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The Next Generation of Peptide Drug Conjugates: Controlled Release of Payload

September 2026

Advancing the Next Gen pre|CISION® Platform: Tumor-activated PDCs Delivering Payloads with Optimized Kinetics in a Small Molecule Format

pre|CISION® masks a potent payload behind a peptide that is cleavable only by FAP, a protease abundant in solid-tumor stroma and near-absent in healthy tissue, widening the therapeutic index of potent payloads that are otherwise dose-limited by toxicity

Lead Asset

FAP-Exd (AVA6103) is the First of our Next Gen PDC

Next Gen pre|CISION® PDC is FAP-activated**, controlled-release exatecan**

Phase 1 FOCUS-01 trial is dosing since March 2026 across 6 indications:

- Gastric Ca
- CRC
- Cervical Ca
- Small Cell Lung Ca
- HR+ Breast Ca
- Pancreatic Ductal Adenocarcinoma

Next Value Drivers

Shots on Goal, Staged in the Clinic

AVA6103

- Initial clinical Proof of Mechanism data 3Q 2026
- Significantly higher tumor concentration, selectivity and activity vs. Enhertu® in vivo

AVA6207

- Gen Three dual payload
- Candidate selection 2H 2026 with data presented to support moving to IND

Why It Wins

pre|CISION® is De-Risked in Patients

FIRST GEN AVA6000 demonstrated:

- **Optimized safety with payload concentrated in the TME**
- **Excellent cardiac safety**, leading regulators to **lift the lifetime maximum dose cap** on its doxorubicin payload
- **Durable RECIST responses** in salivary gland cancer even at low level FAP expression

Sept ‘26	2H 2026	1H 2027	ONGOING	2027
First PK & Safety readout from AVA6103	AVA6207 candidate selection	AVA6103 efficacy readout	Discussions with multiple Pharma partners	Targeted Nasdaq dual listing

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. FAP=fibroblast activation protein; PDC=peptide drug conjugate; TME: tumor microenvironment.

[1]Preclinical in vivo comparison presented at AACR 2026; cross-model, not a clinical head-to-head. Efficacy comparison head-to-head in FAP+/HER2+ gastric cancer PDX

The problem.

The most effective cancer drugs are too toxic for patients

The promise.

Deliver high concentration of the active drug directly to the tumor with an antibody or peptide to reduce systemic exposure

The reality.

Design challenges and toxicity have confronted the first generation peptide drug conjugates – opening an opportunity for a Next Gen approach

The Next Gen pre|CISION® PDC Drug Class Aims to Deliver Activity More Robust Than the ADC

This innovative PDC drug class aims to **achieve the robust efficacy** of the ADC class using innovative **small molecule chemistry**

1

RELEASE payload

in a **tumor-selective** manner

2

PROTECT normal tissues

from the **toxic effects of the payload**

3

MODULATE the PK of the payload

to deliver “like an ADC” with **controlled exposure** in the tumor

pre|CISION Next Gen Controlled Release Peptide Drug Conjugate (PDC) Platform

Designed to achieve all 3 goals using innovative, small molecule chemistry

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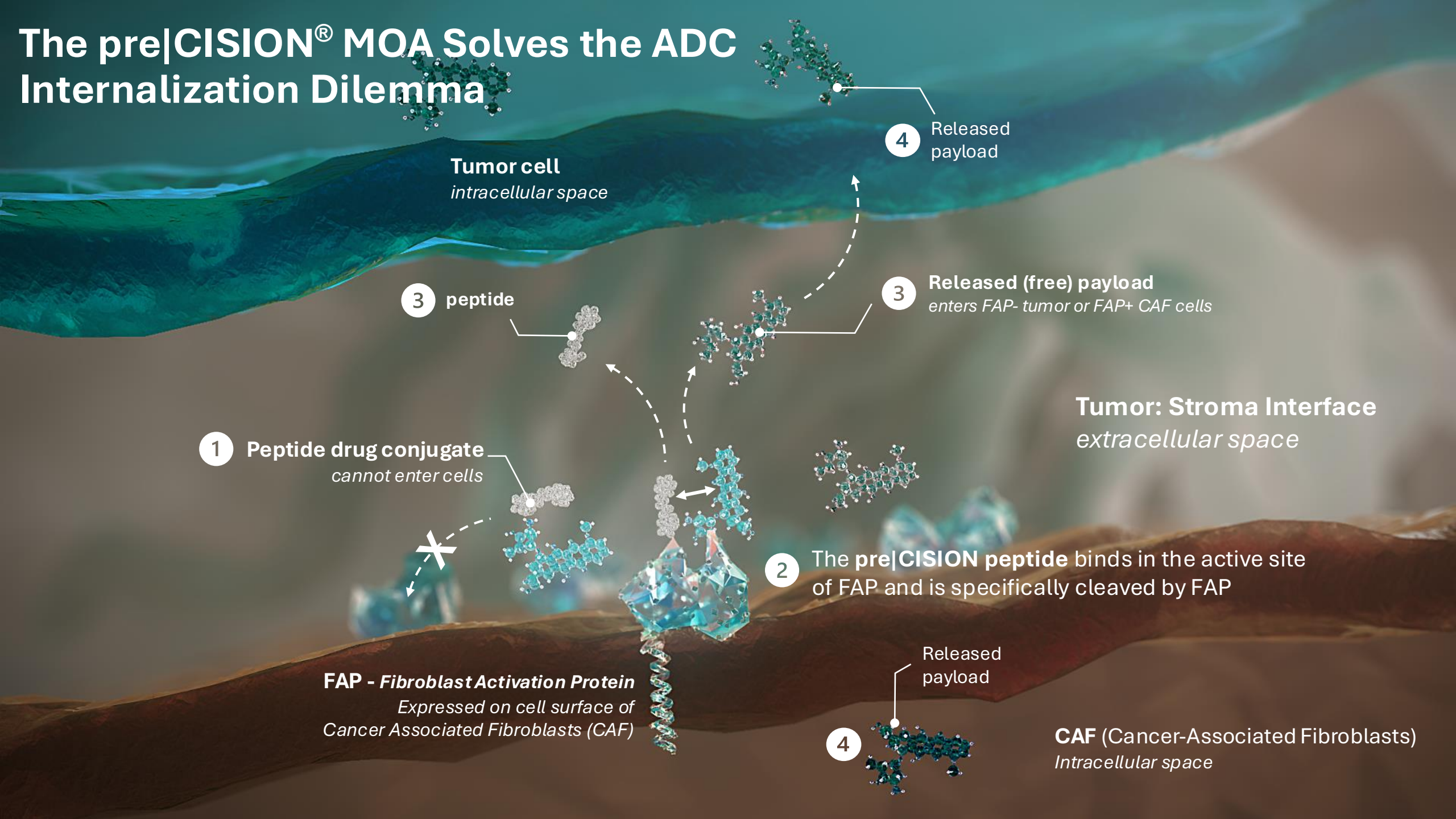
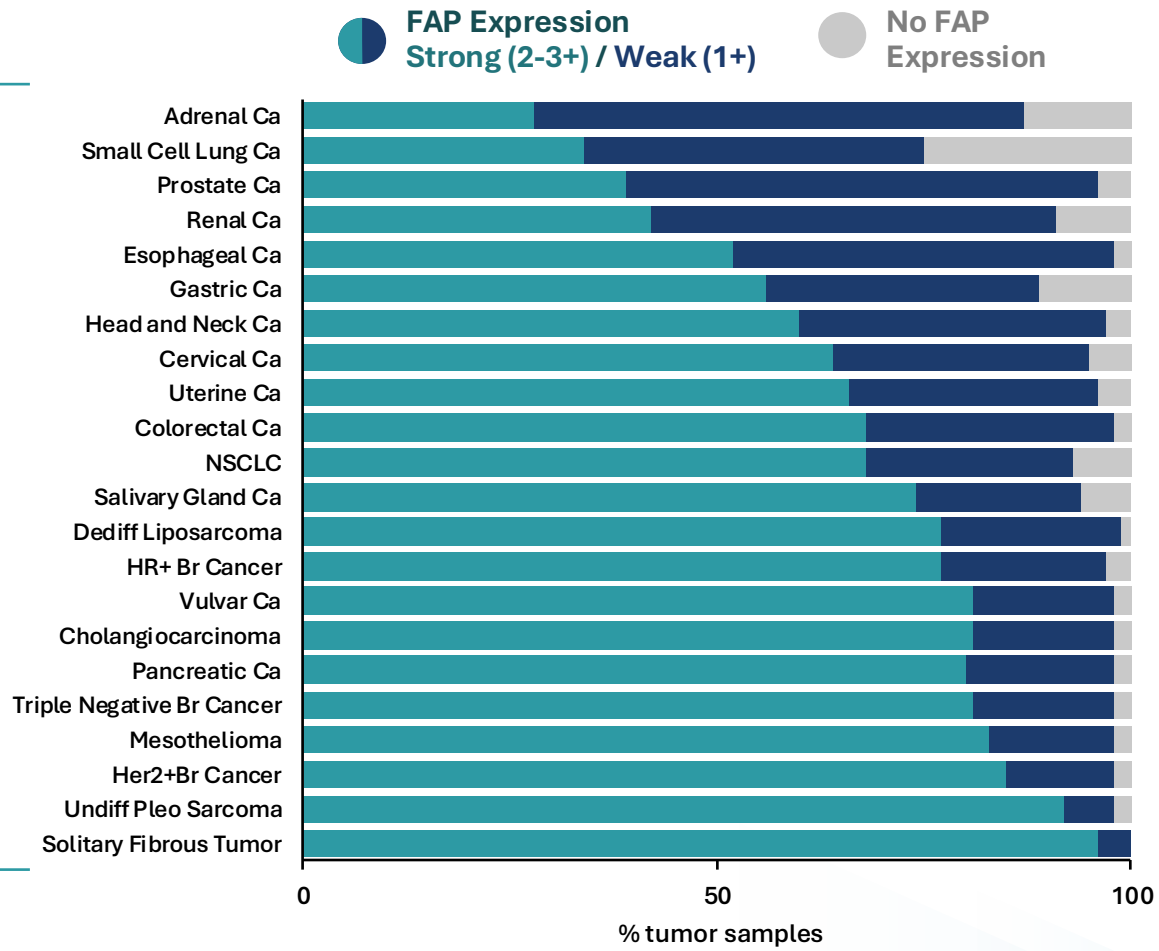

The pre|CISION® MOA Solves the ADC Internalization Dilemma
Tumor cell
intracellular space
4 Released payload
3 peptide
3 Released (free) payload
enters FAP- tumor or FAP+ CAF cells
Tumor: Stroma Interface
extracellular space
1 Peptide drug conjugate
cannot enter cells
2 The pre|CISION peptide binds in the active site of FAP and is specifically cleaved by FAP
FAP - Fibroblast Activation Protein
Expressed on cell surface of Cancer Associated Fibroblasts (CAF)
Released payload
4
CAF (Cancer-Associated Fibroblasts)
Intracellular space

The pre|CISION® Platform Holds a Large Market Opportunity With ~90% of All Solid Tumors Expressing FAP

90%
of solid tumors
express FAP
Fibroblast Activation Protein

- FAP is primarily expressed as a membrane-bound enzyme in the TME
- Only a very small amount of FAP expression is needed to release payload by **pre|CISION®**

FAP expression assessed in >160,000 human tumors using the combined methods of IHC and RNA analysis[1]



Data in the Tempus AI LENS database were analyzed for expression of FAP. Cut-points to define negative, weak and strong were developed using IHC methods and these cut-points were the same across the entire LENS database. Cut-points were set based on known/published positive rates for IHC in 3 diseases: gastric cancer, triple negative cancer and SCLC. Full dataset from this study is available from Avacta. TME=tumor microenvironment, IHC: Immunohistochemistry.

The pre|CISION® PDC Platform Addresses Key Challenges in the ADC Field

	Traditional ADC **CHALLENGES**	pre|CISION® PDC **OPPORTUNITIES**
Payload release	Non-specific protease cleavable linkers result in **organ toxicity** (e.g. pneumonitis, hepatitis)	**Tumor-specific release** by FAP with minimal organ toxicity (e.g. no cardiac toxicity, no pneumonitis or hepatitis)
Dose density to treat resistant disease	Increasing the dose density of an ADC is limited by **the extended PK of the antibody** and **safety of payload in plasma**	**Dose density can be increased** to treat resistant disease given the lack of peptide accumulation and low release of payload in plasma
Internalization dilemma	**Payload is released following slow internalization** and the bystander effect to treat Ag-negative cells is complex	PDC have **simple extracellular release** resulting in kill of FAP+ and FAP- cells
Tumor penetration	**Slow uptake kinetics** (>24 hours) into tumor cells for payload to be released intracellularly	**Payload release observed in minutes** with T_{max} in the tumor less than 1 hour

… all delivered with small molecule chemistry and manufacturing

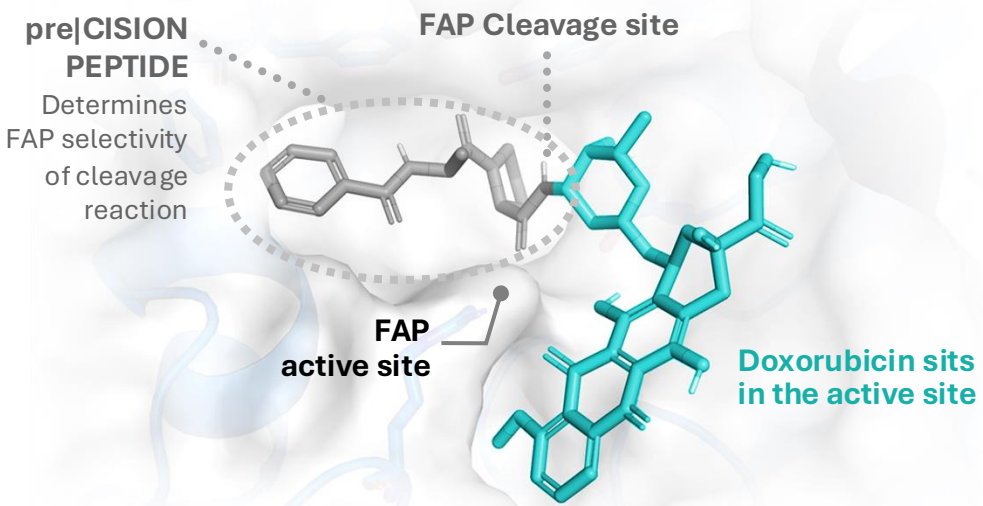
PDC: Peptide drug conjugate | Tmax: Time required to reach Cmax | IHC: immunohistochemistry | Ag: antigen

Avacta's Innovative Pipeline: Three Programs Deploying Our pre|CISION® Platform

	PROGRAM	PAYLOAD	POTENTIAL INDICATIONS	PRE-CLINICAL	IND-ENABLING	PHASE 1	PHASE 2 & 3	MILESTONES
Next Generation Programs	**AVA6103**	**Exatecan** (controlled release)	• Gastric cancer (GC) • Cervical Cancer • Small cell lung cancer (SCLC) • Pancreatic ductal adenocarcinoma (PDAC) • HR+ Breast cancer • Colorectal cancer					Trial in Progress at AACR Pancreatic Cancer and ESMO Congresses in Q4 '26 Efficacy Data H1 '27
	AVA6207	**Dual Payload** Topo Ii/ATRi	Not disclosed					Payloads and Candidate selection to move to IND-enabling studies 2H '26
Pending Partner	**Faridoxorubicin** AVA6000	**Doxorubicin**	• Head and Neck Cancers (Salivary Gland Ca subset) • High grade sarcoma (De-differentiated liposarcoma) • Breast cancer (TNBC/HER2+/HER2low)					Phase 1a/1b data updated at ESMO 2H '26 Further studies pending partnering deal

The pre|CISION® Controlled Release Mechanism Opens the Pipeline to a Large Number of Small Molecules

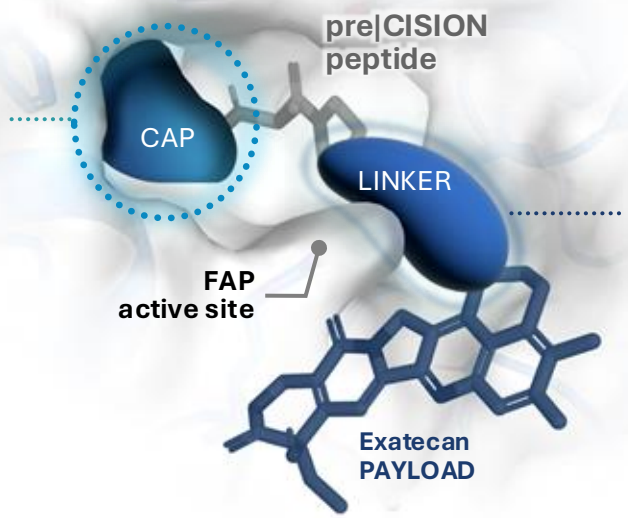
FIRST GEN: Direct Peptide Link



Highly efficient substrate for FAP
enabling rapid cleavage and doxorubicin release
(k_{cat}/Km 39,000)

NEXT GEN: Controlled Release Mechanism

CAPPING GROUP creates a reservoir of intact PDC in the tumor, **slowing PDC clearance**



SELF-IMMOLATIVE LINKER

- Enables **multiple payload classes**
- Variable cleavage efficiency enables **controlled payload release in the tumor**

CONTROLLED RELEASE ENABLES

Greater PK control • Controlled release in the tumor • Expanded pipeline potential

k_{cat}/Km: defined as the catalytic efficiency of an enzyme. A high kcat/Km means the enzyme converts substrate to product more efficiently under low-substrate conditions

Faridoxorubicin: Our First Gen Asset showed Proof of Concept for the pre|CISION® Platform with 4 Key Insights for the Next Gen

FIRST GEN Insights	**Payload concentrates in the tumor**, however this is associated with fast cleavage and a **dose-dependent increase in plasma AUC**	**First Gen pre|CISION dramatically reduced the toxicities** associated with the doxorubicin and eliminates severe cardiac toxicity	**Short plasma half-life of the First Gen PDC** coupled with **very rapid cleavage** results in limited tumor retention	Activity in **salivary gland cancers where tumor cells are negative for FAP** indicates the versatility of the platform to treat 90% of all solid tumors
NEXT GEN Impacts	**Reduce plasma exposure to payload** while maintaining tumor selectivity and tumor concentration is critical	Maintain the **altered biodistribution of the payload** with pre|CISION as this contributes to the reduction in toxicity	Next Gen chemistry delivers a **PDC that is taken up by the tumor and cleaves slowly,** enabling controlled release	**Encouraging activity observed in payload-sensitive indications** and in tumor types where **FAP is only expressed in fibroblasts**

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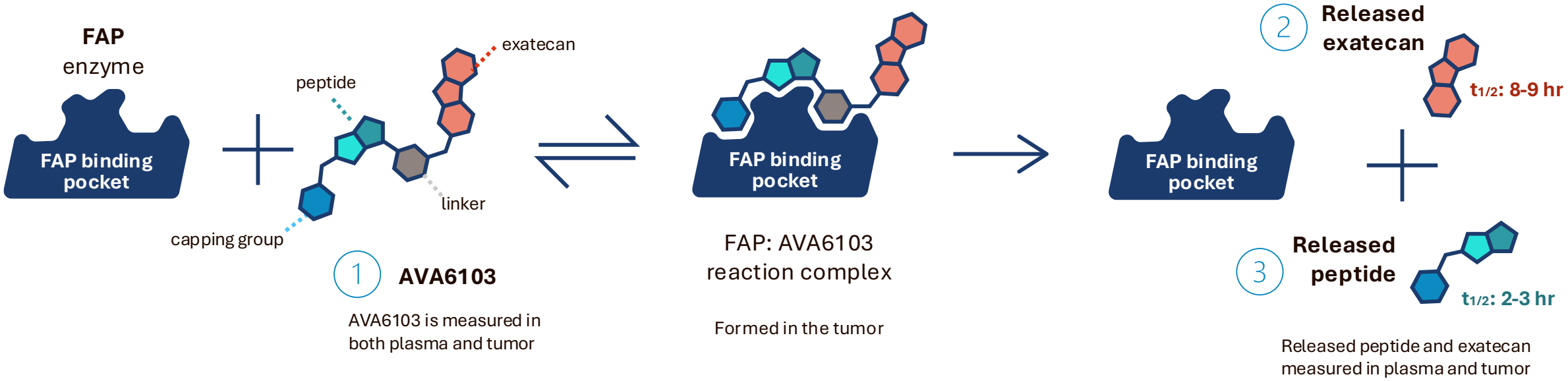
Next Gen: Robust FAP Binding in the Tumor and Slowed Cleavage Enable Controlled Payload Release in the Tumor



The PK studies in animals and humans are **designed to detect the chemical reaction above** that is the basis of the controlled release mechanism

Animal studies use extensive tumor and plasma sampling that is not possible in patients

Clinical plasma PK assays are designed to detect very low levels of the reaction products (exatecan and peptide) released in the bloodstream

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Enzyme and substrate efficiency is described by the ratio of K_{cat}/K_m (very high with AVA6000, very low with AVA6103). The chemical design of controlled release includes low KM (robust binding of the pre|CISION drug to FAP and low to moderate Kcat (determines the rate of cleavage).

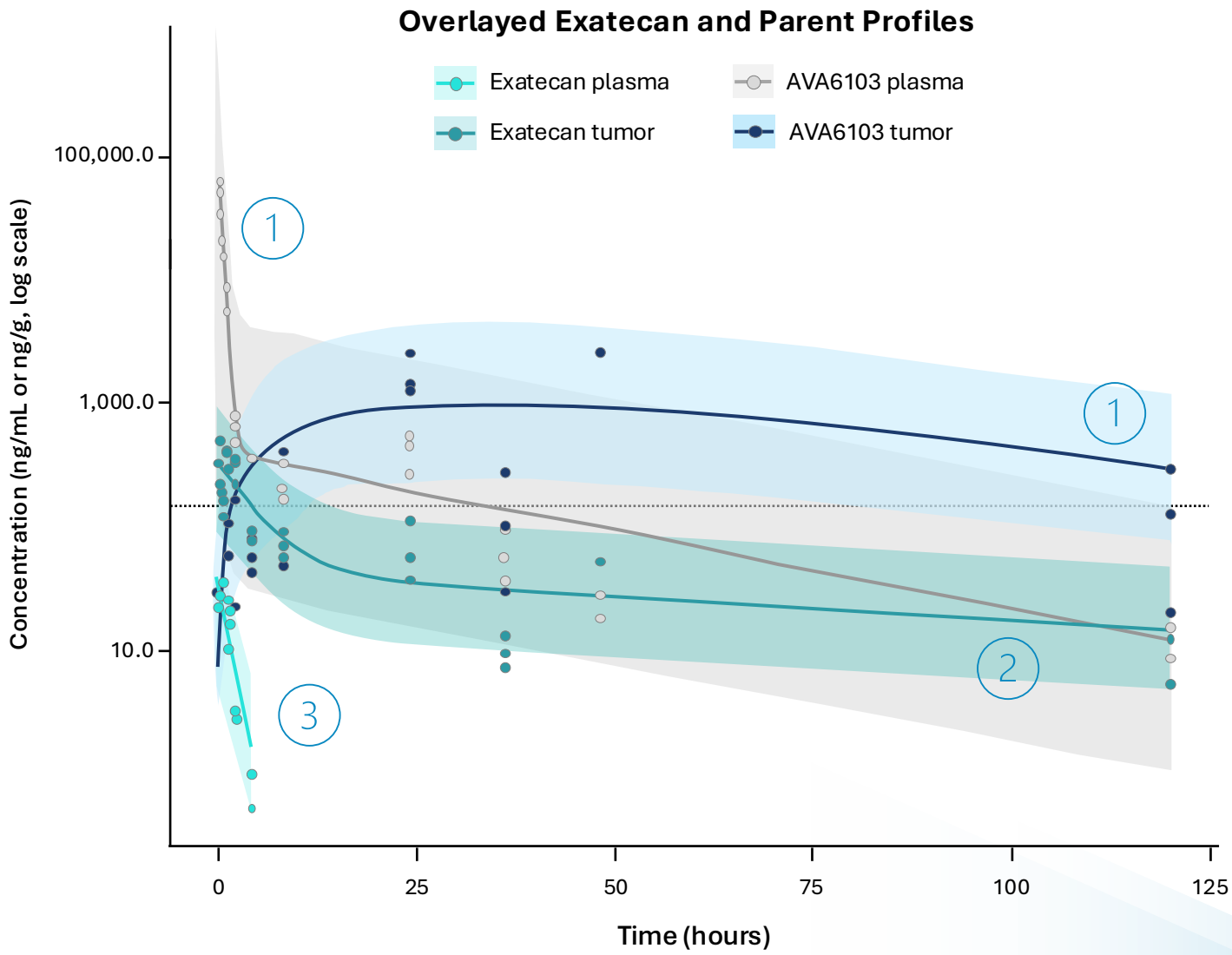
The Controlled Release Mechanism of FAP-Exd (AVA6103) Delivers Exatecan in the Tumor For At Least 5 Days in a Preclinical Model

1. **Payload reservoir:**
The intact PDC disappears from peripheral circulation rapidly and is **detectable in the tumor for at least 5 days**

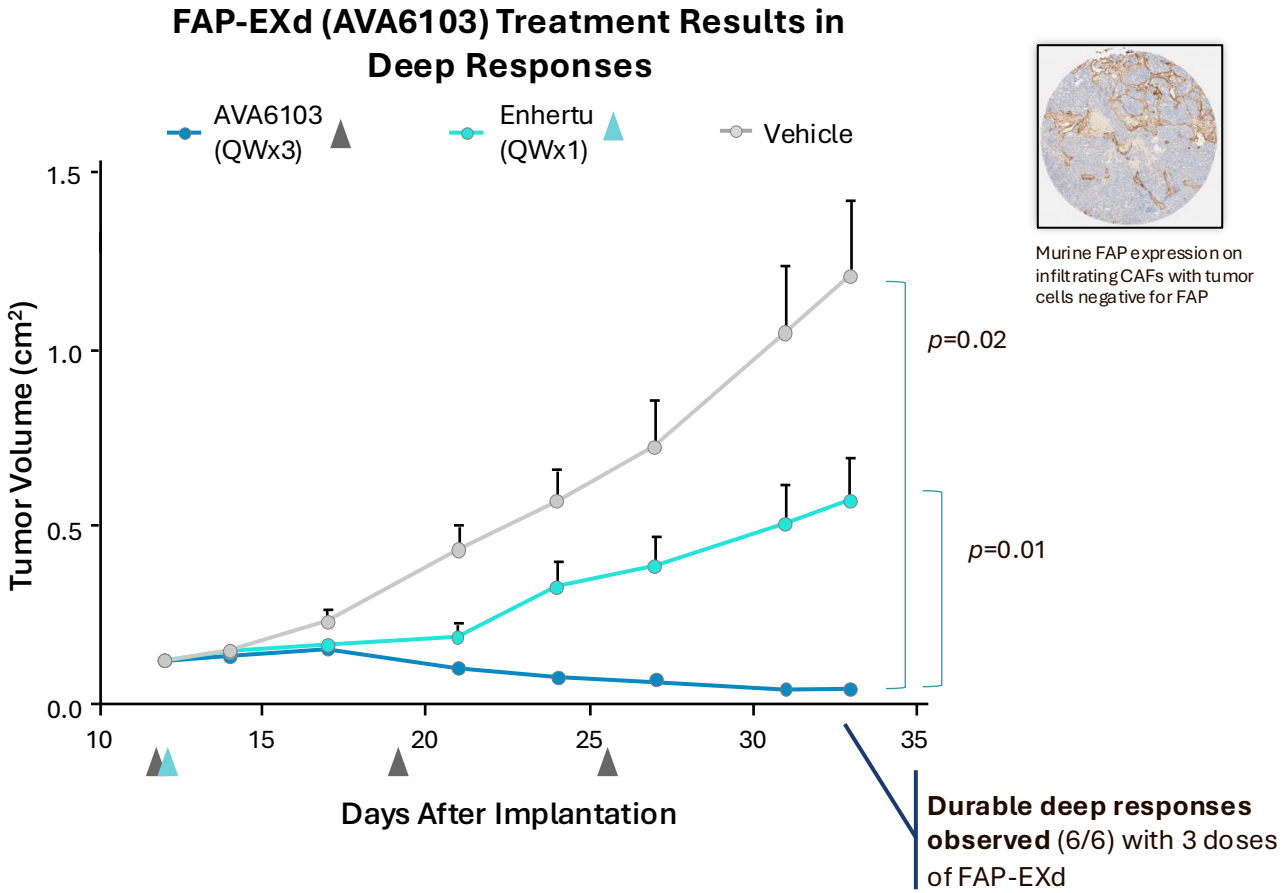
2. **Controlled release:**
Released exatecan is detectable in the tumor within minutes and **released for at least 5 days**

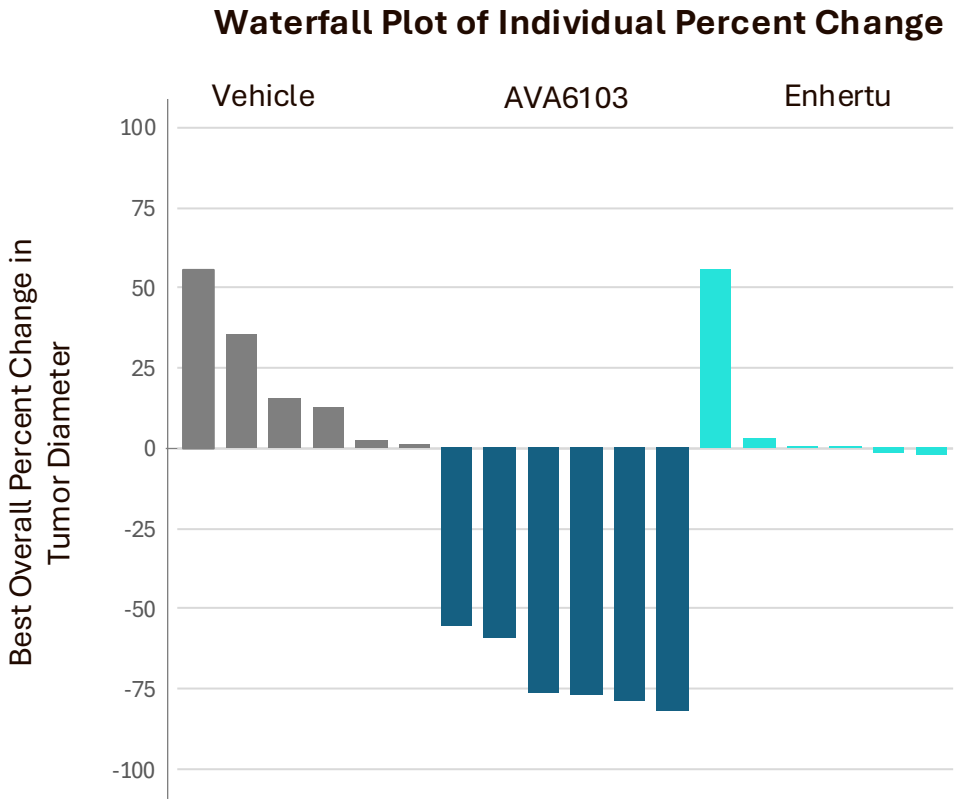
Tumor:plasma:
Plasma levels of released exatecan are not detectable after 4 hours, resulting in **a highly favorable tumor-to-plasma ratio**

3. In human trials, the **plasma assay for exatecan and release peptide** were developed to **detect very low levels of the payload**, assuming the prolonged release in the tumor could be detected in the plasma



FAP-Exd (AVA6103) Demonstrates Better Activity v. Enhertu® in a HER2+ Gastric Cancer PDX Model





FAP-Exd demonstrates superior activity over Enhertu implementing the FAP-Exd Dose Dense regimen
The ability to dose-intensify FAP-Exd is a **key advantage of pre|CISION PDCs over ADCs**

PDX model of HER2+/mFAP+ gastric cancer. . Treatment of established tumors, animals randomized when all tumors >100-150mm^3. Gene expression of PDX models was quantified from RNAseq data. The data was normalized by gene length and library size and presented as TPM values (transcripts per million) using the method of Smid et al. *BMC Bioinformatics*, 2018.

pre|CISION® Tumor-specific FAP Cleavage Mechanism Delivers More Favorable Drug PK Profile than HER2 or TROP2 ADCs

Rapid Tumor Penetration[1]

TUMOR T_{max}: MINUTES v. 24 HRS

CAFs begin **releasing exatecan on contact** with pre|CISION medicines

C_{max} of Free Payload in Tumor

TUMOR C_{max}: Up to 40x HIGHER[2]

Exatecan in the tumor is up to **40x higher** than deruxtecan[1,2]

Tumor Selectivity Index (TSI)*

TSI: NEARLY 3X HIGHER

The TME functions as a **payload reservoir** as plasma exposure decreases

*TSI is defined as the ratio of tumor to plasma payload exposure AUC0-14d [tumor/plasma] and provides a straightforward, quantitative measure of how preferentially a compound distributes to tumor tissue relative to systemic circulation. All data are based on a single dose of AVA6103 v. a single dose of the ADC comparator in antigen-high models.

[1]**Vasalou C**, et al. Quantitative evaluation of trastuzumab deruxtecan pharmacokinetics and pharmacodynamics in mouse models of varying degrees of HER2 expression. CPT Pharmacometrics Syst Pharmacol. 2024 (6):994-1005. doi: 10.1002/psp4.13133 (AZ nonclinical data); | [2]**Okajima, D** et al. Datopotamab Deruxtecan, a Novel TROP2-directed Antibody–drug Conjugate, Demonstrates Potent Antitumor Activity by Efficient Drug Delivery to Tumor Cells. Mol Cancer Ther 2021;20:2329–40 doi: 10.1158/1535-7163.MCT-21-0206

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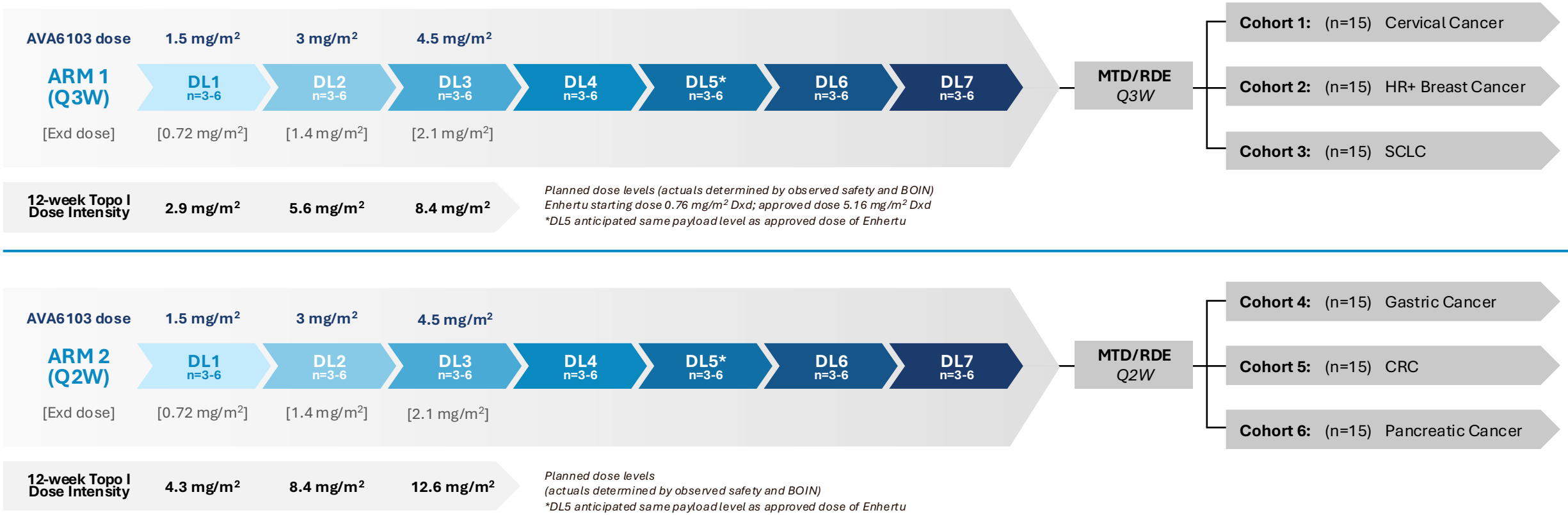
FAP-Exd (AVA6103): Phase 1 Trial Design and Patient Population

Phase 1A Dose Escalation (Parallel Arms)

Phase 1B Dose Expansion Cohorts To Be Determined At RDE



Phase 1 Design: Same payload starting dose as Enhertu Ph I | BOIN design | Parallel Q2W and Q3W arms
Six indications: PDAC, CRC, Gastric/GEJ, Cervical, HR+ Breast cancer, Small cell lung cancer (SCLC)

BOIN=Phase 1 Bayesian optimal interval design; DCR=disease control rate; DL=dose level (actual dose levels determined by safety and statistics (BOIN); DoR=duration of response; FAP=Fibroblast activation protein; GC/GEJ=gastric cancer and gastroesophageal cancer; MTD=maximum tolerated dose; ORR=objective response rate; OS=overall survival; PDAC=pancreatic ductal adenocarcinoma; PFS=progression free survival; Q2W/Q3W=every 2 or 3 weeks; RDE=recommended dose for expansion; SCLC=Small cell lung cancer.

AVA6103 Phase 1: Baseline Characteristics and Disposition

	Dose Level 1 1.5 mg/m^2 0.72 mg/m^2 exatecan n=5	Dose Level 2 3 mg/m^2 1.4 mg/m^2 exatecan n=7	Dose Level 3 4.5 mg/m^2 2.3 mg/m^2 exatecan n=7
Age, median (range)	62 (46-75)	47 (44-64)	58 (42-68)
Sex, (M/F)	4/1	1/6	2/5
ECOG, (0/1)	1/4	2/5	1/6
Prior regimens, median (range)	4 (1-5)	4 (2-8)	5 (2-8)
Heavily pretreated[1]	5/5	7/7	4/7
Prior Topoisomerase I inhibitor treatment, n	Irinotecan (4); Enhertu (1)	Irinotecan (5); None (2)	Irinotecan (4); Enhertu (1); Trodelvy (1); None (1)
Indications	PDAC (4) Gastric (1)	PDAC (1) CRC (4) Cervical (2)	PDAC (2) CRC (2) Breast (3)
Patients ongoing in treatment, n	2/5	5/7	7/7
Patients pending dosing[2], n	0	0	0
Discontinued, n	3	2	0
Patients PK evaluable, n	4	7	5
No. of Cycles of PK evaluable (C1, C2, C3+)	5, 3, 2	7, 6, 3	5, 0, 0

Data cutoff 10 September 2026.

[1] Heavily pretreated defined per Roswinsky et al. (At least 4 cycles of prior platinum-based chemotherapy).

[2] Patients pending dosing have been consented and assigned a slot in the escalation cohort or a backfill slot.

The Safety Profile of AVA6103 Is Highly Favorable Compared with Enhertu® and Exatecan Across the First Three Dose Levels

	Neutropenia	Thrombocytopenia	Nausea/Vomiting	Anemia
AVA6103	**0%**	**5%**	**5%**	**16%**
Enhertu[1]	22%	11%	44%	11%
Exatecan[2]	65%	43%	67%	46%

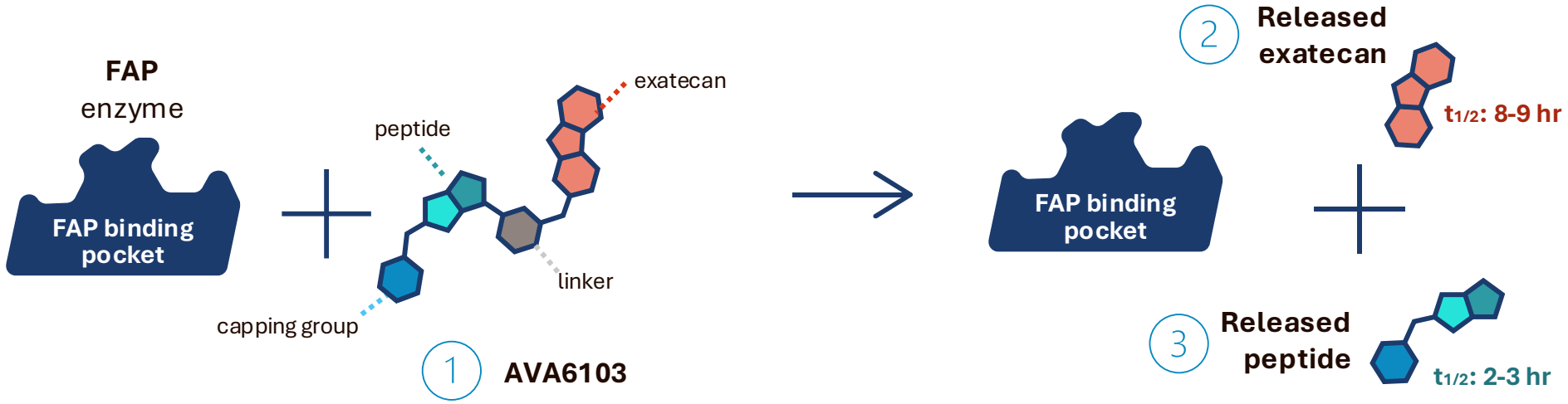
The doses administered in AVA6103 and **Enhertu Phase 1 trials** are similar in absolute payload administered and the third dose level of AVA6103 is approximately 50% higher than the **maximum tolerated dose (MTD) of conventional exatecan**[3]

Thus these data can be directly compared for safety and PK findings

Data cutoff 10 September 2026

[1] Phase 1 Enhertu safety in Q3W IV schedule as per Doi et al. 2017. Lancet Onc 18(11):1512-22. doi: 10.1016/S1470-2045(17)30604-6. One case of febrile neutropenia reported as neutropenia. | [2] Phase 1 exatecan safety in QDx5 schedule per Rowinsky et al. 2000. *JCO* 18(17):3151-63. doi:10.1200/JCO.200.18.17.31. Safety measured by courses of therapy, reported percent of courses per Rowinsky (2000) at the MTD in patients heavily pretreated (HP), 0.3 mg/m^2 QD. HP defined as >4 courses prior platinum therapy. Nausea and vomiting reported as a single event across doses. All patients treated with AVA6103 are considered HP | [3]The safety data are reported as percent of patients experiencing treatment-emergent adverse events of any CTCAE grade across AVA6103 (n=19 pts), Enhertu x 3 dose levels (n=9 pts) and exatecan x 2 MTD dose levels QDx5 (n=13)

Plasma PK Assays in the Clinic Designed to Detect the Controlled Release Mechanism of AVA6103



The clinical plasma PK assays are designed to detect very low levels of the reaction products (exatecan and peptide) released in the bloodstream in the controlled release mechanism

Enzyme and substrate efficiency is described by the ratio of K_{cat}/K_m (very high with AVA6000, very low with AVA6103). The chemical design of controlled release includes low KM (robust binding of the pre|CISION drug to FAP) and low to moderate Kcat (which determines the rate of the cleavage reaction). Half-life of exatecan is published (Rowinsky, et al. 2000) and peptide estimated from AVA6000 where cleavage all occurs in rapid succession.

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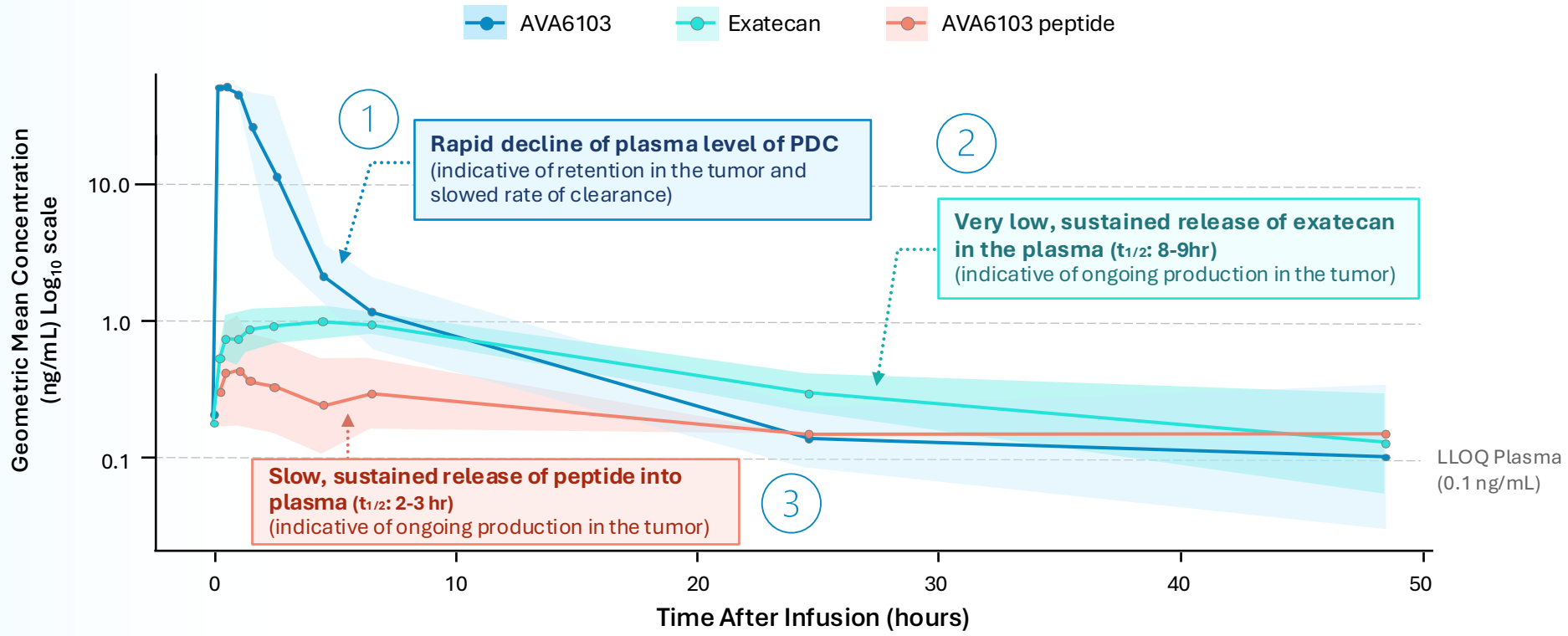
Phase 1 Data Show the Controlled Release Mechanism of AVA6103 is Working in Patients as Designed

Preliminary PK Profile for Cohort 1 Patients

(Cycle 1, n=3 evaluable)

1.5 mg/m^2 dose of AVA6103 administered I.V. over 30 minutes



The **reduction of the k_{cat}/K_m to create the controlled release mechanism** results in changes to the plasma PK of the PDC (1) and the released products (2, 3) of the biochemical reaction (FAP cleavage)

Controlled production of both products of the chemical reaction (exatecan and peptide) is observed for at least 48 hours

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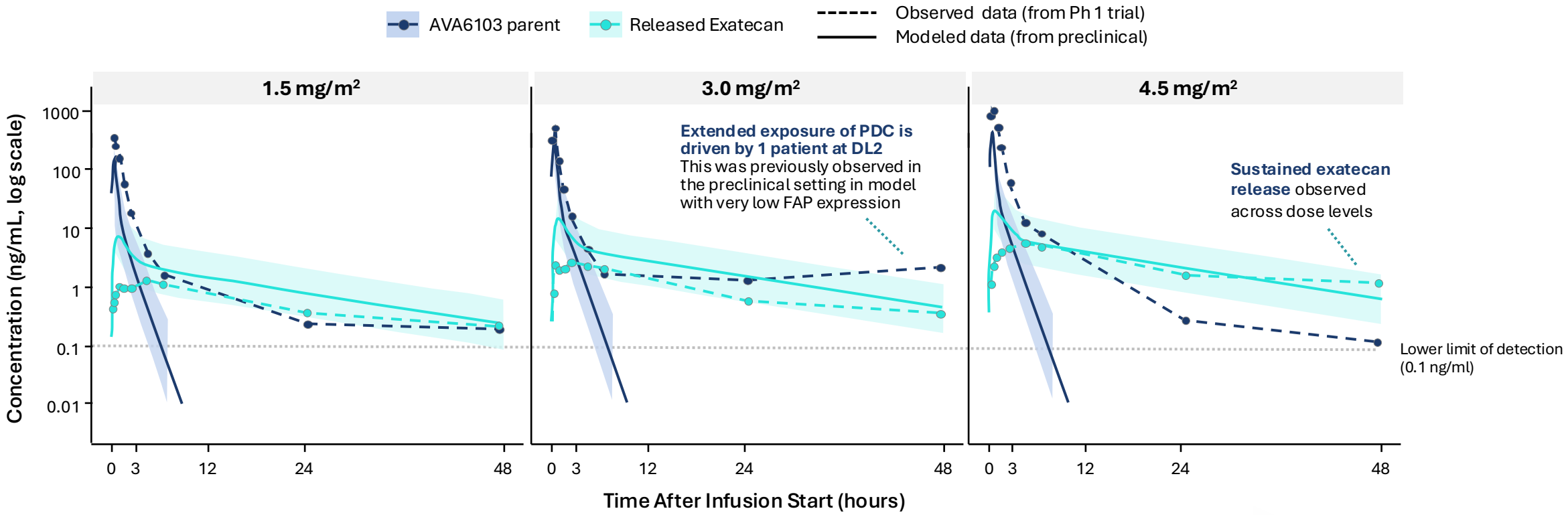
The AVA6103 Phase 1 Plasma PK Replicate the Preclinical Modelled Profile for Controlled Release of Exatecan at First 3 Doses

Observed v. Simulated AVA6103 Parent and Released Exatecan by Dose

colours panels show 1.5, 3 and 4.5 mg/m2 separately; parent and metabolite remain overlaid; Kcat factor = 0.3758; simulation CV = 39%



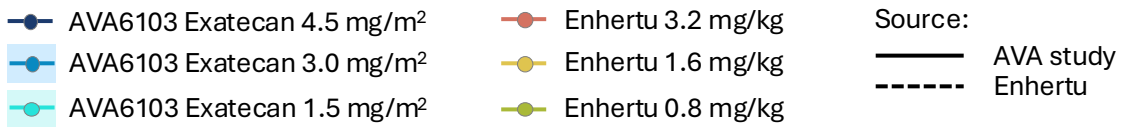
Comparing **preclinical modeled PK** and **clinical trial PK** demonstrates a clear alignment has emerged in the patients and dose levels evaluated to date

These findings increase confidence that the **nonclinical-to-clinical translation is performing as intended**

AVA6103 Releases ~3 Times *Less* Exatecan in Plasma Compared With Equivalent Payload Dose of Enhertu®

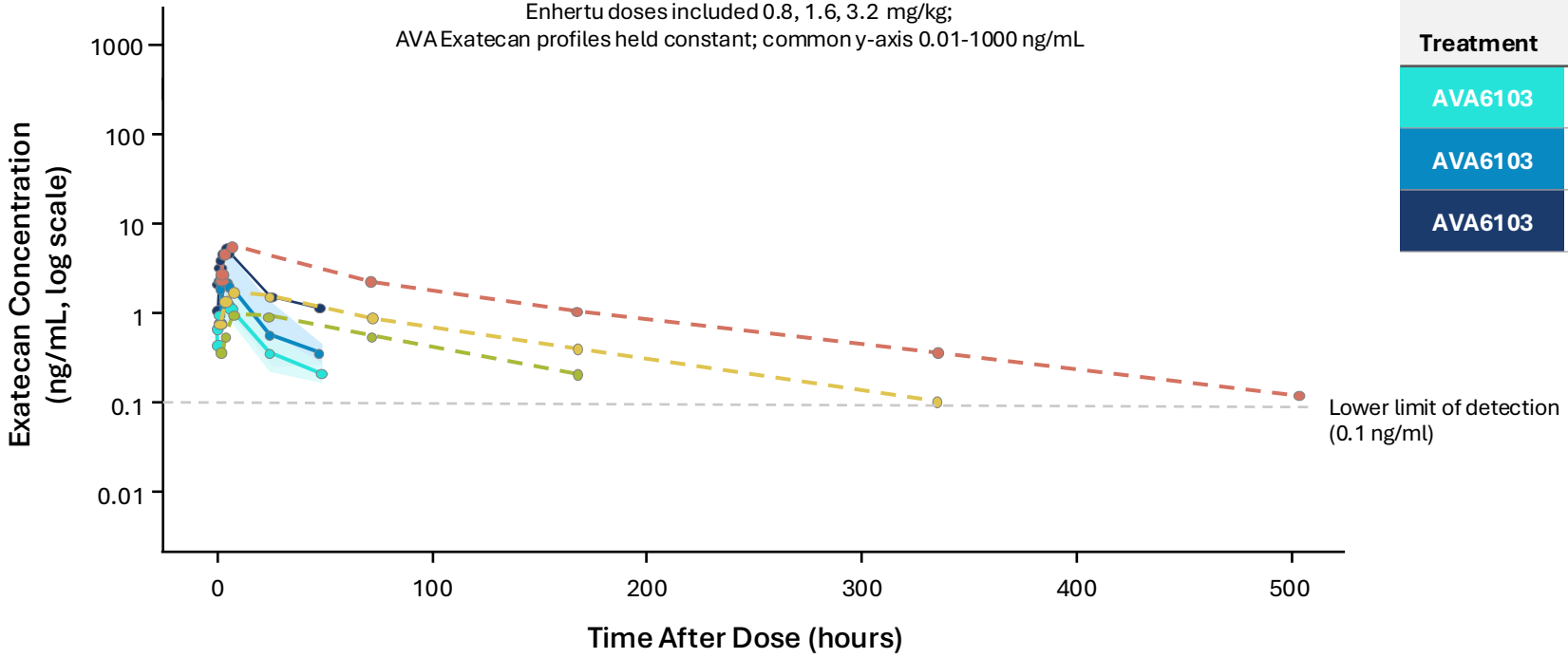
Exatecan and Deruxtecan Concentration:Time Profiles (Log scale)[1]

Exatecan mean with 16th-84th percentile bands, samples below the LLOQ are not displayed for either compound



AVA6103-released Exatecan vs Cumulative Enhertu Profiles: Step 2

Enhertu doses included 0.8, 1.6, 3.2 mg/kg;
AVA Exatecan profiles held constant; common y-axis 0.01-1000 ng/mL



Comparison of Exd Release v. Dxd Release AUC at Two Dose Levels [1]

Treatment	Dose	Equivalent Payload Dose	AUCinf (ng*/h/mL)	Fold Change (v. Enhertu)
AVA6103	1.5mg/m²	0.72 mg/m²	33.0	3.38X
AVA6103	3.0mg/m²	1.4 mg/m²	53.9	3.9X
AVA6103	4.5 mg/m²	2.12 mg/m²	149.9	~3.0[2]

[1] Phase 1 Enhertu Pharmacokinetics in study J101 Q3W IV schedule as per Yin et al. 2020 . | [2] AUC fold change adjusted for slight difference in absolute payload dosing at DL3

Next Gen Controlled Release pre|CISION®: The Path Forward

Phase 1 safety profile of AVA6103 in the clinic is HIGHLY FAVORABLE
compared to both conventional exatecan and Enhertu®,
assessed at similar dose levels in the first cohorts

The comparison of the **preclinical modeled PK** and **clinical trial PK** demonstrates an exceptional alignment through the first 3 dose levels with **controlled release of exatecan evident in patients** for days after dosing

The remarkable consistency of these results with predictive PK modeling based on preclinical studies **increases our confidence that AVA6103 is performing as intended**, and that the robust preclinical data will translate to the clinic

Data demonstrate that a clear mechanistic translation has emerged in the patients evaluated to date
Findings increase confidence that the nonclinical-to-clinical translation is performing as intended

Dose escalation in the trial continues with biopsy and efficacy data to be reported in the near future

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Next Gen pre|CISION: Optimizing the Dual Payload Release Kinetics to Treat Resistant Cancers

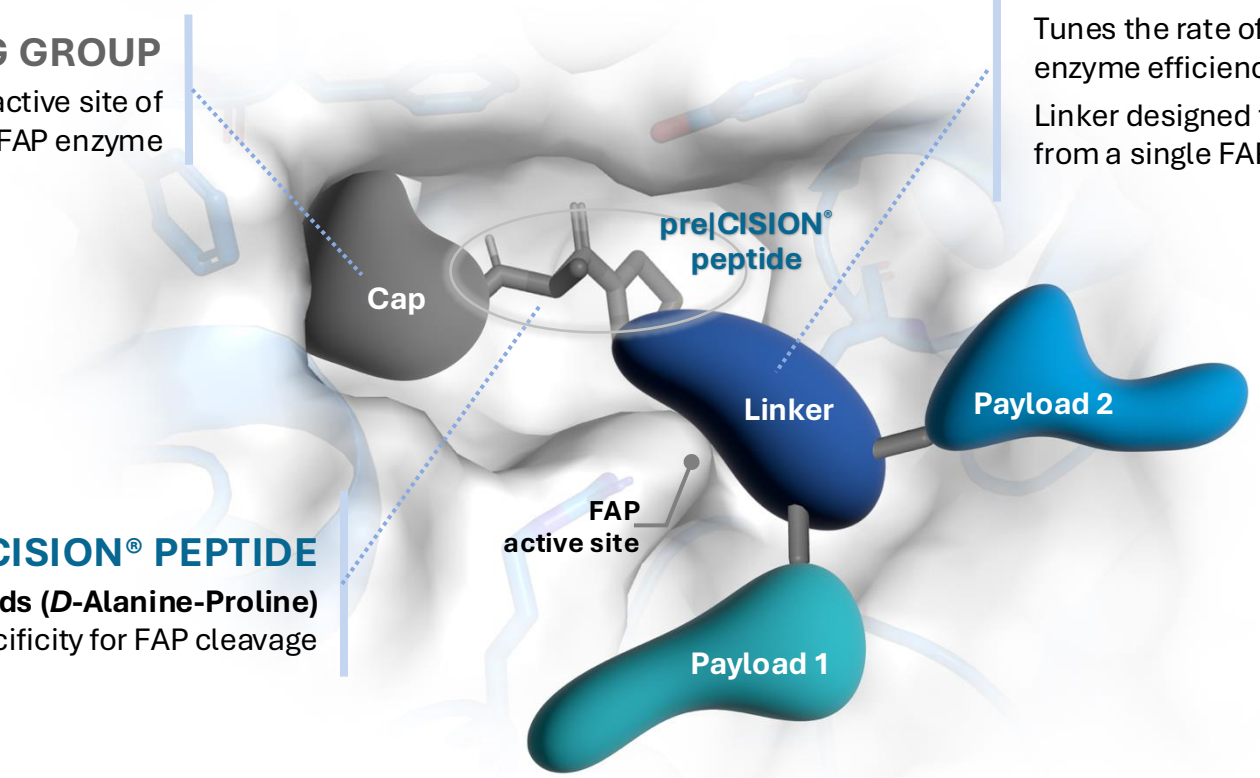
CAPPING GROUP

Sits in the active site of the FAP enzyme

SELF-IMMOLATIVE LINKER

Tunes the rate of peptide cleavage by modulating the enzyme efficiency (k_{cat}/K_M)

Linker designed to **allow two payloads to be released** from a single FAP cleavage event



pre|CISION® PEPTIDE

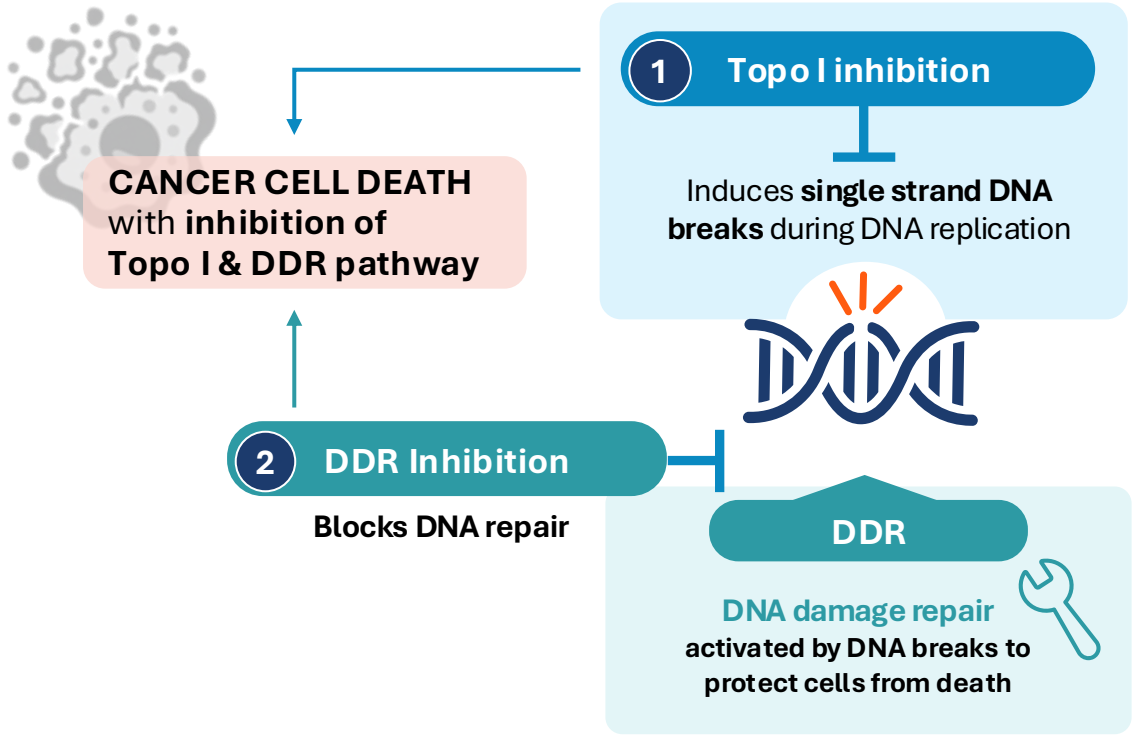
The **two amino acids (*D*-Alanine-Proline)** determine specificity for FAP cleavage

pre|CISION technology is modified to **release two payloads from one cleavage event**

Delivery of the payloads retains the tunable release kinetics

The pre|CISION Dual Payload AVA6207 Treats the Cancer and Prevents Therapy Resistance in One Medicine

The AVA6207 Combination MOA



Combination Clinical Development

- Cancer cells harboring specific genetic changes in the DNA Damage Repair pathways are expected to be highly sensitive to this combination
- These genetic changes are simple to measure in the clinic

There are a number of clinically validated biomarkers that can identify patients with this gen

ATM Loss
NSCLC. PDAC

SLFN11 Loss
SCLC, CRC, Gastric

MSIhigh
CRC

Cyclin CCNE upregulation
Ovarian

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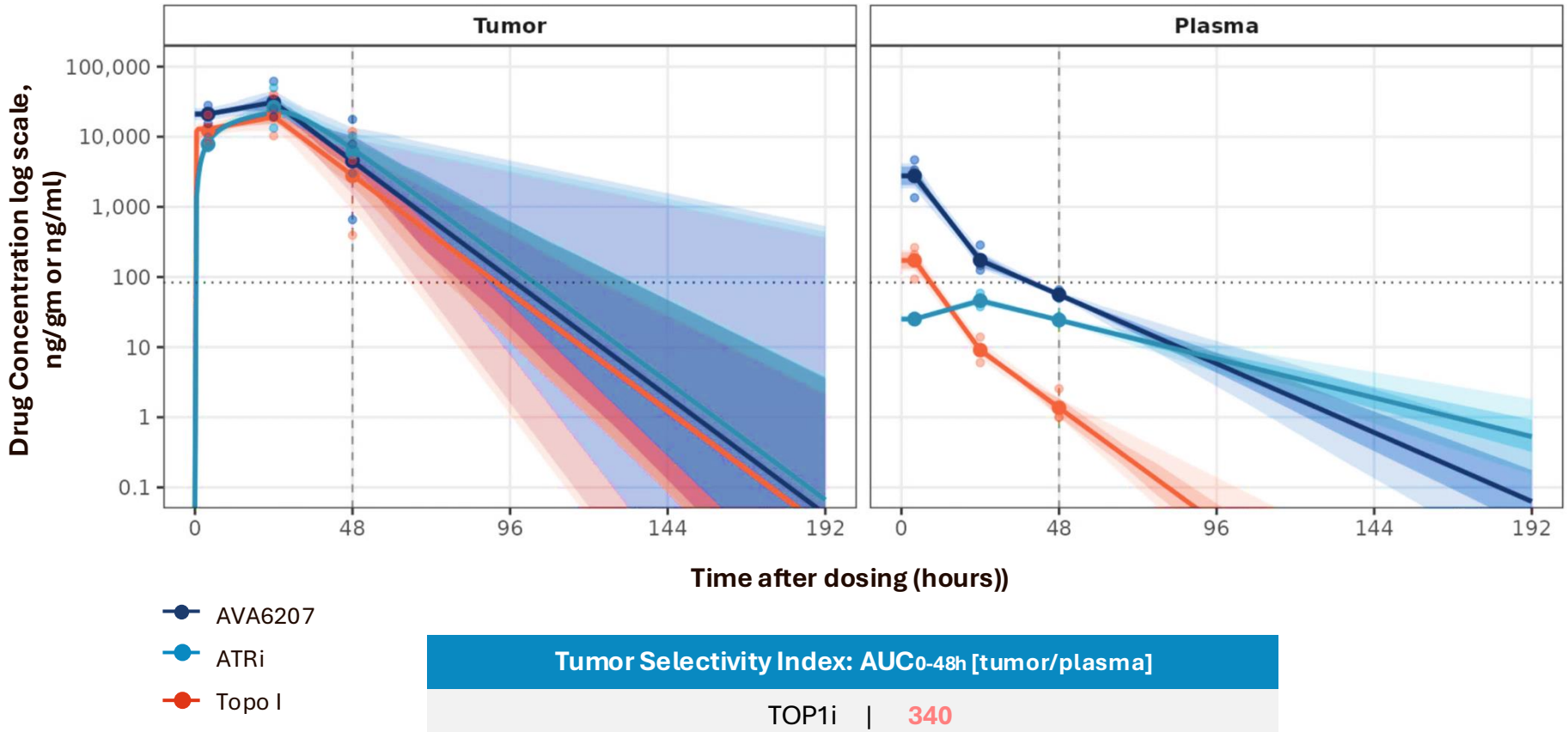
High Intratumoral and Low Plasma Concentrations of TOP1i and ATRi Result in High Tumor Selectivity

- **Levels of dual payload compound-released-TOP1i and -ATRi were assessed** in the tumor and plasma following a single subcutaneous dose of a dual payload compound
- AVA6207 is designed with **high intratumoral concentration** of TOP1i and ATRi concentrations and **low plasma concentration**
- The Tumor Selectivity Index is calculated for each payload and demonstrates **high selectivity in both payloads released**

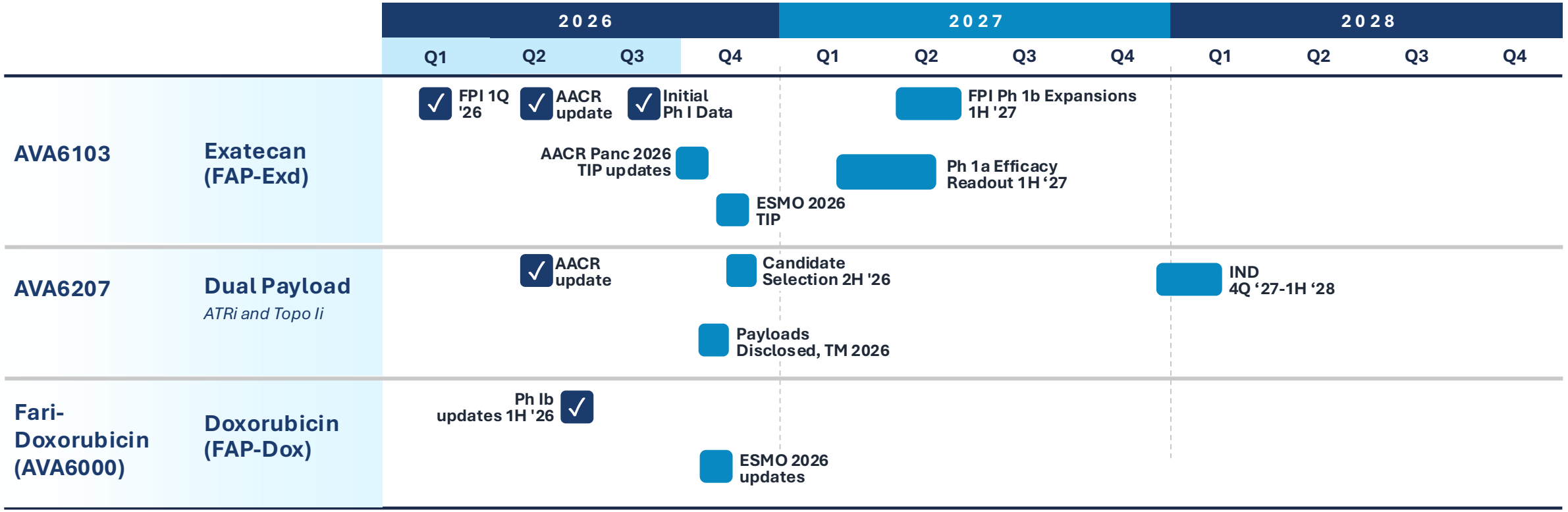


Tumor Selectivity Index: $AUC_{0\text{-}48h}$ [tumor/plasma]	
TOP1i	340
ATRi	527

Plasma and tumor levels of AVA6207 (navy), topo I inhibitor (orange) and ATR inhibitor (teal) were assessed in tumor-bearing mice (HEK-FAP model) following a single dose of 40 mg/kg dosed s.c, Modeling used to predict exposure following the 48 hour timepoint. Tumor selectivity index (TSI) was calculated as the ratio of AUC (tumor)/AUC (plasma). Bootstrapped values up to 48 hours used with 2000 simulations to generate terminal data.

Avacta THERAPEUTICS

Multiple Upcoming Data Catalysts Drive Value Creation



EMSO=European Society for Medical Oncology, Madrid, Spain, 23-27 October 2026; FPI=First Patient In; Ph1a: Phase 1a; SGC=Salivary Gland Cancer; AACR-Panc= AACR Congress on Pancreatic Cancer, San Diego, CA 25-28 September 2026; TIP=Trial in Progress; TM=triple meeting (AACR-NCI-EORTC=International Conference on Molecular Targets and Cancer Therapeutics), Barcelona, November 2026.

The Avacta Leadership Team: Proven Track Record

Christina Coughlin, MD, PhD

Chief Executive Officer

>20 years industry experience

>30 oncology INDs and approvals in oncology, from small molecules to cell therapy

Oncologist and immunologist trained at University of Pennsylvania

Brian Hahn, MBA

Chief Financial Officer

>25 years senior finance and operations experience in biopharma

Led GlycoMimetics' 2014 IPO on Nasdaq

Co-chairman BIO Finance and Tax Committee, served on SEC Advisory Committee on Small and Emerging Companies

David Liebowitz, MD, PhD

Chief Medical Officer

>30 years senior executive experience in clinical development and medical affairs

Hematologist-oncologist trained at University of Chicago and Emory University

Yulii Bogatyrenko

Advisor, Business Development

Principal at Biopharma C&I, leading BD efforts for Avacta

>20 years senior level positions in BD and commercial

Led multiple global drug launches, and numerous industry partnerships

Francis Wilson, DPhil

Chief Scientific Officer

>30 years industry experience

Advanced numerous programs across multiple therapeutic areas

D.Phil. in medicinal chemistry from Oxford University

AMGEN · BAYER · NOVARTIS · Pfizer · Roche · Summit therapeutics · Wyeth

GlycoMimetics, Inc. · IMMUNOCORE · INOVIO · RubiusTherapeutics · xencor · Xenova

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