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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 objects 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.

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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.



ADVANCING A HIGHLY DIFFERENTIATED IMMUNO-ONCOLOGY PIPELINE

ALX Oncology (Nasdaq: ALXO) is advancing a pipeline of candidates based on expertise in protein engineering and oncology led by the CD47 blocker, evorpacept, currently in phase 2 clinical trials



Evorpacept (myeloid checkpoint inhibitor) as a cornerstone therapy

Randomized phase 2 trials enrolling in 3 solid tumor indications: gastric/gastroesophageal cancer and 2 head and neck squamous cell carcinoma trials

Ongoing early clinical trials in 2 hematologic malignancies: myelodysplastic syndromes and acute myeloid leukemia

Continuing to broaden potential uses in new combinations and tumor types.



Building early stage pipeline

Ongoing IND-enabling development of ALTA-002 through 50/50 joint collaboration.

Early preclinical development of tumor-activated antibody platform.



Strong financial position

Cash and equivalents of \$363.7M as of December 31, 2021.

Expected cash runway to mid 2024.

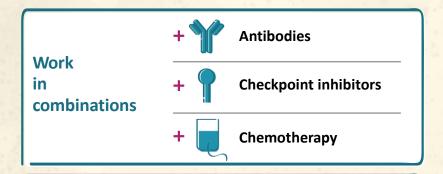
Collaboration partners

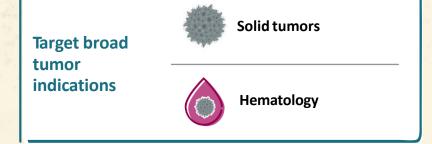
Merck, Eli Lilly, Zymeworks



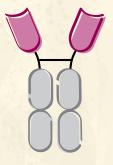
EVORPACEPT'S BROAD CLINICAL DATA SUPPORTS ITS DIFFERENTIATED POTENTIAL

Evorpacept was designed to:





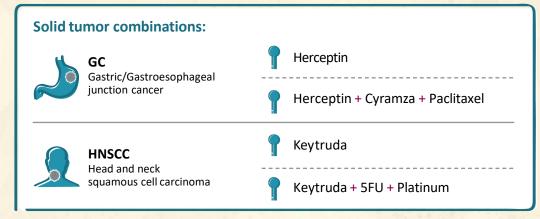


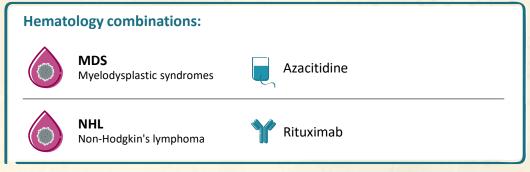


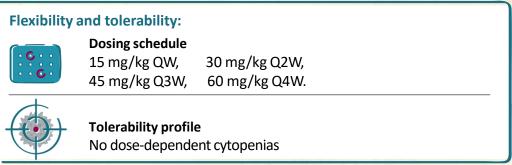
Evorpacept:

A phase 2 CD47 blocker designed to be a cornerstone of cancer treatments

Evorpacept's clinical data shows promising initial activity in:







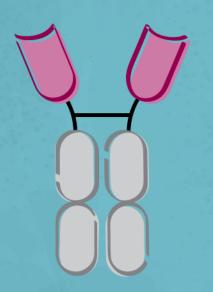


ALX PIPELINE

	Indi	cation	Combination Agent	Discovery	IND Enabling	Phase 1	Phase 2	Phase 3	Fast Track	Collaboration Partner
		HNSCC	Keytruda (ASPEN-03)							♦ MERCK
ies	ORS	Head And Neck Squamous Cell Carcinoma	Keytruda + 5FU + Platinum (ASPEN-04)							MERCK
n Studies	D TUMORS	GC Castria/Castraesanhagaal	Herceptin (ASPEN-01)							
binatio		Gastric/Gastroesophageal Junction Cancer	Herceptin + Cyramza + Paclitaxel (ASPEN-06)							Lilly
pt Com		Breast Cancer	Zanidatamab							zymeworks
Evorpacep	λĐO	MDS Myelodysplastic Syndromes	Azacitidine (ASPEN-02)							
Evo	MATOLOGY	AML Acute Myeloid Leukemia	Azacitidine + Venclexta (ASPEN-05)							
	HEM	NHL Non-Hodgkin's Lymphoma	Rituximab (ASPEN-01)							
ALTA- 002*		Advanced Cancer								TALLAC

^{*}SIRPa Toll-like receptor agonist antibody conjugate (TRAAC)



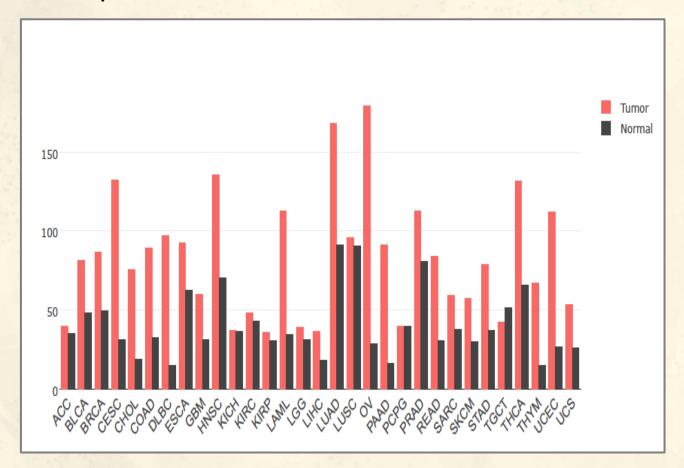


EVORPACEPT (ALX148)

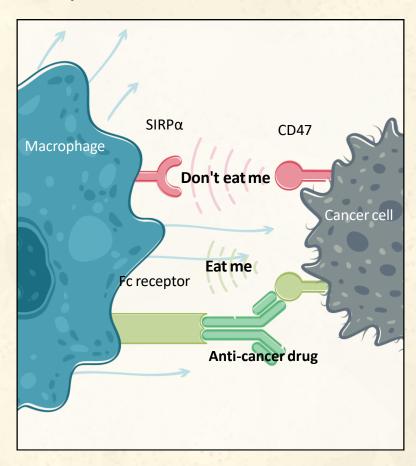


CD47: TUMOR ASSOCIATED ANTIGEN (TAA)- MYELOID CHECKPOINT DUALITY

TAA-Expression levels on cancer and normal cells



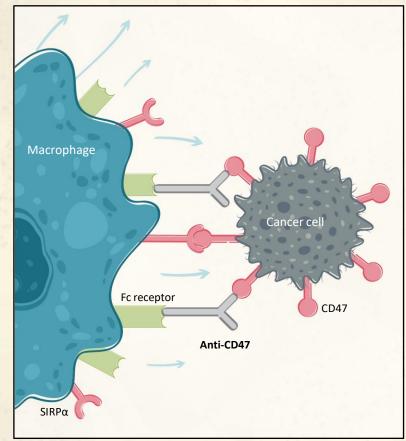
Checkpoint Mechanism: "do not eat me"

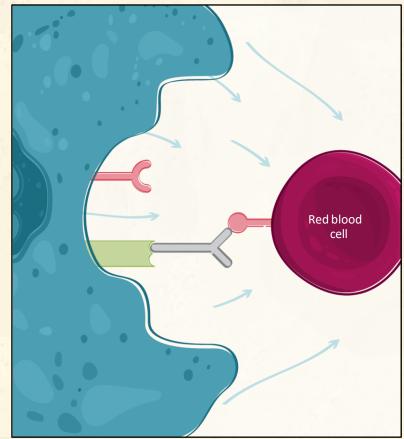


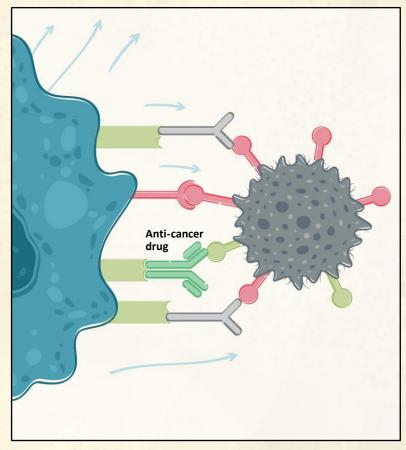


TARGETING CD47 AS TUMOR ASSOCIATED ANTIGEN

But also targets normal cells







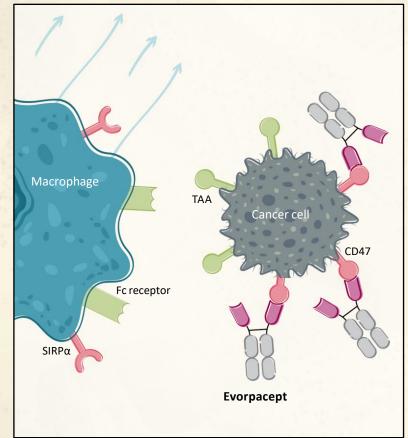
Anti CD47 with active Fc directly targets cancer cells

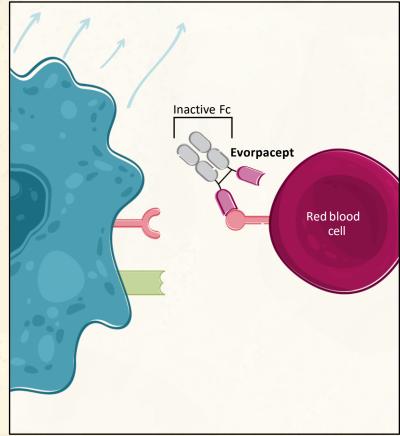


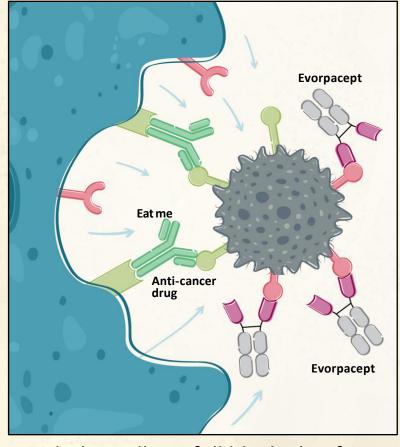
Dose limitations prevent full blockade of CD47 and active Fc competes with combo drug

TARGETING CD47 AS CHECKPOINT: ALX ONCOLOGY'S APPROACH

It spares normal cells







Anti CD47 with inactive Fc binds and block CD47-SIRP α interaction





EVORPACEPT: METICULOUSLY DESIGNED CD47 BLOCKER



Inactive Fc domain eliminates binding activity



No dose dependent cytopenia

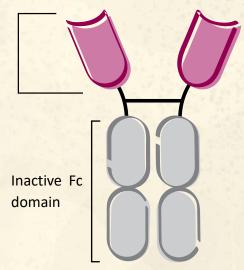
Presence of Fc domain ensures slow clearance and long half-life



Less frequent dosing and more flexibility

Designed for safety and efficacy

High affinity CD47 binding domains of SIRP α



- ~Half the molecular weight of an antibody
- Increases solid tumor penetration
- Cross-reactive to human, monkey, mouse
- Standard antibody manufacturing process



EVORPACEPT DEMONSTRATES CONSISTENT TOLERABILITY PROFILE

Treatment related adverse events	evorpacept + Herceptin + Cyramza + chemo (N=18)		evorpacept + Keytruda + chemo (N=13)		evorpacept + Keytruda (N=52)		evorpacept + 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%)	-	and the same	- 17	5 (9.6%)	-	-	-
AST increased	-	-	1 1 1 1 1	- /	9 (17.3%)	-	-	-
Platelets decreased	-	-		-	4 (7.7%)	2 (3.8%)	-	-
ALT increased	-	-	000000	- 1,000	7 (13.5%)	1 (1.9%)	-	-
Pruritus	2 (11.1%)	-		-14//1-15/8	5 (9.6%)	-	-	-
Pyrexia	-	-	The state of the s	-	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	-	-	- N - N	1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1	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	-	-	929 Fine - 1	-	2 (3.8%)	-	2 (9.1%)	-
Alkaline phosphatase incr	-	-	-72.K. 12		3 (5.8%)	-	-	_
Arthralgia	-	-			3 (5.8%)	-	-	_
WBC decreased	-	-	200	- 1	3 (5.8%)	-	-	_
Myalgia	-	-	244	-	2 (3.8%)	-	-	_
Diarrhea	3 (16.7%)	-	-	_	-	_	-	_
Urticaria	3 (16.7%)	-			-	-	-	-
Lymphocyte count decreased	1 (5.6%)	1 (5.6%)	17 . 25 - 1002	-	-	-	-	-
Headache	1 (5.6%)	-	P		-	-	-	-
Stomatitis	1 (5.6%)	-	-	- 1/3·	-	-	-	-
Back pain	1 (5.6%)	-	-	To - " "	-	-	-	-
Vision blurred	1 (5.6%)	-		- w	-	-	-	-
Abdominal pain / abdominal pain upper	1 (5.6%)	-		10 11 11 11 11 11 11	-	-	-	-
Hypersensitivity	-	-	1 (7.7%)	1 (7.7%)	-	-	-	-
Pneumonitis	-	-	1 (7.7%)	4 4 4 4 5	-	-	-	-
Constipation	-	-	-	-	-	-	3 (13.6%)	-
Vomiting	-	-			-	-	2 (9.1%)	-



EVORPACEPT'S INITIAL CLINICAL ACTIVITY IS MAGNIFIED IN SURVIVAL-BASED ENDPOINTS ACROSS SOLID TUMOR TYPES IN MULTIPLE TRIALS

Population	≥2L HE	R2+ GC	1L HI	NSCC		INSCC Naïve)
Combination (N-evaluable)	evorpacept + Herceptin + Cyramza + paclitaxel (N=18)		evorpacept + 5FU + ; (N=	-	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 + 5F	U + platinum	single age	nt Keytruda



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

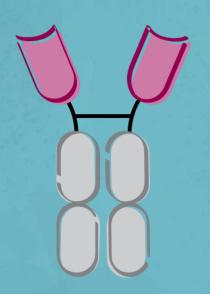
ASPEN-02

Population	Previously unt myelodysplasti with TP!	Relapsed / refractory MDS	
Combination	Evorpacept + azacitidine	Magrolimab + azacitidine ¹	Evorpacept + azacitidine
N-evaluable	5	4	9
CR	2	2	-
mCR	1 with HI	1	5*
SD	1		2

ASPEN-01

Population	≥2L aggressive non-	≥2L aggressive non-Hodgkin's lymphoma				
Combination	Evorpacept + Rituximab ²	Magrolimab + Rituximab³				
N-evaluable	21	38				
ORR	8	11				
(%)	(38%)	(29%)				
CR	1	2				
(%)	(5%)	(5%)				
PR	7	9				
(%)	(33%)	(24%)				



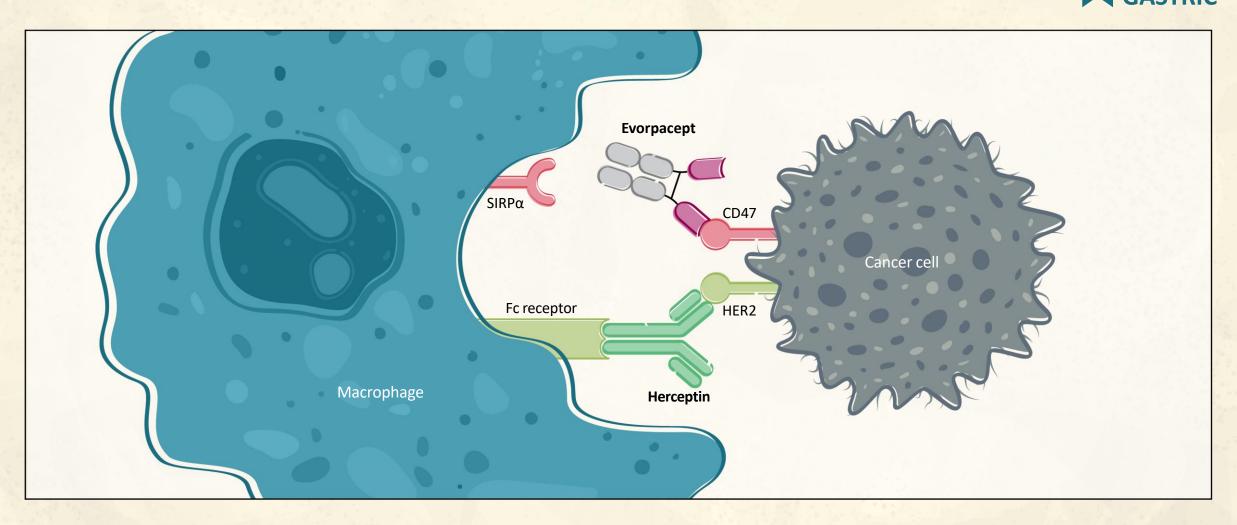


ASPEN-06: EVORPACEPT (ALX148) IN HER2+ GASTRIC/GEJ CANCER



GC TRIAL: EVORPACEPT + HERCEPTIN MECHANISM OF ACTION





Evorpacept 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 RAINBOW¹	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%



PHASE 1B GC TRIALS: PATIENT BASELINE CHARACTERISTICS

		evorpacept + Herceptin ≥2L GC (N=20)	evorpacept + Herceptin + Cyramza/chemo ≥2L GC (N=18)	
Median age, years (range)		58 (45-79)	67.5 (36-83)	
6000	М	15	13	
Sex, n	F	5	5	
	Asian	13	15	
Race, n	White	6	3	
	Other	1		
	0	7	8	
ECOG PS, n	1	13	10	
Progressed upon prior anti-HER2 therapy, n (%	6)	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 (%)	metastasis, n (%) 17 (85)		15 (83)	



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.

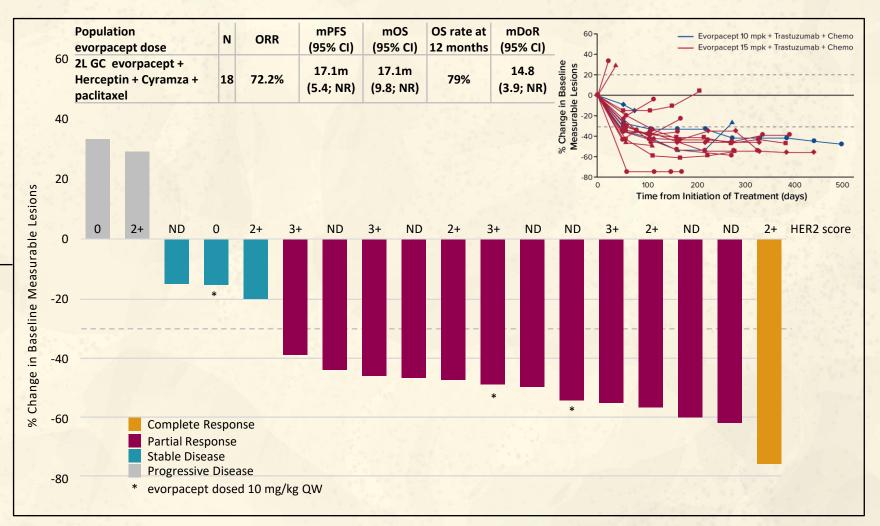


evorpacept 10 and 15 mg/kg (QW)

- + Herceptin
- + Cyramza
- + paclitaxel



- safety of combination
- anti-cancer activity





Data Cutoff September 1, 2021. ND = Not Done. NR = Not Reached.

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



Randomized Phase 2: First patient enrolled March 2022



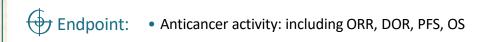
2L or greater HER2 positive GC with prior HER2 targeted therapy



evorpacept 30 mg/kg (Q2W)

- + Herceptin
- + Cyramza
- + paclitaxel

- + Herceptin
 - + Cyramza
 - + paclitaxel



vs.

Randomized Planned Phase 3:



Patients:

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



evorpacept 30 mg/kg (Q2W)

- + Herceptin
- + Cyramza
- + paclitaxel

- + Cyramza
 - + paclitaxel

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



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

Phase 1b GC trial:



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



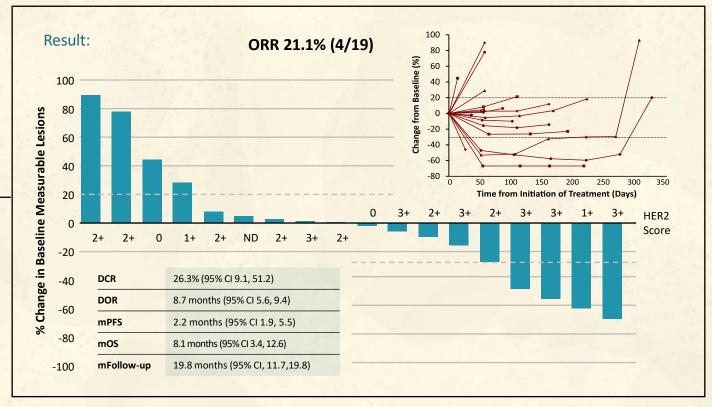
evorpacept 10 mg/kg once a week (QW)

+ Herceptin

8 mg/kg once, then 6 mg/kg every three weeks (Q3W)



- maximum tolerated dose
- anti-cancer activity

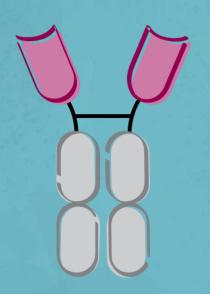


Notes: Data Cutoff October 1, 2020. Patients who received at least one dose of evorpacept in the expansion phase, had a baseline assessment, and at least one post-baseline disease assessment; One patient with clinical progression not included in plots.

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



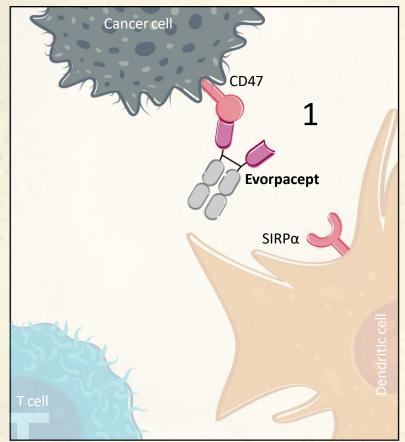


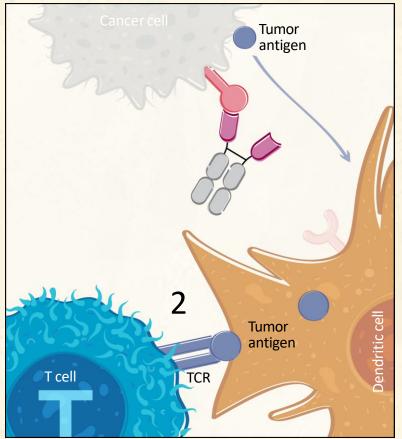
ASPEN-03 AND ASPEN-04: EVORPACEPT (ALX148) IN 1L HNSCC

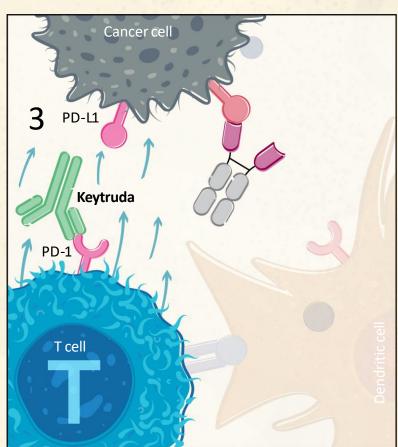


HNSCC TRIAL: EVORPACEPT + KEYTRUDA MECHANISM OF ACTION











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

OS RATE AT 12 MONTHS PREDICTIVE OF OVERALL SURVIVAL



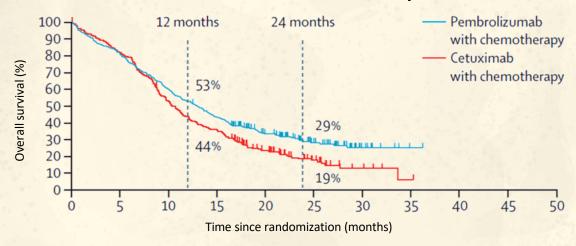
Population	N	ORR (%)	PFS (m) [95% CI]	OS Rate at 12 m	OS (m) [95% CI]	Follow Up (m) [95% CI]
KEYNOTE-048: 1L HNSCC pembrolizumab + 5FU/platinum 1L	281	36%	4.9 [4.7–6.0]	53%	13.0 [10.9–14.7]	13 [6.4–26.6]
KEYNOTE-048: 1L HNSCC cetuximab + 5FU/platinum	300	36%	5.1 [4.9–6.0]	44%	10.7 [9.3–11.7]	10.7 [6.6–19.7]
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]

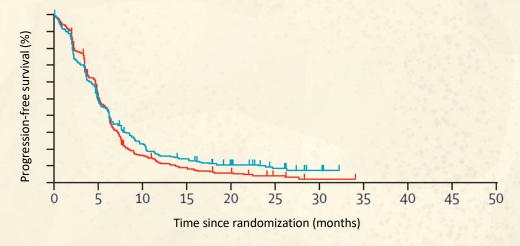


IMMUNO-ONCOLOGY AGENTS IN CPI NAÏVE HNSCC POPULATIONS: PFS AND OS AS ENDPOINTS IN KEYNOTE-040 AND 048

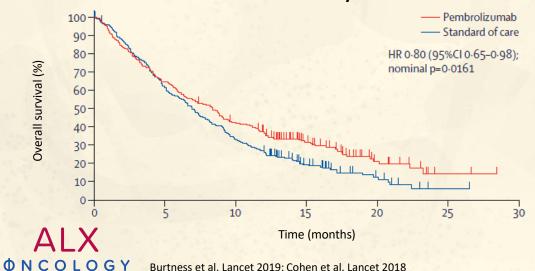


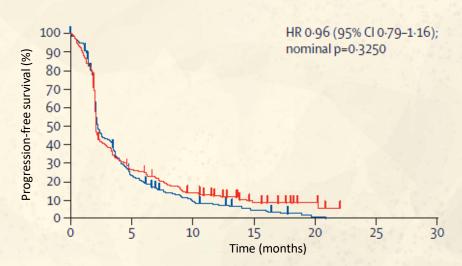
KEYNOTE-048: OS and PFS at the Second Interim Analysis in the 1L HNSCC CPI Naïve Population





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





HNSCC TRIALS: PATIENT BASELINE CHARACTERISTICS

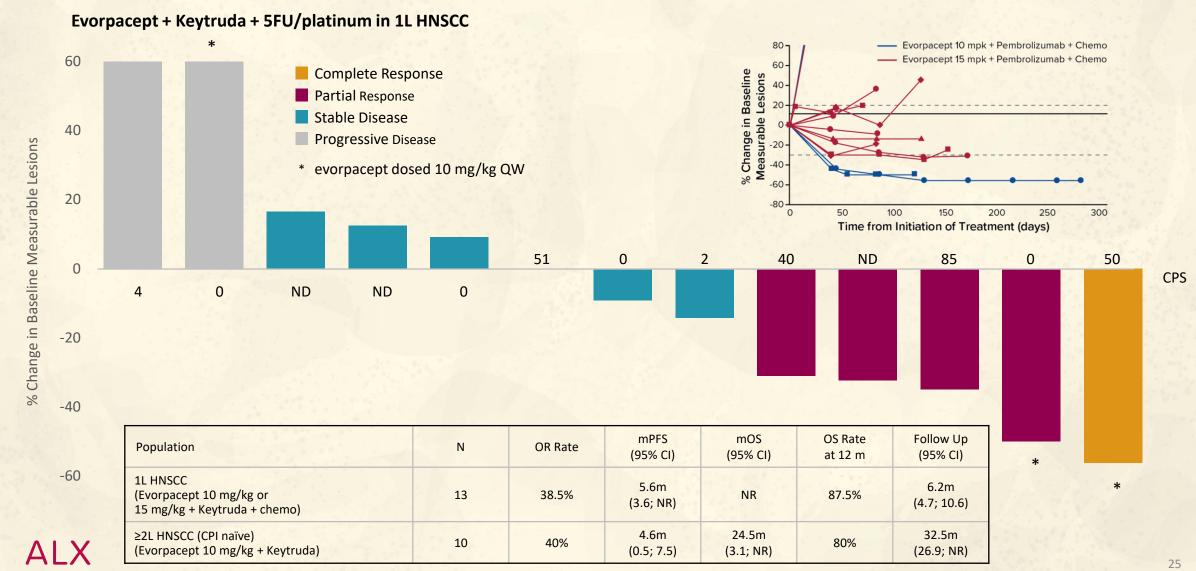


		evorpacept + Keytruda ≥2L HSCC (N=10)	evorpacept + Keytruda + 5FU/platinum 1L HNSCC (N=13)
Median age, years (range)		63 (35-81)	61 (45-70)
6	М	7	12
Sex, n	F	3	1
	Asian	5	10
Race, n	White	4	3
	Black	1	
5000 PC	0	3	8
ECOG PS, n	1	7	5
Progressed upon prior CPI therapy, n (%)		0 (0)	0 (0)
Visceral distant metastasis, n (%)		6 (60)	7 (54)

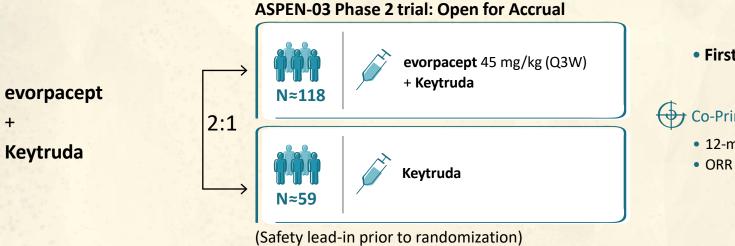


PHASE 1B HNSCC: EVORPACEPT + KEYTRUDA + 5FU/PLATINUM FIRST LINE CHECKPOINT NAIVE

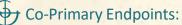




FIRST LINE HEAD & NECK CANCER: PHASE 2 DEVELOPMENT PLAN, ASPEN-03 AND ASPEN-04



First patient enrolled May 2021

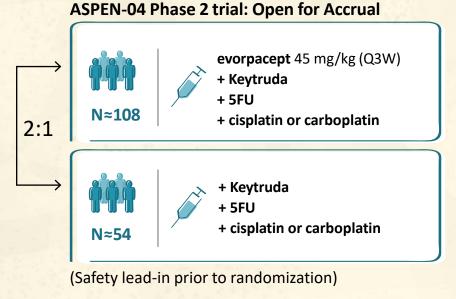


- 12-month OS rate

evorpacept

Keytruda

chemo

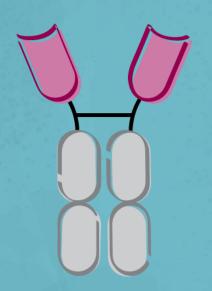


First patient enrolled July 2021

Co-Primary Endpoints:

- 12-month OS rate
- ORR



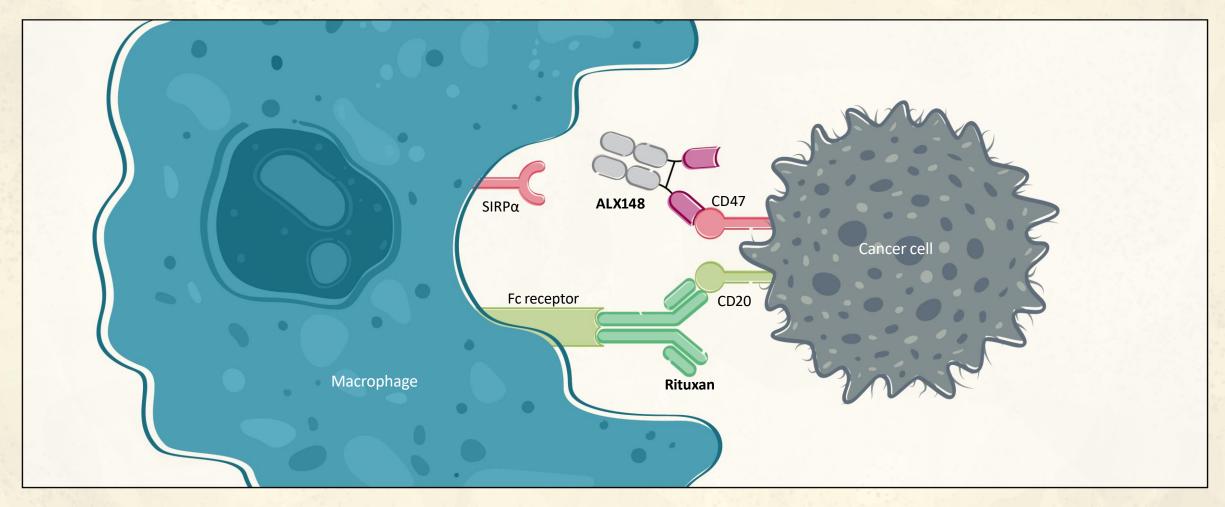


EVORPACEPT (ALX148) IN HEMATOLOGIC MALIGNANCIES



NHL TRIAL: ALX148 + RITUXAN MECHANISM OF ACTION





ALX148 increases antibody dependent cellular phagocytosis in combination with Rituxan



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

		10 mg/kg QW) + uximab	V) + Evorpacept (15 mg/kg (Rituximab		
Population	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

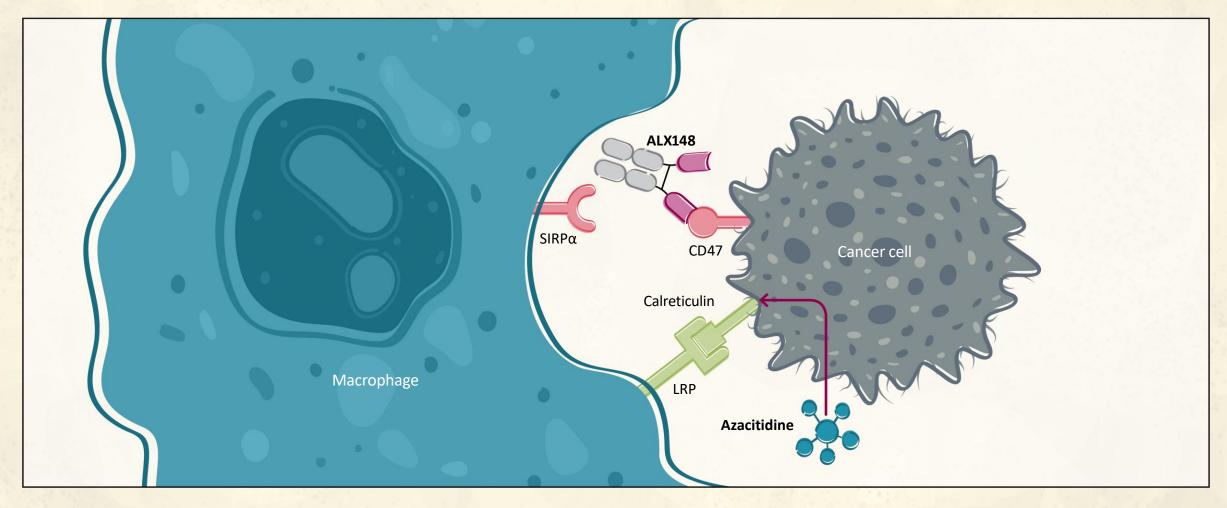


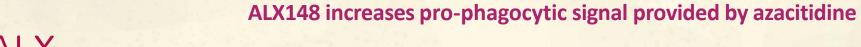
Aggressive = Diffuse Large B-cell Lymphoma and Mantle Cell Lymphoma.

ORR = Objective Response Rate.

MDS TRIAL: ALX148 + AZACITIDINE MECHANISM OF ACTION











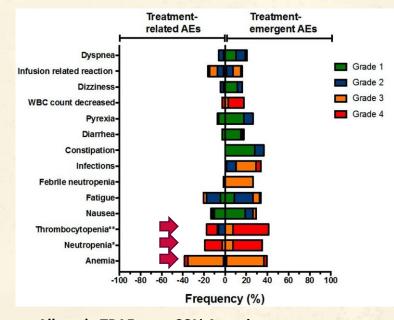
CD47 BLOCKADE IS CLINICALLY VALIDATED WITH SIGNIFICANT OPPORTUNITY TO IMPROVE ON MAGROLIMAB

Best Overall Response	1L MDS N=33
ORR	30 (91%)
CR	14 (42%)
CRi	NA
PR	1 (3%)
MLFS/marrow CR	8 (24%) 4 with marrow CR + HI
Hematologic improvement (HI)	7 (21%)
SD	3 (9%)
PD	0

Magrolimab with azacitidine

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%)

Magrolimab monotherapy



All grade TRAEs: 38% Anemia

19% Neutropenia

18% Thrombocytopenia

Sallman, ASCO 2020

CD47 blocker 5F9 (magrolimab) shows complete responses only when paired with azacitidine,

Sallman, ASCO 2019

and causes frequent incidence of treatment-related, high-grade cytopenia

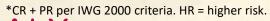


Sallman, ASCO 2020

CRi = complete remission with incomplete hematological recovery

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 MDS³ Venetoclax + azacitidine	38	40%	8%	





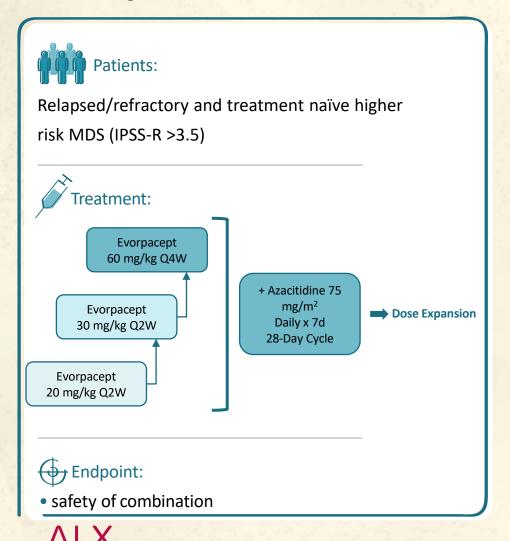
evorpacept in

MDS

MDS TRIAL: DESIGN AND PATIENT BASELINE CHARACTERISTICS

Phase 1 Design

ONCOLOGY



Patient Baseline Characteristics

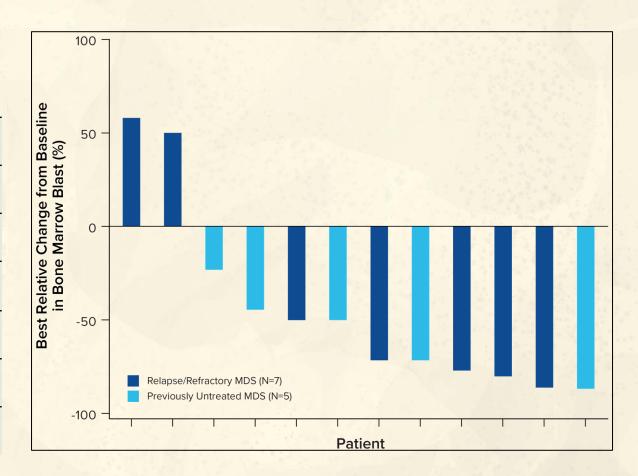
		evorpacept + azacitidine (N=22)
Median age, years (range)		70.5 (56 – 81)
Sex, n	F	8
	M	14
Race, n	White	17
	Black	4
	Unknown	1
ECOG PS, n	0	6
	1	16
	2	0
MDS Status, n	Previously untreated HR-MDS	9
	Therapy related	6
	Relapsed/Refractory MDS	13
	 Prior HMA treatment 	13
IPSS-R Score	Mean	6.0
	Median	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	Very Good	0
Diagnosis, n (%)	Good	2 (9%)
	Intermediate	0
	Poor	2 (9%)
	Very Poor	8 (36%)
	Not Available	10 (45%)

evorpacept in MDS

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 *			
Н	0	0	0			
SD	2	1	2			
PD	1	1	1			





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)



evorpacept

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

azacitidine



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)



evorpacept

recommended phase 2 dose

azacitidine

VS.

azacitidine



• complete response rate (CRR)



AML TRIAL PLANS, ASPEN-05



Phase 1 Dose Escalation and Expansion: Open for Accrual



Patients:

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



evorpacept

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

- + Venclexta
- + azacitidine



 safety of combination, recommended phase 2 dose

Phase 2:



Patients:

Previously untreated AML who are not

considered suitable for intensive

induction therapy



evorpacept

recommended phase 2 dose

- + Venclexta
- + azacitidine



• complete remission rate

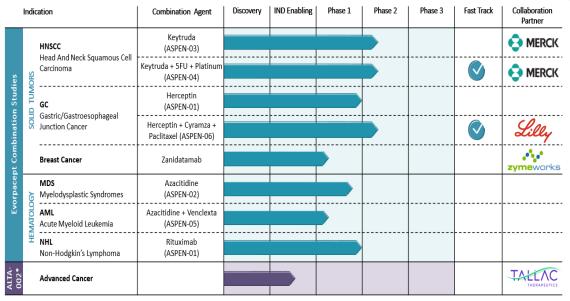


ALX ONCOLOGY MILESTONES AND FINANCIAL INFORMATION



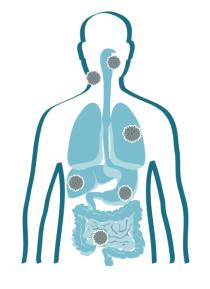
EVORPACEPT IS DESIGNED TO BE A CORNERSTONE OF CANCER TREATMENTS

Evorpacept's ongoing clinical development plan encompasses significant development opportunities...



*SIRPa Toll-like receptor agonist antibody conjugate (TRAAC)

And is designed to be active across more tumor types and anticancer combinations



Continued expansion of immuno-oncology activity across tumor types



Combined with standard of care and emerging anti-cancer modalities



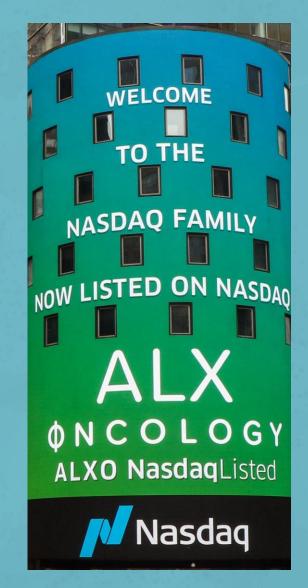
2022 FOCUSED ON DRIVING CLINICAL DEVELOPMENT

	Completed	2022	2023	2024
	ASPEN-01 (Phase 1b) Updated gastric/GEJ and HNSCC trial data at SITC	ASPEN-06 Initiation (Phase 2/3) Randomized gastric/GEJ cancer trial	ASPEN-06 (Phase 2) Randomized gastric/GEJ cancer trial readout	ASPEN-03 (Phase 2) Randomized HNSCC trial readout with pembrolizumab
	ASPEN-02 (Phase 1a) Initial MDS trial readout at ASH	ASPEN-02 (Phase 1b) MDS dose optimization trial readout	ASPEN-05 (Phase 1a) AML trial readout	ASPEN-04 (Phase 2) Randomized HNSCC trial readouts with pembrolizumab and chemo
90	ASPEN-03 Initiation (Phase 2) Randomized HNSCC trial with pembrolizumab	Ongoing collaborations (Zymeworks) and Investigator Sponsored Trials (NHL)		
Evorpacept	ASPEN-04 Initiation (Phase 2) Randomized HNSCC trial with pembrolizumab and chemo			
	ASPEN-05 Initiation (Phase 1a) AML trial			
Preclinical pipeline	Built pipeline through ScalmiBio acquisition and Tallac collaboration	Select clinical development candidates from preclinical pipeline	File IND for ALTA-002	



FINANCIAL INFORMATION

- Consummated IPO on July 21, 2020
- Closed follow-on offering on December 14, 2020
 - Gross proceeds of \$208.0 million
 - 2.737 million shares at \$76 per share
- Cash and cash equivalents as of December 31, 2021:
 - \$363.7 million
- Expected cash runway through mid-2024

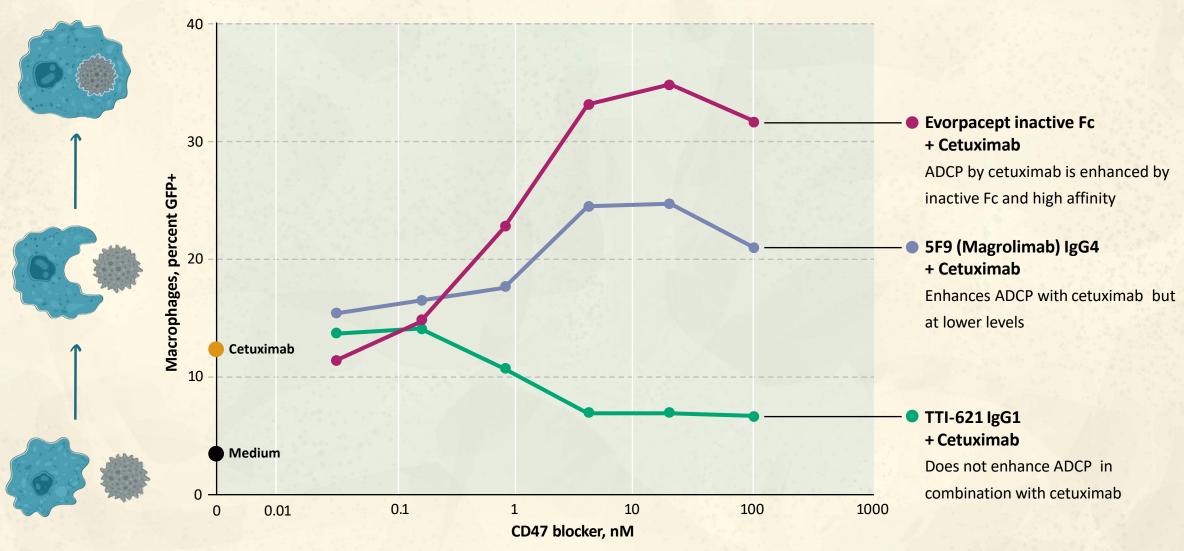




APPENDIX

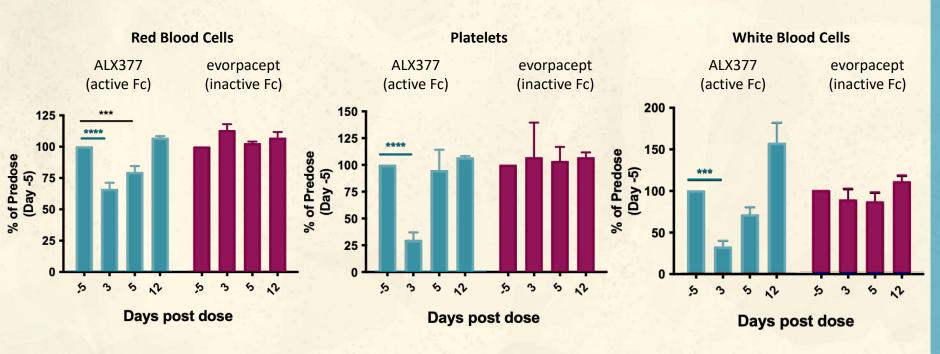


EVORPACEPT DEMONSTRATES SUPERIOR PHAGOCYTOSIS





INACTIVE FC SHOWS REDUCED CYTOPENIA IN MICE IN VITRO



Inactive Fc is the core determinant of safety profile

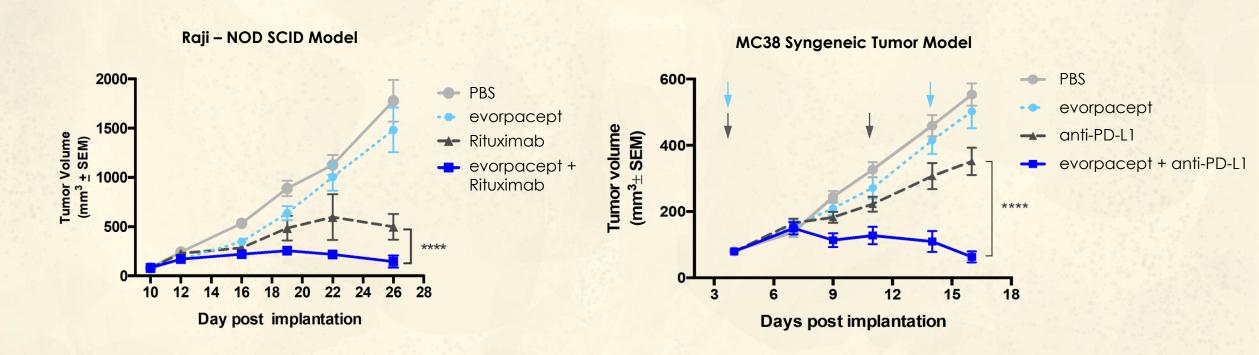
CD-1 mice received 30 mg/kg IV single dose

****p<0.0001, ***p<0.001

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



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

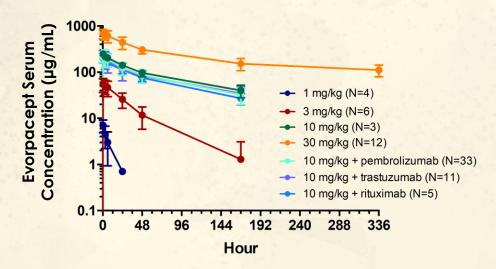


Mouse cross-reactivity allows for better clinical translation of preclinical models: presence of CD47 sink and mouse models with intact immune system



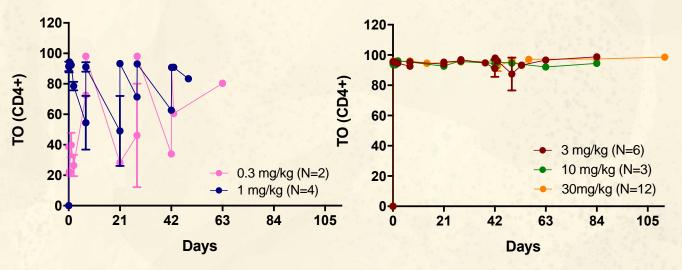
EVORPACEPT CLINICAL PHARMACOKINETICS AND CD47 TARGET OCCUPANCY

Evorpacept Serum Levels for Cycle 1 Day 1



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

CD47 Target Occupancy by Evorpacept



- Near complete CD47 target occupancy (TO) by evorpacept 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

evorpacept
in
NHL

Selected hematologic, treatment related	evorpacept + Rituximab (N=33) ¹			- Rituximab 26) ²	5F9 (magrolimab) + Rituximab (n=115) ³	
adverse events	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

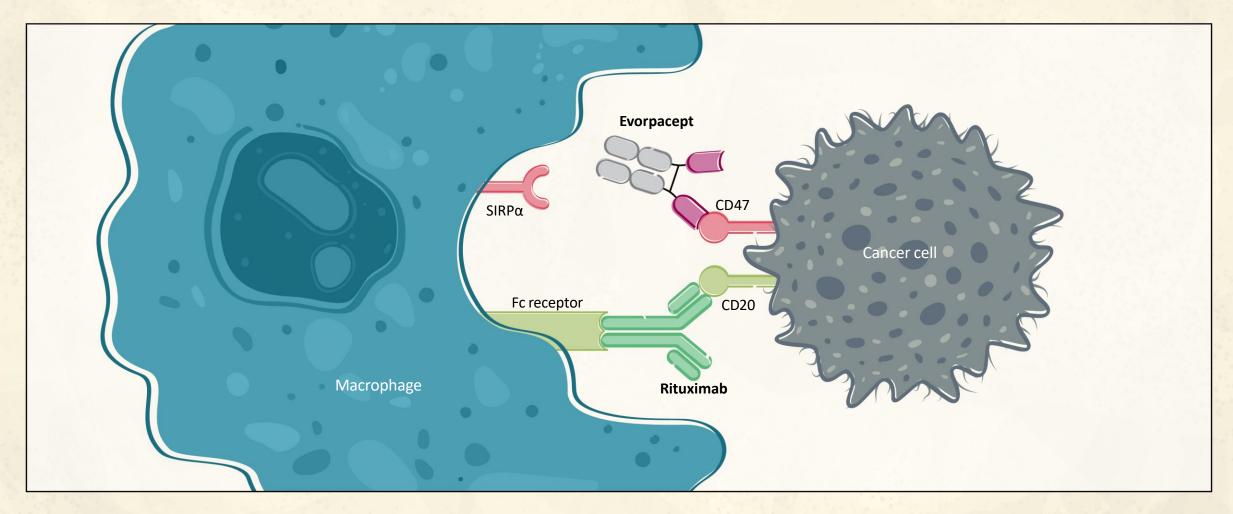
³EHA 2019 Abstract S867

Evorpacept:
Tolerability profile
compares favorably to
other CD47 blockers



NHL TRIAL: EVORPACEPT + RITUXIMAB MECHANISM OF ACTION





Evorpacept increases antibody dependent cellular phagocytosis in combination with Rituximab



NHL PROOF-OF-PRINCIPLE TRIAL



Phase 1b NHL cohorts



relapsed/Refractory NHL, prior regimen with Rituximab



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)		
	Follicular	5	3		
D.:	Marginal Zone (MZL)	2	1		
Primary Disease, r	Mantle Cell (MCL)	4	1		
	DLBCL	11	6		
Median Age, Years	s (range)	66 (32-80)	64 (53-78)		
-	M	17	6		
Sex, n	F	5	5		
	Asian	18	9		
Race, n	White	4	2		
	0	7	2		
ECOG, PS, n	1	15	9		
Median Prior The	rapy, n (range)	3 (1-7)	3 (1 -5)		

Data Cutoff October 1, 2020





evorpacept + Rituximab (N=33)

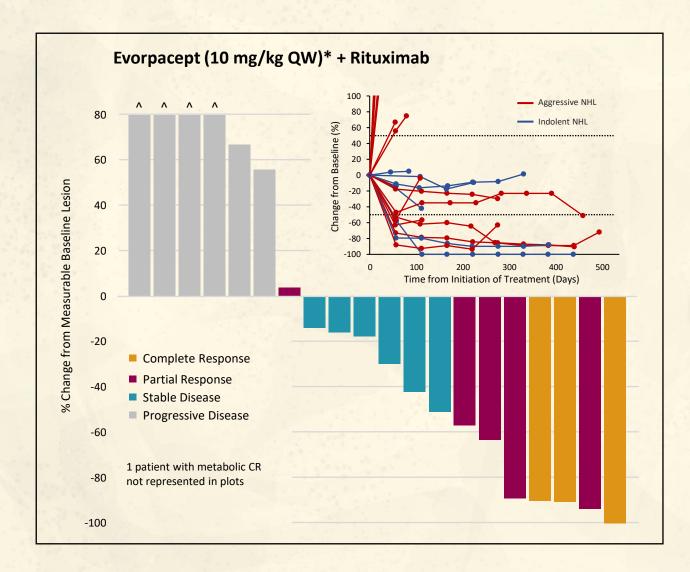
Treatment Related Adverse Event	Total n (%)	≥Grade 3 n (%)
Rash	8 (24.2)	
Fatigue	4 (12.1)	_
Nausea	2 (6.1)	-
Neutrophil Count Decreased	2 (6.1)	2 (6.1)
Anemia	2 (6.1)	1 (3.0)
Myalgia	2 (6.1)	
Pruritus	2 (6.1)	

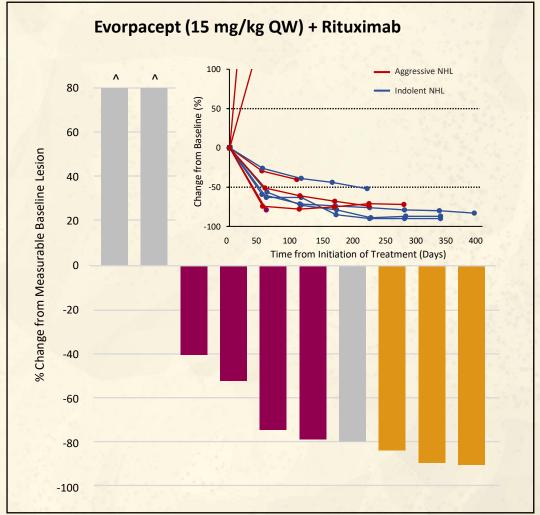
Data Cutoff: October 1, 2020



NHL: CLINICAL ACTIVITY OF EVORPACEPT + RITUXIMAB BY PATIENT



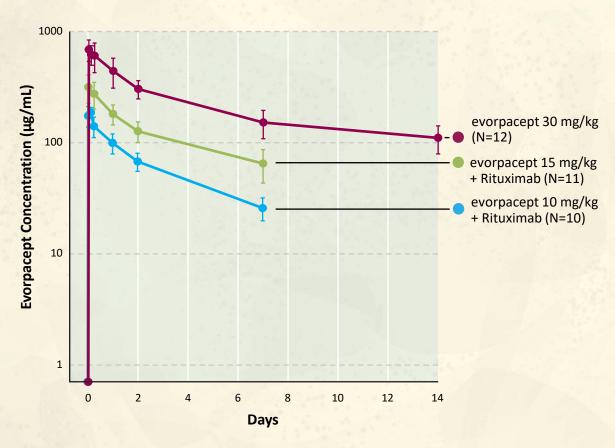


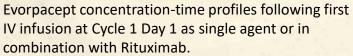


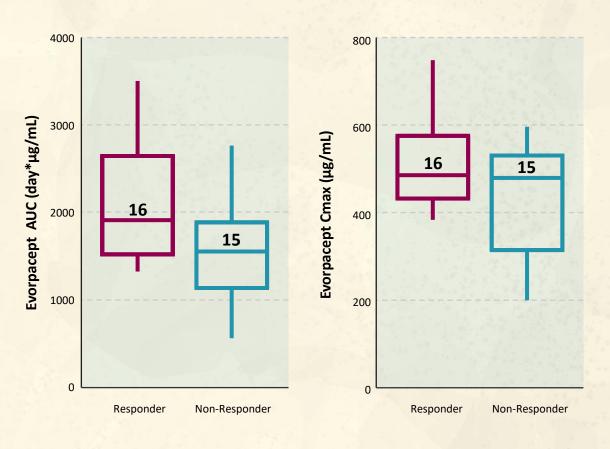


NHL: EVORPACEPT CLINICAL PHARMACOKINETICS AND EXPOSURE-RESPONSE ANALYSIS









^{*}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).



Data Cutoff October 1, 2020

NON-HODGKIN LYMPHOMA PROOF-OF-PRINCIPLE SUMMARY





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 evorpacept + 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 RAINBOW¹	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/paclitaxel²	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 evorpacept (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 Arm⁴	62	11%	3.9	3.5	8.4	29%	



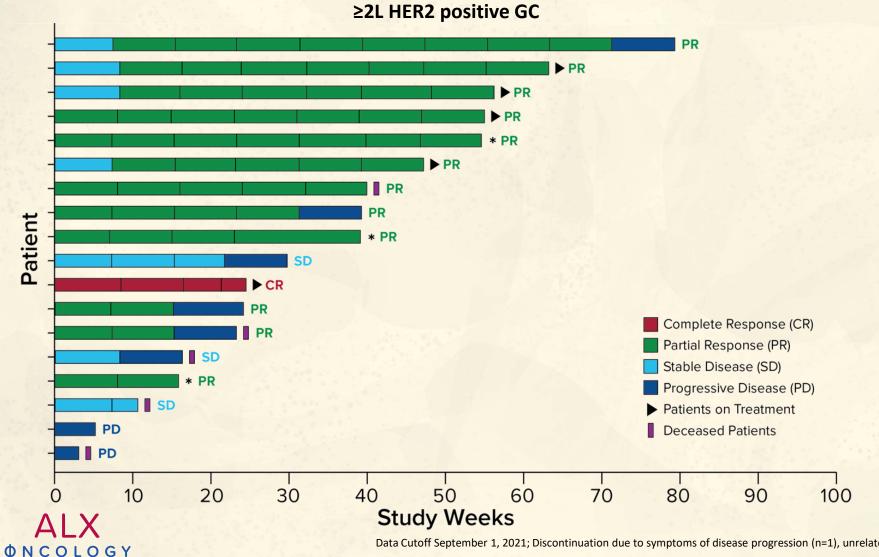
PHASE 1B ≥2 LINE GC TRIAL: EVORPACEPT + HERCEPTIN + CYRAMZA + PACLITAXEL TREATMENT EMERGENT ADVERSE EVENTS

Evorpacept + Trastuzumab + Ramucirumab + Paclitaxel (N=18) / Adverse Event, n (%)

Grade		ALL Causality Evorpacept - r						
Evorpacept Dose QW	G1-2	G3	G4	G1-2	G3	G4		
Neutrophil Count Decreased	3 (17)	5 (28)	3 (17)	70 - 7				
Epistaxis	9 (50)	4 - 2 / - 10 Y		Te 1 - 1	-9	100 11 11 12 13		
Peripheral Neuropathy / Peripheral Sensory Neuropathy	8 (44)	1 (6)		and the Common terms of	_	-432		
Decreased Appetite	8 (44)		- M-	Section 1	- 13 _ - 1	Lo later to		
Fatigue	7 (39)	1 (6)		2 (11)	Cart - 1.			
Anemia	3 (17)	4 (22)	_	1 (6)				
Hypertension		6 (33)	1 1 1 1 0 2	_		- "		
Abdominal Pain / Abdominal Pain Upper	5 (28)			1 (6)		polesi – Kajaka		
Headache	5 (28)	-2/1	10 -6 10	1 (6)				
Stomatitis	5 (28)	5	26.3	1 (6)		1 1 2 7 1		
Alanine Aminotransferase Increased	4 (22)			-				
Alopecia	4 (22)			-				
Aspartate Aminotransferase Increased	3 (17)	1 (6)	-	-	547 v= 1.33	10 To		
Asthenia	3 (17)	1 (6)		4				
Diarrhea	4 (22)		-	3 (17)	_	3700-100		
Insomnia	4 (22)	= / \	_	-		Section 1		
Rash/Dermatitis Acneiform	4 (22)	4 4 4 3		4 (22)		CARL NO.		
Pruritis	3 (17)		_	2 (11)	_	- 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1		
Urticaria	3 (17)	_	_	3 (17)				
Back Pain	2 (11)	_		1(6)				
Diverticulitis	1 (6)	1 (6)		-	_			
Dysphagia	1 (6)	1 (6)	- 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1	1		N/=121 = //		
Hypophosphatemia	1 (6)	1 (6)	_	-				
Platelet Count Decreased	1 (6)	1 (6)		_		= \ / / _		
Hydronephrosis		1 (6)		V 17		-		
Lymphocyte Count Decreased		1 (6)		-	1 (6)			
Non-Cardiac Chest Pain		1 (6)	_	-		- 012		
Urinary Tract Infection	T	1 (6)	C4 _ 4 . (4) (4)		20-1	1 1 1 1 4 2 1 1 1 1 1 1 1 1 1 1 1 1 1 1		
Vision Blurred	1 (6)			1 (6)	# A - 1 11	A. J. A. B.		

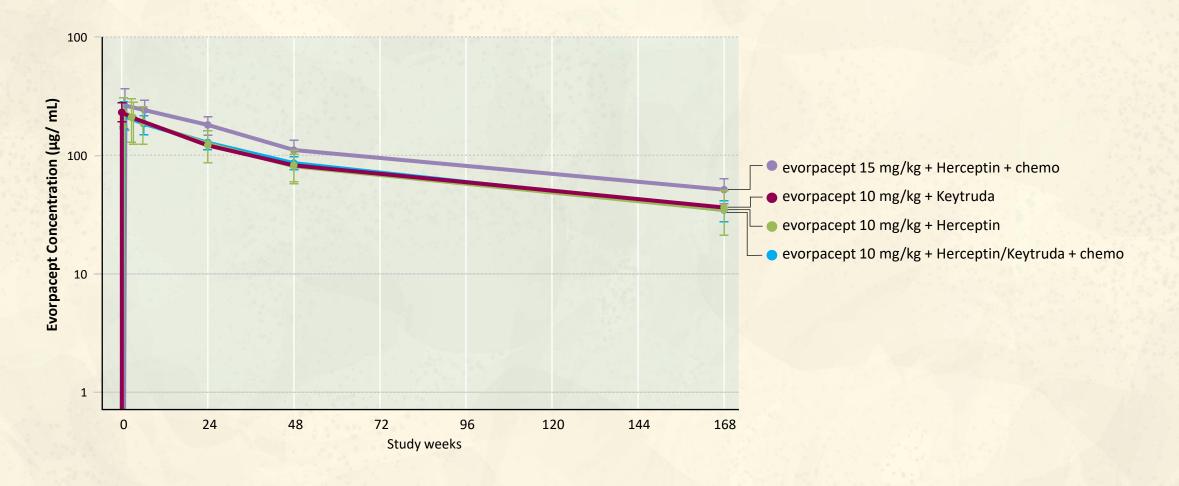


PHASE 1B ≥2 LINE GC TRIAL: **EVORPACEPT + HERCEPTIN + CYRAMZA + PACLITAXEL** BEST OVERALL AND DURATION OF RESPONSE WHILE ON TREATMENT

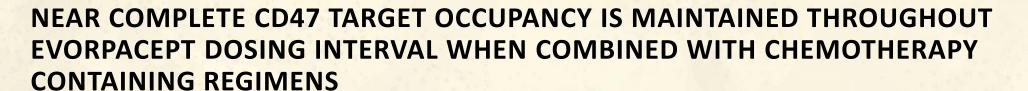


EVORPACEPT PK FOLLOWING COMBINATION THERAPIES IS COMPARABLE WITH AND WITHOUT CHEMOTHERAPY

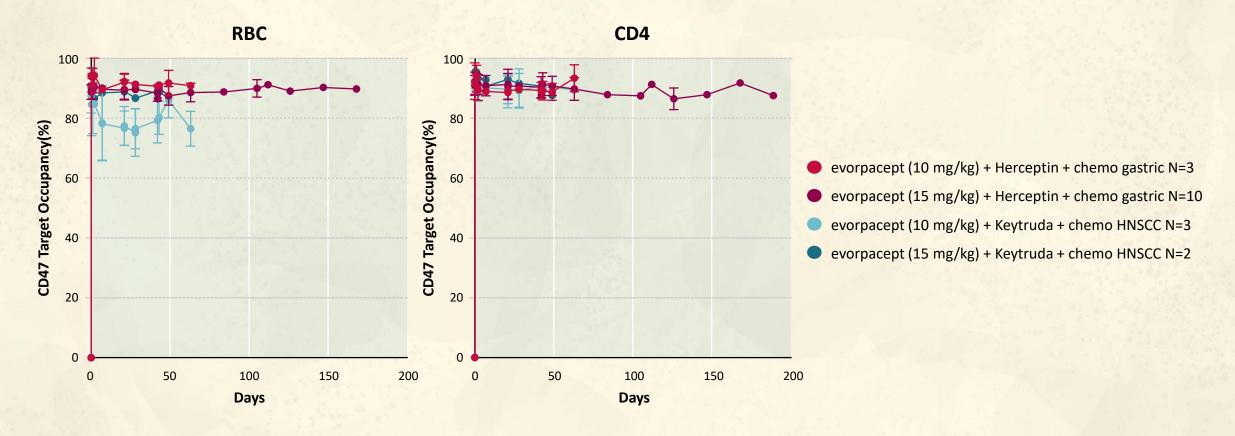










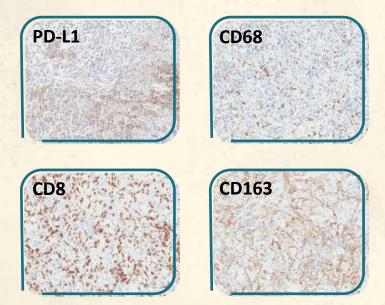




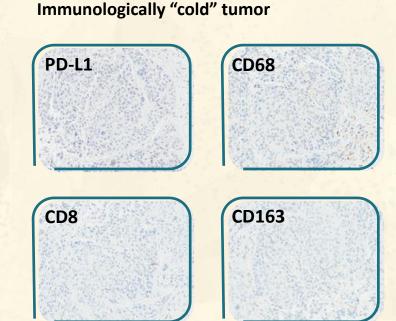
PRE-TREATMENT IHC RESULTS IN HOT AND COLD TUMORS HNSCC PATIENT 1 (PD-L1 POSITIVE) AND PATIENT 2 (PD-L1 NEGATIVE)



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

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.





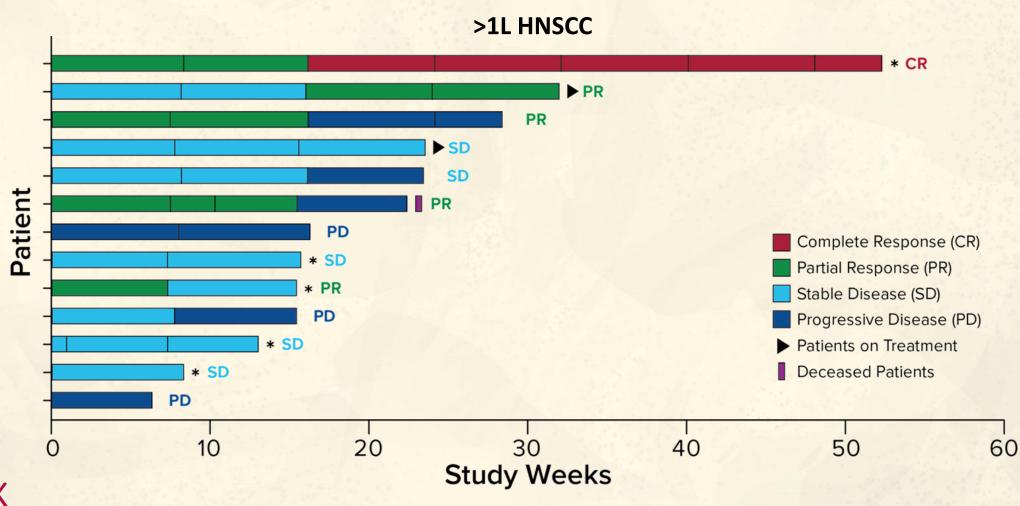
PHASE 1B FIRST LINE HNSCC: EVORPACEPT + KEYTRUDA + 5FU/PLATINUM TREATMENT RELATED ADVERSE EVENTS

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

Grade	Α	ALL Causality			Evorpacept - Related			
Evorpacept Dose QW	G1-2	G3	G4	G1-2	G3	G4		
Anemia	4 (31)	4 (31)	21/41/	- ×-!	1 (8)			
Nausea	8 (62)	-	V9-77	-	e			
Stomatitis	7 (54)	1 (8)	-	<u>-</u>	=	<u> -</u>		
Neutrophil Count Decreased / Neutropenia	2 (15)	5 (38)	2 / -	1 (8)	=	- Y-13		
Platelet Count Decreased /Thrombocytopenia	7 (54)	16 -	-	-	3	7- %		
Fatigue	5 (38)	'& <u>-</u>	<u> </u>	1 (8)		R 4 - 19 1		
Alanine Aminotransferase Increased	3 (23)	1 (8)	-					
Dysphagia	1 (8)	1 (8)	\ ·		- /	_		
Hypersensitivity	1 (8)	3/ I-	1 (8)	- /		1 (8)		
Pneumonia	1 (8)	1 (8)	- L	-	-	-		
Pneumonitis	2 (15)		_	1 (8)	-	4		
Candida Infection		1 (8)	-	6.3/ <u>+</u>	-	-		
Cardiac Tamponade			1 (8)	165				
Headache		1 (8)			-1-1 <u>-</u>	2.2		
Pericarditis Constrictive		1 (8)	-	- 1	<u> - 12 14 14 14 14 14 14 14 14 14 14 14 14 14 </u>	4		
Supraventricular Tachycardia		1 (8)		-	<u>-</u> - 1	1		
Tracheal Obstruction	_	1 (8)		_	= 4	- /		



PHASE 1B FIRST LINE HNSCC: EVORPACEPT + KEYTRUDA + 5FU/PLATINUM BEST OVERALL AND DURATION OF RESPONSE WHILE ON TREATMENT

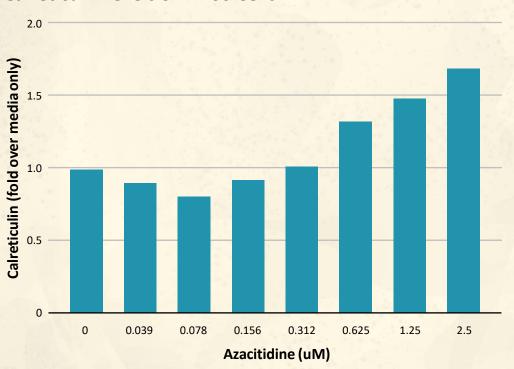


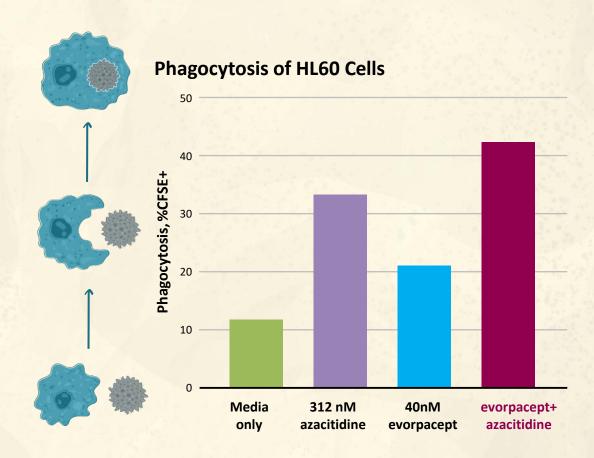
ONCOLOGY

PRECLINICAL: EVORPACEPT INCREASES PHAGOCYTOSIS OF BACKBONE MDS DRUG, AZACITIDINE



Calreticulin levels on HL60 Cells



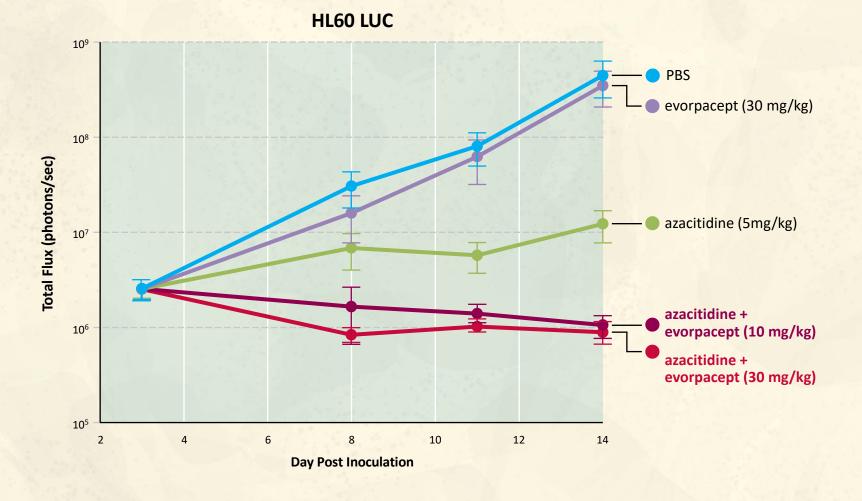


Azacitidine induces calreticulin display. Evorpacept increases phagocytosis in combination with azacitidine.



EVORPACEPT INCREASES TUMOR INHIBITION OF AZACITIDINE

evorpacept in MDS



Combination opportunity in MDS and AML

Disseminated AML mouse model



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

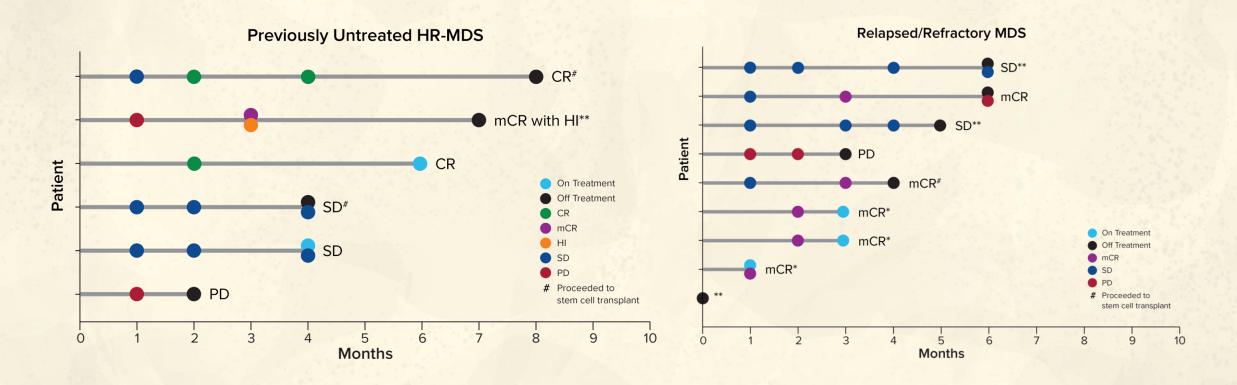
	20 mg/kg Q2W 30 mg/kg Q2W (N=3) (N=3)		60 mg/kg Q4W (N=16)		Total (N=22)		
Adverse Event, n	Grade 1/2	Grade 3/4	Grade 1/2	Grade 3/4	Grade 1/2	Grade 3/4	All Grade n (%)
Blood Creatinine Increased	2	-	1	4 7 - 4	2	-	5 (23)
Constipation	1	4	1		2	1	5 (23)
Diarrhea	1		1	- //	3		5 (23)
Fatigue	-		-	_	4	1	5 (23)
Neutropenia/Neutrophil Count Decreased	-		1	1	1	3	5 (23)
Anemia	1	1	1		1 2	1	4 (18)
Dizziness	-	-	1		3		4 (18)
Dyspnea	1		1	_	2	1	4 (18)
Febrile Neutropenia	_	2			<u> </u>	2	4 (18)
Infusion Related Reaction	_				4	_	4 (18)
Nausea	-	\pm	1		3	_	4 (18)
Abdominal Pain	1		1	S	1		3 (14)
Contusion	1	1	1	3/4-1/	1	1 -	3 (14)
Platelet Count Decreased	E-/-	2	-	1		- / = ,= l	3 (14)
Pneumonia	- Je i	1	-		A -	2	3 (14)
Transfusion Reaction	2	<u>-</u>	475 - N		1	/	3 (14)
Vomiting	1		<u> </u>	N07	2	14 E. C	3 (14)



Data Cutoff October 25, 2021 63

evorpacept in MDS

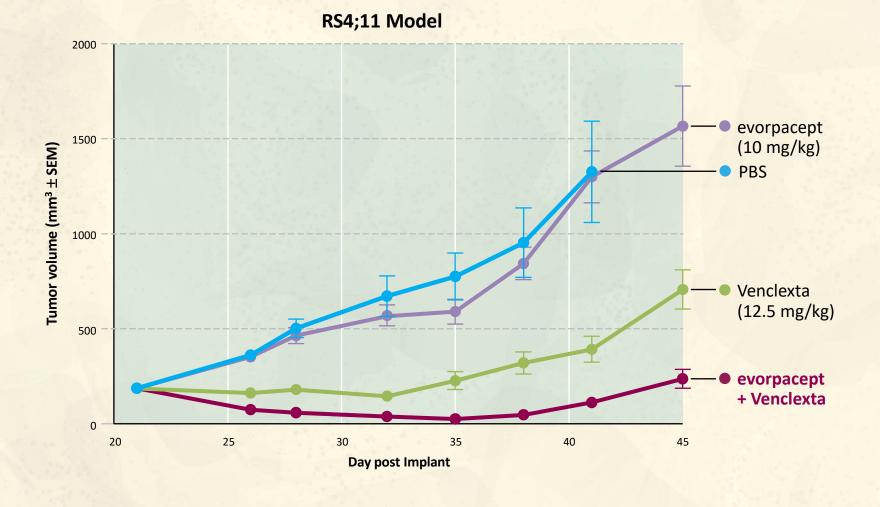
PHASE 1B MDS: EVORPACEPT + AZACITIDINE PREVIOUSLY UNTREATED HR-MDS AND RELAPSED/REFRACTORY MDS DURATION OF RESPONSE





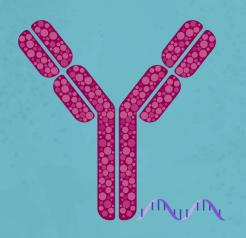
EVORPACEPT INCREASES TUMOR INHIBITION OF VENCLEXTA

evorpacept in AML



Combination opportunity in AML





EARLY STAGE PIPELINE: SIRPα-TRAAC COLLABORATION



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



Provides SIRPα antibody

- CD47-SIPR 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.

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

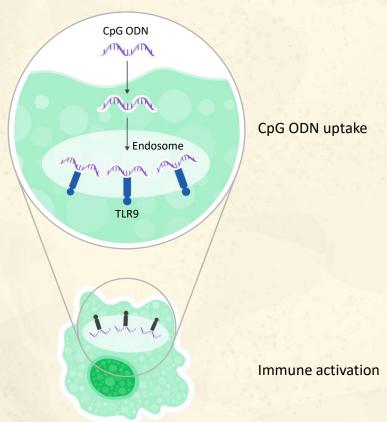
SIRPα TRAAC simultaneously overrides "don't eat me" signals by blocking CD47-SIRPα myeloid checkpoint pathway and induces TLR9-based immune activation in antigen presenting cells (APCs).



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.

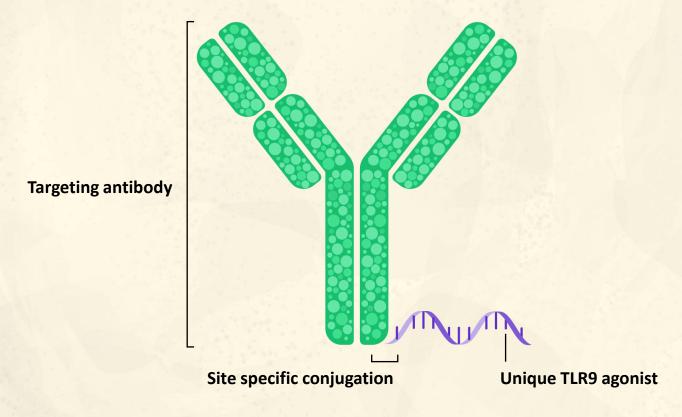
Additional clinical data from SD-101 (Dynavax), IMO-2125 (Idera) and AST-008 (Exicure) further validate TLR9 agonism in cancer.



TALLAC TRAAC PLATFORM: SYSTEMIC, TARGETED IMMUNE ACTIVATION ANTIBODY DIRECTS TLR9 AGONIST (T-CPG) TO SPECIFIC IMMUNE CELLS

TLR9 Agonist Antibody Conjugate (TRAAC):

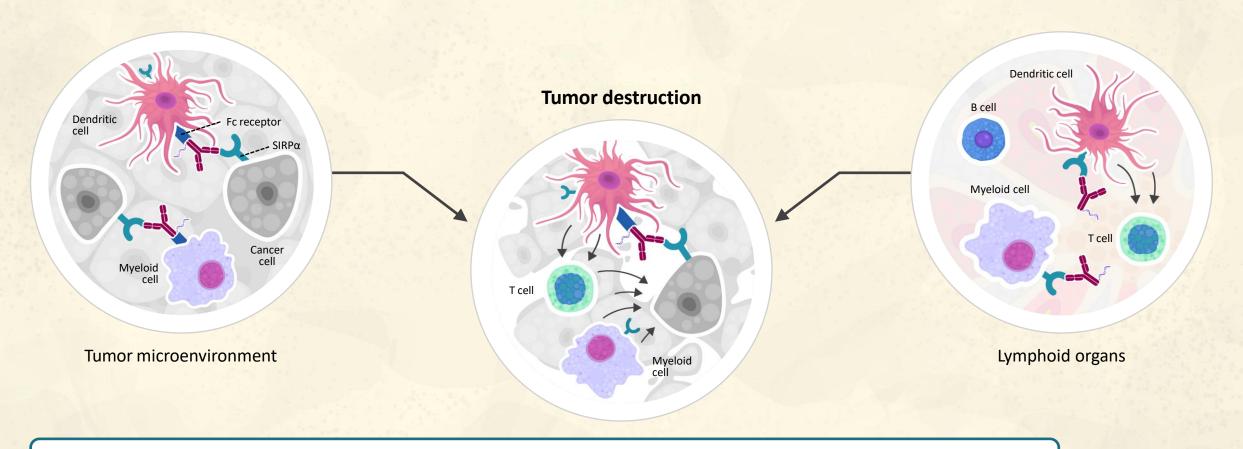
Systemic dosing with cell specific TLR9 activation



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



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



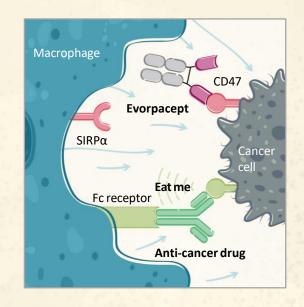
- SIRPα TRAAC binding to myeloid cells targets TLR9 activation in myeloid cells that matter (e.g. dendritic cells).
- SIRPα expression on tumor cells enables tumor microenvironment localization of SIRPα TRAAC.
- SIRPα TRAAC blocks CD47-SIRPα myeloid checkpoint pathway.

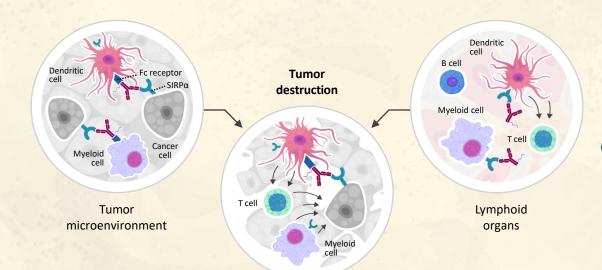


SIRPα TRAAC PROGRAM IS COMPLEMENTARY TO EVORPACEPT

Evorpacept 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.



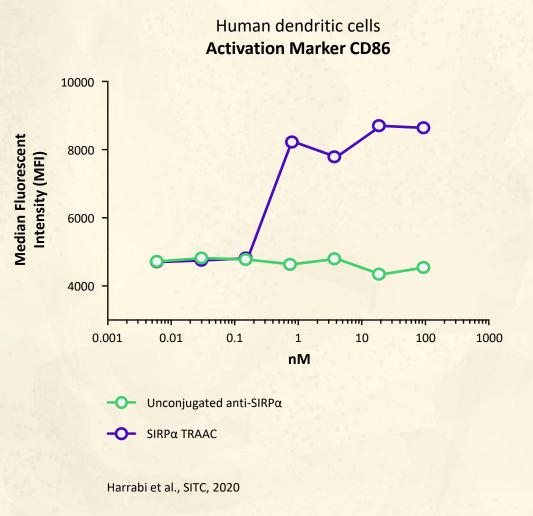


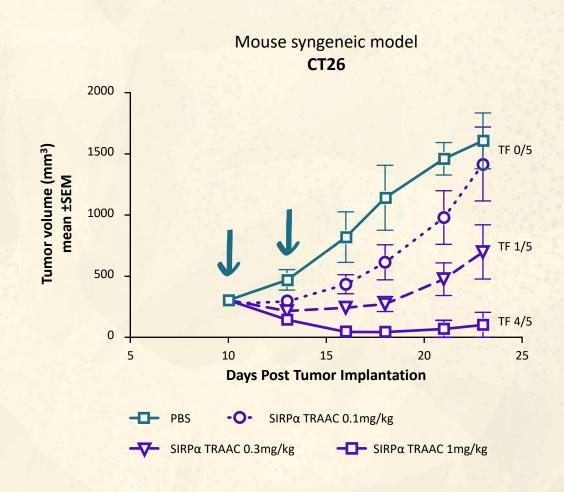
SIRP α TRAAC is an agonistic molecule that directly activates dendritic cells and initiates a coordinated innate and adaptive immune response against cancer.

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



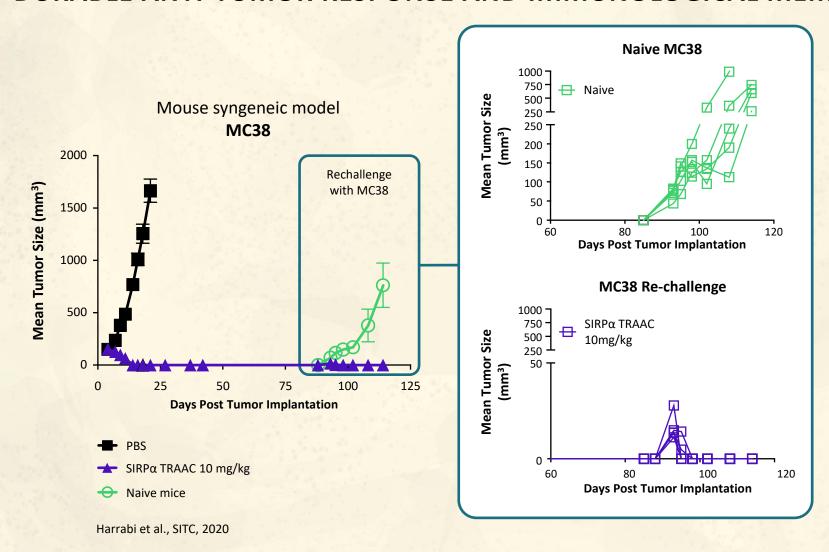
SIRPα TRAAC INDUCES POTENT AND SELECTIVE IMMUNE ACTIVATION AND LEADS TO POTENT SINGLE AGENT ACTIVITY IN TUMOR MODELS







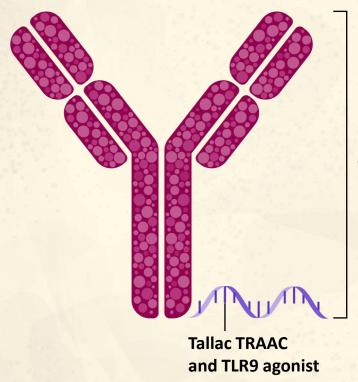
SYSTEMIC ADMINISTRATION OF SIRPα TRAAC GENERATES DURABLE ANTI-TUMOR RESPONSE AND IMMUNOLOGICAL MEMORY



- Established MC38 tumors were eradicated following 10 mg/kg 2q3d systemic treatment with SIRPα TRAAC.
- These tumor free mice were then rechallenged 60-70 days post tumor clearance.
- SIRPα 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



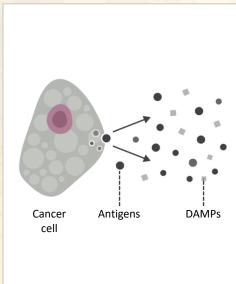
ALX anti-SIRPα antibody

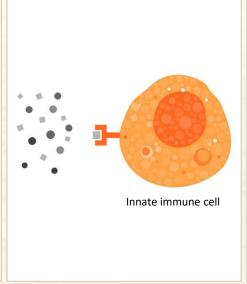
- SIRPα TRAAC binding to myeloid cells targets TLR9 activation in key myeloid cells (e.g. dendritic cells).
- SIRP α expression on tumor cells enables localization of SIRP α TRAAC to tumor microenvironment.
- SIRPα TRAAC blocks CD47-SIRPα myeloid checkpoint pathway.
- Antibody-like PK profile allows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

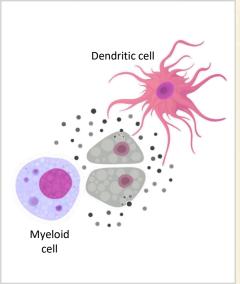
IND expected beginning of 2023

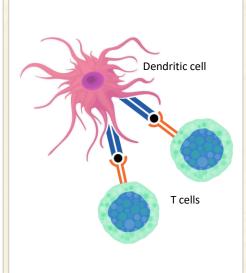


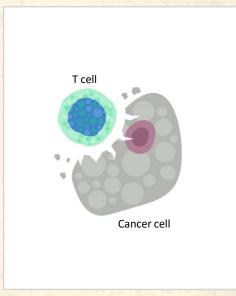
HARNESSING THE POWER OF INNATE AND ADAPTIVE IMMUNE RESPONSES TO CANCER











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

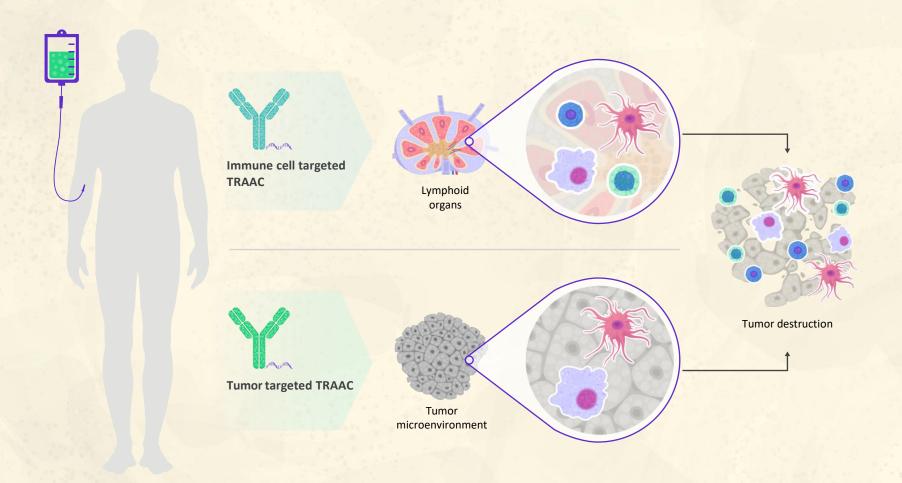
- Successful immune-mediated elimination of cancer requires coordination between the innate and adaptive arms of the immune system.
- Innate immune cells (macrophages, myeloid cells and dendritic cells) may utilize a variety of signaling pathways, including TLR9, TLR7 or 8, STING and CD40, to trigger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

DAMPs: damage-associated molecular patterns PAMPs: pathogen-associated molecular patterns

PRRs: pattern recognition receptors

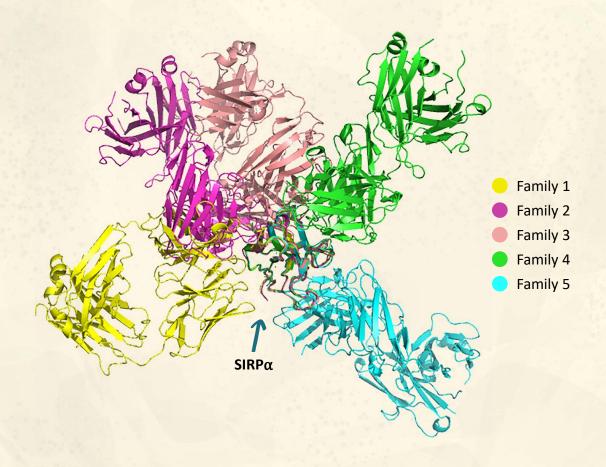


TLR9 AGONIST ANTIBODY CONJUGATE (TRAAC) ENABLES VERSATILE TARGETING OF IMMUNE CELLS THAT MATTER





ALX ONCOLOGY'S SIRPα ANTIBODIES: HIGH AFFINITY AND DIVERSE EPITOPES



ALX's diverse range of SIRPα antibodies

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

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

