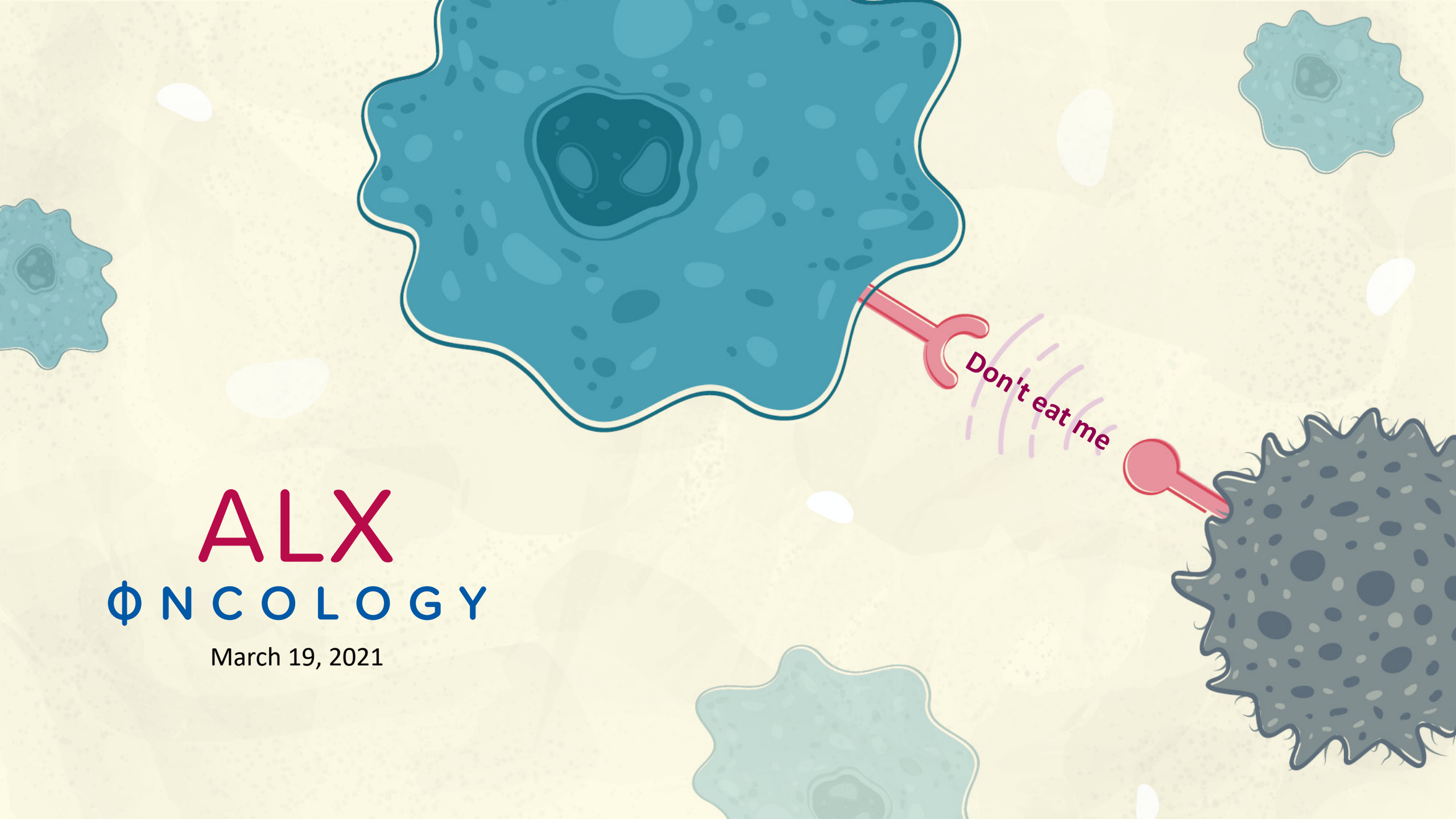

ALX ONCOLOGY

March 19, 2021

DISCLAIMER

This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation, including statements regarding our future 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, are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "would," "expect," "plan," "anticipate," "could," "intend," "target," "project," "believe," "estimate," "predict," "potential," or "continue" or the negative of these terms or other similar expressions.

We have based these forward-looking statements largely on our current expectations and projections about our business, the industry in which we operate and financial trends that we believe may affect our business, financial condition, results of operations and prospects and these forward-looking statements are not guarantees of future performance or development. These forward-looking statements speak only as of the date of this presentation and are subject to a number of risks, uncertainties and assumptions, including, among other things: our history of incurring significant net losses since our inception and our expectation that we will continue to incur significant net losses for the foreseeable future; sufficiency of our cash and cash equivalents to fund our planned operations; the need for additional capital to finance our operations; our limited operating history and absence of products approved for commercial sale; our substantial dependency on the success of our lead product candidate, ALX148, which is in clinical development and which has not completed a pivotal trial; the fact that outcome of preclinical testing and early clinical trials may not be predictive of the success of later clinical trials, and the results of our clinical trials may not satisfy the requirements of the Food and Drug Administration ("FDA") or other comparable foreign regulatory authorities; the possibility that our product candidates may cause significant adverse events or other undesirable side effects when used alone or in combination with other treatments; the fact that the clinical trials of our product candidates are expensive, time consuming and difficult to design and implement and may fail to demonstrate adequate safety, efficacy and potency of our product candidates or provide the basis for marketing approval; the lengthy, time-consuming and inherently unpredictable nature of the regulatory approval processes of the FDA and comparable foreign regulatory authorities, which could lead to our inability to generate product revenue; our ability to obtain, maintain and enforce patent protection and other intellectual property for our product candidates and related technology; our dependency on our key personnel and our ability to successfully attract, motivate and retain highly qualified personnel; the potential adverse impact of COVID-19 on our business, including our ongoing and planned clinical trials and preclinical research; and material weaknesses in our internal control over financial reporting. These risks are not exhaustive. New risk factors emerge from time to time and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements.

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This presentation concerns drugs that are under clinical investigation and which have not yet been approved for marketing by the FDA. It is currently limited by Federal law to investigational use, and no representation is made as to its safety or effectiveness for the purposes for which it is being investigated.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. 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 our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

ALX ONCOLOGY

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ALX ONCOLOGY

OVERVIEW

ALX Oncology (Nasdaq: ALXO) is a clinical-stage immuno-oncology company focused on helping patients fight cancer by developing therapies that block the CD47 checkpoint pathway and bridge the innate and adaptive immune system

Lead product candidate ALX148 initiating multiple Phase 2 trials

CD47 blocker

- Designed for use in combination
- Tolerability profile enables higher dosing
- Higher dosing may enable greater efficacy

Clinical proof-of-principle in both hematologic and solid tumors

Initial focus on solid tumors, MDS, and AML

Early-stage antibody candidate for systemic CpG delivery (SIRPα TRAAC*)

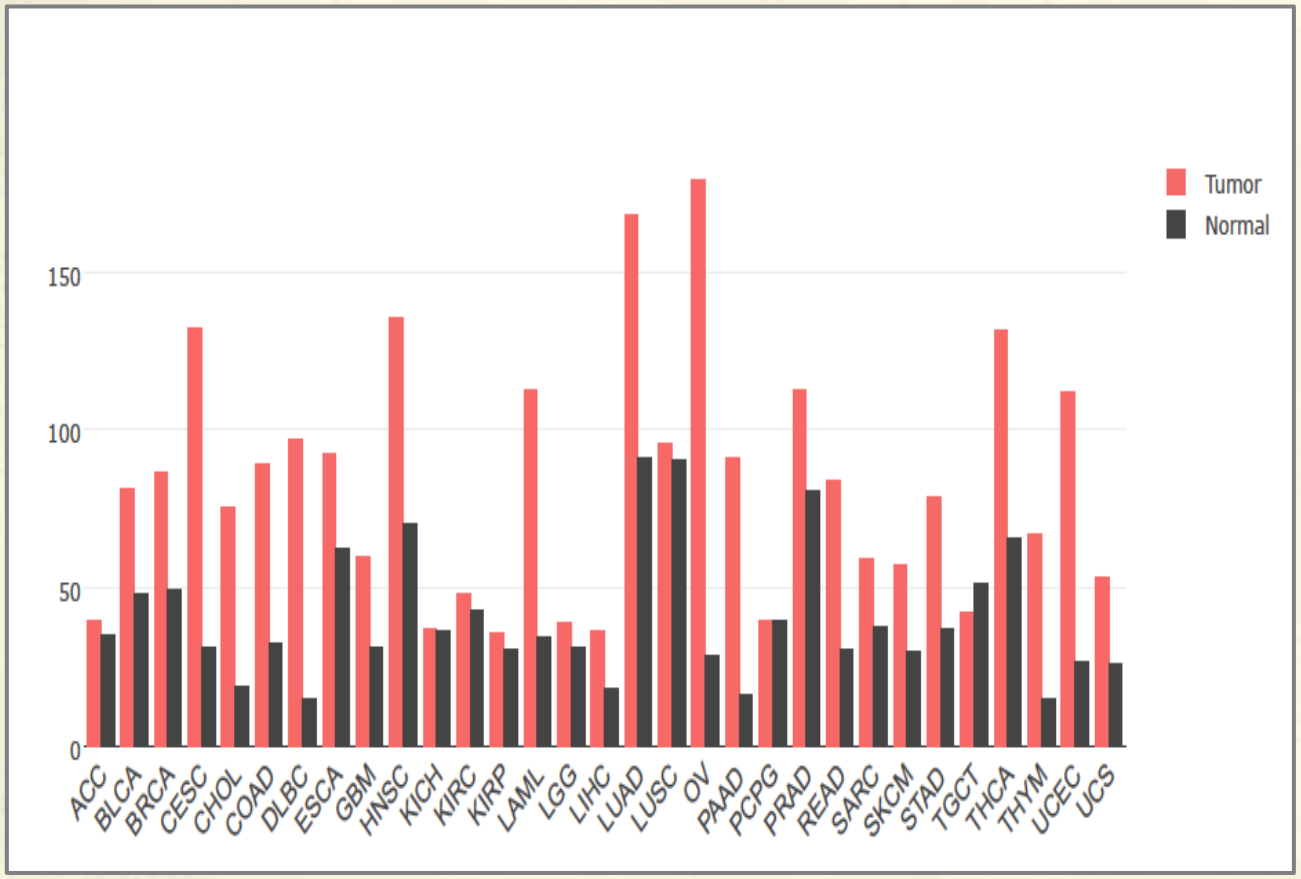
- IND by end of 2022

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

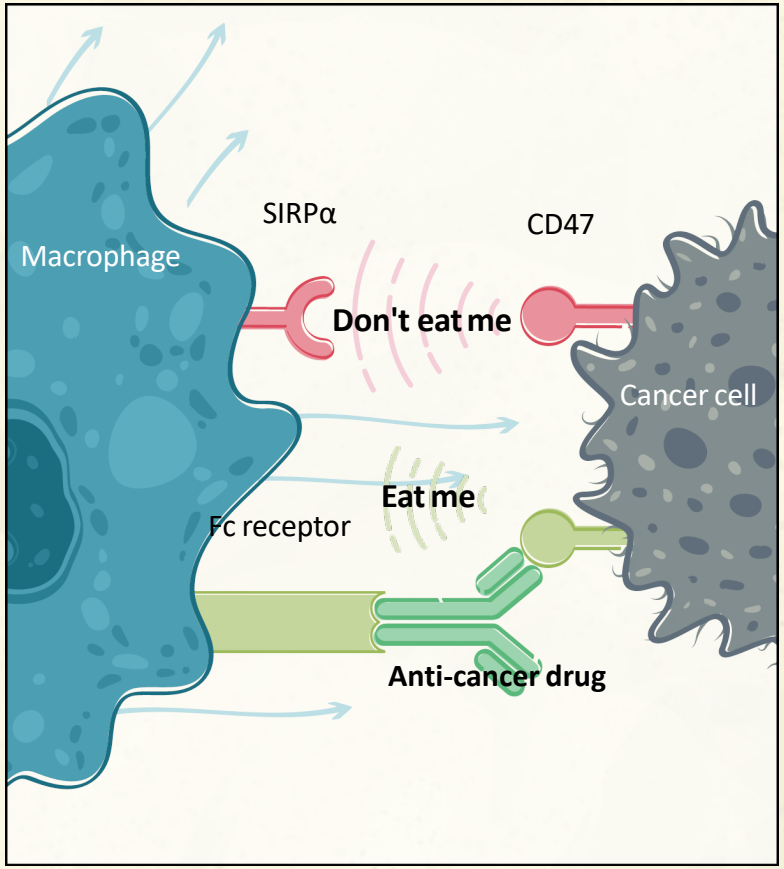
ALX ONCOLOGY

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

TAA-Expression levels on cancer and normal cells



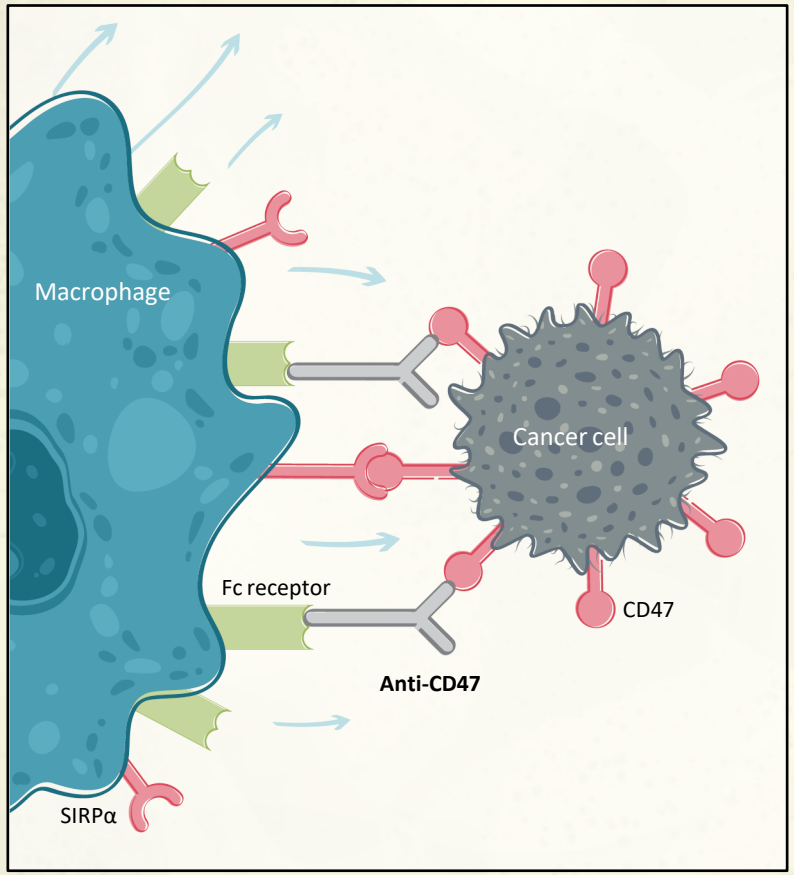
Checkpoint Mechanism: "do not eat me"

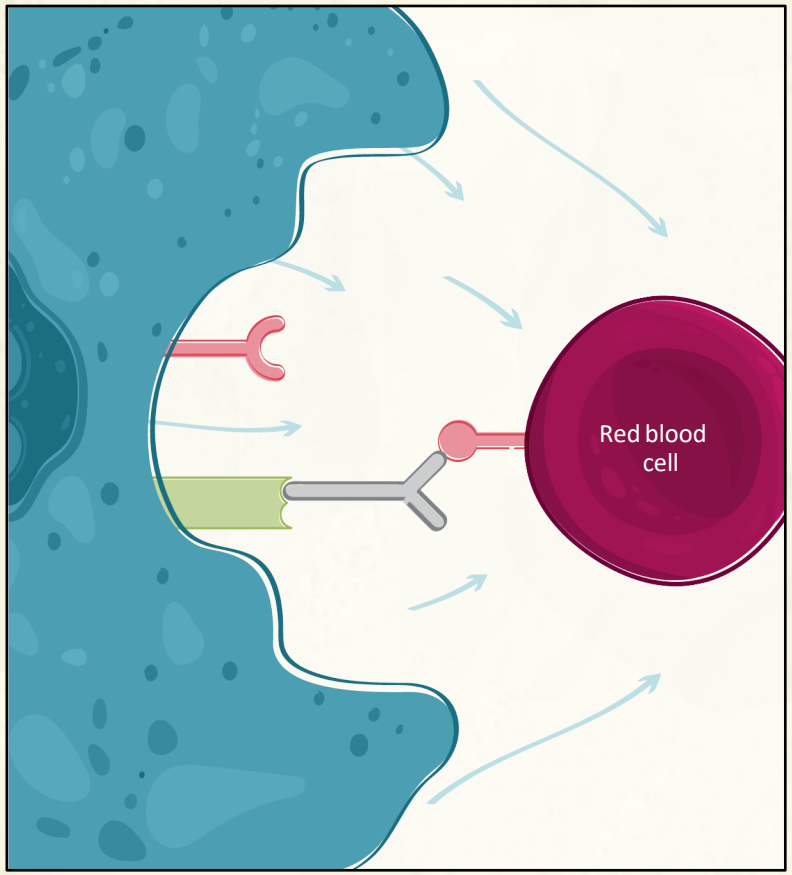


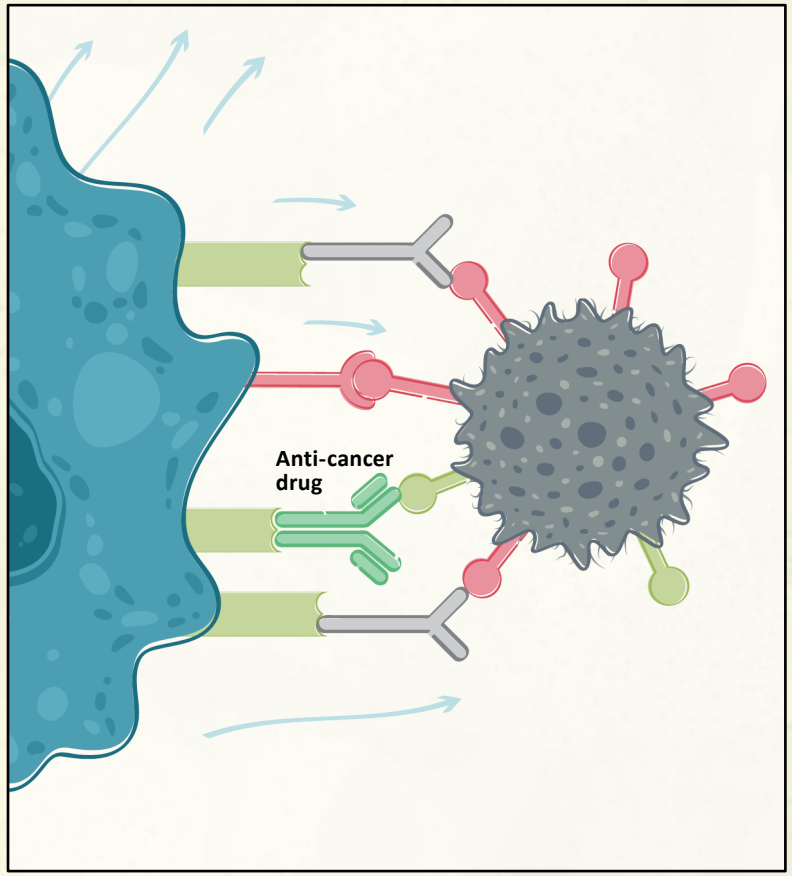
ALX ONCOLOGY

TARGETING CD47 AS TUMOR ASSOCIATED ANTIGEN

But also targets normal cells







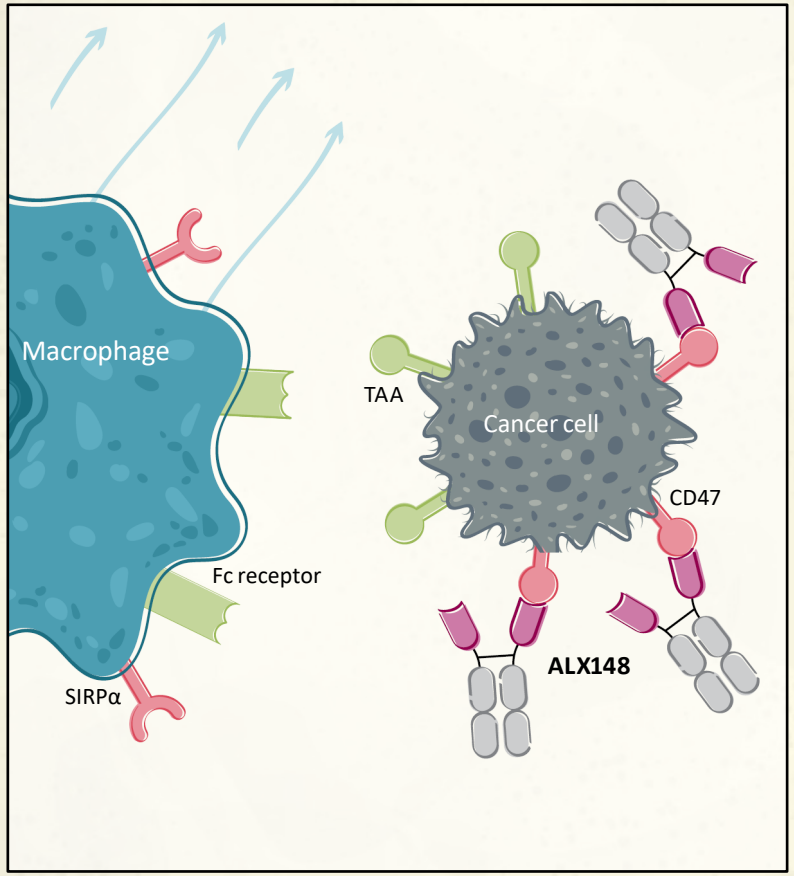
Anti CD47 with active Fc directly targets cancer cells

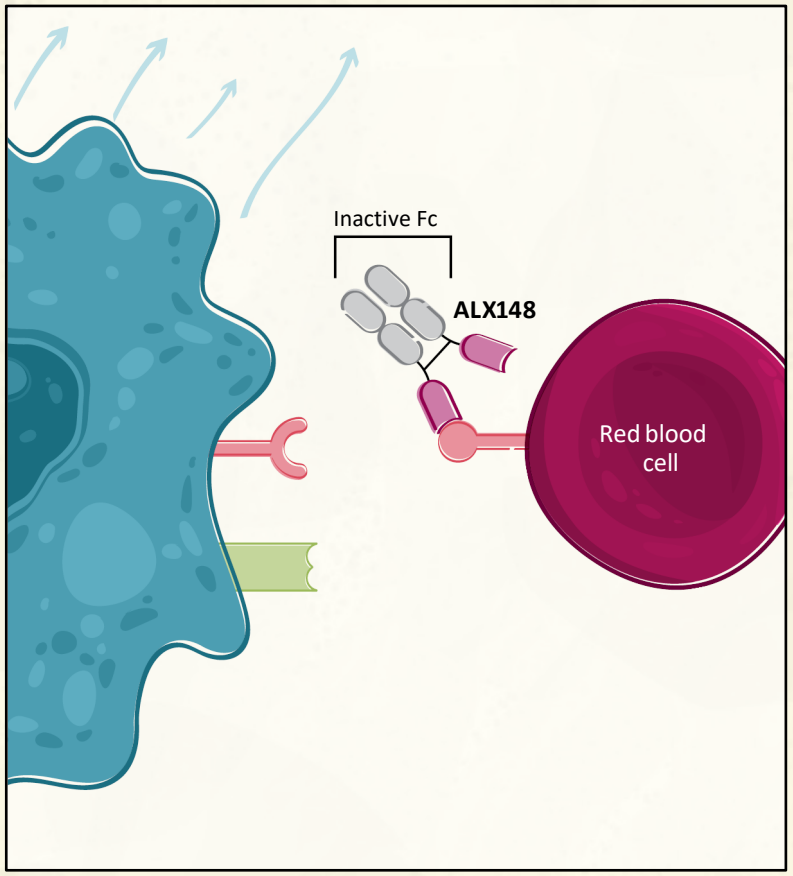
Dose limitations prevent full blockade of CD47 and active Fc competes with combo drug

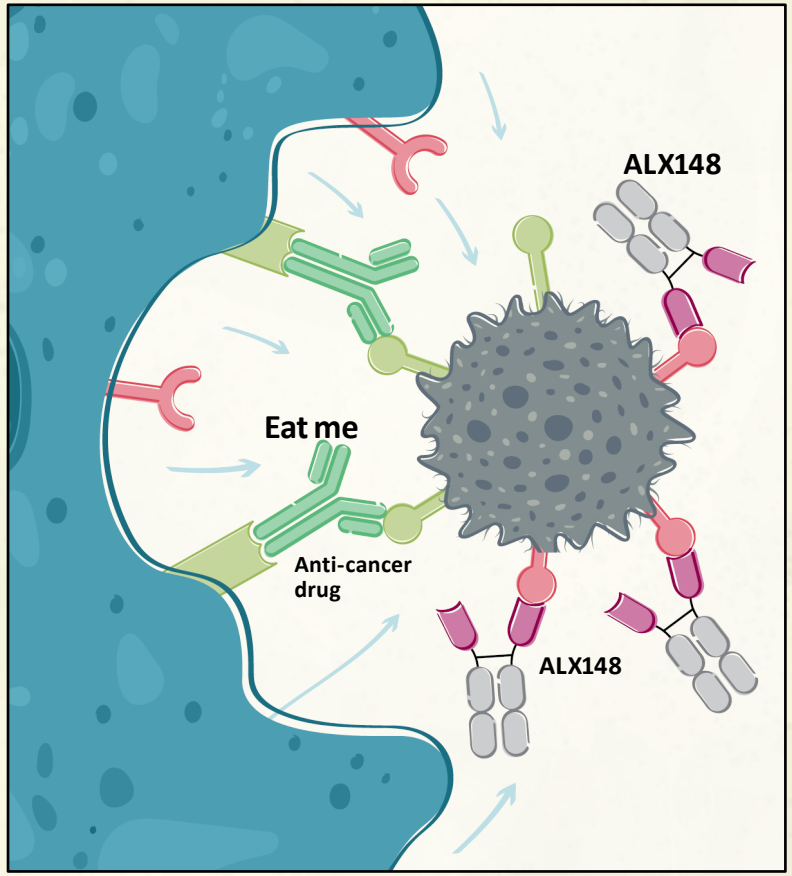
ALX ONCOLOGY

TARGETING CD47 AS CHECKPOINT: ALX ONCOLOGY'S APPROACH

It spares normal cells







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

High dose allows full blockade of CD47 and maximizes activity of combo drug

ALX ONCOLOGY

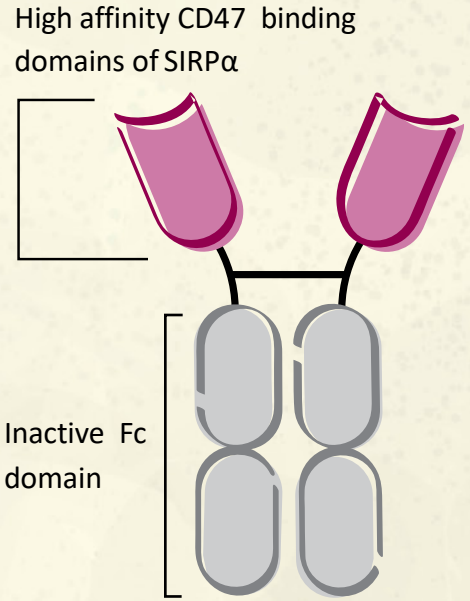
ALX148: METICULOUSLY DESIGNED CD47 BLOCKER

High affinity CD47 binding domain of SIRPα → **Potently blocks CD47 signal on cancer cells**

Inactive Fc domain eliminates binding activity → **No dose dependent cytopenia**

Presence of Fc domain ensures slow clearance and long half-life → **Less frequent dosing**

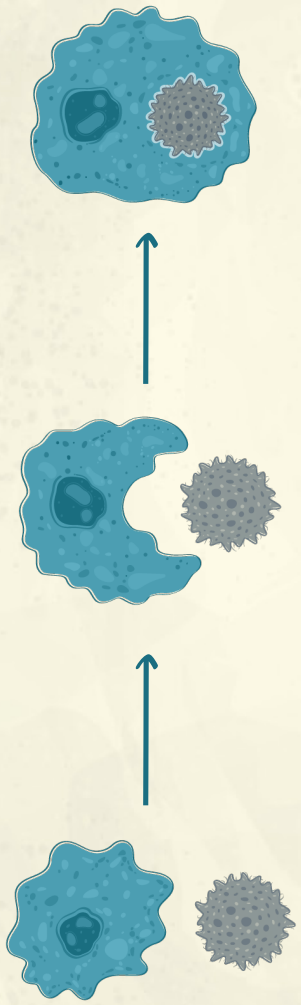
Designed for safety and efficacy

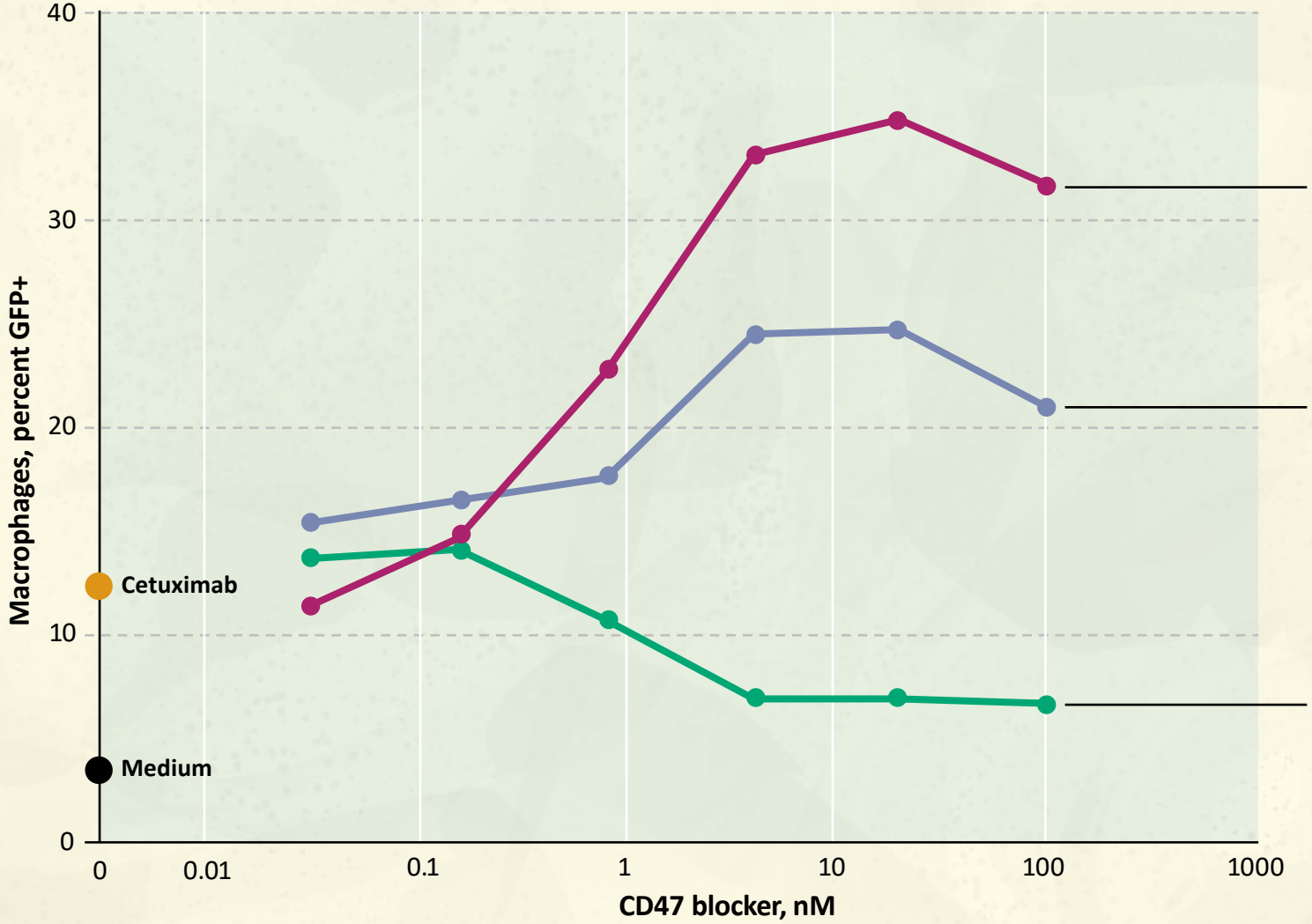


- ~Half the molecular weight of an antibody
- Increases solid tumor penetration
- Cross-reactive to human, monkey, mouse
- Standard antibody manufacturing process

ALX ONCOLOGY

ALX148 DEMONSTRATES SUPERIOR PHAGOCYTOSIS





ALX148 inactive Fc + Cetuximab

ADCP by cetuximab is enhanced by inactive Fc and high affinity

5F9 (Magrolimab) IgG4 + Cetuximab

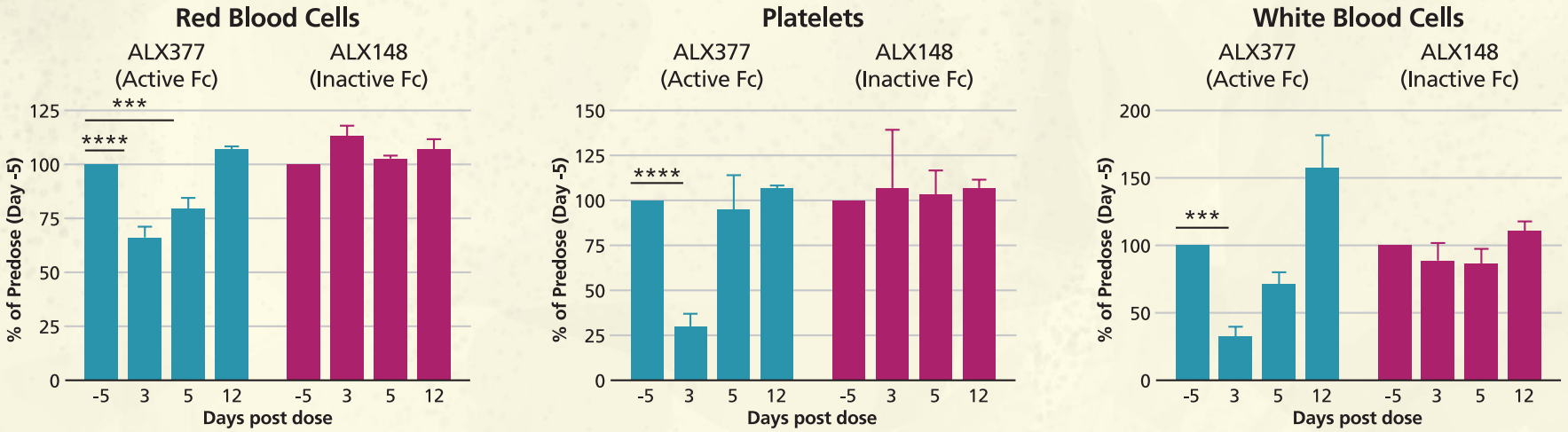
Enhances ADCP with cetuximab but at lower levels

TTI-621 IgG1 + Cetuximab

Does not enhance ADCP in combination with cetuximab

ALX ONCOLOGY

INACTIVE FC SHOWS REDUCED CYTOPENIA IN MICE IN VITRO



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

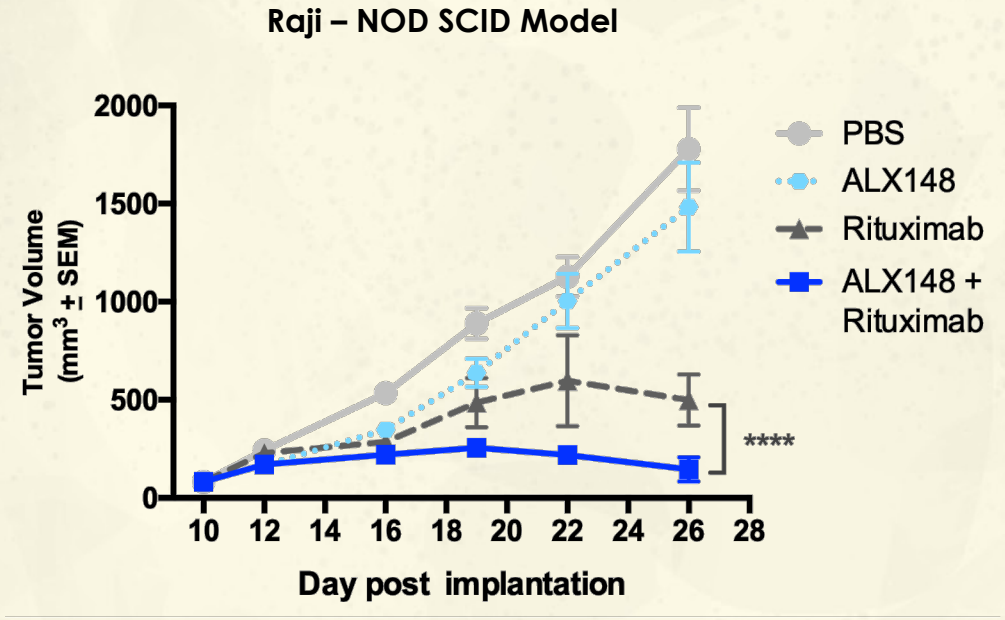
****p<0.0001, ***p<0.001

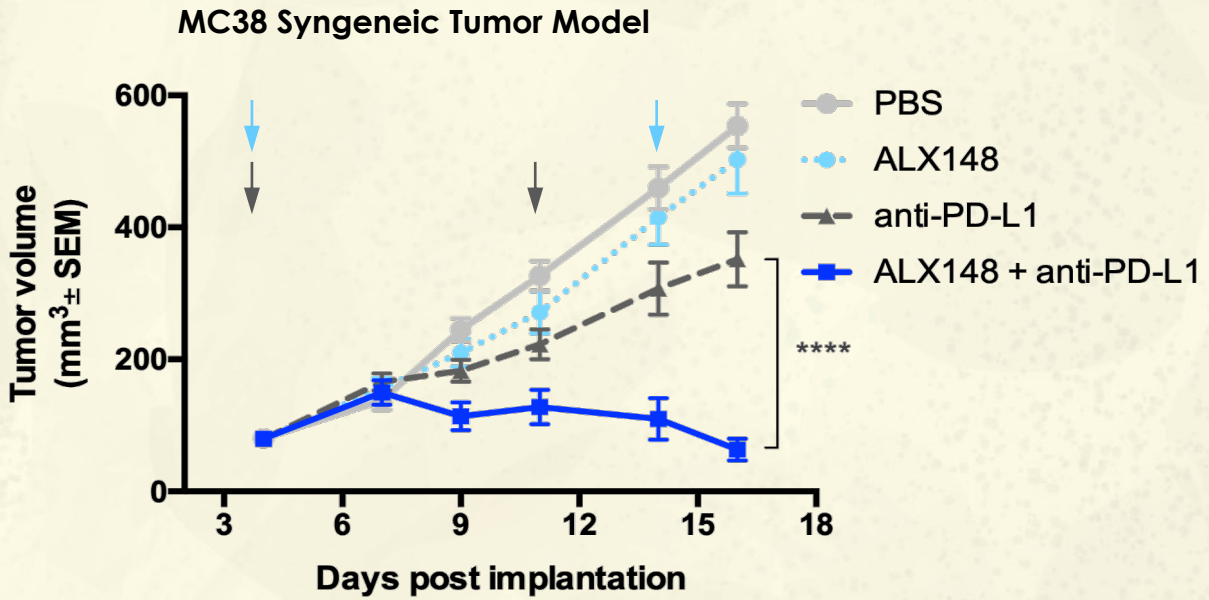
Mouse cross-reactivity allows for safety and efficacy testing in mouse models

Inactive Fc is the core determinant of safety profile

ALX ONCOLOGY

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

ALX ONCOLOGY

ALX148 DEMONSTRATES FAVORABLE TOLERABILITY PROFILE

	Preclinical	Single agent	Combinations
Highest administered dose	**100 mg/kg**[1] with no observable adverse events	**30 mg/kg Q2W**[2] No evidence of dose-dependent cytopenias	**15 mg/kg QW** currently dosed **60 mg/kg Q4W** planned

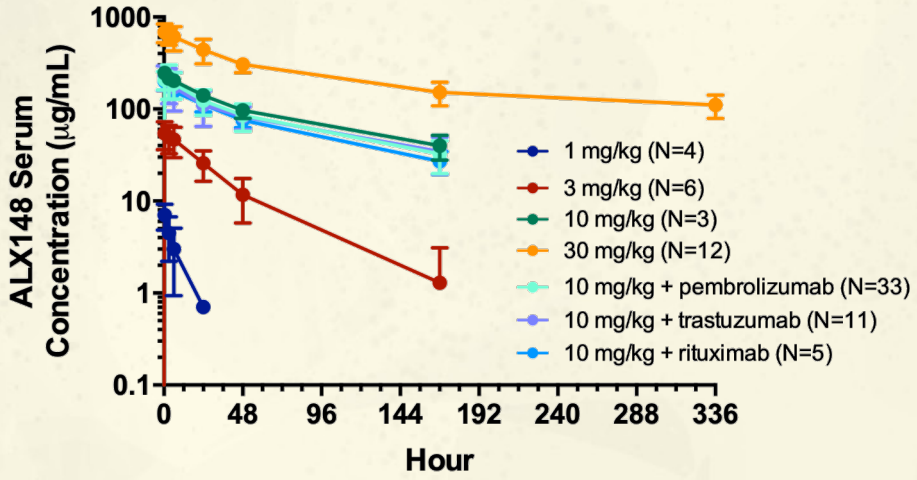
[1]100 mg/kg of ALX148 ≅ 200 mg/kg of a typical antibody
[2]Single agent safety, ALX presentation, ASCO 2018 poster

ALX148
has not yet reached a maximum tolerated dose

ALX ONCOLOGY

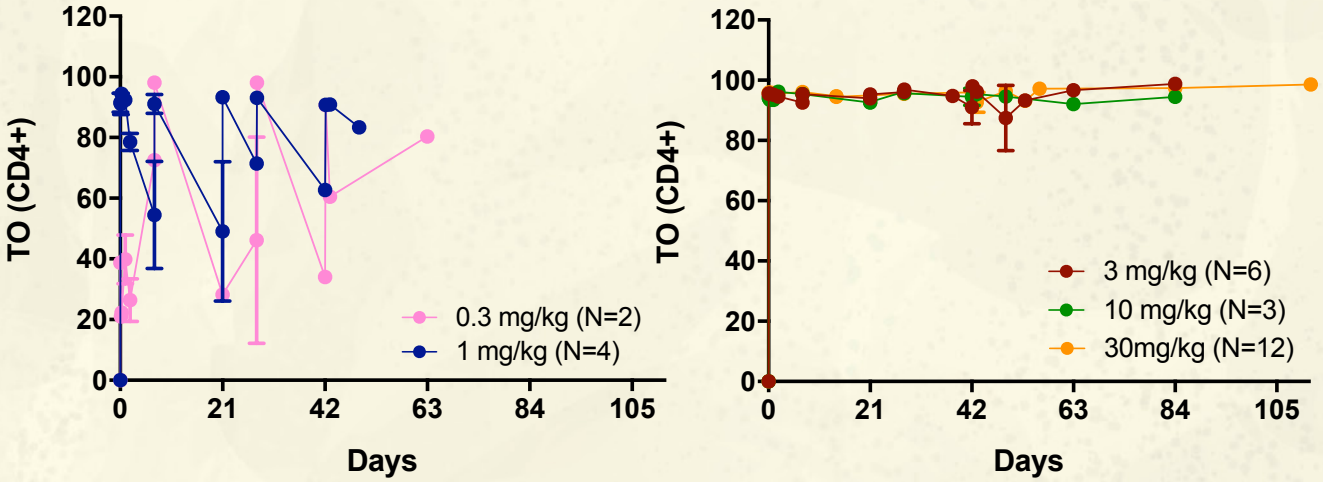
ALX148 CLINICAL PHARMACOKINETICS AND CD47 TARGET OCCUPANCY

ALX148 Serum Levels for Cycle 1 Day 1



- **Steady-state half-life of ALX148 at 10 mg/kg QW is predicted to be ~30 days.**
- ALX148 PK profile is not impacted by combination drugs.

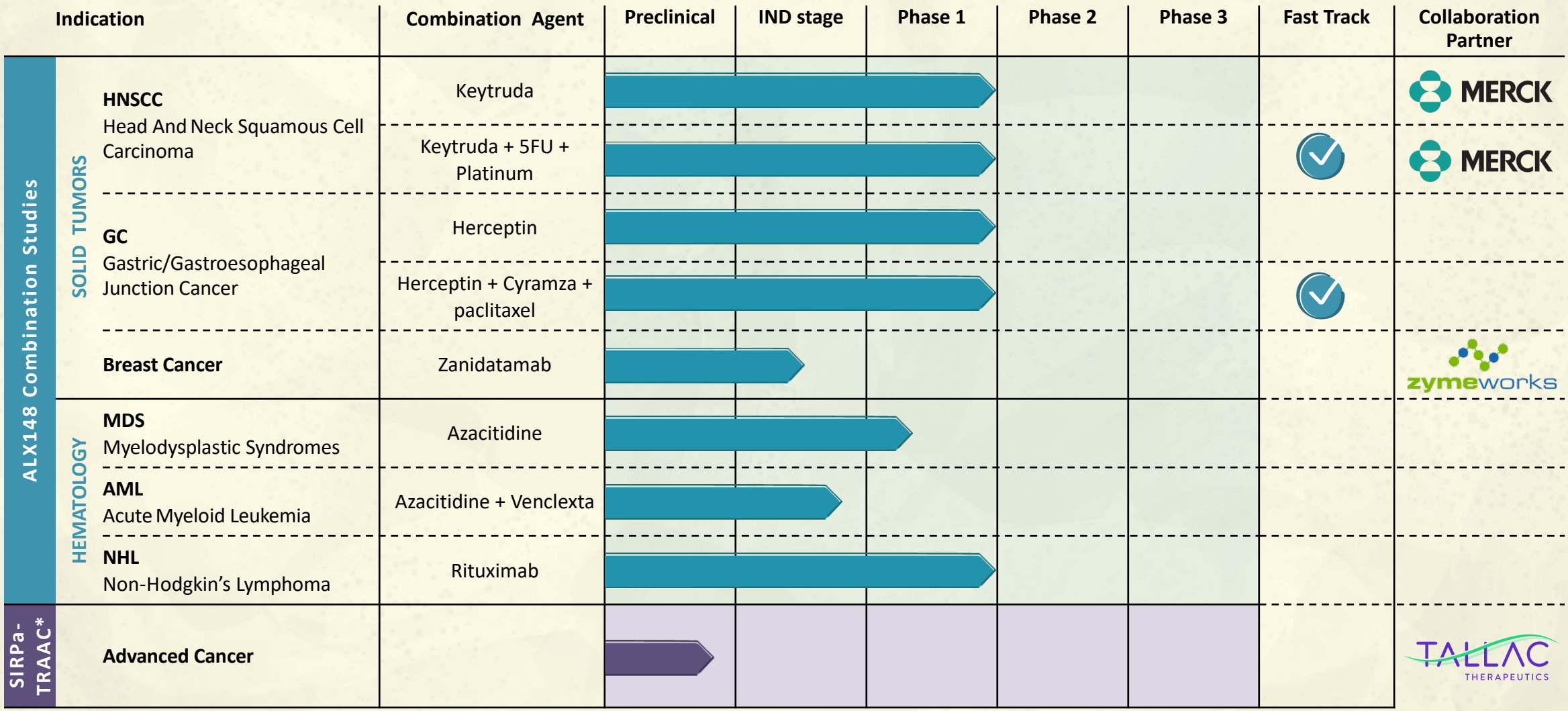
CD47 Target Occupancy by ALX148



- **Near complete CD47 target occupancy (TO) by ALX148 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

ALX ONCOLOGY

ALX PIPELINE



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

ALX ONCOLOGY

ALX148 DEMONSTRATES CONSISTENT TOLERABILITY PROFILE

Treatment related adverse events*	ALX148 + Herceptin + Cyramza + chemo (N=14)		ALX148 + Herceptin (N=30)		ALX148 + Keytruda + chemo (N=5)		ALX148 + Keytruda (N=52)		ALX148 + Rituximab (N=33)	
	Total n (%)	≥Grade 3	Total n (%)	≥Grade 3	Total n (%)	≥Grade 3	Total n (%)	≥Grade 3	Total n (%)	≥Grade 3
Fatigue	**2 (14.0%)**	-	**9 (30.0%)**	-	-	-	**6 (11.5%)**	-	**4 (12.1%)**	-
Rash	**3 (21.0%)**	-	-	-	-	-	**5 (9.6%)**	-	**8 (24.2%)**	-
AST increased	-	-	-	-	-	-	**9 (17.3%)**	-	-	-
Platelets decreased	-	-	**5 (16.7%)**	**2 (6.7%)**	-	-	**4 (7.7%)**	**2 (3.8%)**	-	-
ALT increased	-	-	-	-	-	-	**7 (13.5%)**	**1 (1.9%)**	-	-
Pruritus	**2 (14.0%)**	-	**3 (10.0%)**	-	-	-	**5 (9.6%)**	-	**2 (6.1%)**	-
Pyrexia	-	-	**3 (10.0%)**	-	-	-	**3 (5.8%)**	-	-	-
Decreased appetite	-	-	**3 (10.0%)**	-	-	-	**2 (3.8%)**	-	-	-
Anemia	-	-	**2 (6.7%)**	-	-	-	**5 (9.6%)**	**1 (1.9%)**	**2 (6.1%)**	**1 (3.0%)**
Infusion reaction	-	-	-	-	-	-	**4 (7.7%)**	-	-	-
Neutropenia / Neutrophil count decr	-	-	**2 (6.7%)**	**2 (6.7%)**	-	-	**2 (3.8%)**	**1 (1.9%)**	**2 (6.1%)**	**2 (6.1%)**
Nausea	-	-	**2 (6.7%)**	-	-	-	**2 (3.8%)**	-	**2 (6.1%)**	-
Alkaline phosphatase incr	-	-	-	-	-	-	**3 (5.8%)**	-	-	-
Arthralgia	-	-	-	-	-	-	**3 (5.8%)**	-	-	-
WBC decreased	-	-	-	-	-	-	**3 (5.8%)**	-	-	-
Myalgia	-	-	-	-	-	-	**2 (3.8%)**	-	**2 (6.1%)**	-
Diarrhea	**3 (21.0%)**	-	-	-	-	-	-	-	-	-
Urticaria	**3 (21.0%)**	-	-	-	-	-	-	-	-	-

Treatment related adverse events occurring in ≥2 subjects in all histologies at 10 & 15 mg/kg QW.

*Data cut off: April 1, 2020 for combination cohorts of ALX148 plus Keytruda and Herceptin; October 1, 2020 for combination cohorts of ALX148 plus Rituxan, Keytruda and chemotherapy (5FU, platinum) and Herceptin and chemotherapy (Cyramza, paclitaxel).

ALX ONCOLOGY

Tolerability profile enables broad combination potential

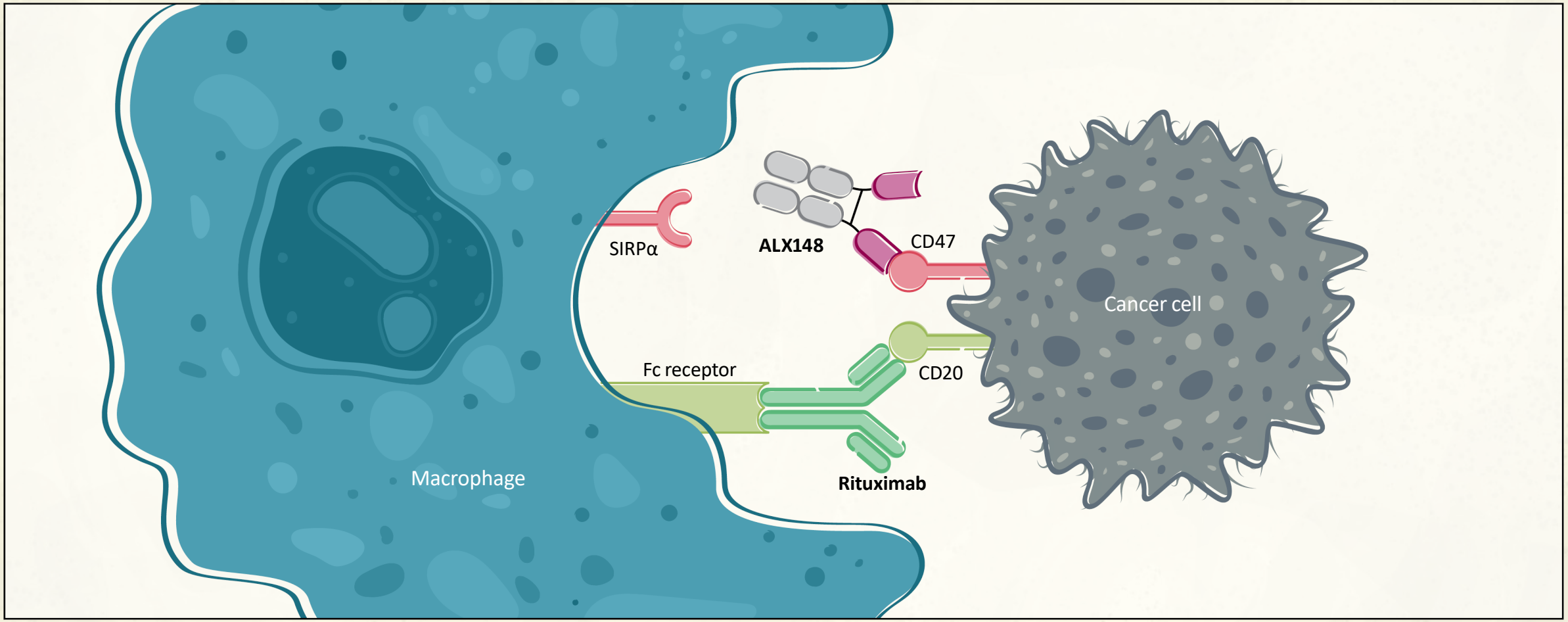
ALX148 HAS INITIAL CLINICAL ACTIVITY ACROSS TUMOR TYPES IN MULTIPLE TRIALS

Population	≥2L HER2+ GC		≥2L HER2+ GC	1L HNSCC		≥2L HNSCC (CPI-Naïve)		≥2L NHL (15mg/kg)
Combination	ALX148 + Herceptin + Cyramza + paclitaxel		ALX148 + Herceptin	ALX148 + Keytruda + 5FU + platinum		ALX148 + Keytruda		ALX148 + Rituximab
N-evaluable	14		19	4		10		10
ORR	**ALX148 64%**	Benchmark 28%	**21%**	**ALX148 75%**	Benchmark 36%	**ALX148 40%**	Benchmark 15%	**70%**
mPFS (months)	**NC**	4.4	**2.2**	**NC**	4.9	**4.6**	2.1	**NC**
mOS (months)	**NC**	9.6	**8.1**	**NC**	13.0	**22.1**	8.4	**NC**
Benchmark regimen	Cyramza + paclitaxel			Keytruda + 5FU + platinum		Single agent Keytruda		

Data as of October 1, 2020. NC = unable to be calculated, ORR = Objective Response Rate, mPFS = median progression free survival, mOS = median overall survival. CPI = checkpoint inhibitor. 2L GC benchmark, Wilke, Lancet Oncology, 2014; 2L HNSCC benchmark, Cohen, Lancet, 2018; 1L HNSCC benchmark, Burtness, Lancet, 2019.

ALX ONCOLOGY

NHL TRIAL: ALX148 + RITUXIMAB MECHANISM OF ACTION



ALX148 increases antibody dependent cellular phagocytosis in combination with Rituximab

ALX ONCOLOGY

NHL PROOF-OF-PRINCIPLE TRIAL

Phase 1b NHL cohorts

Relapsed/Refractory NHL,
prior regimen with Rituximab

Treatment:

ALX148 10 or 15 mg/kg once a week (QW)
+
Rituximab 375 mg/m^2 once a week for 4 weeks, once monthly for 8 months

		ALX148 10 mg/kg QW + Rituximab (n=22)	ALX148 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

ALX ONCOLOGY

NHL PROOF-OF-PRINCIPLE TRIAL

	10 mg/kg QW		15 mg/kg QW	
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%

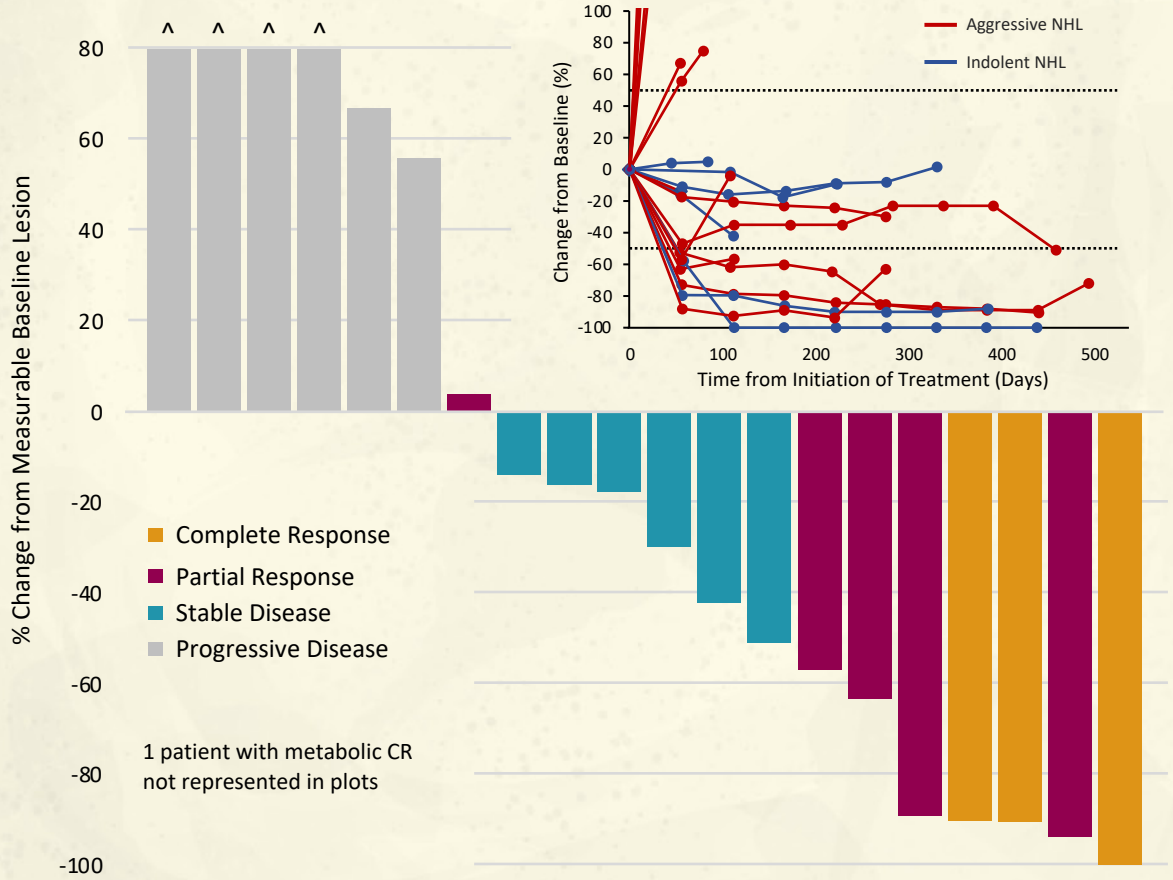
Data Cutoff: October 1, 2020; ASH 2020 Abstract 3016
N = Response Evaluable Patients
Indolent = Follicular Lymphoma and Marginal Zone Lymphoma.
Aggressive = Diffuse Large B-cell Lymphoma and Mantle Cell Lymphoma.
ORR = Objective Response Rate.

ALX148 demonstrated higher response rate at higher dosing

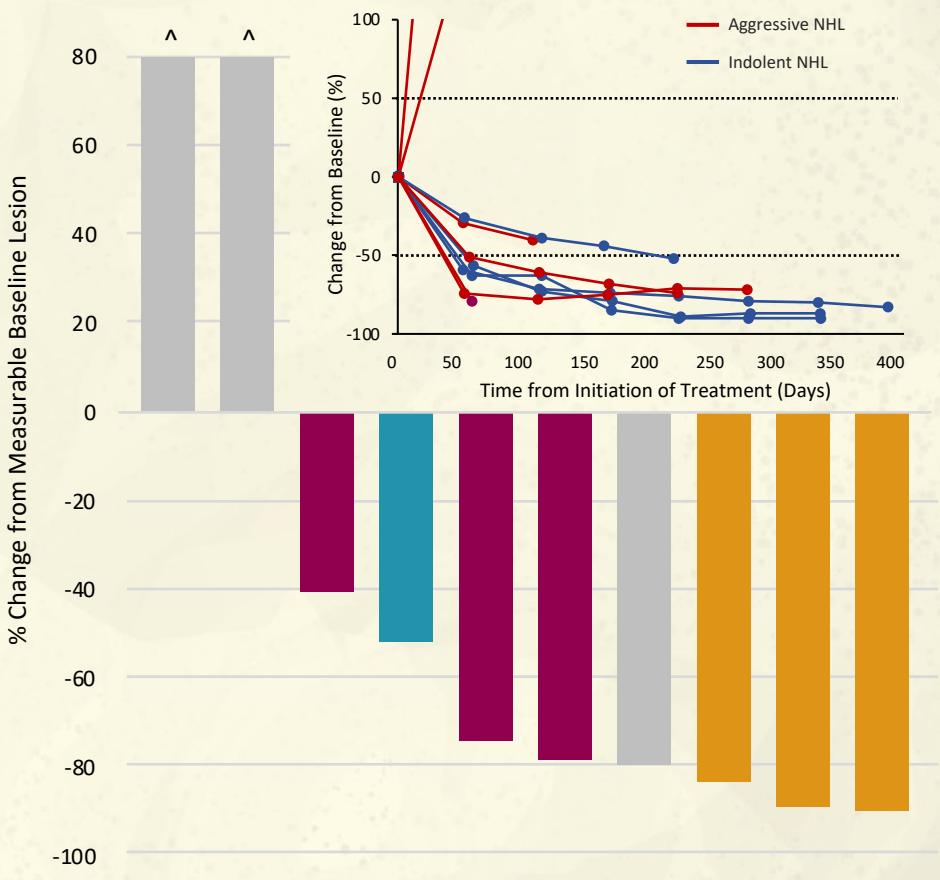
ALX ONCOLOGY

NHL: CLINICAL ACTIVITY OF ALX148 + RITUXIMAB BY PATIENT

ALX148 (10 mg/kg QW)* + Rituximab

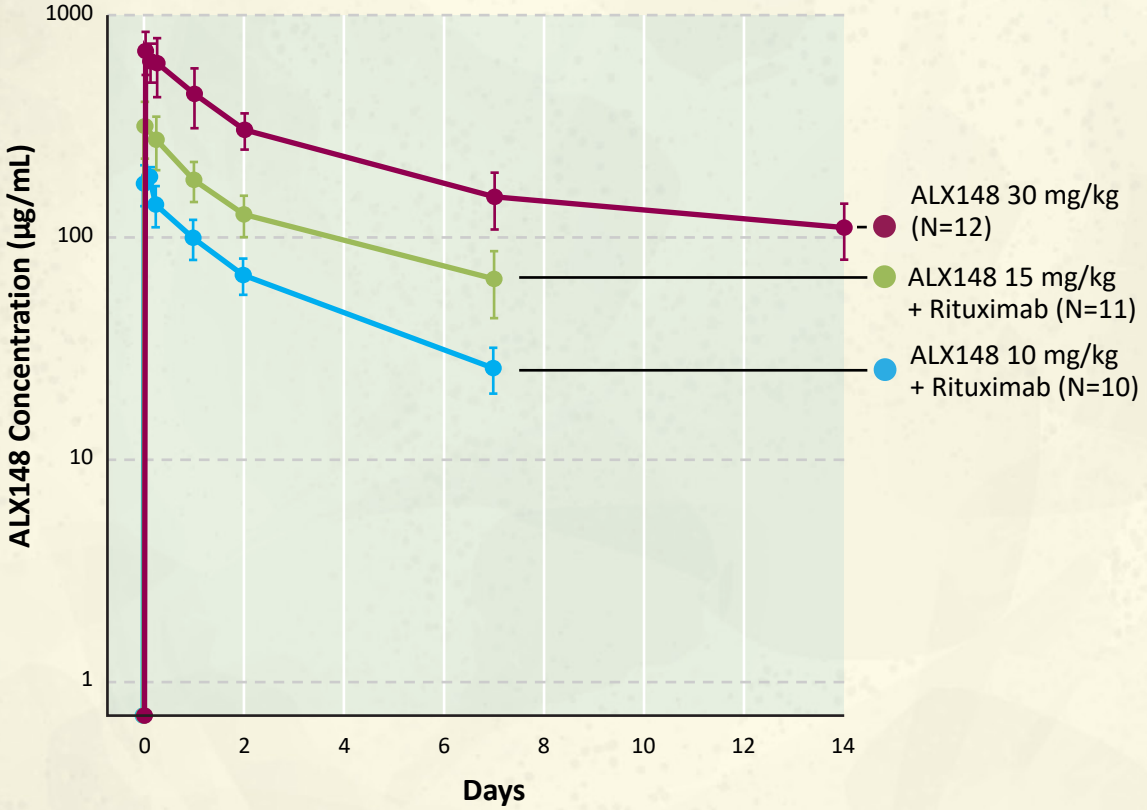


ALX148 (15 mg/kg QW) + Rituximab

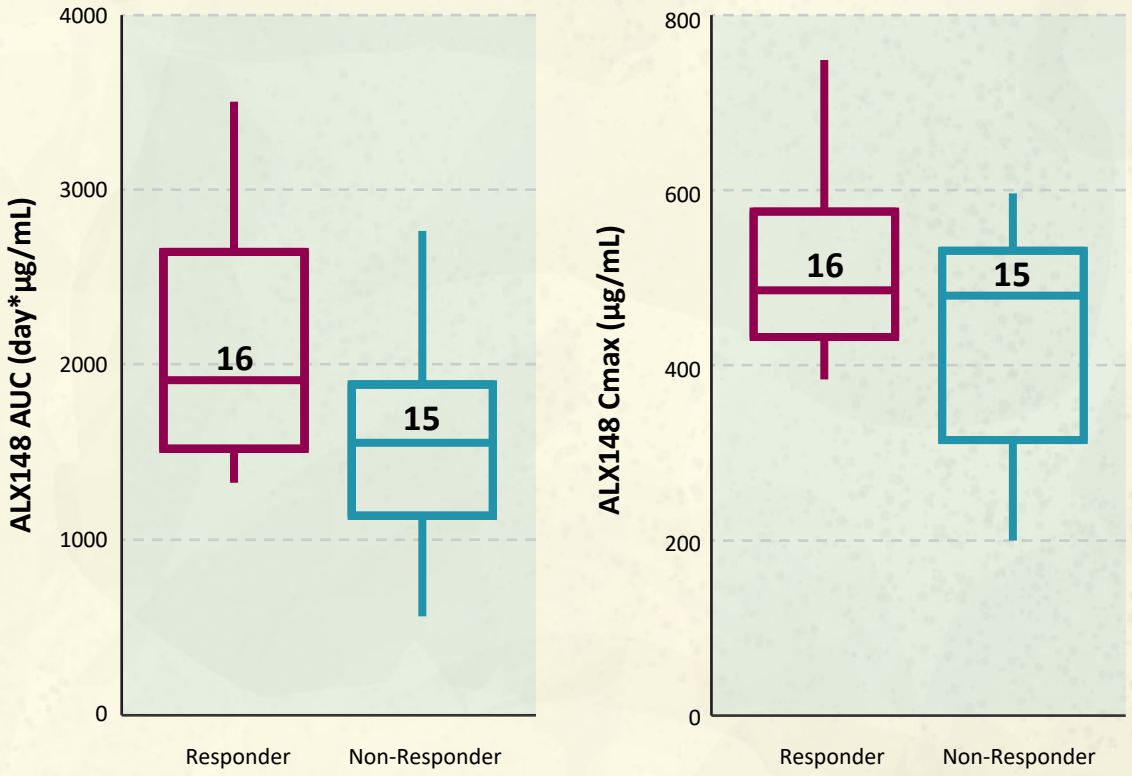


Data Cutoff October 1, 2020; Response evaluable patients; Responses include metabolic response per Lugano Response Criteria.
^ more than 80% increase from baseline. * 1 patient with rapid fatal progressive disease not represented in plot

NHL: ALX148 CLINICAL PHARMACOKINETICS AND EXPOSURE-RESPONSE ANALYSIS



ALX148 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 ALX148 exposure (AUC; p = 0.023) was observed across the exposure range evaluated (10 mg/kg QW - 15 mg/kg QW).

ALX ONCOLOGY

Data Cutoff October 1, 2020

NON-HODGKIN LYMPHOMA PROOF-OF-PRINCIPLE SUMMARY

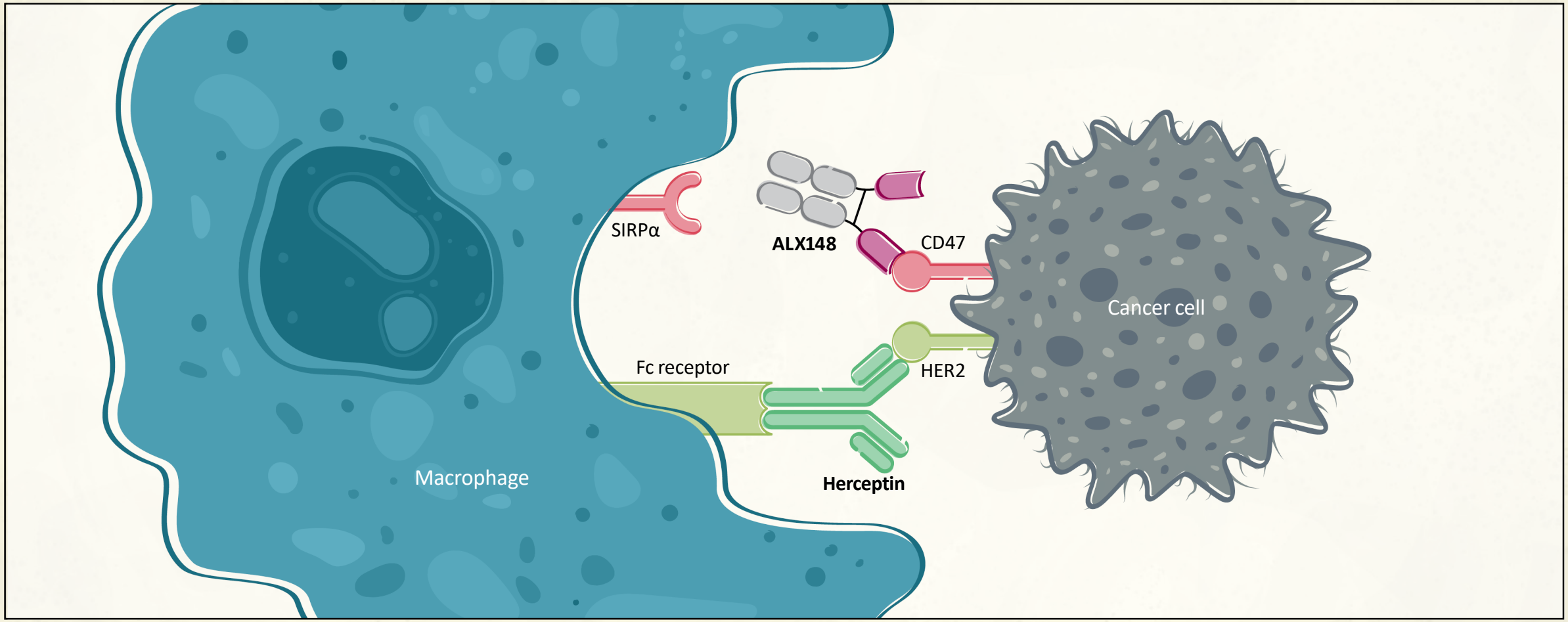
Other agents in CD47 class reduced dosing leading to reduced responses

Higher dosing enabled by ALX148 tolerability profile

Higher dosing of ALX148 led to higher responses

ALX ONCOLOGY

GC TRIAL: ALX148 + HERCEPTIN MECHANISM OF ACTION



ALX148 increases antibody dependent cellular phagocytosis in combination with Herceptin

ALX ONCOLOGY

PHASE 1B GC TRIALS: PATIENT BASELINE CHARACTERISTICS

		ALX148 + Herceptin ≥2L GC (N=20)	ALX148 + Herceptin + Cyramza/chemo ≥2L GC (N=14)
Median age, years (range)		**58 (45-79)**	**63 (36-83)**
Sex, n	M	**15**	**10**
	F	**5**	**4**
Race, n	Asian	**13**	**11**
	White	**6**	**3**
	Other	**1**	**-**
ECOG PS, n	0	**7**	**5**
	1	**13**	**9**
Progressed upon prior anti-HER2 therapy, n (%)		**19 (95)**	**13 (93)**
Progressed upon ≥2 prior anti-HER2 therapy n (%)		**9 (45)**	**1 (7.1)**
Progressed upon prior CPI therapy, n (%)		**9 (45)**	**1 (7.1)**
Visceral distant metastasis, n (%)		**17 (85)**	**13 (93)**

ALX ONCOLOGY

PHASE 1B ≥2 LINE GC TRIAL: ALX148 + HERCEPTIN

Phase 1b GC trial:

Response evaluable patients

N=19 HER2 positive GC

Progressed on prior fluoropyrimidine, Herceptin or platinum.

Treatment:

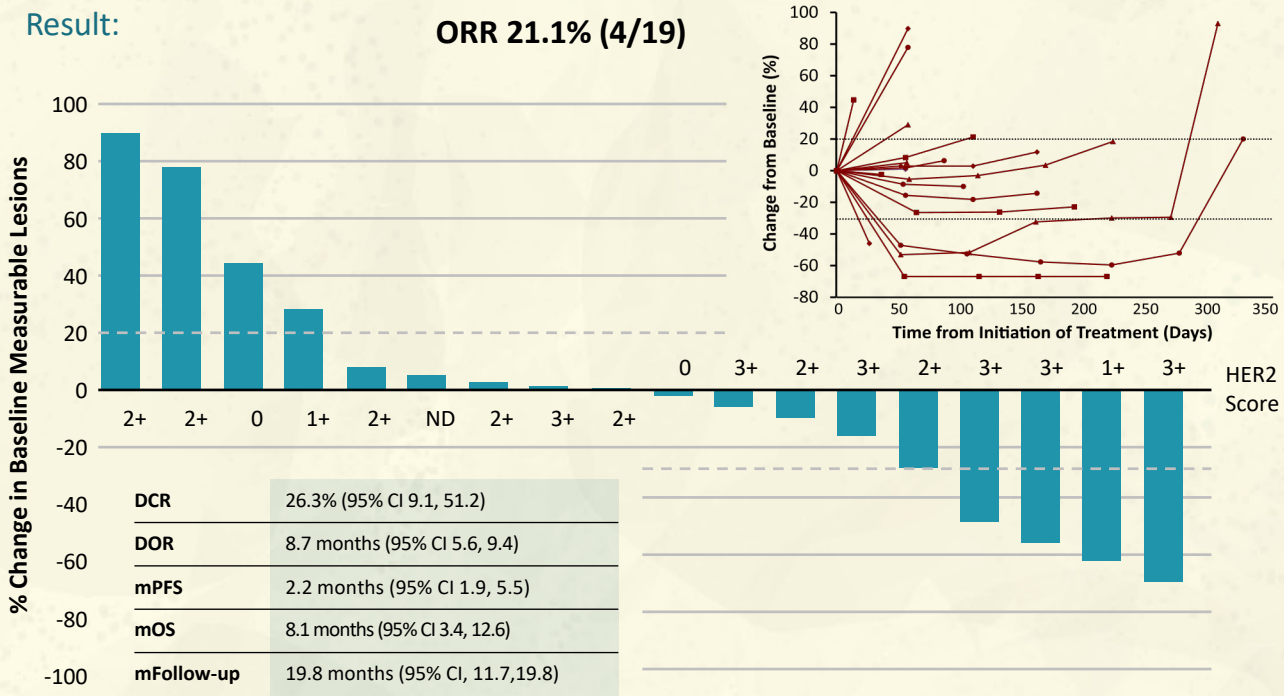
ALX148 10 mg/kg once a week (QW)
+ **Herceptin**
8 mg/kg once, then
6 mg/kg every three weeks (Q3W)

Endpoints:

- maximum tolerated dose
- anti-cancer activity

Result:

ORR 21.1% (4/19)



DCR	26.3% (95% CI 9.1, 51.2)
DOR	8.7 months (95% CI 5.6, 9.4)
mPFS	2.2 months (95% CI 1.9, 5.5)
mOS	8.1 months (95% CI 3.4, 12.6)
mFollow-up	19.8 months (95% CI, 11.7,19.8)

Notes: Data Cutoff October 1, 2020. Patients who received at least one dose of ALX148 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 ALX148 fast track designation for second-line treatment of HER2 positive GC

ALX ONCOLOGY

PHASE 1B ≥2 LINE GC TRIAL: ALX148 + 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.

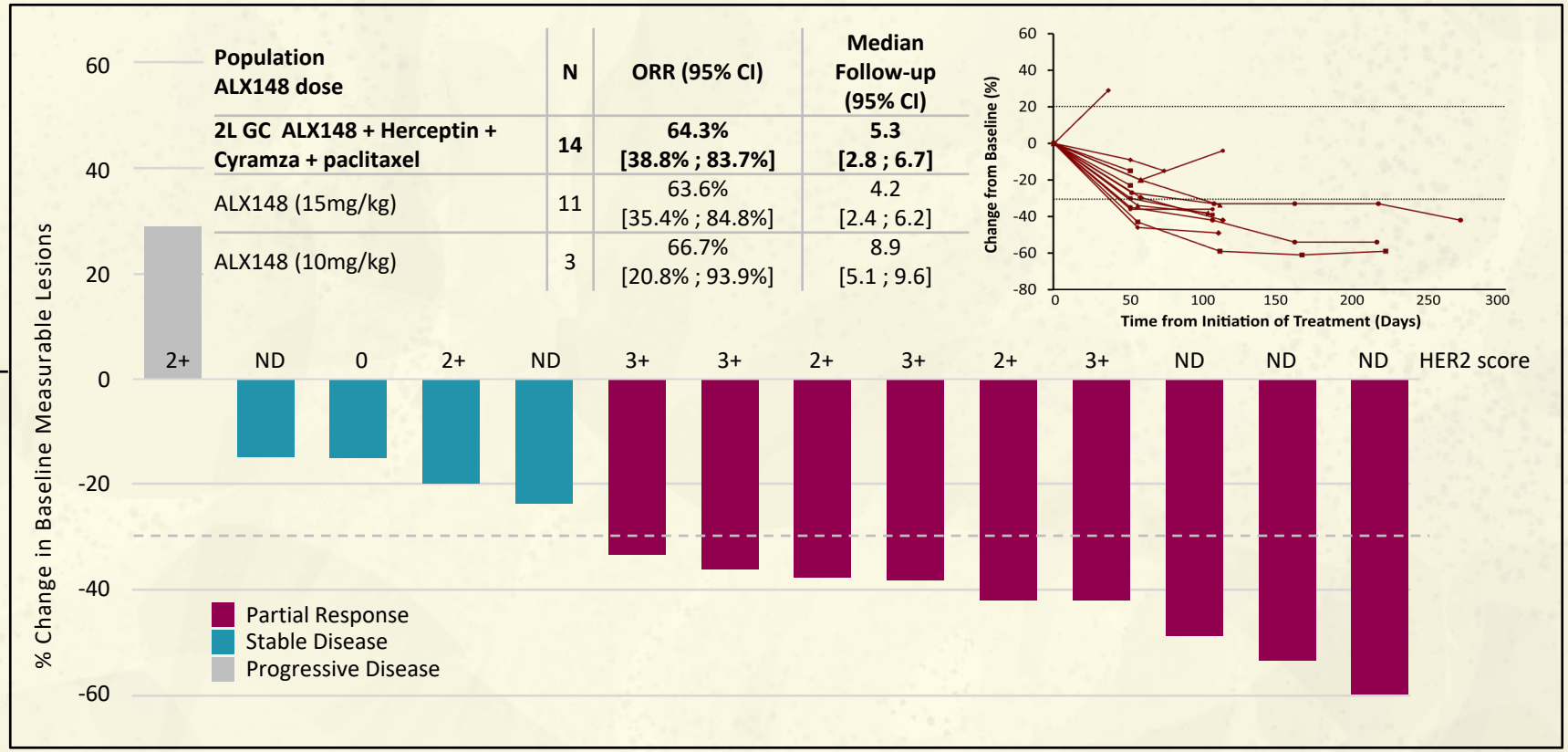
Treatment:

ALX148 10 and 15 mg/kg (QW)
+ **Herceptin**
+ **Cyramza**
+ **Paclitaxel**

Endpoint:

- safety of combination
- anti-cancer activity

Population ALX148 dose	N	ORR (95% CI)	Median Follow-up (95% CI)
2L GC ALX148 + Herceptin + Cyramza + paclitaxel	**14**	**64.3% [38.8% ; 83.7%]**	**5.3 [2.8 ; 6.7]**
ALX148 (15mg/kg)	11	63.6% [35.4% ; 84.8%]	4.2 [2.4 ; 6.2]
ALX148 (10mg/kg)	3	66.7% [20.8% ; 93.9%]	8.9 [5.1 ; 9.6]



Data Cutoff October 1, 2020. ND = Not Done

SECOND LINE GASTRIC CANCER: PLANNED RANDOMIZED PHASE 2 CLINICAL TRIAL

Randomized Planned Phase 2:

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

ALX148 Treatment Arm

ALX148

+ Herceptin

+ Cyramza

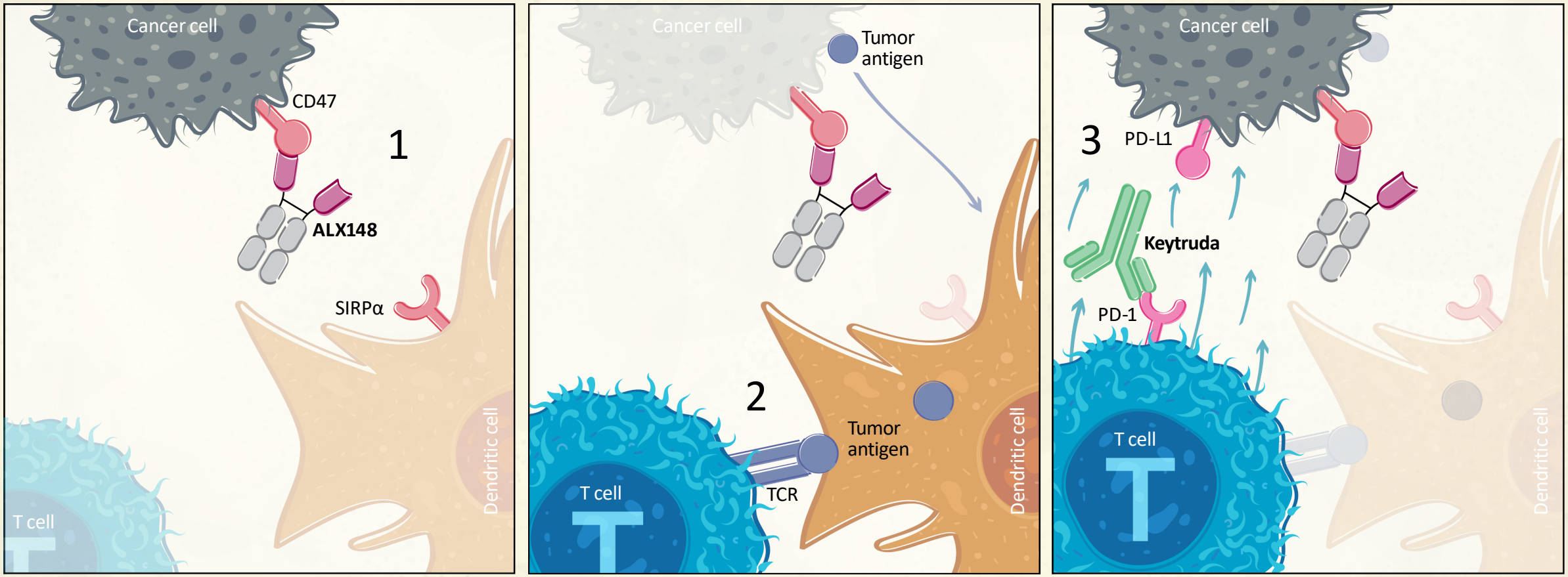
+ Paclitaxel

Endpoint:

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

ALX ONCOLOGY

HNSCC TRIAL: ALX148 + KEYTRUDA MECHANISM OF ACTION



ALX148 activates dendritic cells and enhances cross-priming of T cells

ALX ONCOLOGY

HNSCC STANDARD OF CARE & OPPORTUNITY

		ORR	mPFS (months)	mOS (months)	≥Gr3 TRAEs
1L	**Keytruda** + chemo[1] (KEYNOTE 048)	**36%**	**4.9**	**13.0**	**72%[2]**
	Keytruda monotherapy (KEYNOTE 048)	**17%**	**2.3**	**11.5**	**17%**
2L	**Keytruda monotherapy** (KEYNOTE 040)	**15%**	**2.1**	**8.4**	**13%**

- Keytruda monotherapy ORR of 15% in ≥2L CPI naïve HNSCC
- Significant unmet need
- Increasing use of Keytruda monotherapy[3]
- Keytruda 2020 WW Sales $14.4B[4]

[1]5FU + cisplatin or carboplatin.
[2]83% occurrence in chemo control arm.
[3]Wiley 2019, Real-world treatment patterns for patients with metastatic head and neck squamous cell carcinoma treated with immuno-oncology therapy.
[4]Merck 10-K February 25, 2021

ALX ONCOLOGY

HNSCC TRIALS: PATIENT BASELINE CHARACTERISTICS

		ALX148 + Keytruda ≥2L HNSCC (N=20)	ALX148 + Keytruda + 5FU/platinum 1L HNSCC (N=5)
Median age, years (range)		**62.5 (35-81)**	**61 (45-63)**
Sex, n	M	**15**	**4**
	F	**5**	**1**
Race, n	Asian	**6**	**4**
	White	**12**	**1**
	Other	**2**	**-**
ECOG PS, n	0	**7**	**4**
	1	**13**	**1**
Progressed upon prior CPI therapy, n (%)		**10 (50)**	**0 (0)**
Visceral distant metastasis, n (%)		**12 (60)**	**1 (20)**

ALX ONCOLOGY

PHASE 1B ≥2 LINE HNSCC TRIAL: ALX148 + KEYTRUDA

Phase 1b ≥2L HNSCC trial:

Response evaluable patients

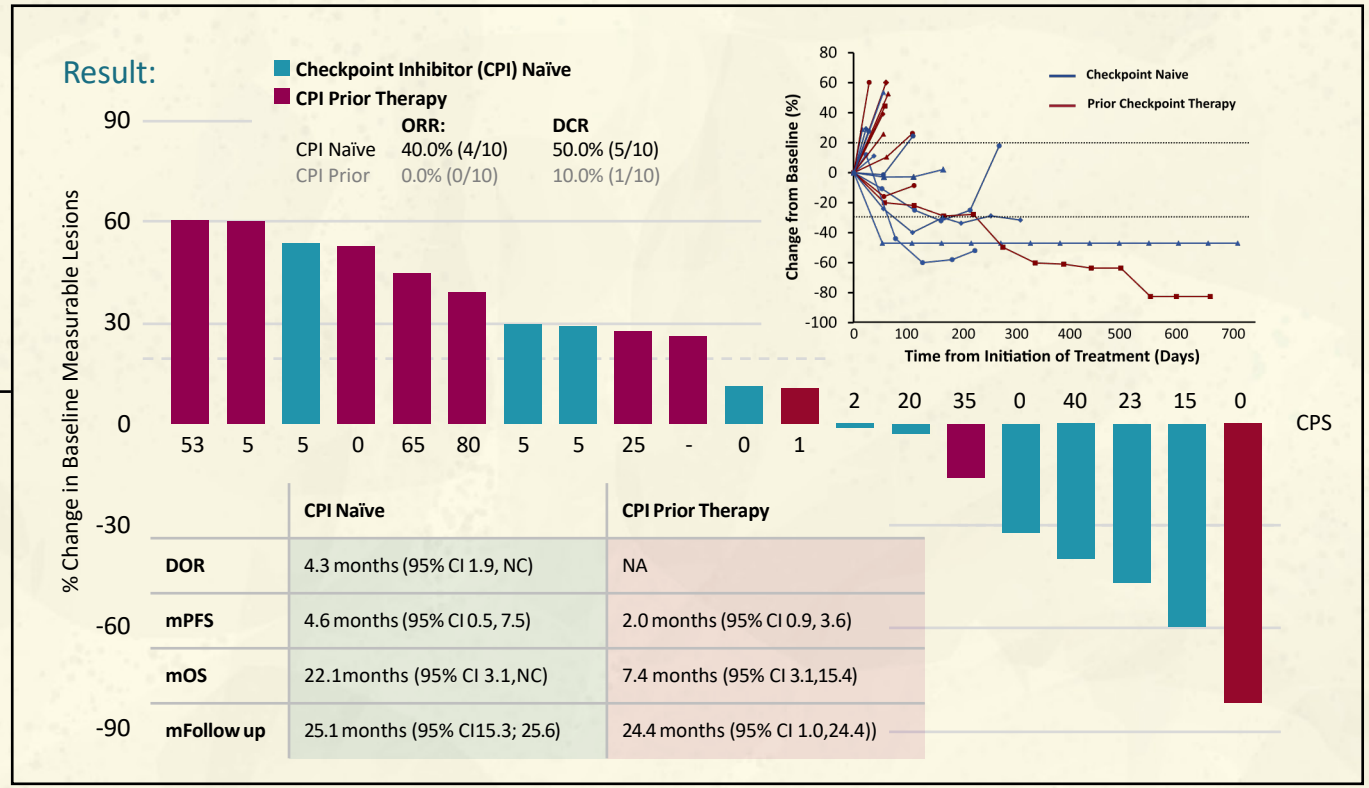
N=20: recurrent/metastatic HNSCC, at least one prior systemic therapy

Treatment:

ALX148 10 mg/kg once a week (QW)
+
Keytruda
200 mg every three weeks (Q3W)

Endpoints:

- maximum tolerated dose
- anti-cancer activity



	CPI Naïve	CPI Prior Therapy
DOR	4.3 months (95% CI 1.9, NC)	NA
mPFS	4.6 months (95% CI 0.5, 7.5)	2.0 months (95% CI 0.9, 3.6)
mOS	22.1months (95% CI 3.1,NC)	7.4 months (95% CI 3.1,15.4)
mFollow up	25.1 months (95% CI15.3; 25.6)	24.4 months (95% CI 1.0,24.4))

Notes: Data Cutoff October 1, 2020. Patients who received at least one dose of ALX148 in the expansion phase, had a baseline assessment, and at least one post-baseline disease assessment.

ORR = Overall Response Rate. **DCR** = Disease Control Rate. **CPS =** Combined Positive Score.

FDA granted ALX148 Fast Track designation for first-line treatment of patients with HNSCC

PHASE 1B HNSCC: ALX148 + KEYTRUDA + 5FU/PLATINUM FIRST LINE CHECKPOINT NAIVE

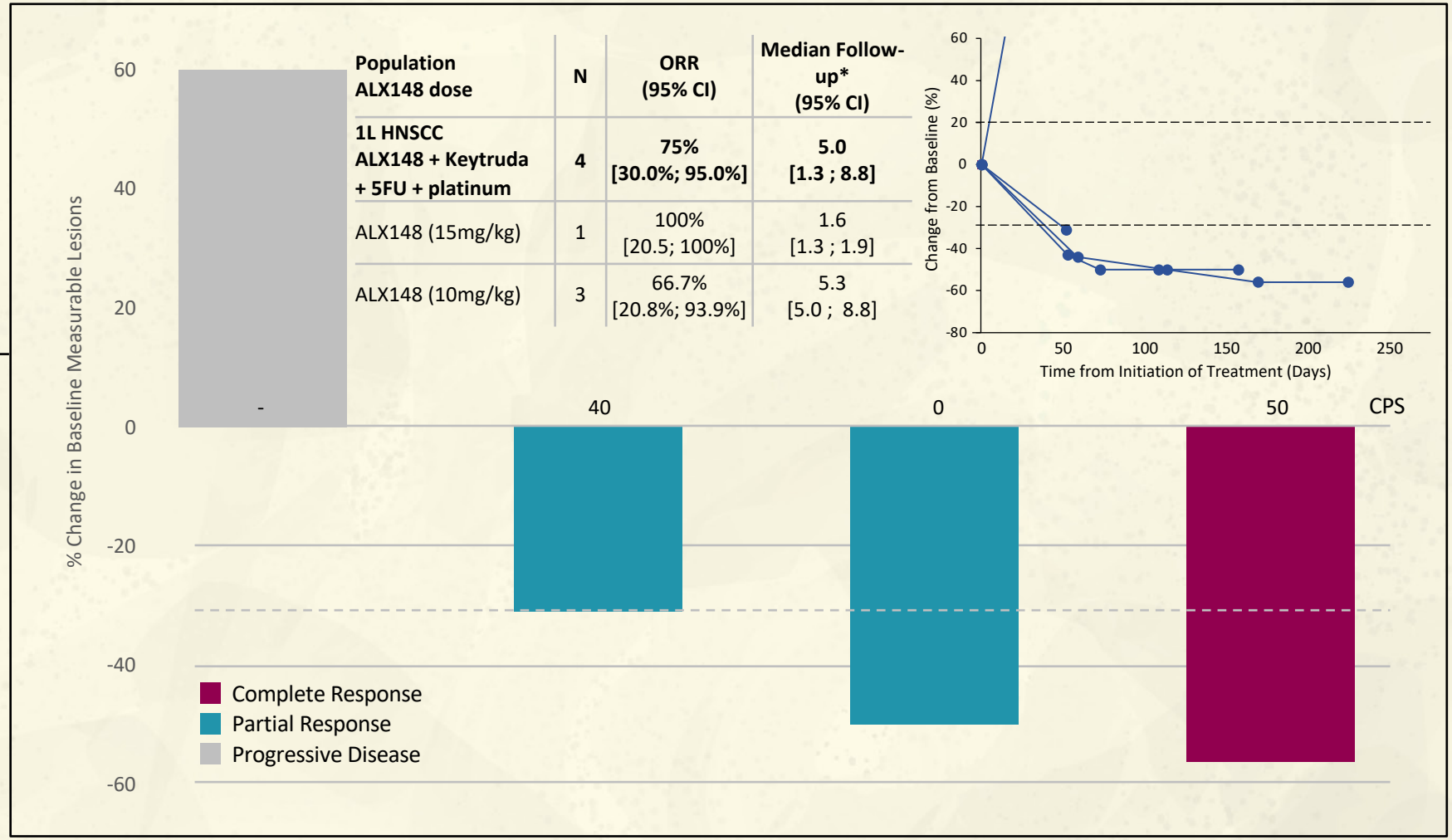
Phase 1b ≥1L HNSCC dose confirmation:

Treatment:

ALX148 10 & 15 mg/kg (QW)
+ Keytruda
+ 5FU
+ Cisplatin or carboplatin

No prior treatment for advanced disease

Population ALX148 dose	N	ORR (95% CI)	Median Follow-up* (95% CI)
1L HNSCC ALX148 + Keytruda + 5FU + platinum	**4**	**75% [30.0%; 95.0%]**	**5.0 [1.3 ; 8.8]**
ALX148 (15mg/kg)	1	100% [20.5; 100%]	1.6 [1.3 ; 1.9]
ALX148 (10mg/kg)	3	66.7% [20.8%; 93.9%]	5.3 [5.0 ; 8.8]



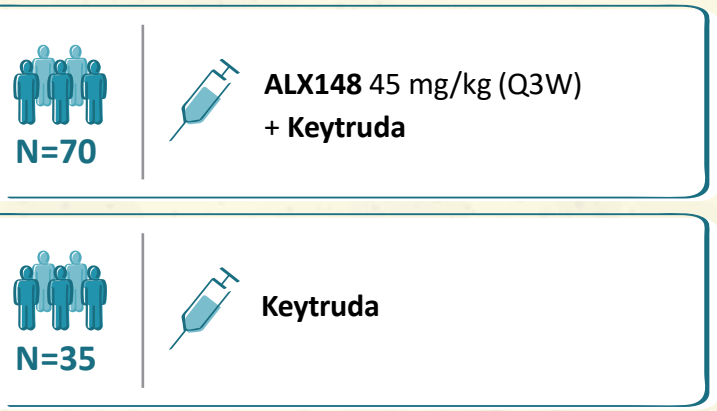
Data Cutoff October 1, 2020
* Intent to treat population: HNSCC (10 + 15 mg/kg) N=5; HNSCC (15 mg/kg) N=2

FIRST LINE HEAD & NECK CANCER: PHASE 2 DEVELOPMENT PLAN

ALX148
+
Keytruda

Phase 2 trial:

2:1



(Safety lead-in prior to randomization)

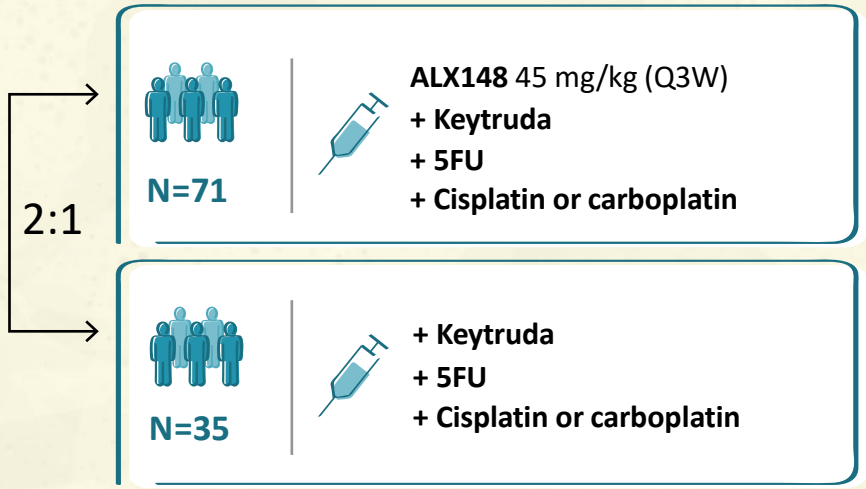
Endpoint:

- ORR (from benchmark of 17% to goal of 33%)

ALX148
+
Keytruda
+
Chemo

Phase 2 trial:

2:1



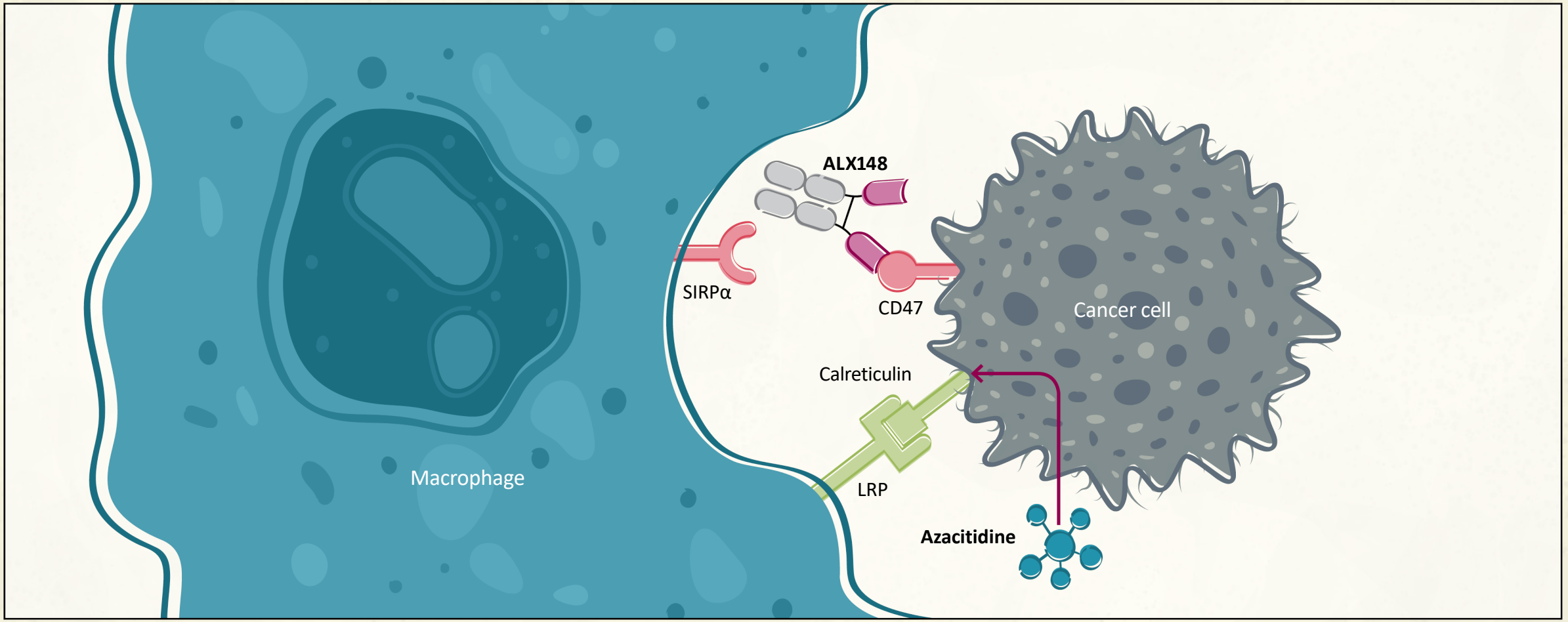
(Safety lead-in prior to randomization)

Endpoint:

- ORR (from benchmark of 36% to goal of 54%)

ALX ONCOLOGY

MDS TRIAL: ALX148 + AZACITIDINE MECHANISM OF ACTION



ALX148 increases pro-phagocytic signal provided by azacitidine

ALX ONCOLOGY

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

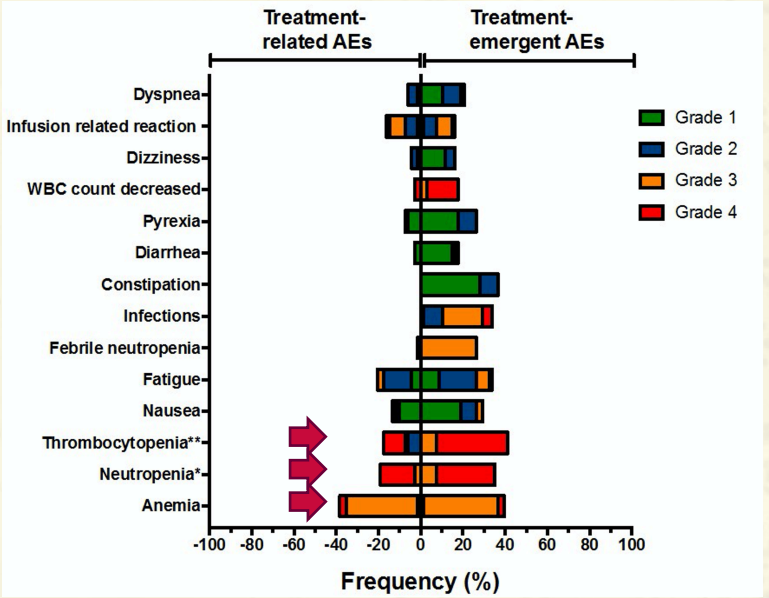
Magrolimab with azacitidine

Sallman, ASCO 2020

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

Sallman, ASCO 2019



All grade TRAEs: **38% Anemia**
19% Neutropenia
18% Thrombocytopenia

Sallman, ASCO 2020

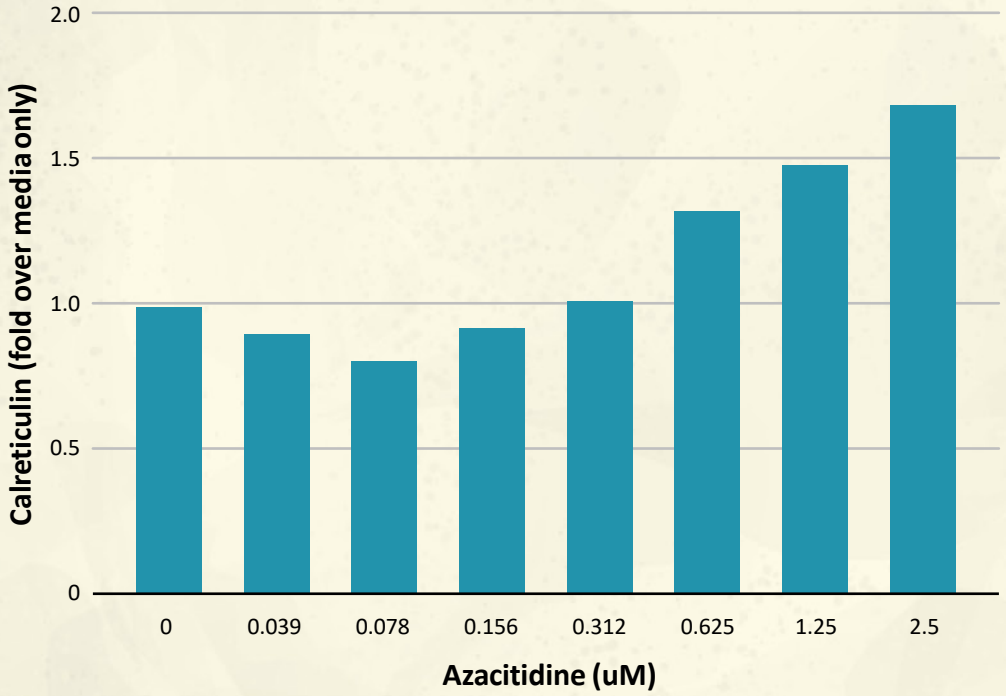
CD47 blocker 5F9 (magrolimab) shows complete responses only when paired with azacitidine, and causes frequent incidence of treatment-related, high-grade cytopenia

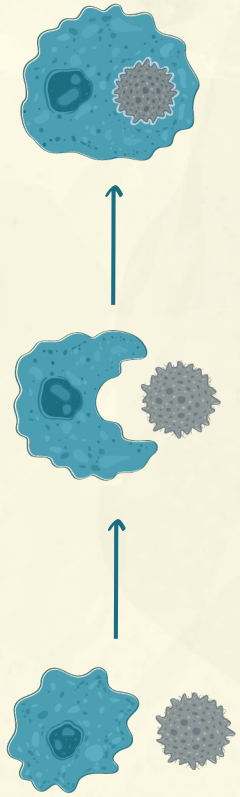
ALX ONCOLOGY

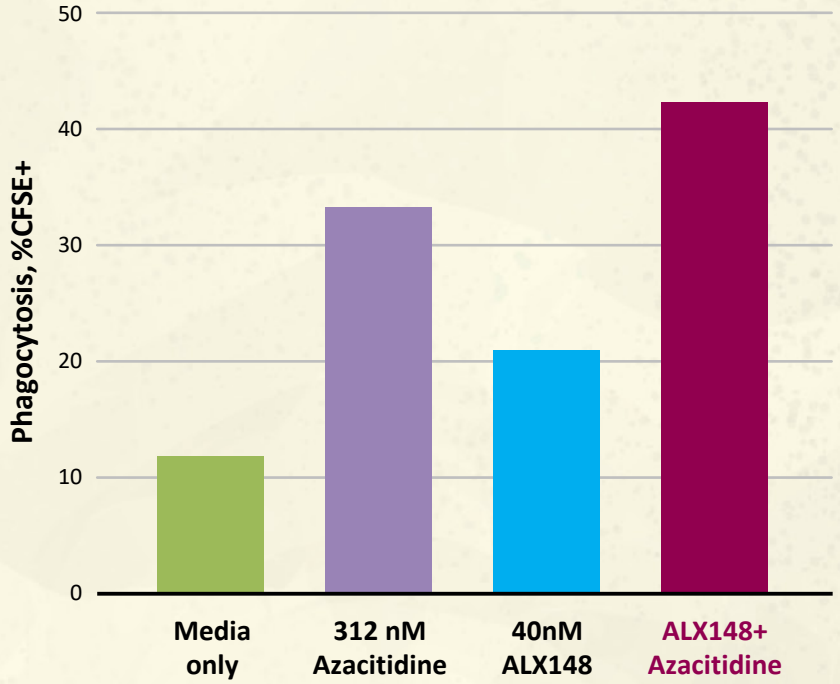
ORR = overall response rate.
CR = complete response rate.
CRi = complete remission with incomplete hematological recovery

MLFS = morphologic leukemia free state
SD = stable disease
PD = progressive disease

PRECLINICAL: ALX148 INCREASES PHAGOCYTOSIS OF BACKBONE MDS DRUG, AZACITIDINE



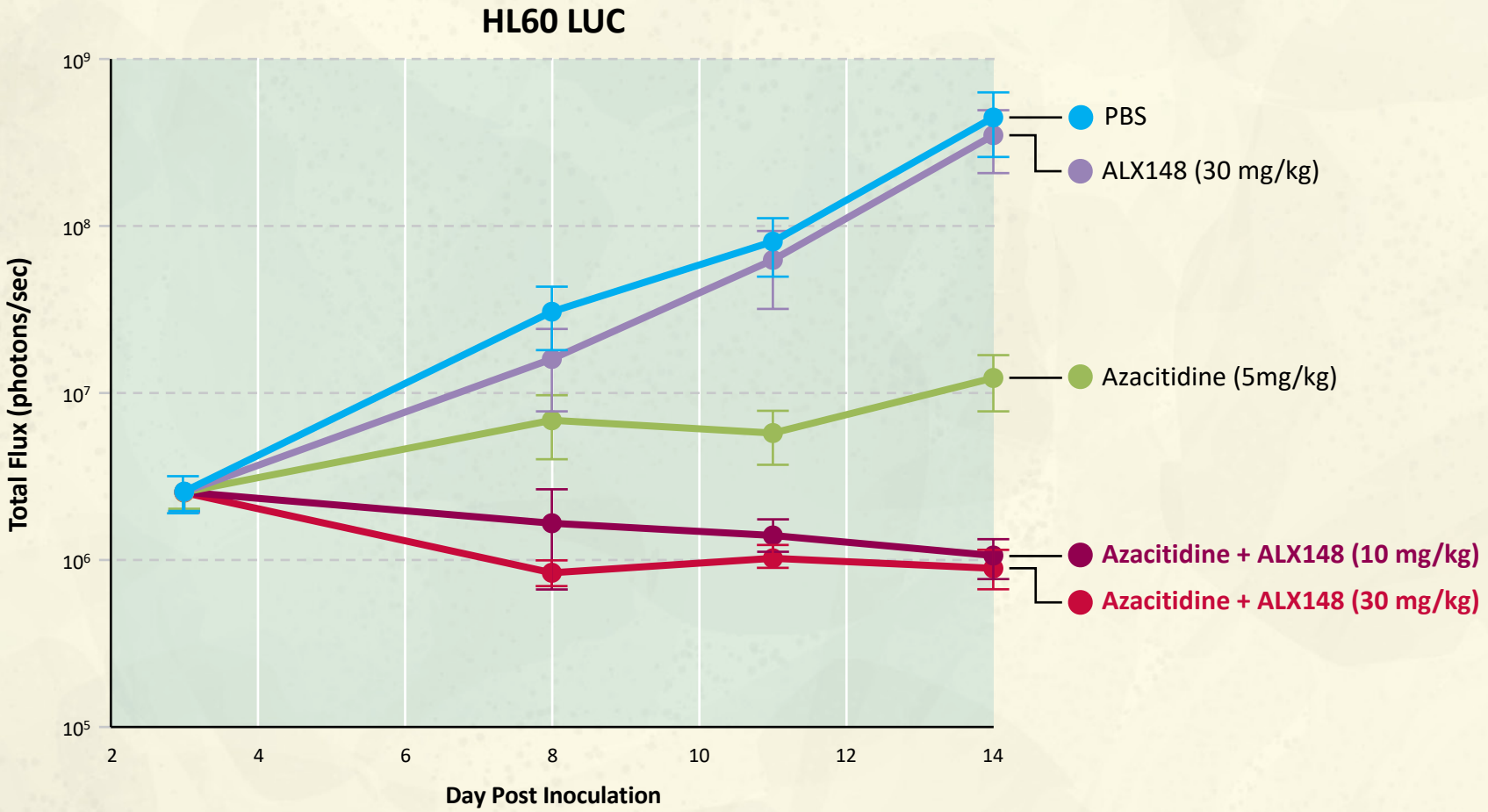




Azacitidine induces calreticulin display.
ALX148 increases phagocytosis in combination with azacitidine.

ALX ONCOLOGY

ALX148 INCREASES TUMOR INHIBITION OF AZACITIDINE

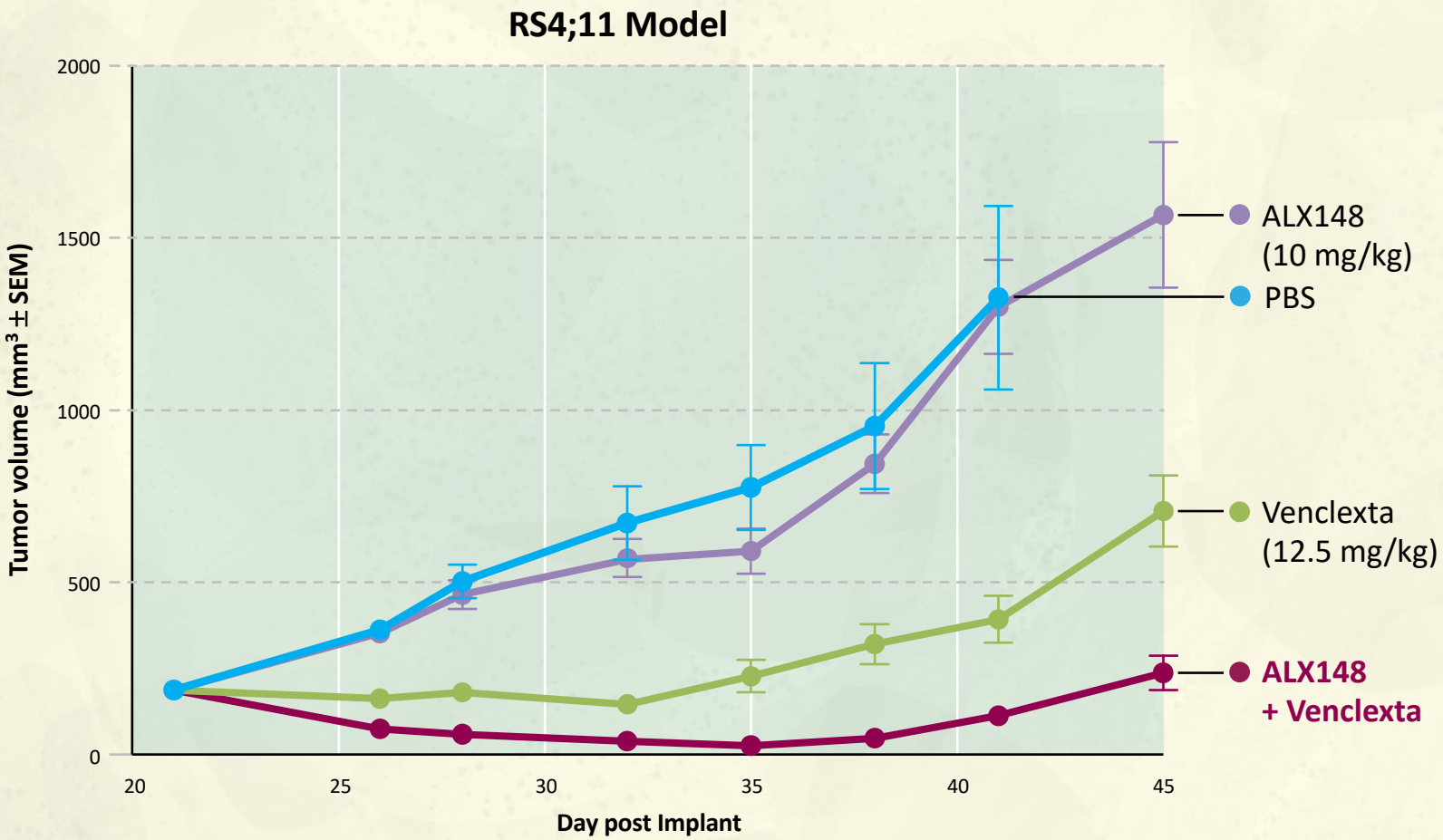


Disseminated AML mouse model

Combination opportunity in MDS and AML

ALX ONCOLOGY

ALX148 INCREASES TUMOR INHIBITION OF VENCLEXTA



Combination opportunity in AML

ALX ONCOLOGY

MDS TRIAL PLANS

Phase 1 trial – Open for Accrual

Patients:

N=~24

R/R and treatment naïve

IPSS-R intermediate,

high, very high risk MDS

Treatment:

ALX148
20 mg/kg (Q2W)
30 mg/kg (Q2W)
or 60 mg/kg (Q4W)
+
Azacitidine
75 mg/m^2 daily for 7 days
of 28 day cycle

Endpoint:

- safety of combination

Phase 2 Randomized Trial

Patients:

Treatment naïve

IPSS-R intermediate, high, very

high risk MDS

Treatment:

ALX148
Recommended phase 2 dose
+
Azacitidine

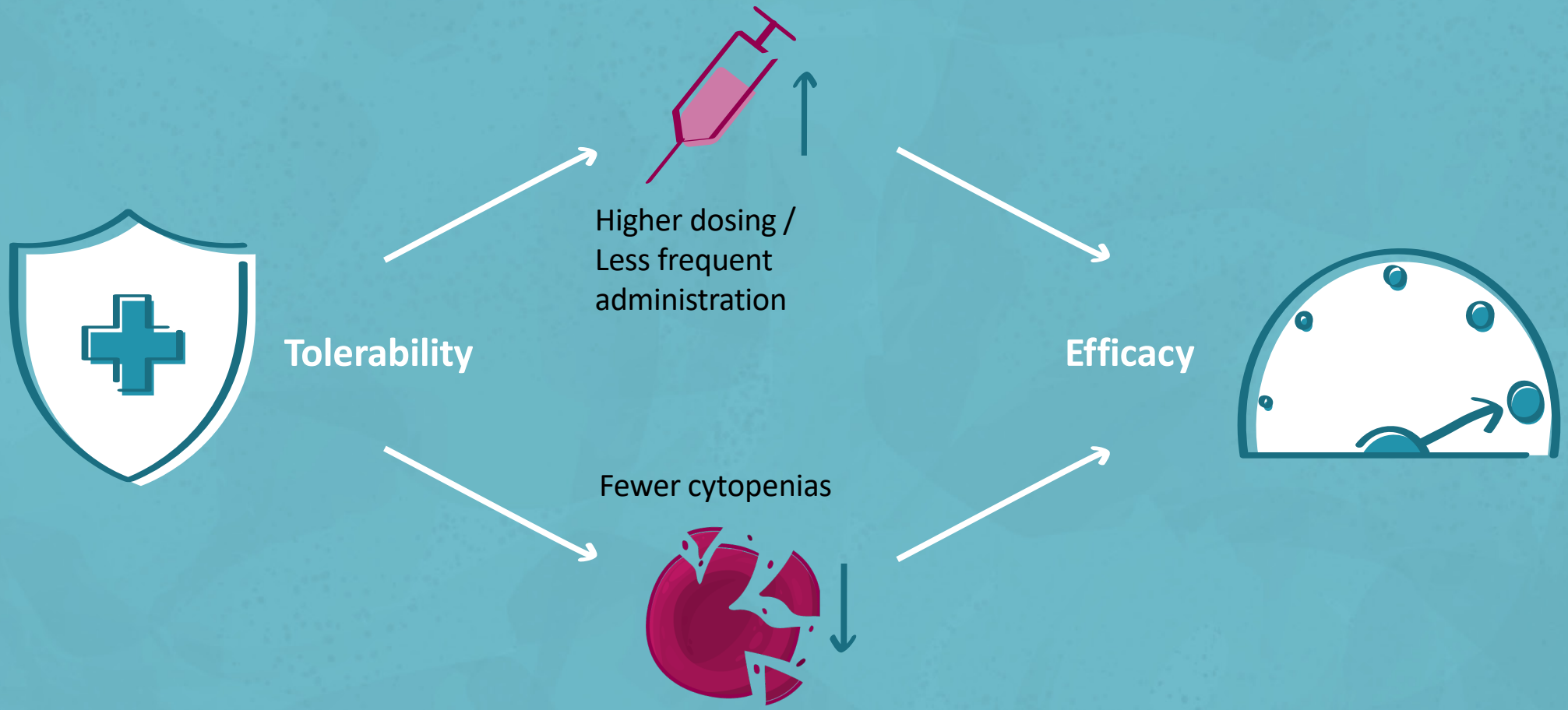
vs.
Azacitidine

Endpoint:

- complete response rate (CRR)
(from benchmark of 17% to goal of 35%)

ALX ONCOLOGY

ALX148 DIFFERENTIATION POTENTIAL ENABLED BY TOLERABILITY



ALX148 SUMMARY

ALX148 tolerability profile enables combination with range of agents

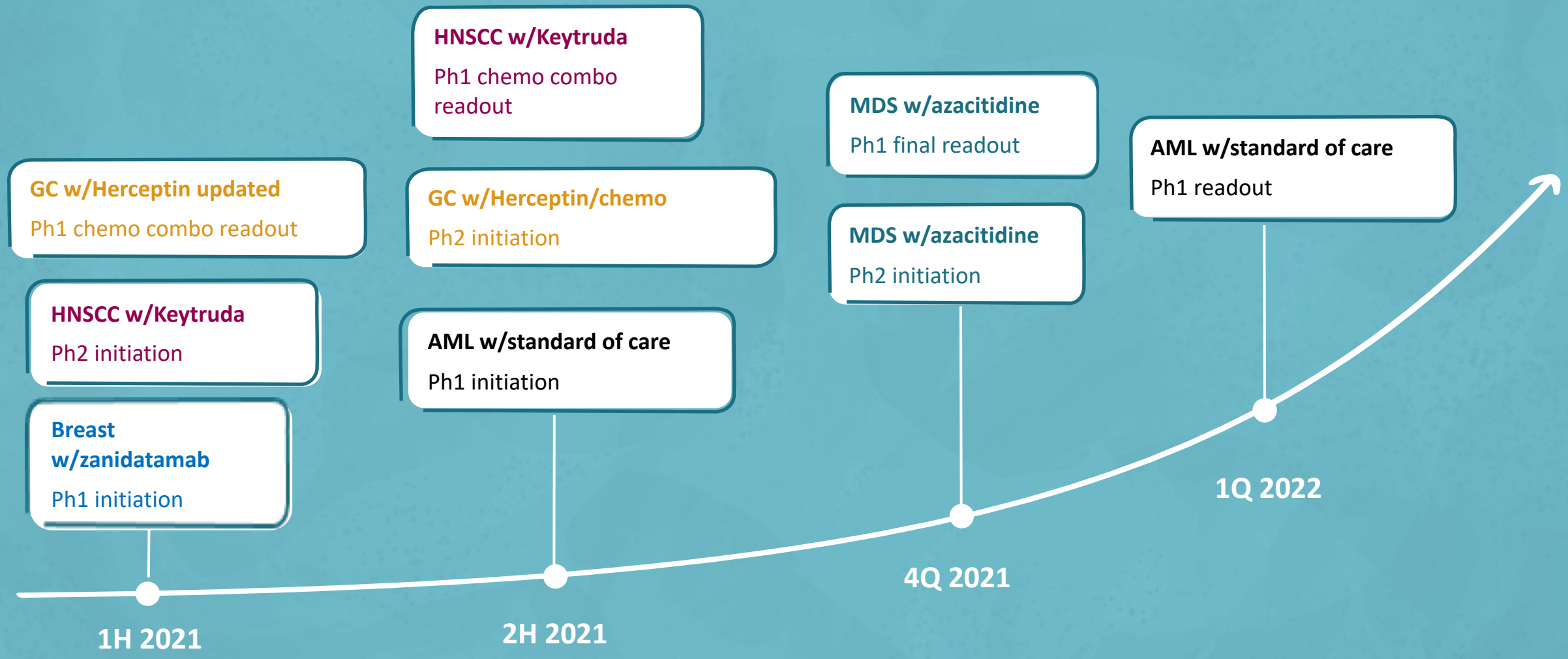
ALX148 Higher dosing and smaller molecular weight facilitate tumor penetration for greater efficacy

Clinical proof-of-principle in hematologic and solid tumors

ALX148 is the only CD47 blocker to show encouraging response data in solid tumor indications

ALX ONCOLOGY

ALX148 DEVELOPMENT PROGRESS AND FUTURE PLANS



ALX ONCOLOGY

EARLY STAGE PIPELINE:
SIRPα-TRAAC COLLABORATION

ALX ONCOLOGY

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

Provides SIRPα antibody

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

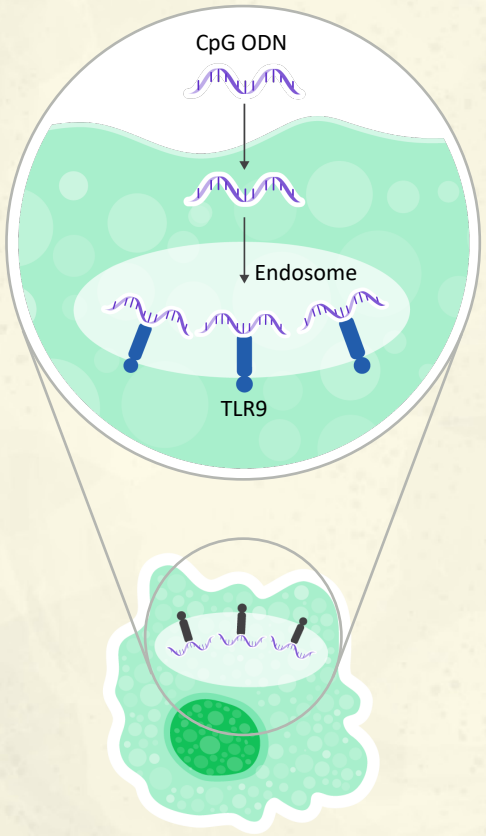
SIRPα TRAAC induces targeted immune activation, bridging innate and adaptive immune responses.

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

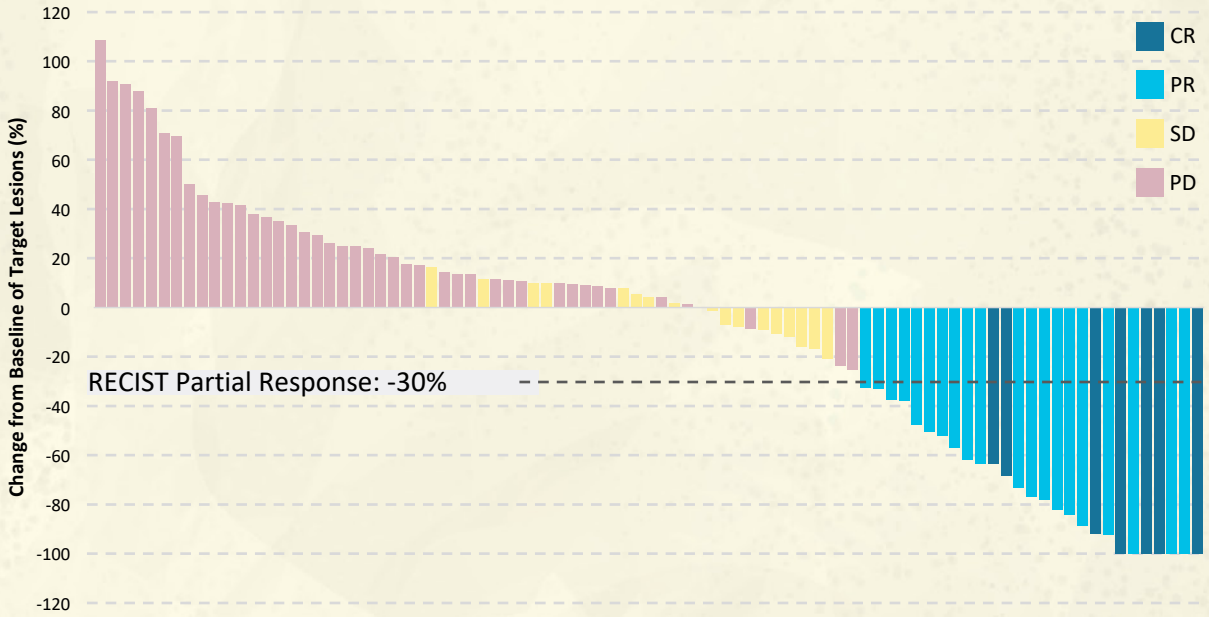
TLR9 stimulation by CpG ODN leads to immune cell activation



CpG ODN uptake

Immune activation

Intratumoral programs have demonstrated clinical activity



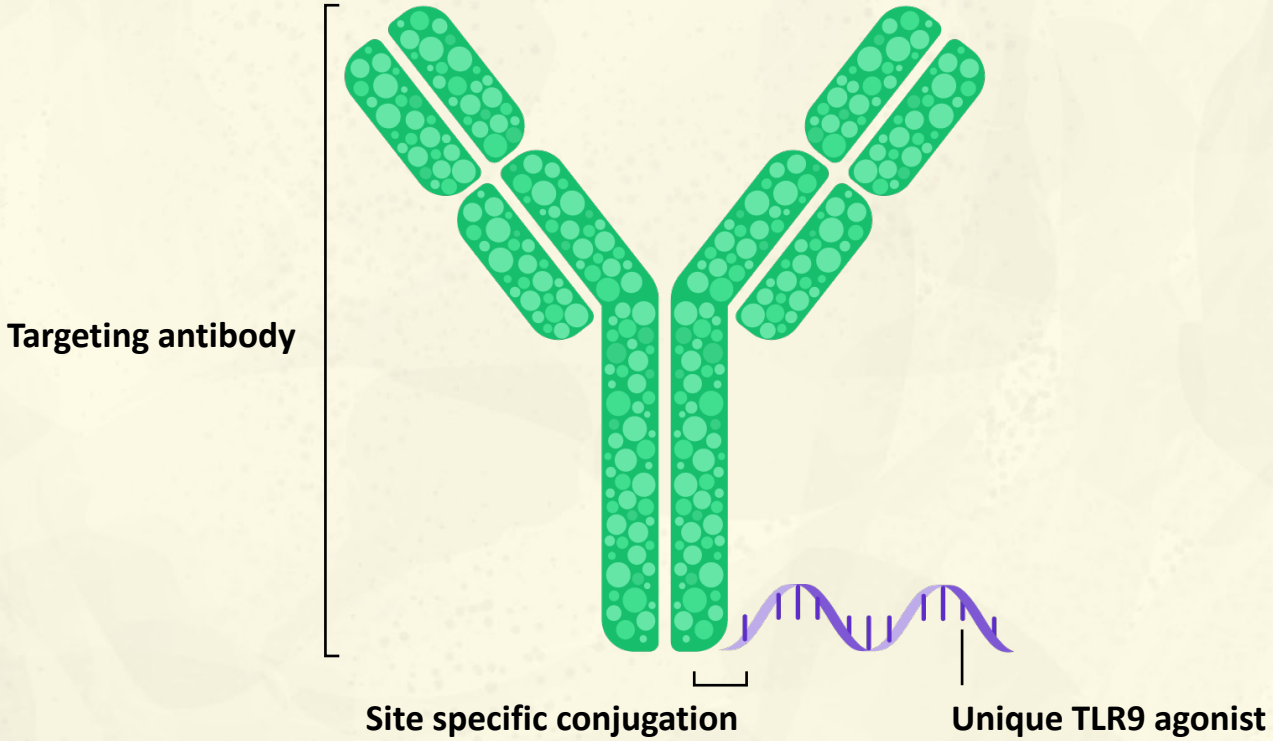
CMP-001 (Checkmate) + Pembrolizumab in Anti-PD-1 Refractory Melanoma, [1-4]Checkmate, S1 2020.

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

ALX ONCOLOGY

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

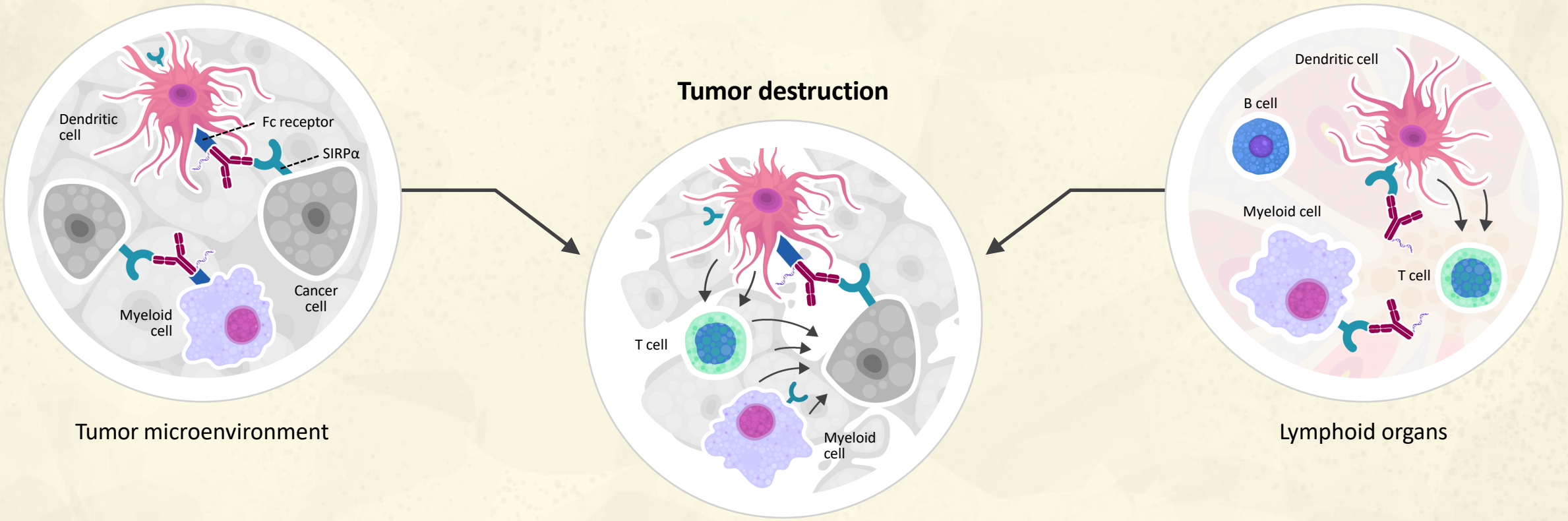
TLR9 Agonist Antibody Conjugate (TRAAC):
Systemic dosing with cell specific TLR9 activation



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

ALX ONCOLOGY

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



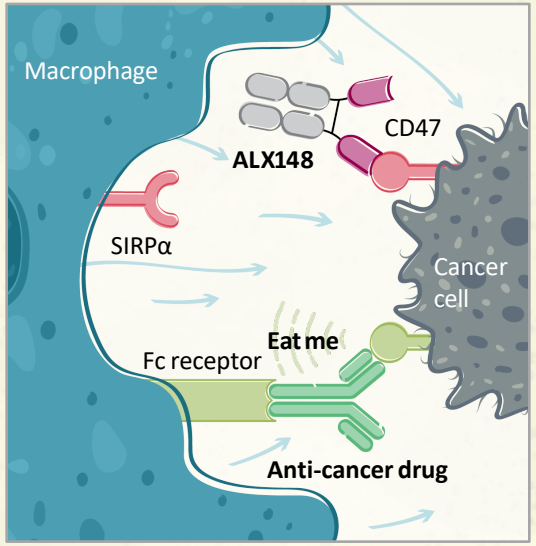
- SIRPα TRAAC binding to myeloid cells targets TLR9 activation in myeloid cells that matter (e.g. dendritic cells).
- SIRPα expression on tumor cells enables tumor microenvironment localization of SIRPα TRAAC.
- SIRPα TRAAC blocks CD47-SIRPα myeloid checkpoint pathway.

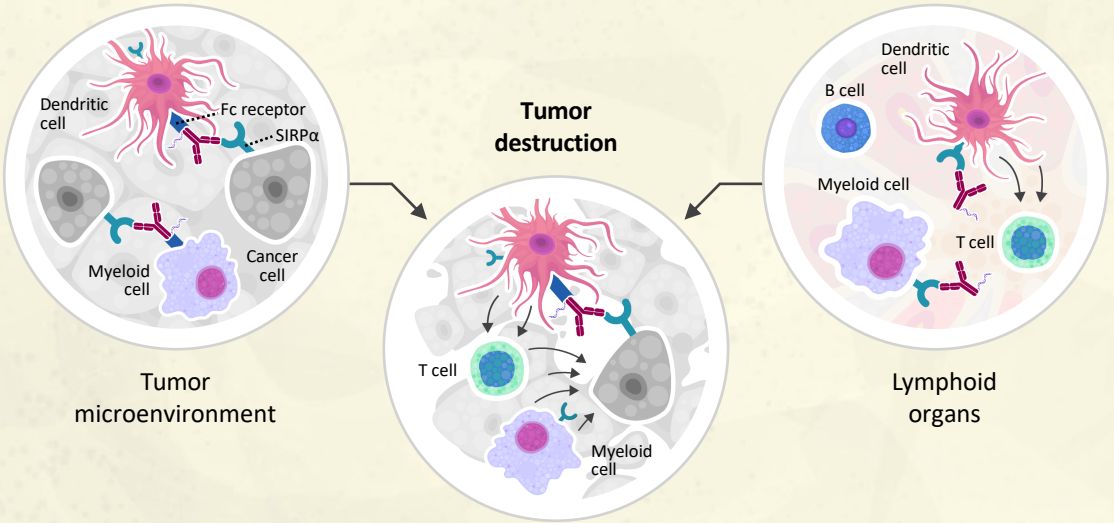
ALX ONCOLOGY

SIRPα TRAAC PROGRAM IS COMPLEMENTARY TO ALX148

ALX148 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.





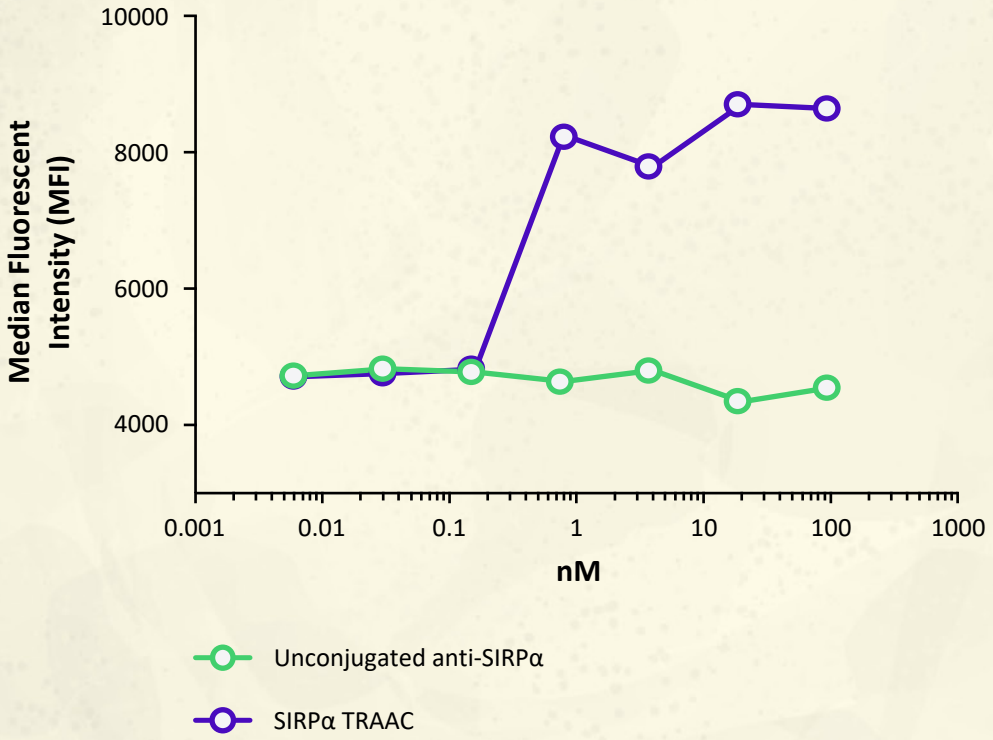
SIRPα TRAAC is an agonistic molecule that directly activates dendritic cells and initiates a coordinated innate and adaptive immune response against cancer.

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

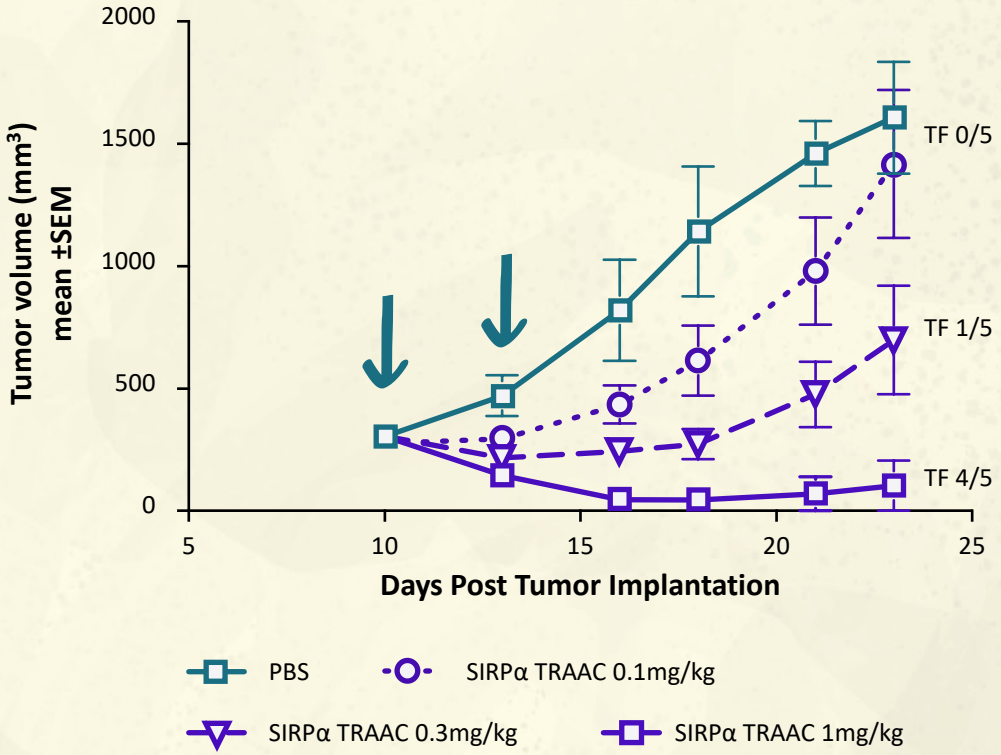
ALX ONCOLOGY

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

Human dendritic cells
Activation Marker CD86



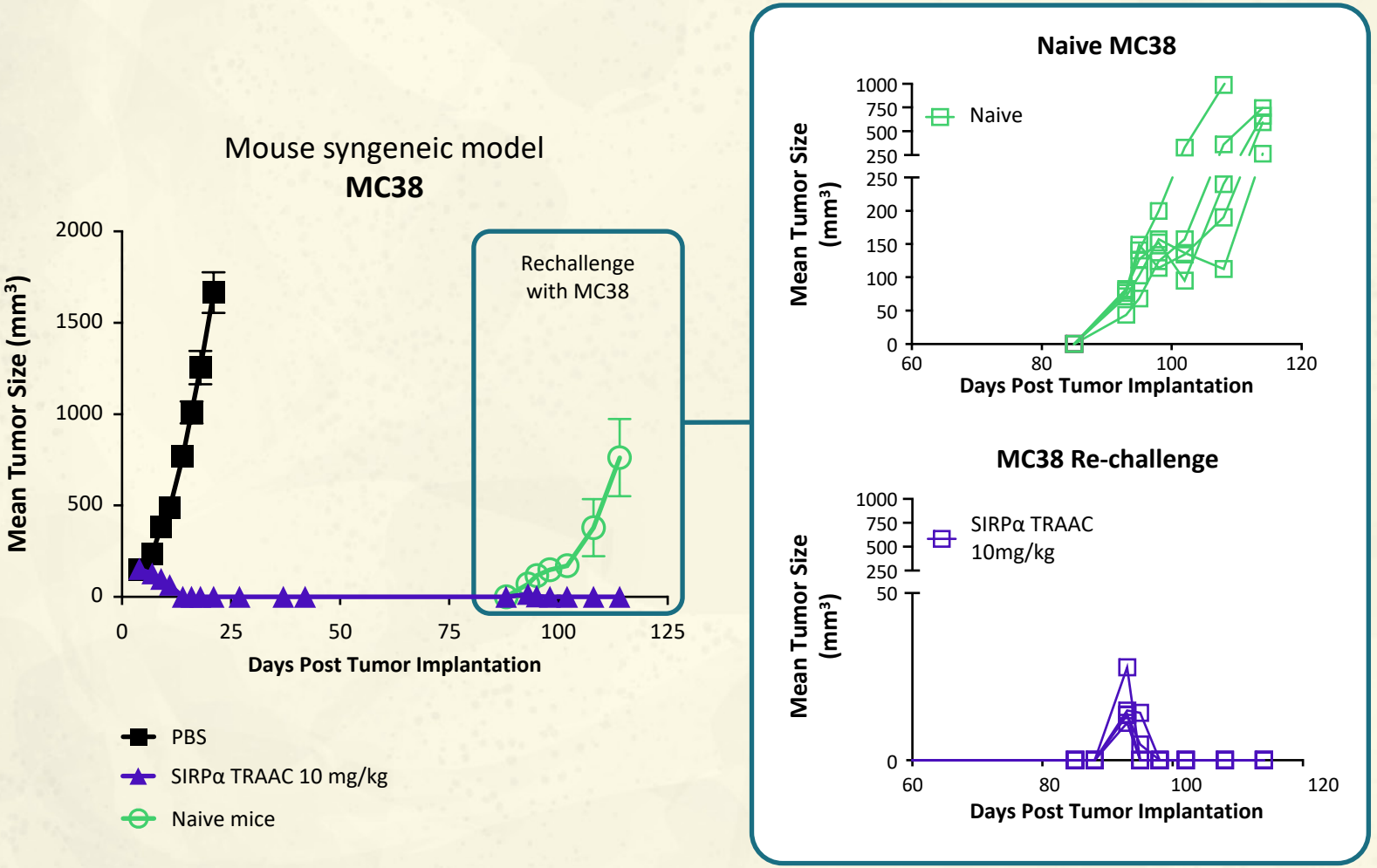
Mouse syngeneic model
CT26



Harrabi et al., SITC, 2020

ALX ONCOLOGY

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

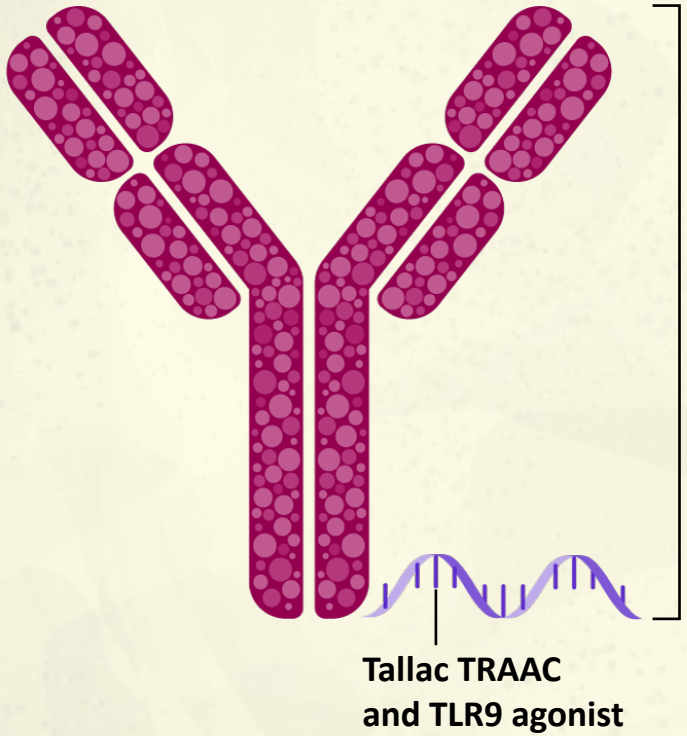


Harrabi et al., SITC, 2020

- Established MC38 tumors were eradicated following 10 mg/kg 2q3d systemic treatment with SIRPα TRAAC.
- These tumor free mice were then re-challenged 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.

ALX ONCOLOGY

SIRPα TRAAC: TARGETING IMMUNE ACTIVATION TO WHERE IT MATTERS



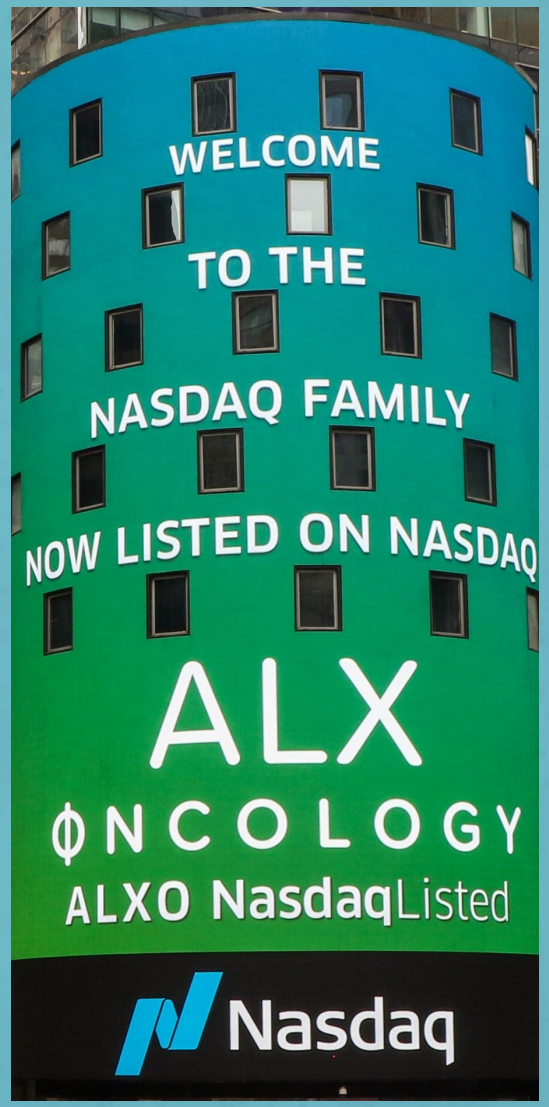
- SIRPα TRAAC binding to myeloid cells targets TLR9 activation in key myeloid cells (e.g. dendritic cells).
- SIRPα expression on tumor cells enables localization of SIRPα TRAAC to tumor microenvironment.
- SIRPα TRAAC blocks CD47-SIRPα myeloid checkpoint pathway.
- Antibody-like PK profile allows for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

IND expected end of 2022

ALX ONCOLOGY

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, 2020:
  - $434.2 million
- Expected cash runway through 2024



ALX ONCOLOGY

STRONG INTELLECTUAL PROPERTY

Robust patent position

- COM patent expiry: 2036 (w/o PTE)
- U.S. patent 10,259,859 granted
- Exclusive, worldwide rights to high affinity SIRPα licensed from Stanford
- Key IP generated in-house
- Non-exclusive, worldwide rights to a broad background CD47 patent portfolio from Stanford

ALX ONCOLOGY

WHY INVEST IN ALX ONCOLOGY: LEADER IN CD47 THERAPY

CD47 is a novel immune checkpoint pathway with clinical proof-of-concept

Clinical proof-of-principle in hematologic and solid tumors

ALX148 is a CD47 blocker with potential for greater efficacy and tolerability due to unique mechanism of action

Growing pipeline in myeloid biology

ALX ONCOLOGY

BACKUP SLIDES

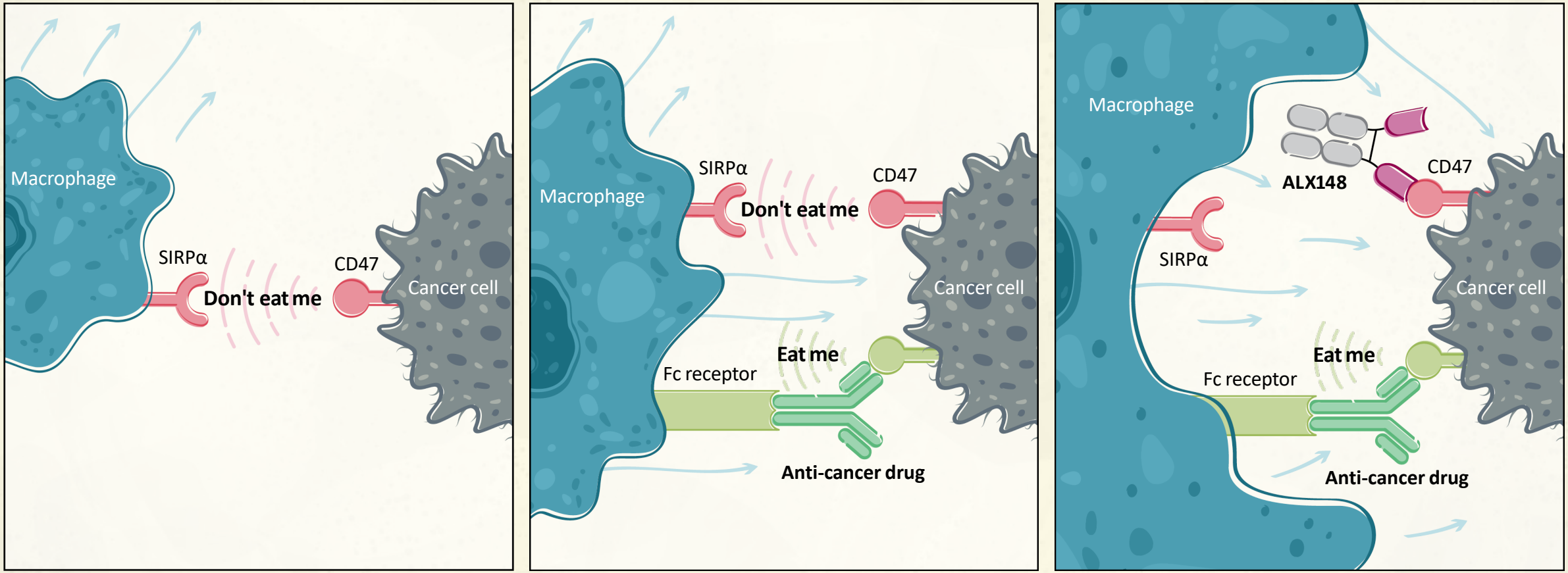
ALX ONCOLOGY

CD47 MECHANISM OF ACTION AS MYELOID CHECKPOINT

Basal state:

Anti-cancer drug alone:

ALX148 combined with anti-cancer drug:



ALX148: designed to work with partner anti-cancer drugs to maximize phagocytosis of cancer cells

ALX ONCOLOGY

NHL TOLERABILITY

Selected hematologic, treatment related adverse events	ALX148 + Rituxan (N=33)[1]		CC-90002 + Rituxan (n=26)[2]		5F9 (magrolimab) + Rituxan (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%**

[1]ASH 2020 Abstract 3016
[2]ASH 2019 Abstract 4089
[3]EHA 2019 Abstract S867

ALX148:
Tolerability profile compares favorably to other CD47 blockers

ALX ONCOLOGY

MAGROLIMAB NHL RESPONSE RATES AND DOSING

DLBCL w/ Rituxan	Ph1	Ph2
N	21	38
Dosing (mg/kg)	up to 30 **Weekly**	30 and 45 **Every Other Week**
ORR	48%	29%
CR	33%	5%
PR	14%	24%

Reduced dosing led to reduced overall response rate in NHL

ORR = overall response rate.
CR = complete response rate.
PR = partial response rate.

EHA 2019 Abstract S867

ALX ONCOLOGY

NHL: PRELIMINARY CLINICAL TOLERABILITY

ALX148 + Rituximab (N=33)

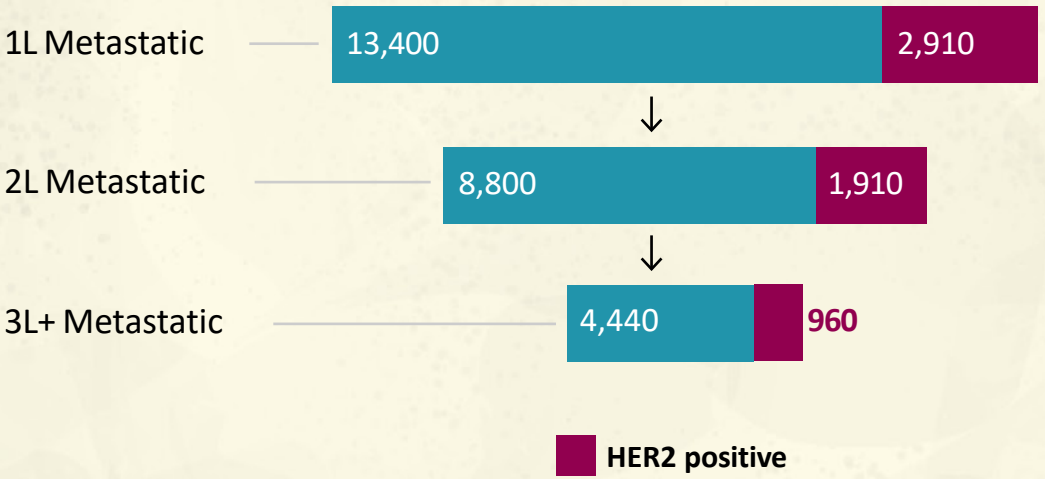
Treatment Related Adverse Event	Total n (%)	≥Grade 3 n (%)
Rash	**8 (24.2)**	—
Fatigue	**4 (12.1)**	—
Nausea	**2 (6.1)**	—
Neutrophil Count Decreased	**2 (6.1)**	**2 (6.1)**
Anemia	**2 (6.1)**	**1 (3.0)**
Myalgia	**2 (6.1)**	—
Pruritus	**2 (6.1)**	—

Data Cutoff: October 1, 2020

ALX ONCOLOGY

HER2 POSITIVE GC UNMET NEED

2020 US patient population
by line of systemic therapy[1]



- Herceptin is an anti-HER2 antibody that has multiple FDA approvals in patients with HER2 positive cancers
- Clinical trials show that re-treatment with Herceptin has no activity in 2L HER2 positive gastric cancer[3]

5-year OS in metastatic gastric cancer is only 6%[2]

ALX ONCOLOGY

[1]DRG Gastroesophageal Cancer published December 2019, HER2+ rate of ~17%.
[2] SEER 18
[3]Makiyama J. Clin Oncology 2020

PHASE 1B ≥2 LINE GC TRIAL: ALX148 + HERCEPTIN + CYRAMZA + PACLITAXEL TREATMENT RELATED ADVERSE EVENTS

Treatment Related Adverse Events

ALX148 (10/15 mg/kg QW) + Herceptin + Cyramza + paclitaxel (N=14)

Adverse Event	Total n(%)
Diarrhea	**3 (21)**
RASH*	**3 (21)**
Urticaria	**3 (21)**
Fatigue	**2 (14)**
Pruritus	**2 (14)**
Lymphocyte count decreased	**1 (7)**
Abdominal pain	**1 (7)**

*RASH: Rash, Dermatitis

≥ Grade 3 Adverse Events

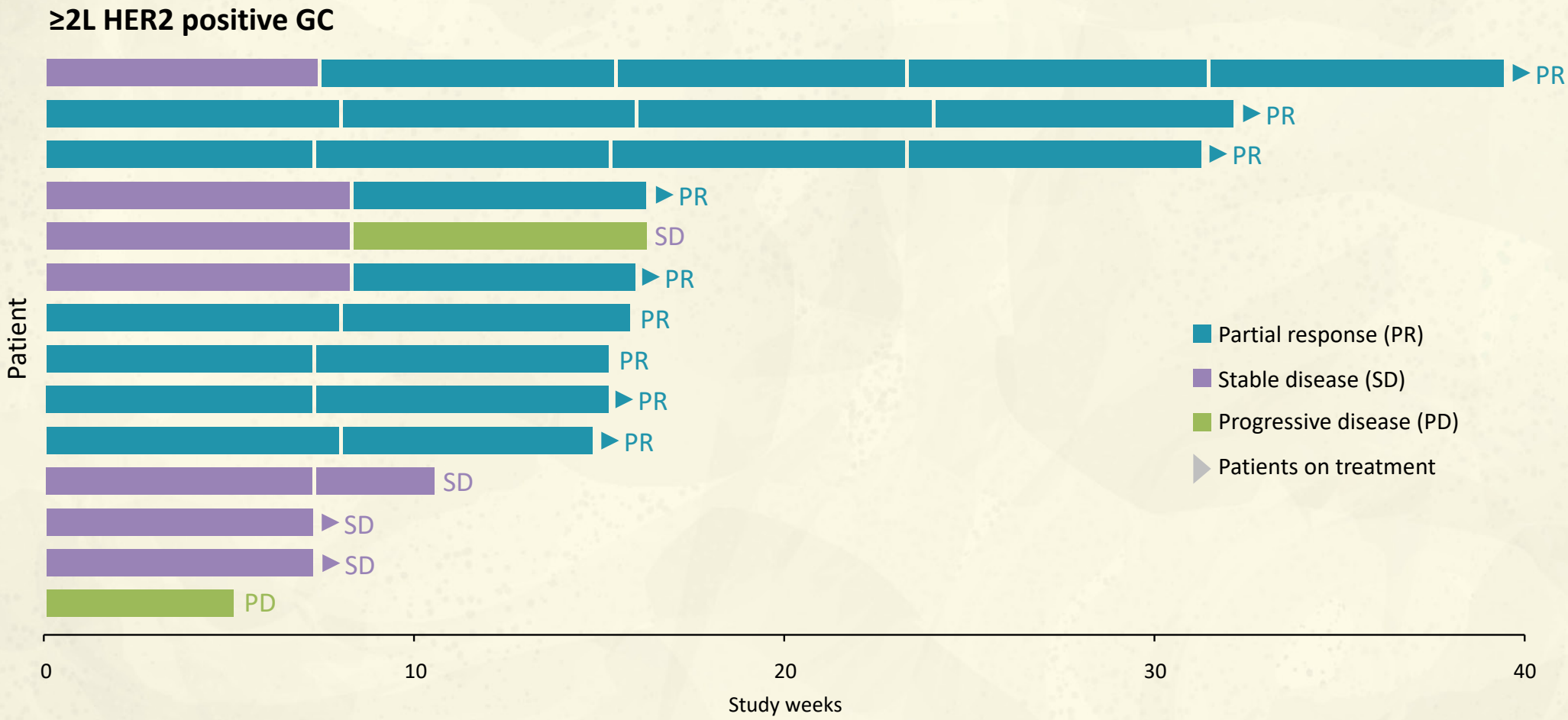
ALX148 (10 and 15 mg/kg QW) + Herceptin + Cyramza + paclitaxel (N=14)

Adverse Event	Total n(%) All Causality		Total n(%) Related	
	Grade 3	Grade 4	Grade 3	Grade 4
Neutrophil count decreased	**5 (36)**	**1 (7)**	-	-
Hypertension	**5 (36)**	-	-	-
Anemia	**1 (7)**	-	-	-
Hypophosphatemia	**1 (7)**	-	-	-
Lymphocyte count decreased	**1 (7)**	-	**1 (7)**	-
Platelet count decreased	**1 (7)**	-	-	-
Urinary tract infection	**1 (7)**	-	-	-

Data Cutoff October 1, 2020

ALX ONCOLOGY

PHASE 1B ≥2 LINE GC TRIAL:
ALX148 + HERCEPTIN + CYRAMZA + PACLITAXEL
BEST OVERALL AND DURATION OF RESPONSE WHILE ON TREATMENT



PHASE 1B FIRST LINE HNSCC: ALX148 + KEYTRUDA + 5FU/PLATINUM TREATMENT RELATED ADVERSE EVENTS

No TRAEs were reported in 1L HNSCC patients (n=5)

≥ Grade 3 Adverse Events

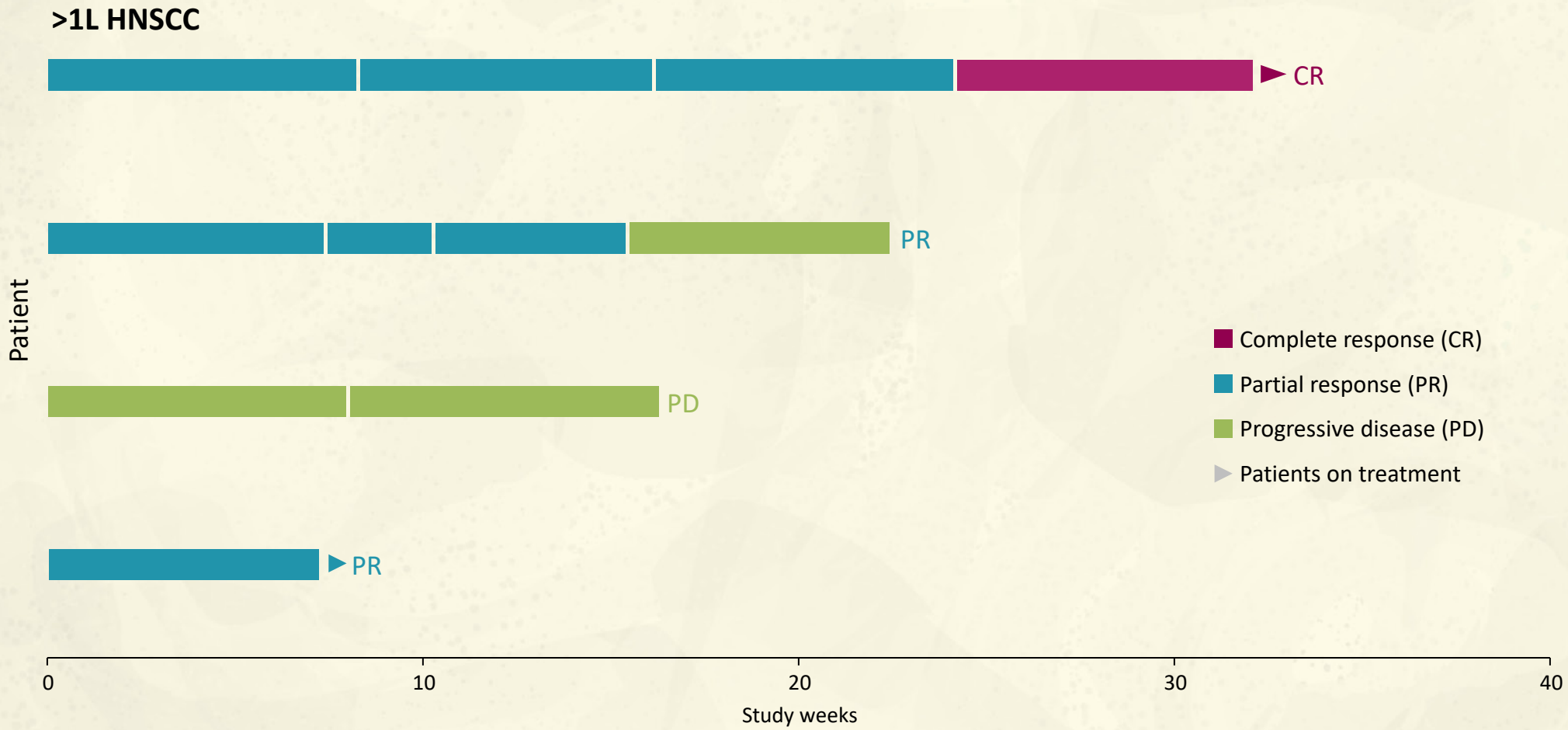
ALX148 (10 and 15 mg/kg QW) + Keytruda + 5FU + platinum (N=5)

Adverse Event	Total n(%) All Causality		Total n(%) Related	
	Grade 3	Grade 4	Grade 3	Grade 4
Neutrophil count decreased	**1 (20)**	-	-	-
Anemia	**1 (20)**	-	-	-
Cardiac tamponade	-	**1 (20)***	-	-
Dysphagia	**1 (20)**	-	-	-
Pericarditis constrictive	**1 (20)***	-	-	-
Supraventricular tachycardia	**1 (20)***	-	-	-

*Events occurred in a single patient with malignant pericardial effusion

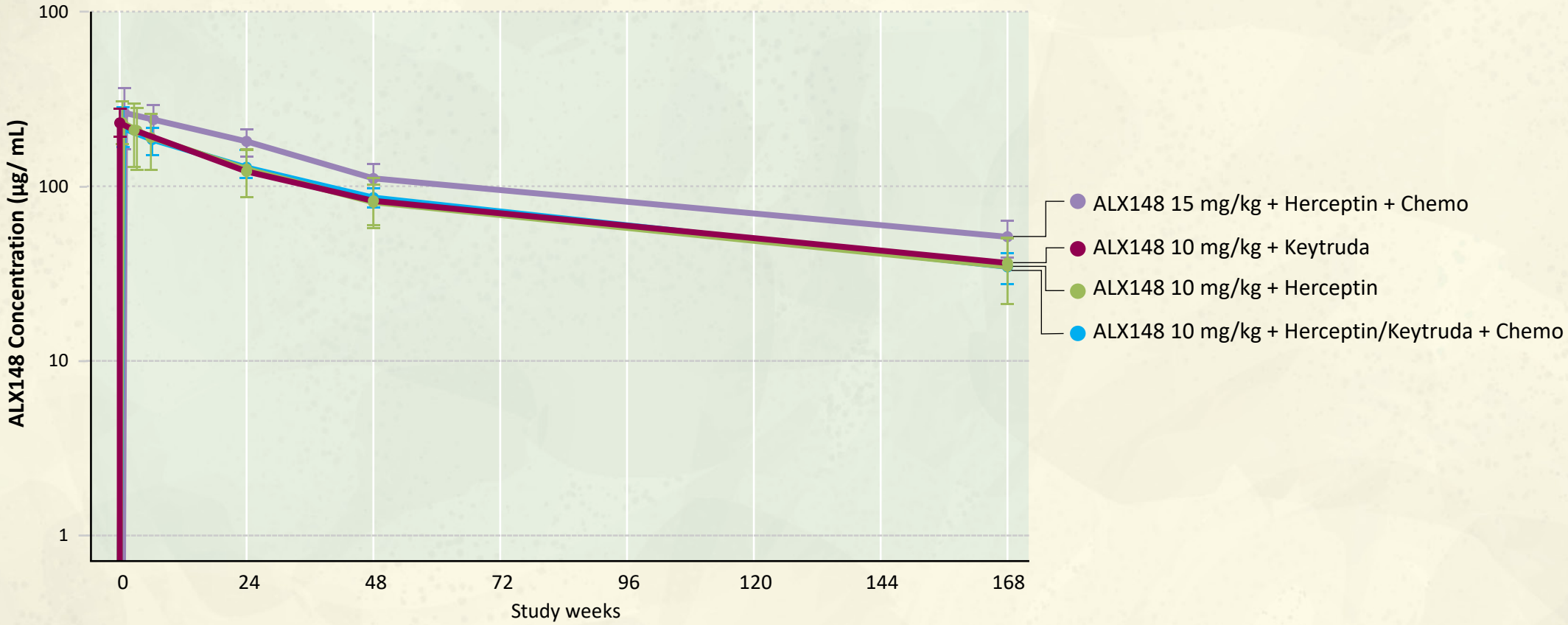
Data Cutoff Oct 1 2020

PHASE 1B FIRST LINE HNSCC: ALX148 + KEYTRUDA + 5FU/PLATINUM BEST OVERALL AND DURATION OF RESPONSE WHILE ON TREATMENT



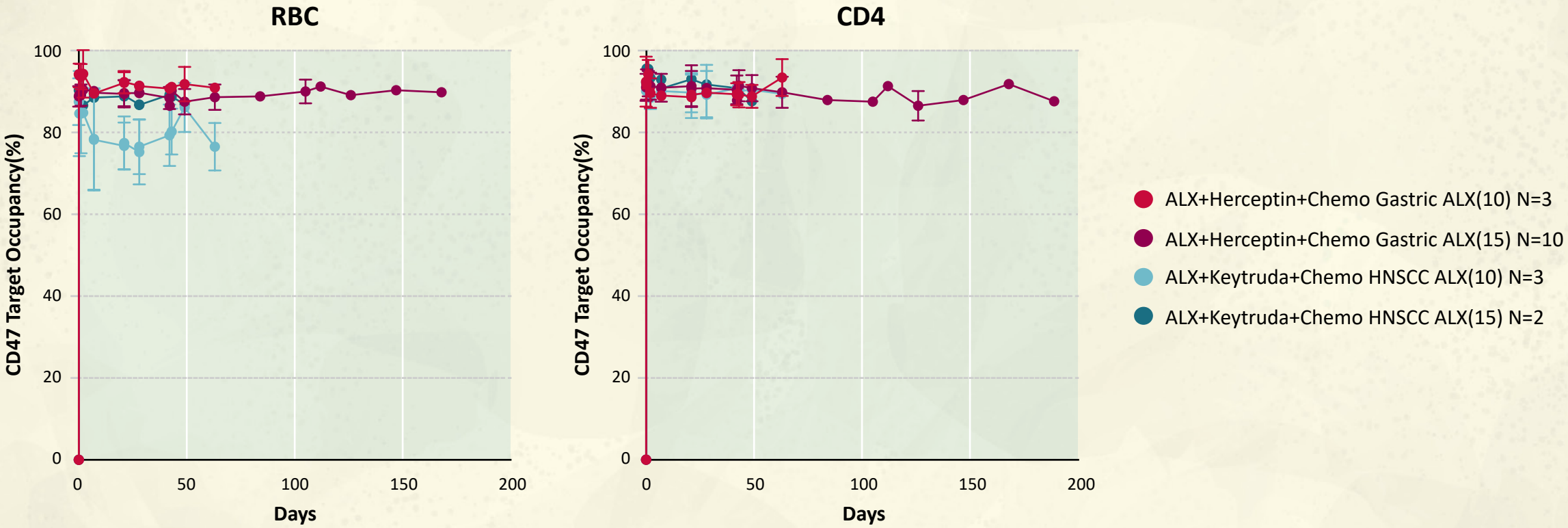
ALX ONCOLOGY

ALX148 PK FOLLOWING COMBINATION THERAPIES IS COMPARABLE WITH AND WITHOUT CHEMOTHERAPY



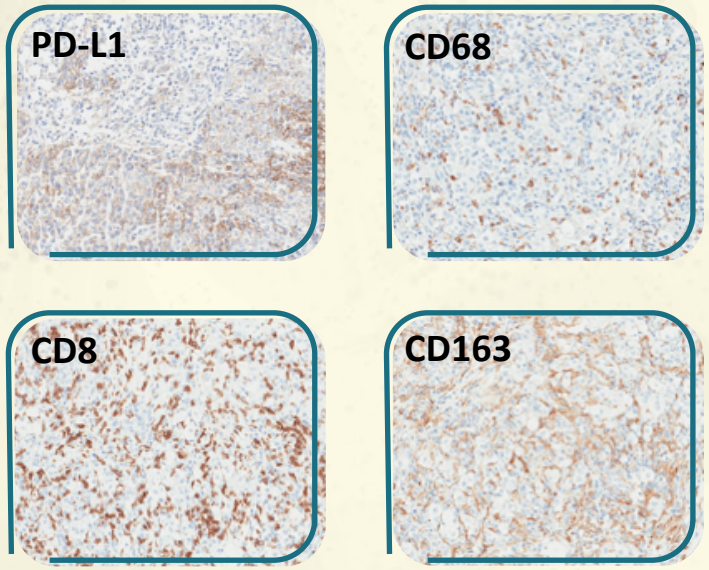
ALX ONCOLOGY

NEAR COMPLETE CD47 TARGET OCCUPANCY IS MAINTAINED THROUGHOUT ALX148 DOSING INTERVAL WHEN COMBINED WITH CHEMOTHERAPY CONTAINING REGIMENS

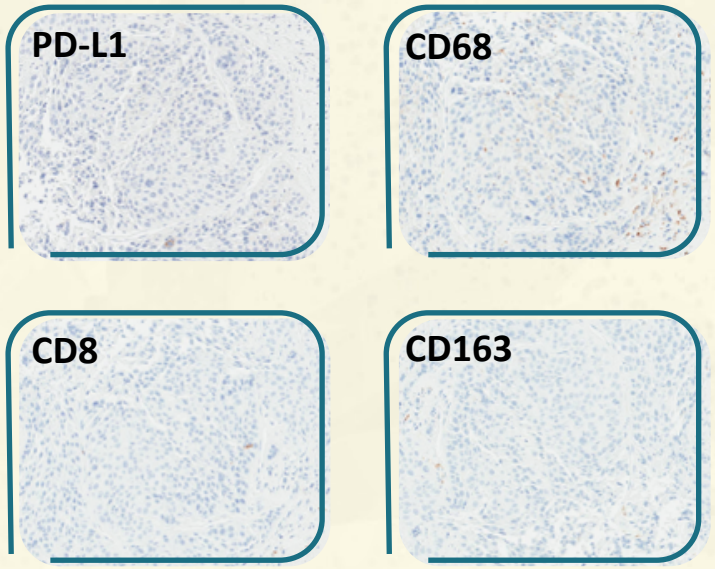


PRE-TREATMENT IHC RESULTS IN HOT AND COLD TUMORS
HNSCC PATIENT 1 (PD-L1 POSITIVE) AND PATIENT 2 (PD-L1 NEGATIVE)

Patient 1 Best Overall Response: CR
Immunologically "hot" tumor



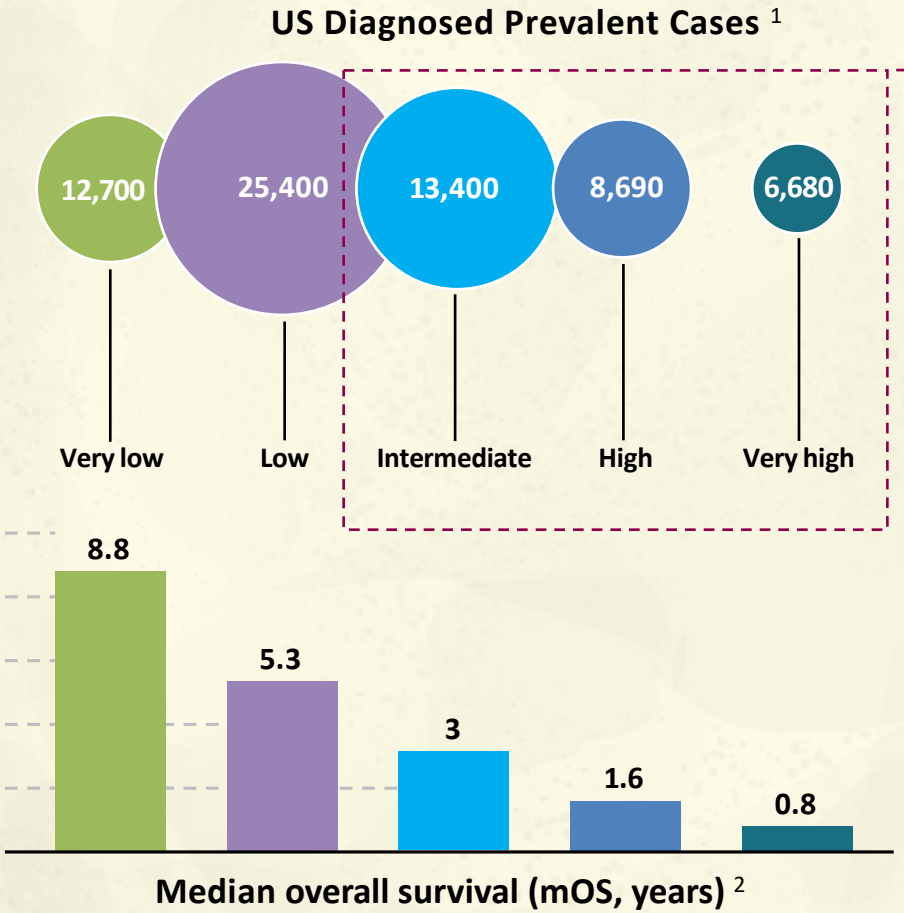
Patient 2 Best Overall Response: PR
Immunologically "cold" tumor



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: 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.

MDS OPPORTUNITY



Higher Risk (HR) MDS

Bone marrow transplant

<10%
Receive allogeneic transplant [3]

Azacitidine, Decitabine

17%
Treated with azacitidine achieve a CR [4]

Overall MDS

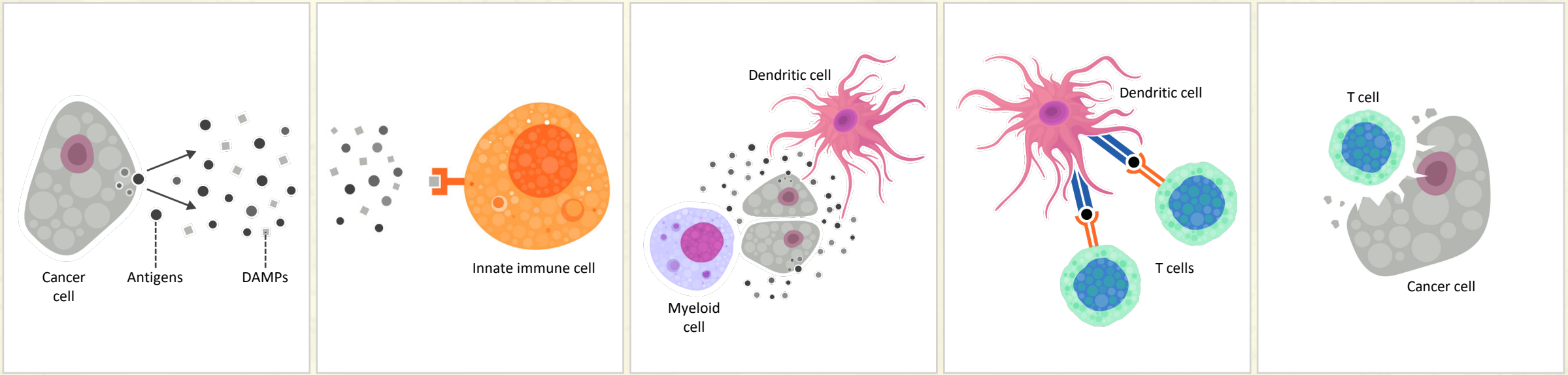
Nearly all pts transfused due to cytopenias

41 of 100
Will die from cytopenia-related causes [5]

Higher risk MDS patients are an area of high unmet need.

Revised international prognostic scoring system (IPSS-R).
[1]Estimated: Decision Resource Group 2019 MDS Report
[2]Greenberg, *Blood,* 2012
[3]Zeidan, *Leukemia & Lymphoma*, 2018
[4]Fenaux, *Lancet Oncology,* 2009
[5]Steensma, *Leukemia & Lymphoma,* 2015

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

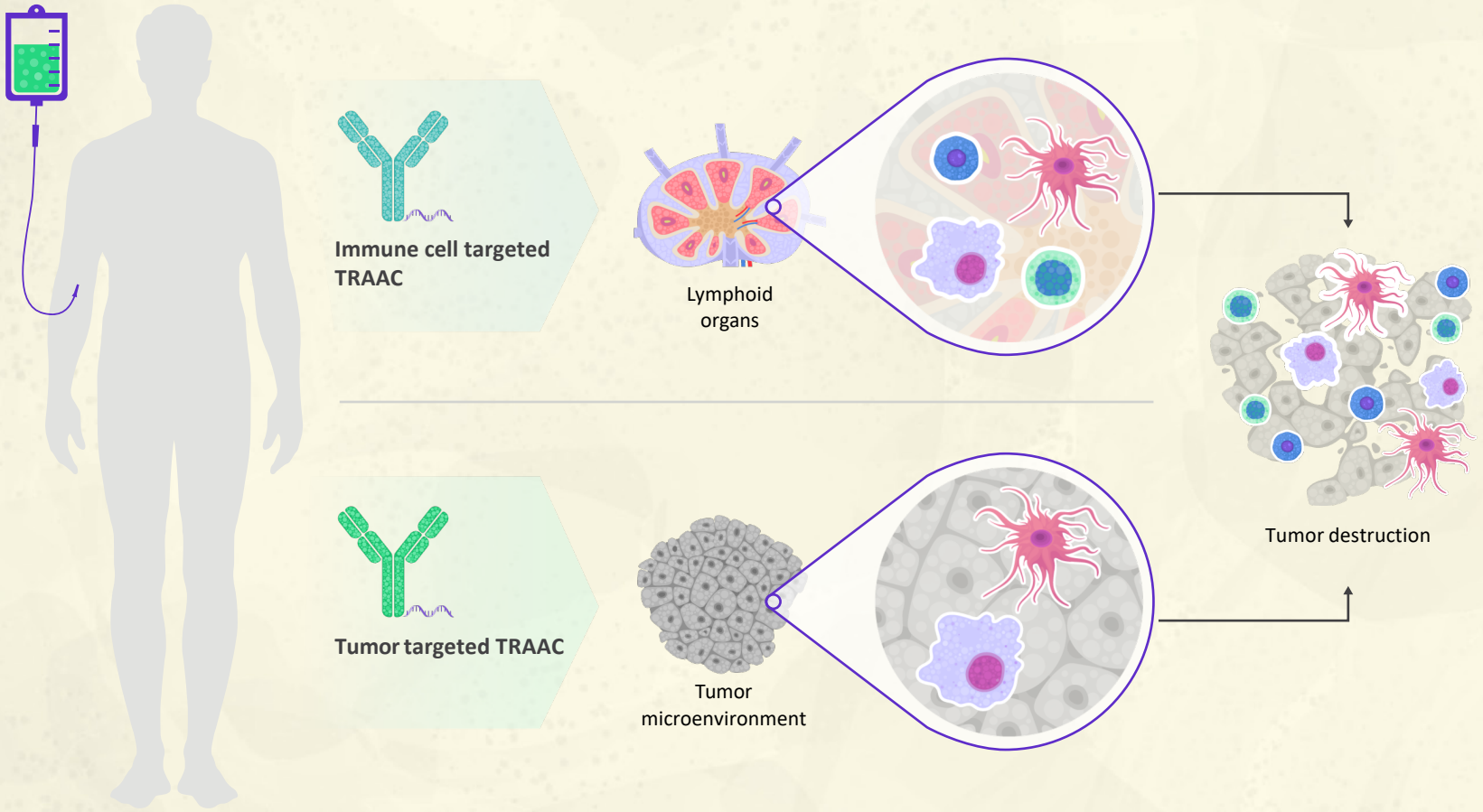
5. Recognition and elimination of tumor by T cells

- 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

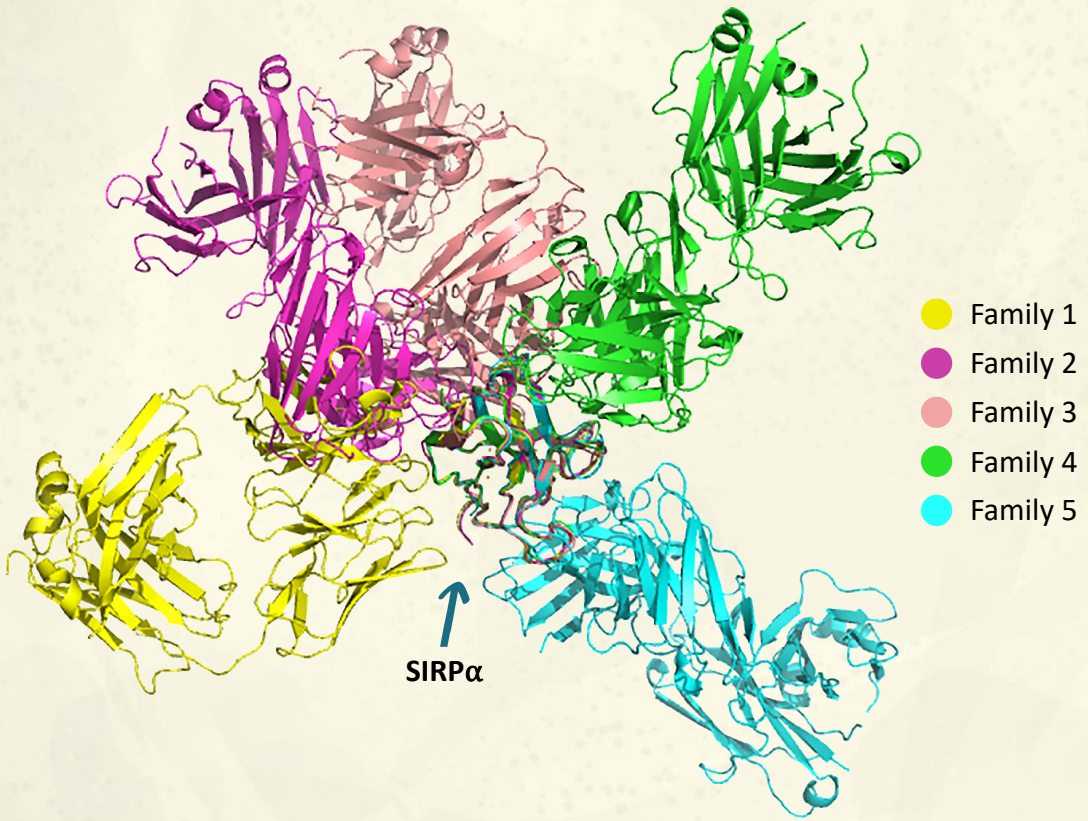
ALX ONCOLOGY

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



ALX ONCOLOGY

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



ALX's diverse range of SIRPα antibodies

Diversity allows selection of best-in-class SIRPα antibodies:

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

ALX ONCOLOGY