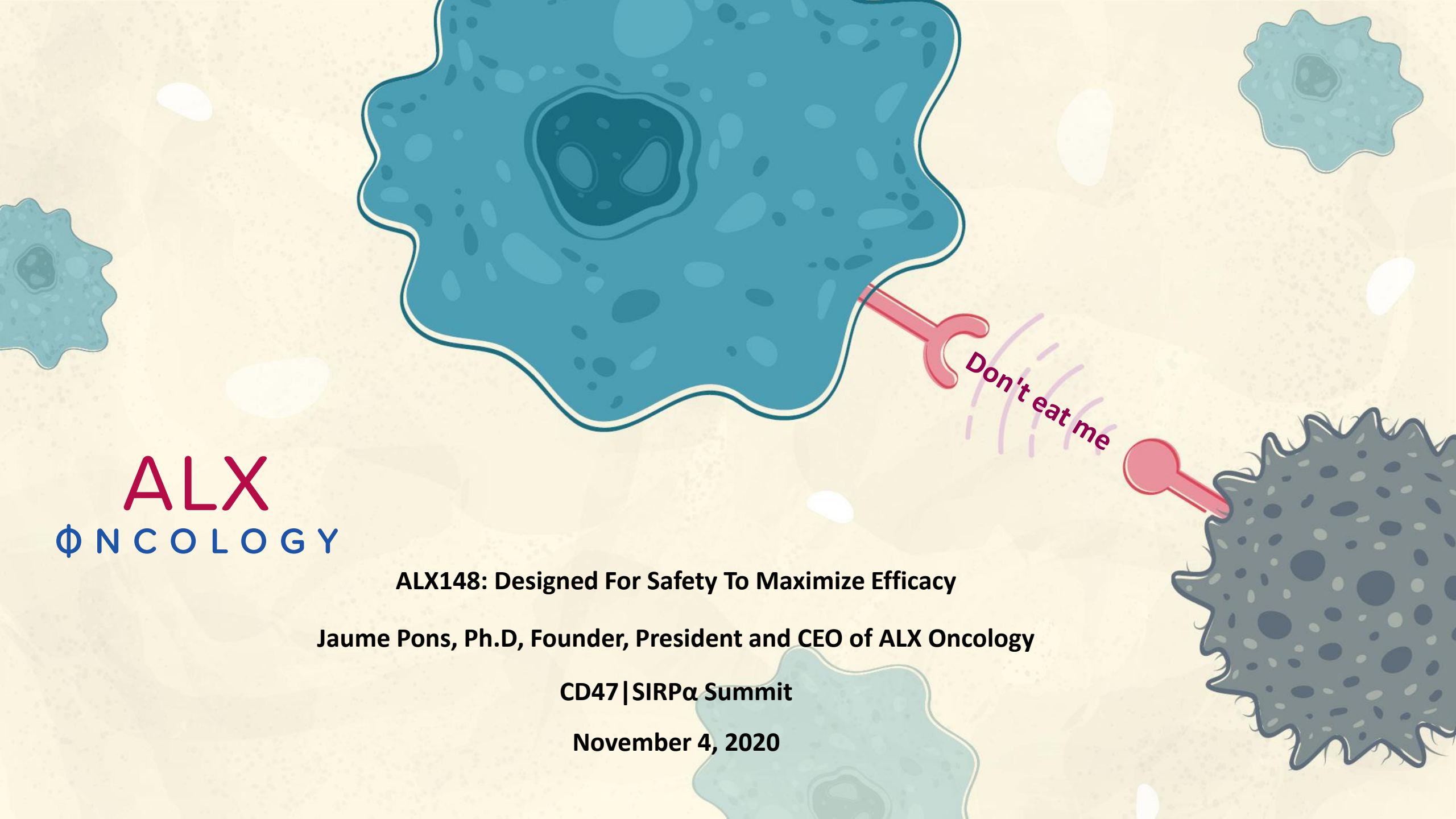

ALX ONCOLOGY

ALX148: Designed For Safety To Maximize Efficacy

Jaume Pons, Ph.D, Founder, President and CEO of ALX Oncology

CD47|SIRPα Summit

November 4, 2020

DISCLAIMER

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

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This presentation also contains estimates and other statistical data based on independent industry publications or other publicly available information, as well as other information based on our internal sources. We have not independently verified the accuracy or completeness of data contained in these industry publications and other publicly available information. Accordingly, we make no representations as to the accuracy or completeness of that data and you are cautioned not to give undue weight to such estimates.

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

CD47 blocker

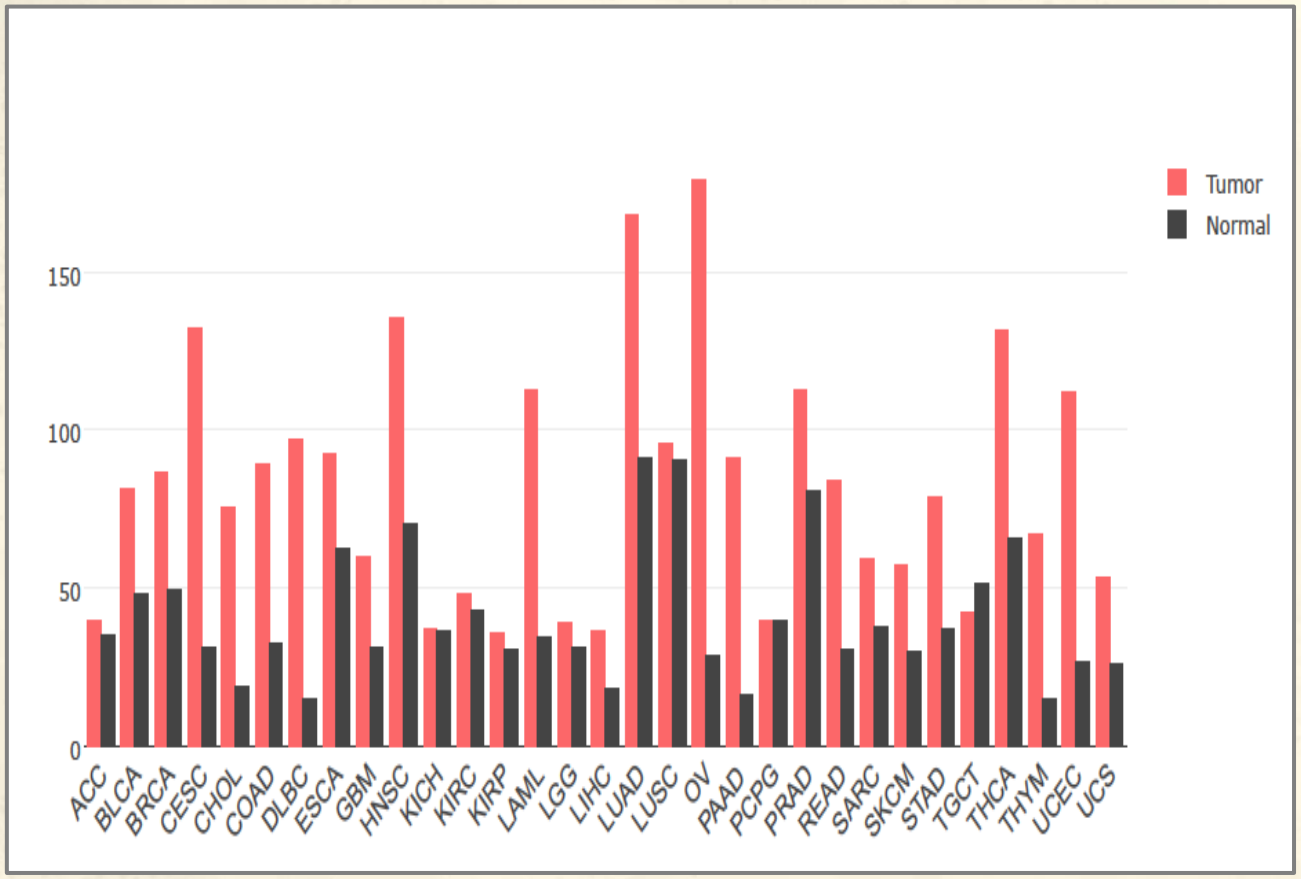
- Designed for use in combination
- Tolerability profile enables higher dosing
- Higher dosing may enable greater efficacy

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

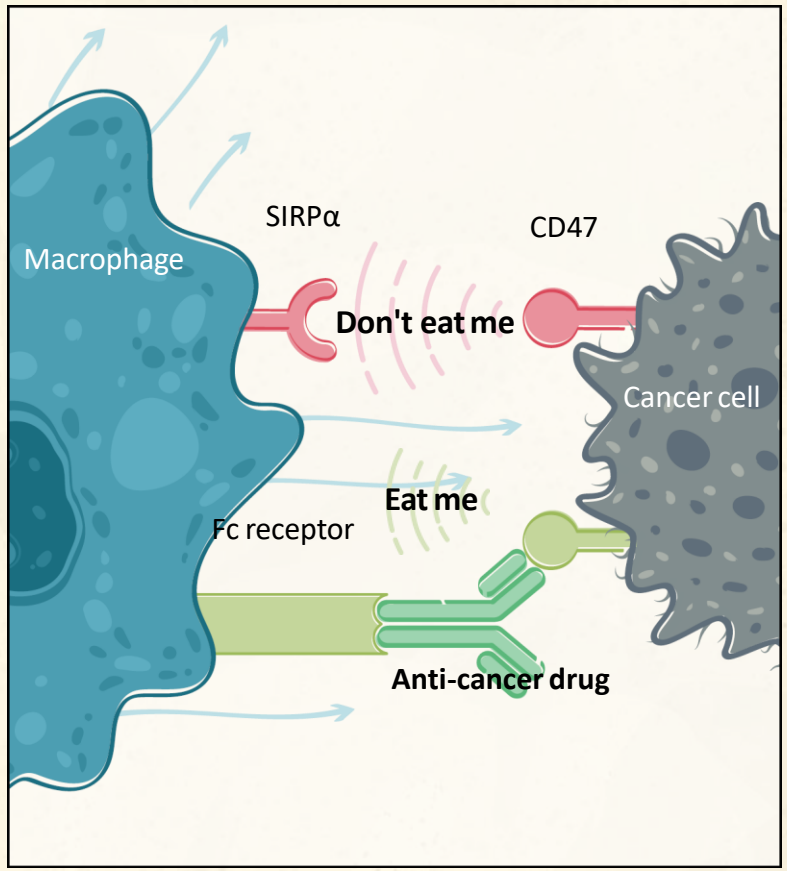
Initial focus on MDS, AML and solid tumors

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

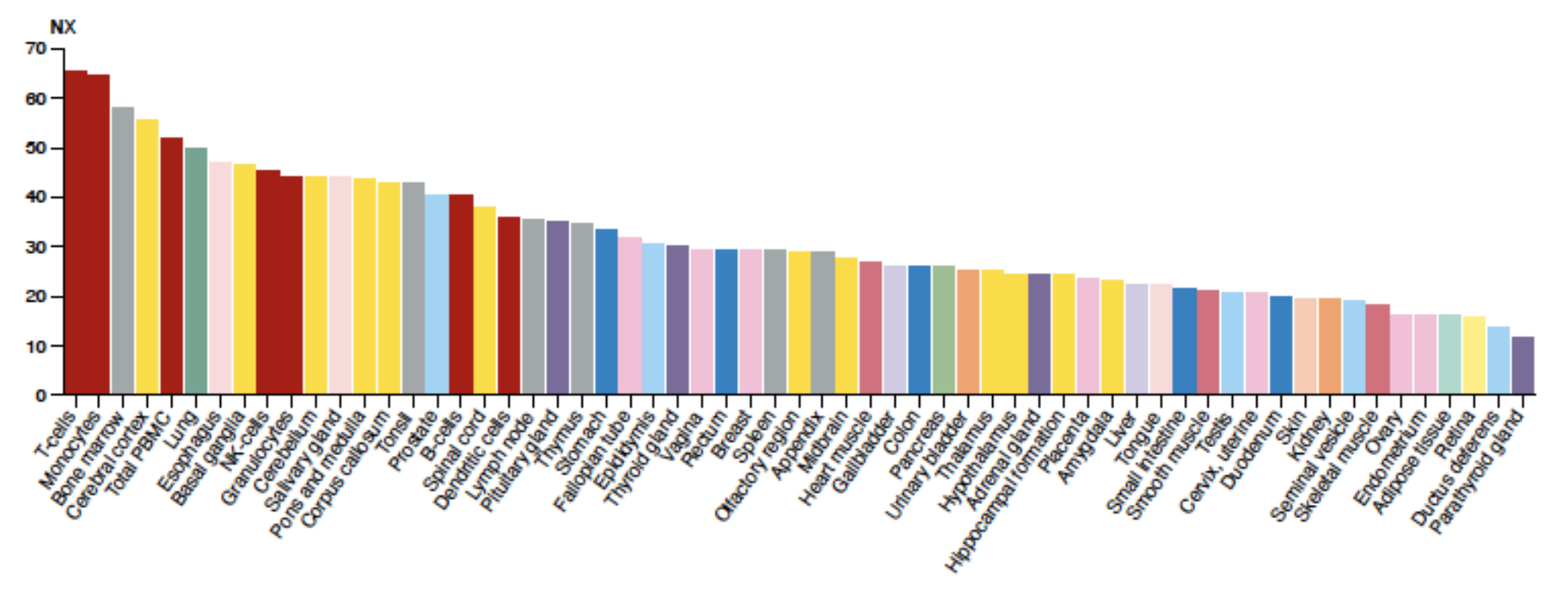
TAA-Expression levels on cancer and normal cells



Checkpoint Mechanism: “do not eat me”



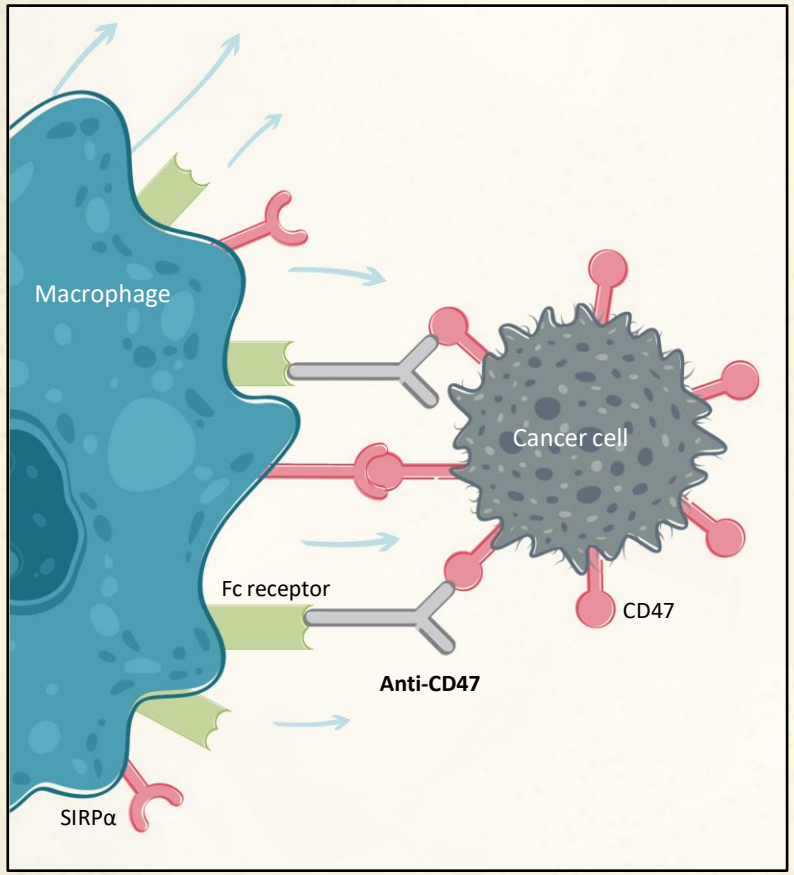
CD47 RNA EXPRESSION ACROSS NORMAL TISSUES

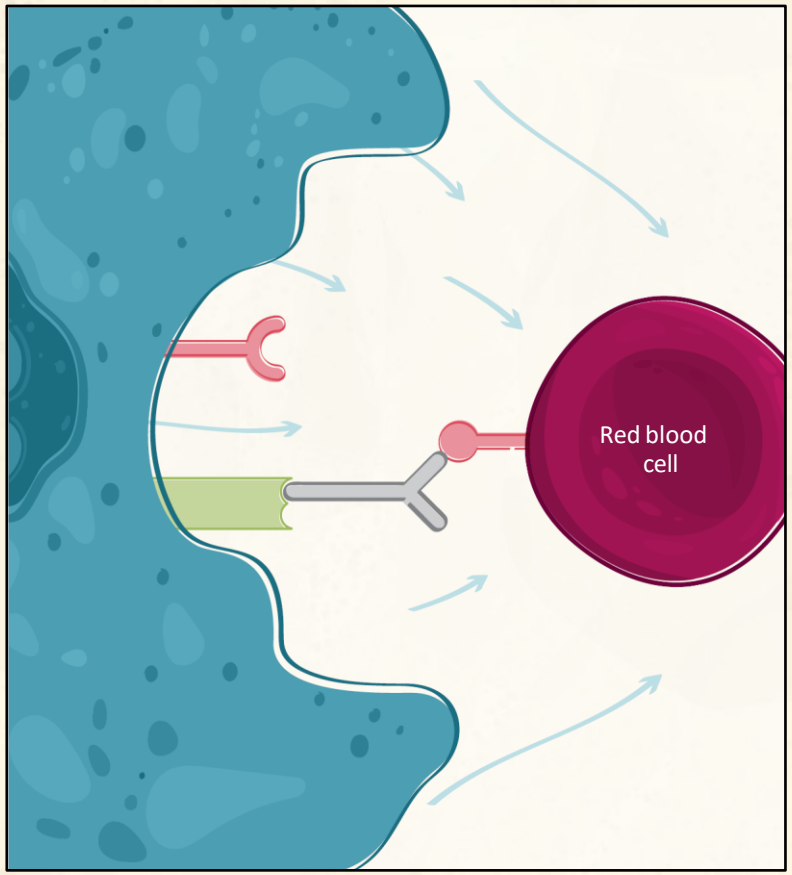


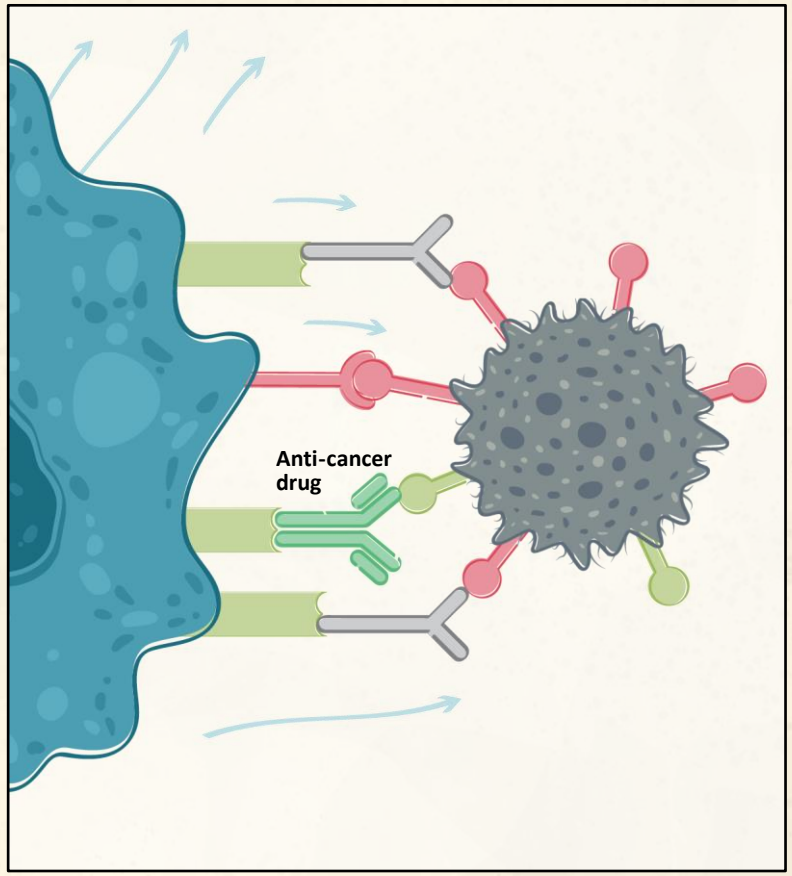
Using CD47 directly targeting cancer cells as TAA will be limited by on-target off-tissue toxicity

TARGETING CD47 AS TUMOR ASSOCIATED ANTIGEN

But also targets normal cells





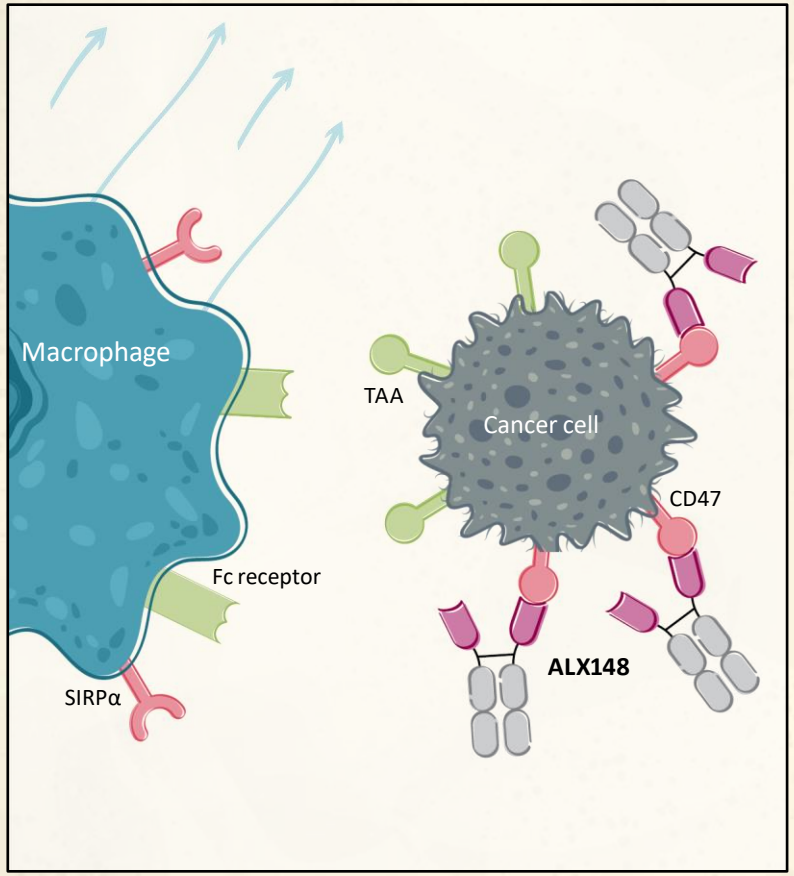


Anti CD47 with active Fc directly targets cancer cells

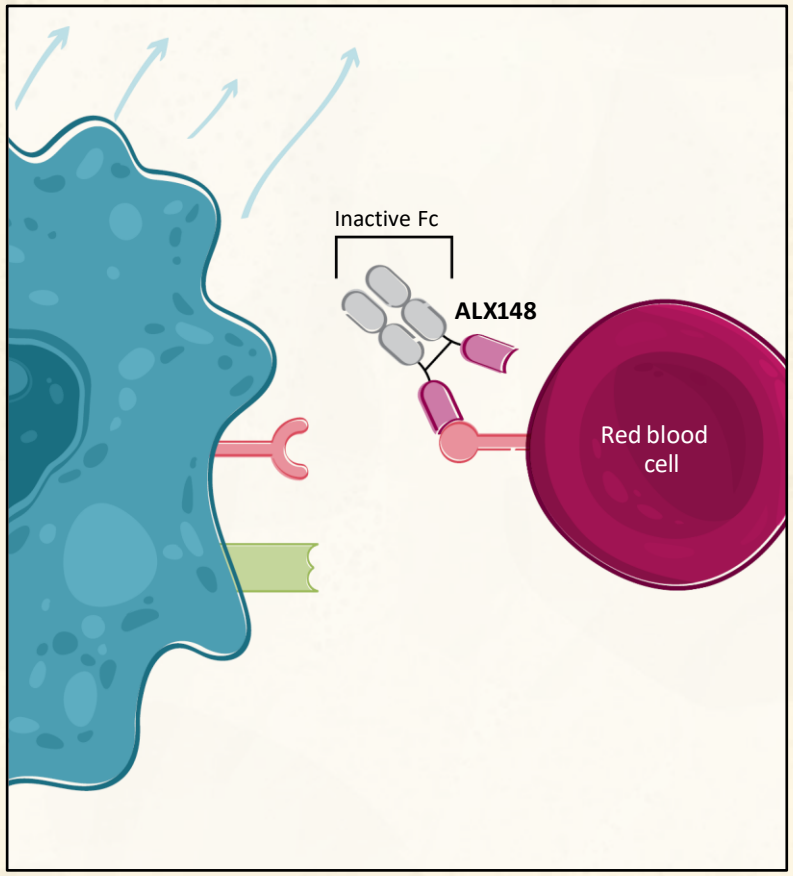
Dose limitations prevent full blockade of CD47 and active fc competes with combo drug

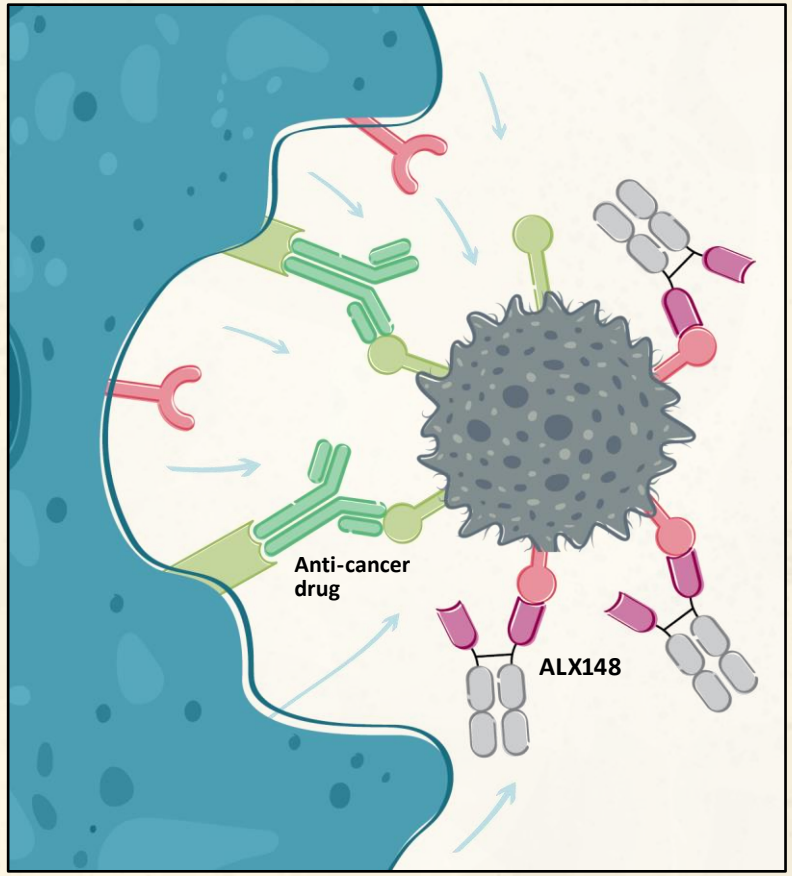
TARGETING CD47 AS CHECKPOINT: ALX ONCOLOGY'S APPROACH

It spares normal cells



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





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

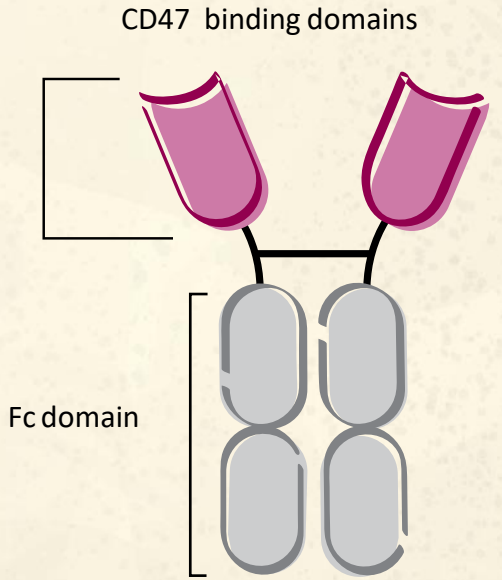
AFFINITY TO CD47 AND FCγ RECEPTORS

Name	Fc Domain (Human)	KD human CD47 (nM)	KD mouse CD47 (nM)	Effector function
ALX148	IgG1 DEAD	0.14	9	-
ALX216	IgG4 S228P	0.14	9	++
ALX377	IgG1 wt	0.14	9	++++
ALX126	IgG1 DEAD	3	65	-
5F9 (magrolimab)*	IgG4 S228P	7	NB	++
TTI-621*	IgG1 wt	500	NB	++++
TTI-622*	IgG4 S228P	500	NB	++

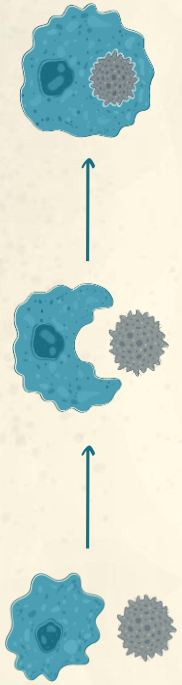
*molecules produced at ALX Oncology, based on public information

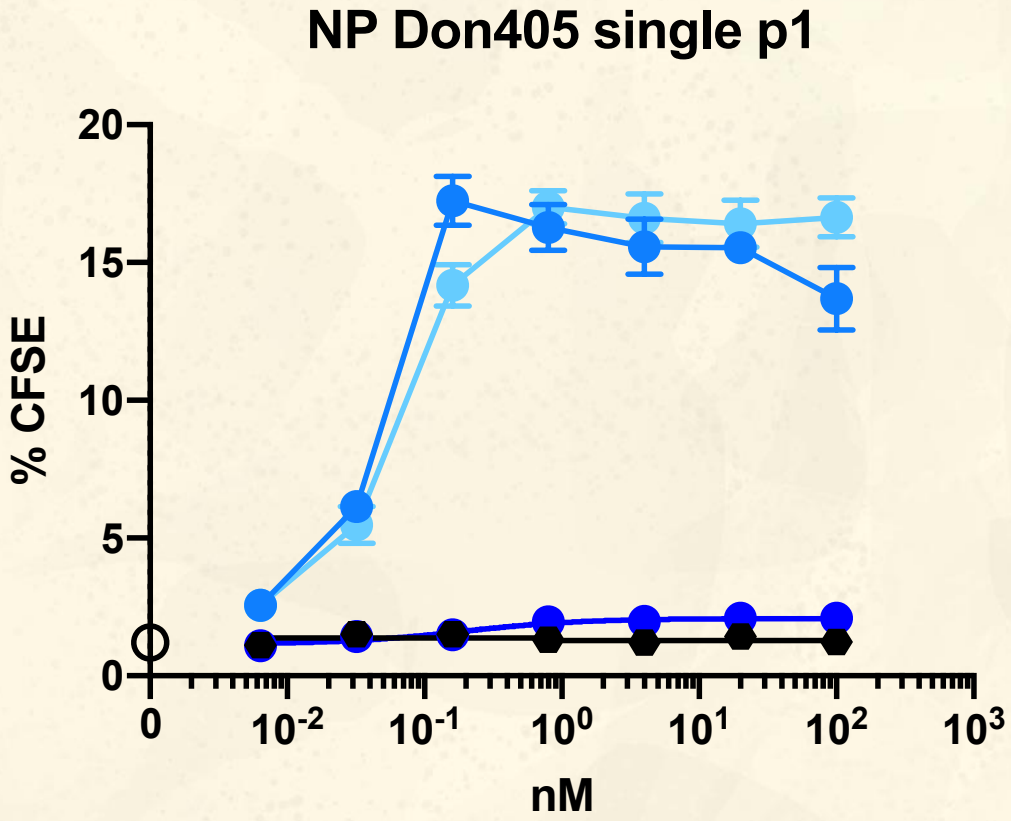
Fc Domain	CD16a (KD nM)	CD32a (KD nM)	CD32b/c (KD nM)	CD64 (KD nM)
IgG1	370	400	2000	0.004
IgG4 S228P	3000	810	850	1
IgG1 DEAD	NB	NB	NB	NB

KD measured by SPR at ALX Oncology



SINGLE AGENT: INCREASE EFFECTOR FUNCTION ENHANCES PHAGOCYTOSIS





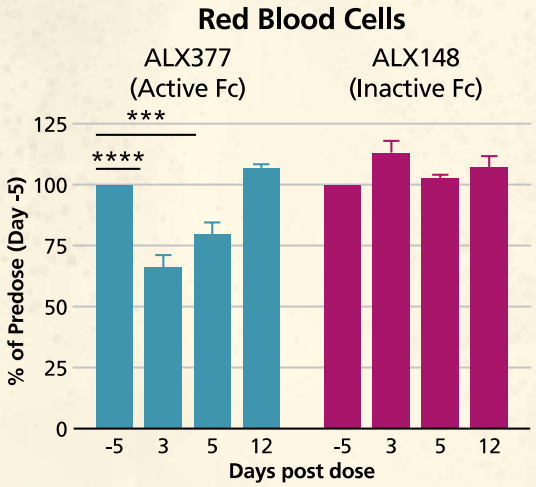
ALX148
ALX216 (IgG4)
ALX377 (IgG1)
Active IgG1 Control
media only

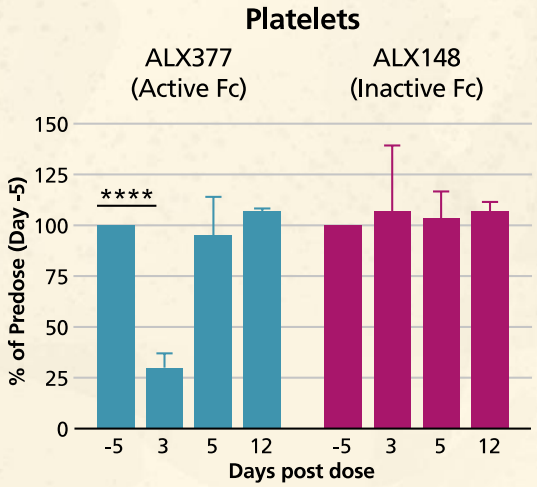
DLD1 cancer cells

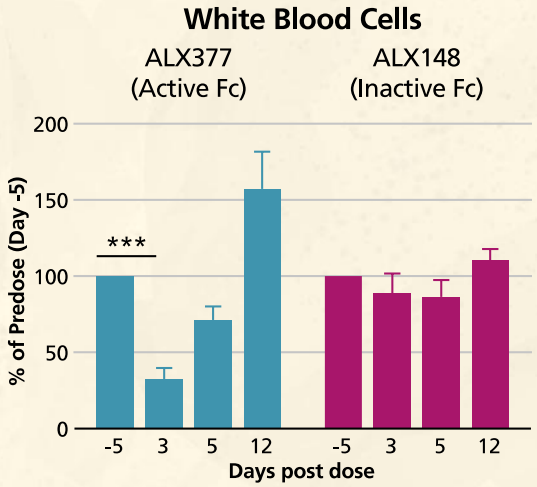
All molecules with same high affinity CD47 binding domain (KD 140 pM)

SINGLE AGENT: INCREASED EFFECTOR FUNCTION INCREASES CYTOPENIAS

All molecules with same high affinity CD47 binding domain
(KD 9 nM for MOUSE CD47)







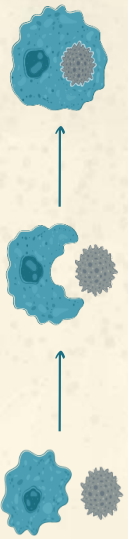
CD-1 mice received 30 mg/kg IV single dose

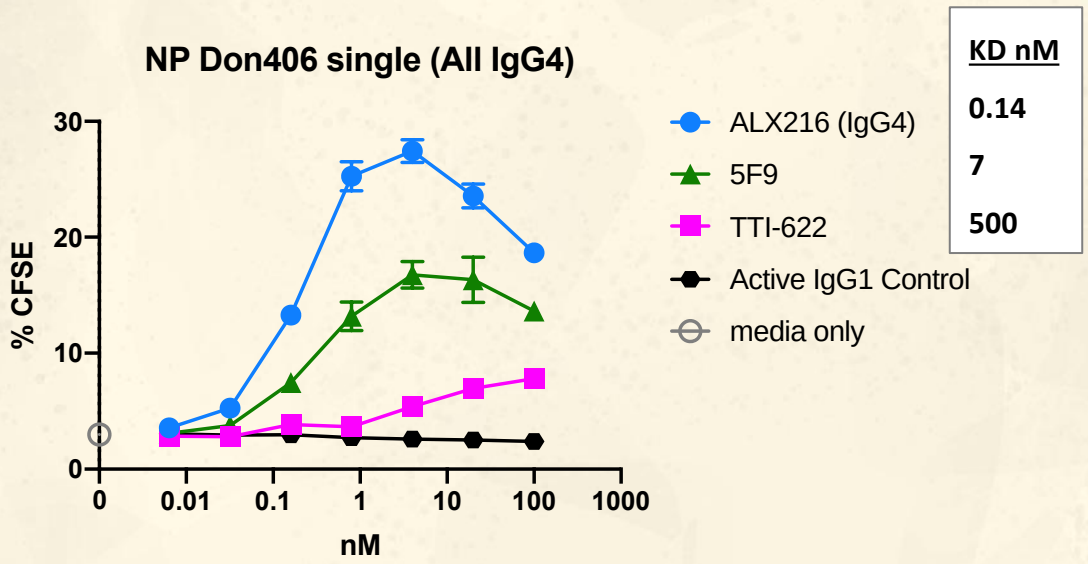
****p<0.0001, ***p<0.001

Inactive Fc is the core determinant of safety profile

SINGLE AGENT: INCREASE OF CD47 AFFINITY ENHANCES PHAGOCYTOSIS

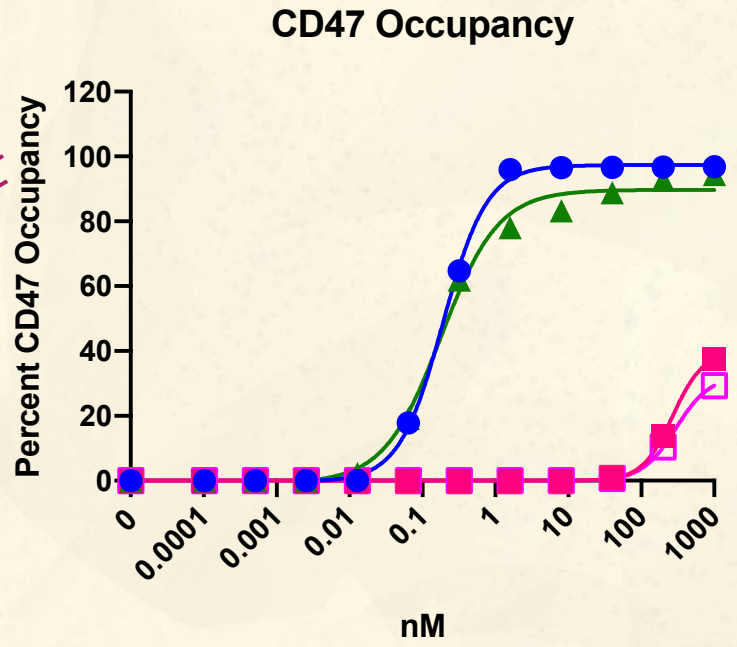
All molecules with same effector function (IgG4)





KD nM
0.14
7
500

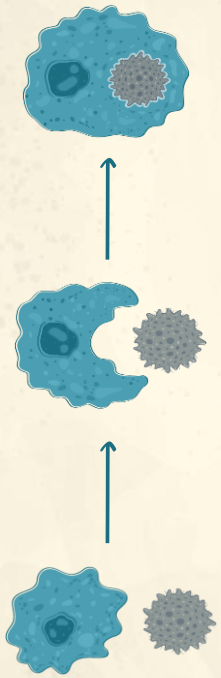
DLD1 cancer cells

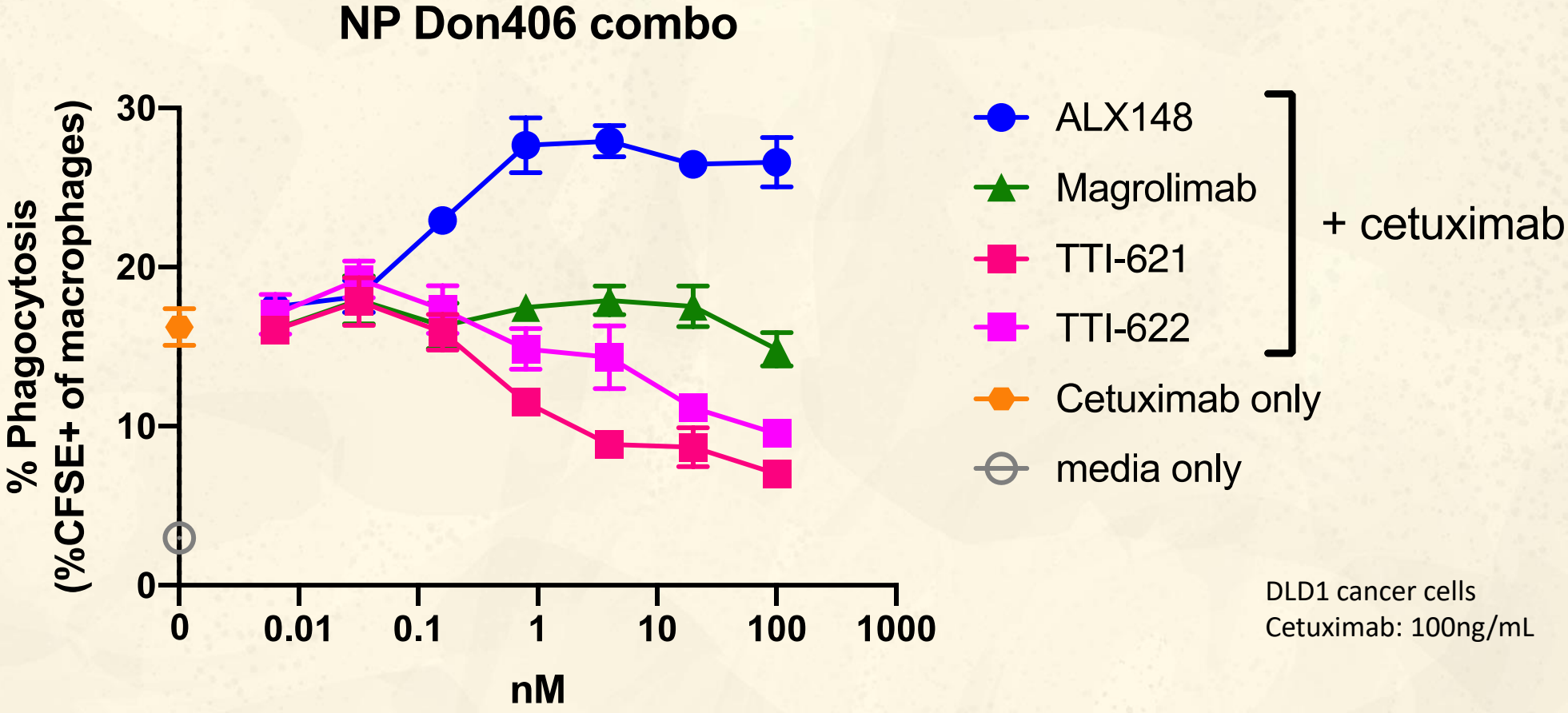


EC50s ng/ml :	
ALX148	25.1
5F9	74.4
TTI-621	> 150
TTI-622	> 150

CD47 as TAA does not require 100% receptor occupancy, but there is higher activity at higher occupancy

COMBINATION: ACTIVE FC DOES NOT ENHANCE ANTICANCER ANTIBODY

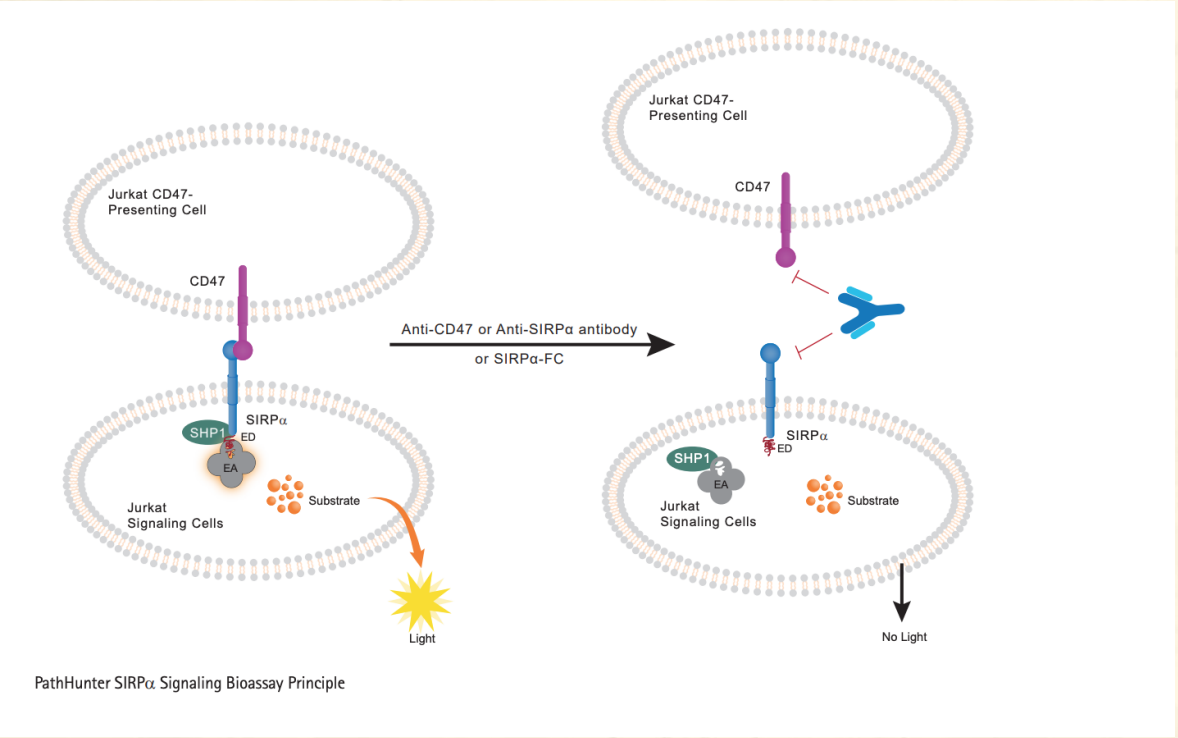




DLD1 cancer cells
Cetuximab: 100ng/mL

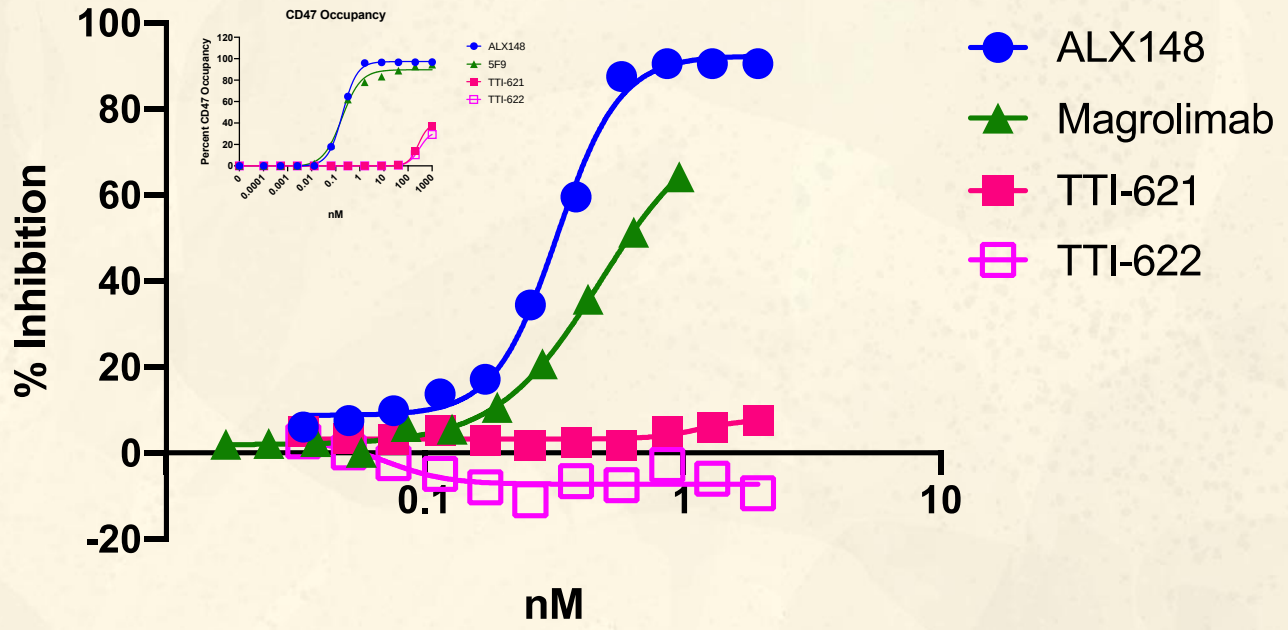
High affinity and no effector function maximizes the phagocytosis of combo drug in vitro

CD47 AS CHECKPOINT: HIGH AFFINITY IS REQUIRED TO INHIBIT CELL/CELL INTERACTION



PathHunter SIRPα Signaling Bioassay Principle

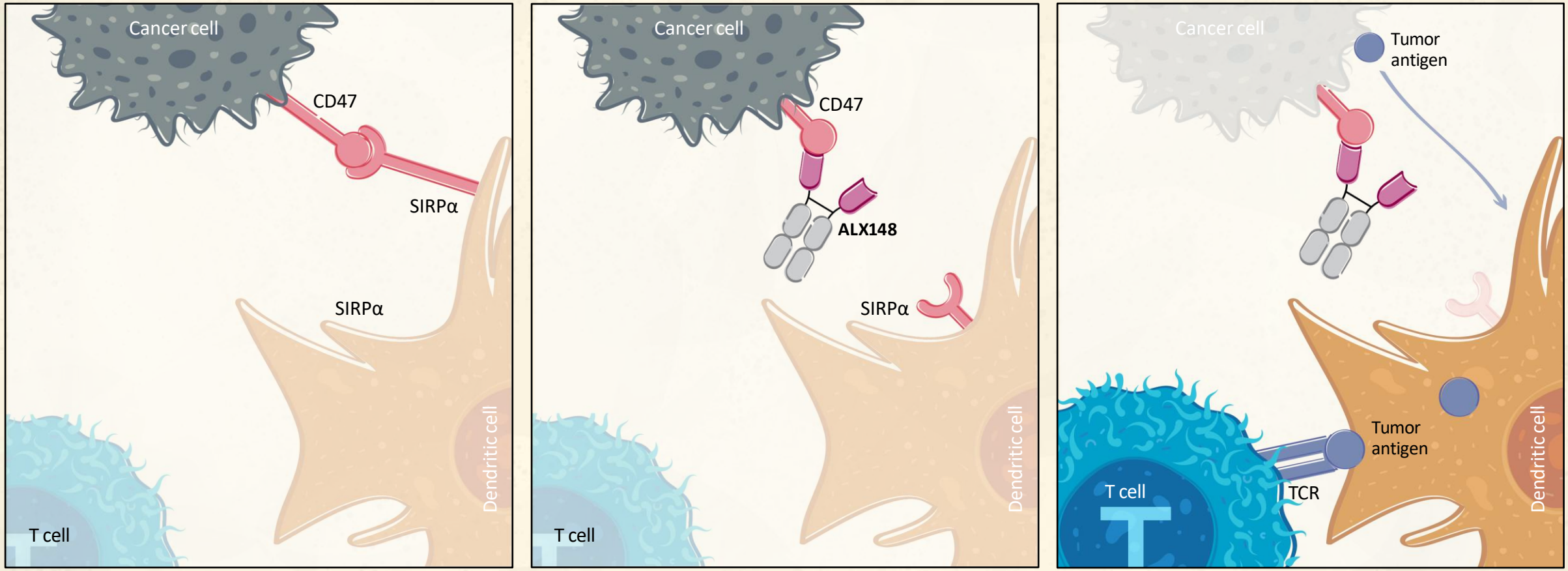
% Inhibition of SIRPa Signaling using the DIscoverX PathHunter SIRPa Signaling Bioassay



100% inhibition is achieved at near complete receptor occupancy level

CD47-SIRPα INTERACTION INHIBIT DENDRITIC CELLS IN TUMOR MICROENVIRONMENT

Blockade of CD47 activate DCs



CD47-Sirpα interaction inhibits Dendritic cells and keep macrophages in M2 phenotype

Activated DC present antigens to T-cells and these get activated and attack cancer cells

CD47 AS CP: HIGH AFFINITY IS REQUIRED TO DEREPRESS DC IN VIVO

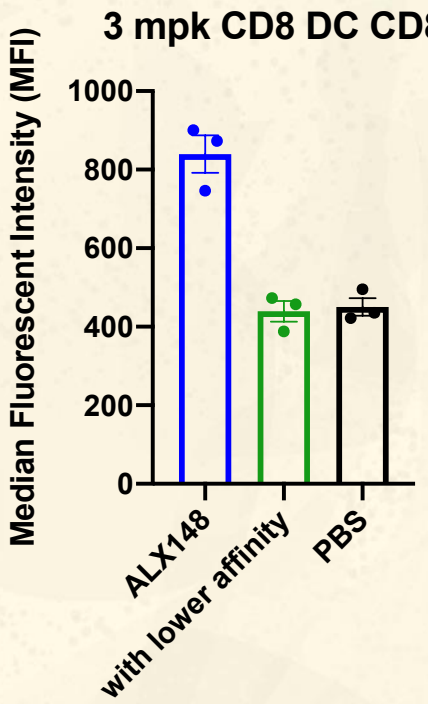
3 mpk ALX148 or ALX126

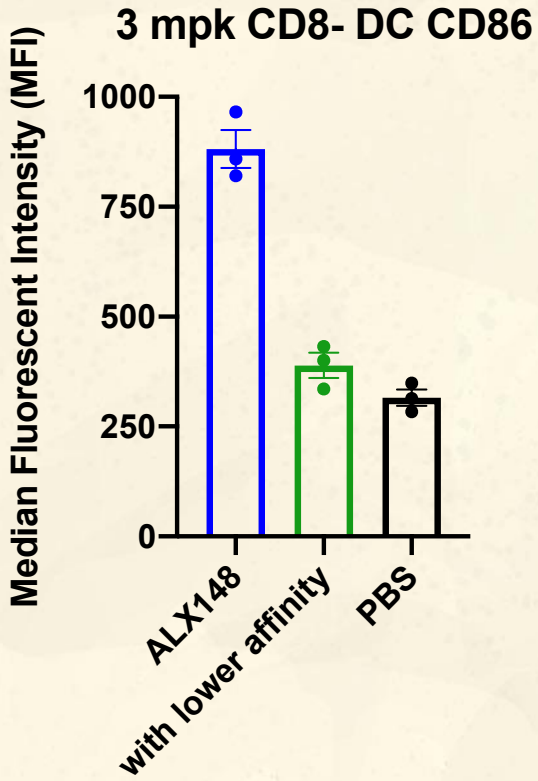
I.P.

3.5hrs

Harvest Spleen

Measure activation of DCs by flow cytometry





mCD47:
ALX148 = KD 9 nM
ALX126 = KD 65 nM

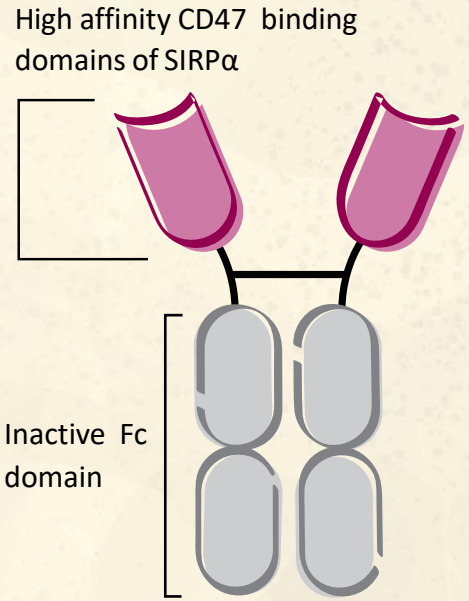
ALX148: METICULOUSLY DESIGNED CD47 BLOCKER

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

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

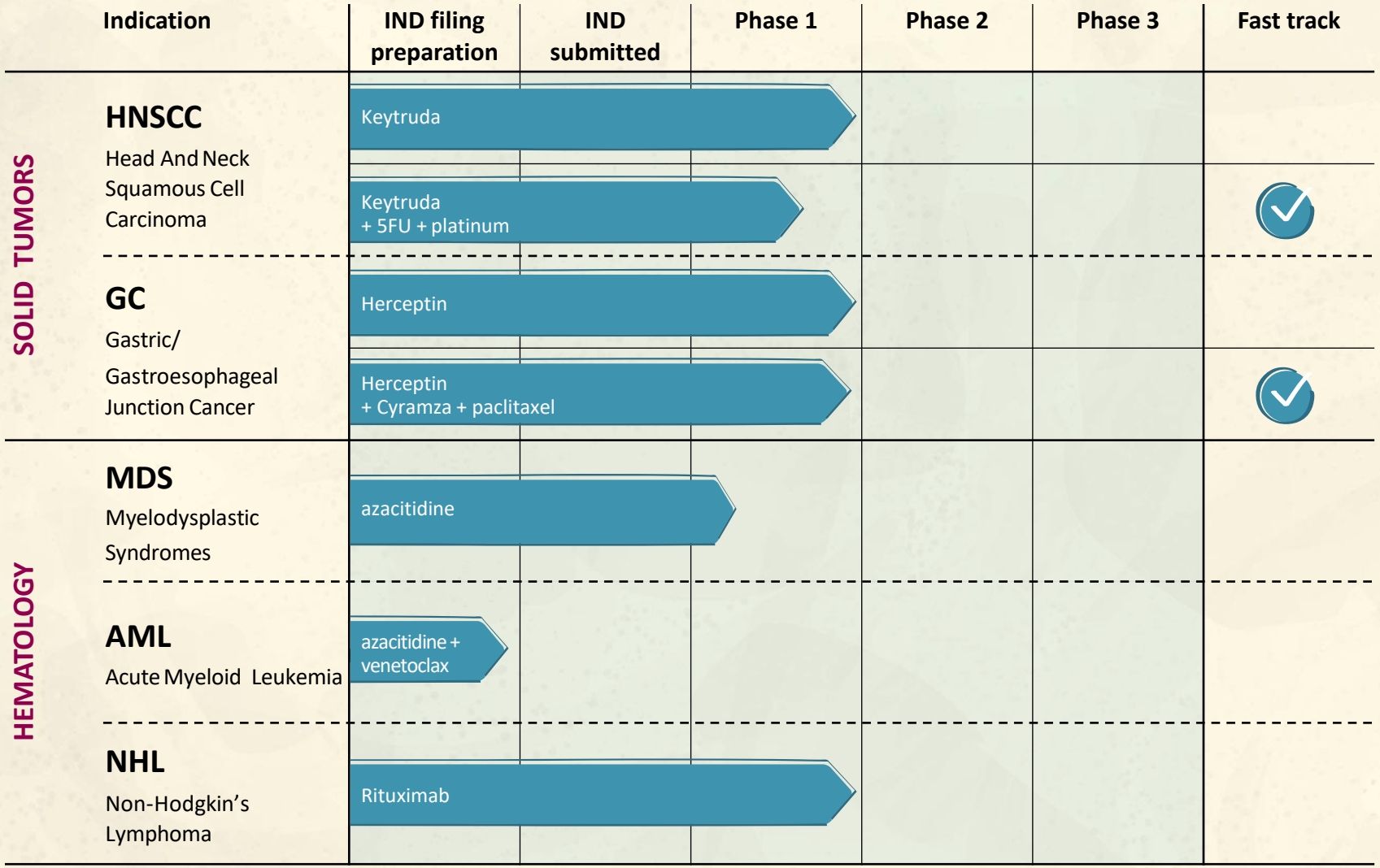
Presence of Fc domain ensures slow clearance and long half-life → **Less frequent dosing**

Designed for safety and efficacy



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

PIPELINE: COMBINATION TRIALS WITH ALX148



>150

patients dosed with ALX148 since 2017

ALX148 DEMONSTRATES CONSISTENT TOLERABILITY PROFILE

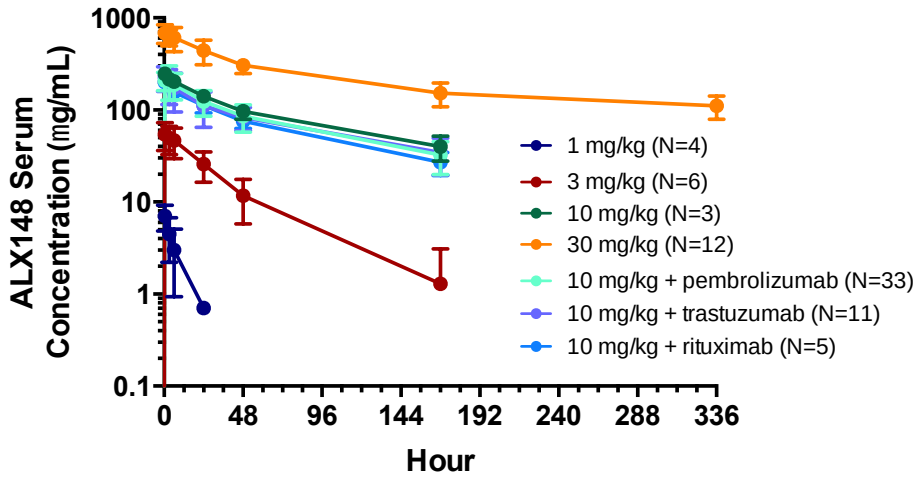
Treatment related adverse events	ALX148 + Rituxan (N=33)		ALX148 + Keytruda (N=52)		ALX148 + Herceptin (N=30)	
	Total n (%)	≥Grade 3	Total n (%)	≥Grade 3	Total n (%)	≥Grade 3
Fatigue	**3 (9.1%)**	-	**6 (11.5%)**	-	**9 (30.0%)**	-
Rash	**6 (18.2%)**	-	**5 (9.6%)**	-	-	-
AST increased	-	-	**9 (17.3%)**	-	-	-
Platelets decreased	-	-	**4 (7.7%)**	**2 (3.8%)**	**5 (16.7%)**	**2 (6.7%)**
ALT increased	-	-	**7 (13.5%)**	**1 (1.9%)**	-	-
Pruritus	-	-	**5 (9.6%)**	-	**3 (10.0%)**	-
Pyrexia	-	-	**3 (5.8%)**	-	**3 (10.0%)**	-
Decreased appetite	-	-	**2 (3.8%)**	-	**3 (10.0%)**	-
Anemia	**2 (6.1%)**	**1 (3.0%)**	**5 (9.6%)**	**1 (1.9%)**	**2 (6.7%)**	-
Infusion reaction	-	-	**4 (7.7%)**	-	-	-
Neutropenia / Neutrophil count decr	**2 (6.1%)**	**2 (6.1%)**	**2 (3.8%)**	**1 (1.9%)**	**2 (6.7%)**	**2 (6.7%)**
Nausea	**2 (6.1%)**	-	**2 (3.8%)**	-	**2 (6.7%)**	-
Alkaline phosphatase incr	-	-	**3 (5.8%)**	-	-	-
Arthralgia	-	-	**3 (5.8%)**	-	-	-
WBC decreased	-	-	**3 (5.8%)**	-	-	-
Myalgia	-	-	**2 (3.8%)**	-	-	-

Treatment related adverse events occurring in ≥2 subjects in all histologies at 10 & 15 mg/kg QW.
Data Cutoff 1 April 2020.

Tolerability profile may enable broad combination potential

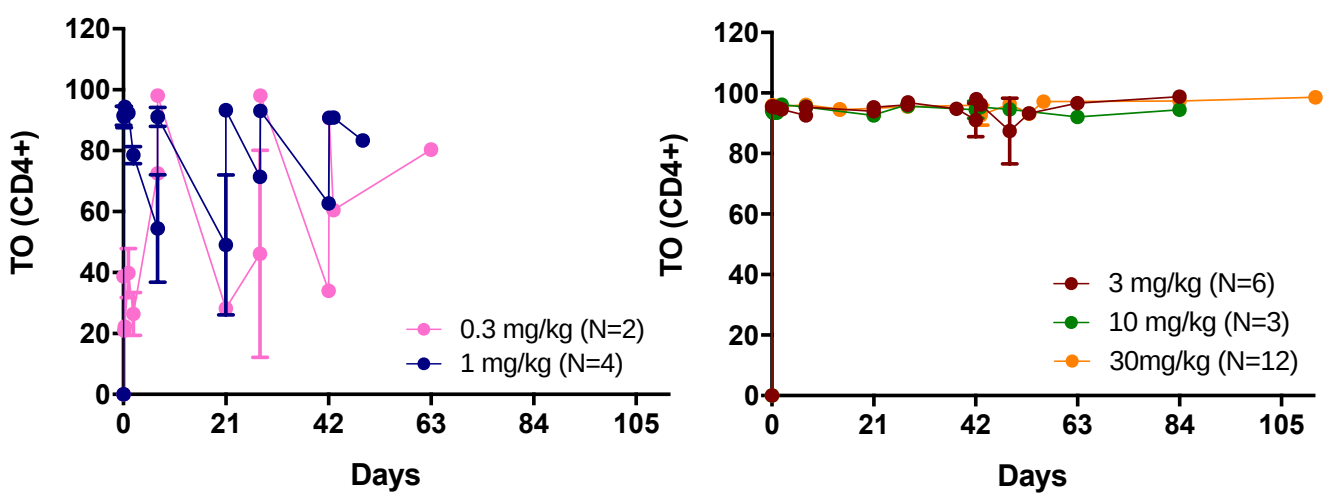
ALX148 CLINICAL PHARMACOKINETICS AND CD47 TARGET OCCUPANCY

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 (10 mg/kg QW) 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 throat

NHL PROOF-OF-PRINCIPLE TRIAL

Phase 1b NHL cohorts

Relapsed/Refractory NHL,
prior regimen with Rituxan

Treatment:

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

	10 mg/kg QW		15 mg/kg QW	
Population	N	ORR	N	ORR
All	**22**	**40.9%**	**11**	**54.6%**
Aggressive	15	33.3%	7	42.9%
Indolent	7	57.1%	4	75.0%

EHA 2020 Abstract EP1247
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

ALX148 HAS INITIAL CLINICAL ACTIVITY ACROSS TUMOR TYPES IN MULTIPLE TRIALS

Population	≥2L HER2+ GC		≥2L HER2+ GC	≥2L HNSCC (CPI-Naïve)		1L HNSCC		≥2L NHL
Combination	ALX148 + Herceptin + Cyramza + paclitaxel		ALX148 + Herceptin	ALX148 + Keytruda		ALX148 + Keytruda + 5FU + platinum		ALX148 + Rituxan
N-evaluable	9		19	10		3		33
ORR	**ALX148 66%**	Benchmark 28%	21%	**ALX148 40%**	Benchmark 15%	**ALX148 66%**	Benchmark 36%	**54.6%**
Benchmark regimen	Cyramza + paclitaxel			Single agent Keytruda		Keytruda + 5FU + platinum		

Solid tumor data as of June 30, 2020. NHL data as of April 1, 2020 EHA June 2020 Abstract EP1247. ORR = Objective Response Rate, CPI = checkpoint inhibitor. Ram/pac benchmark in GC from RAINBOW Ph3 trial (Wilke, Lancet Oncology, 2014); Keytruda single agent benchmark from KEYNOTE-40 Ph3 trial (Cohen, Lancet, 2018). Keytruda + chemo benchmark from KEYNOTE-48 Ph3 trial (Burtness, Lancet, 2019).

SUMMARY

- High affinity and no effector function is required to fully inhibit CD47 safely
- ALX148 tolerability profile enables combination with a wide range of agents
- Demonstrated clinical tolerability with anti-cancer antibodies and chemotherapy
- Clinical proof-of-principle in hematologic and solid tumors

ACKNOWLEDGEMENTS

- Stanford University scientists that originated the idea
- Current and past ALX Oncology members that brought it here
- Clinicians that believe in ALX148 potential
- Patients and their families that trust us