Avacta THERAPEUTICS

Expanding the reach of highly potent cancer therapies

July 2025

Forward-looking statements

This presentation contains forward-looking statements. Such statements include, but are not limited to, statements regarding the Company's research, preclinical and clinical development activities, plans and projected timelines for AVA6000 and the Avacta pipeline, plans regarding regulatory filings, our expectations regarding the relative benefits of our product candidates versus competitive therapies, and our expectations regarding the therapeutic and commercial potential of our product candidates. The words "believe," "may," "should," "will," "estimate," "promise," "plan", "continue," "anticipate," "intend," "expect," "potential" and similar expressions (including the negative thereof) are intended to identify forward-looking statements. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements.

Risks that contribute to the uncertain nature of the forward looking statements include: our preclinical studies and clinical trials may not be successful; the U.S. Food and Drug Administration (FDA) may not agree with our interpretation of the data from clinical trials of our product candidates; we may decide, or the FDA may require us, to conduct additional clinical trials or to modify our ongoing clinical trials; we may experience delays in the commencement, enrollment, completion or analysis of clinical testing for our product candidates, or in the reporting of data from such clinical testing, or significant issues regarding the adequacy of our clinical trial designs or the execution of our clinical trials may arise, which could result in increased costs and delays, or limit our ability to obtain regulatory approval; our product candidates may not receive regulatory approval or be successfully commercialized; unexpected adverse side effects or inadequate therapeutic efficacy of our product candidates could delay or prevent regulatory approval or commercialization; and we may not be able to obtain additional financing.

These statements are not guarantees of future performance and undue reliance should not be placed on them. Such forward-looking statements necessarily involve known and unknown risks and uncertainties, which may cause actual performance and financial results in future periods to differ materially from any projections of future performance or result expressed or implied by such forward-looking statements.

Although forward-looking statements contained in this presentation are based upon what management of the Company believes are reasonable assumptions, there can be no assurance that forward-looking statements will prove to be accurate, as actual results and future events could differ materially from those anticipated in such statements. The Company undertakes no obligation to update forward-looking statements if circumstances or management's estimates or opinions should change except as required by applicable securities laws. The reader is cautioned not to place undue reliance on forward-looking statements.

Avacta Therapeutics: Pioneering a novel, differentiated class of pre|CISION® medicines to revolutionize drug delivery

Innovative pre|CISION® Platform

- **pre|CISION®** targets delivery of payload directly in the tumor, sparing healthy tissue
- **pre|CISION®** repurposes a range of oncology drugs to significantly reduce toxicity/ side effects for patients
- Very significant market opportunity: **~90% of solid tumors potentially treatable**
- Leveraging AI to capture the full market opportunity in both wholly owned and partnered medicines and drive smarter trials

The pre|CISION® Pipeline

- **AVA6000 (FAP-Dox)** Phase I completed: **dramatic reduction in toxicities** and **encouraging clinical activity** in salivary gland cancer (SGC) and high-grade soft tissue sarcoma (HG-STS). Initial data in expansion cohorts late in 2025 in SGC, HG-STS and triple negative breast cancer
- **AVA6103 (FAP-EXd)**: Innovative chemistry creates a **pre|CISION® -enabled sustained release delivery** of the potent topo I inhibitor exatecan directly in the tumor with planned Phase 1 start in 1Q 2026

Company Positioning

- **AIM-listed** (#AVCT) pure-play oncology therapeutics company with new management and cash runway into Q1 2026 following the divestment of the diagnostics division
- Exploring opportunities for a potential dual listing on NASDAQ
- Experienced management team, located in both London and US
- Intention to explore licensing and partnering opportunities. Avacta is seeking a partnership to develop AVA6000

The Avacta Leadership Team: Proven Track Record

Christina Coughlin, MD, PhD

Chief Executive Officer and Head of R&D

Chris is an oncologist and immunologist, trained at the University of Pennsylvania

She has >18 years of industry experience including >30 oncology INDs and approvals across small molecules and cell therapy in oncology

Brian Hahn, MBA

Chief Financial Officer

Brian has >25 years of senior financial and operations experience in biopharma, including 15 years as CFO of GlycoMimetics, Inc., where he led the company's 2014 IPO on Nasdaq.

He serves as co-chairman of the BIO Finance and Tax Committee, and has also served on the SEC Advisory Committee on Small and Emerging Companies

David Liebowitz, MD, PhD

Chief Medical Officer

David is a hematologist-oncologist who trained at the University of Chicago and Emory University with >30 years of industry experience

He was previously SVP of early-stage clinical development at Inovio Pharma and held senior roles including CMO at Xencor, Vaxart, and Amgen.

Yulii Bogatyrenko

Advisor, Business Development

Yulii is a Principal at Biopharma C&I, leading BD efforts for Avacta. Previously, he held senior level positions in business development and commercial, including having led multiple global drug launches, and numerous industry partnerships at Pfizer/Wyeth, Bayer Healthcare and Teva Specialty Pharmaceuticals

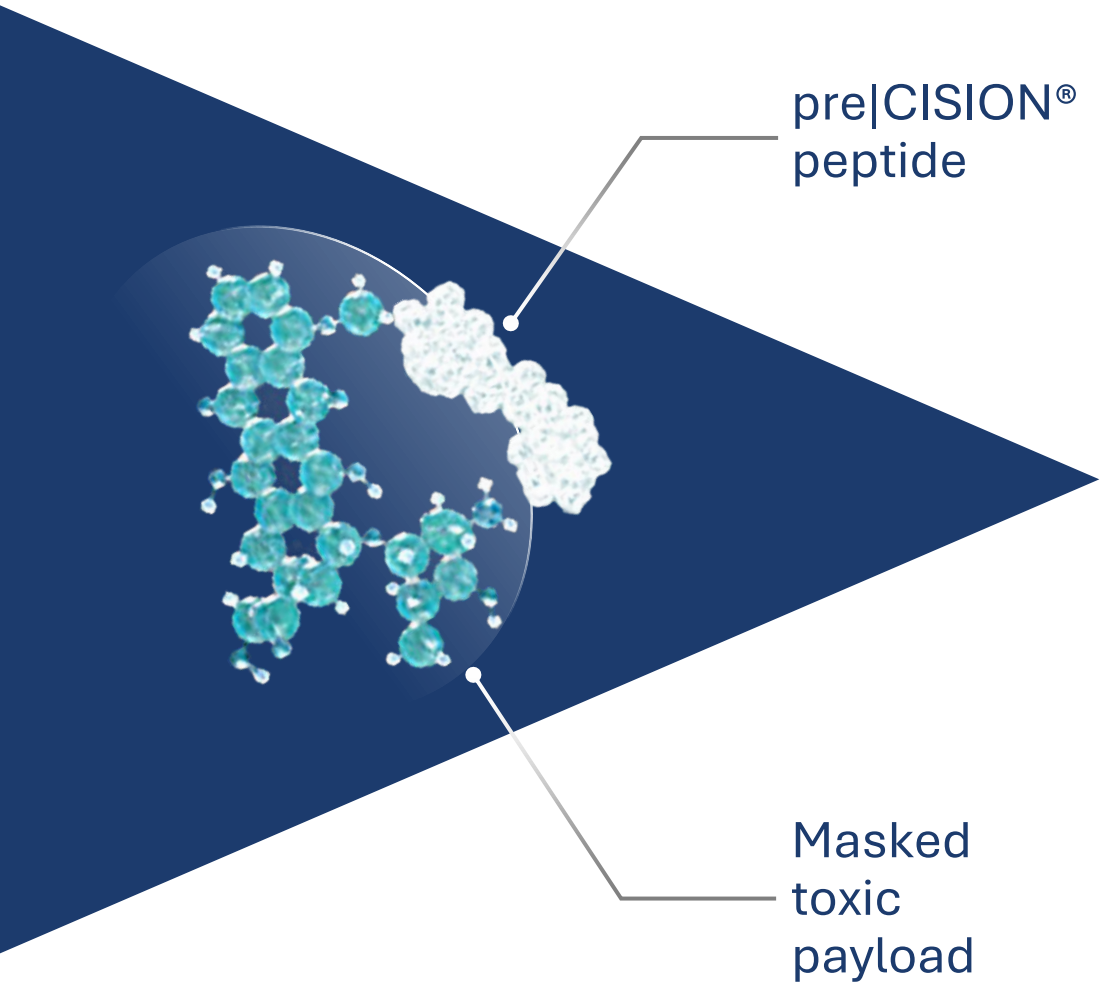
Michelle Morrow, PhD

Chief Scientific Officer

Michelle has >17 years of experience in in oncology research in the biotech and pharma industry

Michelle has significant experience leading discovery and preclinical research teams, including multiple INDs and approvals in oncology

pre|CISION®: FAP-Activated Drug Delivery Platform



Silent in the bloodstream.
Active in the tumor.

Drug release is triggered *ONLY* at the tumor site by FAP, a protein found in tumor-supporting cells

FAP: Fibroblast activation protein

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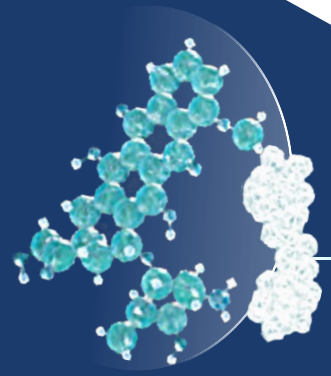
The pre|CISION® Peptide Masks the Toxic Effects of Cancer Drugs and Releases the Active Drug Directly in the Tumor



pre|CISION®
peptide

MASKED
Toxic payload

Masks the toxic effects of a payload in the tissues

Tumor-specific payload release with the FAP enzyme via the bystander effect

Enhances the tumor exposure of payload while limiting the blood exposure

Enables prolonged treatment beyond that permitted with conventional therapy which translates to enhanced survival

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FAP-Dox (AVA6000): Phase 1 Trial Delivers Key Findings

FAP-Dox eliminates the severe cardiac toxicity of doxorubicin

6-20% cardiac tox v. 0%

Dramatically reduces hematologic and GI toxicities of doxorubicin

Limited severe neutropenia

Concentrates released doxorubicin in the tumor 100-fold over plasma

100:1 tumor concentration

Evidence of preliminary activity in salivary gland cancer and sarcoma

Encouraging activity

Phase 1 Trial shows benefits over conventional doxorubicin

[1]Data from Coherent Market Insights, Chemotherapy Market Size and Share Analysis – Global Trends 2025-2032

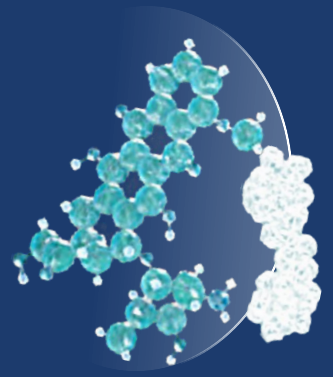
Succeeding where others have failed with FAP Targeting

Traditional
FAP Targeting methods have limited success
via passive targeting

These methods only target the cancer associated fibroblasts, **leaving tumor cells untouched**

Avacta's pre|CISION
FAP Targeting methods having tremendous success
via direct targeting

By leveraging the **Bystander Effect** **pre|CISION medicines will** successfully target & kill cancer associated fibroblasts, and **kill FAP-negative tumor cells**



pre|CISION leverages FAP to activate the drug by cleaving the peptide in the tumor

FAP Radiotherapeutic approaches also leverage the bystander effect to kill tumor cells

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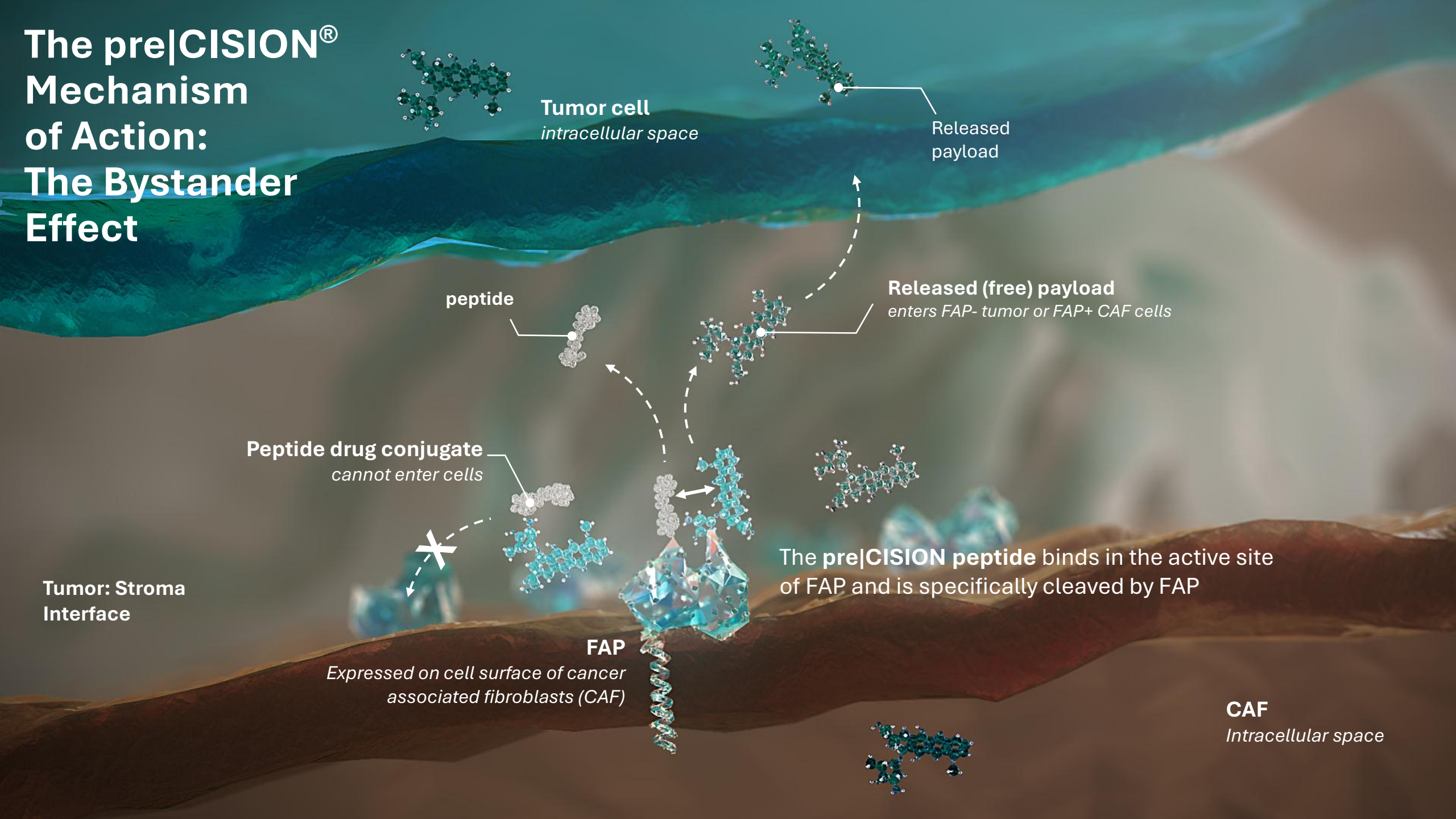
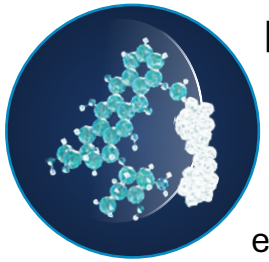
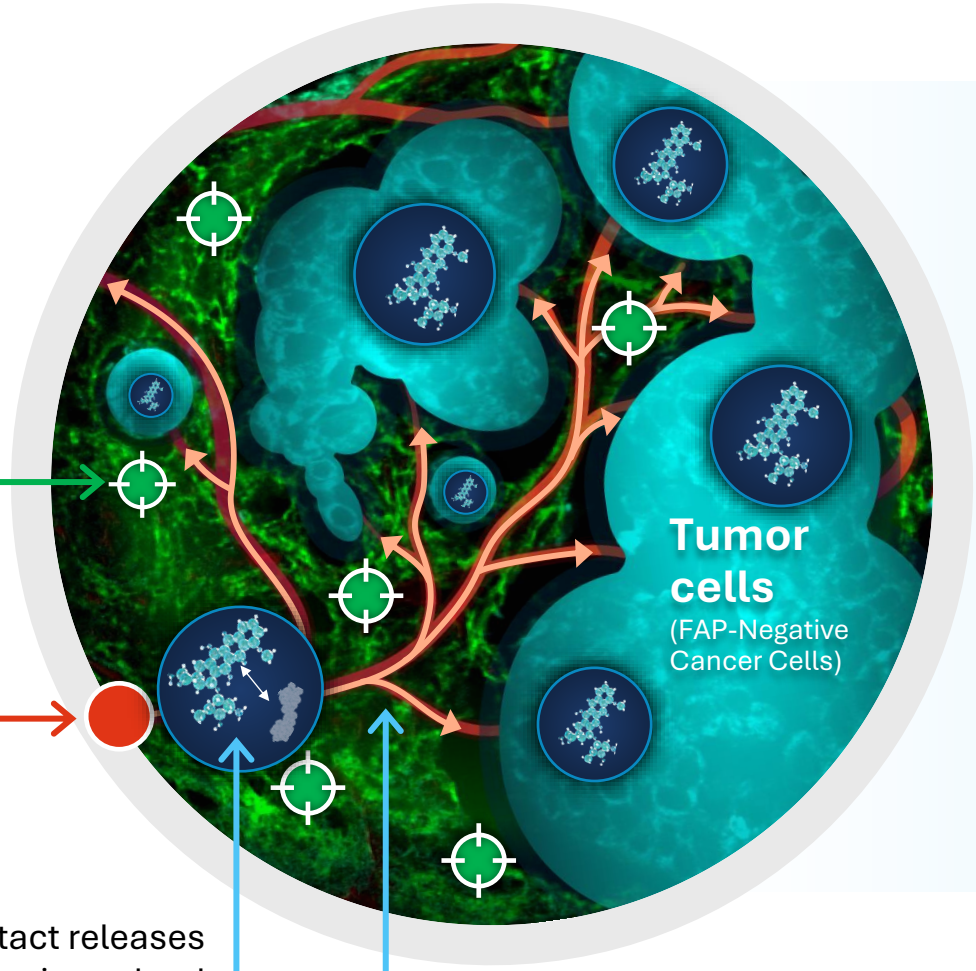
The pre|CISION® Mechanism of Action: The Bystander Effect



The pre|CISION® Bystander Effect



pre|CISION® medicines **target FAP** which is highly expressed in the tumor environment and interwoven with the **tumor's blood supply**



FAP contact releases the toxic payload

Toxic payload distributed to the **tumor cells via the blood supply** ***- The Bystander Effect***

Leveraging FAP in the tumor environment represents a new approach to deliver toxic payloads to the tumor and spare healthy tissue

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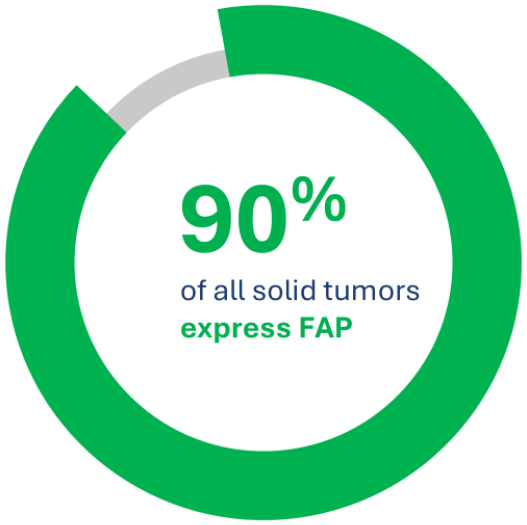
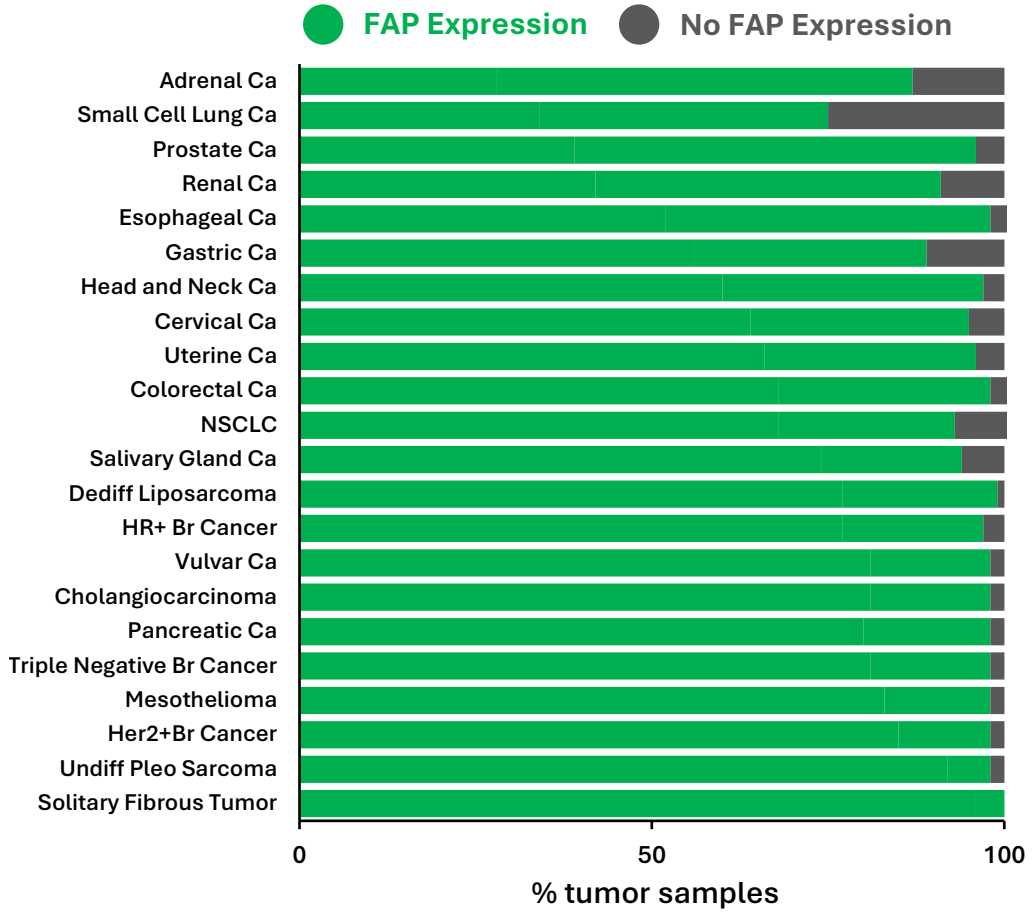
Widespread FAP Expression Across Tumors Supports Broad pre|CISION® Platform Market Opportunity





TEMPUS

Collaboration

Avacta's first two medicines have the **potential to impact over a million lives per year**

Notes: Data in the Tempus AI LENS database were analyzed for expression of FAP. Cut-points to define negative, weak and strong were the same across the entire database and were set based on known/published positive rates for IHC in 3 diseases: gastric cancer, triple negative cancer and SCLC. Generally, negative correlates with 0+ stroma staining, weak expression correlates with 1+ stroma staining, and strong expression correlates with 2-3+ stroma staining. No samples were excluded from the analysis, and total N per indication is indicated in brackets. The lowest expression levels were in hematologic malignancies (data not shown)

FAP-Dox (AVA6000): pre|CISION-enabled doxorubicin

Phase 1 results and path forward

Rationale for Doxorubicin as the First pre|CISION® Payload

1

Doxorubicin is an **approved oncology drug** with a prolonged half life of 35 hrs & known activity in 3 solid tumor indications:

1. Breast cancer
2. Soft tissue sarcomas
3. A subset of head & neck cancer, and salivary gland cancer

2

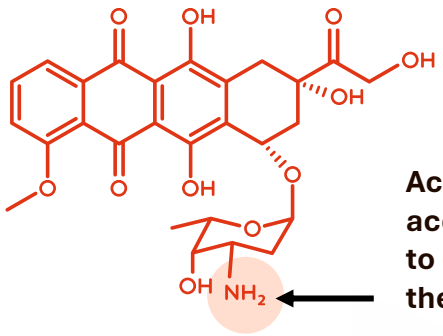
Higher lifetime doses of Doxorubicin cause severe **cardiomyopathy** (known rate of 6-20%). **Limits dosing in the clinic**

Successful implementation of pre|CISION w/ Doxorubicin **should severely reduce or eliminate this toxicity**

3

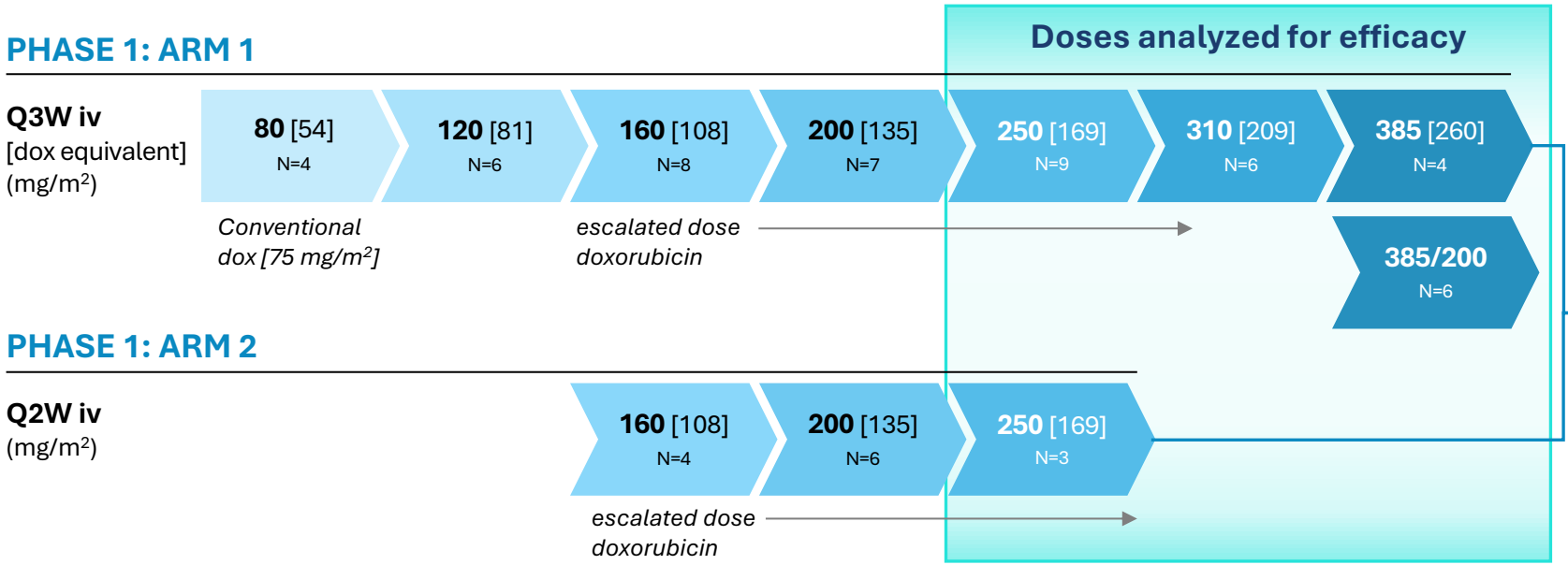
The early chemistry of pre|CISION required an activated & accessible nitrogen to link the peptide which does not occur in most drugs

Doxorubicin chemical structure



Activated, accessible nitrogen to directly conjugate the peptide

FAP-Dox (AVA6000) Phase 1 Trial Design and Patient Population



PHASE 2

Recommended dose for expansion (RDE)
(mg/m²)

EXPANSION COHORTS (PHASE 1B)

Triple Negative Breast Cancer
PD-L1-negative, BRC*Awt*

High-grade Soft Tissue Sarcoma
(UPS, DDLPS, 1L/2L)

Salivary Gland Cancer
(SGC), any histology, 1L/2L

PHASE 1 DOSE ESCALATION: PATIENT POPULATION AND METHODS

Patients

FAP-positive cancers including:

- Sarcoma
- Pancreatic
- Colorectal
- Head & neck

Expression levels

- FAP-high (uniform expression)
- FAP-mid (heterogeneous)

Anthracycline limits

- Prior ≤350 mg/m²
- Trial max ≤550 mg/m²

Primary Endpoint

- Safety (primary)

Secondary Endpoint

- Efficacy by FAP level (secondary)

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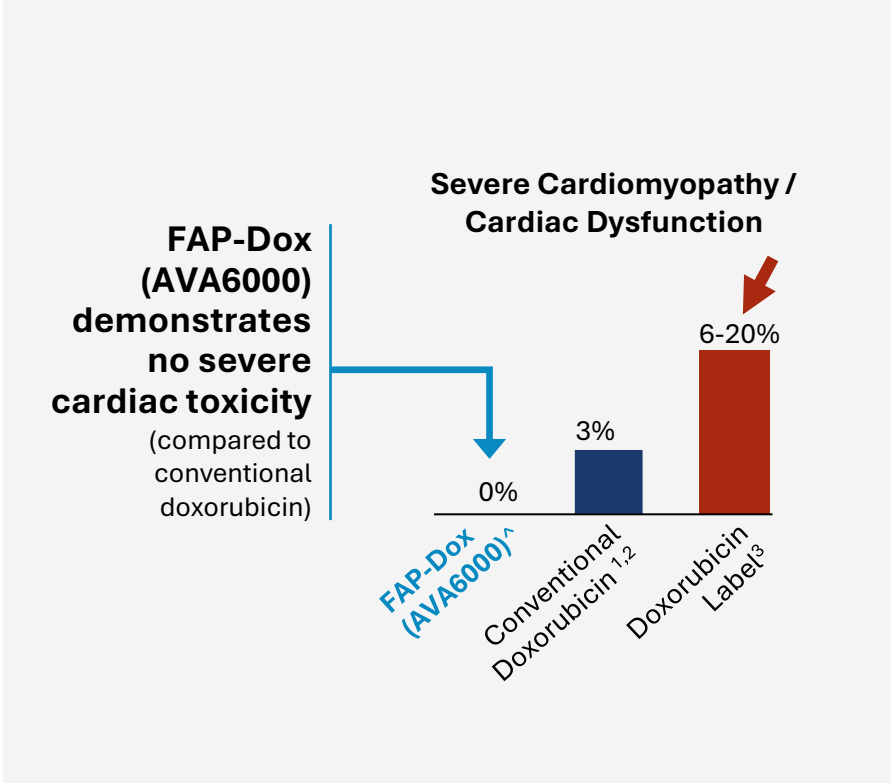
pre|CISION® Eliminates the Severe Cardiac Toxicity of Doxorubicin

FAP-Dox has **no severe cardiac toxicity** despite doses approaching 4x the MTD of conventional doxorubicin (75 mg/m^2)

Bone marrow toxicities are dramatically reduced when comparing FAP-Dox versus conventional dose doxorubicin

Alopecia is generally limited to hair thinning (grade 1) and not complete hair loss

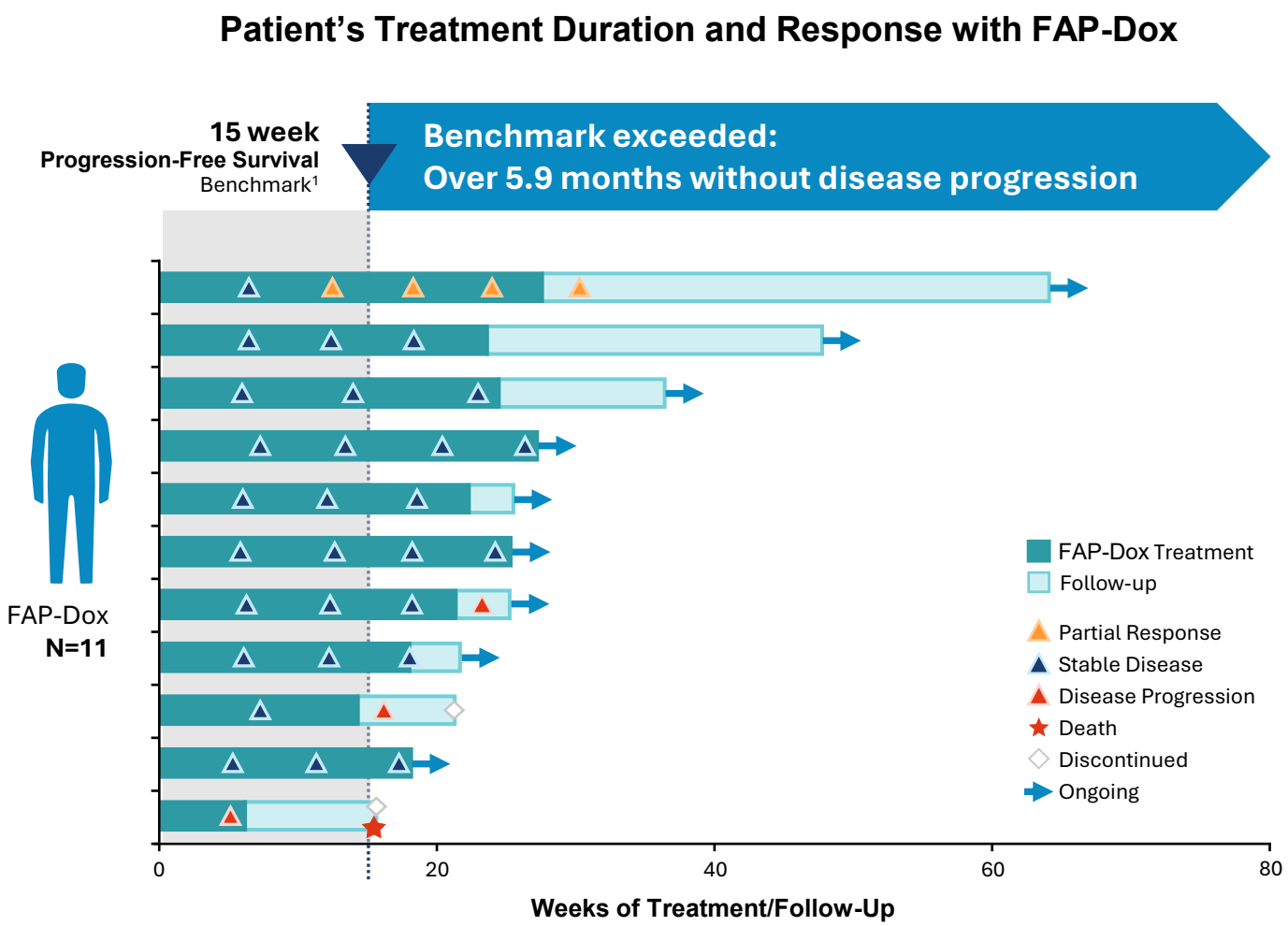
There are no reports of ADC-linked **toxicities associated w/ non-specific release of the payload** (no pneumonitis, ocular toxicity or liver toxicity)



Conventional doxorubicin at the cumulative doses we are using is known to cause severe cardiac dysfunction in 6-20% of patients. FAP-Dox has eliminated this toxicity

Data cutoff 15 February 2025. 1. Tap, WD et al. 2020. *JAMA* 323:1266. 2. Jones, RL et al. 2021. *Clin Ca Res*. 3. Doxorubicin package insert (at 550mg/m2 max)
Updated from Twelves et al. 2024 ESMO Annual Meeting

FAP-Dox Durability & KOL Confidence in Salivary Gland Cancer



"FAP-Dox would completely become standard of care for Adenoid Cystic Carcinoma (ACC), your comparator is lenvatinib which has 12% ORR and horrible toxicity … "

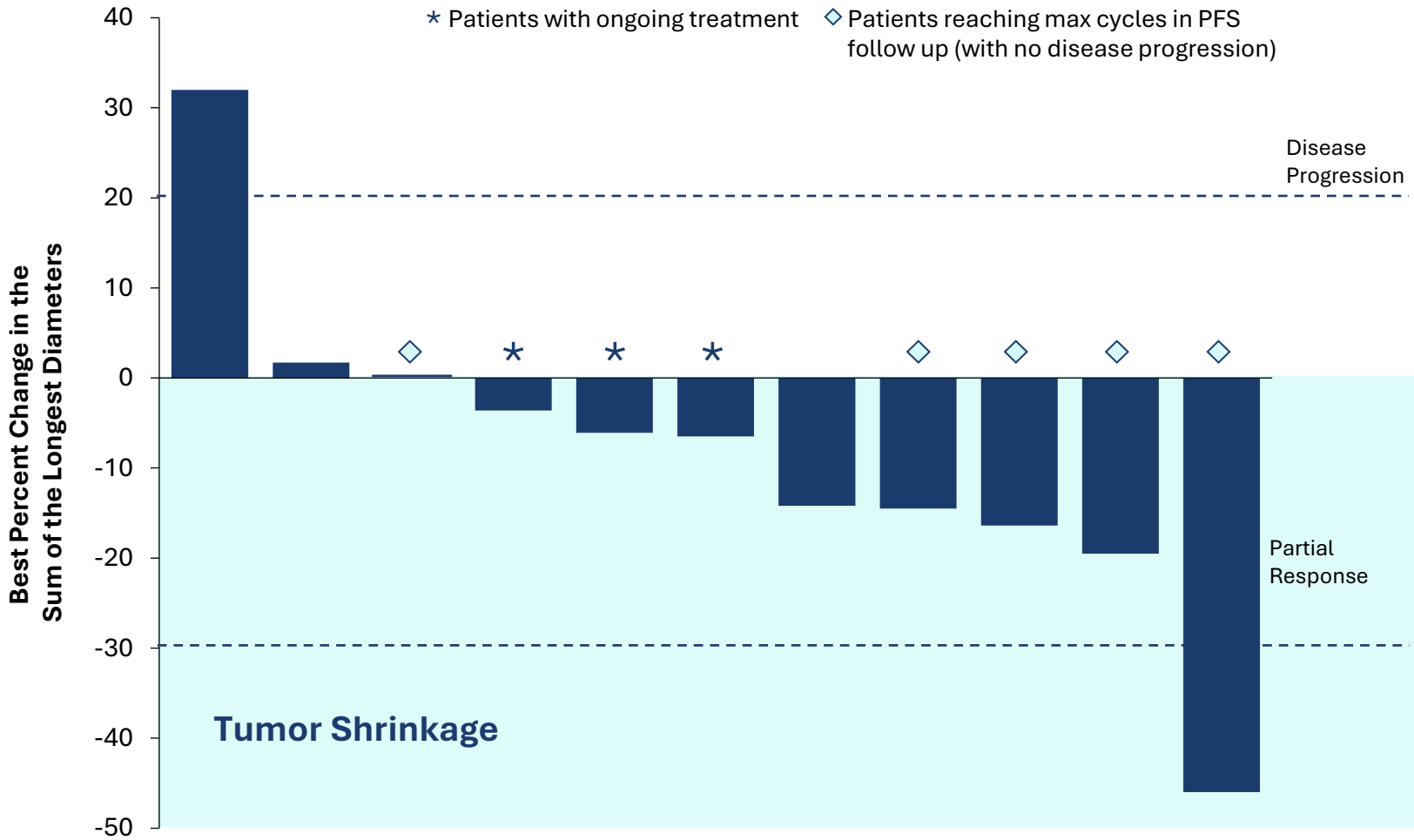
Consultant in Medical Oncology
NHS Foundation Trust

" … I'd use this very widely in 2L+ for patients who hadn't received it yet… there's no approved therapy in this space and with this data there's no reason not to prescribe this … "

Professor of Clinical Medicine
UCSF

Data cutoff 10 April 2025
All pts with the diagnosis of salivary gland cancer treated at or above the 250 mg/m2 dose level, regardless of schedule. Median follow up 25.3 weeks
1. Licitra et al. ESMO 2024.

FAP-Dox: Preliminary Data Demonstrate Durable Tumor Shrinkage in Patients with Salivary Gland Cancers



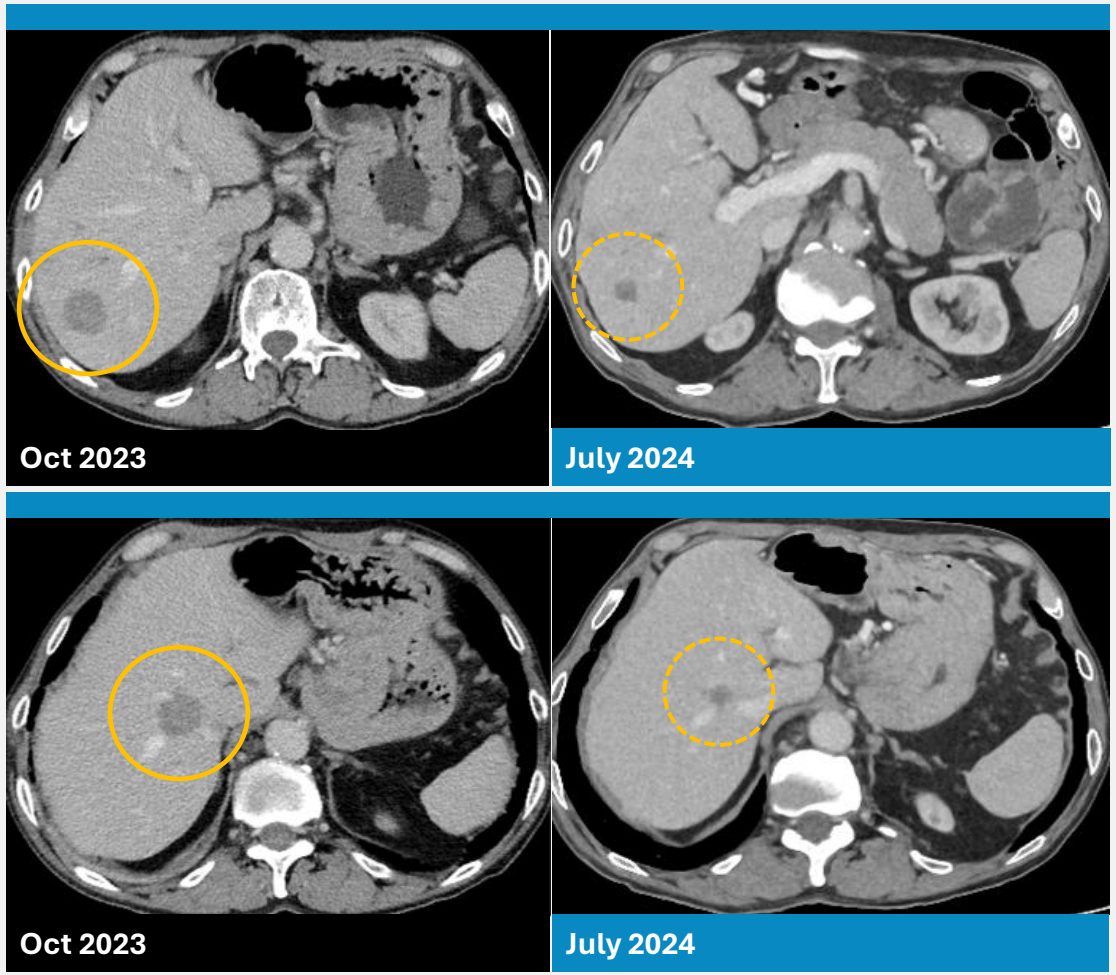
Multiple responders in the trial with durable tumor shrinkage

>90%

disease control rate
in an indication that is reported to be chemorefractory

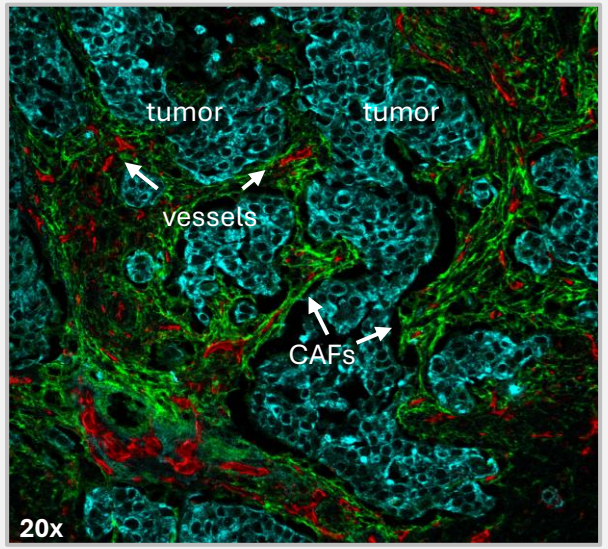
Data cutoff 10 April 2025
All patients with the diagnosis of salivary gland cancer treated at or above 250 mg/m2 regardless of schedule (n=11)

FAP-Dox (AVA6000): Deep prolonged PR in Salivary Gland Cancer



Durable RECIST response in the setting of FAP-negative tumor cells

- 79 yo male with SGC (ductal histology). Prior therapy: triptorelin/ bicalutamide followed by disease progression
- Enrolled in the AVA6000 trial (Oct 2023) in the 385 mg/m2 Q3W cohort; highest level of intratumoral doxorubicin of any patient at 24 hours post-dose
- Confirmed partial response at 12 weeks; duration of response >18 weeks. Pt discontinued after reaching lifetime max of doxorubicin exposure; PR continuing in follow-up



Tumor cells are negative for FAP (aqua)
FAP-positive Cancer Associated Fibroblast (CAF) populations (green) at the tumor-stroma interface with vessels co-localized in stroma (red)

- PanCK (tumor)
- CD31 (blood vessels)
- FAP (CAFs)

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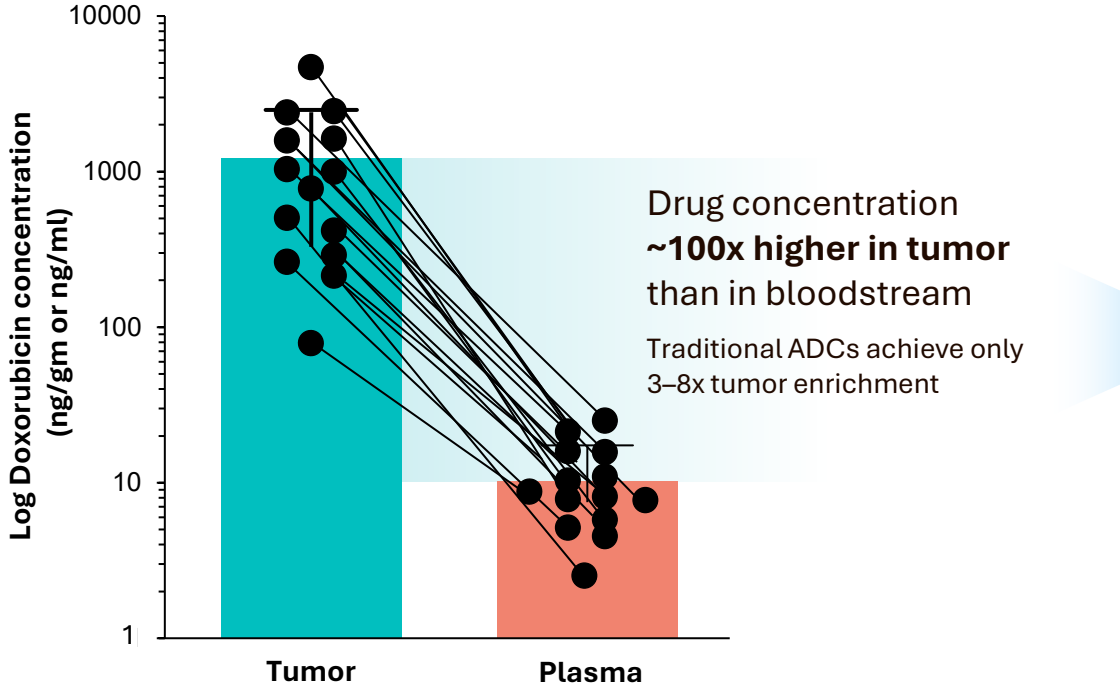
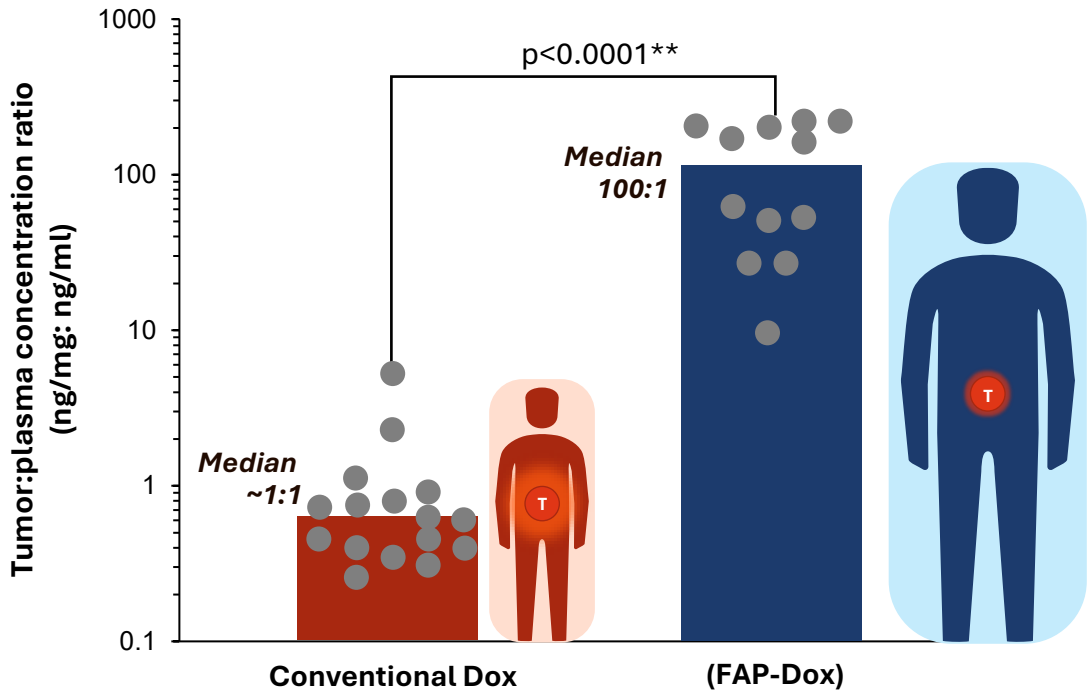
pre|CISION®: FAP-Dox Demonstrates Superior Tumor Targeting

Dox Concentration
24 hr After 1st Dose of FAP-Dox[1]



Tumor to Plasma Ratio of Active Doxorubicin After First Dose[2]



FAP-Dox tumor targeting far exceeds conventional dox.
It achieves 100× higher concentration in the tumor vs. conventional dox, which shows no tumor targeting (~1:1 tumor:plasma)

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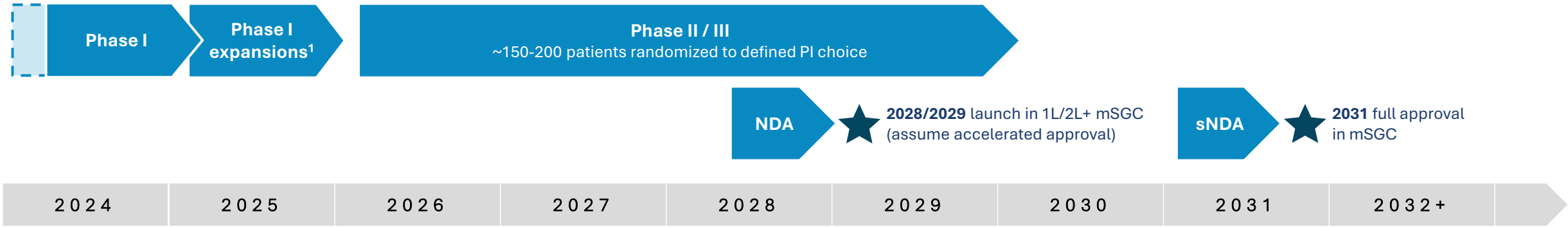
1. Lahu, G et al. AACR 2025, data cutoff 10 April 2025.
2.. Stallard et al. (1990) *Cancer Chemother Pharmacol*. 25,: 286–290

Clinical Development: Rapid route to market in an orphan indication with TNBC to expand the label

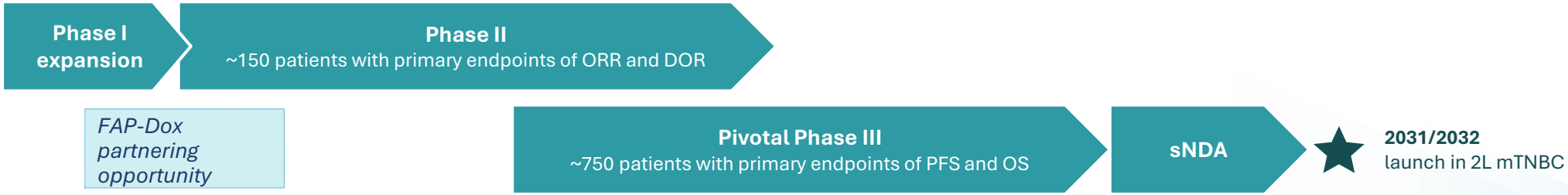
EARLY DEVELOPMENT → **SGC INDICATION: 1st APPROVAL** → **TNBC: 2nd APPROVAL**

Salivary gland cancer (SGC) Indication



Triple Negative Breast Cancer (TNBC)

PD-L1-negative and BRCA*wt*



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Commercial Analysis of FAP-Dox Reveals Advantages to the Initial Orphan Indication Supporting the Breast Cancer Indication

	Salivary Gland Cancer (SGC)		Triple Negative Breast Cancer (TNBC)		HR+ and HER2+ Breast Cancer
Addressable Population	 **1L/2L Indication** **~1000/yr (US) 2500/yr global** FAP-Dox is indicated after hormonal therapy		 **1L/2L (US population estimates)** **~5000/yr** (doxorubicin naïve) *–or–* **~15000/yr** (doxorubicin pretreated)		 **Label expansion opportunities** to adjuvant/neoadjuvant therapy and additional subsets of metastatic breast cancer
Pricing Implications	**Advantages of first indication:** • Orphan pricing • Avoids the IRA		**Unlikely to see diminution in orphan price** given Trodelvy® pricing in this indication		Similar to TNBC Indication
	USA	GLOBAL	USA	GLOBAL	
Launch Timing	**Late 2028/ early 2029**	**2031**	**2031**	**2032**	**2033-2035** (Global)
US-only Peak Revenue	**~$127 M** (early)	**~$250 M** (later)	**~$740M** (doxorubicin naïve population only)	**~$1.5B**	**Analysis ongoing**

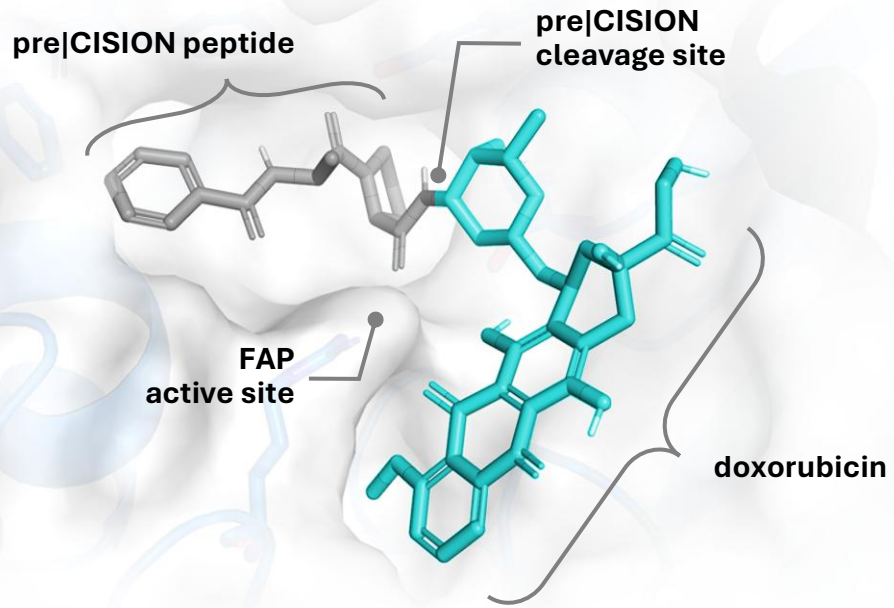
Source: L.E.K. analysis of AVA6000 CDP and commercial opportunities

FAP-Exd (AVA6103): pre|CISION-enabled exatecan

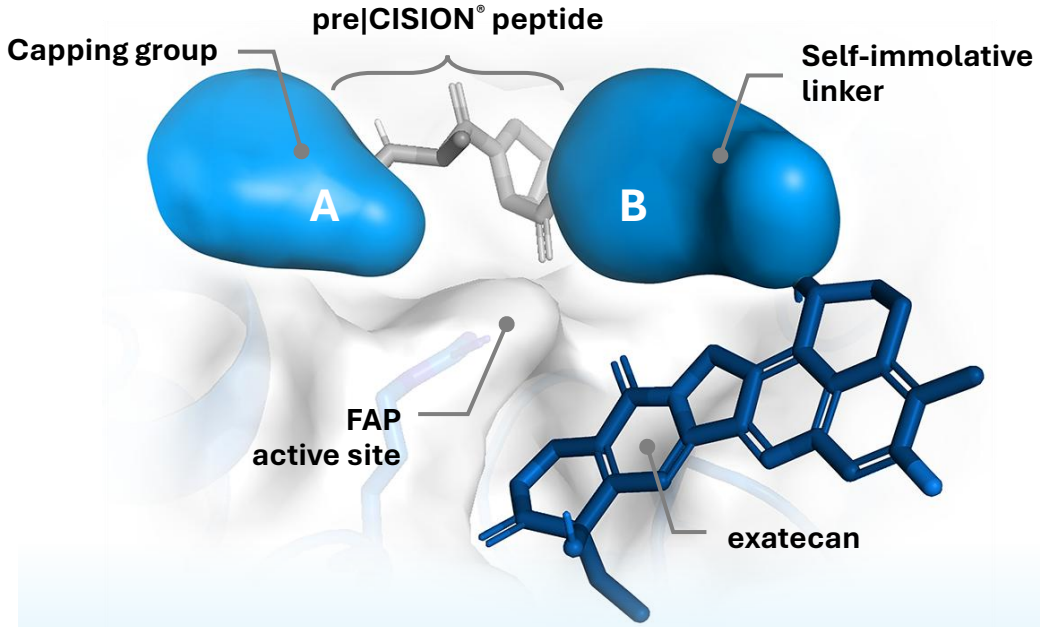
Potent Topoisomerase I Peptide Drug Conjugate

FAP-EXd (AVA6103): Sustained Tumor Release of a Highly Potent Payload via the pre|CISION Platform

pre|CISION-Dox (FAP-Dox) in the FAP Docking Model



pre|CISION-Exd (FAP-EXd) in the FAP Docking Model



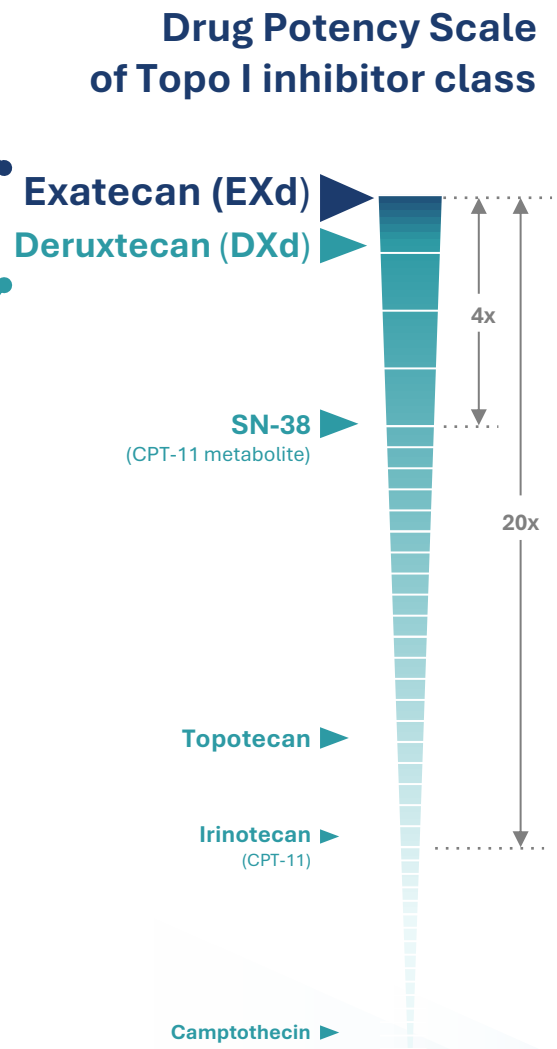
pre|CISION Sustained Release Mechanism: Delivery of payloads with a short half-life for prolonged periods of time directly in the tumor

Exatecan is an ideal payload for the next evolution of the pre|CISION platform: Rationale

1. **Exatecan (EXd) is the most potent topo I inhibitor w/ single agent activity** in Ph 2 trials in several key FAP-positive indications

2. **Deruxtecan (DXd) has similar potency and is an APPROVED highly successful ADC (Enhertu®),** however with a lower membrane permeability compared with exatecan

3. **Exatecan (EXd) previously failed in the clinic due to a limited therapeutic index & significant PK issues**
   - Short half-life of ~9 hours which is insufficient for the effective inhibition of the topoisomerase I enzyme



Kumazawa, E. et al. Potent and broad antitumor effects of DX-8951f, a water-soluble camptothecin derivative, against various human tumors xenografted in nude mice. Cancer Chemother Pharmacol 42, 210–220 (1998). https://doi.org/10.1007/s002800050807 | Esteva, F (2003). A Phase II Study of Intravenous Exatecan Mesylate (DX-8951f) Administered Daily for 5 Days Every 3 Weeks to Patients with Metastatic Breast Carcinoma. Cancer 98(5):900-906. | Abou-Alfa GK et al. Randomized phase III study of exatecan and gemcitabine compared with gemcitabine alone in untreated advanced pancreatic cancer. J Clin Oncol. 2006 Sep 20;24(27):4441-7. doi: 10.1200/JCO.2006.07.0201.

FAP-EXd (AVA6103) Optimizes Tumor-Specific Delivery with Key Attributes

1

Optimized Dosing of exatecan

The **maximum tolerated dose of FAP-EXd is 75x that of conventional exatecan** (30x molar ratio of pure payload) allowing greater tumor concentration

2

FAP-EXd is inert in the absence of FAP

FAP-EXd is completely inert in the absence of FAP+ CAFs unless FAP+ CAFs are present

3

FAP-EXd optimizes sustained tumor release

We observe **high tumor levels of both PDC and released exatecan over multiple days,** whereas conventional exatecan disappears from circulation and the tumor in hours

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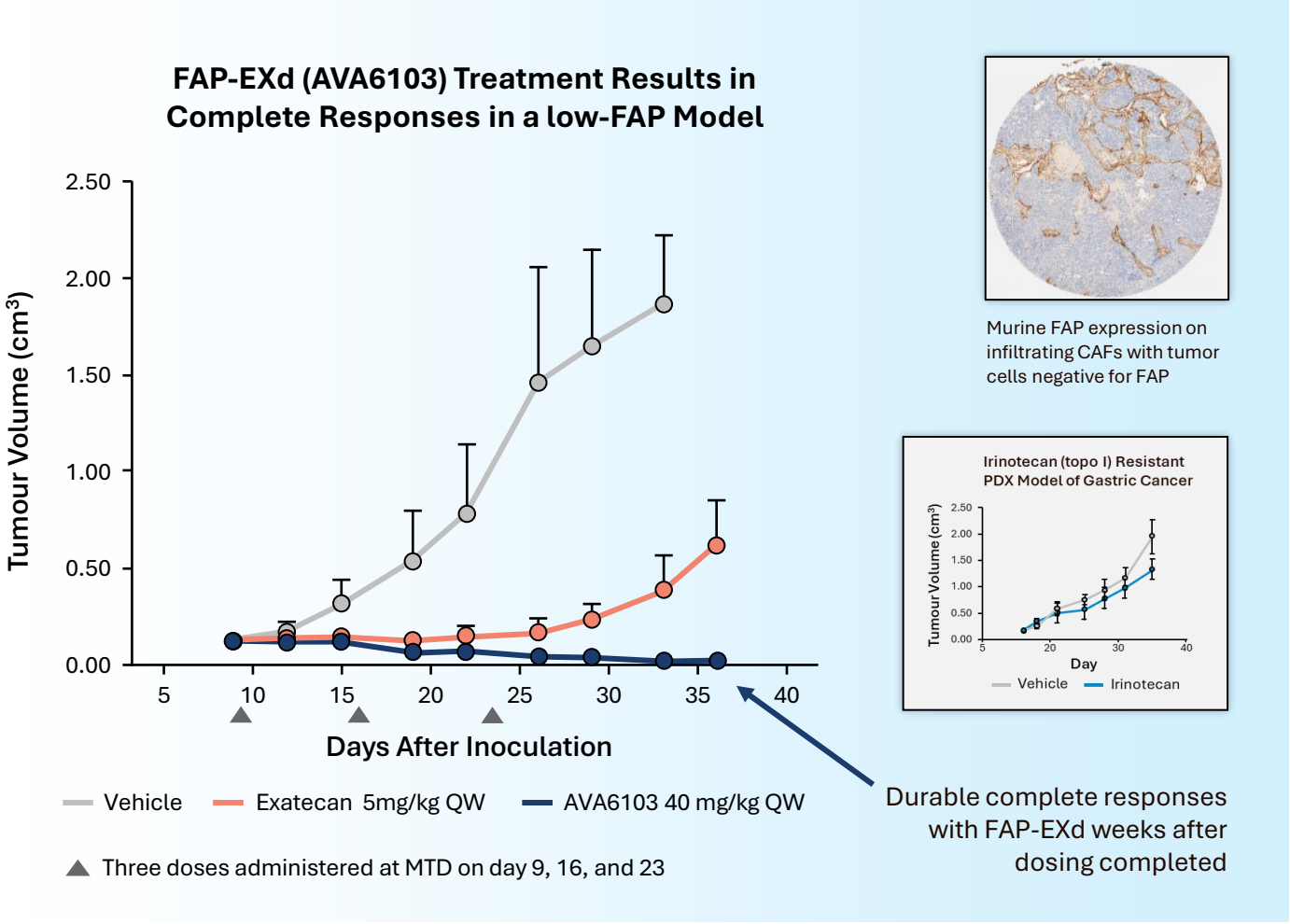
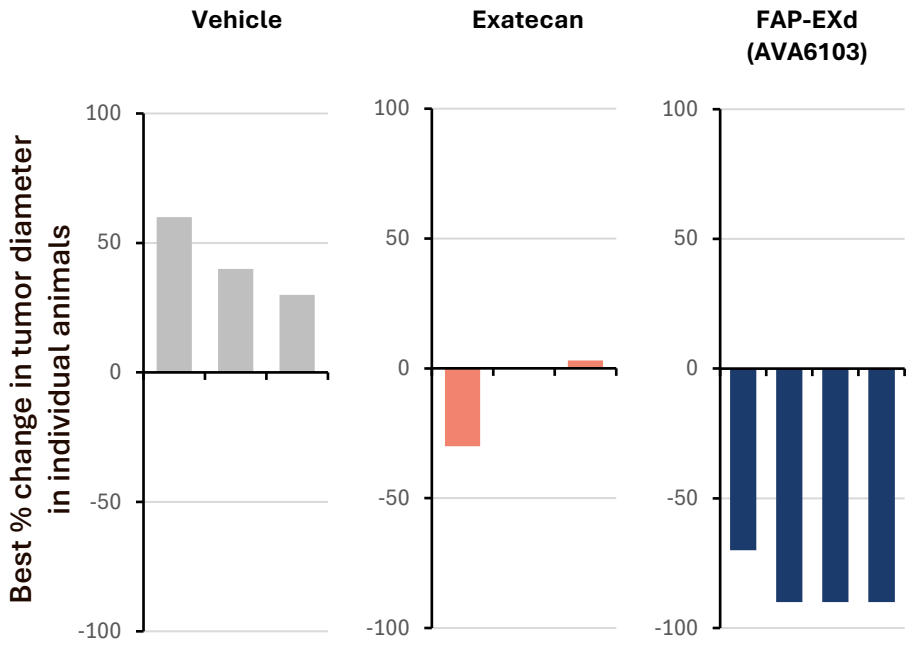
FAP-EXd Demonstrates Complete Responses in a Topo I-resistant PDX Model of Gastric with FAP Expression Only on Murine CAFs





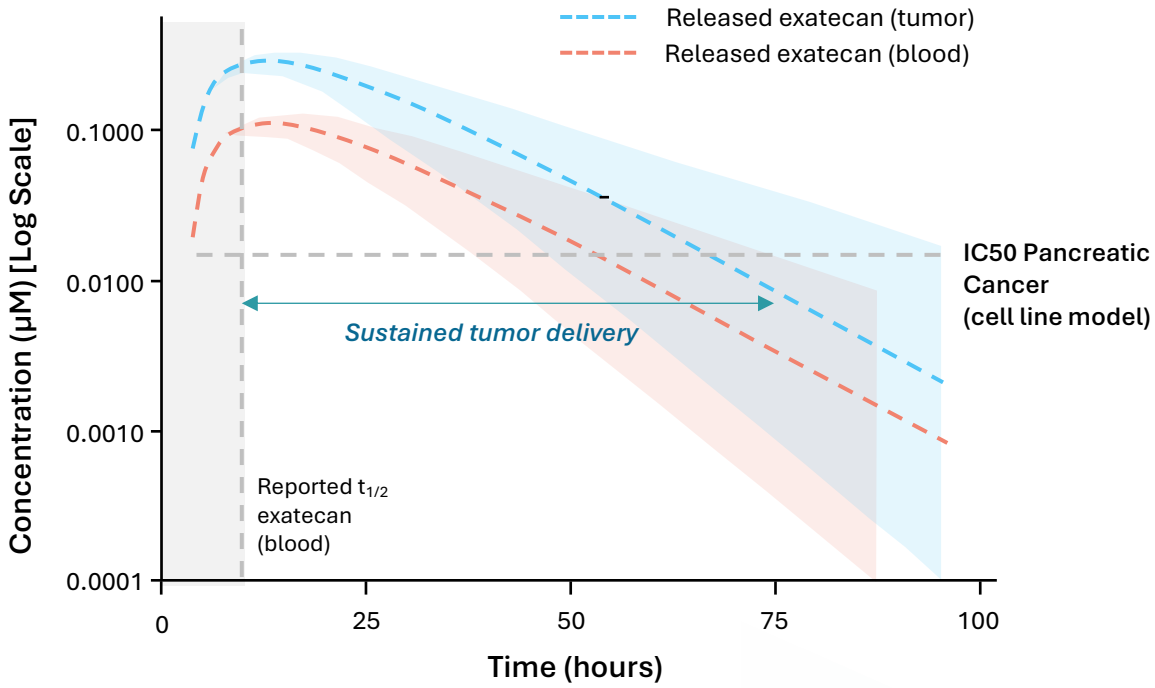
Sarc4183 is a soft tissue sarcoma PDX model with expression of human FAP directly on tumor cells (IHC inset). AVA6103 and exatecan dosing at MTD x 4 doses. Durable complete regressions observed in 7/8 animals with AVA6103

Tumor & Plasma PK with AVA6103 Demonstrate Sustained Tumor Release Resulting in 2-3 Days Above the IC50 in the Tumor

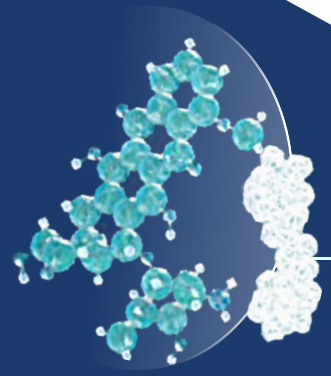
1. Tumor and plasma PK studies demonstrate a highly favorable ratio when comparing concentrations of released exatecan

2. Using these data, **longitudinal PK simulations of released exatecan** demonstrate tumor exposure predicted above the IC50 concentration for **over 60 hours** following a single low dose (15 mg/kg) of FAP-EXd

Modeling of Tumor & Plasma Concentration at low-dose AVA6103 suggests prolonged tumor exposure



FAP-EXd (AVA6103) Represents a Large Market Opportunity



pre|CISION®
peptide

FAP-EXd (AVA6103)

The topoisomerase I inhibitor chemo mechanism is **used widely in oncology**

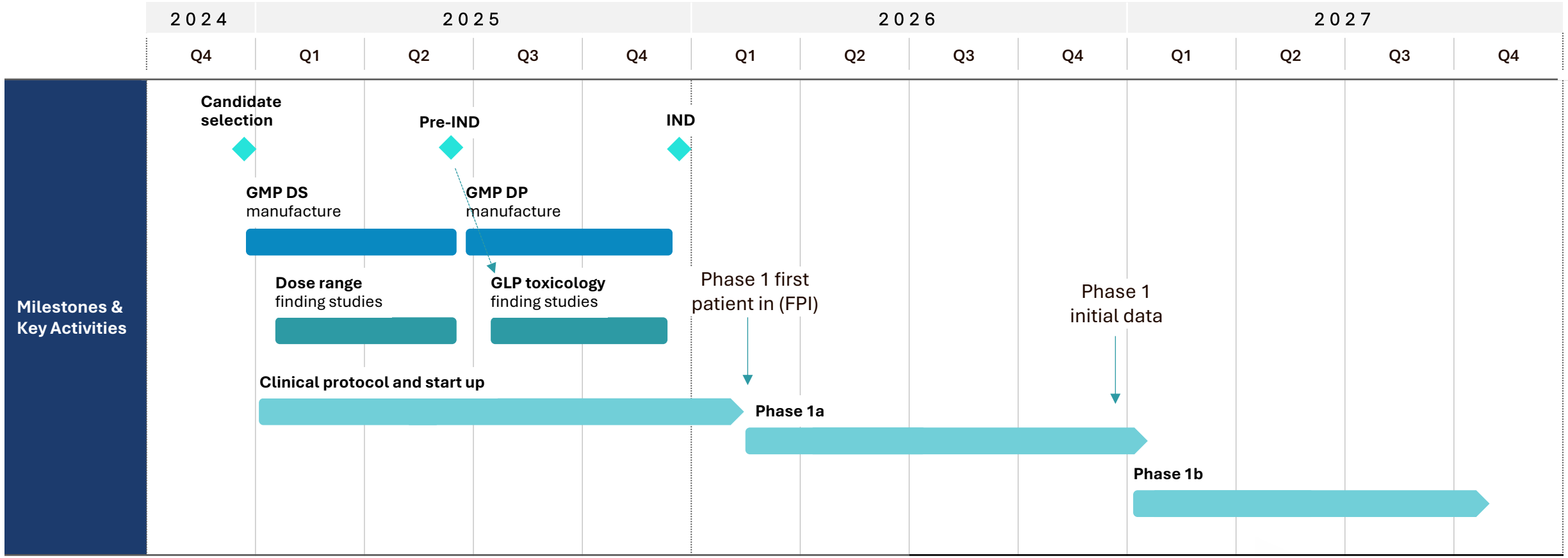
There are approved topo I inhibitors with much lower potency (topotecan and irinotecan)

Leveraging the Tempus collaboration to **identify optimal indications** (SLFN11 and FAP modeling)

Multiple large market opportunities for FAP-Exd include pancreatic cancer, gastric cancer, small cell and non-small cell lung cancer, breast cancer, among others

Avacta THERAPEUTICS

FAP-EXd (AVA6103) Planned for Phase 1 to Initiate in Q1 2026



pre|CISION: 3 IP Families Align with the Product Pipeline

pre|CISION® Platform Foundational IP

The background platform IP of the **pre|CISION® FAP-cleavable peptide drug conjugates** is owned by Bach Bio with an exclusive license to Avacta. Patent expiry in 2035

FAP-Dox (AVA6000) will be further protected with formulation, manufacturing, patient population and dosing IP. US Orphan drug designation with regulatory exclusivity

pre|CISION® Sustained Release Program IP

The **sustained release pre|CISION® mechanism** delivers payloads with precisely tunable kinetics

FAP-EXd (AVA6103): First program developed by Avacta with a novel, sustained released mechanism of action based on the FAP-cleavable peptide. The Program IP is owned by Avacta, based on the foundational patents with Program patent expiry in 2045

pre|CISION® Biologic Drug Conjugate IP

The background platform IP of the pre|CISION® FAP-cleavable biologic drug conjugates is co-owned by Avacta and Tufts with an exclusive sublicense to Avacta via Bach Bio. Patent expiry in 2041

Avacta holds an enviable IP position with multiple families and new foundational IP around the sustained release mechanism of pre|CISION delivery

Avacta THERAPEUTICS

Thank You

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