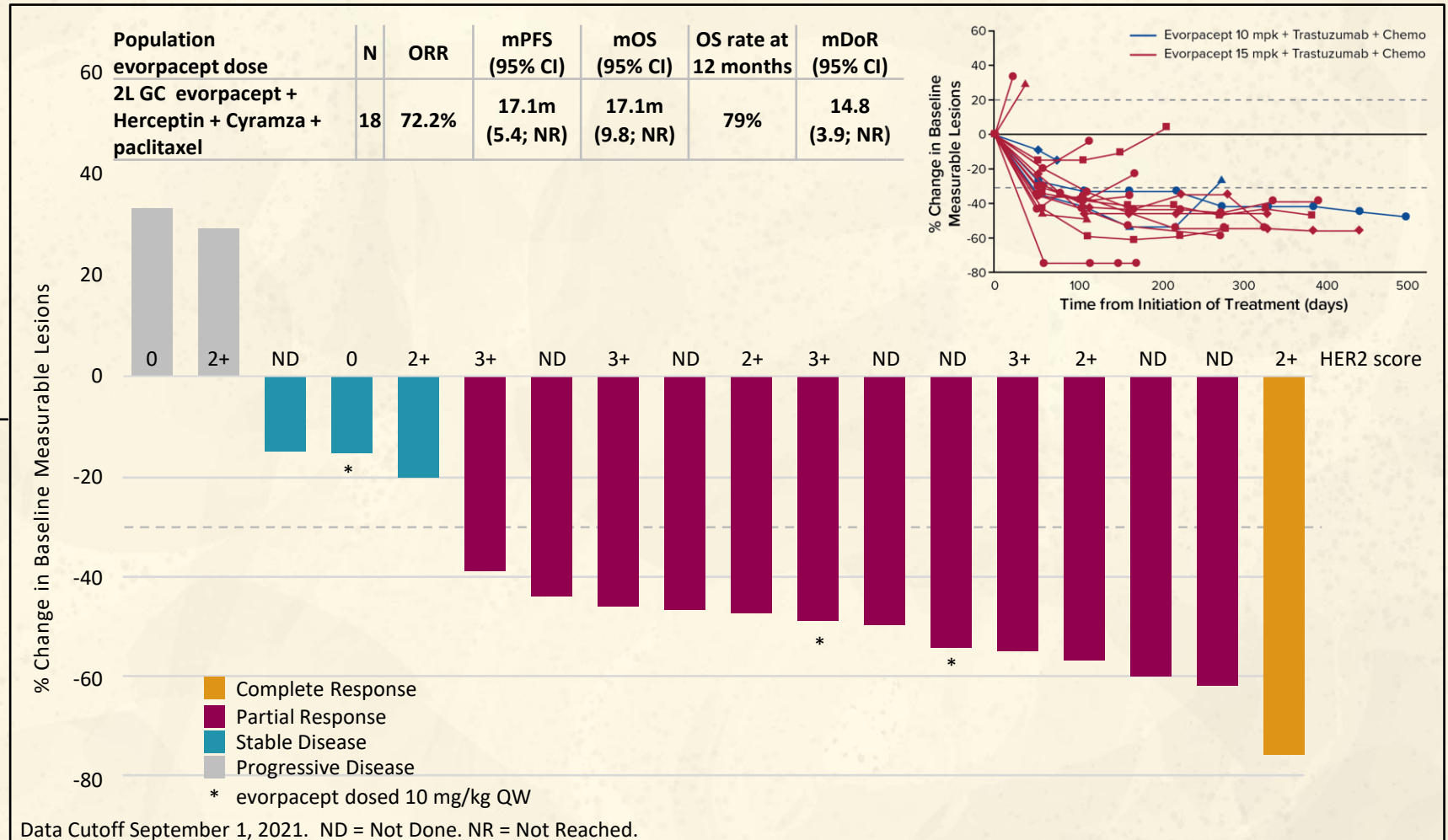
Treatment:

evorpacept 10 and 15 mg/kg (QW)
+ **Herceptin**
+ **Cyramza**
+ **paclitaxel**



Endpoint:

- safety of combination
- anti-cancer activity



Data as of February 1, 2022. NC = not calculable, (95% CI)
mOS not reached (CI: 9.84-NC) with median follow up of 18.7 months (CI: 9.28-21.7)

SECOND LINE GC: RANDOMIZED PHASE 2 CLINICAL TRIAL, ASPEN-06

Randomized Phase 2: Ongoing



Patients:
N≈122

2L or greater HER2 positive GC
with prior HER2 targeted therapy



Treatment:
1:1 randomization

evorpacept 30 mg/kg (Q2W)

+ Herceptin

+ Cyramza

+ paclitaxel

vs.

+ Herceptin

+ Cyramza

+ paclitaxel



Endpoint: • Anticancer activity: including ORR, DOR, PFS, OS

Randomized Planned Phase 3:



Patients:

2L or greater HER2 positive GC
with prior HER2 targeted therapy



Treatment:

evorpacept 30 mg/kg (Q2W)

+ Herceptin

+ Cyramza

+ paclitaxel

vs.

+ Cyramza

+ paclitaxel



Endpoint: • Anticancer activity: including OS, PFS, ORR, DOR

ASPEN-01 PHASE 1B ≥ 2 LINE GC TRIAL: EVORPACEPT + HERCEPTIN

Phase 1b GC trial:

 Response
evaluable patients

N=19 HER2 positive GC
progressed on prior fluoropyrimidine,
Herceptin or platinum.

 Treatment:

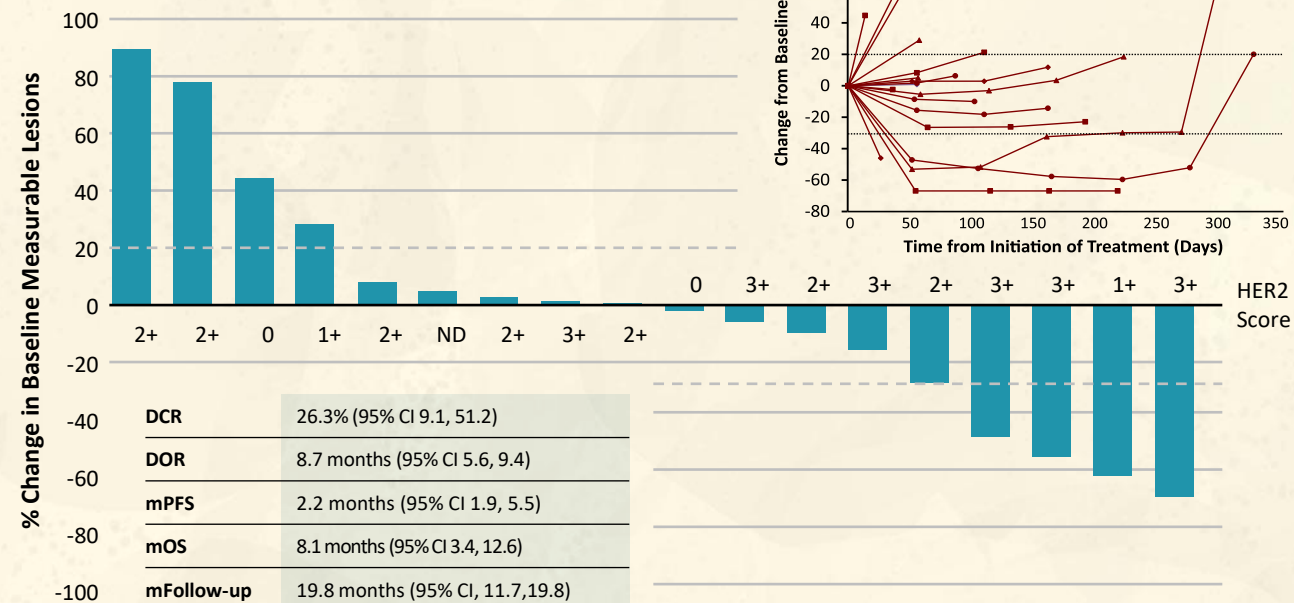
evorpacept 10 mg/kg
once a week (QW)
+ **Herceptin**
8 mg/kg once, then
6 mg/kg every three weeks (Q3W)

 Endpoints:

- maximum tolerated dose
- anti-cancer activity

Result:

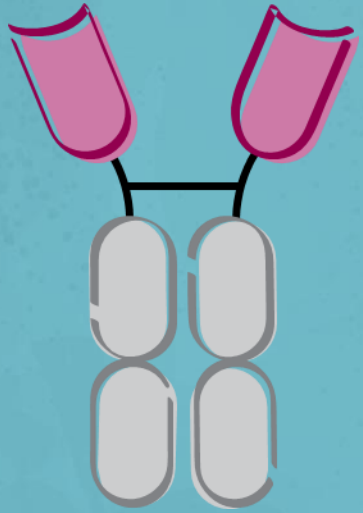
ORR 21.1% (4/19)



Notes: Data Cutoff October 1, 2020. Patients who received at least one dose of evorpacept in the expansion phase, had a baseline assessment, and at least one post-baseline disease assessment; One patient with clinical progression not included in plots.

ORR = Overall Response Rate. **ND** = Not Done. **HER2** Score retrospectively assessed using archival tissue by a central IHC lab.

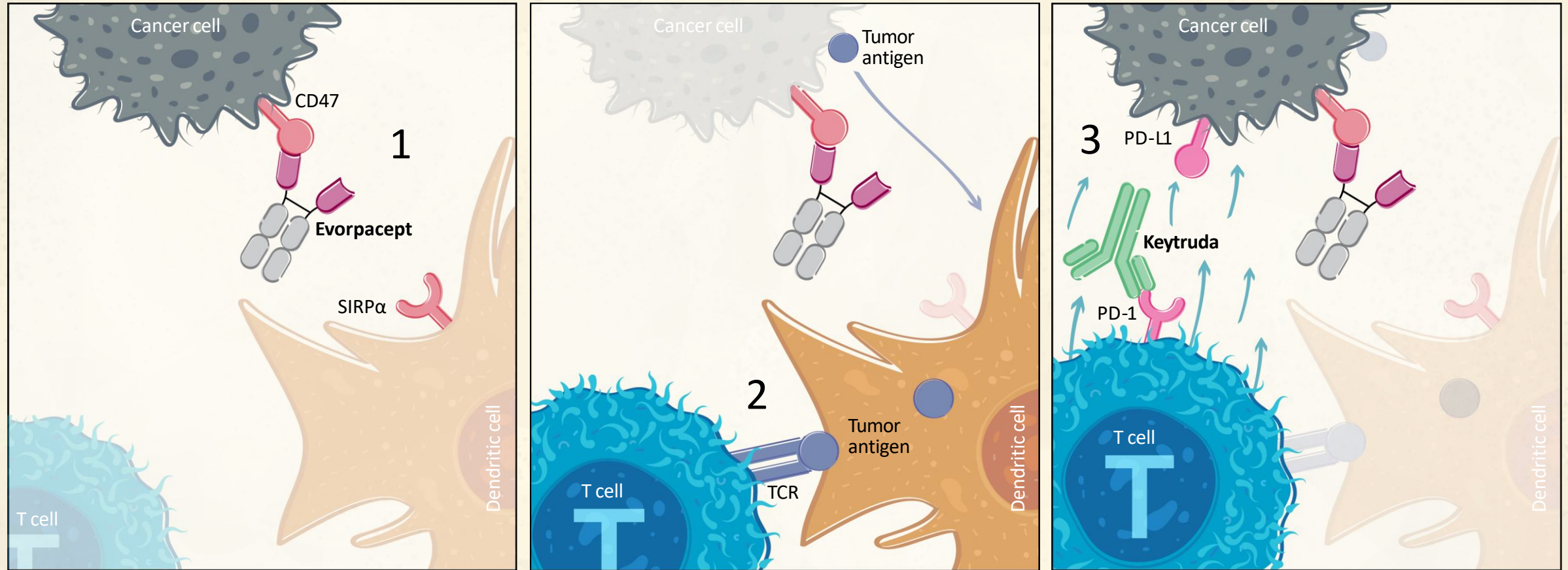
FDA granted evorpacept fast track designation for second-line treatment of HER2 positive GC



**ASPEN-03 AND ASPEN-04:
EVORPACEPT (ALX148)
IN 1L HNSCC**

HNSCC TRIAL: EVORPACEPT + KEYTRUDA MECHANISM OF ACTION

evorpaccept
in
HNSCC



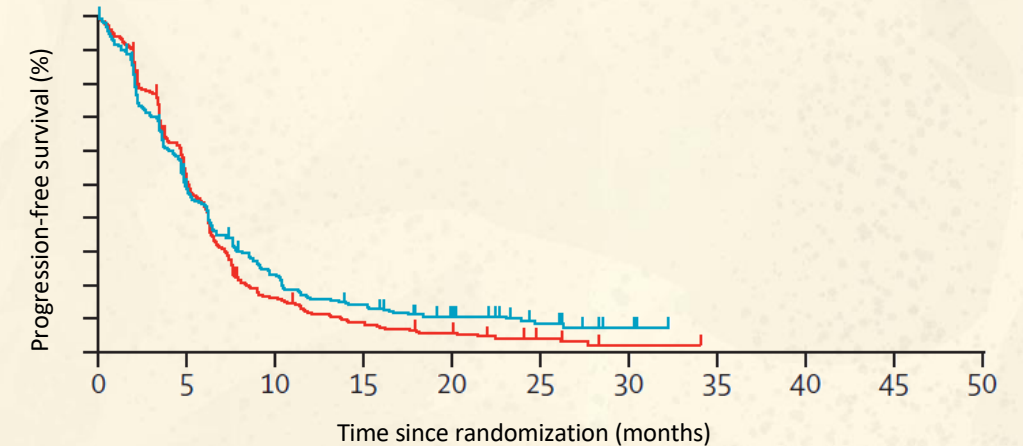
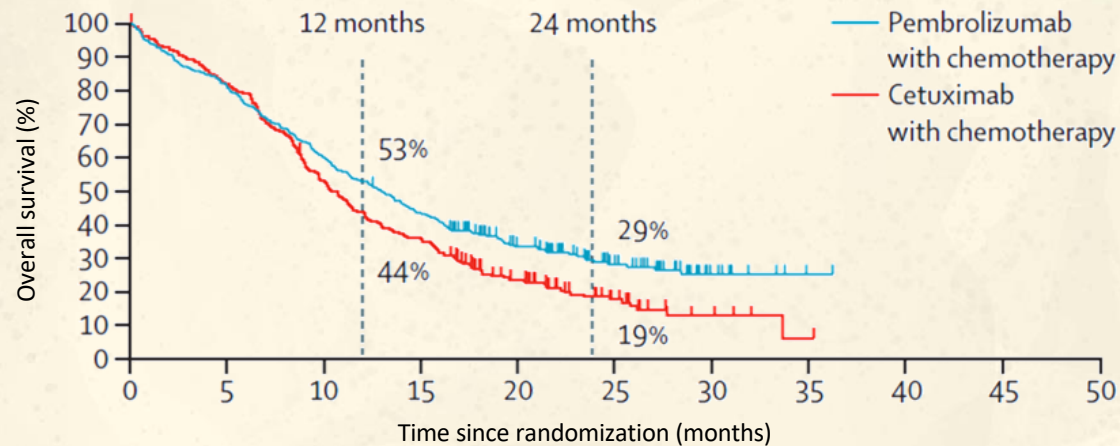
Evorpaccept activates dendritic cells and enhances cross-priming of T cells

OS RATE AT 12 MONTHS PREDICTIVE OF OVERALL SURVIVAL

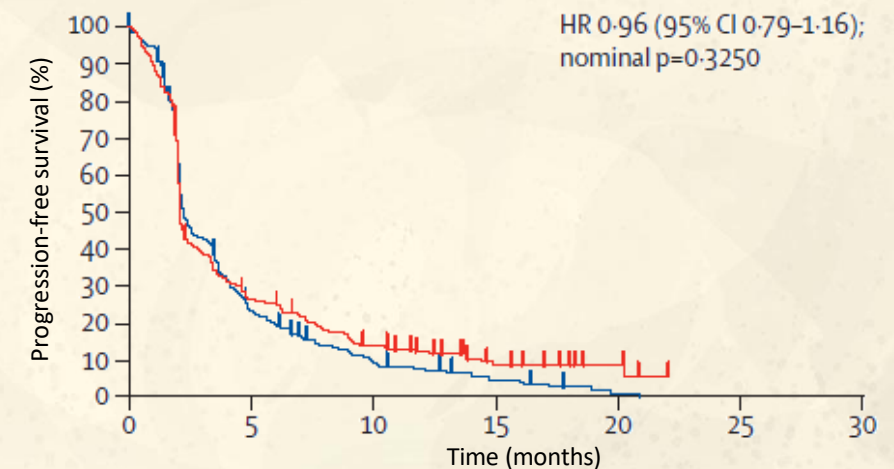
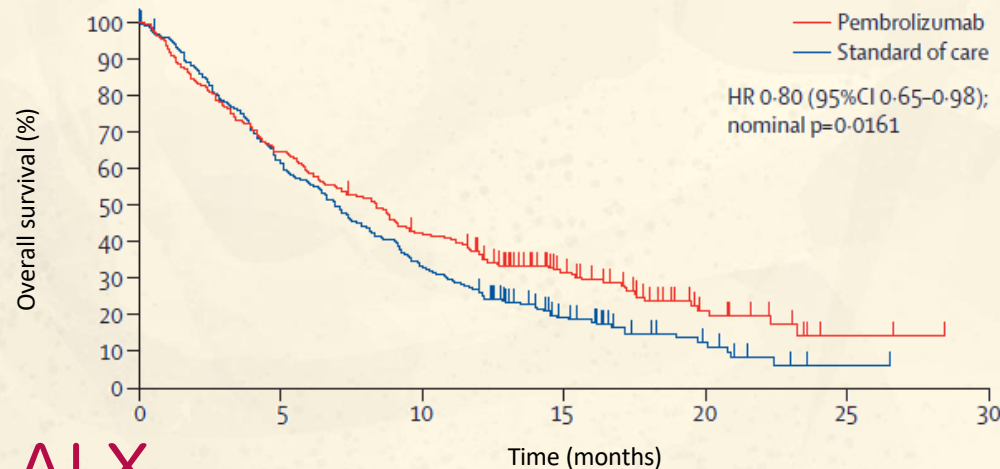
Population	N	ORR (%)	PFS (m) [95% CI]	OS Rate at 12 m	OS (m) [95% CI]	Follow Up (m) [95% CI]
KEYNOTE-048: 1L HNSCC pembrolizumab + 5FU/platinum	281	36%	4.9 [4.7–6.0]	53%	13.0 [10.9–14.7]	13 [6.4–26.6]
KEYNOTE-048: 1L HNSCC cetuximab + 5FU/platinum	278	36%	5.1 [4.9–6.0]	44%	10.7 [9.3–11.7]	10.7 [6.6–19.7]
KEYNOTE-040: 2L HNSCC (CPI naïve) pembrolizumab	247	14.6%	2.1 [2.1–2.3]	37%	8.4 [6.4–9.4]	8.4 [3.3–14.5]
KEYNOTE-040: 2L HNSCC (CPI naïve) Phys Choice: methotrexate, docetaxel, or cetuximab	248	10.1%	2.3 [2.1–2.8]	26.5%	6.9 [5.9–8.0]	7.1 [3.7–12.4]

IMMUNO-ONCOLOGY AGENTS IN CPI NAÏVE HNSCC POPULATIONS: PFS AND OS AS ENDPOINTS IN KEYNOTE-040 AND 048

KEYNOTE-048: OS and PFS at the Second Interim Analysis in the 1L HNSCC CPI Naïve Population



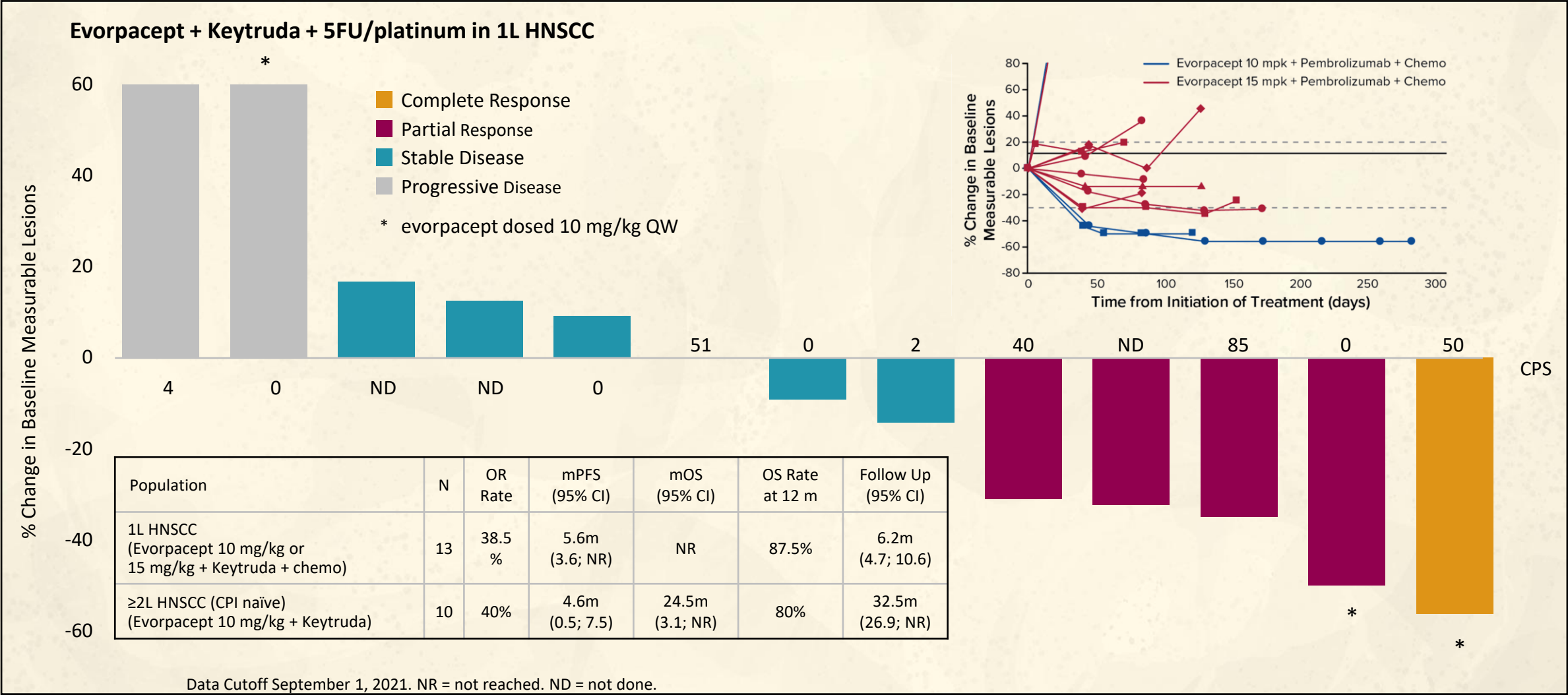
KEYNOTE-040: OS and PFS at the Final Analysis in the 2L HNSCC CPI Naïve Population



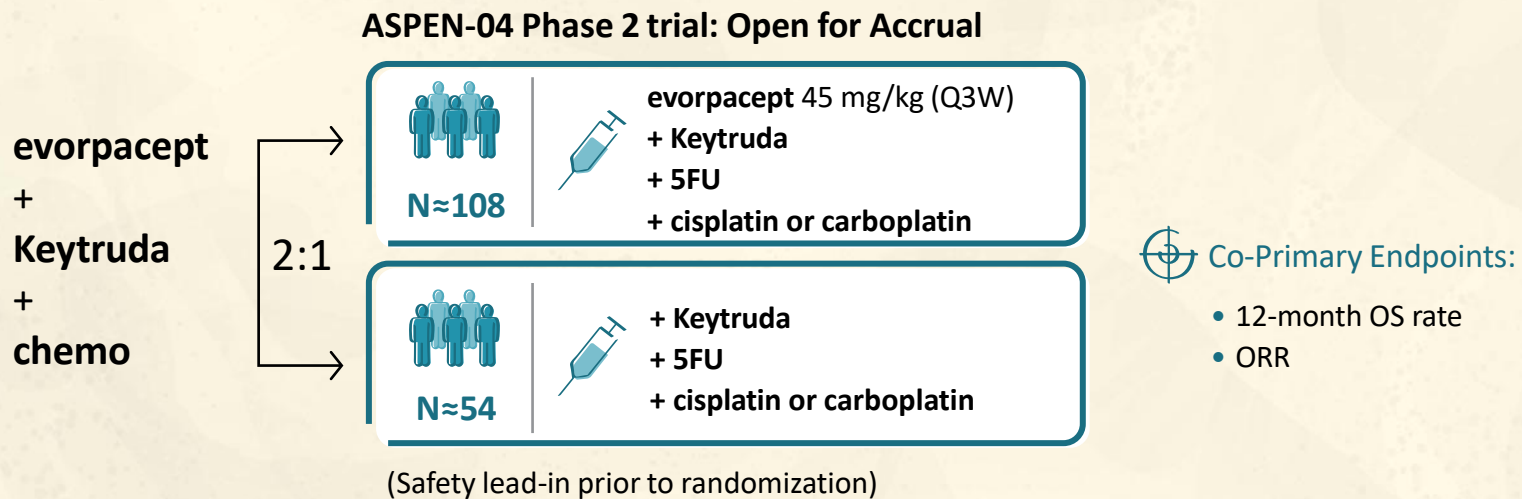
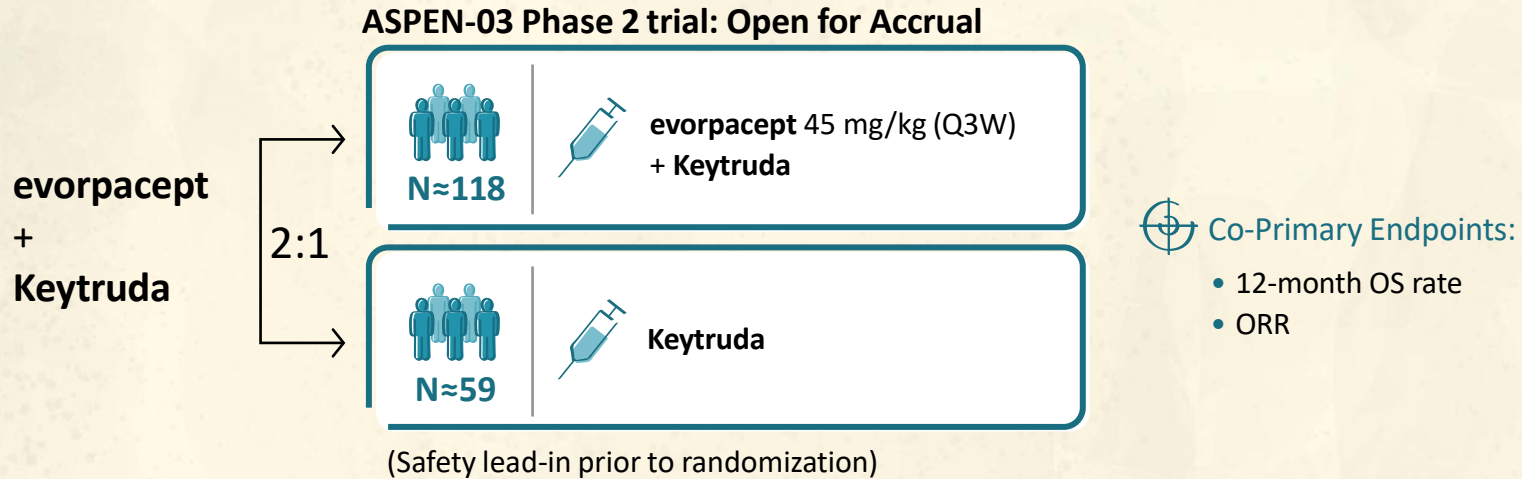
ASPEN-01 HNSCC TRIALS: PATIENT BASELINE CHARACTERISTICS

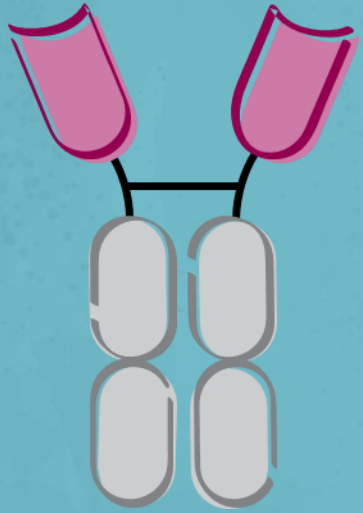
		evorpaccept + Keytruda ≥2L HSCC (N=10)	evorpaccept + Keytruda + 5FU/platinum 1L HNSCC (N=13)
Median age, years (range)		63 (35-81)	61 (45-70)
Sex, n	M	7	12
	F	3	1
Race, n	Asian	5	10
	White	4	3
	Black	1	-
ECOG PS, n	0	3	8
	1	7	5
Progressed upon prior CPI therapy, n (%)		0 (0)	0 (0)
Visceral distant metastasis, n (%)		6 (60)	7 (54)

ASPEN-01 PHASE 1B HNSCC: EVORPACEPT + KEYTRUDA + 5FU/PLATINUM FIRST LINE CHECKPOINT NAIVE



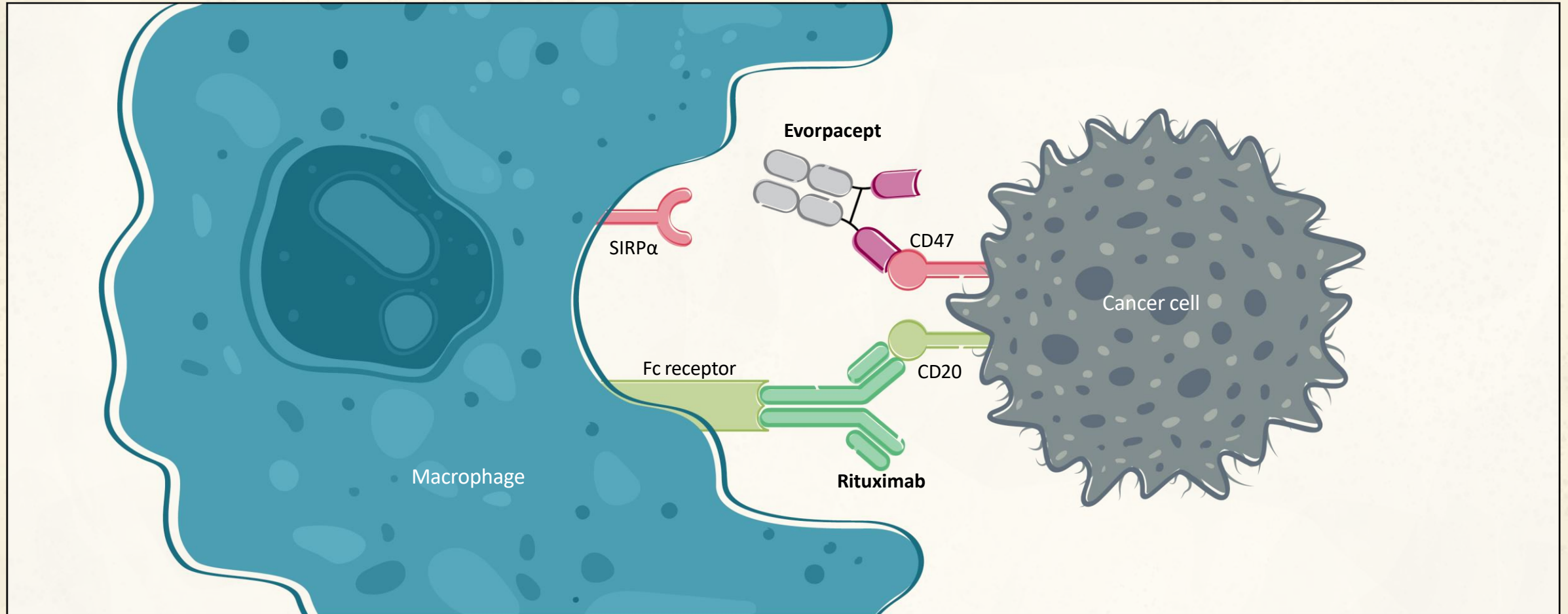
FIRST LINE HEAD & NECK CANCER: PHASE 2 DEVELOPMENT PLAN, ASPEN-03 AND ASPEN-04





EVORPACEPT IN HEMATOLOGIC MALIGNANCIES

NHL TRIAL: EVORPACEPT + RITUXIMAB MECHANISM OF ACTION



Evorpaccept increases antibody dependent cellular phagocytosis in combination with Rituximab

EVORPACEPT SHOWS CLINICAL ACTIVITY IN HEMATOLOGIC MALIGNANCY: ASPEN-01 NHL

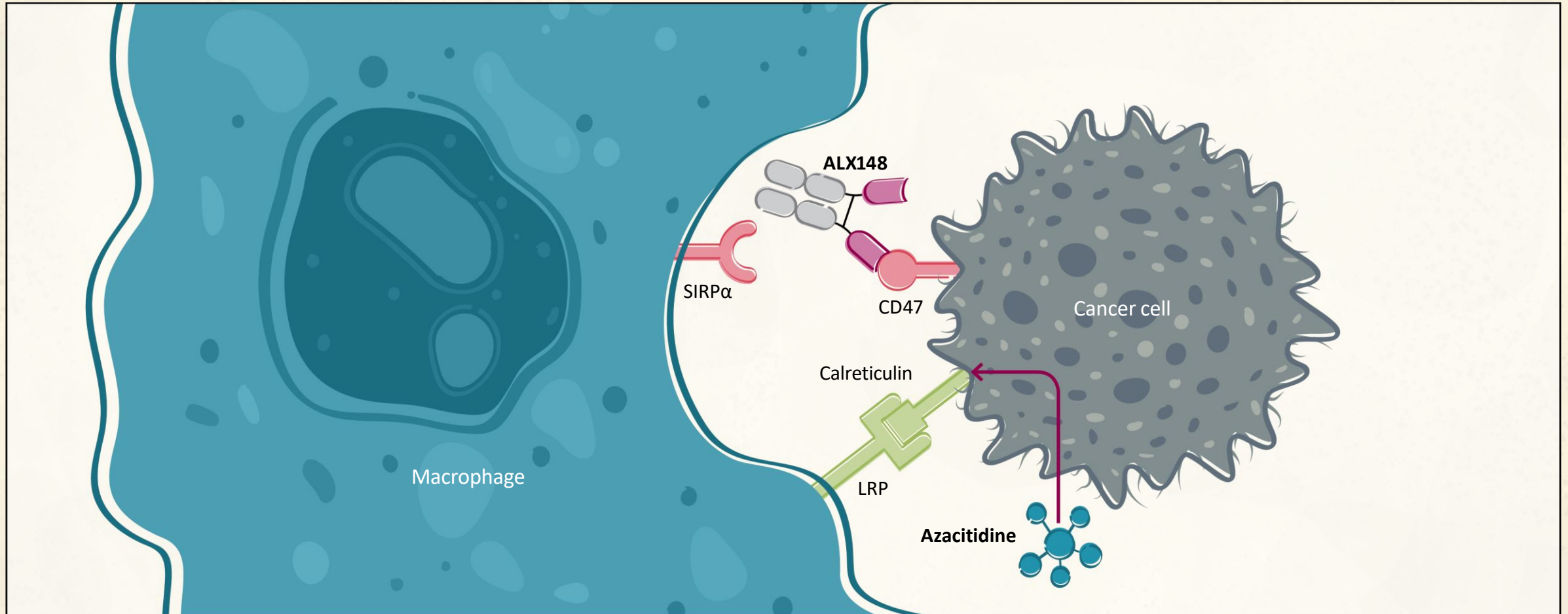
evorpacept
in
NHL

Population	Evorpacept (10 mg/kg QW) + Rituximab		Evorpacept (15 mg/kg QW) + Rituximab	
	N	ORR	N	ORR
All	22	40.9%	10	70.0%
Aggressive	15	33.3%	6	50.0%
Indolent	7	57.1%	4	100.0%

**Evorpacept
demonstrated higher
response rate
at higher dosing**


MDS TRIAL: ALX148 + AZACITIDINE MECHANISM OF ACTION

ALX148
in
MDS



ALX148 increases pro-phagocytic signal provided by azacitidine

CD47 BLOCKADE IS CLINICALLY VALIDATED WITH SIGNIFICANT OPPORTUNITY TO IMPROVE ON MAGROLIMAB

Best Overall Response	R/R AML/MDS 5F9 mono N=10
ORR	1 (10%)
CR	0 
CRi	0
PR	0
MLFS/ marrow CR	1 (10%)
HI	-
SD	7(70%)
PD	2 (20%)


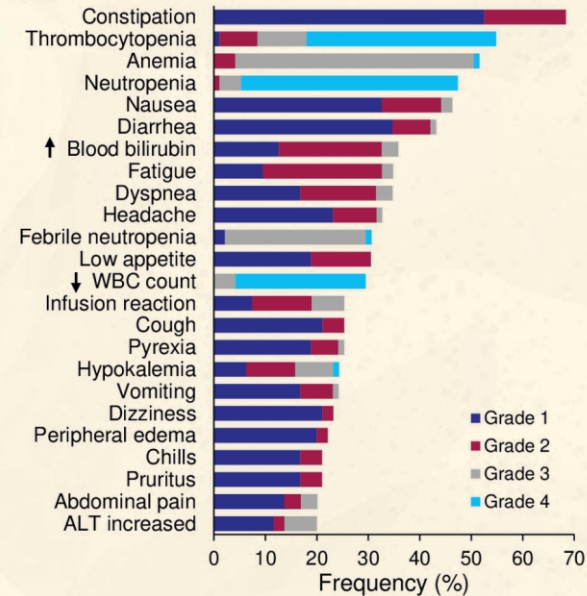
Outcome	All (N = 95)*
ORR, % [†]	74.7
CR, % (95% CI) 	32.6 (23.4, 43.0)
Marrow CR, %	31.6
Any HI, %	58.9
Marrow CR with HI, %	16.8
SD with HI, %	10.5
DCR, median (95% CI), mo	11.1 (7.6, 13.4)
Time to CR, median (range), mo	3.7 (1.7, 7.2)
DOR, median (95% CI), mo	9.8 (8.8, 12.9)
Time to OR, median (range), mo	1.9 (0.7, 10.9)
Conversion to RBC transfusion independence, n/N (%) [‡]	13/37 (35.1)
PFS, median (95% CI), mo	11.6 (9.0, 14.0)
OS, median (95% CI), mo	NR (16.3, NR)

Figure 3. TEAEs by Grade (N = 95)*



Magrolimab monotherapy⁽¹⁾

Magrolimab with azacitidine in 1L higher risk MDS⁽²⁾
38% received 30 mg/kg QW and 59% 30 mg/kg Q2W
magrolimab maintenance dose

- Gr3/4 TEAE (all causality): 47% anemia; 46% neutropenia; 46% thrombocytopenia
- 60% of ≥Gr3 TEAE related to magrolimab
- Gr5 TEAE (all causality): intracranial hemorrhage, myocardial ischemia, leukemia, pulmonary embolus, sepsis, pneumonia, COVID19

**CD47 blocker 5F9 (magrolimab) shows complete responses only when paired
with azacitidine, and causes frequent incidence of treatment-related, high-grade cytopenia**

CLINICAL ACTIVITY OF HISTORICAL BENCHMARKS IN PATIENTS WITH MDS

Population	N	ORR	CRR	mOS (m)
1L Phase 3 AZA-002: 1L HR-MDS ¹ Azacitidine	179	29%*	17%	24.5
Retrospective analysis: 1L HR-MDS with TP53 mutation and complex cytogenetics ² Azacitidine	261	~63%	~22%	10.7
2L Phase 2: 2L MDS ⁴ Guadecitabine	56	14%	4%	7.1
2L+ Phase 1b: ≥2L MDS ³ Venetoclax + azacitidine	38	40%	8%	-

ASPEN-02 MDS TRIAL: DESIGN AND PATIENT BASELINE CHARACTERISTICS

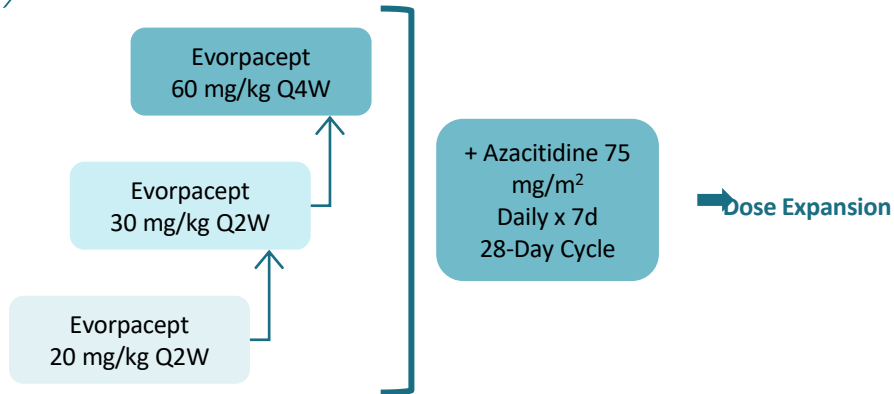


Patients:

Relapsed/refractory and treatment naïve higher risk MDS (IPSS-R >3.5)



Treatment:



Endpoint:

- safety of combination

Patient Baseline Characteristics

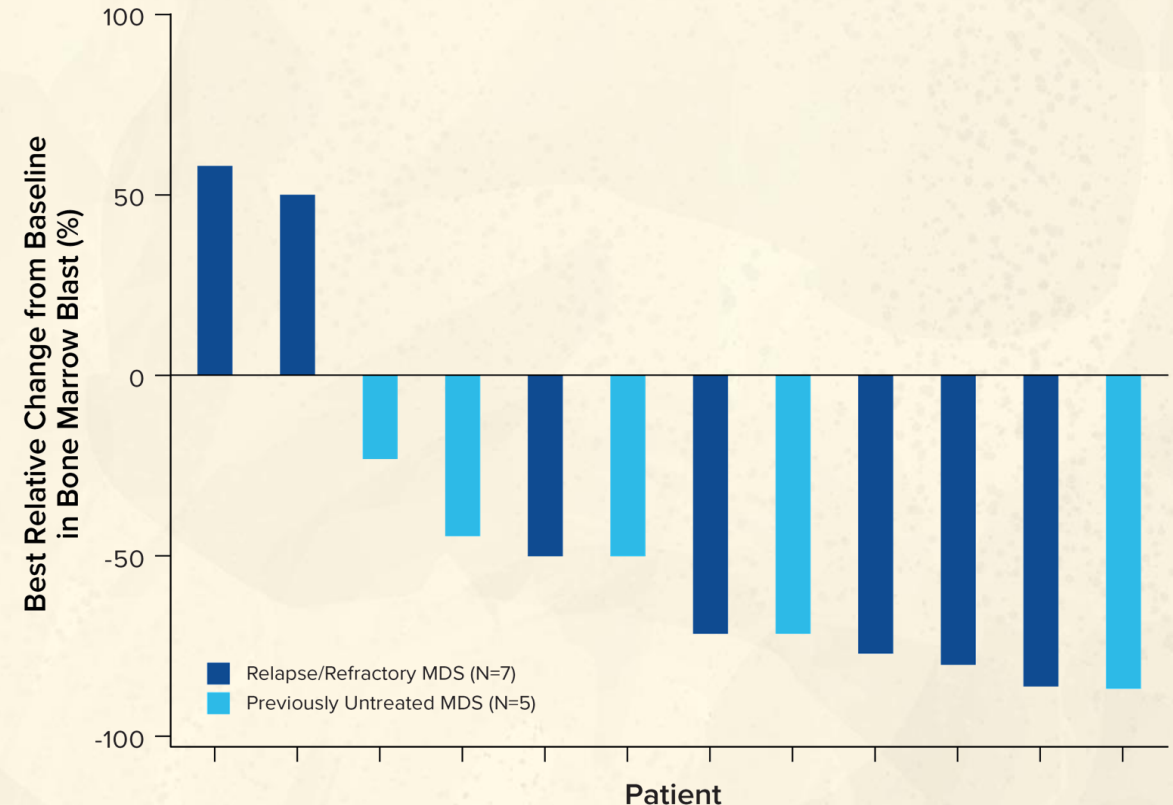
		evorpaccept + azacitidine (N=22)
Median age, years (range)		70.5 (56 – 81)
Sex, n	F	8
	M	14
Race, n	White	17
	Black	4
	Unknown	1
ECOG PS, n	0	6
	1	16
	2	0
MDS Status, n	Previously untreated HR-MDS	9
	• Therapy related	6
	Relapsed/Refractory MDS	13
	• Prior HMA treatment	13
IPSS-R Score	Mean	6.0
	Median	5.8
	Min-Max	1.0-10.0
Mutation Status, n (%)	TP53	8 (36%)
	ASXL1	4 (18%)
	TET2	3 (14%)
	DNMT3A	2 (9%)
	SF3B1	1 (4.5%)
	SRSF2	1 (4.5%)
	RUNX1	1 (4.5%)
Cytogenetic Risk at Diagnosis, n (%)	Very Good	0
	Good	2 (9%)
	Intermediate	0
	Poor	2 (9%)
	Very Poor	8 (36%)
	Not Available	10 (45%)

Data as of October 25, 2021

ASPEN-02 PHASE 1A MDS: EVORPACEPT + AZACITIDINE FOR PREVIOUSLY UNTREATED HIGHER RISK (HR) MDS AND RELAPSED/REFRACTORY MDS

Initial Patients' Data Presented at ASH 2021

	Previously Untreated HR-MDS (N=6)	Previously Untreated HR-MDS with TP53 mutation (N=5)	Relapsed/Refractory MDS (N=9) [#]
ORR	3	3	5 *
CR	2	2	0
PR	0	0	0
Marrow CR	1 with HI	1 with HI	5 *
HI	0	0	0
SD	2	1	2
PD	1	1	1



Data Cutoff 25Oct2021; Response evaluable population (n=15); *includes 3 unconfirmed responses; [#]One subject with an unrelated G5 event prior to first disease assessment; On graphic, 2 subjects (1 Relapsed/Refractory and 1 Previously Untreated) with missing data and the previously described subject with an unrelated G5 event not represented.

ORR – Objective response rate; CR – Complete response; PR – Partial response; HI – Hematologic improvement; SD – Stable disease; PD – Disease progression

MDS TRIAL PLANS, ASPEN-02

Phase 1 Dose Escalation: Accrual Complete



Patients:

N~18

Relapsed/refractory and treatment naïve
higher risk MDS (IPSS-R >3.5)



Treatment:

evorpacept
20 mg/kg (Q2W)
30 mg/kg (Q2W)
or 60 mg/kg (Q4W)
+
azacitidine



Endpoint:

- safety of combination

Phase 1 Dose Expansion: Open for Accrual



Patients:

N~40

Treatment naïve higher risk MDS
(IPSS-R >3.5)



Treatment:

evorpacept
40 mg/kg (Q4W)
or 60 mg/kg (Q4W)
+
azacitidine



Endpoint:

- safety of combination

Phase 2 Randomized Trial



Patients:

Treatment naïve higher risk MDS
(IPSS-R >3.5)



Treatment:

evorpacept
recommended phase 2 dose
+
azacitidine
vs.
azacitidine



Endpoint:

- complete response rate (CRR)

ASPEN-05 AML TRIAL: DESIGN AND PATIENT BASELINE CHARACTERISTICS

Phase 1 Design

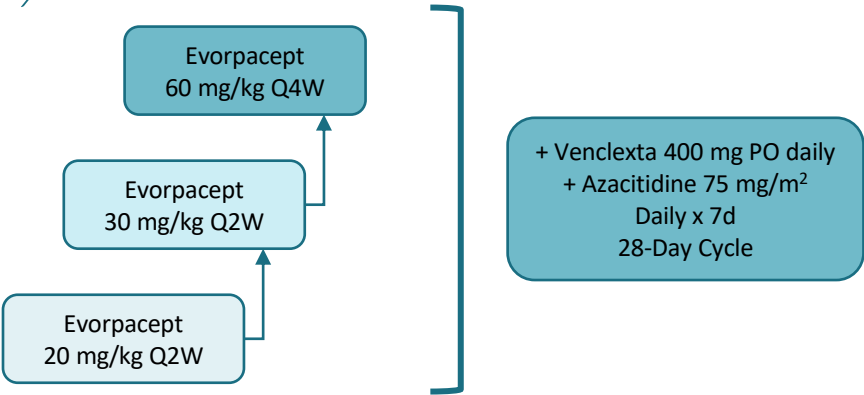


Patients:

Relapsed/refractory and newly diagnosed AML who are not considered suitable for intensive induction therapy



Treatment:



Endpoint:

- safety of combination

Patient Baseline Characteristics

		evorpacept + azacitidine + Venclexta (N=14)
Age, Years (median, range)		71 (50-82)
Sex, n	Male	10
	Female	4
Race, n	White	8
	Black or African American	2
	Native Hawaiian or Other Pacific Islander	1
	Asian	3
AML Status, n	Relapsed or Refractory	11
	• Number of Prior Treatment Regimens (median, range)	1 (1-2)
	• Prior Venetoclax, n	9
	• Venetoclax-Naïve, n	2
	• Prior Hypomethylating Agents, n	5
	Newly Diagnosed	3
WHO AML Classification at Screening, n	AML with Myelodysplasia-Related Changes	5
	Therapy-Related Myeloid Neoplasms	2
	AML, NOS	4
	Unknown/Missing	3
Cytogenetic Risk at Screening, n	Intermediate	1
	Adverse	13
Bone Marrow Myeloblast Percentage at Baseline (median, range)		27 (5-84)
Mutation Status, n (%)	DNMT3A	3 (21%)
	RUNX1	2 (14%)
	ASXL1	2 (14%)
	TP53 Mutation	11 (79%)
	Other	8 (57%)

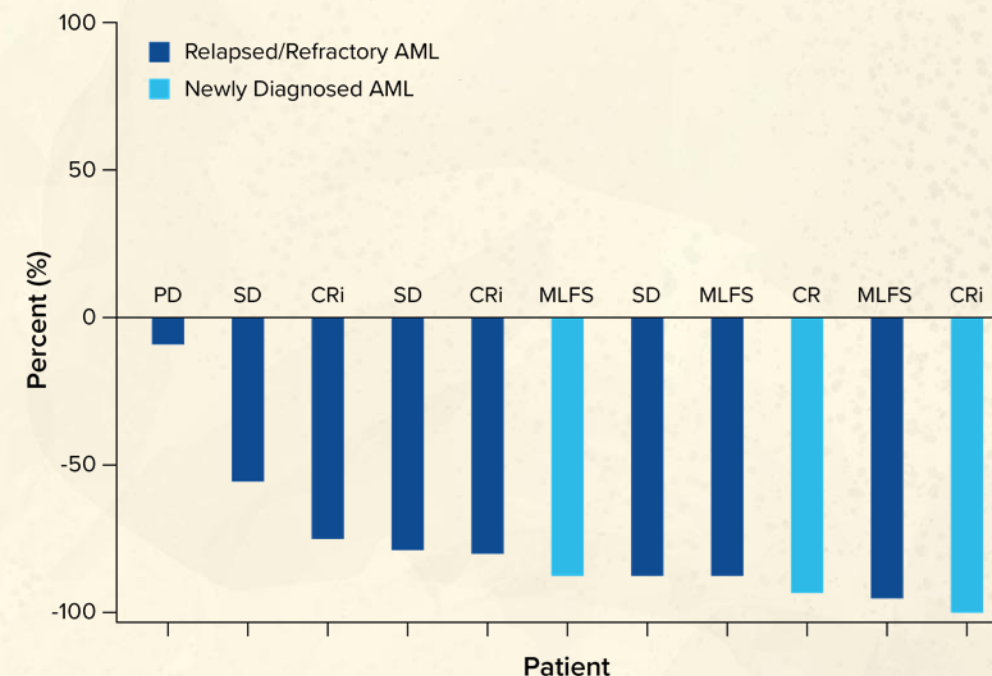
ASPEN-05 AML TRIAL: EVORPACEPT + AZACTIDINE + VENCLEXTA FOR NEWLY DIAGNOSED AND RELAPSED/REFRACTORY AML

Initial Patients' Data Presented at ASH 2022

Best Overall Response (Response Evaluable Patients)[#]

	Newly Diagnosed (N=3)	Rel/Ref (N=10)		Overall (N=13)* n (%)
		VEN-Naïve (N=2)	Prior VEN (N=8)*	
ORR	3	2	2	7 (54)
CR	1	0	0	1 (8)
CRi	1	2	0	3 (23)
PR	0	0	0	0
MLFS	1	0	2	3 (23)
SD	0	0	4	4 (31)
PD	0	0	1	1 (8)

Best Percent Change in Bone Marrow Blast % from Baseline (Available Samples)



Note: One subject with missing data, two subjects with no post-baseline disease assessment (1 DLT, 1 death).

AML TRIAL PLANS, ASPEN-05

Phase 1 Dose Escalation and Expansion: On pause after dose escalation



Patients: Relapsed/refractory AML or previously untreated AML who are not considered suitable for intensive induction therapy



Treatment

evorpacept

20 mg/kg (Q2W)
30 mg/kg (Q2W)
or 60 mg/kg (Q4W)

+ Venclexta
+ azacitidine



Endpoint: • safety of combination, recommended phase 2 dose

Phase 2:



Patients: Previously untreated AML who are not considered suitable for intensive induction therapy



Treatment

evorpacept

recommended phase 2 dose

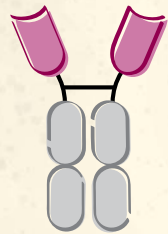
+ Venclexta
+ azacitidine



Endpoint: • complete remission rate

ALX ONCOLOGY MILESTONES AND FINANCIAL INFORMATION

UPCOMING MILESTONES



Evorpcept

**Early clinical and
research pipeline**



2023

Gastric Cancer (Phase 2)

ASPEN-06 Randomized gastric/GEJ cancer trial data presentation in 2H 2023

MDS (Phase 1b)

ASPEN-02 MDS dose optimization trial presentation in 2H 2023

AML (Phase 1b)

ASPEN-05 Initiation of AML dose optimization trial in 2H 2023

Urothelial Carcinoma (Phase 1)

ASPEN-07 Initiate dosing of urothelial carcinoma with enfortumab vedotin-ejfv trial in 1H 2023

Continue Supporting Ongoing and Planned Clinical Collaborations

- Breast cancer (I-SPY, Zymeworks)
- NHL, CRC, Ovarian (Investigator Sponsored Trials)

ALTA-002 (Phase 1) initiation

File IND in 1H 2023

ADC pipeline

Identify clinical development candidates in 2H 2023

2024

Head & Neck Cancer (Phase 2)

ASPEN-03 Completion of randomized HNSCC trial with pembrolizumab

Head & Neck Cancer (Phase 2)

ASPEN-04 Completion of randomized HNSCC trial with pembrolizumab and chemo

MDS (Phase 2)

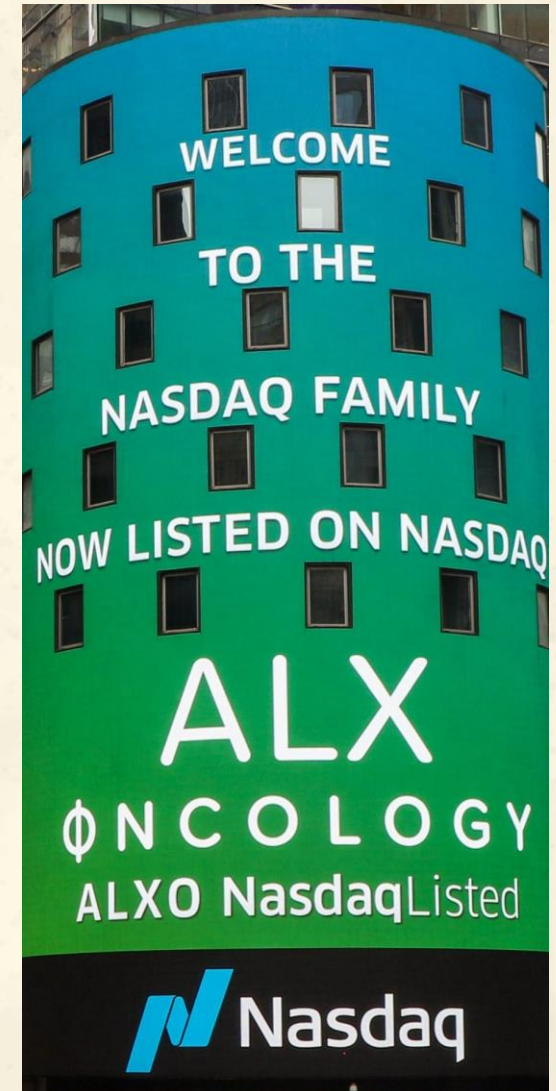
Initiation of randomized MDS trial

Gastric Cancer (Phase 3)

ASPEN-06 Initiation of randomized gastric trial

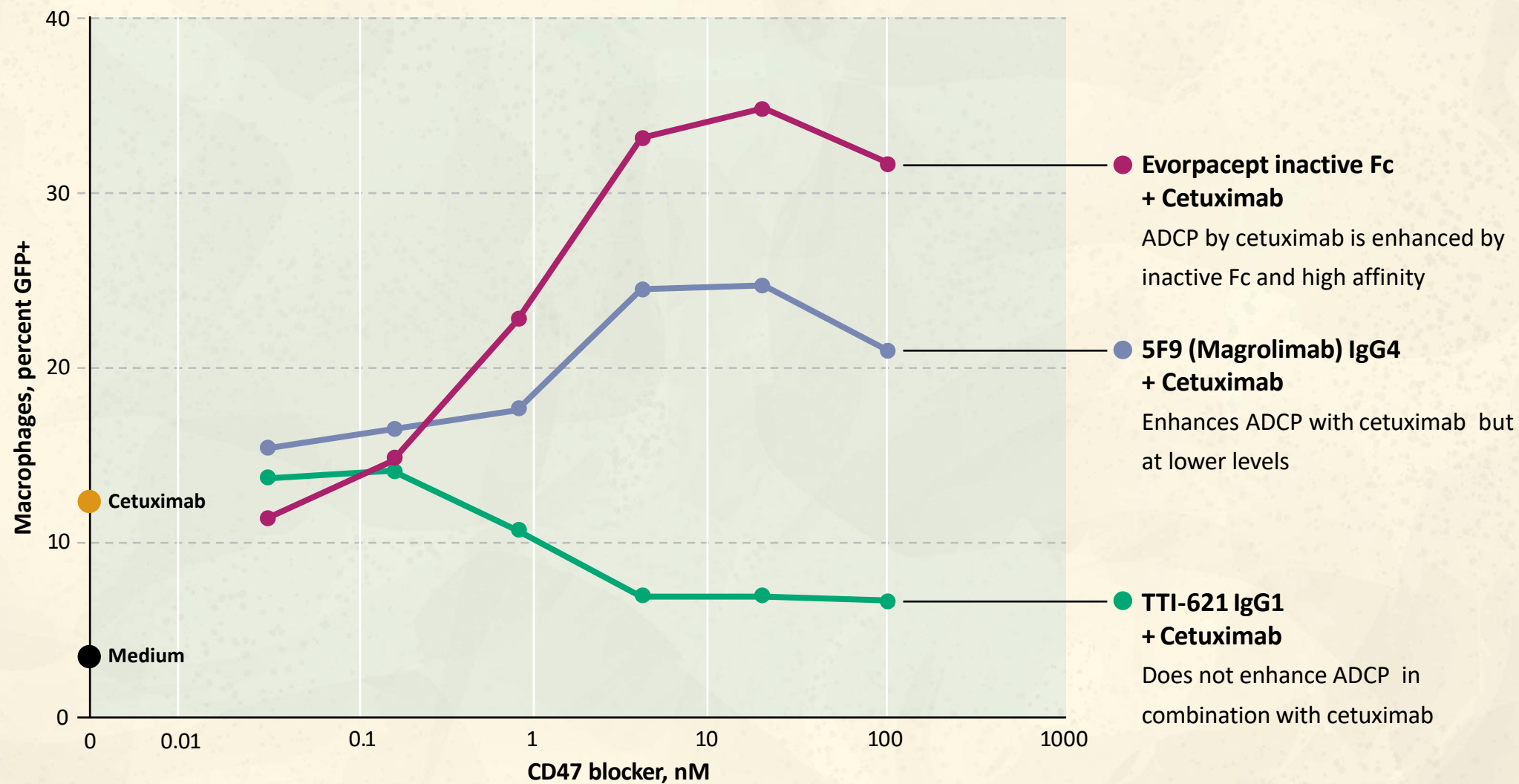
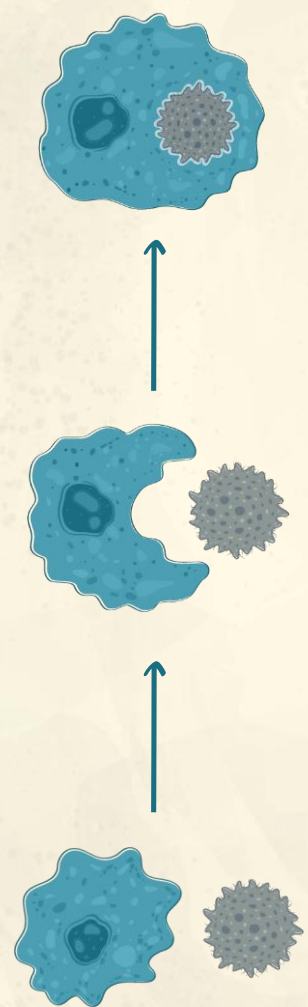
FINANCIAL INFORMATION

- Approximately \$545M in net proceeds raised to date including:
 - \$170 million IPO in July 2020 and \$195 million follow on in December 2020
- \$100M loan facility
 - \$10M drawn as of October 31, 2022
- Cash, cash equivalents and investments balance as of December 31, 2022:
 - \$282.9M
- Expected cash runway through mid-2025

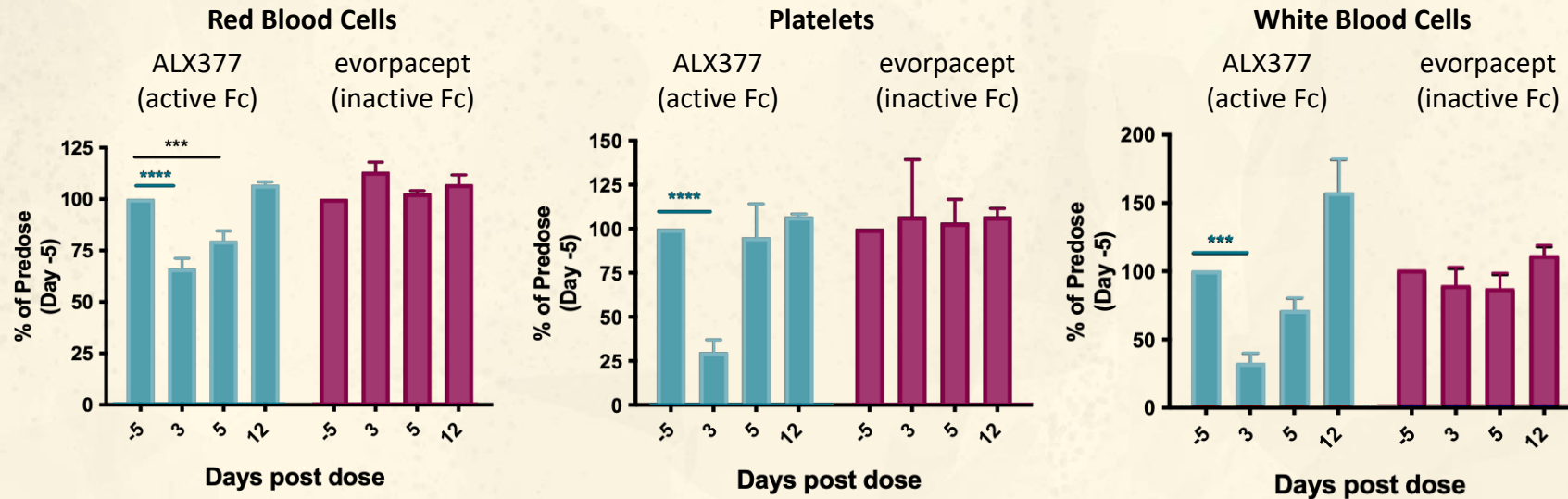


APPENDIX

EVORPACEPT DEMONSTRATES SUPERIOR PHAGOCYTOSIS



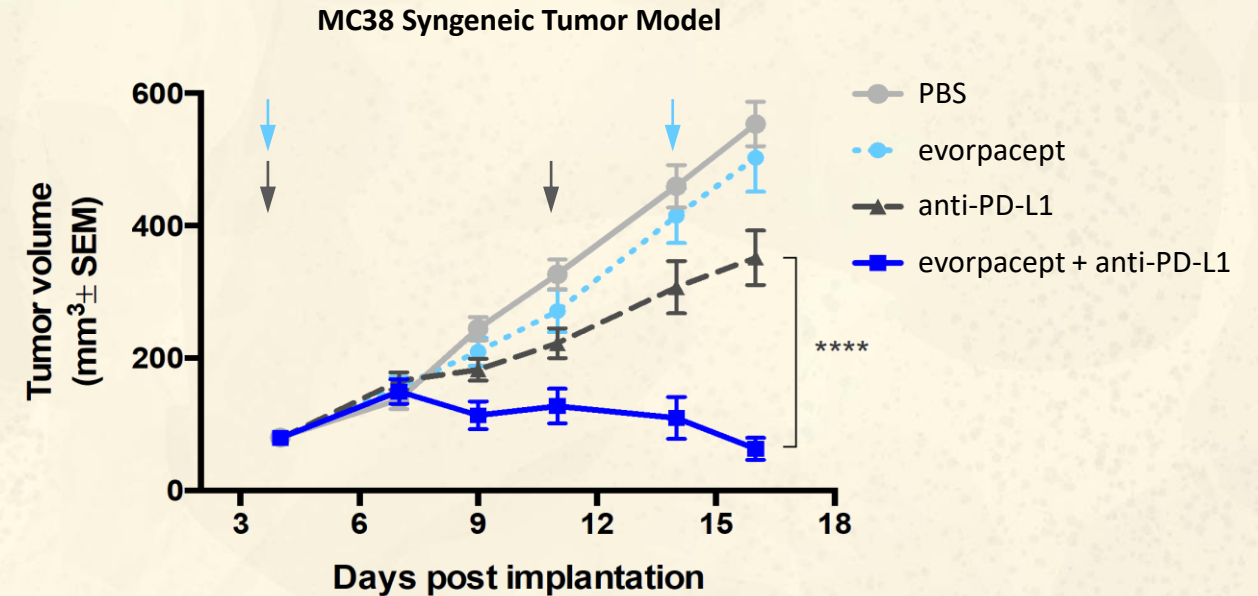
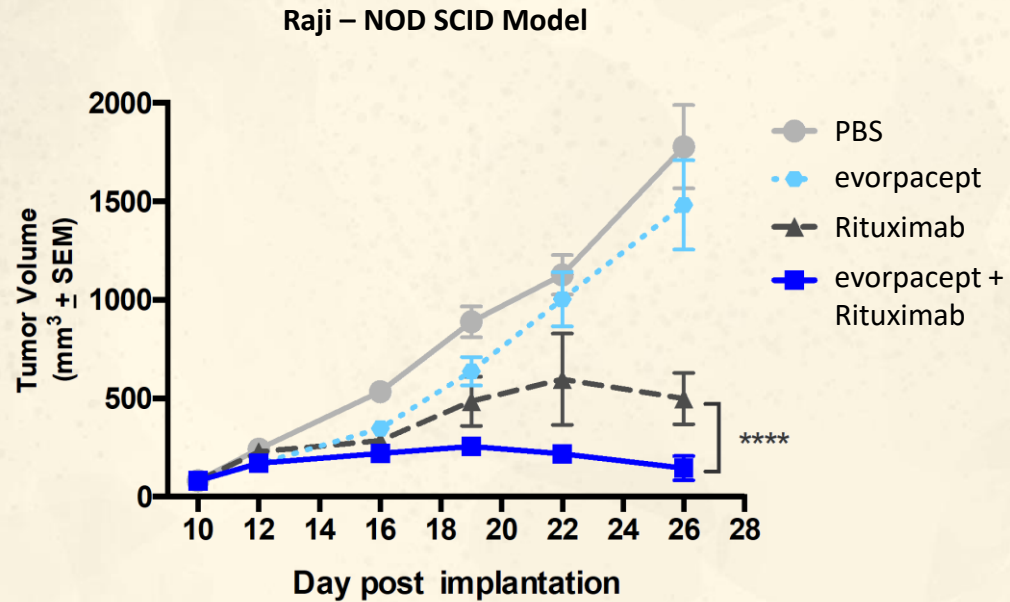
INACTIVE FC SHOWS REDUCED CYTOPENIA IN MICE IN VITRO



Inactive Fc is the core determinant of safety profile

Mouse cross-reactivity allows for safety and efficacy testing in mouse models

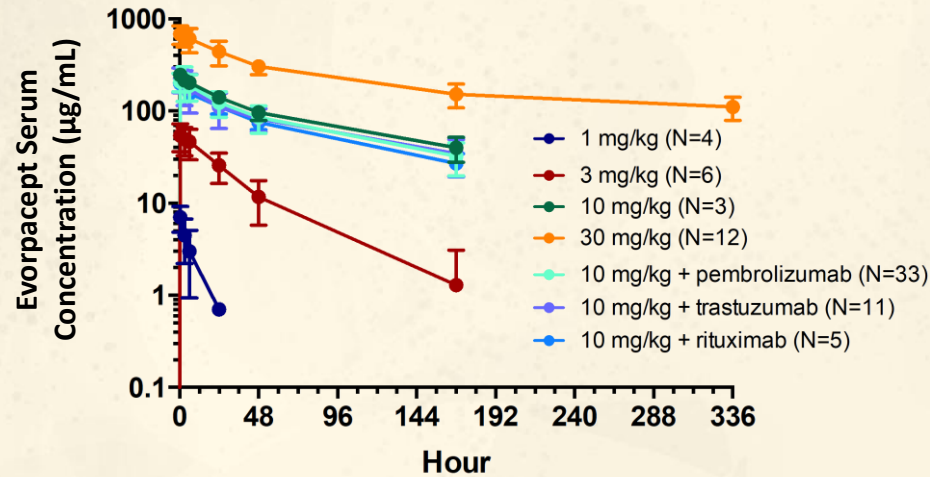
COMBINATION EFFICACY WITH TAA (RITUXIMAB) ANTIBODIES AND CHECKPOINT INHIBITOR (ATEZOLIZUMAB)



Mouse cross-reactivity allows for better clinical translation of preclinical models:
presence of CD47 sink and mouse models with intact immune system

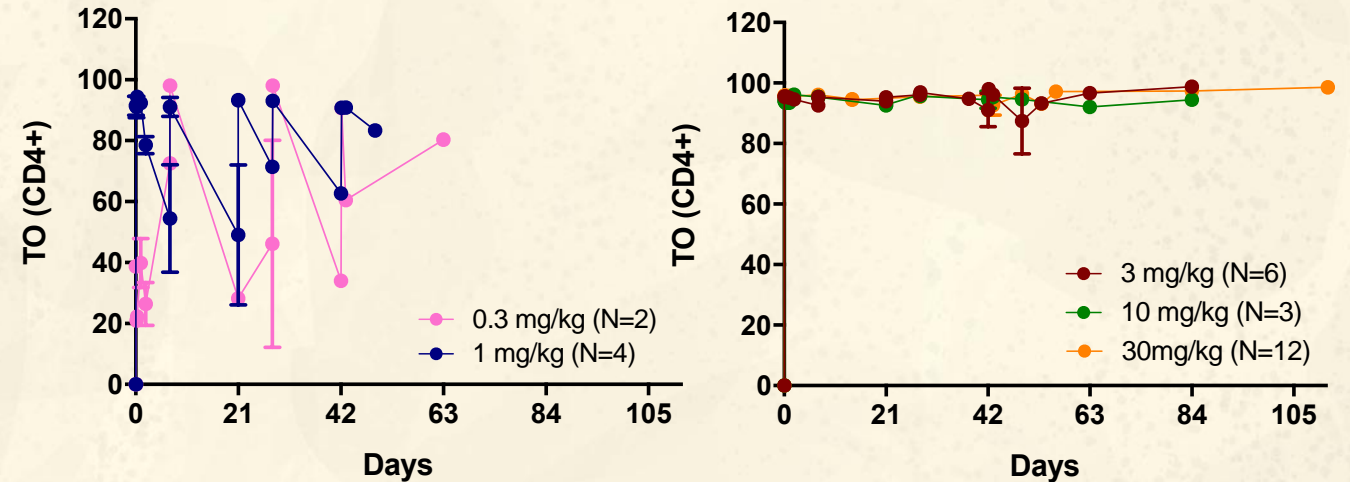
EVORPACEPT CLINICAL PHARMACOKINETICS AND CD47 TARGET OCCUPANCY

Evorpaccept Serum Levels for Cycle 1 Day 1



- Steady-state half-life of evorpaccept at 10 mg/kg QW is predicted to be ~30 days.
- Evorpaccept PK profile is not impacted by combination drugs.

CD47 Target Occupancy by Evorpaccept



- Near complete CD47 target occupancy (TO) by evorpaccept is maintained at ≥ 3 mg/kg QW across dosing interval
- 0.3 and 1 mg/kg QW show near complete target occupancy at peak, but not at trough

NHL TOLERABILITY

Selected hematologic, treatment related adverse events	evorpacept + Rituximab (N=33) ¹		CC-90002 + Rituximab (n=26) ²		5F9 (magrolimab) + Rituximab (n=115) ³	
	Total % (n)	≥Grade 3	Total % (n)	≥Grade 3	Total %	≥Grade 3
Neutropenia	6% (2)	6% (2)	50% (13)	39% (10)	~13%	~7%
Thrombocytopenia/ Decreased Platelets	-	-	35% (9)	23% (6)	~20%	~13%
Anemia	6% (2)	3% (1)	12% (3)	4% (1)	~30%	~15%

¹ASH 2020 Abstract 3016

²ASH 2019 Abstract 4089

³EHA 2019 Abstract S867

Evorpacept:
Tolerability profile
compares favorably to
other CD47 blockers

ASPEN-01 NHL PROOF-OF-PRINCIPLE TRIAL

Phase 1b NHL cohorts



relapsed/Refractory NHL,
prior regimen with Rituximab



Treatment:

evorpacept 10 or 15 mg/kg
once a week (QW)
+
Rituximab 375 mg/m² once a week for
4 weeks, once monthly
for 8 months

		evorpacept 10 mg/kg QW + Rituximab (n=22)	evorpacept 15 mg/kg QW + Rituximab (n=11)
Primary Disease, n	Follicular	5	3
	Marginal Zone (MZL)	2	1
	Mantle Cell (MCL)	4	1
	DLBCL	11	6
Median Age, Years (range)		66 (32-80)	64 (53-78)
Sex, n	M	17	6
	F	5	5
Race, n	Asian	18	9
	White	4	2
ECOG, PS, n	0	7	2
	1	15	9
Median Prior Therapy, n (range)		3 (1-7)	3 (1 -5)

Data Cutoff October 1, 2020

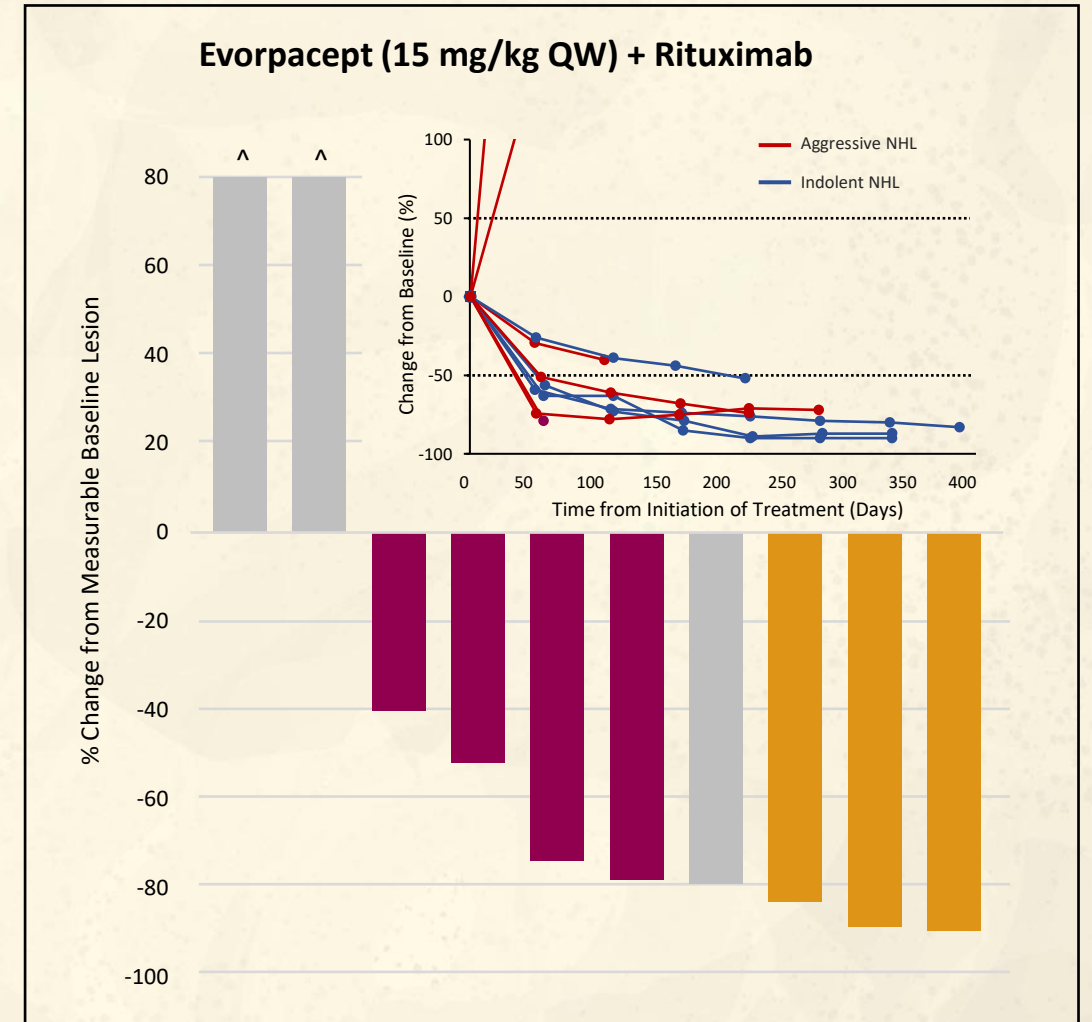
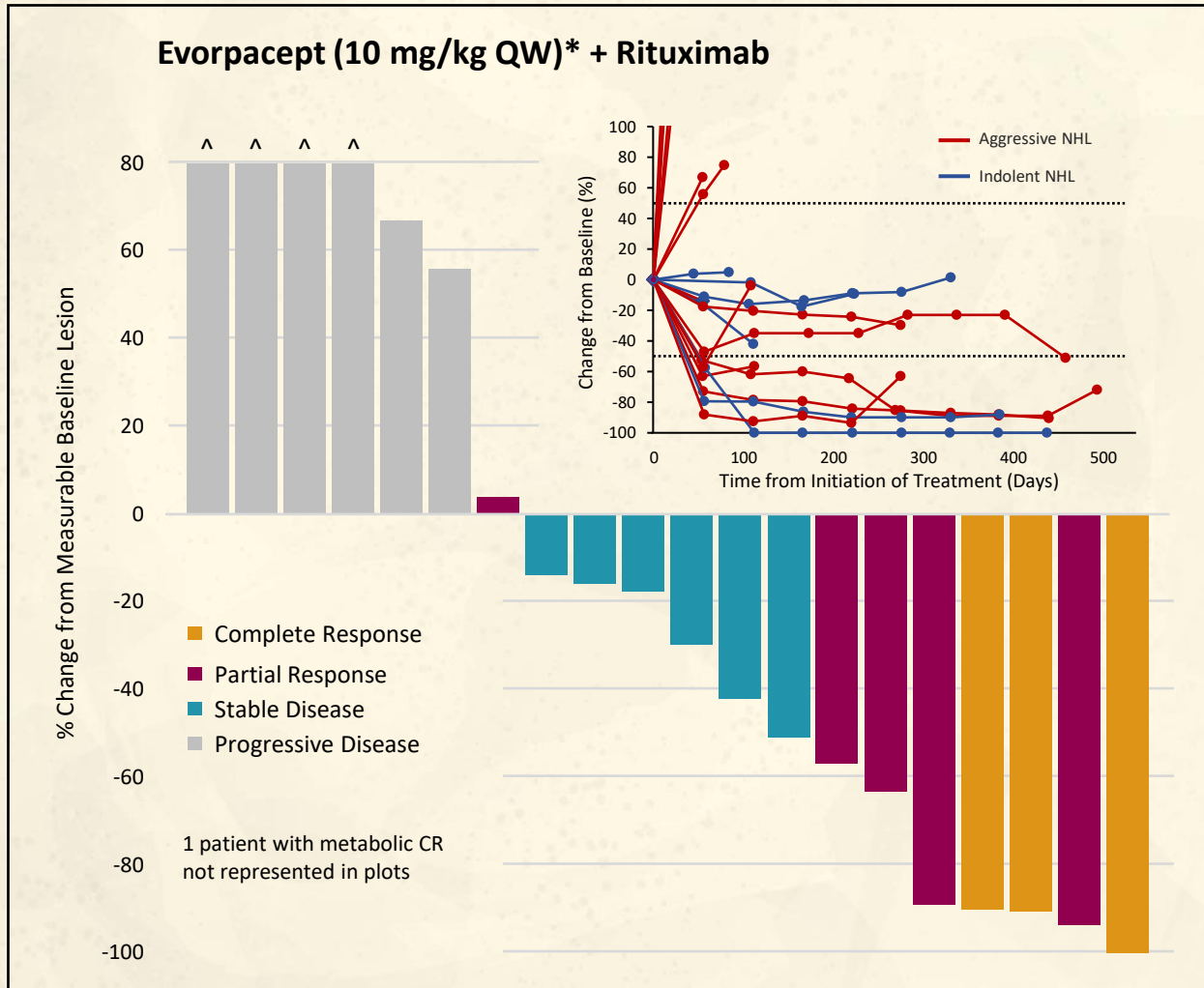
ASPEN-01 NHL: PRELIMINARY CLINICAL TOLERABILITY

evorpacept + Rituximab (N=33)

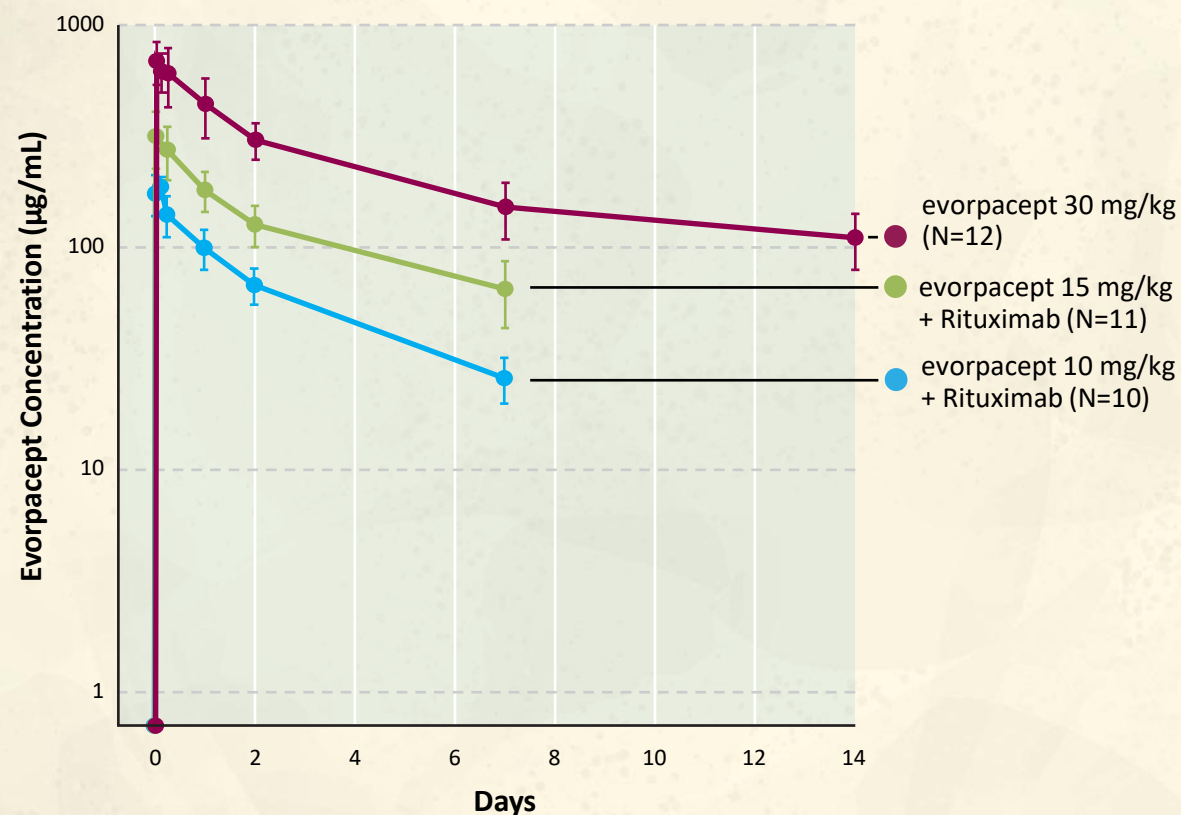
Treatment Related Adverse Event	Total n (%)	≥Grade 3 n (%)
Rash	8 (24.2)	—
Fatigue	4 (12.1)	—
Nausea	2 (6.1)	—
Neutrophil Count Decreased	2 (6.1)	2 (6.1)
Anemia	2 (6.1)	1 (3.0)
Myalgia	2 (6.1)	—
Pruritus	2 (6.1)	—

Data Cutoff: October 1, 2020

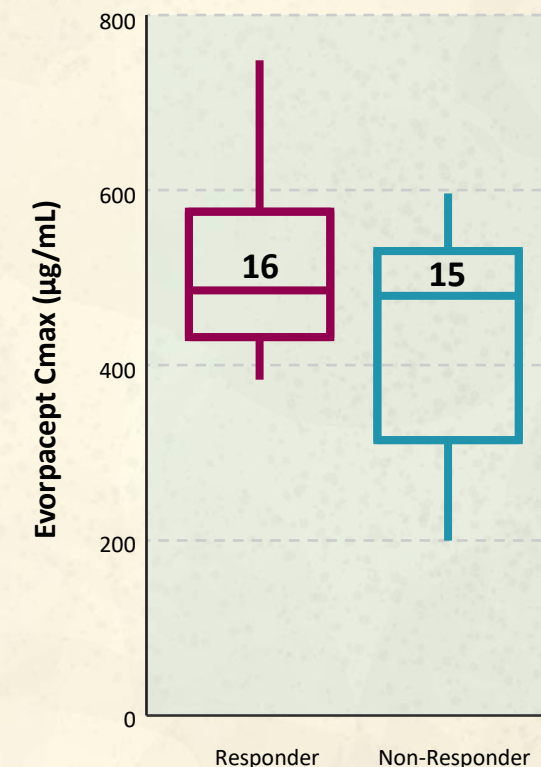
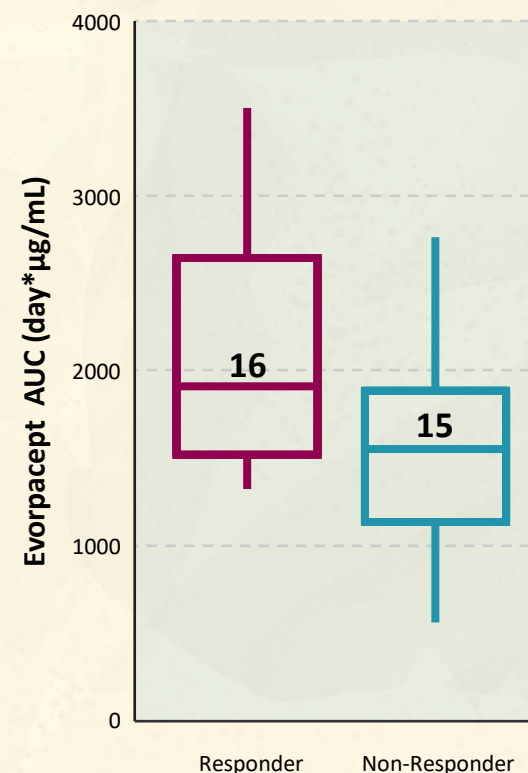
ASPEN-01 NHL: CLINICAL ACTIVITY OF EVORPACEPT + RITUXIMAB BY PATIENT



ASPEN-01 NHL: EVORPACEPT CLINICAL PHARMACOKINETICS AND EXPOSURE-RESPONSE ANALYSIS



Evorpacept concentration-time profiles following first IV infusion at Cycle 1 Day 1 as single agent or in combination with Rituximab.



*A significant improvement in patients with clinical response (PR,CR) with increased evorpacept exposure (AUC; $p = 0.023$) was observed across the exposure range evaluated (10 mg/kg QW - 15 mg/kg QW).

NON-HODGKIN LYMPHOMA PROOF-OF-PRINCIPLE SUMMARY

evorpacept
in
NHL



**Other agents in CD47 class
reduced dosing leading to reduced
responses**



**Higher dosing enabled by
evorpacept tolerability profile**



**Higher dosing of evorpacept
led to higher responses**

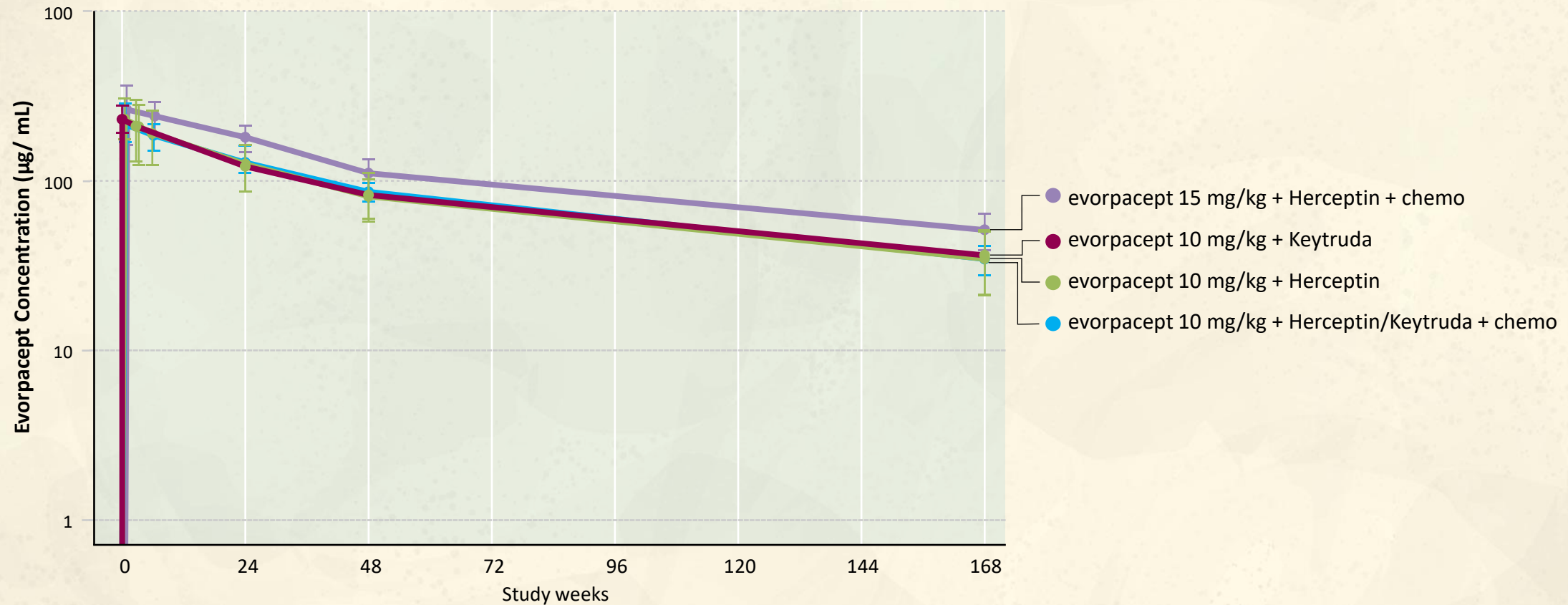
CLINICAL ACTIVITY OF EVORPACEPT COMBINATIONS IN RESPONSE EVALUABLE PATIENTS WITH ≥2L HER2 POSITIVE GC CANCER

HER2 GC Population	N	ORR	DOR (m) [95% CI]	PFS (m) [95% CI]	OS (m) [95% CI]	OS rate at 12 m	Follow up (m) [95% CI]
2L GC evorpaccept + Herceptin + Cyramza + paclitaxel	18	72%	14.8 [3.9–NR]	17.1 [5.4–NR]	17.1 [9.8–NR]	79%	14.5 [7.2–19.0]
≥2L Gastric ramucirumab/ paclitaxel RAINBOW ¹	330	28%	4.4 [IQR 2.8–7.5]	4.4 [4.2–5.3]	9.6 [8.5–10.8]	40%	
≥2L Gastric trastuzumab/ram/paclitaxel ²	50	52%	5.1 [3.3–6.9]	7.4 [6.5–8.3]	13.6 [9.6–17.5]	-	22.9
2L Gastric trastuzumab- Deruxtecan DESTINY 02 ³	79	38%	8.1 [4.1–NE]	5.5 [4.2–7.3]	-	-	5.7
≥3L Gastric trastuzumab- Deruxtecan DESTINY 01 ⁴	126	41%	11.3 [5.6–NE]	5.6 [4.3–6.9]	12.5 [9.6–14.3]	52%	
≥2L Gastric evorpaccept (10 mg/kg) + Herceptin	19	21%	8.7 [5.6; NR]	2.2 [1.9 ; 5.5]	8.1 [3.4 ; 12.6]	38%	27.0
≥3L Gastric Irinotecan or Paclitaxel DESTINY 01 Control Arm ⁴	62	11%	3.9	3.5	8.4	29%	

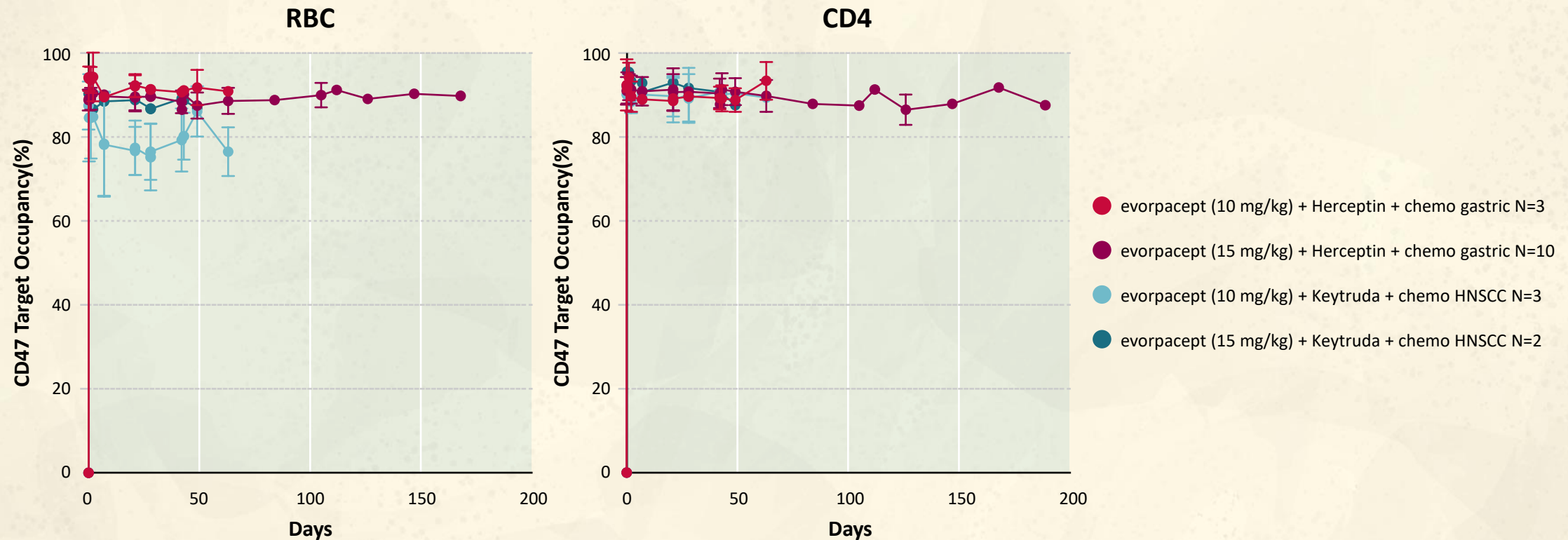
ASPEN-01 PHASE 1B ≥2 LINE GC TRIAL: EVORPACEPT + HERCEPTIN + CYRAMZA + PACLITAXEL TREATMENT EMERGENT ADVERSE EVENTS

Grade	Evorpacept + Trastuzumab + Ramucirumab + Paclitaxel (N=18) / Adverse Event, n (%)					
	ALL Causality			Evorpacept - related		
Evorpacept Dose QW	G1-2	G3	G4	G1-2	G3	G4
Neutrophil Count Decreased	3 (17)	5 (28)	3 (17)	–	–	–
Epistaxis	9 (50)	–	–	–	–	–
Peripheral Neuropathy / Peripheral Sensory Neuropathy	8 (44)	1 (6)	–	–	–	–
Decreased Appetite	8 (44)	–	–	–	–	–
Fatigue	7 (39)	1 (6)	–	2 (11)	–	–
Anemia	3 (17)	4 (22)	–	1 (6)	–	–
Hypertension	–	6 (33)	–	–	–	–
Abdominal Pain / Abdominal Pain Upper	5 (28)	–	–	1 (6)	–	–
Headache	5 (28)	–	–	1 (6)	–	–
Stomatitis	5 (28)	–	–	1 (6)	–	–
Alanine Aminotransferase Increased	4 (22)	–	–	–	–	–
Alopecia	4 (22)	–	–	–	–	–
Aspartate Aminotransferase Increased	3 (17)	1 (6)	–	–	–	–
Asthenia	3 (17)	1 (6)	–	–	–	–
Diarrhea	4 (22)	–	–	3 (17)	–	–
Insomnia	4 (22)	–	–	–	–	–
Rash/Dermatitis Acneiform	4 (22)	–	–	4 (22)	–	–
Pruritis	3 (17)	–	–	2 (11)	–	–
Urticaria	3 (17)	–	–	3 (17)	–	–
Back Pain	2 (11)	–	–	1 (6)	–	–
Diverticulitis	1 (6)	1 (6)	–	–	–	–
Dysphagia	1 (6)	1 (6)	–	–	–	–
Hypophosphatemia	1 (6)	1 (6)	–	–	–	–
Platelet Count Decreased	1 (6)	1 (6)	–	–	–	–
Hydronephrosis	–	1 (6)	–	–	–	–
Lymphocyte Count Decreased	–	1 (6)	–	–	1 (6)	–
Non-Cardiac Chest Pain	–	1 (6)	–	–	–	–
Urinary Tract Infection	–	1 (6)	–	–	–	–
Vision Blurred	1 (6)	–	–	1 (6)	–	–

EVORPACEPT PK FOLLOWING COMBINATION THERAPIES IS COMPARABLE WITH AND WITHOUT CHEMOTHERAPY



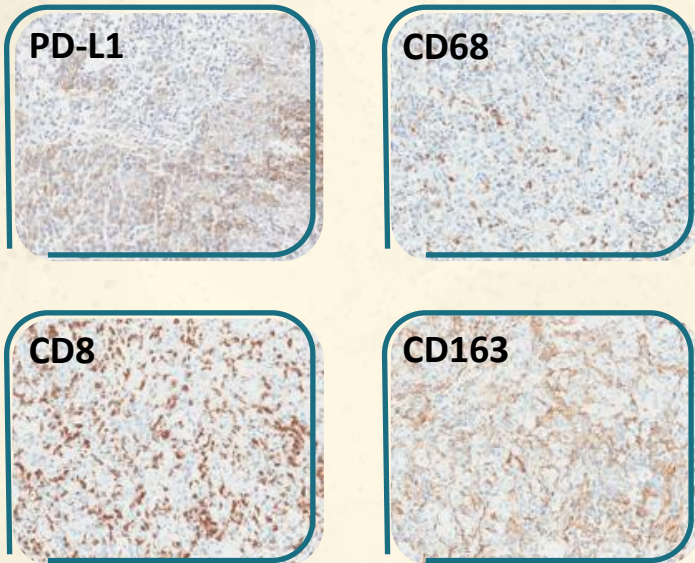
NEAR COMPLETE CD47 TARGET OCCUPANCY IS MAINTAINED THROUGHOUT EVORPACEPT DOSING INTERVAL WHEN COMBINED WITH CHEMOTHERAPY CONTAINING REGIMENS



PRE-TREATMENT IHC RESULTS IN HOT AND COLD TUMORS

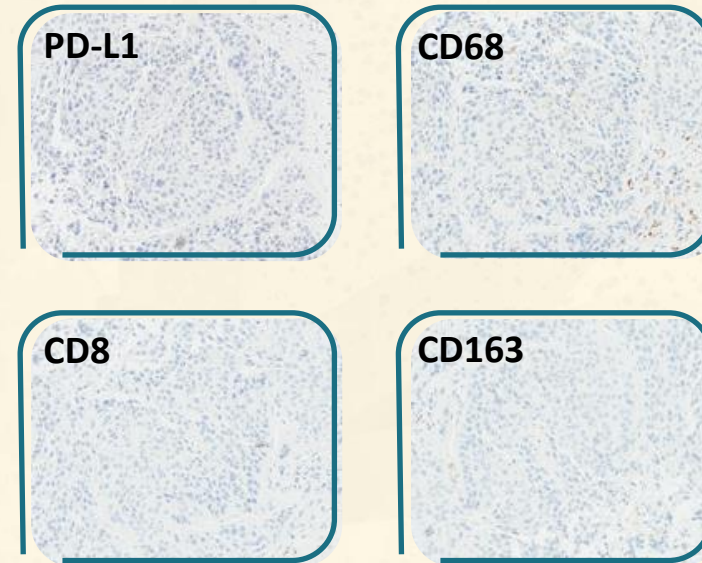
HNSCC PATIENT 1 (PD-L1 POSITIVE) AND PATIENT 2 (PD-L1 NEGATIVE)

Patient 1 Best Overall Response: CR
Immunologically “hot” tumor



Patient 1: HNSCC (CPS 50) characterized as immunologically “hot” with a high density of infiltrating T lymphocytes (CD8) and tumor associated macrophages and myeloid cells (CD68 and CD163).

Patient 2 Best Overall Response: PR
Immunologically “cold” tumor



Patient 2: HNSCC (CPS 0) characterized as immunologically “cold” where immune cells are excluded from the tumor core while present at lower density in the peri-tumoral regions.

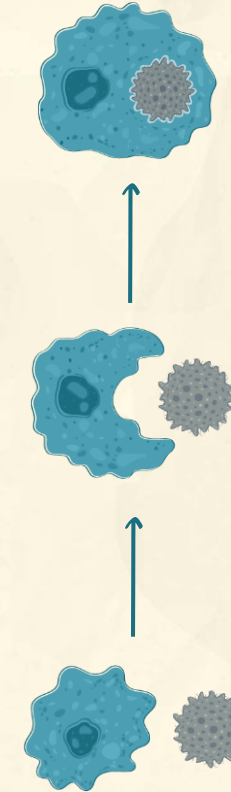
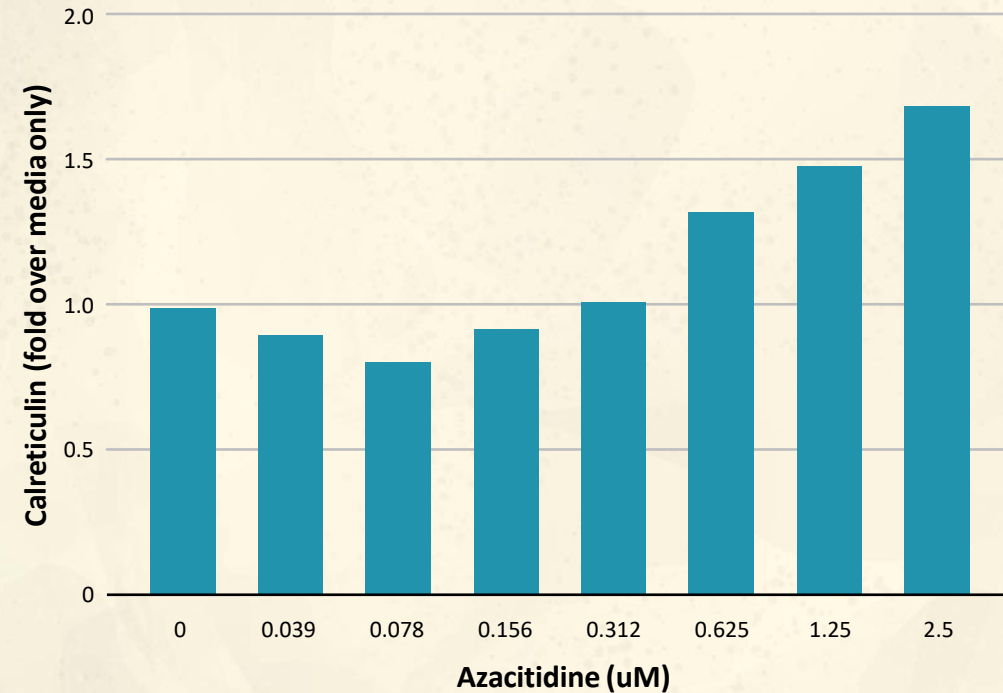
ASPEN-01 PHASE 1B FIRST LINE HNSCC: EVORPACEPT + KEYTRUDA + 5FU/PLATINUM TREATMENT RELATED ADVERSE EVENTS

Evorpacept + Pembrolizumab + 5FU + Platinum (N=13) / Adverse Event, n (%)

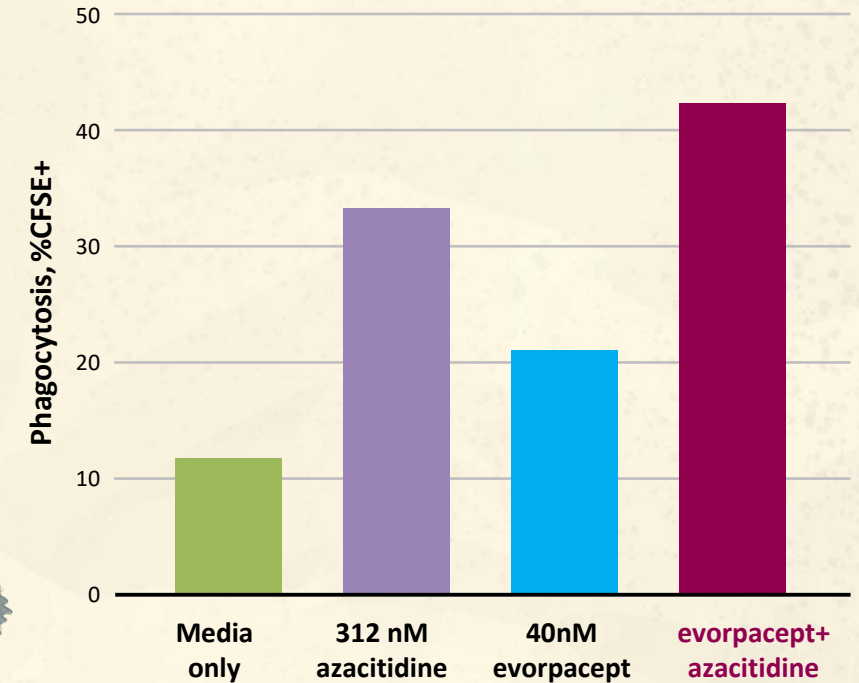
Grade Evorpacept Dose QW	ALL Causality			Evorpacept - Related		
	G1-2	G3	G4	G1-2	G3	G4
Anemia	4 (31)	4 (31)	–	–	1 (8)	–
Nausea	8 (62)	–	–	–	–	–
Stomatitis	7 (54)	1 (8)	–	–	–	–
Neutrophil Count Decreased / Neutropenia	2 (15)	5 (38)	–	1 (8)	–	–
Platelet Count Decreased /Thrombocytopenia	7 (54)	–	–	–	–	–
Fatigue	5 (38)	–	–	1 (8)	–	–
Alanine Aminotransferase Increased	3 (23)	1 (8)	–	–	–	–
Dysphagia	1 (8)	1 (8)	–	–	–	–
Hypersensitivity	1 (8)	–	1 (8)	–	–	1 (8)
Pneumonia	1 (8)	1 (8)	–	–	–	–
Pneumonitis	2 (15)	–	–	1 (8)	–	–
Candida Infection	–	1 (8)	–	–	–	–
Cardiac Tamponade	–	–	1 (8)	–	–	–
Headache	–	1 (8)	–	–	–	–
Pericarditis Constrictive	–	1 (8)	–	–	–	–
Supraventricular Tachycardia	–	1 (8)	–	–	–	–
Tracheal Obstruction	–	1 (8)	–	–	–	–

PRECLINICAL: EVORPACEPT INCREASES PHAGOCYTOSIS OF BACKBONE MDS DRUG, AZACITIDINE

Calreticulin levels on HL60 Cells



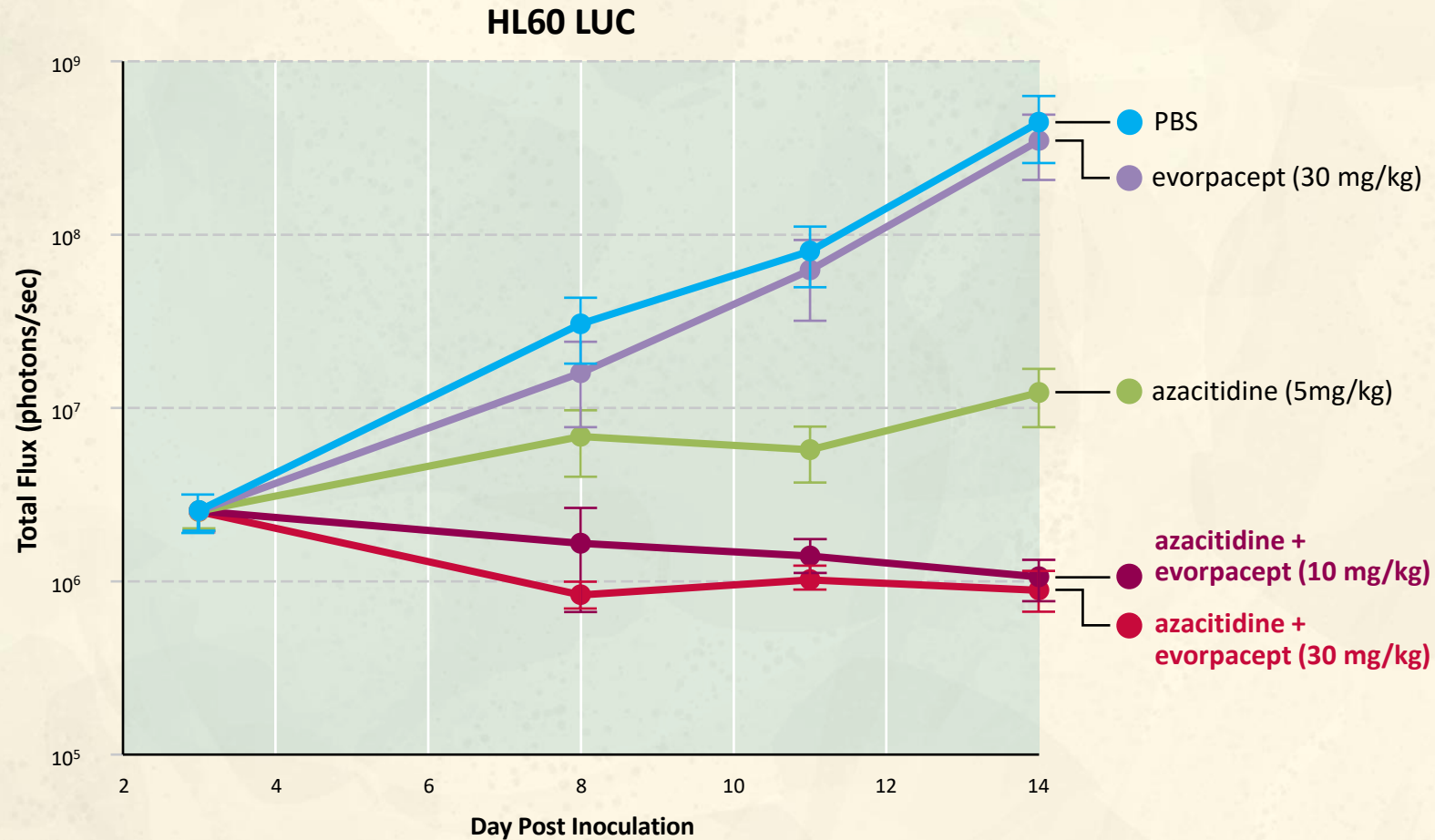
Phagocytosis of HL60 Cells



Azacitidine induces calreticulin display.

Evorpaccept increases phagocytosis in combination with azacitidine.

EVORPACEPT INCREASES TUMOR INHIBITION OF AZACITIDINE



Combination
opportunity in MDS
and AML

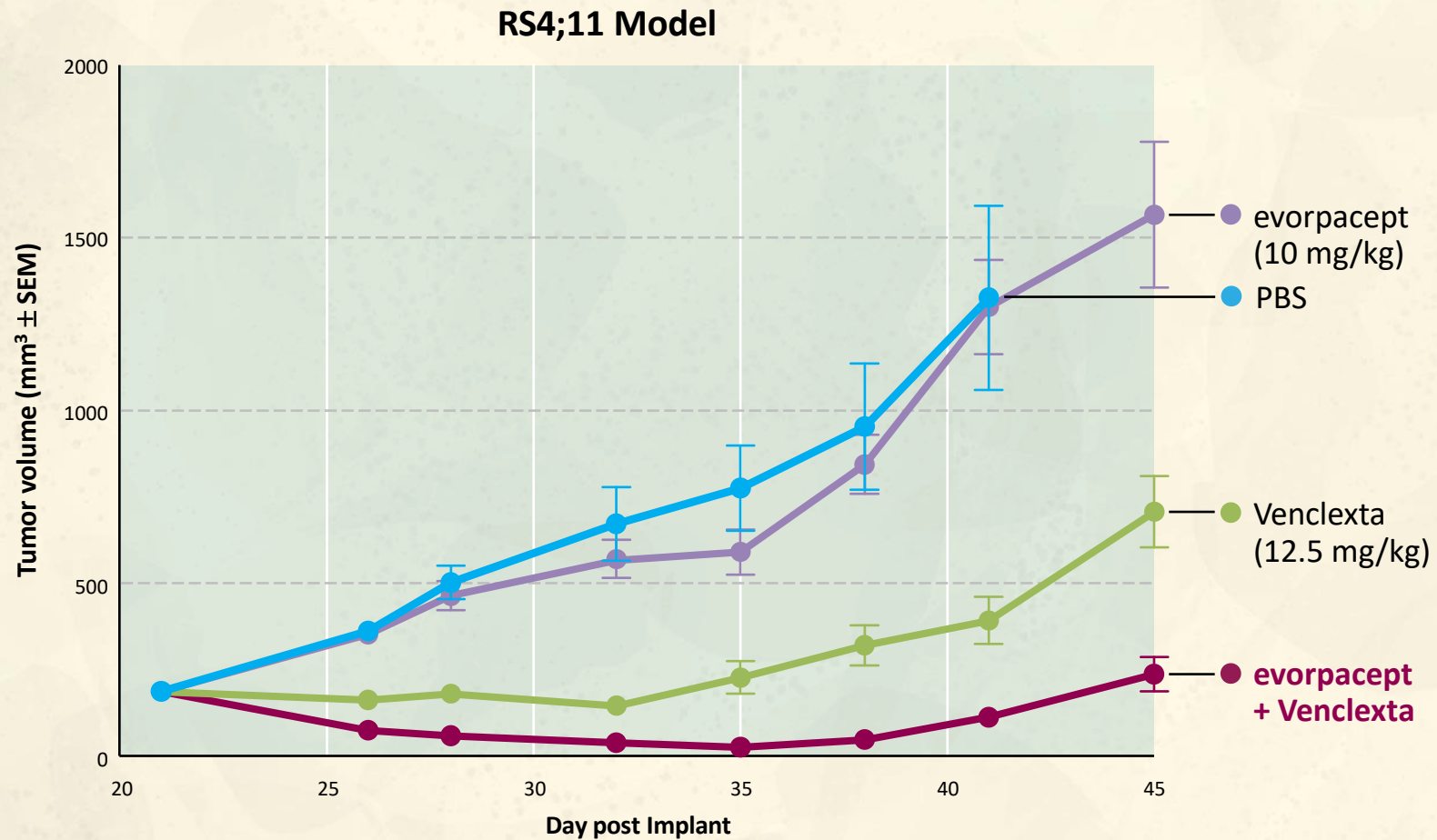
Disseminated AML mouse model

ASPEN-02 PHASE 1B MDS: EVORPACEPT + AZACYTIDINE PREVIOUSLY UNTREATED HR-MDS AND RELAPSED/REFRACTORY MDS ADVERSE EVENTS

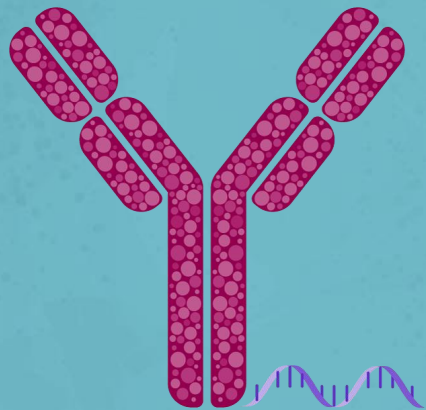
Adverse Event, n	20 mg/kg Q2W (N=3)		30 mg/kg Q2W (N=3)		60 mg/kg Q4W (N=16)		Total (N=22)
	Grade 1/2	Grade 3/4	Grade 1/2	Grade 3/4	Grade 1/2	Grade 3/4	All Grade n (%)
Blood Creatinine Increased	2	–	1	–	2	–	5 (23)
Constipation	1	–	1	–	2	1	5 (23)
Diarrhea	1	–	1	–	3	–	5 (23)
Fatigue	–	–	–	–	4	1	5 (23)
Neutropenia/Neutrophil Count Decreased	–	–	–	1	1	3	5 (23)
Anemia	1	1	1	–	–	1	4 (18)
Dizziness	–	–	1	–	3	–	4 (18)
Dyspnea	1	–	–	–	2	1	4 (18)
Febrile Neutropenia	–	2	–	–	–	2	4 (18)
Infusion Related Reaction	–	–	–	–	4	–	4 (18)
Nausea	–	–	1	–	3	–	4 (18)
Abdominal Pain	1	–	1	–	1	–	3 (14)
Contusion	1	–	1	–	1	–	3 (14)
Platelet Count Decreased	–	2	–	1	–	–	3 (14)
Pneumonia	–	1	–	–	–	2	3 (14)
Transfusion Reaction	2	–	–	–	1	–	3 (14)
Vomiting	1	–	–	–	2	–	3 (14)

EVORPACEPT INCREASES TUMOR INHIBITION OF VENCLEXTA

evorpacept
in
AML



Combination
opportunity
in AML



EARLY STAGE PIPELINE: SIRP α -TRAAC COLLABORATION

ALX ONCOLOGY AND TALLAC THERAPEUTICS 50/50 JOINT COLLABORATION ON NOVEL SIRP α ANTIBODY – TLR9 AGONIST CONJUGATE (SIRP α TRAAC)



Provides
SIRP α antibody

- CD47-SIRP α is a dominant myeloid checkpoint mechanism where SIRP α is expressed on myeloid and dendritic cells as well as on a range of tumor cells.
- SIRP α expression on tumor cells enables tumor microenvironment localization of SIRP α TRAAC.



Provides
TRAAC platform
and TLR9 agonist

- Toll-like receptor 9 (TLR9) is a key receptor in the innate immune response.
- Synthetic CpG oligonucleotides (CpG ODNs) are potent TLR9 agonists that stimulate antitumoral cytokine production and immune activation.
- Novel Toll-like receptor agonist antibody conjugation platform (TRAAC) enables systemic delivery of targeted TLR9 activation.

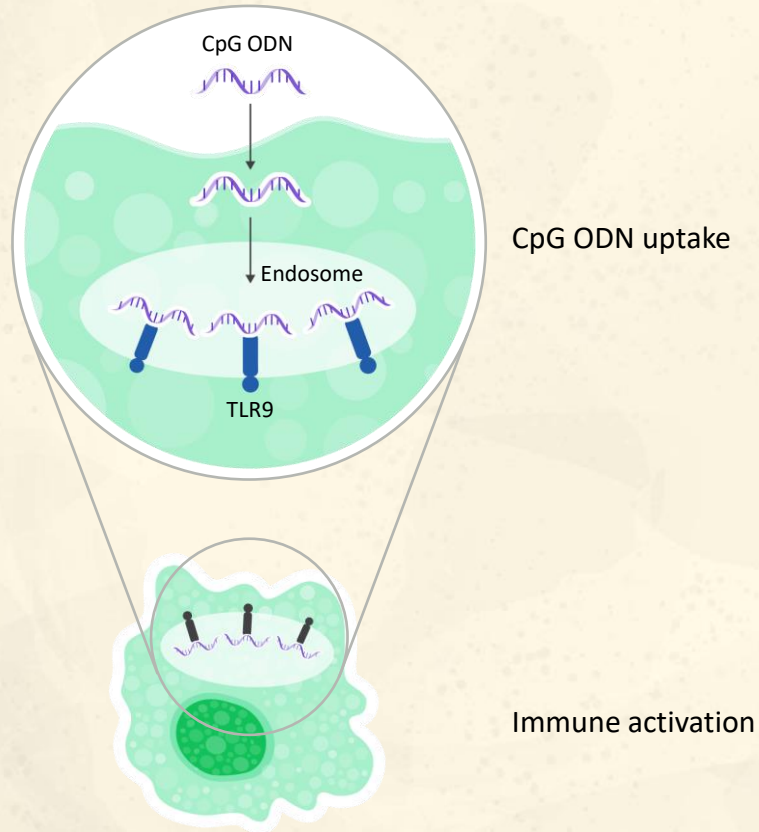
SIRP α TRAAC induces targeted immune activation, bridging innate and adaptive immune responses.

SIRP α TRAAC simultaneously overrides “don’t eat me” signals by blocking CD47-SIRP α myeloid checkpoint pathway and induces TLR9-based immune activation in antigen presenting cells (APCs).

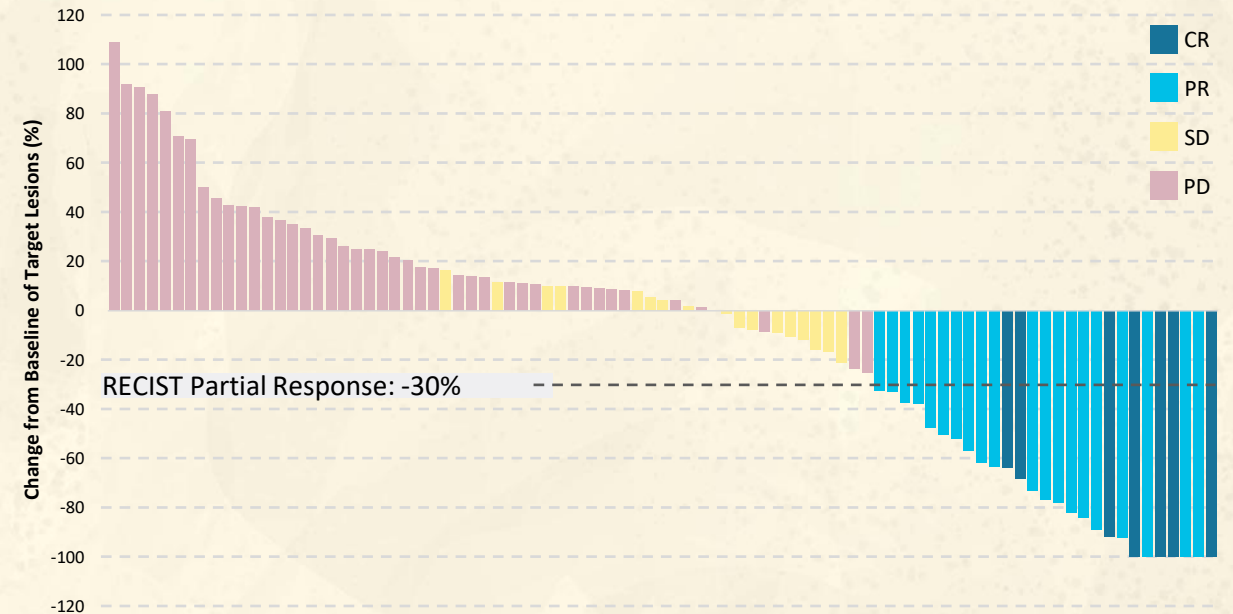
TOLL-LIKE RECEPTOR 9 (TLR9): A KEY INNATE PATHWAY

PROOF-OF-CONCEPT DATA IN MELANOMA PATIENTS WITH INTRATUMORAL TLR9 AGONISTS

TLR9 stimulation by CpG ODN leads to immune cell activation



Intratumoral programs have demonstrated clinical activity



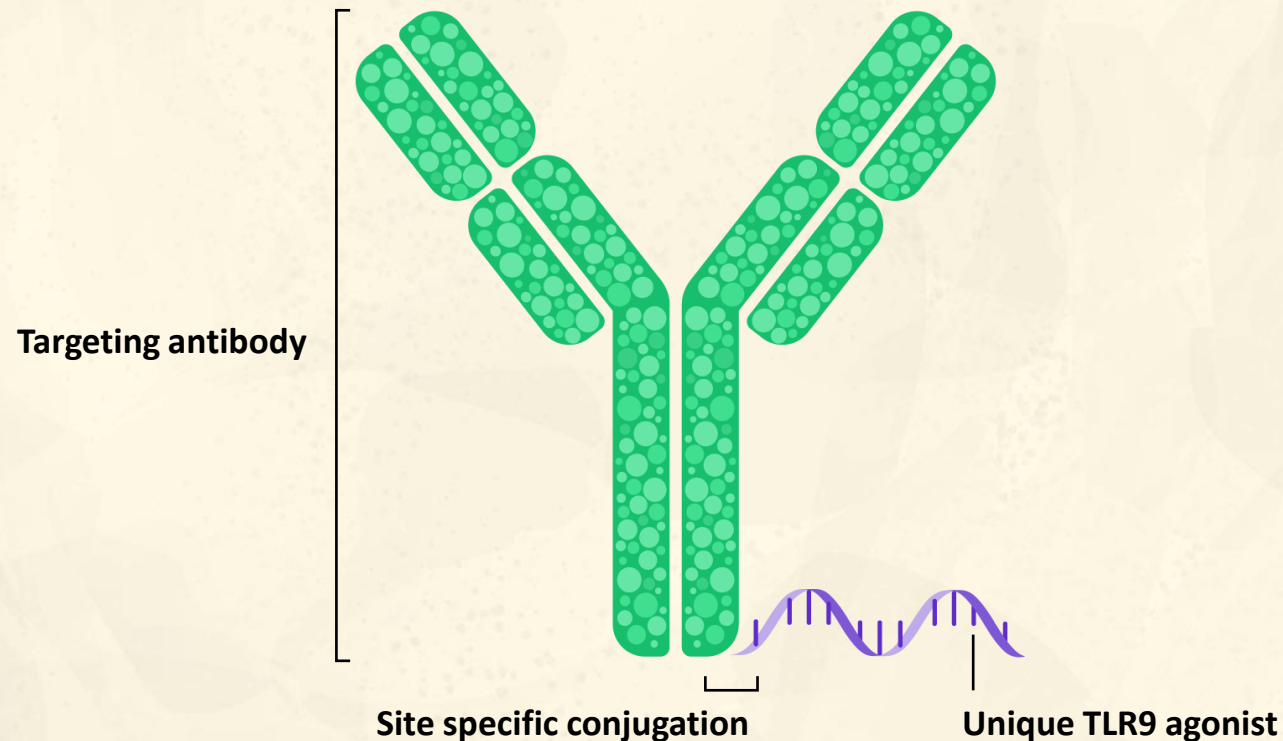
CMP-001 (Checkmate) + Pembrolizumab in Anti-PD-1 Refractory Melanoma, ¹⁻⁴Checkmate, S1 2020.

Additional clinical data from SD-101 (Dynavax), IMO-2125 (Idera) and AST-008 (Exicure) further validate TLR9 agonism in cancer.

TALLAC TRAAC PLATFORM: SYSTEMIC, TARGETED IMMUNE ACTIVATION

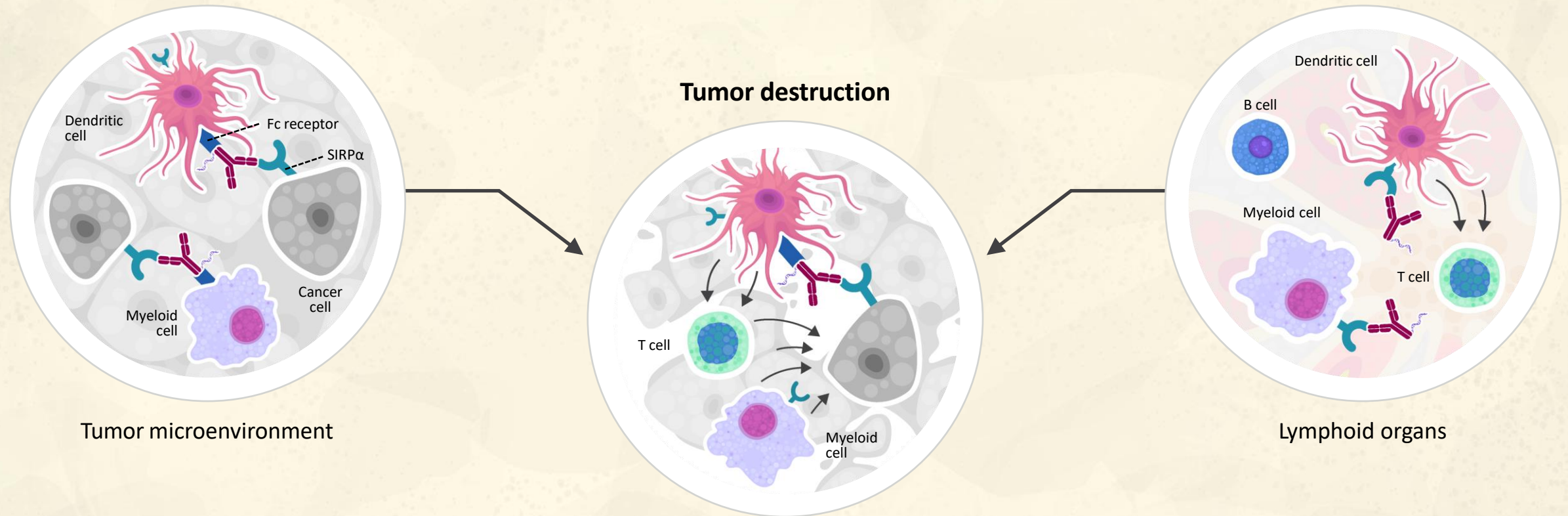
ANTIBODY DIRECTS TLR9 AGONIST (T-CPG) TO SPECIFIC IMMUNE CELLS

TLR9 Agonist Antibody Conjugate (TRAAC):
Systemic dosing with cell specific TLR9 activation



Targeted-CpG (T-CpG) designed specifically for compatibility with antibody conjugation, superior PK, receptor-mediated uptake and TLR9 stimulation

SIRP α IS EXPRESSED ON MYELOID AND DENDRITIC CELLS AS WELL AS SELECTED TUMOR TYPES

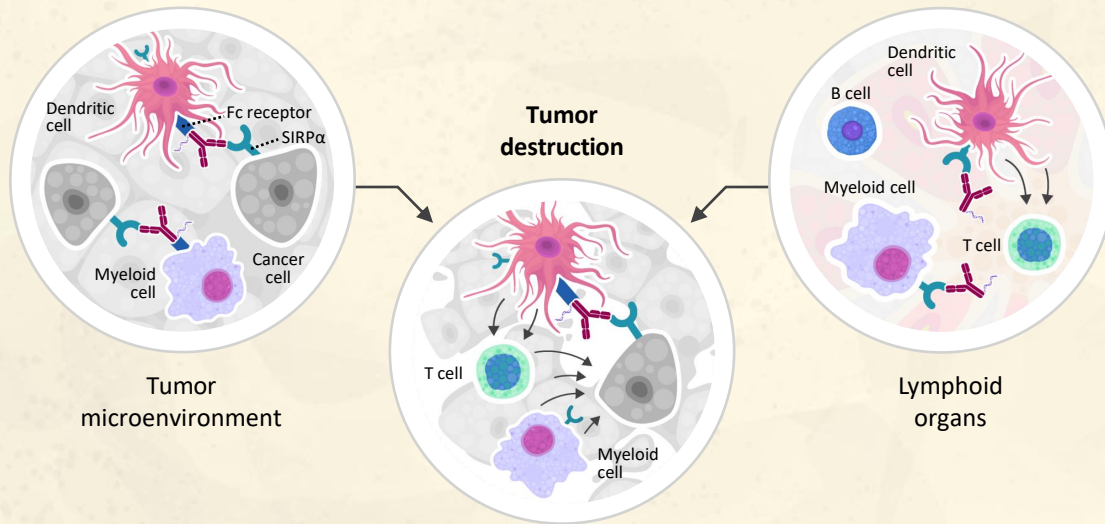
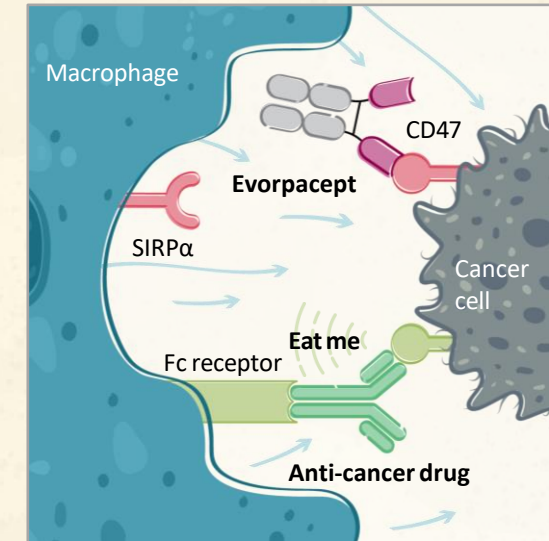


- SIRP α TRAAC binding to myeloid cells targets TLR9 activation in myeloid cells that matter (e.g. dendritic cells).
- SIRP α expression on tumor cells enables tumor microenvironment localization of SIRP α TRAAC.
- SIRP α TRAAC blocks CD47-SIRP α myeloid checkpoint pathway.

SIRP α TRAAC PROGRAM IS COMPLEMENTARY TO EVORPACEPT

Evorpaccept is an antagonistic molecule designed to maximize the activity of a wide array of anti-cancer agents by blockade of the CD47 myeloid checkpoint.

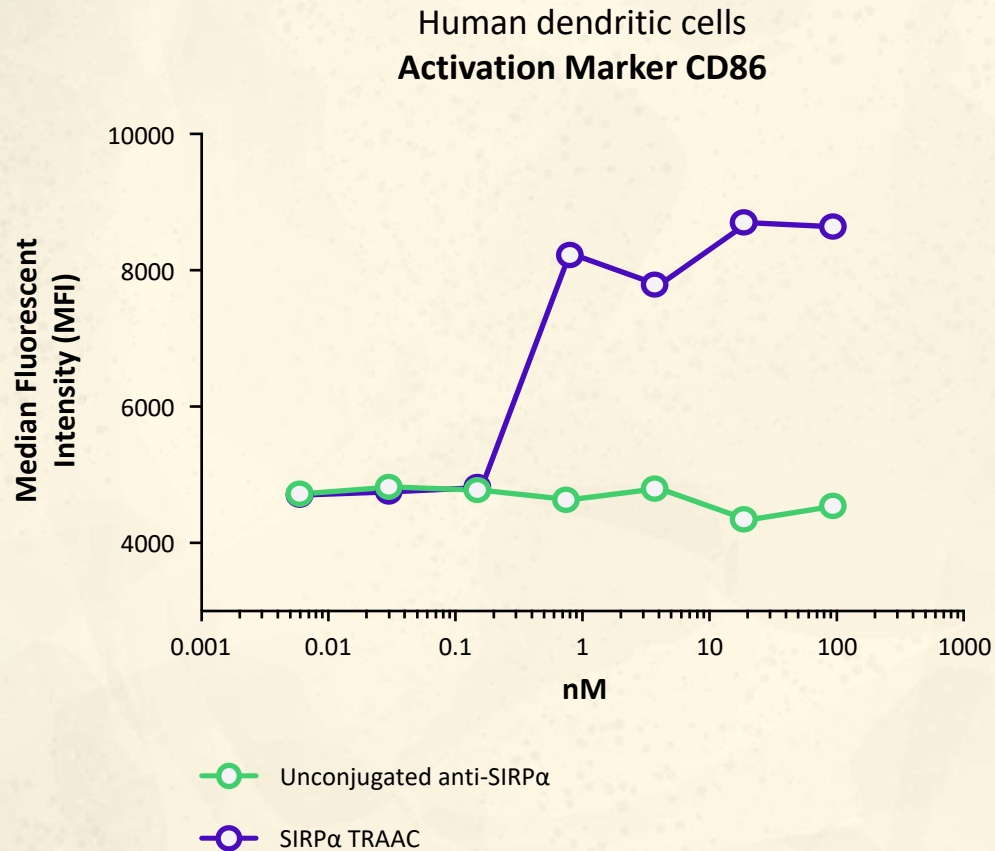
Removal of the CD47 inhibitory signal requires constant, full blockade of the pathway.



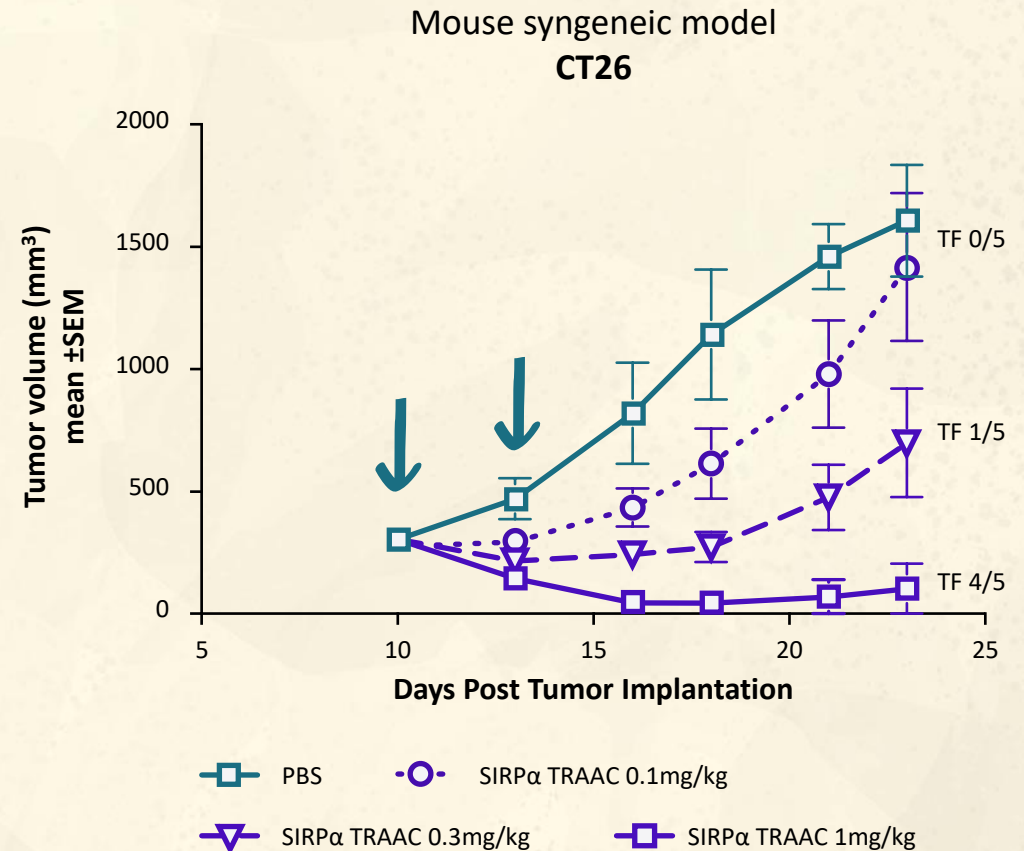
SIRP α TRAAC is an agonistic molecule that directly activates dendritic cells and initiates a coordinated innate and adaptive immune response against cancer.

In the case of agonistic molecules (TLR9 agonist), constant blockade is not required.

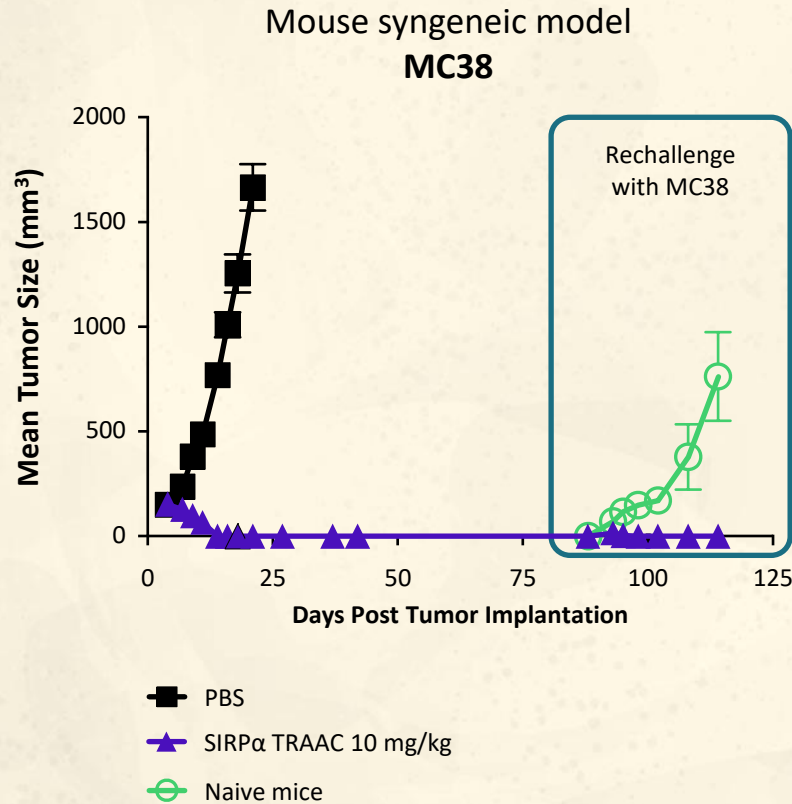
SIRP α TRAAC INDUCES POTENT AND SELECTIVE IMMUNE ACTIVATION AND LEADS TO POTENT SINGLE AGENT ACTIVITY IN TUMOR MODELS



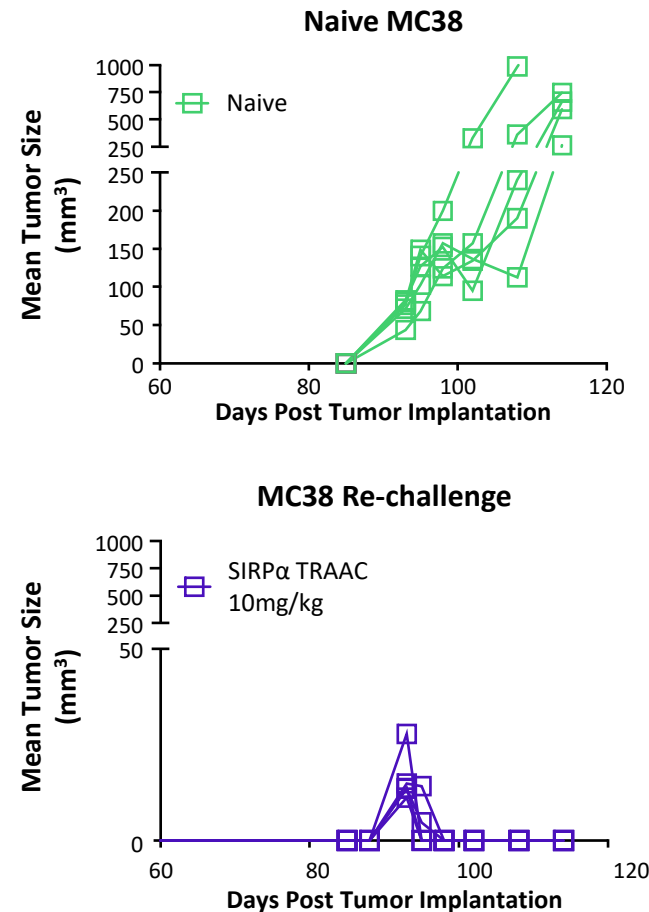
Harrabi et al., SITC, 2020



SYSTEMIC ADMINISTRATION OF SIRP α TRAAC GENERATES DURABLE ANTI-TUMOR RESPONSE AND IMMUNOLOGICAL MEMORY

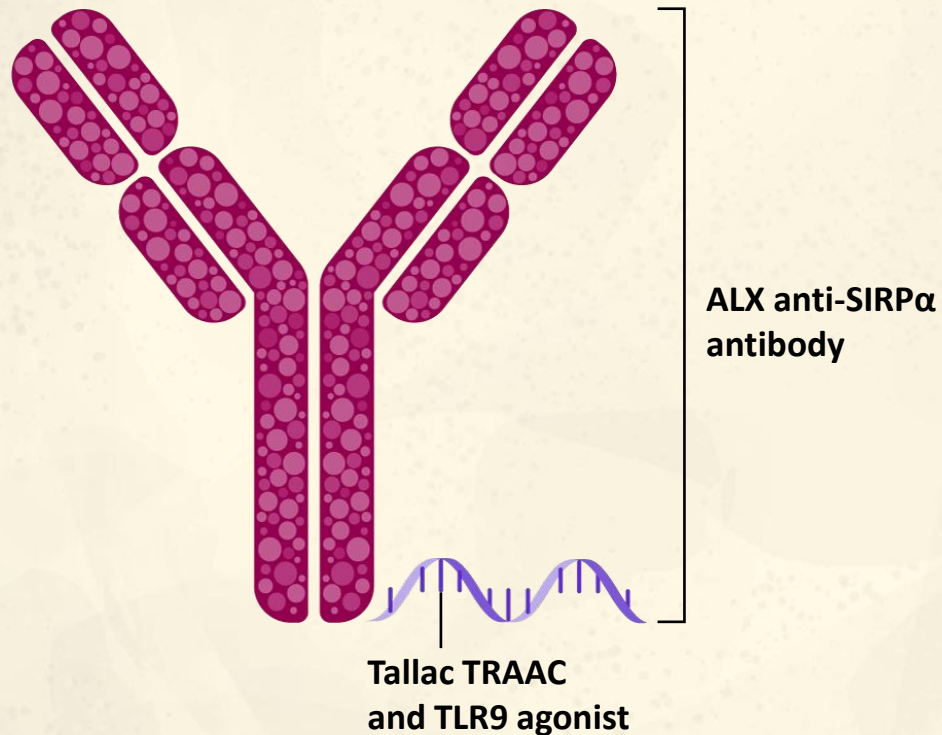


Harrabi et al., SITC, 2020



- Established MC38 tumors were eradicated following 10 mg/kg 2q3d systemic treatment with SIRP α TRAAC.
- These tumor free mice were then re-challenged 60-70 days post tumor clearance.
- SIRP α TRAAC treatment group demonstrated immune protection from the tumor re-challenge.
- Naïve age-matched mice were used as control for tumor growth.

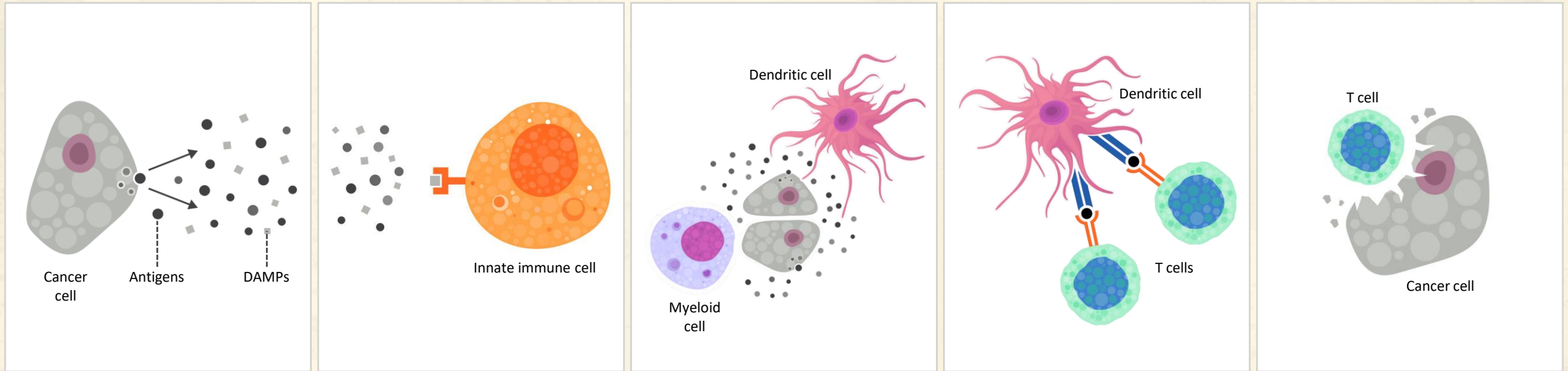
ALTA-002: TARGETING IMMUNE ACTIVATION TO WHERE IT MATTERS



- SIRPα TRAAC binding to myeloid cells targets TLR9 activation in key myeloid cells (e.g. dendritic cells).
- SIRPα expression on tumor cells enables localization of SIRPα TRAAC to tumor microenvironment.
- SIRPα TRAAC blocks CD47-SIRPα myeloid checkpoint pathway.
- Antibody-like PK profile allows for convenient dosing.
- Antibody conjugate produced through established manufacturing processes.

IND expected beginning of 2023

HARNESSING THE POWER OF INNATE AND ADAPTIVE IMMUNE RESPONSES TO CANCER



1. Release of PAMPs/DAMPs and tumor antigens

2. Detection by PRRs on innate immune cells

3. Amplification of innate immune cell activation

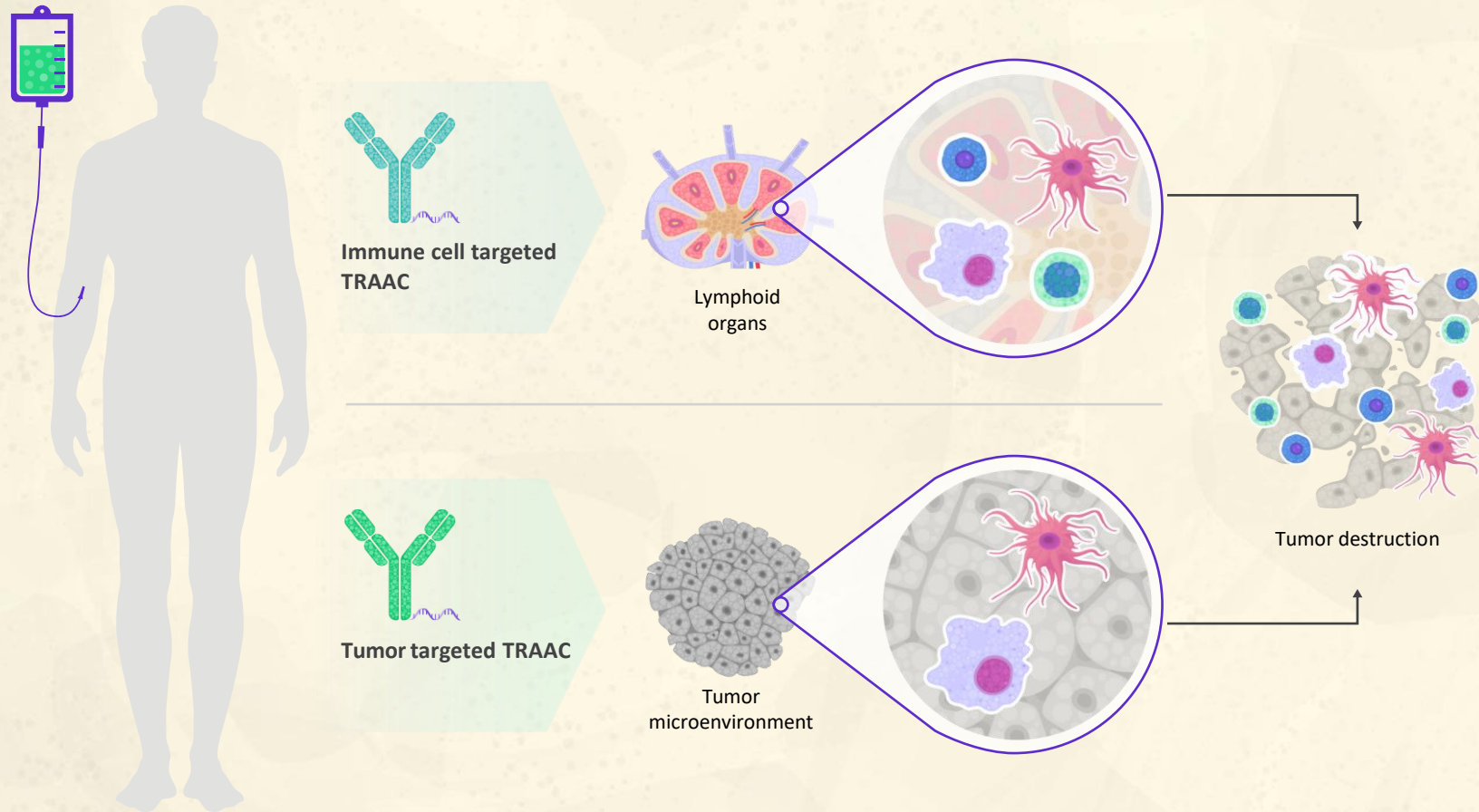
4. Antigen presentation and activation of T cells

5. Recognition and elimination of tumor by T cells

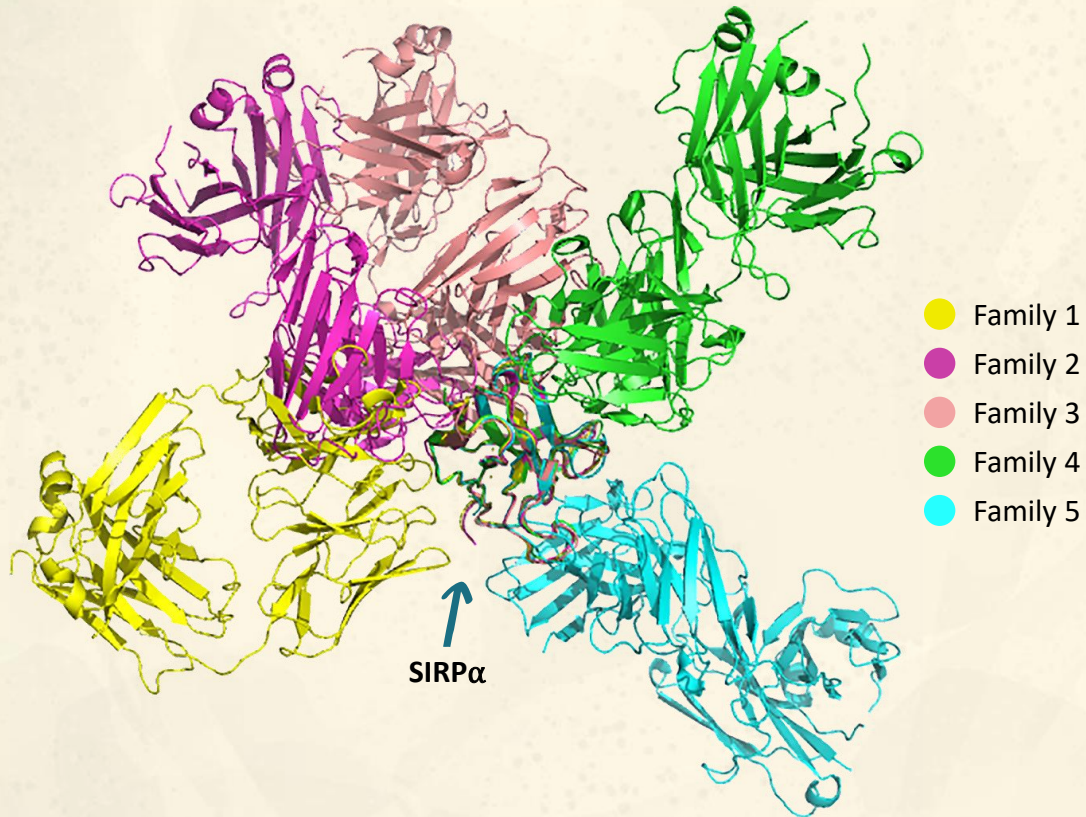
- Successful immune-mediated elimination of cancer requires coordination between the innate and adaptive arms of the immune system.
- Innate immune cells (macrophages, myeloid cells and dendritic cells) may utilize a variety of signaling pathways, including TLR9, TLR7 or 8, STING and CD40, to trigger proinflammatory programs and engage the adaptive immune system.

DAMPs: damage-associated molecular patterns
PAMPs: pathogen-associated molecular patterns
PRRs: pattern recognition receptors

TLR9 AGONIST ANTIBODY CONJUGATE (TRAAC) ENABLES VERSATILE TARGETING OF IMMUNE CELLS THAT MATTER



ALX ONCOLOGY'S SIRP α ANTIBODIES: HIGH AFFINITY AND DIVERSE EPITOPES



ALX's diverse range of SIRP α antibodies

Diversity allows selection of best-in-class SIRP α antibodies:

- Binds human SIRP α variants V1 and V2
- Cross reacts with rodent, monkey and human SIRP α
- Wide range of affinities
- Full coverage of SIRP α domain 1 surface allows selection for optimal epitope