

Advancing Antibody Drug Conjugates with Novel Payloads Targeting RNA Splicing

AKARI THERAPEUTICS

Corporate Presentation
November 2026

NASDAQ: AKTX
akaritx.com

Forward-Looking Statements

This presentation includes expressed or implied forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended (the Exchange Act), about Akari Therapeutics, Plc (the "Company") that involve risks and uncertainties relating to future events and the future performance of the Company. Actual events or results may differ materially from these forward-looking statements. Words such as "will," "could," "would," "should," "expect," "plan," "anticipate," "intend," "believe," "estimate," "predict," "project," "potential," "continue," "future," "opportunity," "will likely result," "target," variations of such words, and similar expressions or negatives of these words are intended to identify such forward-looking statements, although not all forward-looking statements contain these identifying words. Examples of such forward-looking statements include, but are not limited to, express or implied statements regarding: the business combination and related matters, including, but not limited to, post-closing operations and the outlook for the Company's business; the Company's targets, plans, objectives or goals for future operations, including those related to its product candidates; financial projections; future economic performance; and the assumptions underlying or relating to such statements. These statements are based on the Company's current plans, estimates and projections. By their very nature, forward-looking statements involve inherent risks and uncertainties, both general and specific. A number of important factors, including those described in this communication, could cause actual results to differ materially from those contemplated in any forward-looking statements. Factors that may affect future results and may cause these forward-looking statements to be inaccurate include, without limitation: the risk that the Company may not realize the anticipated benefits of its merger with Peak Bio, Inc. (the "Merger") in the time frame expected, or at all; the ability to retain and hire key personnel; potential adverse reactions or changes to business relationships resulting from the Merger; the potential impact of unforeseen liabilities, future capital expenditures, revenues, costs, expenses, earnings, synergies, economic performance, indebtedness, financial condition and losses on the future prospects, business and management strategies for the management, expansion and growth of the combined business; uncertainties as to the long-term value of the Company's American Depositary Shares ("ADSs") (and the ordinary shares represented thereby), including the dilution caused by the Company's issuance of additional ADSs (and the ordinary shares represented thereby) in connection with the Merger; risks related to global as well as local political and economic conditions, including interest rate and currency exchange rate fluctuations; potential delays or failures related to research and/or development of the Company's programs or product candidates; risks related to any loss of the Company's patents or other intellectual property rights; any interruptions of the supply chain for raw materials or manufacturing for the Company's product candidates, the nature, timing, cost and possible success and therapeutic applications of product candidates being developed by the Company and/or its collaborators or licensees; the extent to which the results from the research and development programs conducted by the Company, and/or its collaborators or licensees may be replicated in other studies and/or lead to advancement of product candidates to clinical trials, therapeutic applications, or regulatory approval; uncertainty of the utilization, market acceptance, and commercial success of the Company's product candidates; unexpected breaches or terminations with respect to the Company's material contracts or arrangements; risks related to competition for the Company's product candidates; the Company's ability to successfully develop or commercialize its product candidates; potential exposure to legal proceedings and investigations; risks related to changes in governmental laws and related interpretation thereof, including on reimbursement, intellectual property protection and regulatory controls on testing, approval, manufacturing, development or commercialization of any of the Company's product candidates; the Company's ability to maintain listing of its ADSs on the Nasdaq Capital Market. While the foregoing list of factors presented here is considered representative, no list should be considered to be a complete statement of all potential risks and uncertainties. More detailed information about the Company and the risk factors that may affect the realization of forward-looking statements is set forth in the Company's filings with the SEC, copies of which may be obtained from the SEC's website at www.sec.gov. The Company assumes no, and hereby disclaims any, obligation to update the forward-looking statements contained in this presentation.

Akari: Driving Novel ADC Payload Innovation to Address Market Needs and Create Near-Term Value Acceleration

1

ADC Payload Innovation

- Urgent need for new payloads:
  - PH1: Targeting RNA splicing through disrupting the spliceosome
- PH1 is a potent cytotoxic and immuno-therapeutic payload
- PH1 designed to circumvent ADC payload resistance mechanisms
- PH1 has potential for unique synergy with:
  - Anti-PD1/PD-L1 therapies
  - Topo1 Inh. payloads (dual)

2

Internal Development

- AKTX-101: TROP2; IND enabling activities, Phase 1 mid-2027
- AKTX-102: CEACAM5, Preclinical

External / Partner Opportunities

- Collaboration with Whitehawk Therapeutics to evaluate PH1/TOP1 dual ADC payloads
- Market comps show strong interest in novel ADC technologies:
  - Acquisitions of Myricx, Tubulis

3

Near-Term Value

- AKTX-101: Near-Term IND/Phase 1 Trial
- Pipeline Expansion:
  - CEACAM5, others
- Collaborations → Partnerships

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PH1 Payload Powers a Growing ADC Pipeline

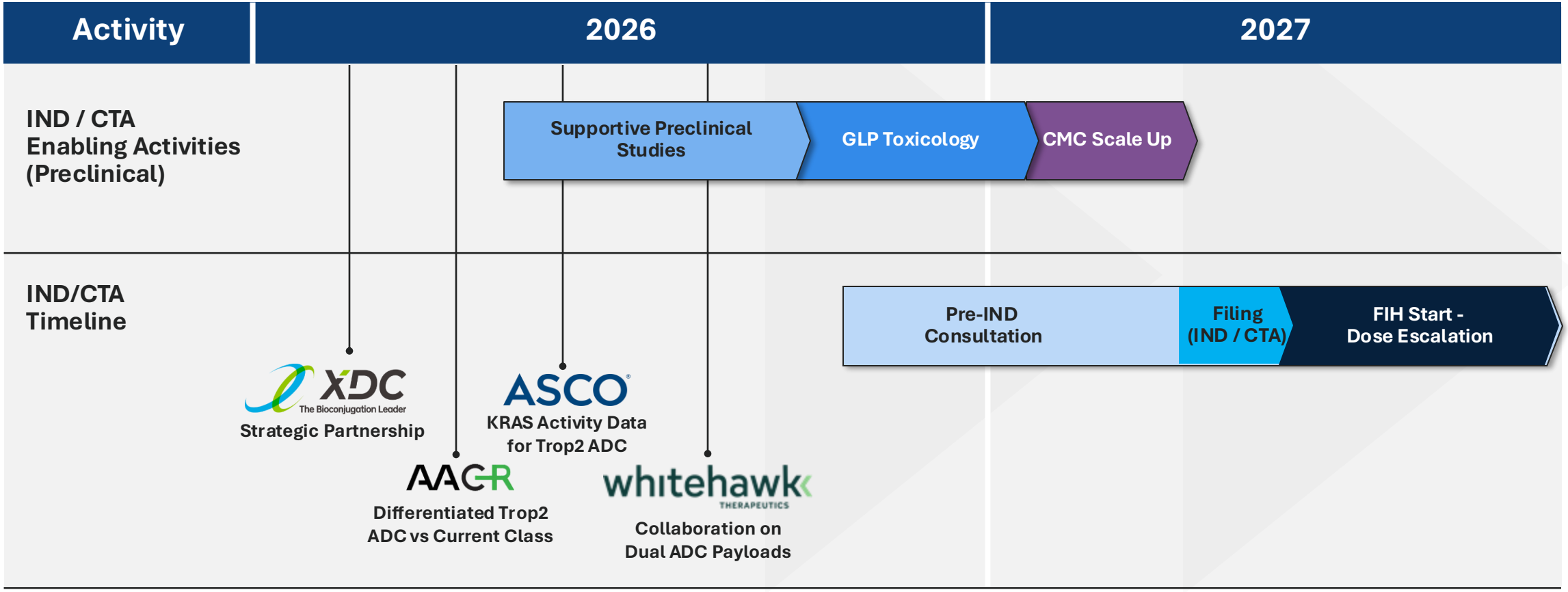
Lead Asset AKTX-101 in IND-Enabling Studies; Phase 1 Initiation Targeted by Mid-2027

ADC Programs (All PH1 Payload Based)	Indication	Discovery	Preclinical	IND Enabling	Highlights
AKTX-101 (TROP2-PH1)	**Urothelial Cancer; Other Solid Tumors**				- Initiated IND enabling activities - GLP tox data expected Q4 2026 - Phase 1 IND filing expected by mid-2027 - Focus on urothelial cancer and other select tumors (breast, lung)
AKTX-102 (CEACAM5-PH1)	**Colon, Gastric, and Lung Cancers**				- Developing best-in-class antibody/PH1 ADC - Opportunity in colon, pancreatic, gastric, lung

PH1 Proprietary ADC Payload Enables Expanding Beyond AKTX-101 and AKTX-102

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AKTX-101 Path to Clinic: Projected Phase 1 Initiation By Mid-2027



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ADCs Have Transformed Cancer Treatment and Continue to Drive Significant R&D Investment and Deal Flow

Clinical success, commercial adoption and unprecedented investment have established ADCs as a cornerstone of oncology therapeutics

17

Approved ADCs Globally[1]

400+

ADCs in Clinical Development[2]

>$75B

Strategic ADC Transactions Since 2023[3]

Commercial ADC Success Has Been Built on Two Dominant Payload Classes

Product	Payload	2025 Sales (Approx.)[4]
Enhertu	Topoisomerase I Inhibitor	~$5.0B
Padcev	Microtubule Inhibitor	~$3.5B
Kadcyla	Microtubule Inhibitor	~$2.3B
Adcetris	Microtubule Inhibitor	~$1.8B
Polivy	Microtubule Inhibitor	~$1.7B
Trodelvy	Topoisomerase I Inhibitor	~$1.4B
Elahere	Microtubule Inhibitor	~$0.8B
2025 Sales		**>$16.5 Billion**

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1. FDA Drugs@FDA database (accessed June 2026); includes all FDA-approved antibody-drug conjugates through 2025; 2. Li et al. Antibody–Drug Conjugates (ADCs): Current and Future Biopharmaceuticals. Signal Transduction and Targeted Therapy, 2025; 3. Nature Biopharma Dealmakers, *Deal bonanza for ADC assets continues*, 2025; 4. Based on 2025 reported revenues from AstraZeneca/Daiichi Sankyo (Enhertu), Pfizer and Astellas (Padcev), Roche (Kadcyla, Polivy), Gilead (Trodelvy), Takeda (Adcetris), and AbbVie (Elahere)

Recent ADC Deal Flow Highlights Strong Pharma Demand for Next-Generation ADC Technologies to Further Accelerate Category

Recent Landmark ADC Transactions

Up to $1.5 Billion

MyricxBio

acquired by

NOVARTIS

July 6, 2026

Up to $1 Billion

DualityBio 映恩生物

licensed by

Genentech

August 28, 2026

Up to $1.3 Billion

和黄医药 HUTCHMED

licensed by

GSK

September 3, 2026

Underscored by Numerous Additional ADC Deals Over the Past Two Years

>37	>$6 Billion	>$24 Billion
Total ADC Deals	Upfront Capital Committed	Total Potential Deal Value

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Source: Company press releases; BioCentury; Evaluate Pharma; GlobalData; PitchBook (2023–2026)

Despite ADC Successes in Earlier Lines of Therapy, Relapsed Patients Have No Options for ADC Sequencing Due to Lack of Novel Payloads

Sequential Use of ADCs With Same Payload Class Has Significantly Less Efficacy Vs. Prior Line of ADC Therapy

Breast Cancer

			Enhertu® DESTINY-Breast06[2]		SATEEN Study Results (Trodelvy®)	
Study	**Drug Sequencing**	**Setting**	**ORR**	**mPFS**	**ORR**	**mPFS**
SATEEN[1]	Enhertu → Trodelvy	**HER2+ metastatic breast cancer**	**62.0%**	**13.2 mo**	**3.7%**	**2.3 mo**

Urothelial Cancer

			Disitamab vedotin earlier line of therapy		Enfortumab vedotin subsequent line of therapy	
Study	**Drug Sequencing**	**Setting**	**ORR**	**mPFS**	**ORR**	**mPFS**
RC48G001 **(Powles T, ASCO-GU, 2026)**[3]	disitamab vedotin → enfortumab vedotin	HER2+ metastatic urothelial carcinoma	**54.9%**	**5.7 mo**	**18.2%**	**N/R**
	disitamab vedotin → enfortumab vedotin	HER2-low metastatic urothelial carcinoma	**52.6%**	**5.7 mo**	**12.5%**	**N/R**

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1. Tarantino P, Yu L, Faggen M, et al. *Efficacy and safety of sacituzumab govitecan (SG) plus trastuzumab in patients with HER2-positive metastatic breast cancer after prior trastuzumab deruxtecan (T-DXd): Results from the Phase II SATEEN trial.* Presented at: ESMO Breast Cancer Congress 2026; May 6–8, 2026; Berlin, Germany. Late-Breaking Abstract LBA4; 2. DESTINY Breast06 Trial Data: https://www.enhertuhcp.com/en/her2-low-her2-ultralow-breast/efficacy/her2-low 3. Rosa K. Disitamab vedotin elicits antitumor activity in HER2-expressing advanced urothelial carcinoma. OncLive. Published February 27, 2026. Accessed August 3, 2026

Akari's PH1 ADC Payload Can Solve For This Sequencing Issue: Targeting RNA Splicing To Drive Differentiated Efficacy and Safety

PH1: A Differentiated ADC Payload Designed to Expand Therapeutic Potential of ADCs

Novel Mechanism of Action

Disrupts RNA splicing, depleting RNAs essential to cancer cell survival and growth

Potent Cytotoxic Activity

Demonstrates sub-nanomolar activity across multiple cancer cell lines and tumor models

Designed to Overcome Common Resistance Mechanisms

Poor substrate for MDR/ABC transporters associated with resistance to many established payload classes

Supports potential activity in multidrug-resistant tumors

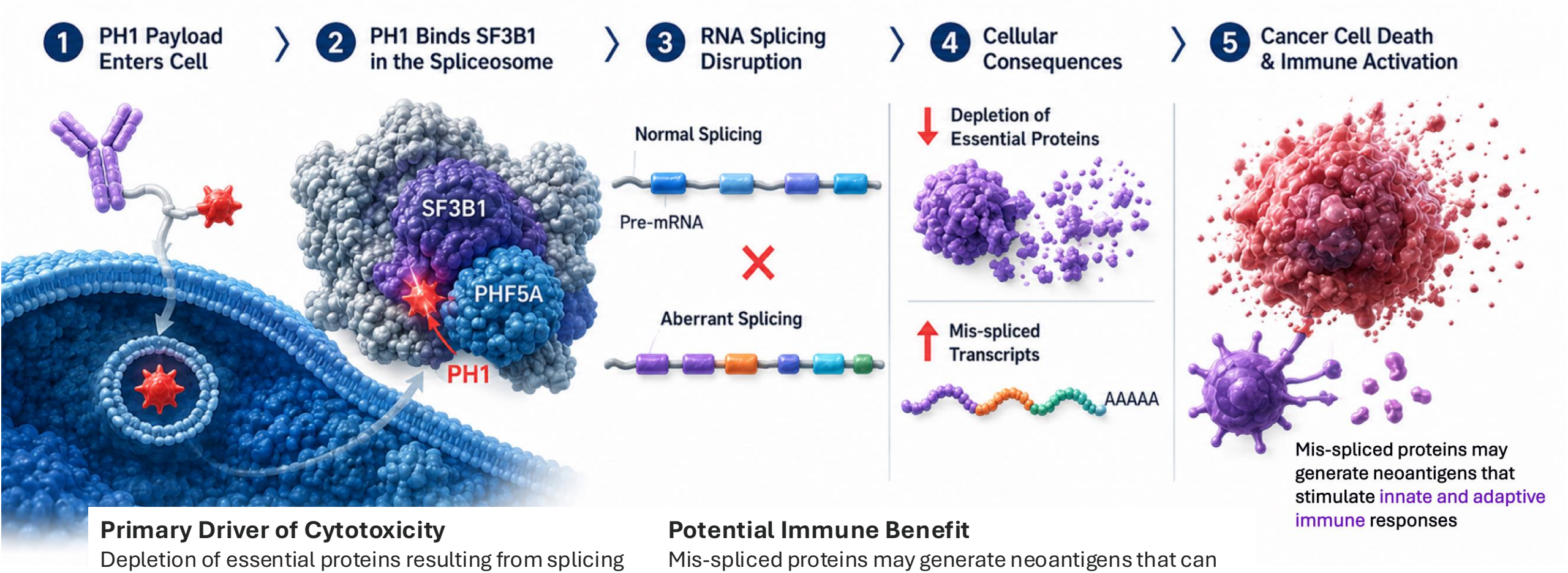
Potential Adaptive Immune Activation

Mis-spliced RNAs may generate novel neoantigens capable of stimulating innate and adaptive immune responses

PH1 ADC Payload Fills a Critical Void in Patients That Have Relapsed After 1st Line ADC Therapies That Utilize Microtubule or Topo1 Inhibitor Payloads

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PH1 Targets the SF3B1 Spliceosome Complex to Disrupt RNA Splicing and Drive Cancer Cell Death



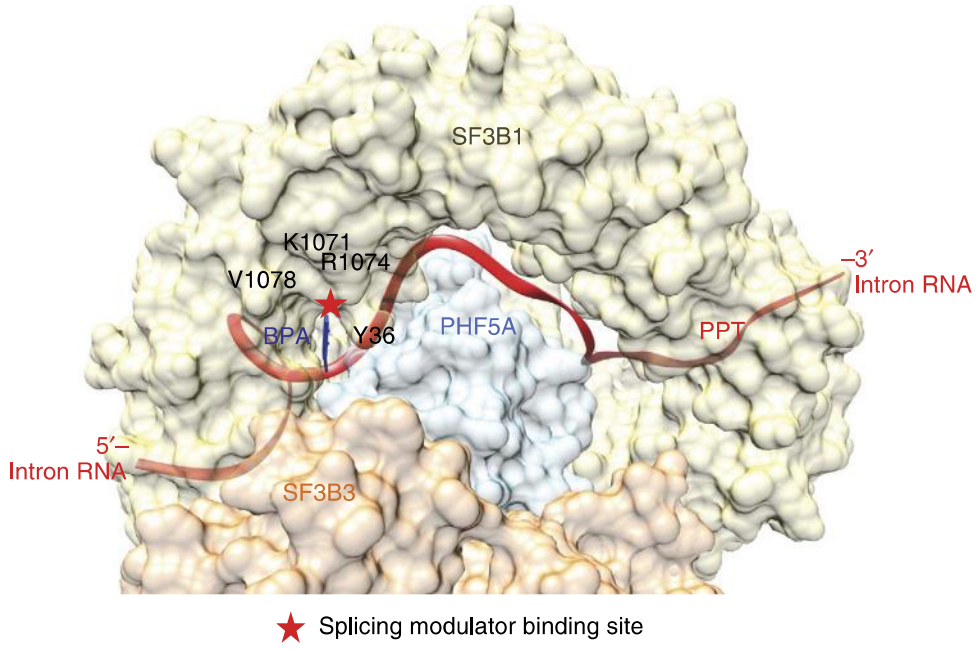
Primary Driver of Cytotoxicity
Depletion of essential proteins resulting from splicing disruption drives potent and broad cytotoxic activity

Potential Immune Benefit
Mis-spliced proteins may generate neoantigens that can stimulate adaptive anti-tumor immune responses

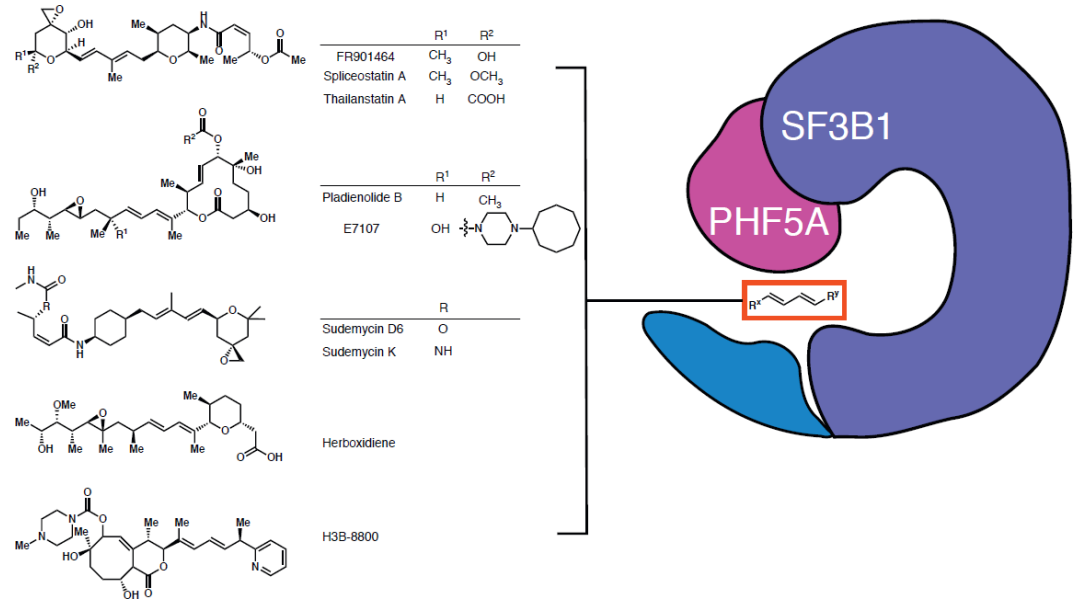
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PH1 Selectively Targets the SF3B1 Spliceosome Complex

PH1 is a Novel, Proprietary 3rd Generation Thailanstatin Analog with Differentiated Binding/Functionality



Model of SF3B1 and PHF5α binding to intron BPA and location of resistance mutations



Binding to SF3B1 disrupts normal spliceosome function and prevents productive RNA splicing

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Teng et al., 2017 Nat Comm

PH1 Induces Broad RNA Mis-Splicing: Drives Both Cytotoxicity and Generation of Neoantigens to Activate the Immune System

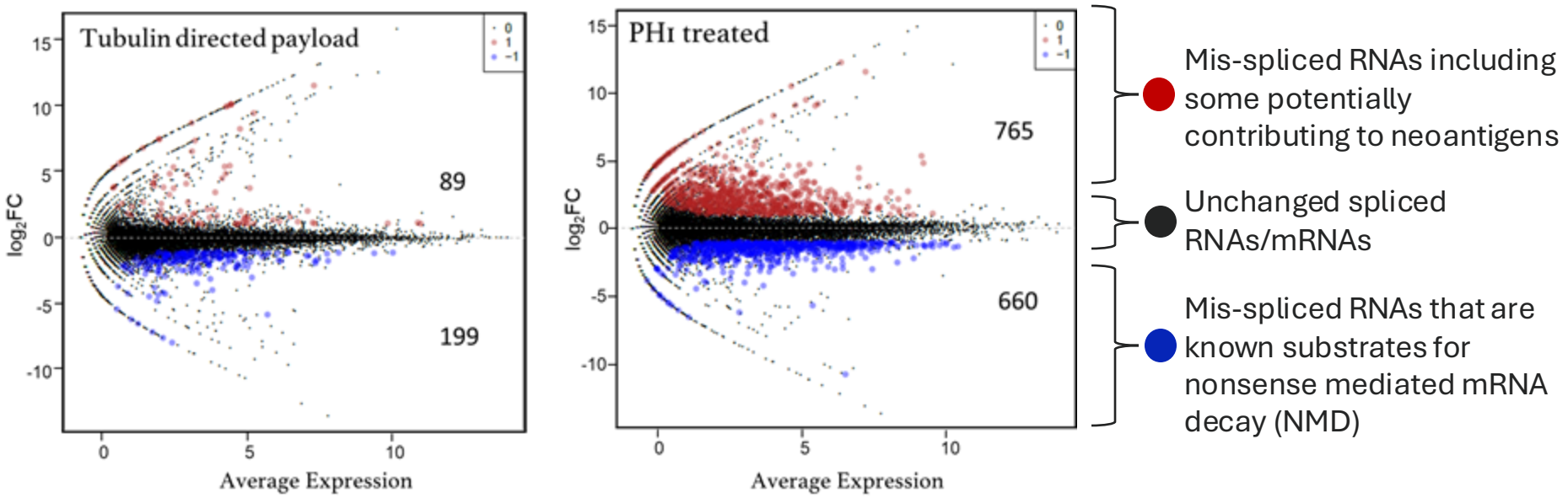
PH1 Generates ~9× More Potential Neoantigen-Generating RNA Transcripts Than DM4 payload (Microtubule Inhibitor)

1 – 2 punch of PH1:

(1) Potent cytotoxicity through disruption of RNA splicing

(2) Generation of potential neoantigen-generating transcripts that may support adaptive immune activation

Average (n=4 wells) expression of spliced RNA transcripts showing effect of DM4 (L, tubulin directed payload) and PH1 payloads (R) compared to control



- Each dot represents a specific RNA derived by splicing
- PH1 markedly increases the number and diversity of mis-spliced transcripts, including ~3× more predicted NMD substrates and ~9× more potential neoantigen-generating transcripts than DM4

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PH1 Combines Potent Cytotoxicity with a Differentiated Therapeutic Profile

Attribute	PH1 Payload	Topo1 Inhibitors	Microtubule Inhibitors
Optimal Potency as a Cytotoxic Agent	< 5 nanomolar IC50	5-20x nanomolar IC50	< 1 nanomolar IC50
Susceptible to MDR Transporters	No – payload not subject to this tumor resistance mechanism	Yes – Creates tumor resistance	Yes - Creates tumor resistance
Immune Activation	• B cell expansion / IgM antibody cell killing • Neutrophil expansion • Macrophage activation	Immunogenic Cell Death (ICD)	Immunogenic Cell Death (ICD)
Mitigate Off-Target Toxicities (Y/N)	**Yes** • **Non-cleavable linker** • **Non-permeable linker-payload**	**No** • Cleavable linker • Permeable linker-payload	**No** • Cleavable linker • Permeable linker-payload
Observed Side Effects	• Transient/Reversible Transaminitis • Mild reduction in platelets(Reversible)	• Significant off-target toxicities and therapy discontinuations • ILD (DXd), Gut Toxicity, Ulcers	• Significant off-target toxicities and therapy discontinuations • Neuropathies, Ocular and Bone Marrow toxicities

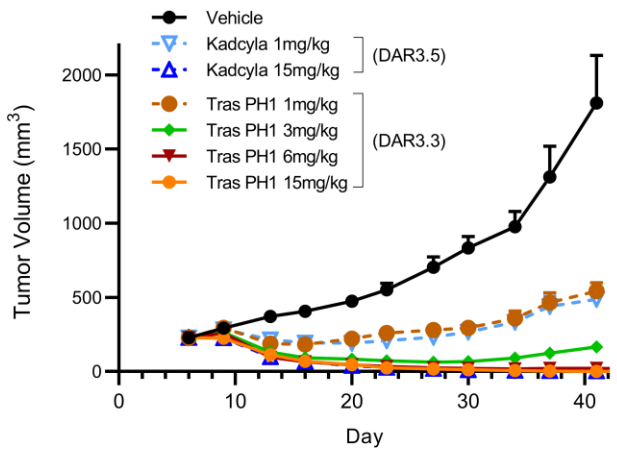
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PH1 Payload Demonstrates Potent Cytotoxicity Vs. Highly Potent Microtubule Inhibitor Payload:

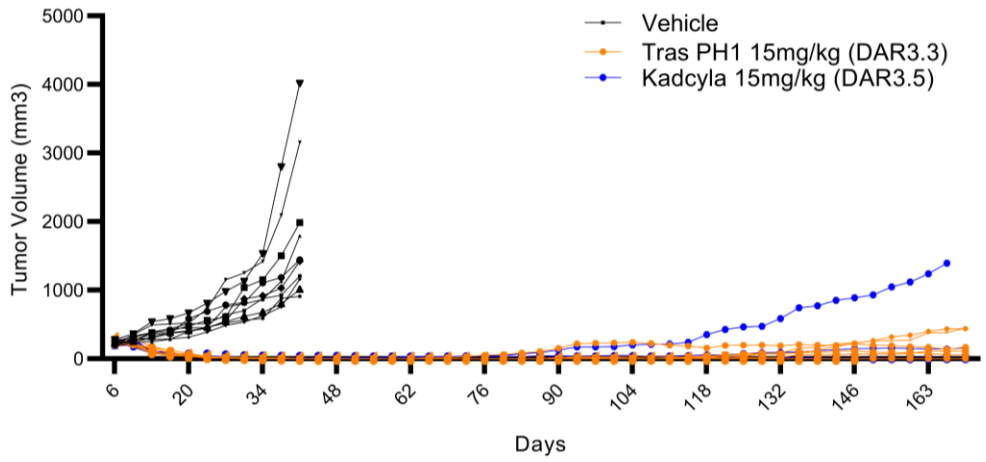
Trastuzumab PH1 vs. Kadcyla in Her2^{High} NCI-N87 Model

Trastuzumab conjugated ADC termed Tras PH1 exhibited durable anti-tumor efficacy in NCI-N87 xenograft tumors vs. Kadcyla (microtubule payload)

Dose-response (n=10 per arm)



Durable-response (n=10 per arm)

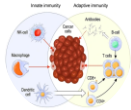


Kadcyla-treated tumor rebound evident

Tras PH1-treated tumor rebound is minimal

Source(s): Data on file, Internal Akari Research Program reports
Data published at https://doi.org/10.1158/1538-7445.AM2021-1832

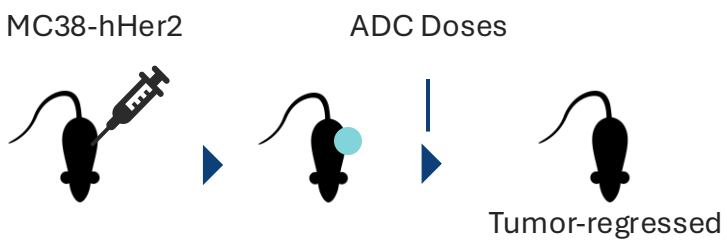
PH1 Payload's Immunotherapy Mode of Action Uniquely Drives Complete Remission and Immunological Memory Against Tumor



PART 1:
Potent Activity of PH1 ADC as a Single Agent and with Checkpoint Inhibitor

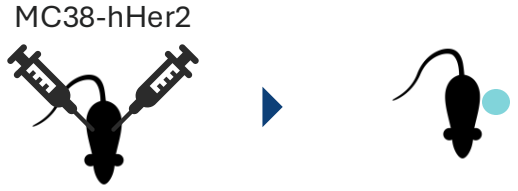
PART 2:
PH1 Payload Induces Tumor-Specific Immunological Memory Against Cancer Cells

ADC Treatment and Survival Curves

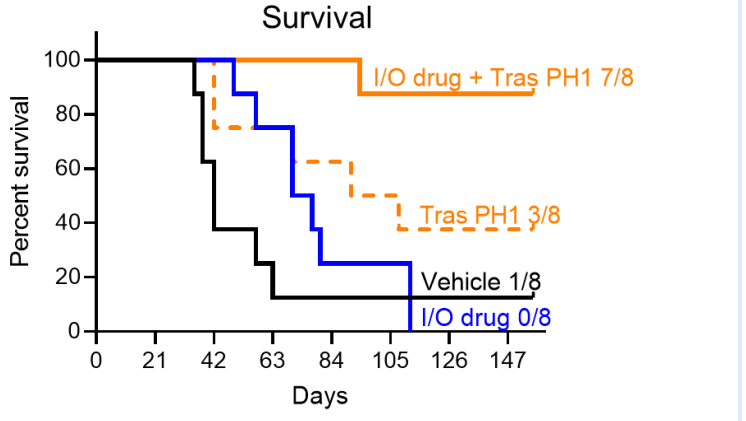


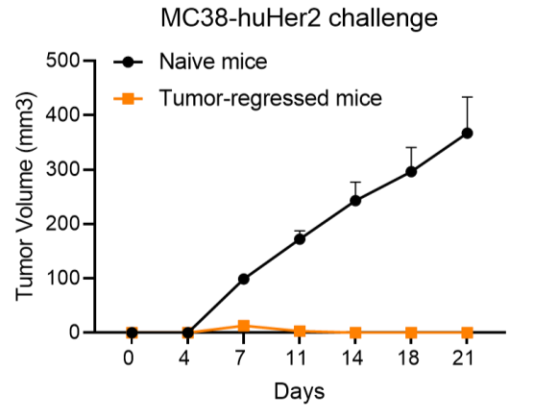
Rechallenge Regressed Mice Only (7/8 From Combination Arm)

Rechallenge



Inject 2 Groups of Mice: 1) Tumor Regressed Mice From Part 1 2) Control group that is naïve to ADC treatment





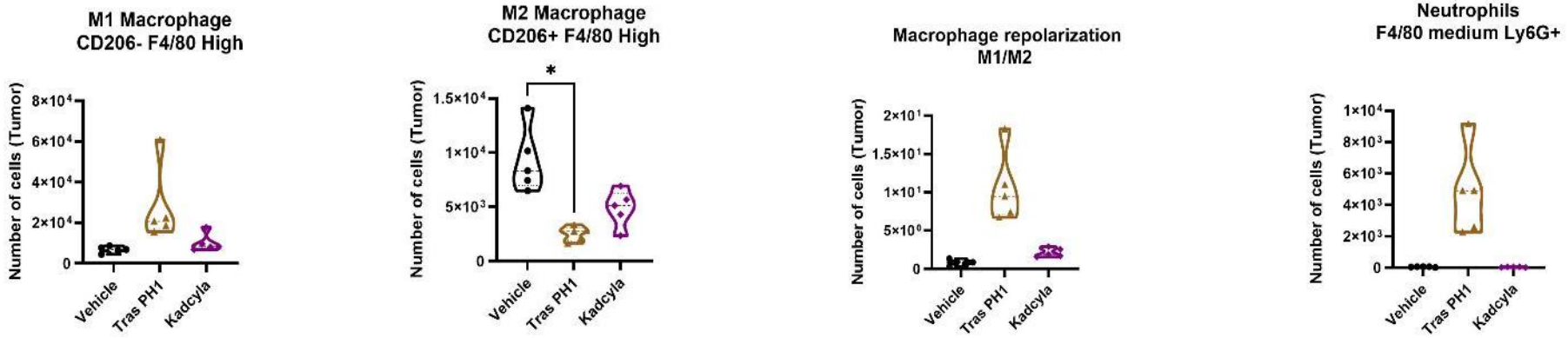
Demonstrates PH1 Payload Creates Immunological Memory That Enables Immune System to Prevent Tumor Growth When Mice are Rechallenged

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Trastuzumab PH1 Durable Remissions Are Driven by Activating Both Innate and Adaptive Immune System – Unlike Other ADC Payloads

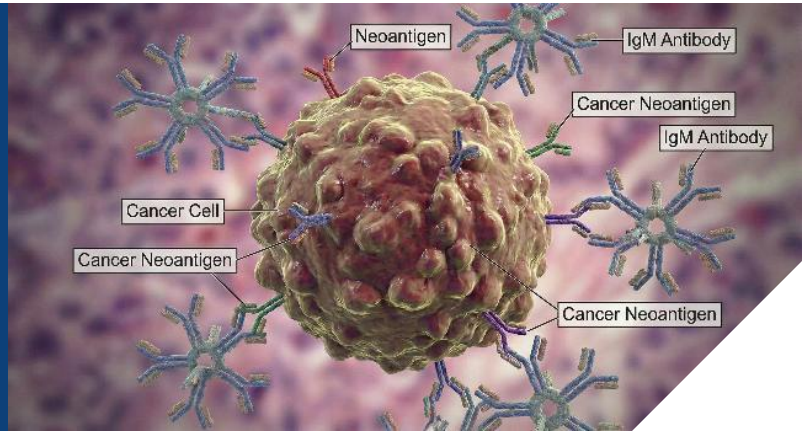
Tras-PH1 [1] Immune Mode of Action Driven by Unique Innate and Adaptive Immune Response

Increase in Pro-Inflammatory Macrophages and Neutrophils for Tras-PH1 vs Kadcyla®



Expands B Cells and IgM Antibodies vs. Kadcyla®

- IgM antibodies target cancer cells via neoantigens
- IgM-targeted cells marked for destruction by complement system



[1] Data using trastuzumab antibody conjugated to PH-1 payload (Tras PH1)

https://akaritx.com/wp-content/uploads/2025/11/SITC-Nov-2025-e-Poster-AKTX.pdf

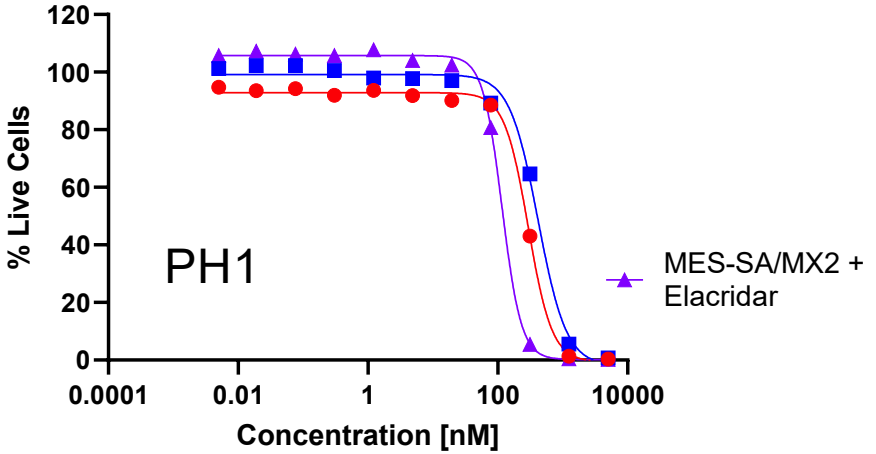
PH1 Payload Designed to Overcome

Traditional Resistance Mechanisms Experienced by Currently Approved or in Development ADC Payloads

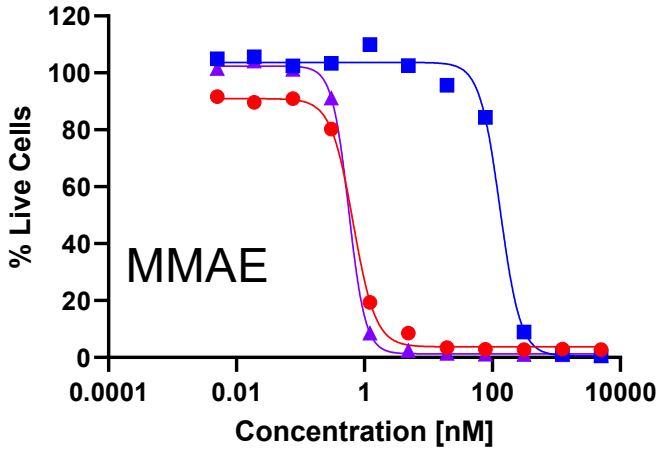
Method: Normal MES-SA cells & MES-SA cells selected for overexpression of MDR transporter 1/2 exposed *in vitro* to PH1 or anti-tubulin payload MMAE (monomethyl auristatin E), in presence or absence of MDR 1/2 inhibitor elacridar

- MES-SA uterine sarcoma cell line
- MES-SA cells expressing high levels of MDR transporter 1 and 2 which can pump toxins out of cells
- MES-SA cells with high MDR expression + MDR inhibitor elacridar

PH1 potency unaffected by overexpression of multidrug resistance (MDR) transporters



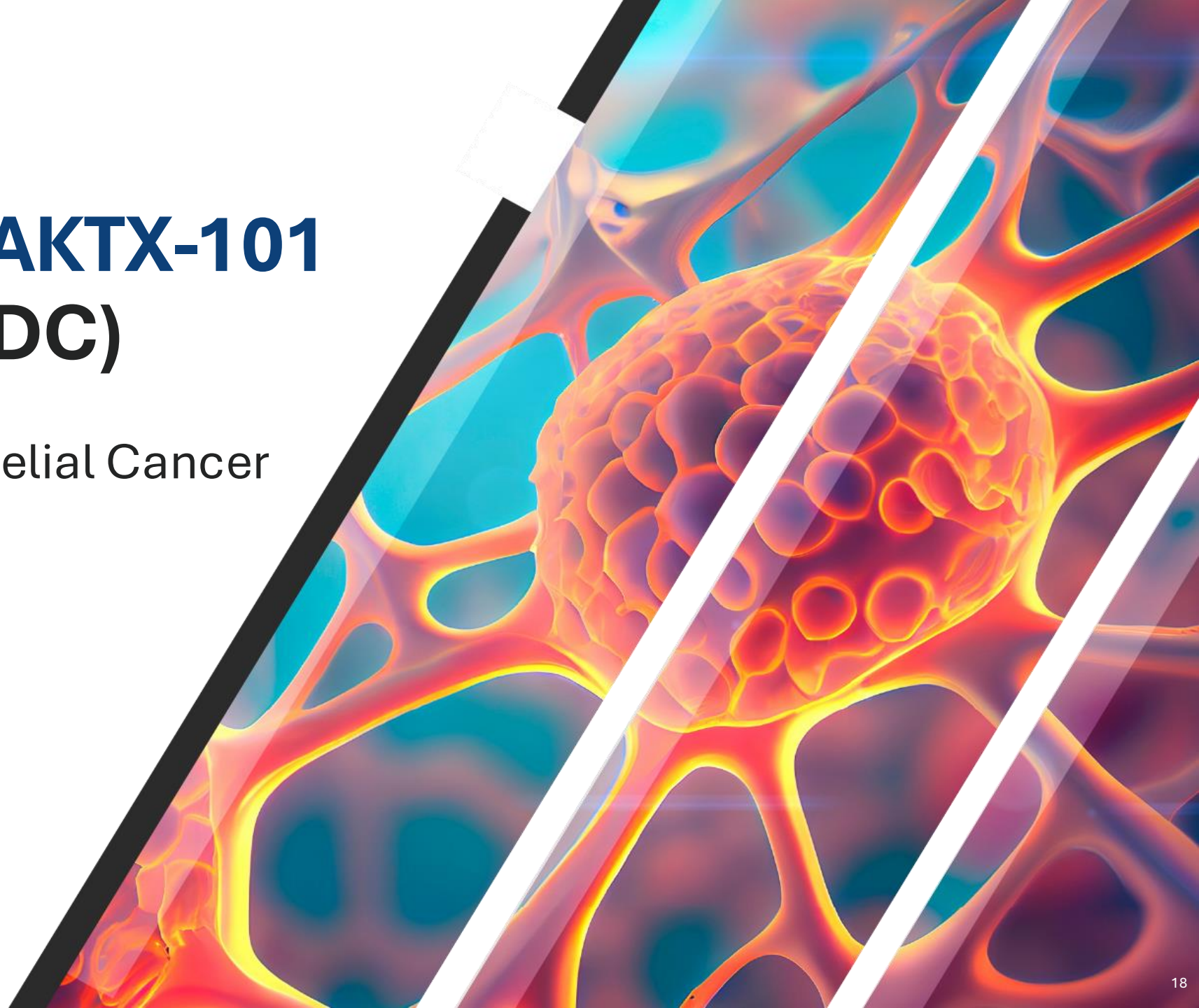
200X higher MMAE concentration required to kill cells overexpressing MDRs; inhibition of MDR 1/2 by elacridar restores MMAE potency



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Lead Program AKTX-101 (TROP2 PH1 ADC)

Initially Targeting Urothelial Cancer

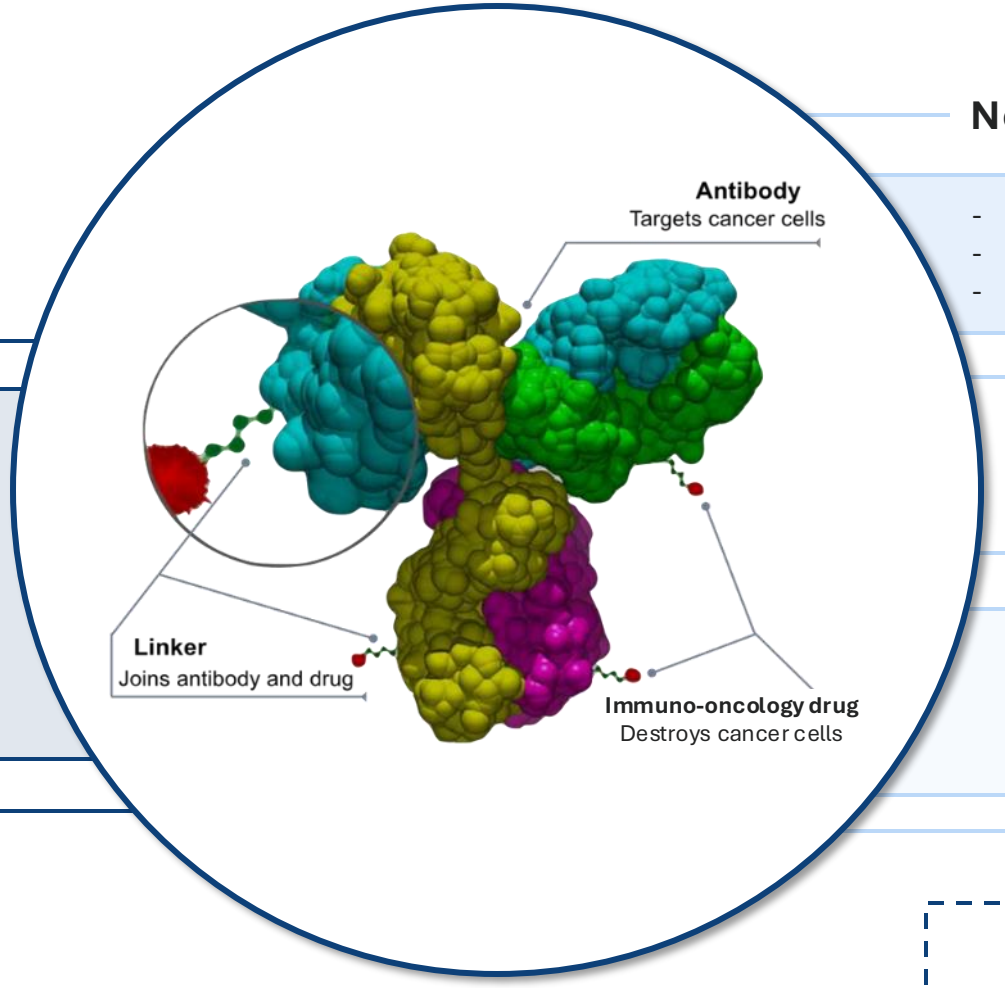


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AKTX-101: Highly Differentiated TROP2 ADC with Novel PH1 Payload

Reduced Off-Target Effects

- AKTX-101 uses L22 lysine non-cleavable linker
- This linker provides best performance vs. traditional val-cit and val-ala linkers
- Linker-Payload associated with reduced off-target effects



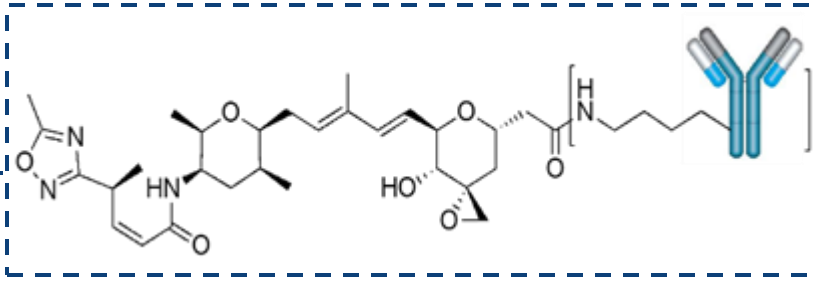
Novel Antibody With Cell Killing Intact

- Employs proprietary TROP2 antibody
- Higher affinity than hRS7 (Sacituzumab)
- Potent ADC action while mitigating ADA immunogenicity

Potent Cytotoxicity and Immune Activation

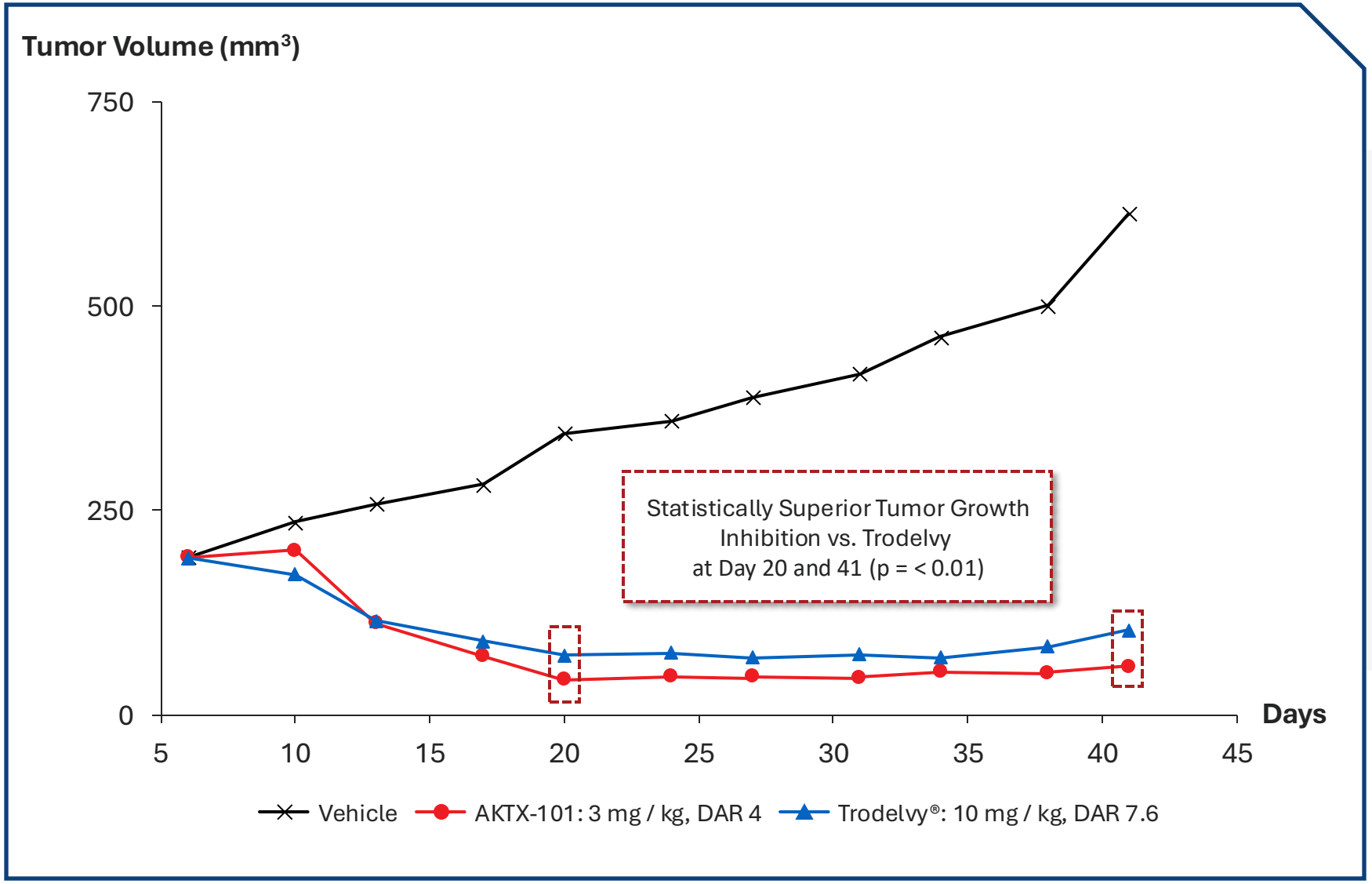
- AKTX-101 employs 3rd generation proprietary Thailanstatin toxin
- Single digit nanomolar potency in several tumors
- Unique innate and adaptive immune activation

Chemical Structure of Toxin Attached to TROP2 Antibody



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AKTX-101 Demonstrates Superior Tumor Regression at Significantly Lower Dose/DAR Compared to Trodelvy® (Topo1 ADC)



Method

NCI-N87 cell xenograft model

Dosing regimen: Day 1 and Day 8 for both AKTX-101 and Trodelvy® to align with approved dosing schedule for Trodelvy®

Results

At Day 150 (end of study), AKTX-101 @ 3 mg/kg, DAR4 had 5/10 tumors regressed

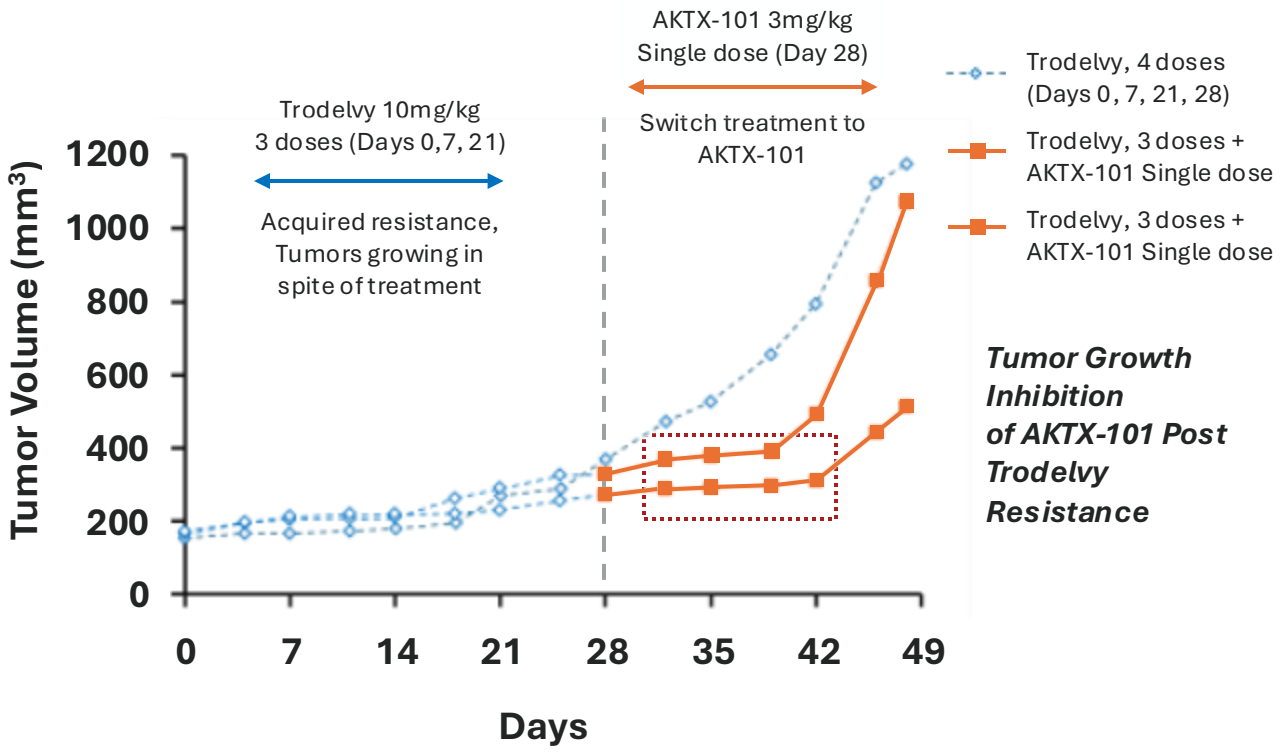
vs.

Trodelvy® at @ 10 mg/kg, DAR 7.6 with 2/10 tumors regressed

Source: Akari Internal Data

AKTX-101 Demonstrates Strong Activity in Urothelial Cancer Models Resistant to TRODELVY® and PADCEV®, Highlighting Prominent Role for ADC Sequencing

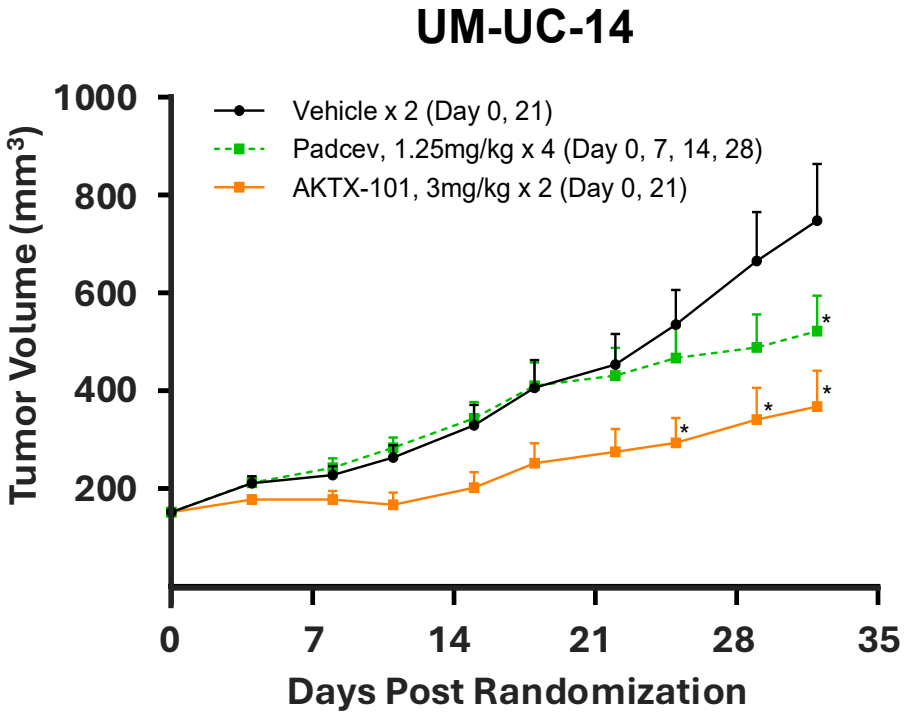
Trodelvy-Resistant SW-780 Tumors



Tumor Growth Inhibition of AKTX-101 Post Trodelvy Resistance

Padcev Resistant Model, UM-UC-14

AKTX-101 efficacy in an upper tract urothelial carcinoma model that responds poorly to Padcev



Source: Internal Data

AKTX-101(Trop2 PH1) Has a Differentiated Safety Profile vs Current TROP2 ADCs

Significant Safety Warning/Precautions	Trodelvy® (SN-38 payload)	Datroway® (Dxd payload)	Sac-TMT (Tirumotecan payload)	AKTX-101 (PH1 payload)
Interstitial Lung Disease (ILD)		X		
Stomatitis		X	X	
Diarrhea	X			
Neutropenia	X		X	
Anemia			X	

AKTX-101 Current NHP Findings Focused In 3 Key Areas

- Skin rash/scaling (known on-target toxicity for TROP2 ADCs)
- Reduction in platelets (reversible)
- Liver enzyme elevations (reversible)

<u>On-going Work (Initiated in Q4):</u>

1. GLP Toxicology Study underway to define ADC-related toxicities, reversibility, formal HNSTD

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Source: Akari Internal Data, Non-Human Primate Toxicology Studies, Okajima et al 2021 and Cardillo et al 2011, Approved Product Package Inserts, Merck Data Releases (website)

In Urothelial Cancer, 2nd Line Opportunity is Valuable and Makes an Attractive Strategic Entry Point for AKTX-101

DIAGNOSIS: metastatic Urothelial Carcinoma (mUC)

PREFERRED 1ST LINE THERAPY (Standard of Care for All-Comers)



Enfortumab Vedotin + Pembrolizumab

(Regardless of Cisplatin Eligibility)

DISEASE PROGRESSION

50% of Patients Relapse in ~12 Months

2ND LINE THERAPY (Post-EV+Pembro) **~75,000 Patients***

Platinum-based Chemotherapy

(Gem/Cis or Gem/Carbo)



Erdafitinib

(If FGFR2/3 Alteration +)

Unmet Needs:

- Improved PFS, duration of response and overall response
- Limited toxicities in frail and elderly population
- No overlapping toxicities with 1L therapies (e.g. neuropathies)

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*Patient Estimates include US, EU5, Japan, China; Data Monitor, Decision Resources Group, Kantar Health, and internal estimates; Powles et al: N Engl J Med 2024;390:875-888

Urothelial Cancer is Part of a Larger Opportunity for AKTX-101, Based on Solid Tumors That Have High TROP2 Expression

Cancer Type	New Annual Cases	Annual Deaths
Initial Target: Urothelial	85,000	17,000
Breast	319,750	42,680
Lung	226,650	124,730
Colorectal	154,270	52,900
Head and Neck	72,680	16,680
Pancreatic	67,440	51,980
Oral Cavity	59,660	12,770
Gastric	30,300	10,780
Esophagus	22,070	16,250
Ovarian	20,890	12,730
Cervical	13,360	4,320
Total Potential Target TROP2 Patient Population	**987,070**	**345,820**

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First-in-Human Phase 1a Trial Explores Safety, Therapeutic Dose and Identifies Efficacy Signals in Range of Solid Tumors

Phase 1b Expansion into Urothelial Cancer Drives Potential Accelerated Path

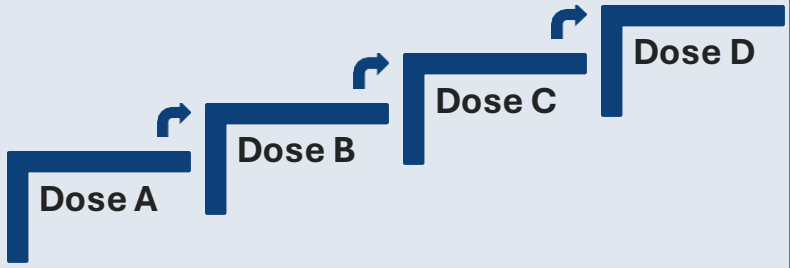
Dose Escalation Phase 1a
12-24 Patients

Recommended Dose

1b Expansion In Urothelial

Potential for Other Solid Tumor Exploratory Arms

TROP2 Expressing Tumors (Urothelial, Gastric, Lung, Breast, Others)



Design: Simon 2-stage

Urothelial Stage 1 → **Urothelial** Stage 2

Exploratory Solid Tumor Stage 1 → **Exploratory Solid Tumor** Stage 2

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Partnerships Are Accelerating The Development and Value of PH1 ADC Payload



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Akari Has Entered into Two Strategic Partnerships to Advance the PH1 Payload Through Multiple Ways: Internal Pipeline and Dual Payload ADCs

Independent Collaborations Spanning Manufacturing Scale-Up and Dual-Payload Discovery

WuXi XDC

Development & Manufacturing Partnership

- Global ADC leader spanning novel payloads, linkers and conjugation technologies
- Scale-up and GMP supply of the PH1 payload for AKTX-101
- End-to-end CMC and nonclinical work to enable the IND package by mid-2027
- Proprietary highly efficient cell lines for ADC production

Accelerates AKTX-101 toward first-in-human

whitehawk THERAPEUTICS

Dual-Payload Research Collaboration

- Combines Akari's PH1 spliceosome modulator with Whitehawk's TOP1i ADC platform
- Akari leads the design, execution and evaluation of the preclinical studies
- Interim data as studies progress, with joint review to guide future development

Opens a first-in-class dual-payload ADC frontier

External Validation of PH1 — and a Broader Frontier for Akari's ADC Platform

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Akari is Positioned to Lead the Next Generation of ADC Payload Innovation

Differentiated Payload Platform

- First-in-class ADC payload targeting RNA splicing to attack cancer
- PH1 is a potent cytotoxic and immuno-therapeutic as a single agent
- Designed to circumvent key limitations of current payload classes and can be sequenced after 1st line ADCs
- Broad potential to create multiple ADCs across variety of antigens

Advancing Lead Program: AKTX-101, TROP2 PH1 ADC

- Initial focus in urothelial cancer with expansion opportunities in Breast, Lung, Gastric, Head & Neck, Others
- Supported by differentiated preclinical efficacy and safety profile

Plan to Initiate Phase 1 FIH (First-in-Human) by Mid-2027

Significant Value Inflections Possible Through Next 18 Months

- ADC comparables suggest potential for significant upside at catalyst timepoints: Phase 1 initiation and potential partnerships

Key Target Milestones The Next 18 Months

✓ 2H 2026: GLP Toxicology Results

✓ Mid-2027: Phase 1 First-in-Human Study Initiation

✓ On-going: PH1 ADC Strategic Collaborations and Partnerships

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Supplemental Corporate Information:

Akari Website: https://akaritx.com

Recent Press Releases:

1. Akari Therapeutics Secures Australian Patent Approval
2. ASCO Abstract Acceptance Highlights Potential for AKTX-101 ADC to Treat KRAS Mutant Tumors
3. Akari Therapeutics Announces Strategic Partnership with WuXi XDC to Advance Development of Its Novel ADC Payload Targeting RNA Splicing
4. Akari Therapeutics Files Key Patent and Unveils Second ADC Program AKTX-102 Targeting CEACAM5 Expressing Solid Tumors

Recent Data / Posters / Presentations:

1. 2026 AACR Poster: Rationale for the development of a differentiated Trop2 ADC in solid tumors of the bladder, lung, and breast
2. 2025 SITC Poster: A novel splicing-targeted ADC payload drives immune activation, synergy with checkpoint inhibitors, and enhanced therapeutic potential beyond cytotoxicity
3. 2023 AACR Poster: Rationale for the development of a differentiated Trop2 ADC

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