

Silencing the Fc, completely

Stealth-Body is a human IgG1 scaffold with no measurable binding to any FcγR tested and none to C1q — with half-life, thermostability and expression titer preserved. It has been validated in our own lead asset and in third-party antibodies.

CrossPoint Therapeutics | 2026 Q4



Company at a glance

Stealth-Body-enabled drug developer advancing proprietary therapeutics and platform partnerships.



What we have built

A differentiated Fc-silencing scaffold	A human IgG1 Fc with no measurable binding to FcγRs or C1q, with no measurable ADCC or CDC in the assays tested while retaining FcRn binding. Serum half-life was longer than LALA-PG under matched conditions.
A cross-format Fc platform	The same Fc has been applied to multiple unrelated antibodies without material loss of expression titer or thermostability, including a cytotoxic ADC, a second internal program, and third-party antibodies.
Lead asset validation in CPA-001	CPA-001 (EGFR, MMAE, DAR 4) achieved an MED of 0.6 mg/kg in A431 CDX and complete regression at 1 mg/kg across three CDX models. Published MMAE-based EGFR ADCs have reported MEDs generally in the 3–5 mg/kg range².

1. Including non-dilutive government grants. 2. Cross-study comparison only; models, dosing schedules and study conditions differ.

Leadership Team

Experienced leadership spanning business strategy, drug development, and antibody engineering



Tae-Erk Kim, Ph.D.
CEO

*Business Development &
Strategy*

- Ph.D. in Technology & Innovation Management, University of Leeds
- Former Head of Business Development, Korea Drug Development Fund (KDDF)
- Former CEO, Lead Compass Investment
- Evaluated and managed portfolios across 150+ drug development programs



Kihwan Chang, Ph.D.
CTO

Antibody Discovery to IND

- Ph.D. in Chemistry, Yonsei University
- R&D leadership experience at Curogen, Orum Therapeutics, CJ Healthcare
- Expertise in antibody discovery, optimization, and ADC development
- Extensive patent and development experience across oncology, infectious disease, and immuno-oncology



Prof. Sang Taek Jung
CTA

Antibody & Fc Engineering

- Professor, Seoul National University
- Expertise in antibody libraries, affinity maturation, half-life extension, and Fc engineering
- 115 patents and 22 government-funded R&D programs
- 10 technology-transfer deals to 8 companies, totaling KRW 26 billion
- Vice President, Korean Antibody Society

R&D and Business Development



Pedro Lee

Head of Biology

- Biology lead for Stealth-Body-based drug programmes
- Target validation and cell-based assay strategy
- In vitro pharmacology and in vivo efficacy support
- Biological evaluation across new modalities



Seongguk Cho, Ph.D.

Head of Preclinical Development

- Preclinical development for lead and follow-on assets
- Nonclinical efficacy, safety and TK study planning
- External study coordination and data review
- IND-enabling development strategy



Jia Lee

Head of Business Development

- Out-licensing BD, Daewoong Pharmaceutical
- Business development, KDDF
- Biopharma consulting, IQVIA and Syneos
- Global partnering, licensing and strategy at CrossPoint

FcγR-Related Toxicity Across Antibody Modalities

Fc effector function and FcγR-mediated interactions can contribute to safety liabilities across multiple antibody modalities.

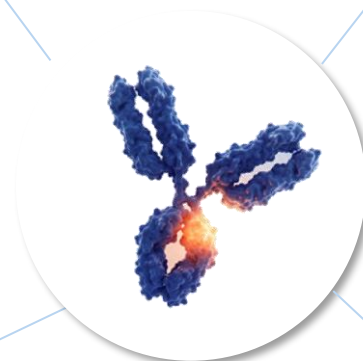
Antagonistic antibodies

- Fc effector function may drive normal-cell depletion and hematologic toxicity
- Magrolimab highlights normal-cell CD47 binding and Fc-related safety considerations

T-cell engagers

- FcγR-mediated clustering can trigger target-independent T-cell activation and CRS
- FcγR-rich tissues may further increase off-target activation and toxicity

(Sci Rep. 2017 May 30;7(1):2476.)



FcγR-mediated uptake ·
clustering · effector function

Agonistic antibodies

- Many TNFRSF agonists rely on FcγR-mediated clustering for activity
- Excessive clustering may increase cytokine release and liver toxicity, limiting the achievable therapeutic dose

(Human Vaccines & Immunotherapeutics 2020,16,2, 377–387)

Antibody-drug conjugate

- FcγR-mediated uptake can deliver intact ADCs into non-target immune cells
- This may contribute to payload-associated off-target toxicity and dose limitation

(Pharmacology & Therapeutics 200 (2019) 110–125)

Two Key Pathways Contributing to ADC Toxicity

Linker stability has improved; FcγR-mediated uptake remains a potential liability

① Premature Payload Release

–Improved through linker optimization

- Linker stability has been a major focus of ADC development
- However, clinically relevant toxicities persist even with highly stable linkers

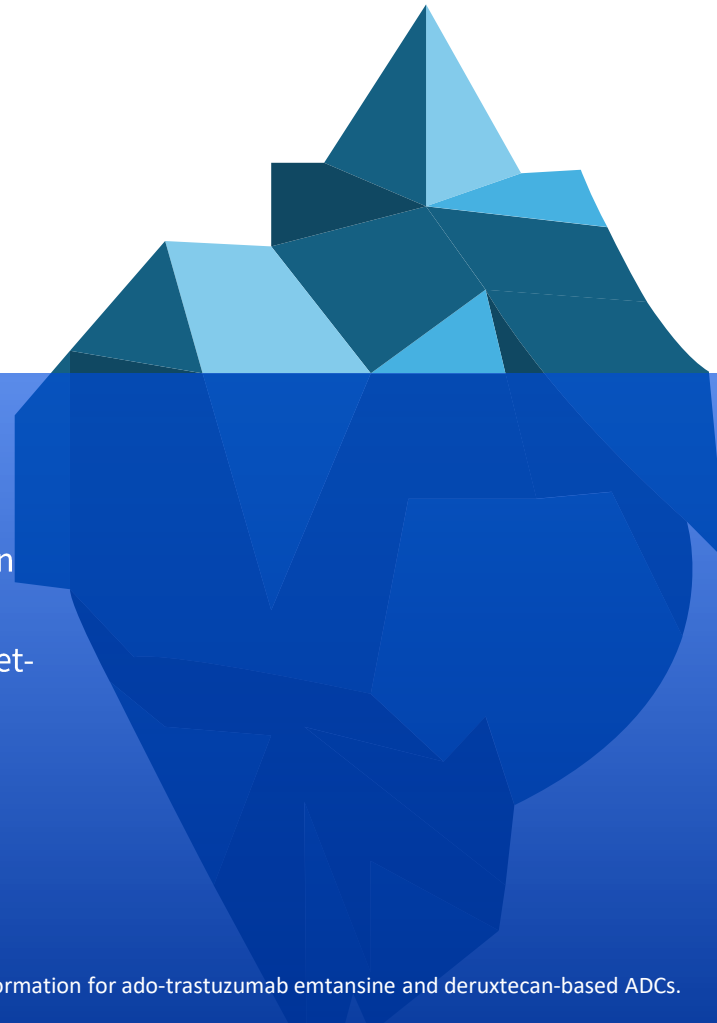
Examples

T-DM1: thrombocytopenia observed despite a non-cleavable linker

DXd ADCs: ILD and other toxicities remain despite high linker stability

② FcγR-Mediated Off-Target Uptake

- FcγR-expressing normal and immune cells can internalize intact ADCs
- Intracellular payload release may lead to target-independent toxicity
- This pathway is not addressed by linker stabilization alone



How the Field Has Tried to Address Fc Liability

Three common approaches — and why we chose selective Fc silencing.

Option 1 Isotype switch

- Reduces effector function, but does not eliminate it
- IgG4 still binds FcγRI and FcγRIIb
- FcγR-mediated uptake may be reduced, but not fully closed
- Requires additional stabilization, typically S228P
- Hinge introduces concerns around Fab-arm exchange and stability

Partial reduction, not complete silencing

Option 2 Remove the Fc

- Use a Fab, scFv, or similar fragment
- Closes the FcγR route completely
- But also removes FcRn-mediated recycling
- Fc removal can substantially shorten systemic exposure
- For conjugates, this can reduce tumor exposure
- Moves outside the standard IgG1 purification and manufacturing platform

Avoids FcγR uptake, but may introduce PK and CMC trade-offs

Option 3 Mutations in IgG1

- Retains the full-length IgG1 scaffold and FcRn-mediated recycling
- Representative approaches include LALA, LALA-PG, N297A/aglycosylation, IgG1σ/FES, and STR
- Degree of FcγR/C1q silencing and biophysical trade-offs vary by mutation set
- Stealth-Body is designed for complete FcγR/C1q silencing while retaining FcRn binding and WT-like developability

Selective Fc silencing with full-length IgG1 properties retained

Design Principle Preserve what you need from IgG1; remove only the Fc functions you do not want.

Stealth-Body Design

FcγR/C1q silenced. FcRn and developability retained.

Component	Design	Experimental Evidence
Fab	Unchanged	- Target selectivity and potency retained - Comparable EC50 to the wild-type counterpart in matched in vitro killing assays
FcγR I / IIa / IIb / IIIa	Removed	- No measurable binding across six human FcγRs tested by BLI - No measurable ADCC and reduced FcγR-mediated uptake in tested systems
C1q	Removed	No detectable complement-dependent cytotoxicity
FcRn	Retained	FcRn binding and recycling function preserved
Developability	Retained	WT-like stability and expression; Q295/N297 and Fc glycosylation preserved
Portability	Transferrable	Applied across ADCs and selected bispecific / third-party antibodies; targeted delivery evaluation ongoing

Head-to-head against LALA and LALA-PG

No measurable FcγR or C1q binding, with FcRn function retained

Trastuzumab backbone. Fc receptor binding assessed by BLI; effector function by cell-based assays; half-life evaluated in human FcRn transgenic mice.

Variant	FcγRI	FcγRIIa	FcγRIIb	FcγRIIIa	C1q	ADCC	CDC	FcRn	Half-life
WT IgG1	++++	+++	+++	++	+++	+++	+++	+++	~9 days
LALA	++	+	+	+	-	++	+	+++	~10 days
LALA-PG	+	-	-	-	+/-	+	+	+++	~12 days
Stealth-Body	-	-	-	-	-	-	-	+++	~14 days

Fc silencing with key antibody properties retained

Thermostability

68.5°C

Comparable to WT IgG1 (69.54°C)

FcRn BINDING

Retained (+++)

pH-dependent FcRn binding and recycling preserved

Serum Half-life

Longer than LALA-PG

Observed in human albumin/FcRn double-transgenic mice

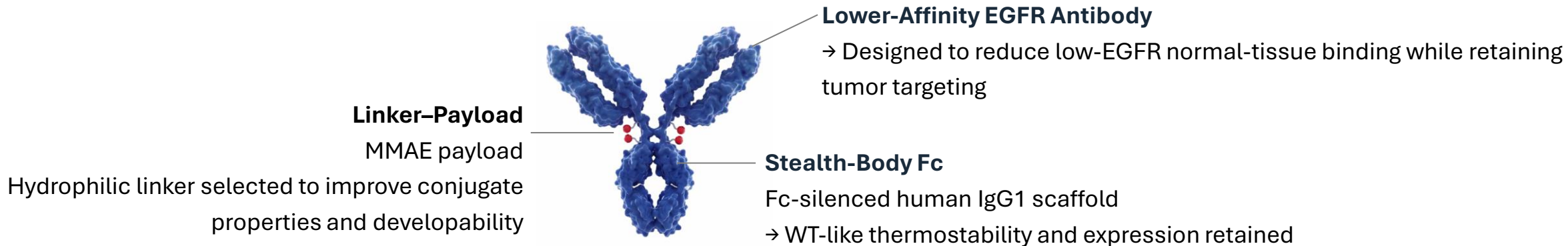
Off-Target Cytotoxicity — Wild-Type vs LALA vs Stealth-Body

Eight antigen-negative cell lines, including FcγR-negative controls, under matched VC-MMAE conditions

Cells	Trastuzumab Fc-WT-VC-MMAE IC50 (nM)	Trastuzumab Fc-LALA-VC-MMAE IC50 (nM)	Trastuzumab Fc-SB-VC-MMAE IC50 (nM)	Cetuximab Fc-WT-VC-MMAE IC50 (nM)	Cetuximab Fc-SB-VC-MMAE IC50 (nM)
THP-1	0.355	4.003	23.130	6.556	No effect
OCI-AML-2	1.854	20.48	No effect	85.09	No effect
MV-4-11	5.293	12.75	No effect	147.2	No effect
U937	6.362	5.368	No effect	53.81	1392
EOL-1	20.630	10.06	No effect	566.600	No effect
HL60	—	—	—	517.000	No effect
Jurkat (No FcγR)	No effect	11.15	No effect	No effect	No effect
HEK293 (No FcγR)	No effect	42.35	No effect	No effect	No effect

- Wild-type ADCs show potent killing in FcγR-positive cells lacking HER2 or EGFR expression.
- LALA reduces off-target killing, but residual activity remains across multiple cell lines.
- Stealth-Body eliminates measurable killing in most cell lines.

CPA-001 — Preclinical Validation of an EGFR ADC



MED of 0.6 mg/kg in A431 CDX

- CPA-001 achieved an MED of 0.6 mg/kg following three doses (D0, D4, D8) in the A431 CDX model
- MEDs in FaDu and MDA-MB-468 were 0.5 and 0.125 mg/kg, respectively
- Reported MEDs from other EGFR-MMAE ADCs are around ~3 mg/kg

Efficacy Impact of Fc Engineering

- Antibody, target, linker, payload, DAR, conjugation method, animal model, and dosing schedule were held constant
- Wild-type Fc and Stealth-Body Fc were compared directly
- Stealth-Body Fc showed a lower MED and improved antitumor activity under matched conditions

Internal in vivo studies, 2025–2026. Published comparator MEDs based on AACR 2018, Oncotarget 2018, Bioorg. Med. Chem. 2024, and publicly available patent literature.

Preclinical Benchmarking of Selected EGFR ADCs

CPA-001 A431 MED: 0.6 mg/kg (three-dose regimen); cross-study efficacy comparisons are contextual.

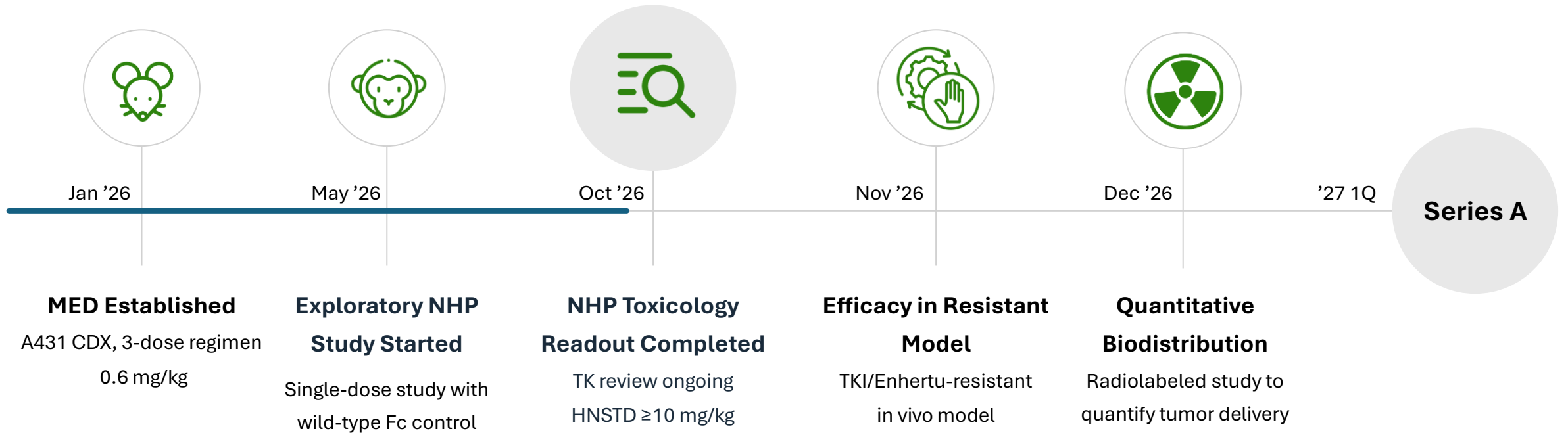
Attribute	ABBV-221	RN765C*	AGCM-22	MRG003	CPA-001
EGFR format	High affinity	Low affinity	High affinity	Highest affinity	Low affinity
Payload / DAR	MMAE / 3	Proprietary / 2	MMAE / 4	MMAE / 4	MMAE / 4
Mouse MED	3 mg/kg	1.5–3 mg/kg	4.5 mg/kg	3 mg/kg	0.6 mg/kg
Schedule	6 doses	single dose	5 doses	3 doses	3 doses (d0/4/8)
HNSTD	4.5 mg/kg	Not shown	Not shown	6 mg/kg	≥10 mg/kg
Estimated TI (cross-species adjusted)	Reported: 6	Not shown	Not shown	Reported: 4.8-8	≥66†

†CPA-001 HED ratio ≥66 uses A431 MED 0.6 mg/kg and NHP HNSTD ≥10 mg/kg. Alternate inputs: 0.5 mg/kg (3 doses) → ≥80; 1.0 mg/kg (single dose) → ≥40. FDA 2005 recommends mg/kg scaling for IV proteins >100 kDa; BSA-HED here is exploratory only. External reported TI figures may not be harmonized; models and regimens differ.

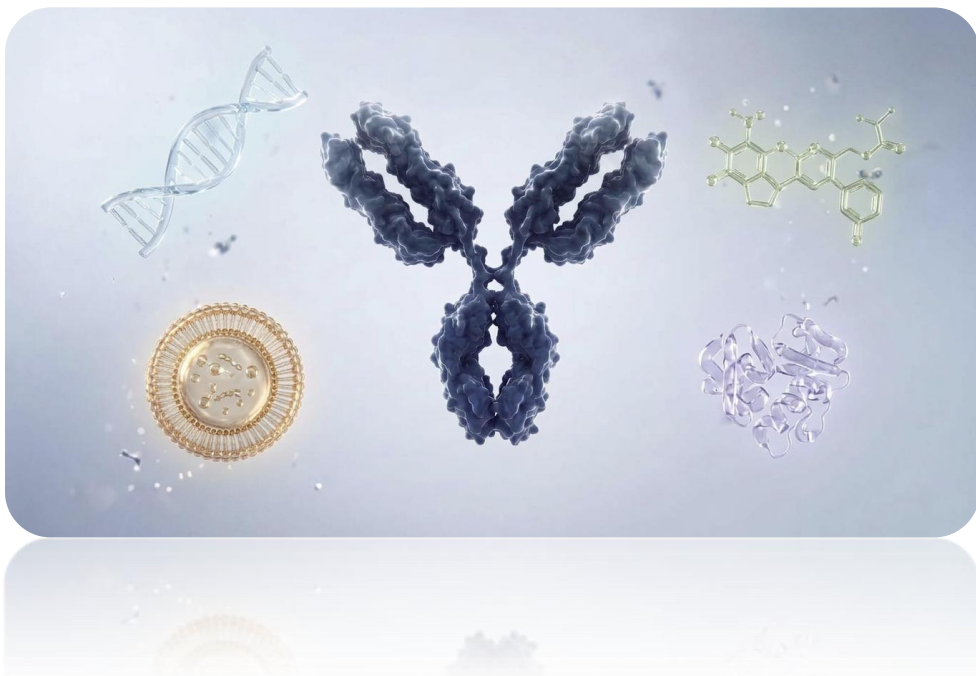
* No reported progress since 2018

Exploratory NHP Toxicology Completed

Cynomolgus study with matched WT Fc comparator completed; HNSTD ≥ 10 mg/kg. IND-enabling work is next.



Expanding Stealth-Body Across Modalities and Partnerships



Gene Delivery

Lipid polymer nanoparticle × Stealth-Body

- A one-year joint research program validated conjugation feasibility, active targeting, and antibody orientation control
- A follow-on program on a defined target is being discussed, with initiation targeted for October 2026
- Expands Stealth-Body into targeted gene delivery

Bispecific ADC

Stealth-Body-Based Bispecific ADC

- Selected for a government R&D program of approximately KRW 5.0 billion
- Enables development of a second proprietary pipeline alongside CPA-001
- Extends Stealth-Body into next-generation ADC formats

New Application Areas

Expanding the Addressable Opportunity

- CNS antibodies: exploring Fc silencing as an approach to reduce Fc effector-related safety liabilities
- Subcutaneous ADC delivery: evaluating whether reduced Fc-mediated uptake may improve tolerability during lymphatic transit
- Additional antibody and conjugate formats are under partner discussion and internal evaluation

Stealth-Body —External Validation and IP Position

Platform Licensing Track Record

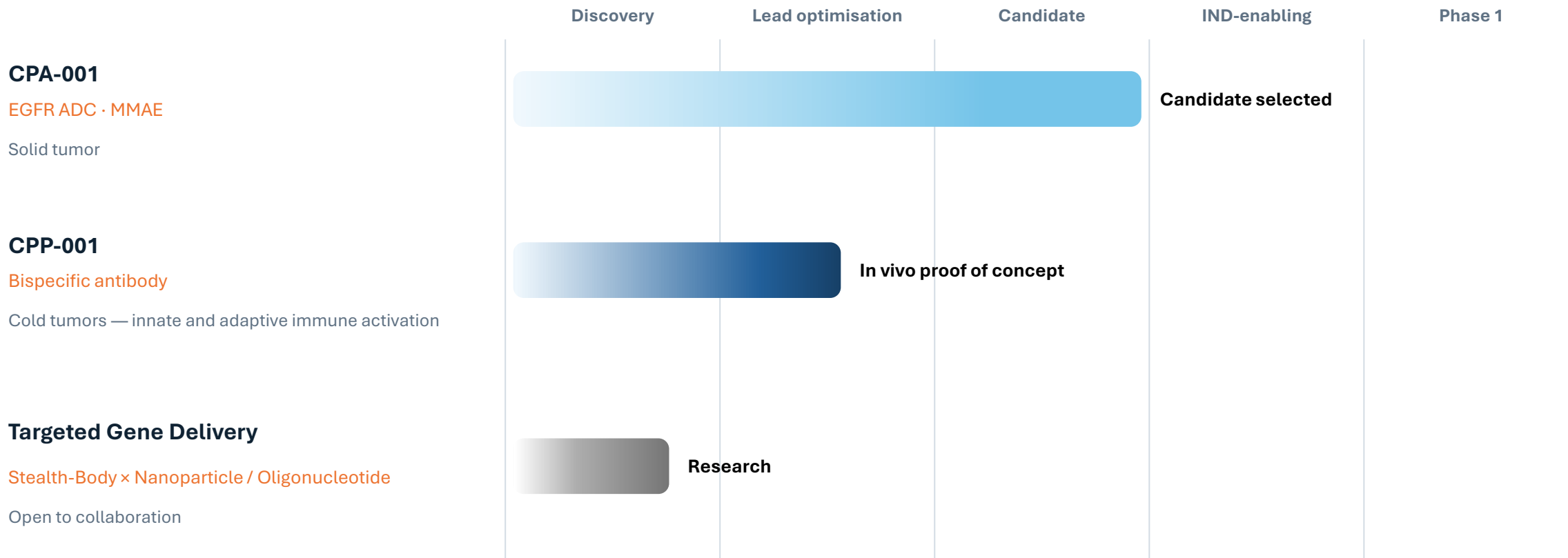
- In June 2025, Stealth-Body was licensed to Y-Biologics, a KOSDAQ-listed antibody company, for a defined set of targets under exclusive and non-exclusive rights.
- The transaction followed an independent material-transfer evaluation using the partner’s own antibodies, providing external validation of the platform’s portability and applicability.

Family	Covers	Status
Human antibody Fc domain variants and uses	Composition of matter — the platform	KR filed · PCT · US / EP / CN / JP national phase
Fc variant–based antibody–drug conjugates	ADC application	KR filed · PCT
Immuno-oncology bispecific antibody and uses	Bispecific / IO application	KR filed · PCT
Derivative filings	Formulation and further applications	KR priority · PCT

Filing status as of August 2026; national-phase entries shown only where applicable. Claims and FTO scope available under CDA.

Independent partner evaluation supports platform portability; pending IP filings cover key downstream applications.

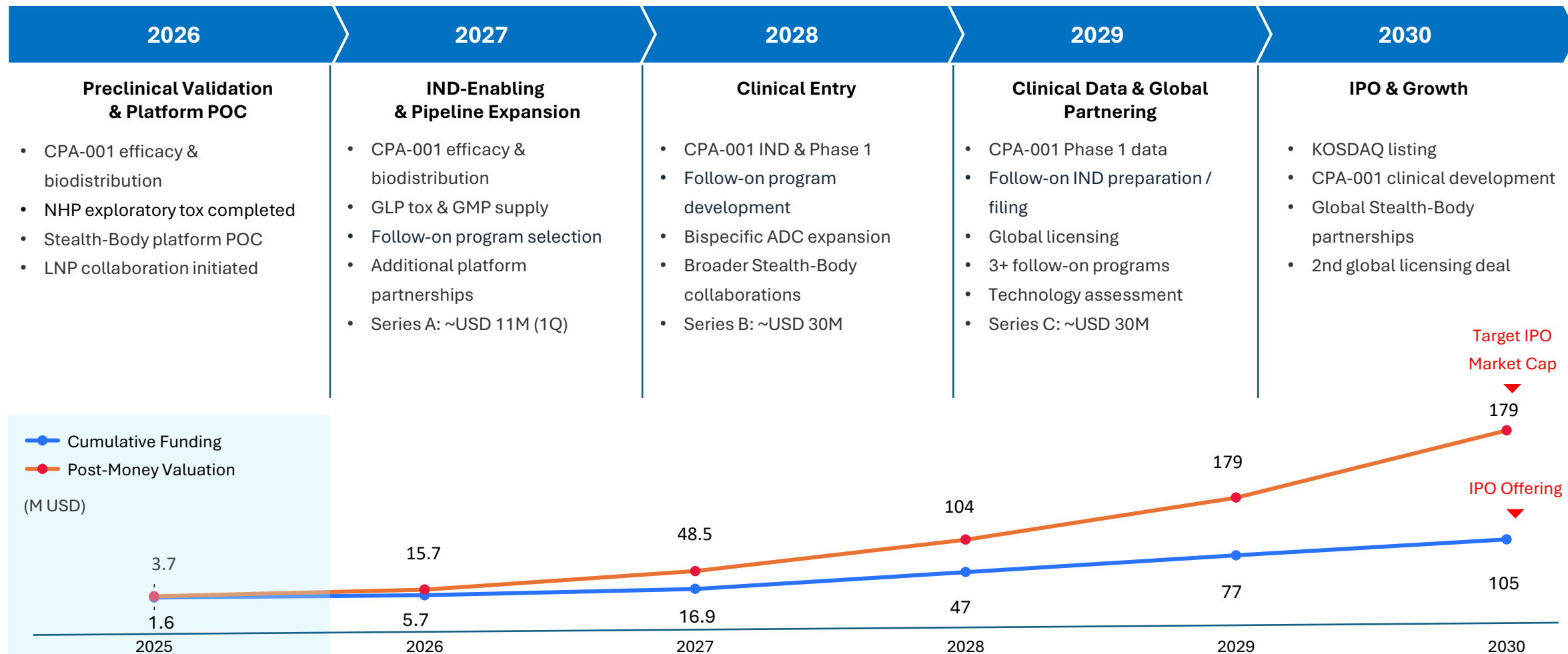
Three programs, one Fc architecture



A single Fc architecture supporting multiple therapeutic formats

Development Roadmap & Key Milestones

Targeting a KOSDAQ listing in 2030 with a minimum market capitalization of ~\$300M, following technology assessment in 2029



Partnering routes

Two value drivers, three partnering routes

Our thesis: FcγR-mediated off-target uptake remains an addressable source of toxicity even as linker stability improves.

PLATFORM

Stealth Body

- No measurable binding to six FcγRs or C1q
- FcRn binding, half-life and developability retained
- Longer half-life than LALA-PG under matched conditions
- Already out-licensed to a third-party antibody company

ASSET

CPA-001 | EGFR ADC

- MED 0.6 mg/kg in A431 CDX
- Complete regression at 1 mg/kg across three CDX models
- Hydrophilic linker · MMAE · DAR 4
- NHP pre-tox/TK package completed; IND targeted for 2028

PARTNERING

Three routes to collaborate

- **Platform license**
 - Target-specific rights to apply Stealth-Body to a partner's antibodies
- **Asset license**
 - Global or regional rights to CPA-001, with or without co-development
- **Research collaboration**
 - Application of Stealth-Body to partner-selected modalities, including bispecifics and antibody conjugates

Next Milestone IND-enabling studies / GMP preparation

Partnering After NHP Toxicology

Following exploratory NHP toxicology, CrossPoint is exploring platform, asset licensing and co-development opportunities.

- | | | |
|-----------|----------------------------|---|
| 01 | Platform evaluation | Evaluate a partner-selected antibody under an MTA feasibility study. |
| 02 | Co-development | Jointly develop selected programs around defined targets. |
| 03 | Licensing | Explore target-specific, regional, or global rights to the platform or selected assets. |

Stealth-Body

Silence unwanted Fc interactions.
Preserve circulation.
Enable precise delivery.

Business Development

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