NON-CONFIDENTIAL PARTNERING DECK

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 Q3

Company at a glance

2022 Founded

1 Platform licensee to date

4 Filed PCT patents US · EP · CN · JP

~$7M Total capital raised to reach NHP toxicology

What we have built

A differentiated Fc-silencing scaffold	• A human IgG1 Fc with no measurable binding to FcγRs or C1q, eliminating detectable ADCC/ADCP/CDC activity 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 range[2].

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

Executive Summary

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

Upcoming Catalyst **NHP pre-tox / TK package — therapeutic index available under CDA**

Leadership Team

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

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

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

PROBLEM

FcγR-Related Toxicity Across Antibody Modalities

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

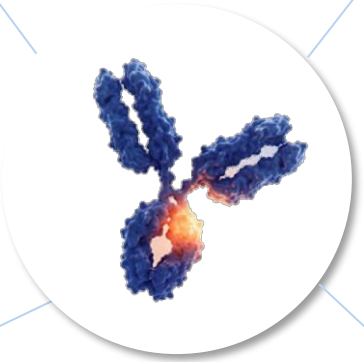
Antagonistic antibodies

- Fc effector function may drive normal-cell depletion and hematologic toxicity
- Magrolimab (IgG4) highlights the clinical challenges associated with Fc-mediated safety liabilities

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

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



FcγR-mediated uptake · clustering · effector function

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.)

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)

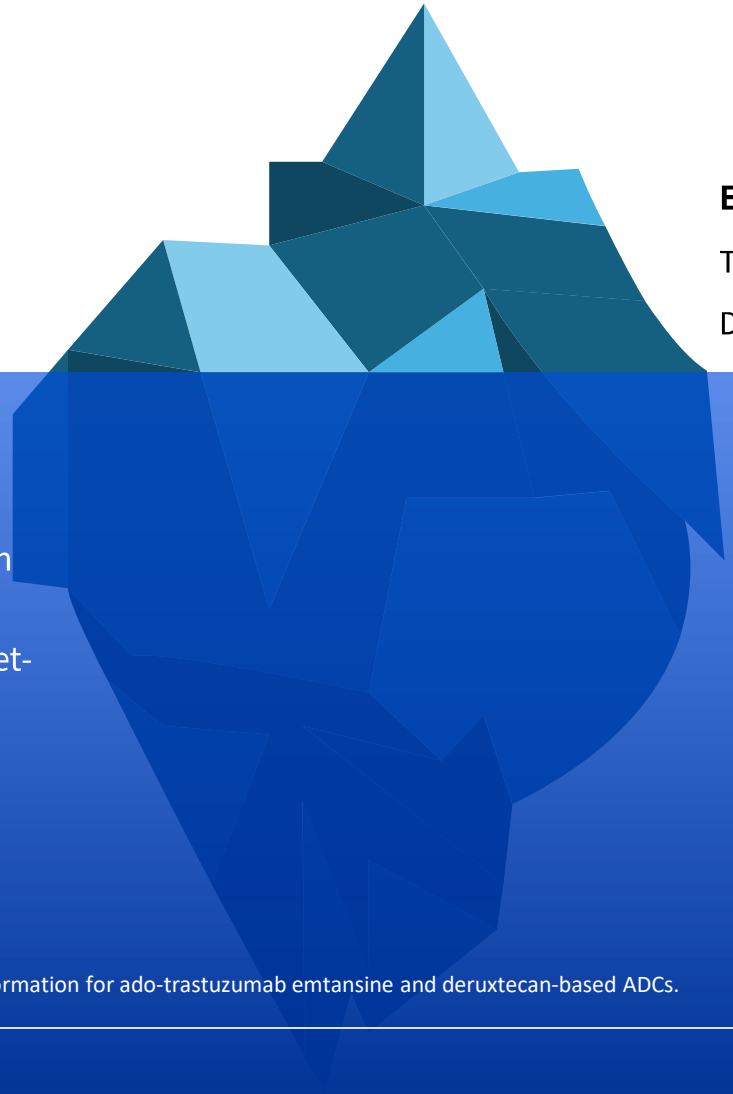
Two Major Routes of ADC Toxicity

Linker stability has improved, but FcγR-mediated off-target uptake remains

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



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

Examples

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

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

Pharmacology & Therapeutics 200 (2019) 110–125; approved prescribing information for ado-trastuzumab emtansine and deruxtecan-based ADCs.

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 • ADCC, ADCP and FcγR-mediated uptake abrogated
C1q	Removed	• No detectable complement-dependent cytotoxicity
FcRn	Retained	• FcRn binding and recycling function preserved
Developability	Retained	• Thermostability and expression titer maintained within the wild-type IgG1 range
Portability	Transferrable	• Same Fc applied across cytotoxic ADCs, agonistic bispecifics and third-party antibodies; evaluation in targeted LNP formats ongoing

Head-to-head against LALA and LALA-PG

Same assay panel, matched experimental conditions

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	++++	+++	+++	++	+++	+++	+++	+++	Reference
LALA	++	+	+	+	-	++	+	+++	Longer
LALA-PG	+	-	-	-	+/-	+	+	+++	Longer
Stealth-Body	-	-	-	-	-	-	-	+++	Longest

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 FcRn transgenic mice under matched study conditions

PROBLEM

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

Eight HER2-/EGFR-negative immune cell lines under matched VC-MMAE conditions

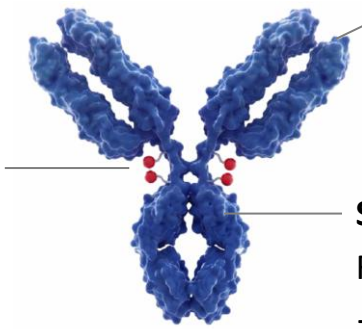
Cells	Trastuzumab Fc-WT-VC-MMAE IC50 (nM)	Trastuzumab Fc-LALA-VC-MMAE IC50 (nM)	Trastuzumab Fc-SB-VC-MMAE IC50 (nM)	Cetuximab Fc-WT-VC-MMAE IC50 (nM)	Cetuximab Fc-SB-VC-MMAE IC50 (nM)
THP-1	0.355	4.003	23.130	6.556	No effect
OCI-AML-2	1.854	20.48	No effect	85.09	No effect
MV-4-11	5.293	12.75	No effect	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 7 of 8 tested cell lines.
- FcγR-negative controls show no corresponding wild-type ADC activity, supporting an FcγR-mediated mechanism.

PROOF ASSET

CPA-001 — Preclinical Validation of an EGFR ADC

Lower-Affinity EGFR Antibody
→ Reduced on-target toxicity and improved tumor penetration



Linker–Payload
MMAE payload
Hydrophilic linker selected to improve conjugate properties and developability

Stealth-Body Fc
Fc-silenced human IgG1 scaffold
→ Improved stability and manufacturability

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
- Published preclinical MEDs for EGFR–MMAE ADCs are generally reported in the 3–5 mg/kg range

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 EGFR–MMAE ADCs

CPA-001 achieved an MED of 0.6 mg/kg, below the reported range of selected EGFR–MMAE ADC comparators.

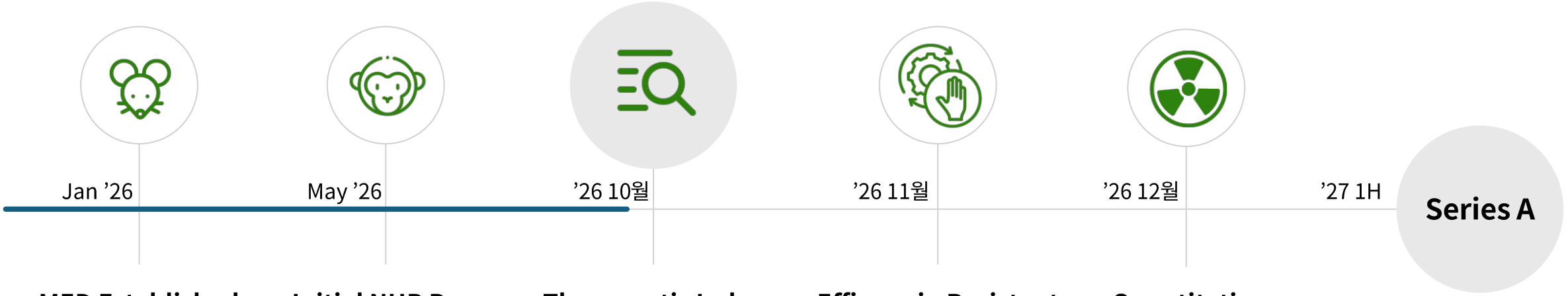
Attribute	ABBV-221	RN765C	AGCM-22	MRG003	CPA-001
EGFR format	High affinity	Low affinity	High affinity	Highest affinity	Low affinity
Payload / DAR	MMAE / 3	MMAE / 2	MMAE / 4	MMAE / 4	MMAE / 4
Mouse MED	3 mg/kg	1.5–3 mg/kg	4.5 mg/kg	3 mg/kg	0.6 mg/kg
Schedule	6 doses	single dose	5 doses	3 doses	3 doses (d0/4/8)
HNSTD	4.5 mg/kg	Not shown	Not shown	6 mg/kg	Under CDA
TI status	Reported: 6	Not shown	Not shown	Reported: 4.8–8	Under CDA

Based on publicly available preclinical data. Study models, dosing schedules, and toxicity assessment criteria differ; values are not from head-to-head studies.

PROOF ASSET

NHP Pre-Toxicology Readout Imminent

Therapeutic index confirmation will support the Series A valuation and serve as a key inflection point for global partnering



MED Established
A431 CDX, 3-dose regimen
0.6 mg/kg

Initial NHP Pre-Toxicology Readout
Single-dose study with wild-type Fc control

Therapeutic Index (TI) Established
NHP TK analysis
HNSTD determination

Efficacy in Resistant Model
Enhertu-resistant in vivo model

Quantitative Biodistribution
Radiolabeled study to quantify tumor delivery

Where Stealth-Body Applies

Application	Rationale for Fc silencing	Current status
Cytotoxic ADCs	Reduces FcγR-mediated off-target uptake and associated toxicity	Validated in vivo across multiple models
Agonistic receptor antibodies (CD40, OX40, CD28)	Limits FcγR-driven clustering and excessive immune activation	Early in vivo validation in bispecific format
Blocking antibodies on shared targets (CD47 and similar)	Reduces unwanted Fc effector activity on normal cells	
Immune modulators, autoimmune (CD40L, CD3-directed)	Minimizes FcγR-mediated interactions with platelets and immune cells	Partner validation ongoing
Fc-dependent antibodies	Not applicable when target-cell depletion depends on Fc effector function	Out of scope

Validation basis **Application areas are supported by internal in vivo studies and/or partner-led evaluations.**

Expanding Stealth-Body Across Modalities and Partnerships

Gene Delivery

Lipid nanoparticle × Stealth-Body

- A one-year joint research program validated conjugation feasibility, active targeting, and antibody orientation control
- Related platform IP is jointly owned, with development rights under discussion
- 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 granted · 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. Application numbers, claim scope and FTO analysis available under CDA.

Third-party licensing validates portability; the IP portfolio protects the platform and key downstream applications.

Three programs, one Fc architecture

CPA-001

EGFR ADC · MMAE

Solid tumor

CPP-001

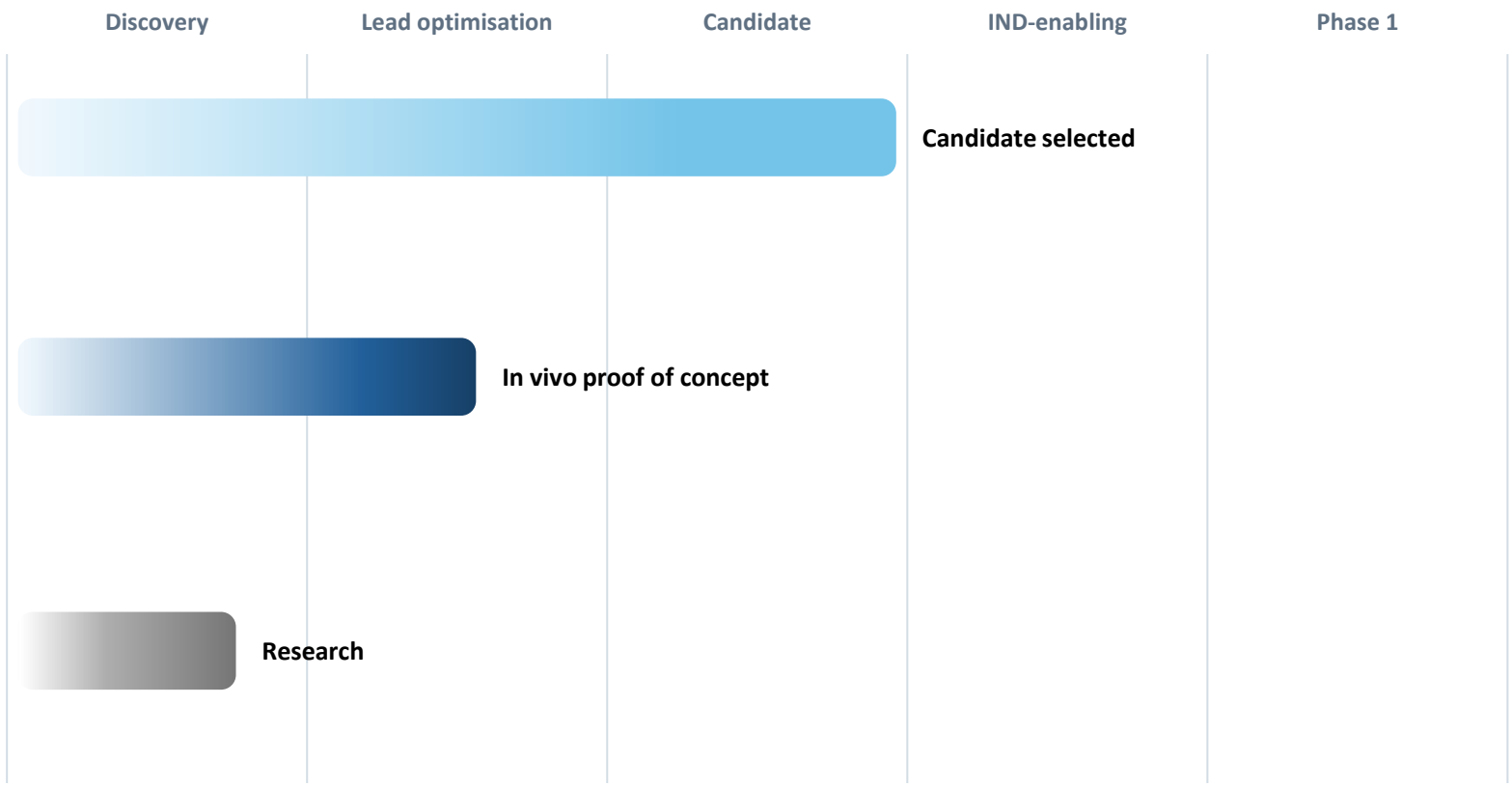
Bispecific antibody

Cold tumors — innate and adaptive immune activation

Targeted Gene Delivery

Stealth-Body × Nanoparticle / Oligonucleotide

Open to collaboration



A single Fc architecture supporting multiple therapeutic formats

Development Roadmap & Key Milestones

The Oct ’26 NHP package marks the next major value inflection point, followed by IND-enabling development and clinical entry.

2026 H2	2027	2028	2029
• NHP pre-tox / TK package completed, TI established • LNP-based gene delivery collaboration initiated • Expanded tumor models and quantitative biodistribution	• GLP toxicology • Mouse/Monkey PK • GMP clinical supply • Series A financing to support IND-enabling development	• CPA-001 IND filing • Phase 1 initiation • Next-generation ADC candidate selected • Gene delivery collaboration candidate advanced	• Technology assessment for IPO • KOSDAQ listing preparation • Target IPO market capitalization: ~$300M

GLOBAL PARTNERING

Global partnering begins as the therapeutic index is established in H2 2026, with platform licensing and CPA-001 asset transactions pursued independently.

FUNDING PLAN

2027 GLP toxicology and GMP manufacturing represent the primary development spend. A KRW 15bn Series A is planned to fund the company through IND filing.

CrossPoint THERAPEUTICS

Partnering Ahead of the Next Data Inflection

CrossPoint is engaging global partners at both the platform and asset level as CPA-001 advances through NHP preclinical development.

01	**Platform evaluation**	Apply Stealth-Body to a partner-selected antibody or modality.
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.

Platform and asset transactions can be structured independently.

Business Development

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