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**UNITED STATES  
SECURITIES AND EXCHANGE COMMISSION  
WASHINGTON, D.C. 20549**

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**FORM 8-K**

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**CURRENT REPORT  
Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934**

**Date of Report (Date of earliest event reported): June 20, 2017**

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**APTEVO THERAPEUTICS INC.**

(Exact Name of Registrant as Specified in its Charter)

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**Delaware**  
(State or Other Jurisdiction of Incorporation)

**001-37746**  
(Commission File Number)

**81-1567056**  
(IRS Employer Identification No.)

**2401 4th Avenue, Suite 1050**  
**Seattle, Washington**  
(Address of Principal Executive Offices)

**98121**  
(Zip Code)

**Registrant's telephone number, including area code: (206) 838-0500**

**Not Applicable**  
(Former Name or Former Address, if Changed Since Last Report)

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Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

- Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
- Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
- Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
- Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

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Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act.

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**Item 7.01 Regulation FD Disclosure.**

Aptevo Therapeutics Inc. (the “Company”) has prepared presentation materials, which it intends to present at the BIO International Convention held in San Diego, California. A copy of the presentation materials to be used in the presentations is attached hereto as Exhibit 99.1.

The information in this Current Report on Form 8-K, including the attached Exhibit 99.1, is being furnished and shall not be deemed “filed” for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the “Exchange Act”), or otherwise subject to the liability of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, whether made before or after the date hereof, except as expressly set forth by specific reference in such filing to this Current Report on Form 8-K.

**Item 9.01 Financial Statements and Exhibits.**

(d) Exhibits

See Exhibit Index attached hereto.

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**SIGNATURE**

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

APTEVO THERAPEUTICS INC.

Date: June 20, 2017

By: /s/ Shawnte Mitchell  
Shawnte Mitchell, Secretary, Vice  
President and General Counsel

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INDEX TO EXHIBITS

<u>Exhibit Number</u>	<u>Description</u>
99.1	Presentation of Aptevo Therapeutics Inc. dated 19-21 June 2017.

Exhibit 99.1



# Aptevo Therapeutics

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**Developing Next-Generation ADAPTIR™ Molecules  
From the Bench to the Clinic**

**BIO International  
19-21 June, 2017**

# Agenda

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- Overview of ADAPTIR Bispecific Technology
- Bispecifics in Immuno-Oncology
- Next Generation ADAPTIR Candidates
- Portfolio of Preclinical Candidates
- ADAPTIR Lead Selection Process
- ADAPTIR Platform: Key CMC Advantages
- Summary

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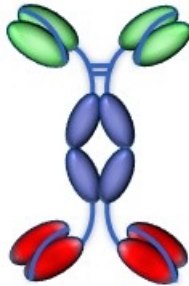
# Overview of ADAPTIR Bispecific Technology

# ADAPTIR – A Modular Technology

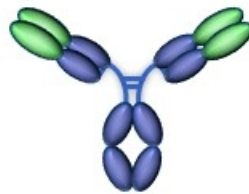
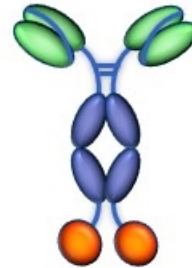
**Monospecific**



**Bispecific**



**Targeted  
Cytokine Delivery**



**mAb**

*Previously known as SMIP  
and SCORPION platforms*



# Overview of ADAPTIR Platform

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Aptevo's monospecific and bispecific antibody platform technology for novel immuno-oncology therapeutics

Robust, flexible bispecific platform fit for different mechanisms of action

- T-cell engagers; redirected T-cell cytotoxicity (RTCC)
- Directed cytokine delivery
- Co-engagement of cell receptors or soluble factors

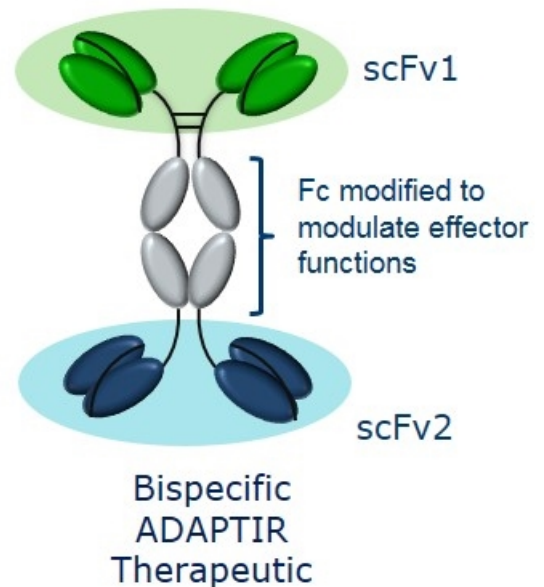
Distinct advantages over other bispecific technologies

- Homodimeric
- Antibody-like half-life (up to 12.5 days)
- Multiple binding domains or ligands can be engineered on ADAPTIR scaffold

Excellent stability and manufacturing characteristics

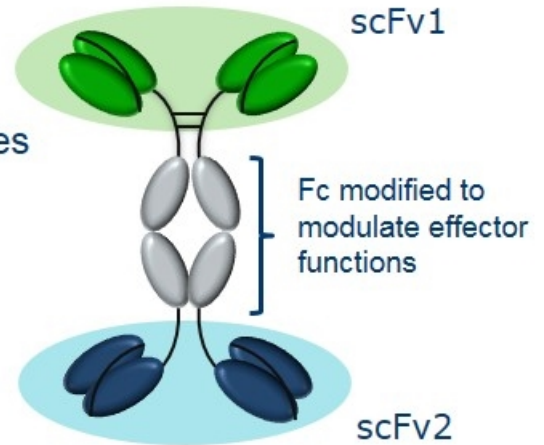
# Unique Features of ADAPTIR Bispecific Platform Technology

- Novel homodimer structure simplifies CHO production with increased manufacturing yields
  - Single gene, ease of CHO cell line production
  - Better yields and cost-of-goods than heterodimers
  - Predictable manufacturability
- Bivalent for both binding domains, improves avidity
  - Translates into improved potency
- Modular structure for versatile function
  - T-cell engagers (CD3 x tumor antigen)
  - Targeted cytokine delivery
  - Targeted activation of immune cells
  - Neutralization of soluble factors
  - Receptor blockade
  - Modified Fc to tailor Fc gamma receptor binding



# Unique Features of ADAPTIR Bispecific Platform Technology (continued)

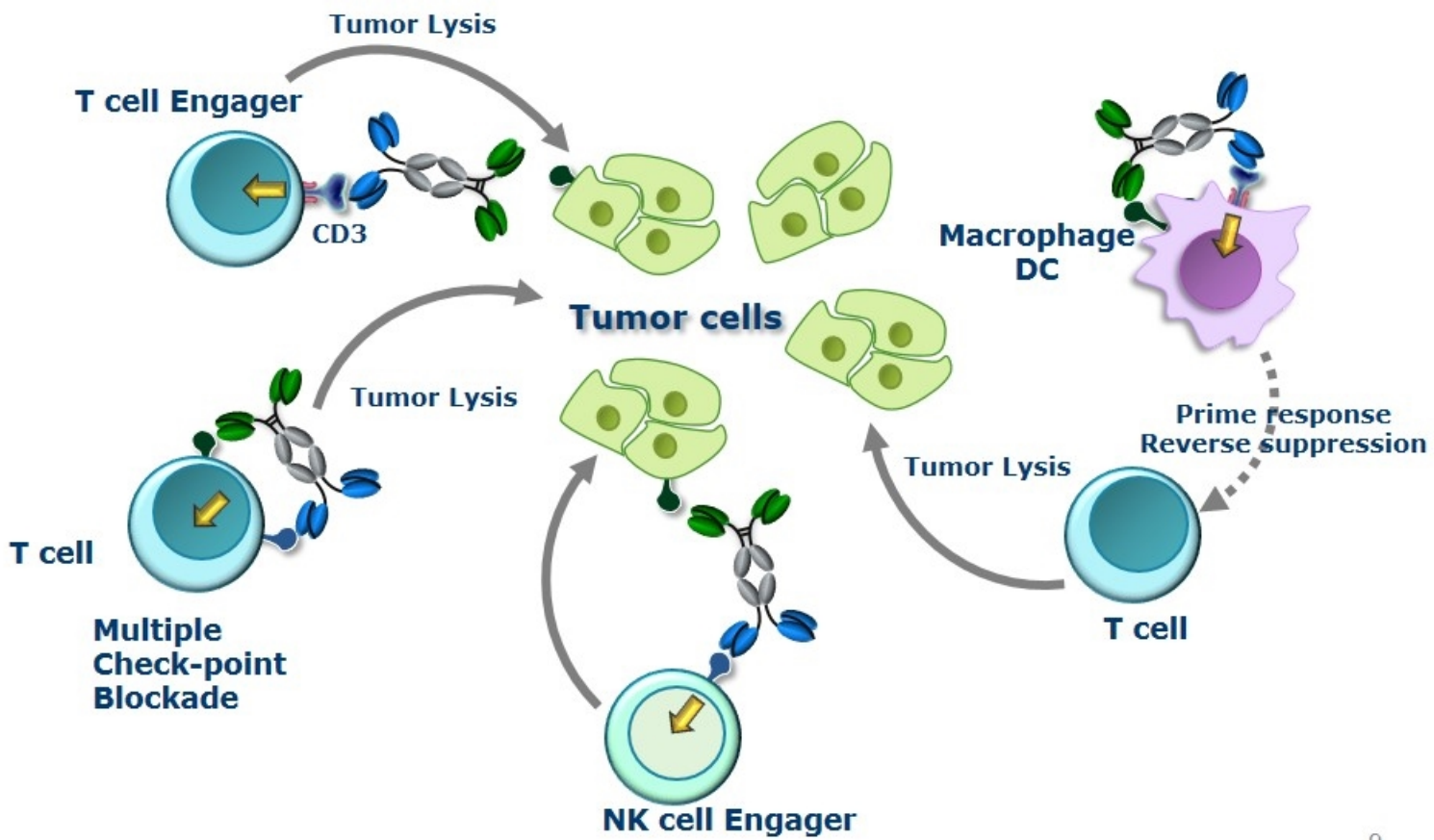
- scFv structures optimized to improve stability and manufacturability
  - Antibody-like melting temperature
  - Long-term stability
- Immunoglobulin hinge-Fc extends half-life, simplifies manufacturing
  - 12.5 days demonstrated in rodents, NHP studies in progress
  - Reproducible and robust manufacturing processes based on Fc capture



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# **ADAPTIR Bispecifics in Immuno-Oncology**

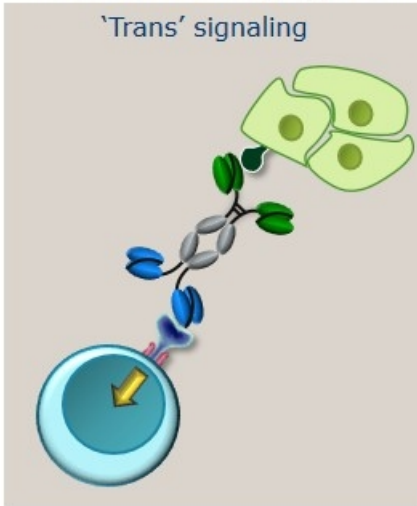
# Diversity of Opportunities to Boost Anti-Tumor Responses Using Bispecific Molecules



- Platform enables multiple modalities to stimulate T-cell function
  - Stimulation of adaptive and innate cell responses
  - Stimulation in trans or cis
  - Engagement of TCR/CD3, costimulatory receptors or cytokine receptors

## T-cell CD3 engagers

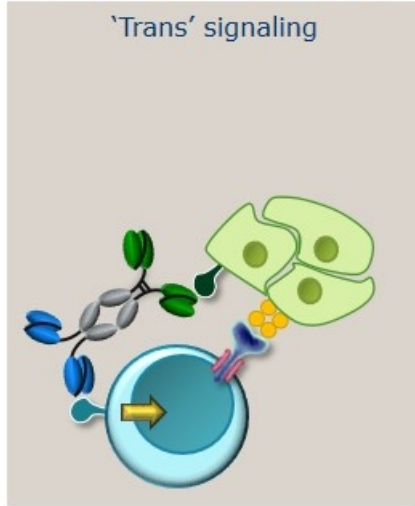
'Trans' signaling



Induces polyclonal T-cell response

## T-cell co-stimulators

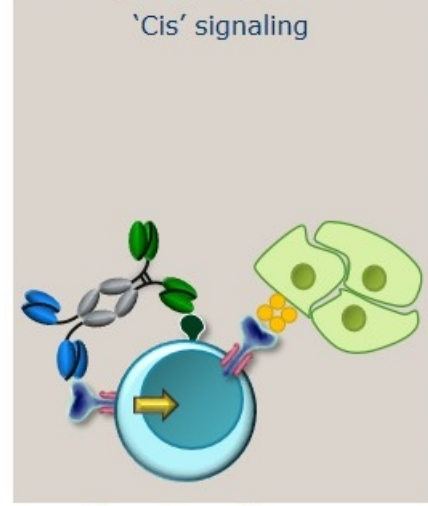
'Trans' signaling



Boosts endogenous T-cell response

## Targeted cytokines

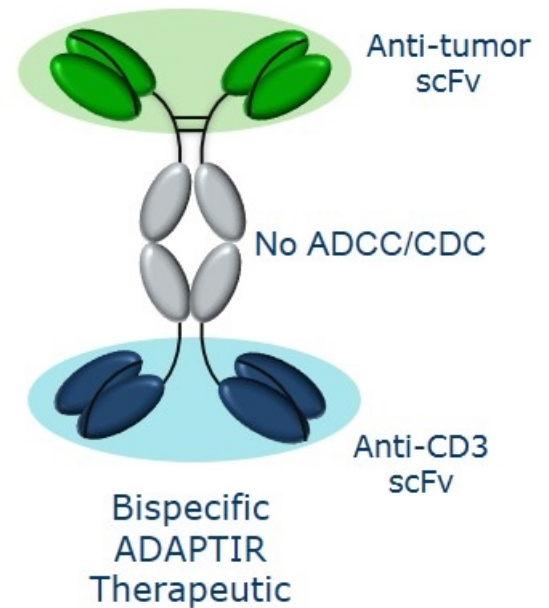
'Cis' signaling



Boosts endogenous adaptive/innate response

# Unique Features of ADAPTIR Bispecific RTCC Candidates and Selection Process

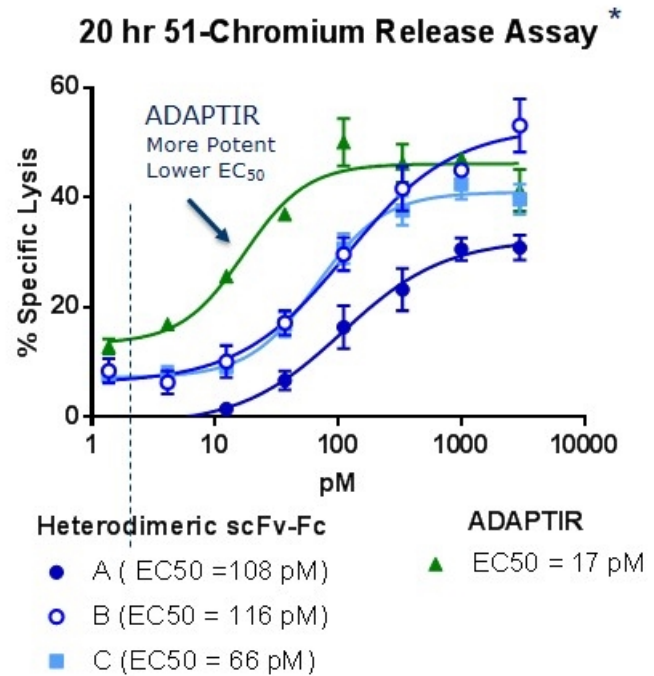
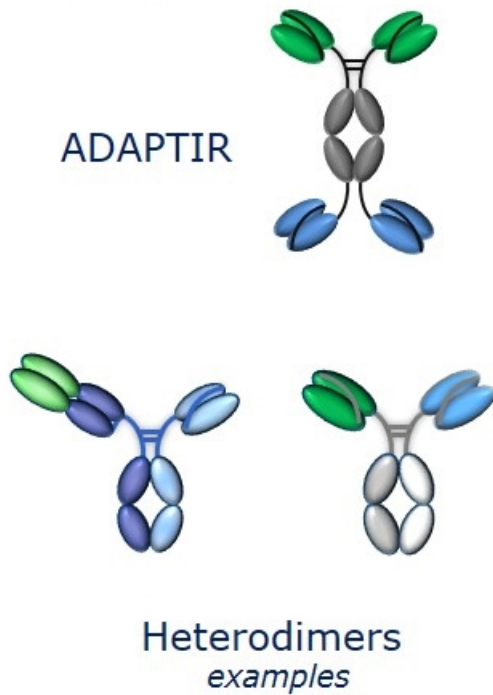
- Novel, proprietary humanized binding domain targeting CD3, cross-reactive with NHP
- Bivalent binding to target increases RTCC potency compared to monovalent bispecifics
- T-cell stimulation results in reduced cytokine release upon T-cell activation\*
- scFv optimized and selected in bispecific format to ensure good manufacturability and half-life
- State-of-the art tools used to identify and remove potential immunogenic sequences



\* MOR209/ES414, A Novel Bispecific Antibody Targeting PSMA For The Treatment of Metastatic Castration-Resistant Prostate Cancer, Hernandez-Hoyos et al. Molecular Cancer Therapeutics, July 12 2016 DOI: 10.1158/1535-7163.MCT-15-0242

# ADAPTIR: Bivalent Interaction with Target Induces more Potent RTCC than Monovalent Heterodimers

- ADAPTIR RTCC molecules have been compared to 3 heterodimer formats targeting the same tumor antigen

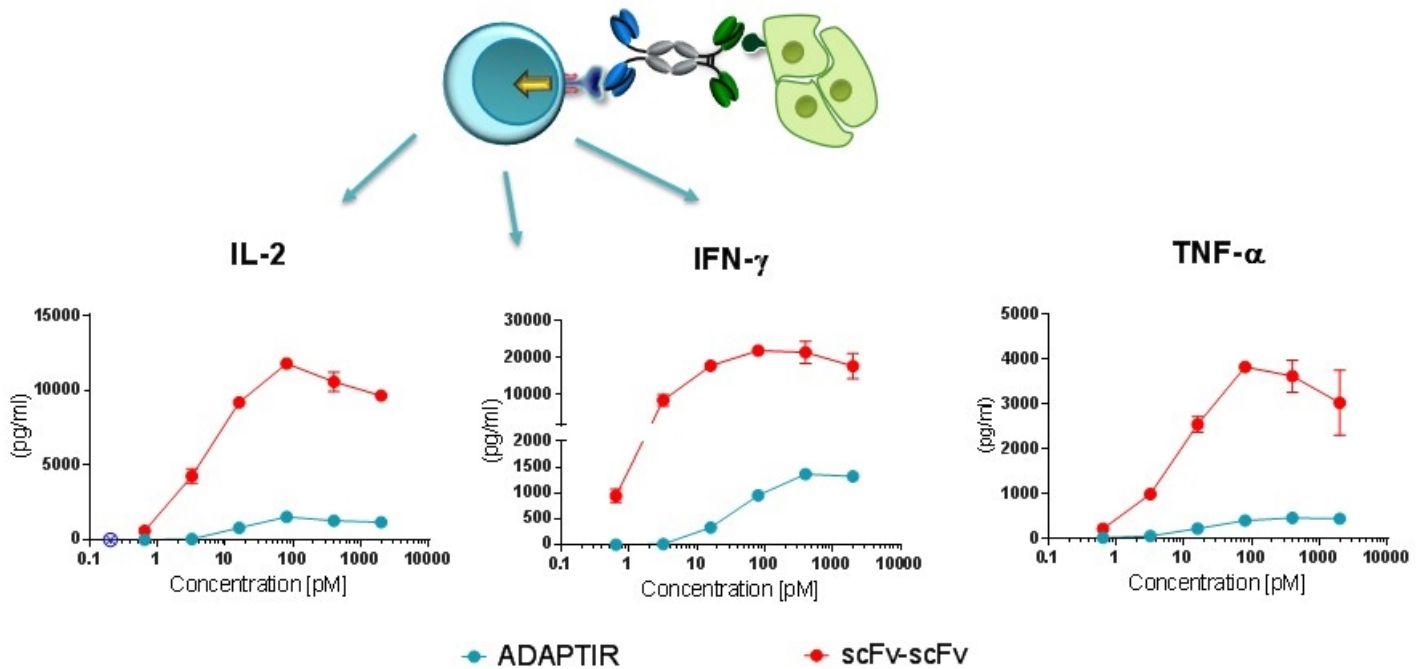


\* Unpublished in vitro preclinical studies



# ADAPTIR RTCC Candidates Induce Lower Levels of Cytokines than Competitor scFv-scFv

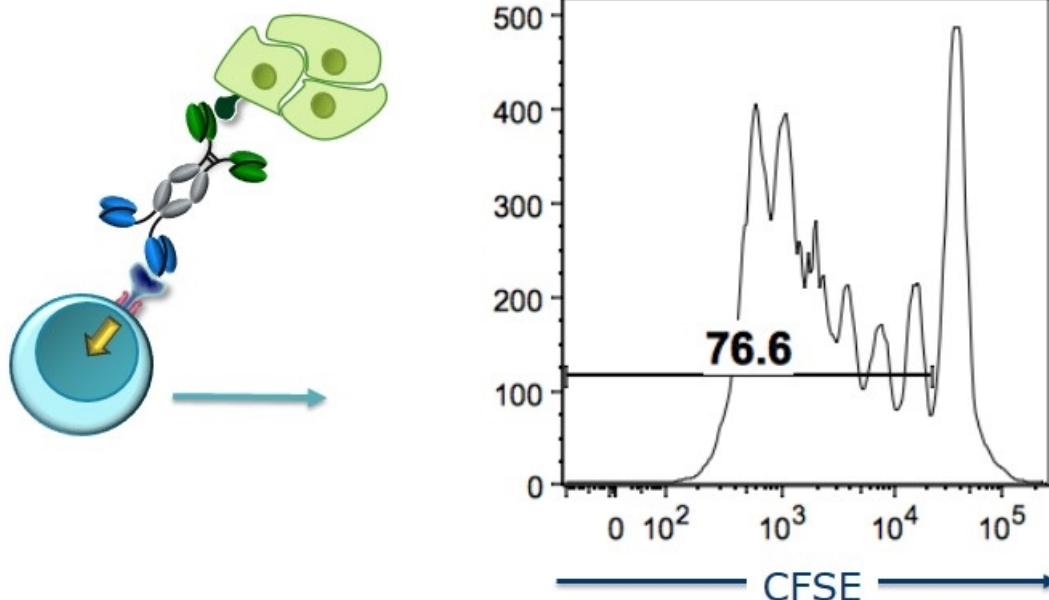
- Compared ADAPTIR to scFc-scFv targeting same tumor antigen in 20 hr activation assay



- 20 hr stimulation of T cells with ADAPTIR and tumor cells
- 3 representative cytokines shown out of 16 cytokines tested

# ADAPTIR RTCC Candidates Induce Robust Proliferation of T cells

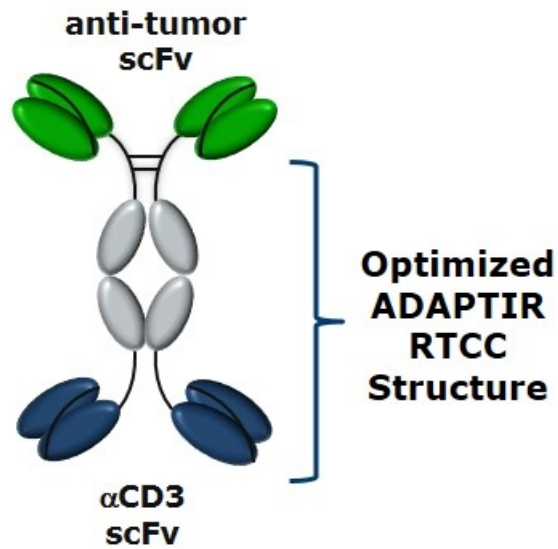
- Four-day proliferation of CD8 T cells in the presence of target cell
- T cells undergo multiple cells divisions and differentiate into effector memory cells



## Next Generation ADAPTIR Bispecifics

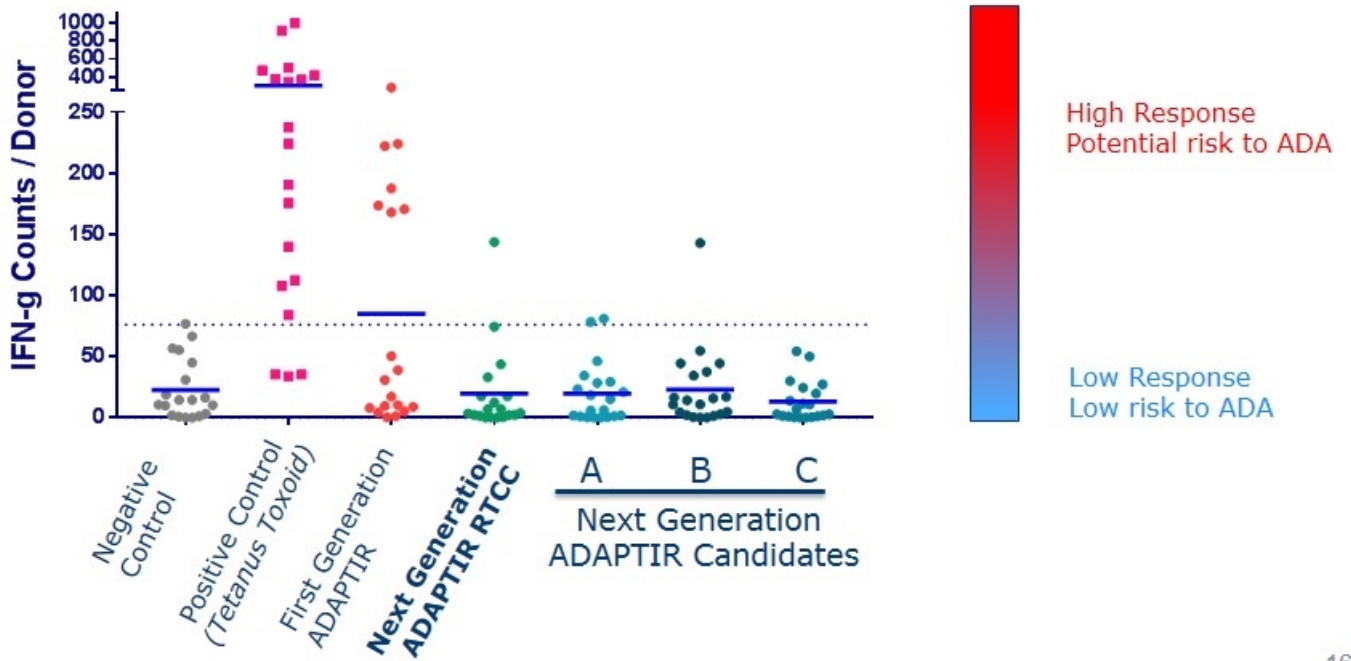
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- Next generation ADAPTIR candidates have been optimized for
  - Improved stability and improved manufacturability
  - Improved half-life; 12.5 day half-life in rodents
  - Removed potential sequences with risk to immunogenicity



# Next Generation ADAPTIR RTCC Candidates Show Improved Immunogenicity Profile – Low ADA Risk

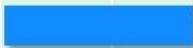

- T-cell assays performed on peptides derived from ADAPTIR RTCC platform and candidates
- IFN-gamma response measured for 20 donors with most common HLA alleles
- **Low mean response observed in Next Generation ADAPTIR candidates; comparable to negative control**



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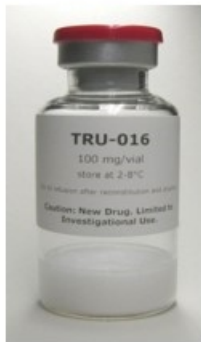
# **ADAPTIR Portfolio of Candidates**

# ADAPTIR Monospecific / Bispecific Portfolio

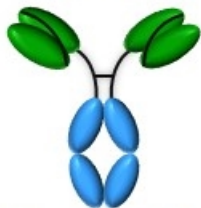
Product Candidate	Technology	Indication	Target	Pre-Clinical	Clinical Development Stage			Milestones/Highlights
					Phase I	Phase II	Phase III	
Otlertuzumab	ADAPTIR Monospecific	CLL	CD37					Executing Phase 1b combination study
MOR209 ES414*	ADAPTIR Bispecific RTCC	mCRPC Immunology	CD3/PSMA					Executing Phase 1 clinical trial
APVO436	ADAPTIR Bispecific RTCC	AML	CD3/CD123					Preclinical in vitro and in vivo POC, lead candidate
ROR1	ADAPTIR Bispecific RTCC	Hematologic and Solid Tumors	Tyrosine Kinase ROR1					Preclinical in vitro and in vivo POC, developing lead candidate
Multiple ADAPTIR Candidates	ADAPTIR Bispecific RTCC / New MOA	Hematologic and Solid Tumors	Undisclosed					Multiple RTCC candidates with novel MOA
ES210	ADAPTIR Targeted cytokine	IBD	IL10/CD86					Preclinical POC in IBD, CHO production cell line

RTCC – Redirected T-Cell Cytotoxicity = T-Cell Engager

\* Partnered with MorphoSys AG



$\alpha$ CD37 scFv



Human IgG<sub>1</sub> Fc

## Description

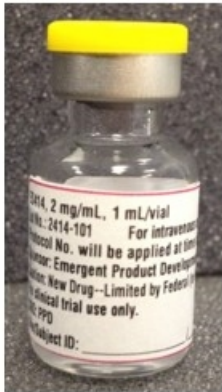
- Humanized monospecific protein therapeutic
- Targets CD37 and its signaling pathway involved in B-cell malignancies
- Built on ADAPTIR (modular protein therapeutic) platform
- Demonstrated anti-tumor activity
- Prolonged serum half-life (mouse /NHP) vs antibody fragments

## Partnering

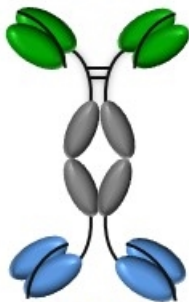
- 100% owned by Aptevo
- Actively pursuing potential partnership opportunities

## Development Status

- 253 subjects treated to date; appears safe and well tolerated
- Ongoing: Phase 2 study for chronic lymphocytic leukemia (CLL)
  - Combination with ibrutinib
  - Preliminary data read-out anticipated in H2 2017
- Multiple clinical trial data published, establishing clinical proof-of-concept
  - PHASE 2 STUDY (16201): Combination of otlertuzumab + bendamustine
  - PHASE 1b STUDY (16009): Combination of otlertuzumab + rituximab



Anti-PSMA



Anti-CD3

## Description

- Humanized bispecific protein therapeutic
- Targets PSMA and CD3, a component of the T-cell receptor
- Demonstrated redirection of T-cells to kill tumor cells expressing PSMA in vitro and in vivo
- Prolonged serum half-life (mouse/NHP) vs antibody fragments

## Partnering

- Co-development/Co-commercialization partnership with MorphoSys AG established August 2014

## Development Status

- Open-label Phase 1 continuous infusion study underway (U.S. & Australia)
- Safety, tolerability, and clinical activity endpoints
- Patients with metastatic castration-resistant prostate cancer (mCRPC)
  - Stage 1: Primary Objective – MTD; Secondary Objectives: tolerability, PK, PD, immunogenicity, cytokine response, and clinical activity
  - Stage 2: Primary Objective – Evaluate clinical activity in patients that have or have not received prior chemotherapy
- Preliminary data read-out anticipated mid-2017



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# **ADAPTIR Preclinical Candidates**

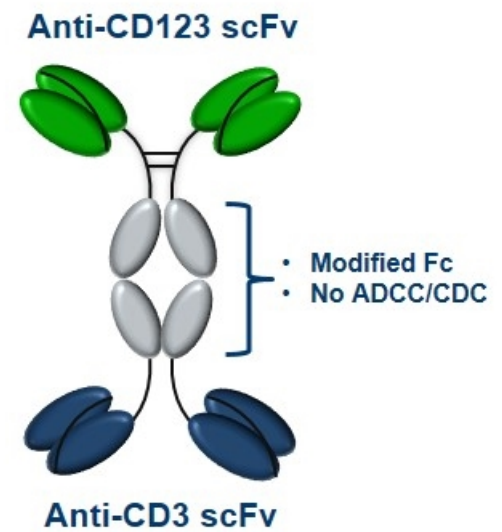
# ADAPTIR - Preclinical Pipeline

- Bispecific ADAPTIR molecules can redirect T-cell cytotoxicity against multiple tumor targets
- ADAPTIR platform can be used to generate bispecifics with novel MOA in immuno-oncology or other indications

Molecule	Target Antigen Type	Target Indication(s)	Development Activity				
			Design	<i>in vitro</i> RTCC	<i>in vivo</i> POC	IND-Enabling	Clinical: Phase 1
$\alpha$ CD123 X $\alpha$ CD3	CD123	Hematologic malignancies	[Yellow bar spanning Design, <i>in vitro</i> RTCC, <i>in vivo</i> POC, and IND-Enabling]				
$\alpha$ ROR1 X $\alpha$ CD3	Tyrosine Kinase (ROR1)	Hematologic malignancies; solid tumors	[Yellow bar spanning Design, <i>in vitro</i> RTCC, and <i>in vivo</i> POC]				
Multiple RTCC candidates	Undisclosed targets	Immuno-oncology	[Yellow bar]				
ADAPTIR with Novel MOA	Undisclosed targets	Immuno-oncology	[Yellow bar]				

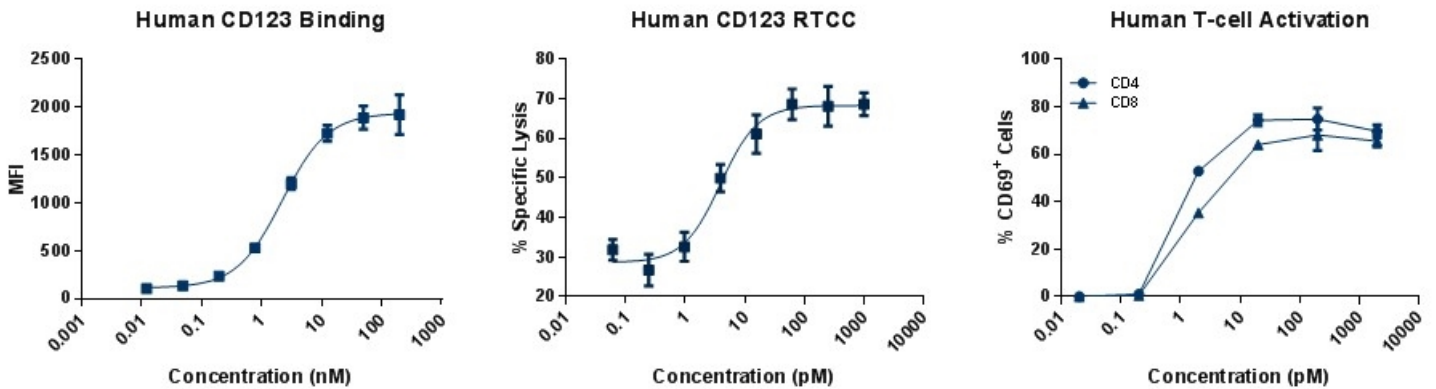
## APVO436: CD123 x CD3 Bispecific ADAPTIR

- Targets multiple hematological malignancies
  - Acute myeloid leukemia, acute lymphoblastic leukemia, hairy cell leukemia, myelodysplastic syndrome, blastic plasmacytoid dendritic cell neoplasm
- Lead and back-up candidates identified with similar *in vitro* properties
  - Binding, activation of T cells, RTCC activity
- Antibody-like half-life in Balb/c mice of >12 days
- Preclinical *in vivo* proof of concept established in xenograft tumor models
- High titer CHO cell clone production levels
- Good manufacturability attributes



# APVO436 Key In Vitro Data

- Derived from a fully human anti-CD123 binding domain generated in humanized mice
- Binds human CD123-expressing cell lines and potently induces RTCC, T-cell activation and proliferation
- Activity is dependent upon the presence of CD123<sup>+</sup> target cells
- Demonstrates good functional activity to cynomolgus CD123 and CD3

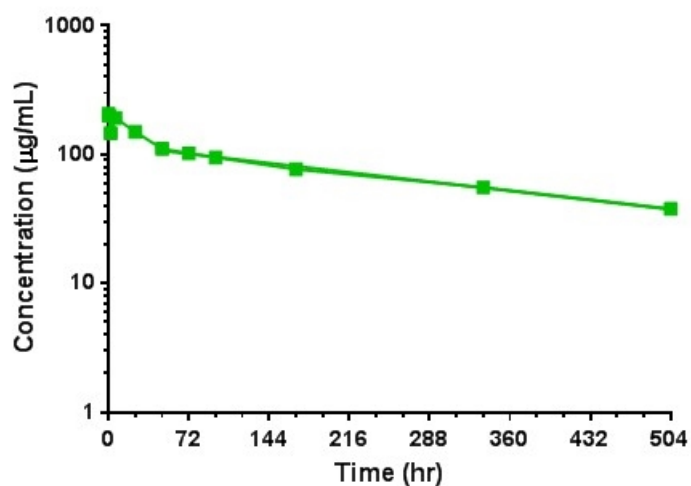


Binding (EC50)	RTCC (EC50)	T-cell Activation (EC50)	T-cell Proliferation (EC50)
2.2 nM	4.4 pM	CD4 (1.4 pM) CD8 (1.8 pM)	CD4 (1.7 pM) CD8 (3.8 pM)

# APVO436 Has Antibody-Like Half-Life in Balb/c Mice

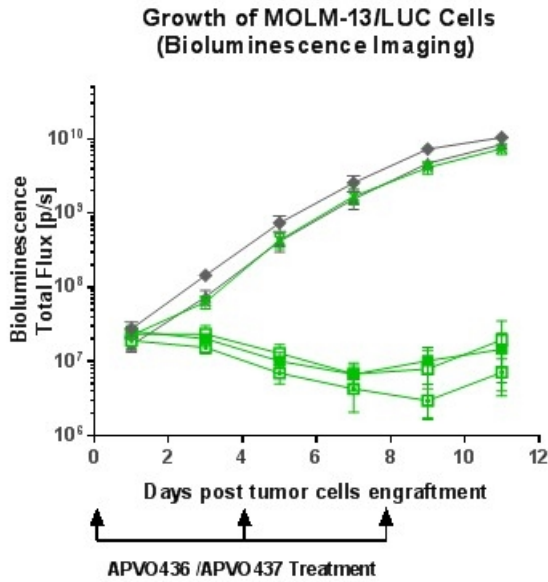
Half – life up to 12.5 days demonstrated in rodents

Serum Concentration

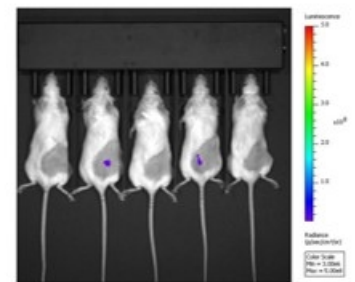
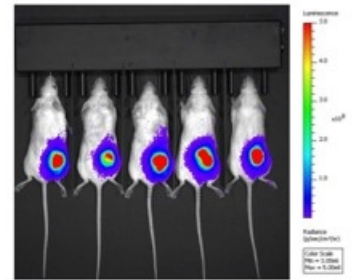


Parameter	APVO436
T <sub>1/2</sub>	301 hours (12.5 days)
Clearance	0.186 ml/hr/kg
Volume	80.84 ml/kg
AUC	37309 hr* µg/ml

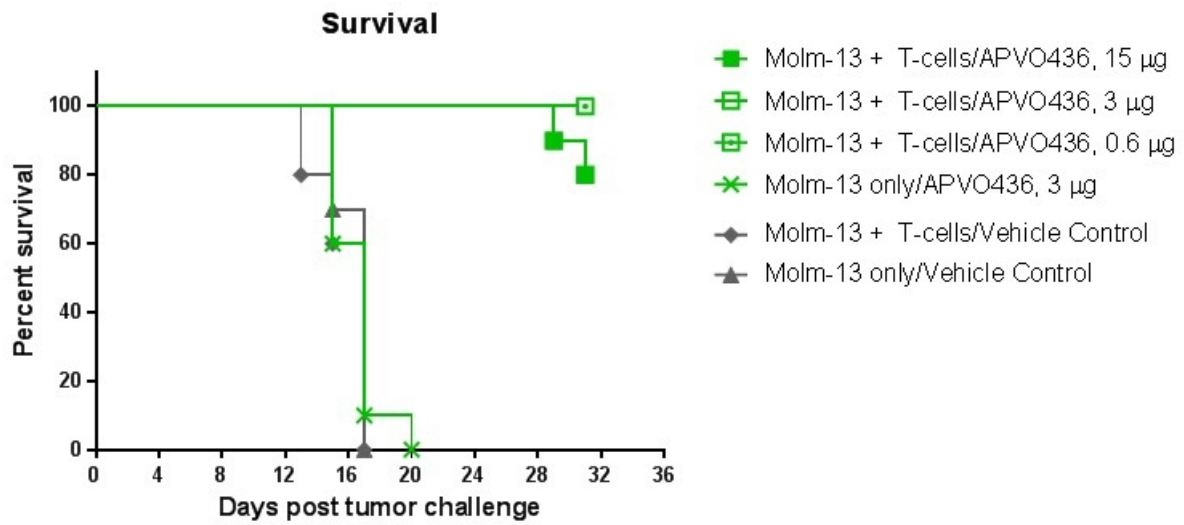
# APVO436 Inhibits Tumor Growth in Xenograft Model of Human AML



- ◆ Molm-13 + T-cells/Vehicle Control
- ▲ Molm-13 only/Vehicle Control
- Molm-13 + T-cells/APVO436, 15 µg
- Molm-13 + T-cells/APVO436, 3 µg
- ◇ Molm-13 + T-cells/APVO436, 0.6 µg
- ✱ Molm-13 only/APVO436, 3 µg



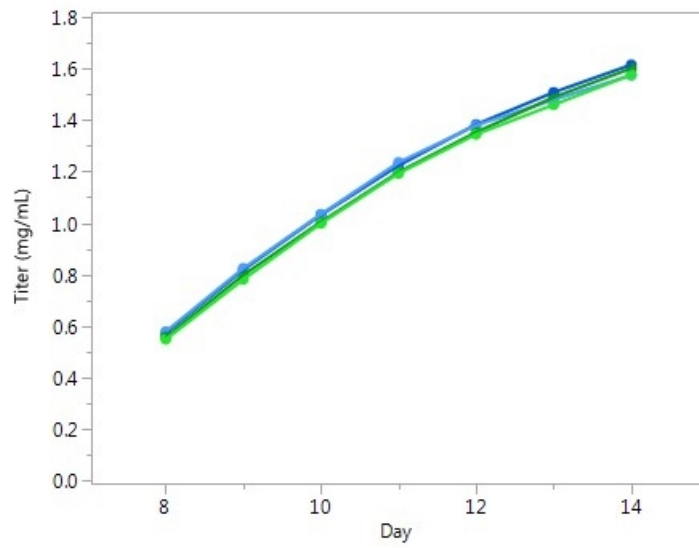
# APVO436 Prolongs Survival in Xenograft Model of Human AML



Treatment Group	Median Survival Time (Days)	Survival Relative to Molm-13 + T cells/Vehicle Control (P value)
Molm-13 + T cells/Vehicle	17	(-)
Molm-13 + T cells/APVO346 15 µg	Undefined	<0.0001
Molm-13 + T cells/APVO346 3 µg	Undefined	<0.0001
Molm-13 + T cells/APVO346 0.6 µg	Undefined	<0.0001
Molm-13 only/Vehicle	17	0.5136
Molm-13 only/APVO346	17	0.0772

## Antibody-like expression levels of APVO436 in CHO Production Cell Lines

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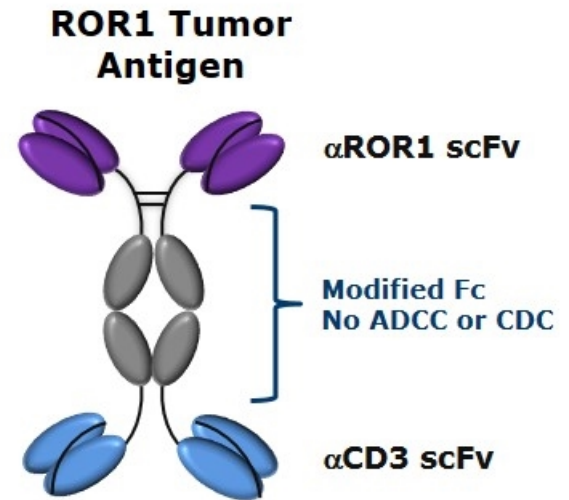


- Reproducible production of more than 1.5 g/L of APVO436 in 10-L cultures



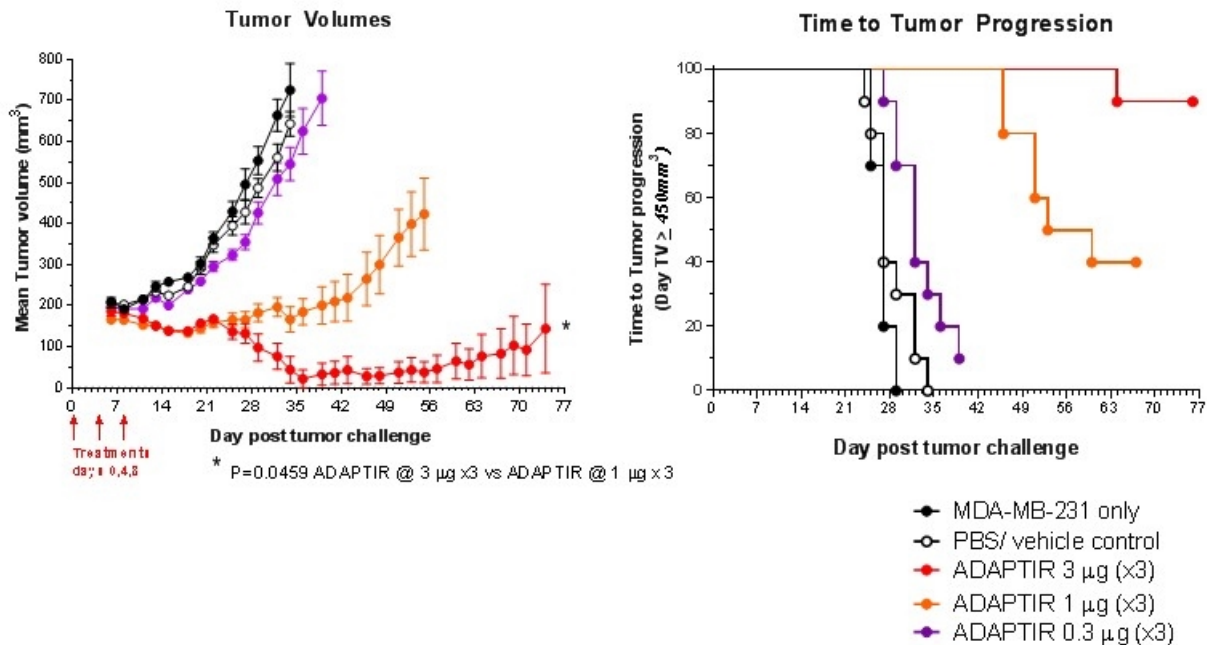
# $\alpha$ ROR1 x $\alpha$ CD3 ADAPTIR Pilot Candidate: Summary

- Novel therapeutic that redirects T cells to kill ROR1-expressing tumor cells
- Active in *in vitro* and *in vivo* studies at very low concentrations
- Competitive with other bispecific antibody formats
  - Prolonged serum half-life in rodents
- NHP cross-reactive
- Targeting clinical development in multiple oncology indications



# $\alpha$ ROR1 x $\alpha$ CD3 Delays Tumor Growth and Improves Survival in a Xenograft Model

- Statistically significant delay of tumor growth in and increase in overall survival in MDA-MB-231 subcutaneous xenograft model
- 8/10 mice at top dose (3  $\mu$ g x3) tumor free at end of study



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# **ADAPTIR Platform Lead Selection Process**

# ADAPTIR Candidates are Screened to Meet Detailed Product Profile Criteria

**Activity and affinity consistent with desired mechanism of action**

**Stable under manufacturing, storage and dosing conditions**



**Stable in serum, no off-target binding, low immunogenicity potential**

# Screening and Optimization of Stable scFvs Enables mAb-like Performance

- scFv domains obtained from human phage library to leverage stable IgG frameworks, or humanized rodents to leverage high affinity human binding domains
- High throughput screening and characterization assays are performed in ADAPTIR format
- scFv must achieve  $T_m > 60$  °C without reliance on additional stabilizing disulfides
- Example of 8 ADAPTIR candidates selected against a single antigen shows multiple scFvs with desired stability criteria:

Molecule	scFv1 $T_m$ , °C	Molecule	scFv1 $T_m$ , °C
ADAPTIR "M"	70	ADAPTIR "R"	73
ADAPTIR "N"	79	ADAPTIR "S"	70
ADAPTIR "O"	64	ADAPTIR "T"	69
ADAPTIR "P"	65	ADAPTIR "U"	74

# ADAPTIR Bispecifics Utilize Modified Fc to Eliminate ADCC and CDC Function

- Thermostability and FcRn binding similar to wt IgG1 Fc

	Molecule	CH2 Tm (°C)	CH3 Tm (°C)	Functions
<b>Thermal Stability</b>	IgG1 Fc WT	70	82	ADCC/CDC <u>capable</u>
	ADAPTIR IgG1 Fc	68	82	ADCC/CDC <u>null</u>

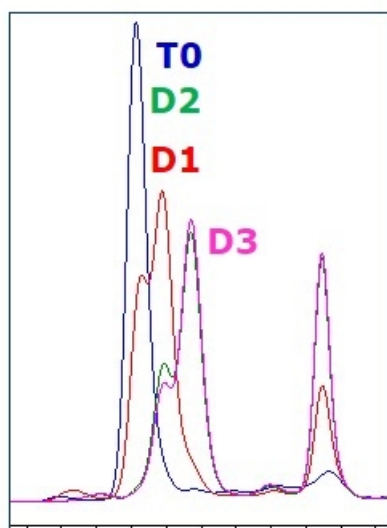
	Molecule	IgG subtype	Molecule Type	KD by SPR (nM)
<b>FcRn Affinity</b>	scFv-Fc-scFv	ADAPTIR IgG1 Fc (ADCC and CDC null)	ADAPTIR bispecific	26
	trastuzumab	IgG1 WT	MAb	64
	etanercept	IgG1 WT	ECD fusion	550

# Standardized Approach for ADAPTIR Candidate Evaluation

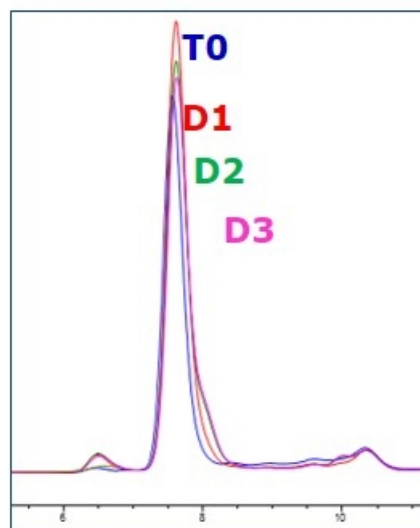
scFvs are screened for functional, conformational and colloidal stability

Category	Assays
Thermostability	DSC (T <sub>m</sub> )
	DSF (T <sub>m</sub> , T <sub>agg</sub> )
Solubility	High-salt solubility screen
	Protein concentration screen
Process compatibility	Process intermediate stability
	Shear stress assessment
Storage stability	Stability at multiple pH, temp, [protein] and platform formulation conditions
Sequence liabilities	PTM prediction and evaluation by MS
	Spatial Aggregation Propensity (SAP) Analyses
Specificity, biological stability	Target binding affinity
	Non-specific binding screens (serum and cell surface)
	Serum stability (binding and function)
	Protease susceptibility

# ADAPTIR Candidates are Screened for Cleavage Susceptibility at Domain Junctions



**Construct "A"**



**Construct "B"**

- New candidates are selected to be resistant to proteolytic cleavage to minimize degradation products during expression and *in vivo* use
- Eliminates need for specific purification step to eliminate LMW contaminants

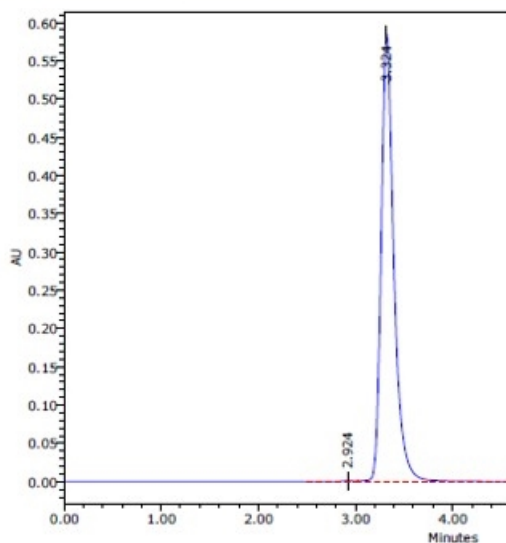


## ADAPTIR Molecules Achieve both Functional and Stability Objectives

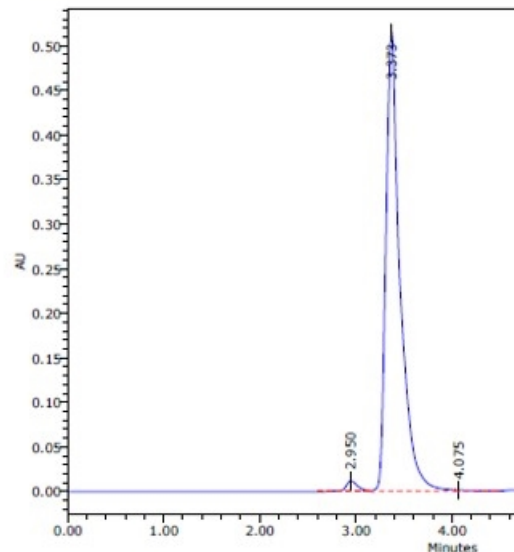
ID	%Protein Loss, Salt Spike	Target Binding KD (nM)	scFv Tm (DSC)	In vitro Activity EC-50 (pM)
Candidate A (Lead)	0	3	65	4
Candidate B	0	176	74	5.6
Candidate C	-95	78	73	22.5

- Standardized process for identifying stable, active constructs
- Research, process, analytical and formulation development teams review data and select clinical lead construct

# ADAPTIR Molecules Are Stable Under Accelerated Stability Conditions (40 °C)



T=0: 0.21% HMW



T=7days at 40°C; 1.9% HMW

- Careful selection of lead facilitates subsequent development and cGMP manufacturing
- Enables rapid progression of candidates to clinic

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# ADAPTIR Platform

## Key CMC Advantages

## EXPERIENCE

- 8 GMP lots across multiple molecules
- History of successful transfer to CMOs

## UPSTREAM

- Standard CHO cell line
- Titers > 1 g/L achieved for early-phase processes
- Successfully scaled to 2,000 L
- Standard commercially-available defined media
- Fed-batch processes with standard production bioreactor residence times

## DOWNSTREAM

- Three chromatography steps, mAb-like capture
- mAb-like viral inactivation and viral filtration
- Purification yields comparable to mAbs

## ANALYTICAL

- Standard mAb assays for:
  - Characterization
  - GMP release
  - Stability testing

## FORMULATION

- Standard excipients
- Frozen DP for early phase clinical
- Currently use lyophilized DP for late phase clinical (>2yr stability)

## EXPERIENCE

8 GMP lots across multiple molecules

## UPSTREAM

- Standard CHO cell line

**Titers > 1 g/L achieved for early-phase processes  
Successfully scaled to 2,000 L**

- Fed-batch processes with standard production bioreactor residence times

## DOWNSTREAM

- Three chromatography steps, mAb-like capture

**Purification yields comparable to mAbs**

## ANALYTICAL

- Standard mAb assays for:
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  - GMP release
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## FORMULATION

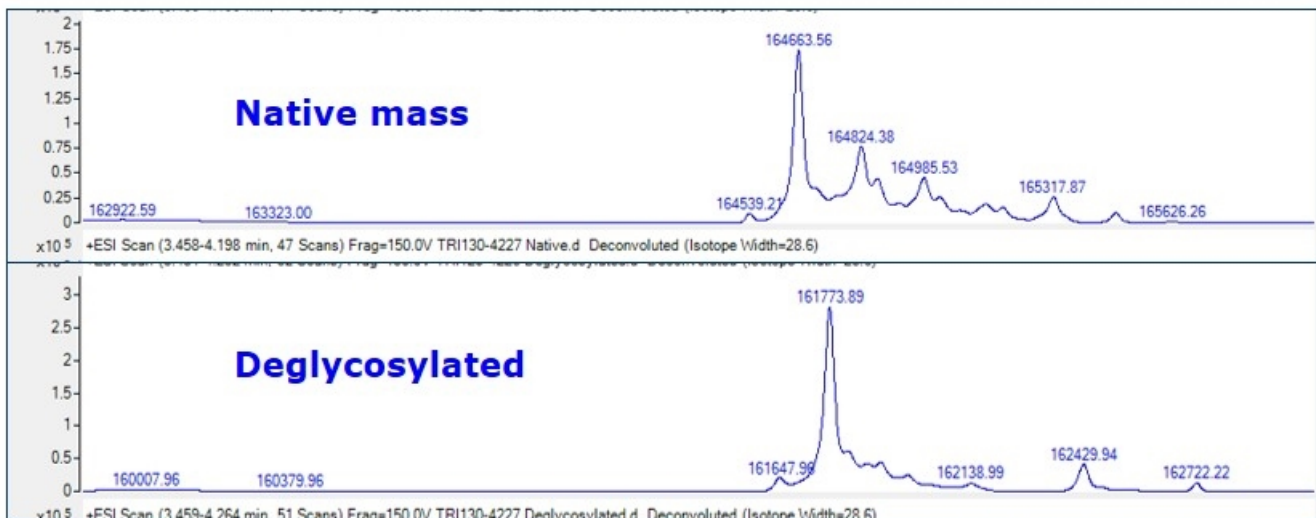
- Standard excipients
- Frozen DP for early phase clinical
- Currently use lyophilized DP for late phase clinical (>2yr stability)

# ADAPTIR Expression Levels Meet Clinical and Commercial Demands

ADAPTIR Candidate Examples	ADAPTIR Structure	Titer (g/L)
scFv-Fc	Mono specific	~ 2.0
scFv-Fc-cytokine	Cytokine Delivery	~ 1.4
scFv-Fc-scFv	Bispecific	~ 1.5

- Cell-culture titers are typically greater than 1 g/L prior to process optimization
- Cell-culture titers greater than 2 g/L have been achieved after process optimization
- ADAPTIR proteins are produced at levels that easily meet clinical & commercial demands

# ADAPTIR Proteins are Well-Characterized Protein Therapeutics



- Standard mAb-like analytical techniques are used for characterization and release testing
- LC-MS analyses of ADAPTIR candidates show:
  - Proper disulfide bond formation
  - Glycosylation pattern consistent with CHO-expressed mAbs

# Rapid PD from “Lead to Clinic”

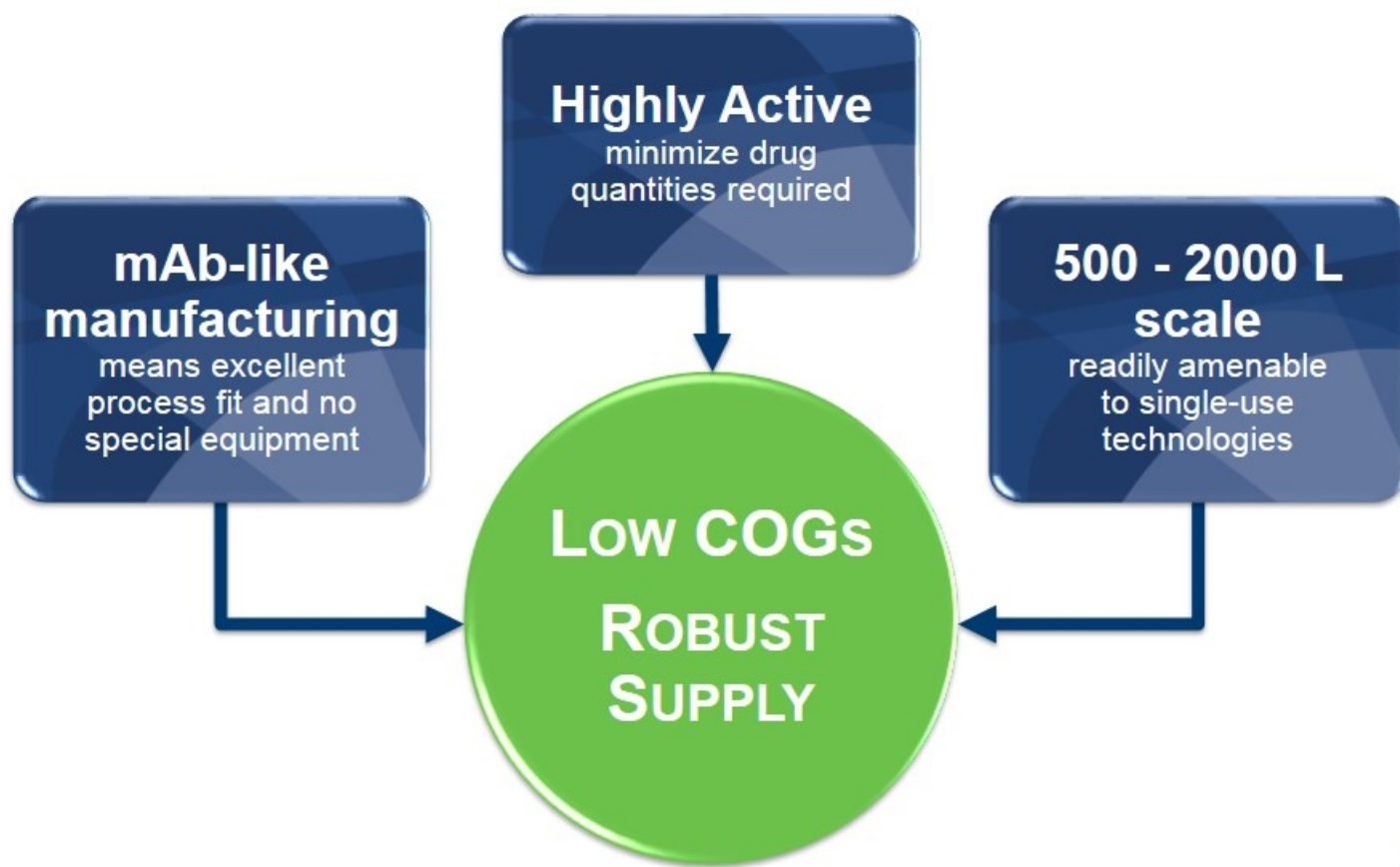
- Aptevo employs a *process & analytical development platform* enabling rapid transfer from research to clinical development

**Lead to Clinic  
in 18 months**





# Highly Favorable Supply Economics



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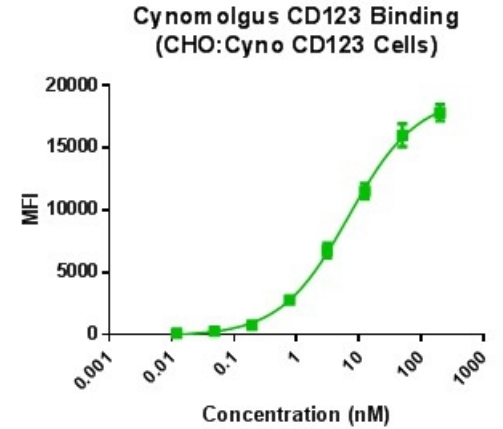
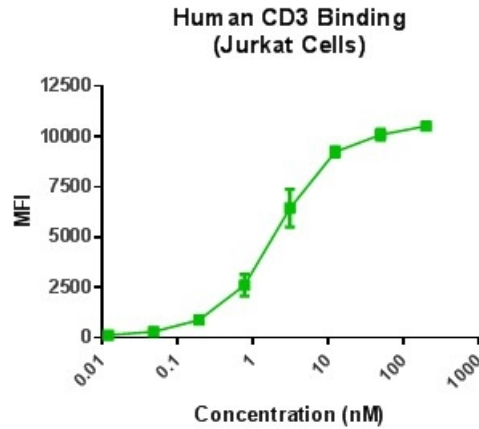
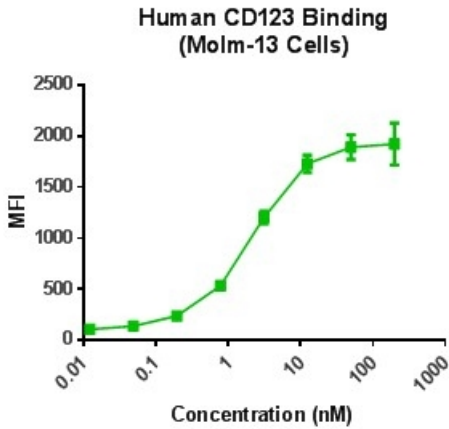
# ADAPTIR Summary

- ADAPTIR is Aptevo's monospecific and bispecific antibody platform technology for generating novel immuno-oncology therapeutics
- ADAPTIR is a robust, flexible platform that can be used to generate bispecific molecules with different mechanisms of action
- ADAPTIR platform has distinct advantages over other bispecific technologies and therapeutic approaches
- ADAPTIR therapeutics have overcome many challenges facing other bispecific strategies
  - Excellent stability, half-life and manufacturing characteristics
  - Ability to reproducibly generate potent molecules with different modes of action, that modulate the immune response to tumors
  - Antibody-like half-life allows for improved dosing protocols

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# Back-up

# APVO436 - ADAPTIR Lead Candidate Binds CD123 and CD3 with High Affinity



## Binding Affinity by Flow cytometry

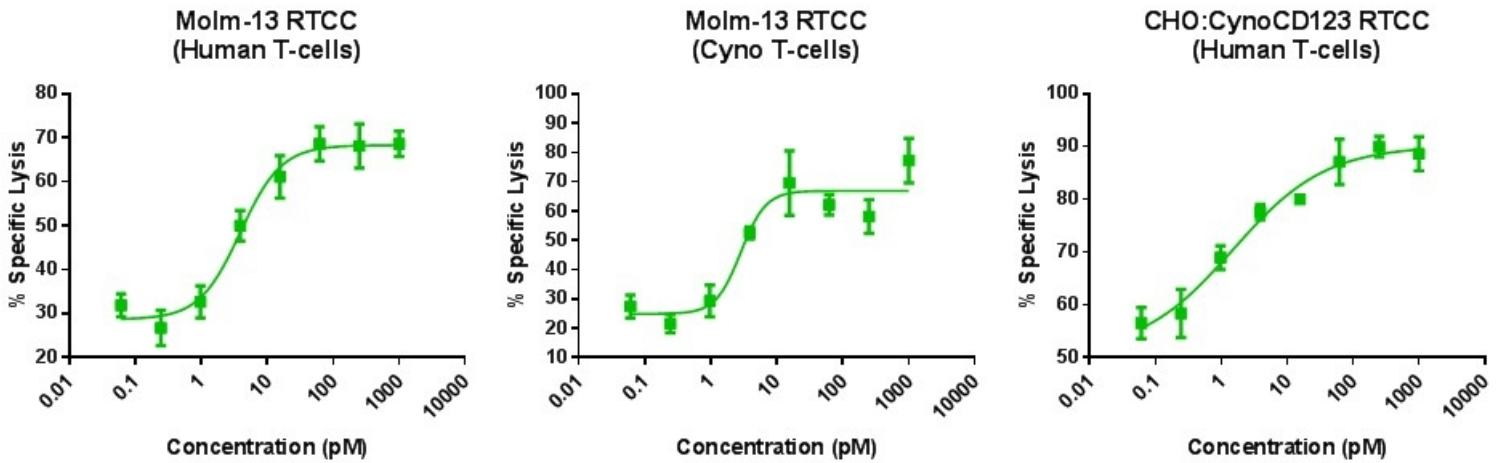
Molm-13 (Human CD123)	Jurkat (Human CD3)	CHO Cyno CD123
2nM	2nM	7nM

## Affinity to human CD123 by Biacore

K <sub>a</sub> (1/Ms)	K <sub>d</sub> (1/s)	K <sub>D</sub> (nM)
1.6 x 10 <sup>5</sup>	3.7 x 10 <sup>4</sup>	2

# APVO436 Induces Redirected T-Cell Cytotoxicity (RTCC) of CD123+ Tumors

RTCC activity demonstrated using both Human and Cynomolgus T cells targeting Molm-3, a CD123+ tumor cell line

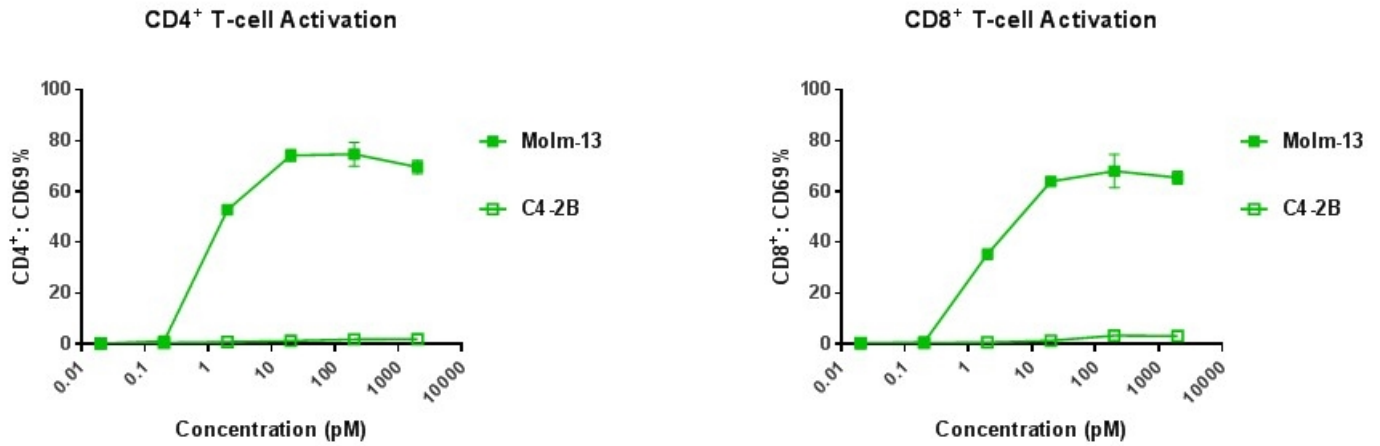


EC<sub>50</sub> values in RTCC assays

Molm-13 (Hu T cells)	Molm-13 (Cyno T cells)	CHO/Cyno CD123 (Hu T cells)
4 pM	2 pM	3 pM

# APVO436 Induces Target Dependent T-cell Activation

APVO436 Induces T cell Activation of CD4 and CD8 T cells and Induces Proliferation of Both T-cell subsets (data not shown)

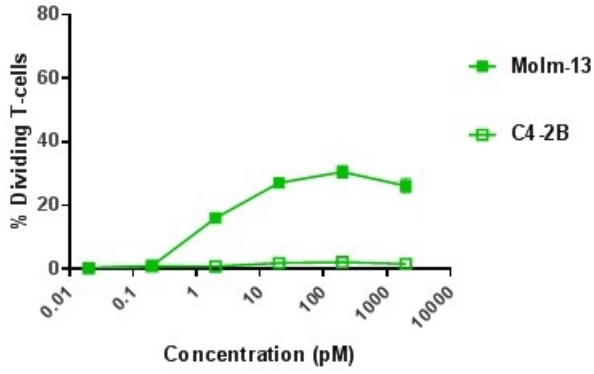


EC<sub>50</sub> values in activation assays

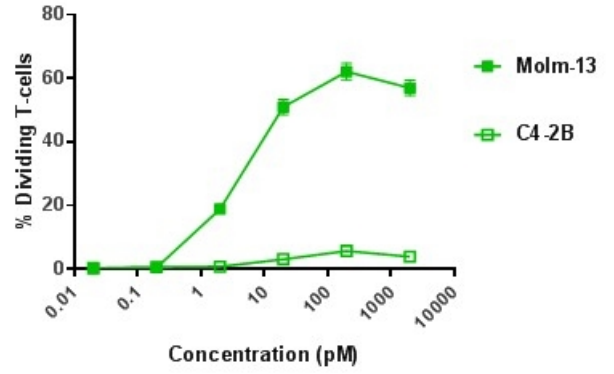
CD4+ T cells	CD8+ T cells
1 pM	2 pM

# APVO436 Induces Target Dependent T-cell Proliferation

CD4<sup>+</sup> T-cell Proliferation



CD8<sup>+</sup> T-cell Proliferation



EC<sub>50</sub> values in activation assays

CD4 <sup>+</sup> T cells	CD8 <sup>+</sup> T cells
2 pM	2 pM



# APVO436 Inhibits Tumor Growth in Xenograft Model of Human AML

