Captor Therapeutics®

A new dawn in drug discovery

Corporate Presentation – Q1 2023 update

May 2023

All images by Piotr Piatek

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Q1 2023 Executive summary

Announcement of the **next steps in the Strategy** - increase of authorised share capital

Target announcements in CT-01, CT-02 and CT-05

CT-01 - additional compelling *in-vivo* efficacy data in preclinical studies - moving towards the clinic as planned

Financial results - almost PLN 16 M spent on R&D, but supported by non-dilutive grant funding which helps preserve capital in these difficult market conditions

Ono - the collaboration has started well with excellent cooperation between the companies and an improvement in cash flow for Captor

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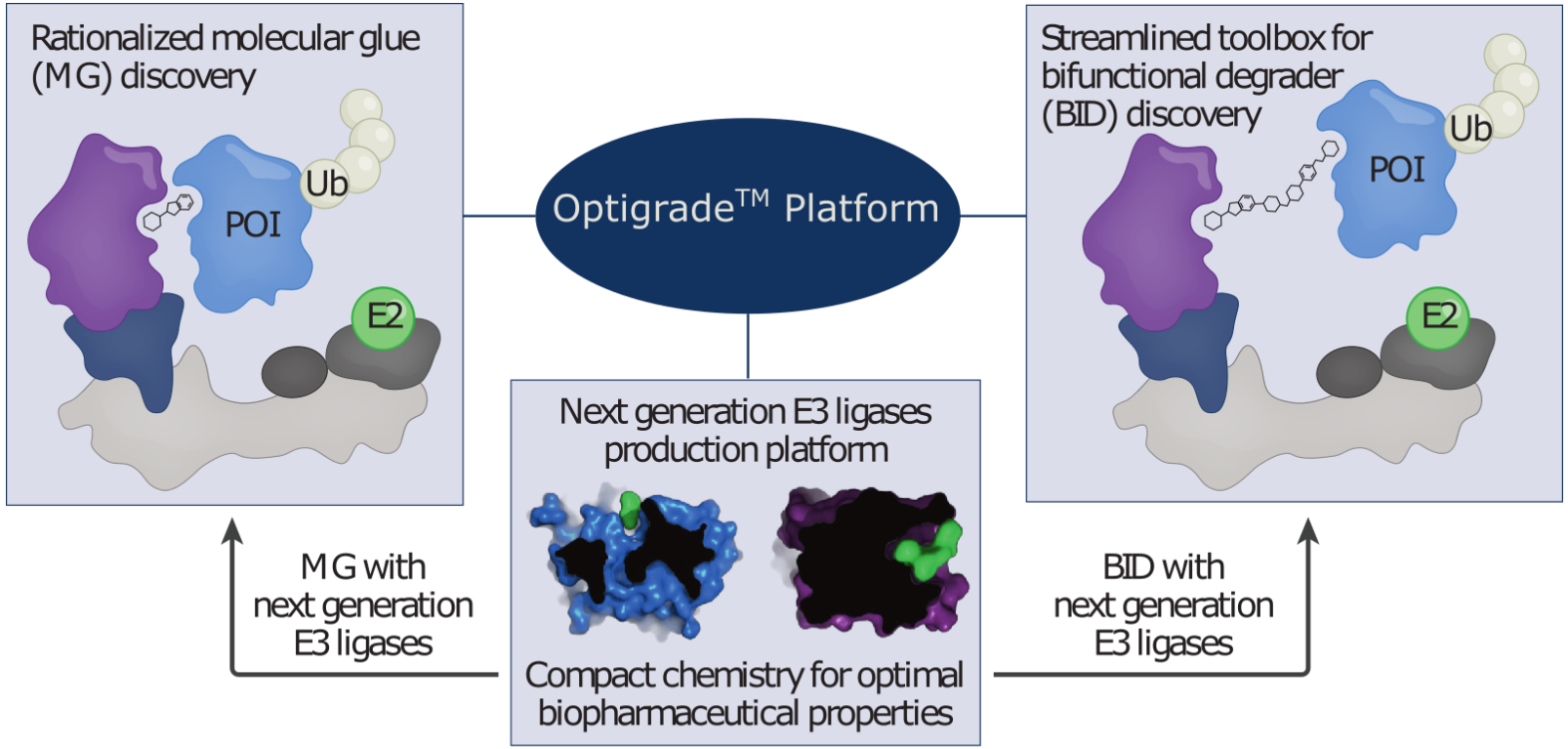
DEVELOPMENT OF PROJECTS AND MOLECULAR TARGETS ANNOUNCEMENT

Optigrade™ discovery platform – importance of structure



Optigrade™ – addressing Molecular Glues, Bifunctional Degraders and novel Ubiquitin E3 Ligases

- Industry leading capability in protein engineering and structural biology
- Unique structure-guided lead optimization paradigm gives high selectivity with good pharmaceutical properties
- Proprietary, focused library of molecular glue compounds with improved chemical stability
- “Silent” ligase ligands for enhanced selectivity of bifunctional degraders (no intrinsic degradation capacity)
- Library of ~100 novel E3 Ubiquitin Ligase proteins

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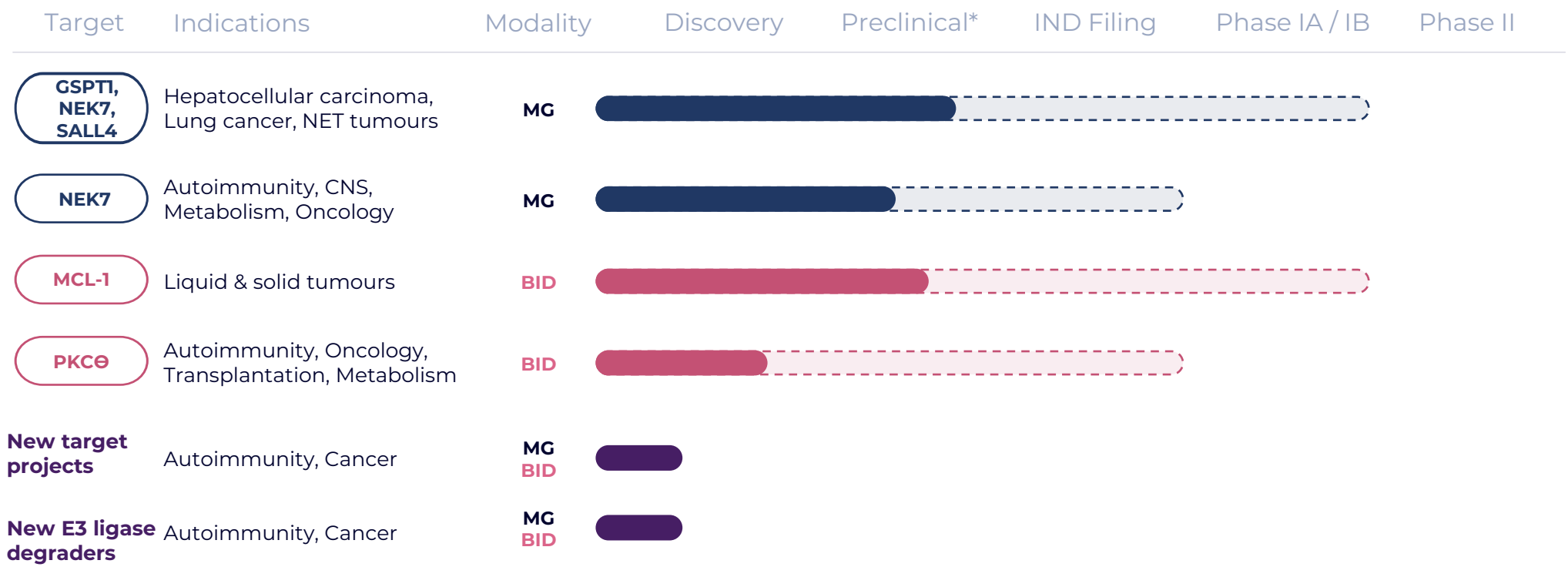
Fully owned pipeline



Preclinical stage include IND-enabling studies, **BID – Bi-functional Degrader; **MG** – Molecular Glue*

Assumed stage at the end of 2025

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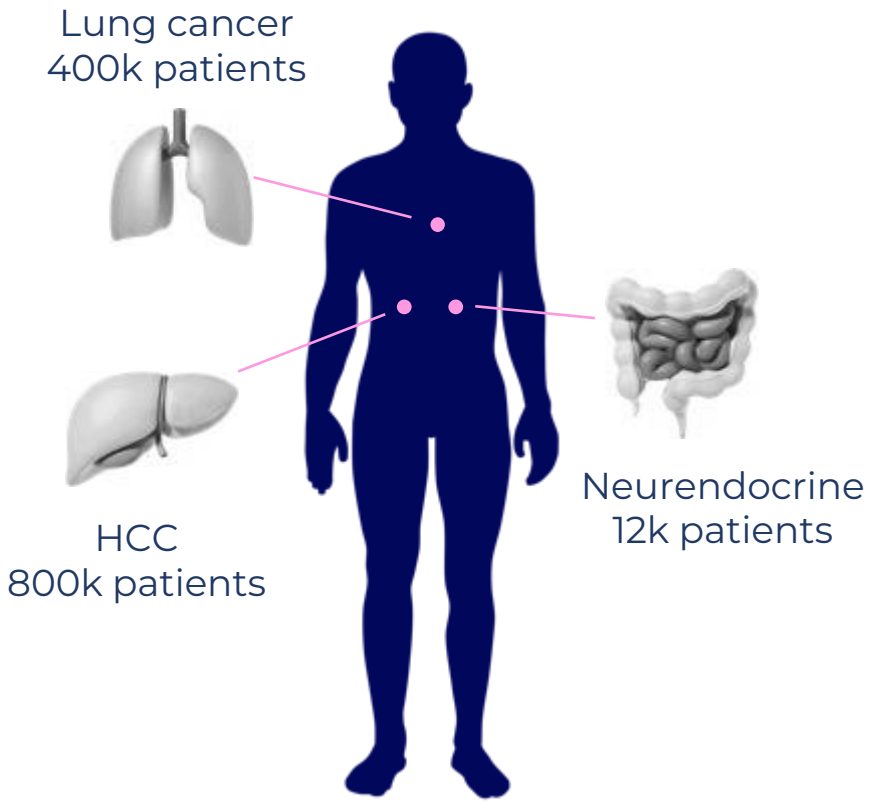
CT-01: Multi-target GSPT1, NEK7 & SALL4 degrader



CPT-6281: first-in-class MG degrader of GSPT1, SALL4 & NEK7

GSPT1 degradation leads to an Integrated Stress Response and induction of apoptosis in HCC cells

SALL4 is expressed foetal liver, silenced in adults, but often re-expressed in HCC and correlates with poor prognosis

NEK7 degradation leads to reduction of IL-1b production – a well-establish procarcinogenic factor. Reduction of IL-1b levels enables activation of the immune response

CPT-6281 is a **pro-drug** activated by an enzyme present at high levels in the liver, lungs and certain gastrointestinal tumors

Only TPD can address 3 undrugged targets GSPT1, SALL4 and NEK7 with one molecule, and in addition, NEK7 has a pathological scaffolding role that cannot be blocked with inhibitors

A unique degradation profile combined with target tissue pro-drug activation for liver, lung and neuroendocrine cancers

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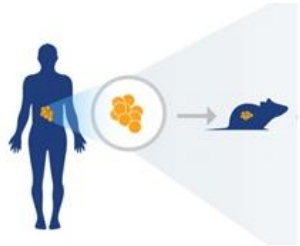
Convincing tumor growth inhibition in HCC PDX models

CrownBio
CONNECTING SCIENCE TO PATIENTS

PDX Models/

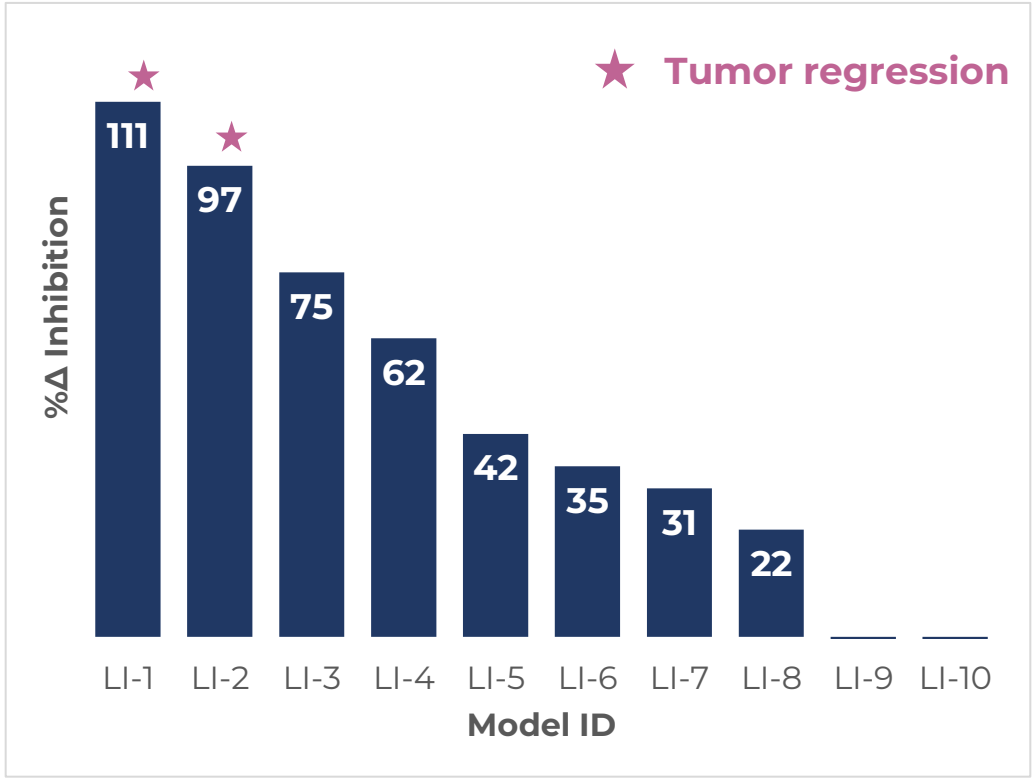


10 randomly selected HCC models, CPT-6281 100mg/kg, BID

3 animals per vehicle & treatment groups

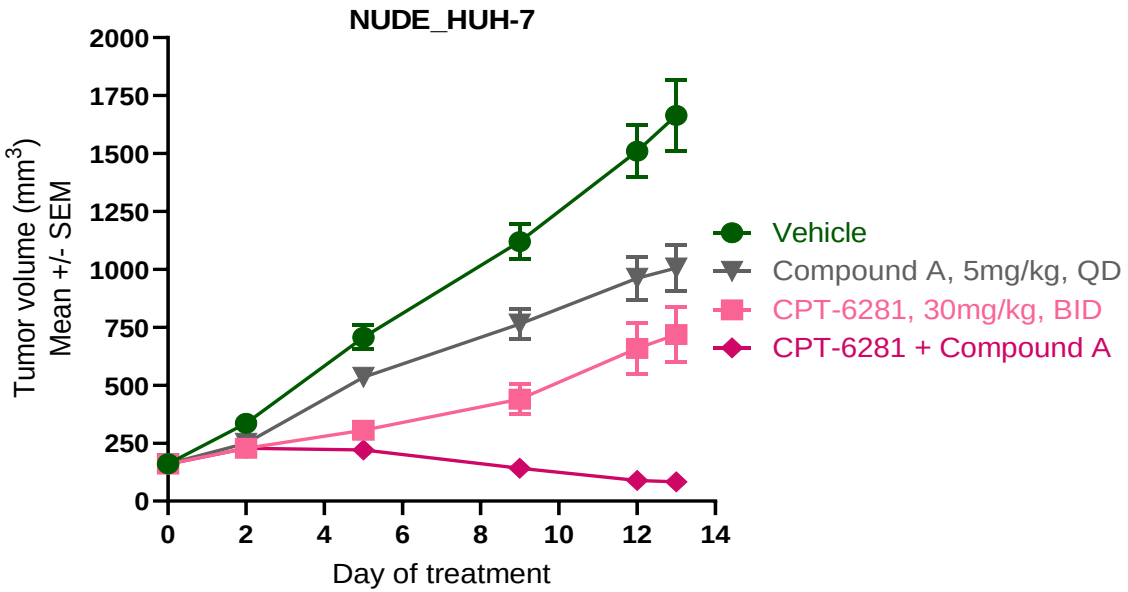
Efficacy demonstrated in 8/10 models; TGI>50% in 4 models, 2 models with regression

Tumor Growth Inhibition



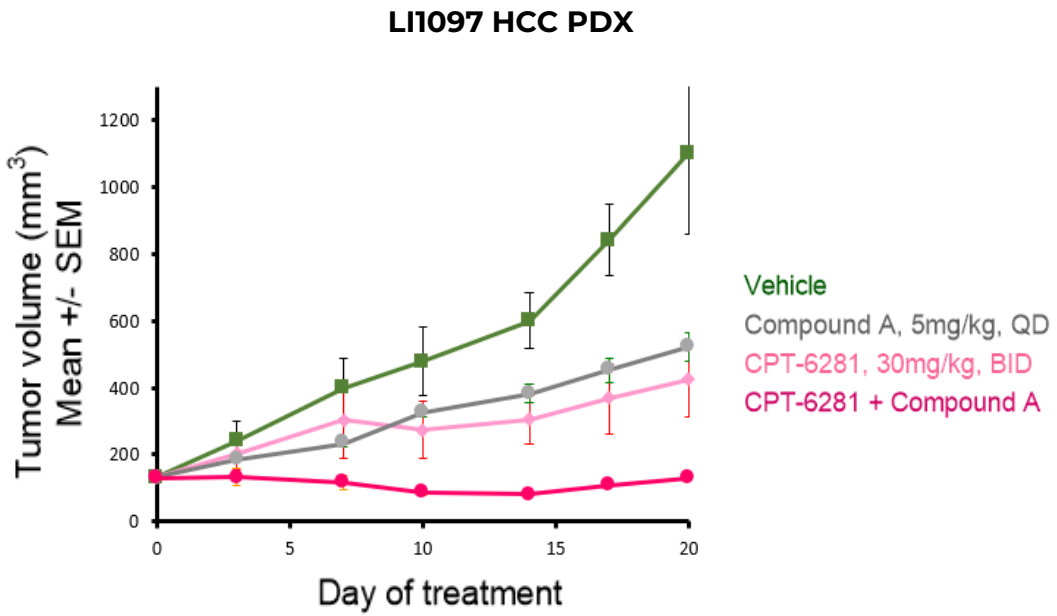
Clear synergy of CPT-6281 with an approved drug

HUH-7 Xenograft Model

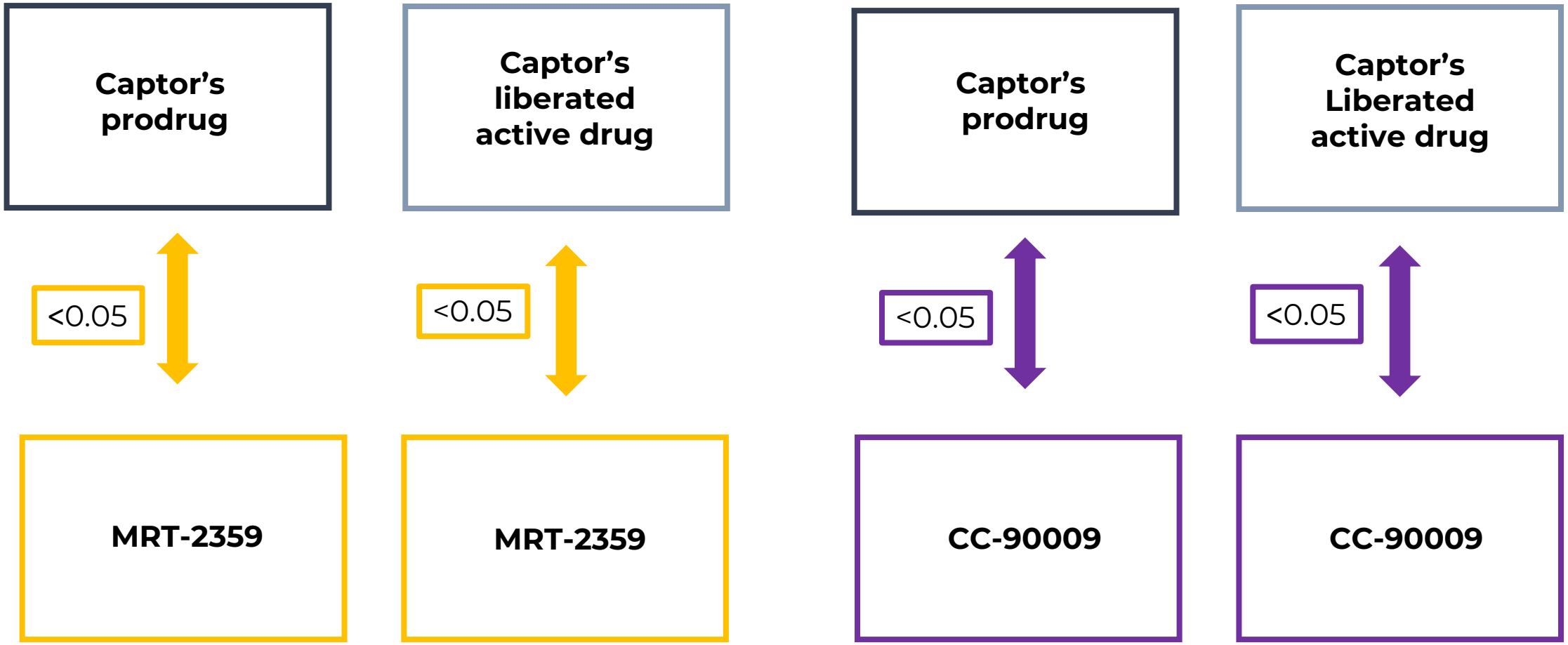


CrownBio
CONNECTING SCIENCE TO PATIENTS

PDX Models/



Captor compound is structurally different to the clinical ones



„Tails" comparison;
Tanimoto comparison; Tanimoto coefficient <0-1>, where 0 means completely different and 1 identical

Based on MorganFingerprints (r=3, 2048)

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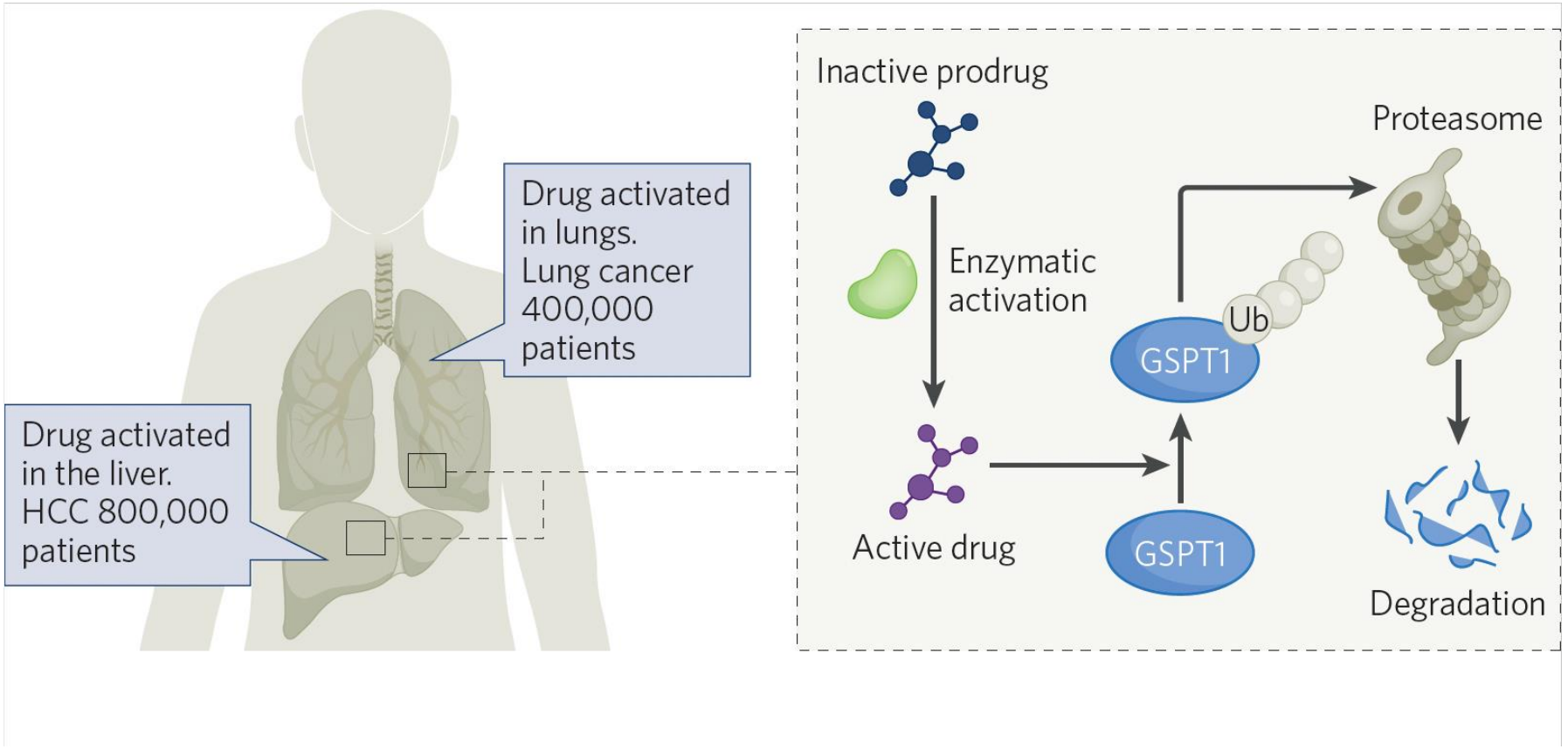
CPT-6281 – pro-drug with target tissue activation



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Development timeline – CPT-6281

Molecular Glue

Initial indication:

hepatocellular carcinoma

Degradation profile

GSPT1, SALL4 + NEK7

Target tissue activated pro-drug

DRF studies complete

GLP toxicology underway

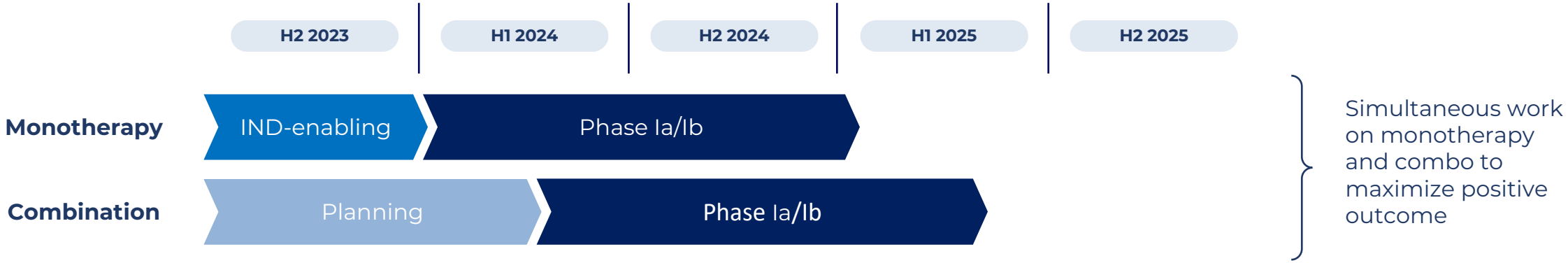
Manufacturing scale-up complete

Expected milestones:

CTA submission for initiation of clinical trials in Q3 2023

Phase Ia/Ib top-line data to be reported by the end of 2024

Combination study data by end 2025



CT-01 – GSPT1

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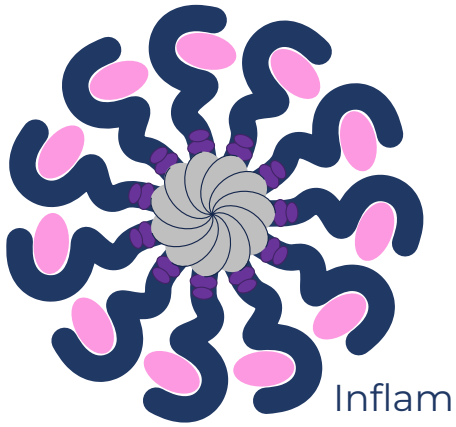
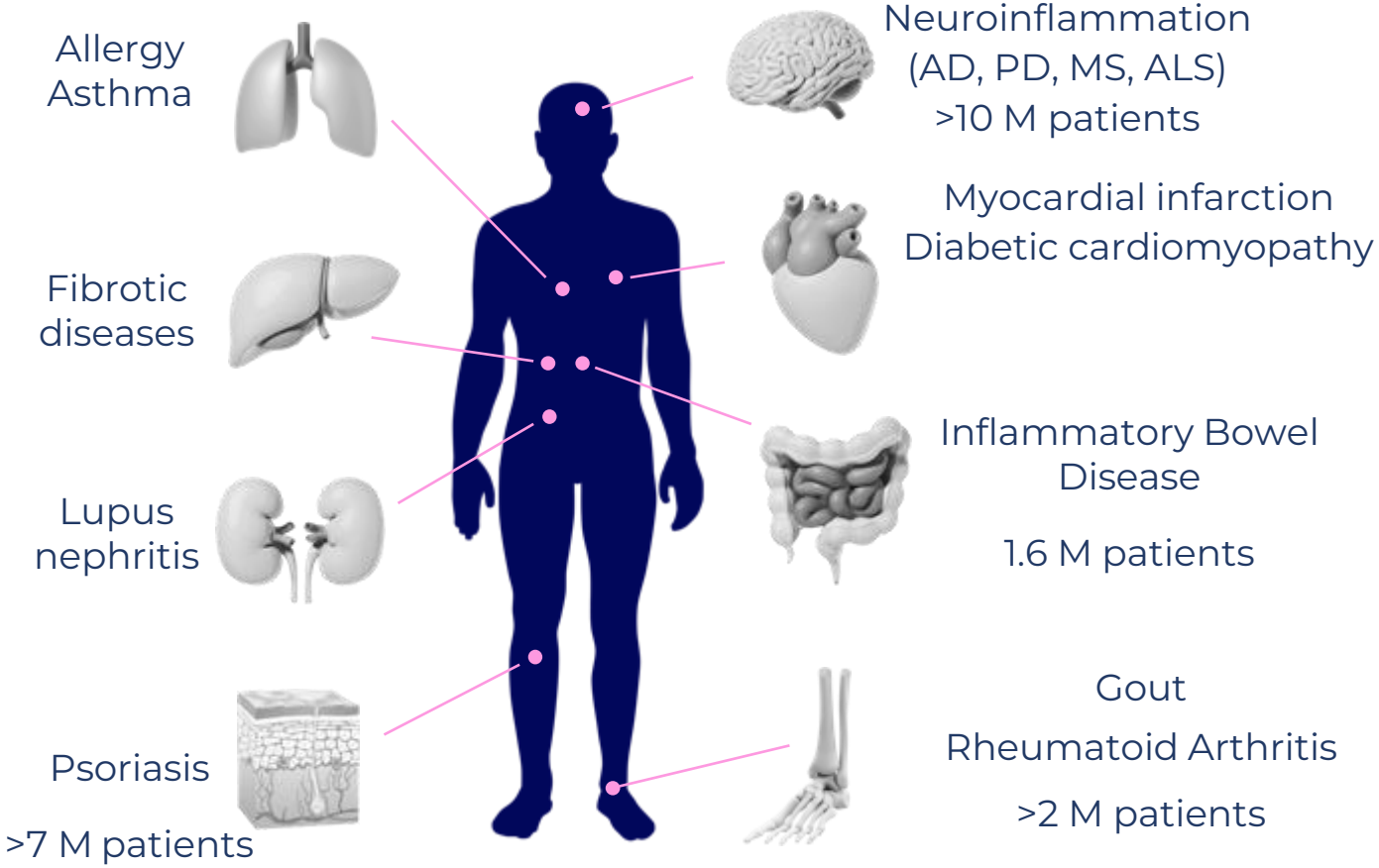
CT-02: Vast market potential for inflammasome modulators



Inflammasome
:NEK7 complex

Caspase 1

IL-1β **IL-18** **Gasdermin**



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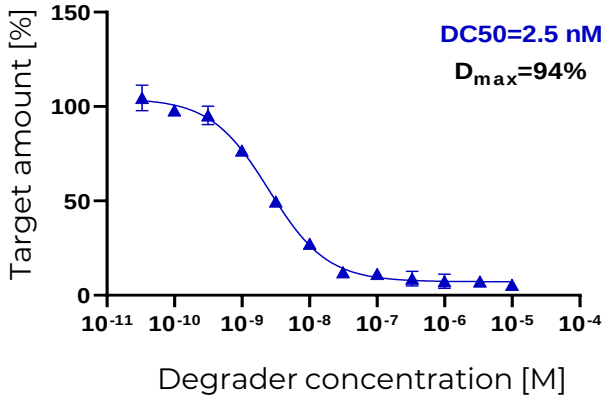
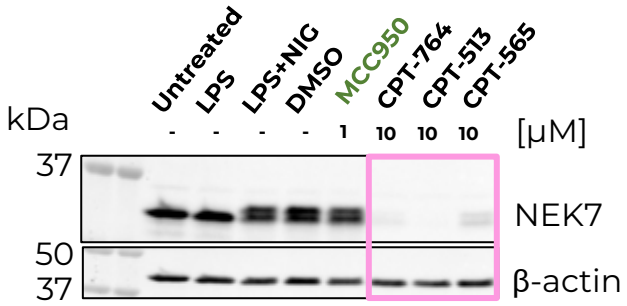
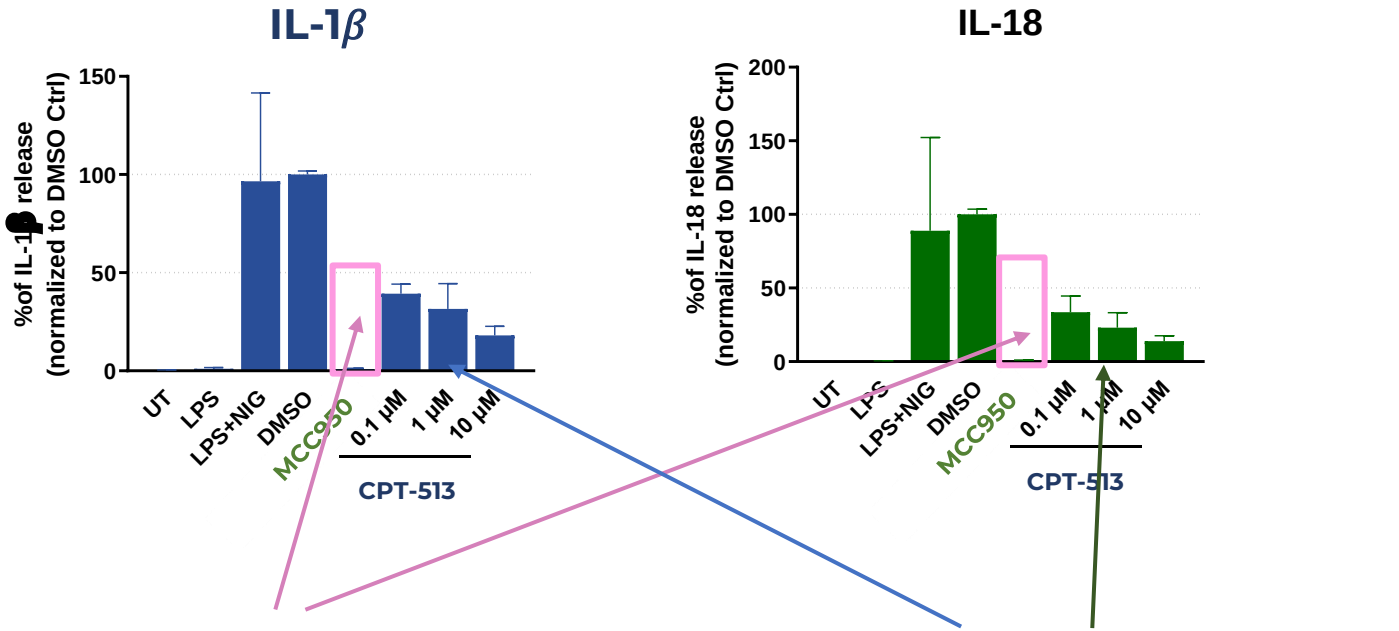
Therapeutic edge in autoimmunity through NEK7 degradation

Potent degradation of NEK7







Inhibitor completely inactivates the system

Degrader partially attenuates the system with a dose response

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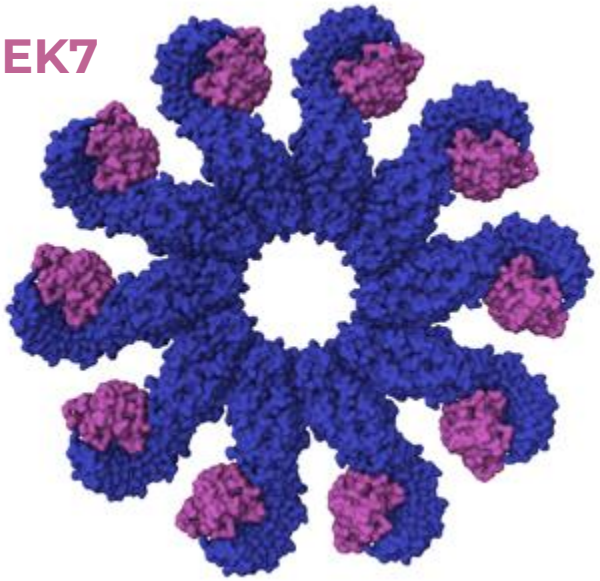
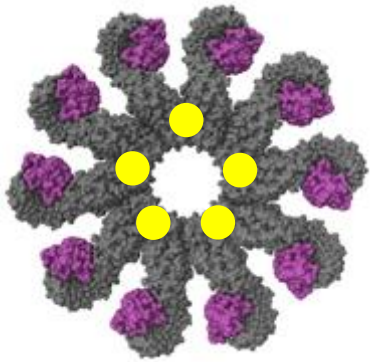
Differentiated intervention in NLRP3 pathway *via* NEK7 degradation

Aberrant activation of NLRP3

Disease development

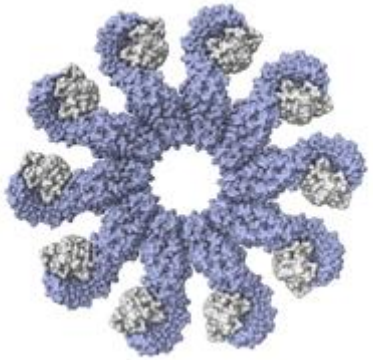


IL-1β
IL-18



NLRP3 **inhibition**
Complete inactivation of NLRP3

Therapeutic response
Increased infections via reduced immunity



NEK7 **degradation**
Partial inactivation of NLRP3

Therapeutic response
Maintained immunity

CT-02 – NEK7

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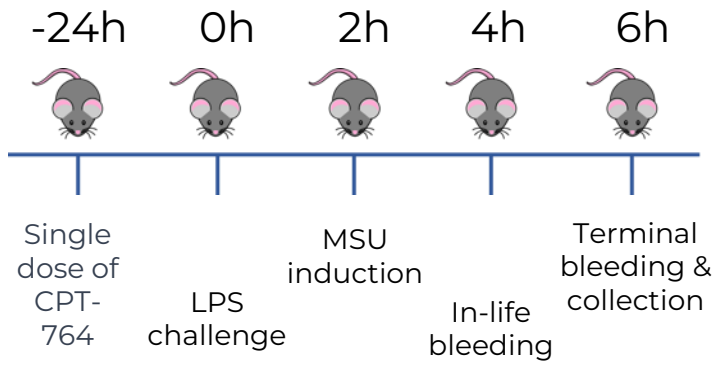
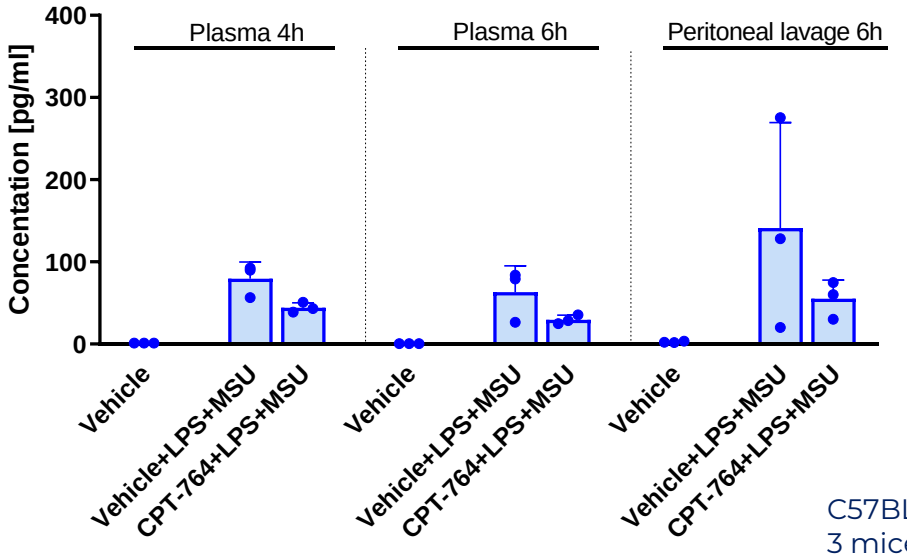
In vivo PoC in peritonitis model & brain penetration

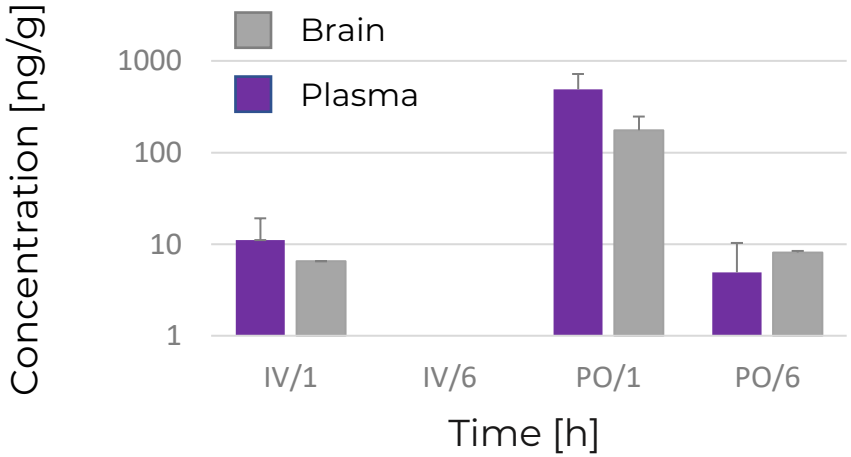


IL-1β levels



C57BL/6-Crbntm1.1Ble/J;
3 mice per group

Plasma and brain concentration of CPT-565



Time point	Brain exposure (ng/g Tissue)	Plasma exposure (ng/ml)	Brain to Plasma ratio
1h (IV)	6.55	11.1	0.59
6 h (IV)	BLQ	BLQ	NC
1 h (PO)	175.2	494.9	0.35
6 h (PO)	8.1	4.9	1.65

NEK7 summary

Molecular Glue

Multiple series of NEK7 molecular glue degraders discovered

Lead compounds show low nanomolar potency and excellent PK properties

In-vivo proof-of-efficacy for systemic degraders achieved in initial study

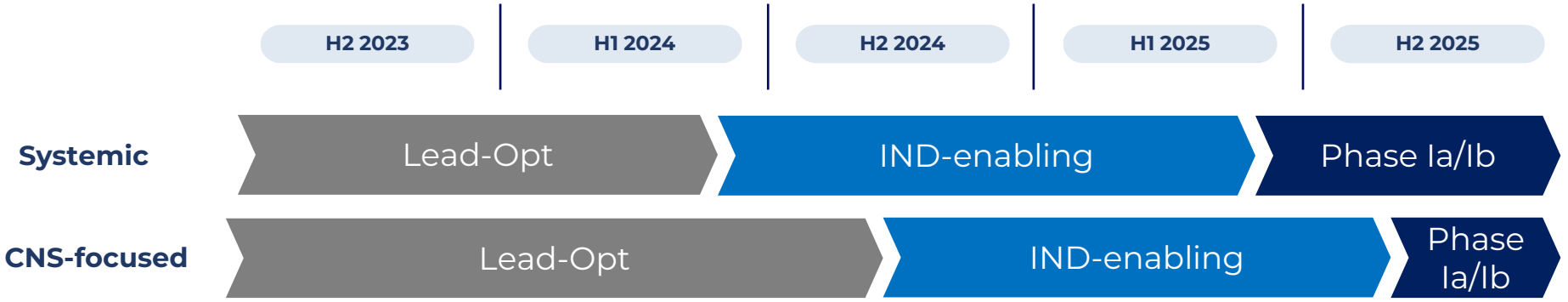
Larger optimised efficacy study reporting soon

Significant brain penetration shown in vivo with subset of molecules

In vivo PoC (efficacy) for the brain-penetrant series in 2023

Ready for IND-enabling studies in H1 2024

Degradation, but not inhibition, switches off both pathological functions of NEK7 – kinase and scaffolding



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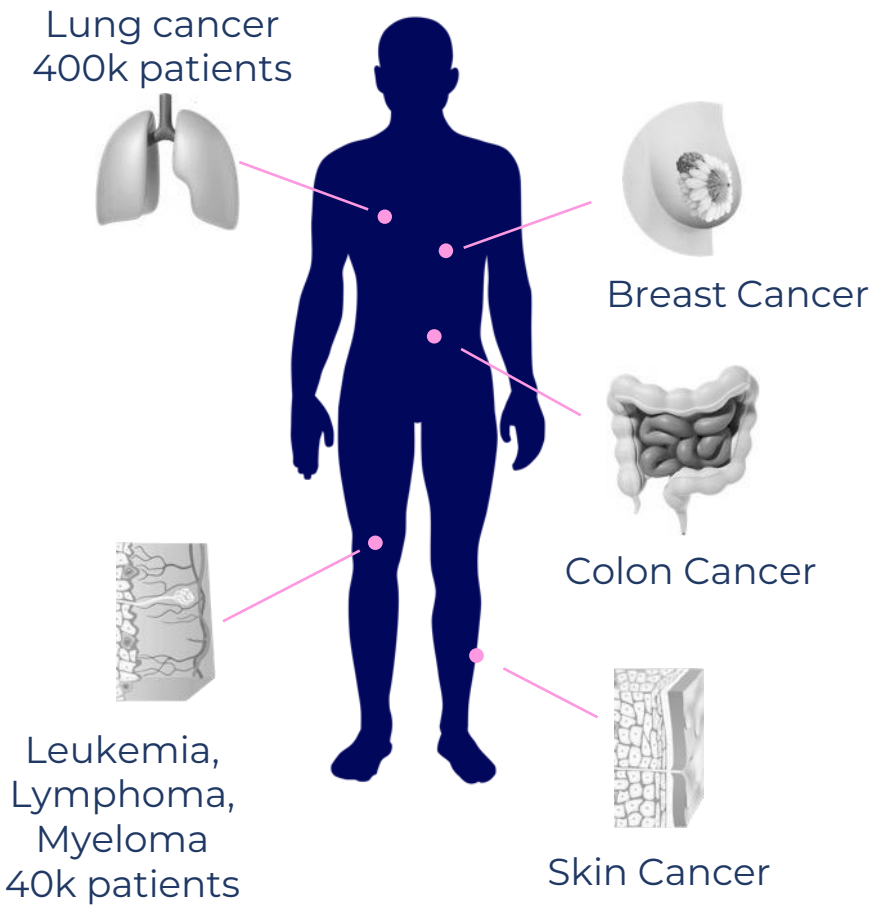
CT-03: MCL-1 – a critical pathway of cancer resistance



MCL-1 is one of the most amplified proteins in cancer

A critical resistance mechanism in haematological and solid tumours

Ablation of MCL-1 protein directly attenuates tumours *in vivo* as monotherapy & sensitizes tumors for other chemo- and targeted therapies

Adequate ablation of MCL-1 requires rapid and sustained action & high target coverage

Use of inhibitors causes accumulation of MCL1 in cancer cells

Degraders have a different mode of action, without accumulation of MCL1

Degradation of ~70% of MCL-1 induces apoptosis, while inhibitors require nearly 100% of target coverage. This, together, with optimized clearance expands the therapeutic window from the perspective of cardiotoxicity

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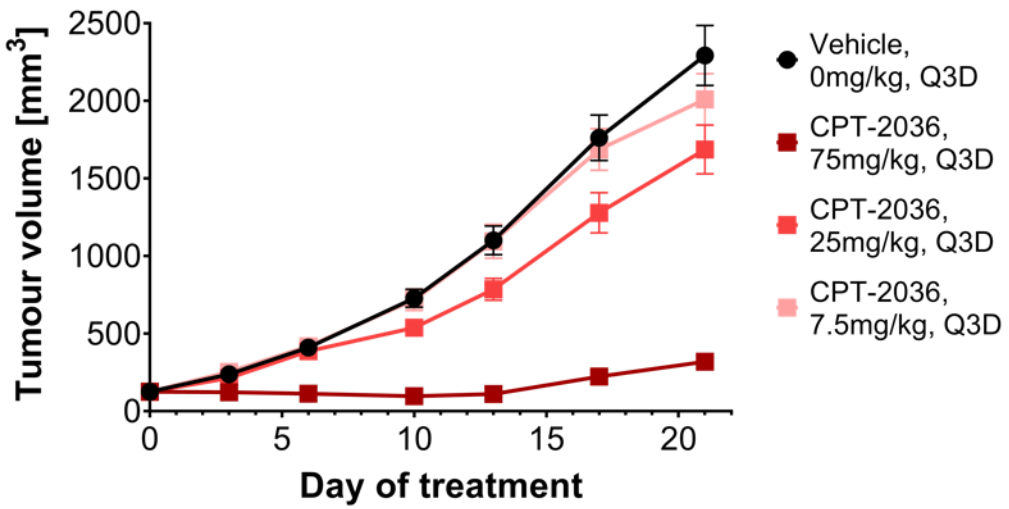
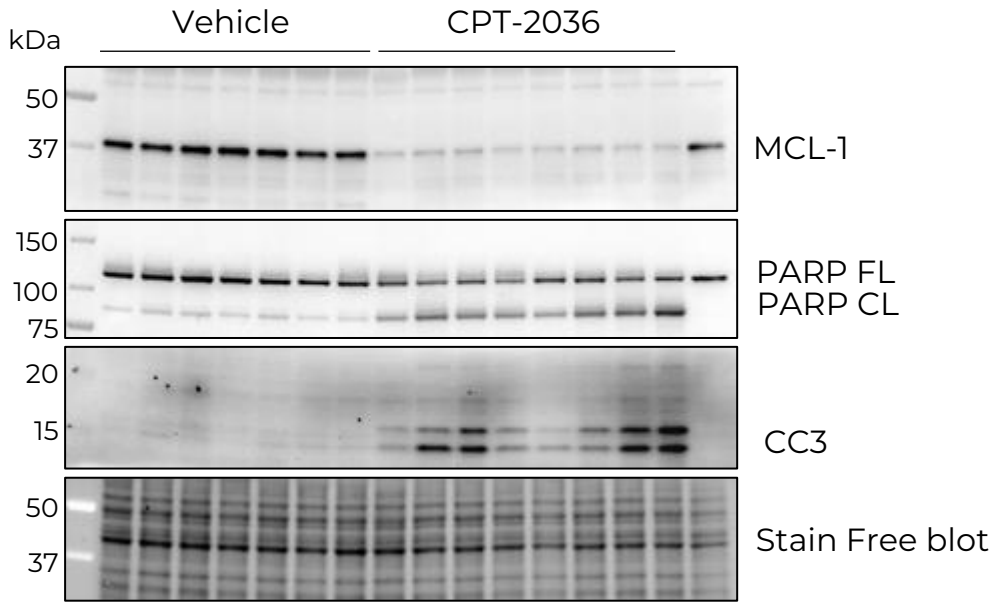
MCL-1 degraders effective in AML and lung cancer models

Strong tumor growth inhibition in intermittent dosing (every 3 days)



Mice with 150 mm^3 MV4-11, IV

Potent MCL-1 degradation and apoptosis activation in SCLC xenograft model



DMS-114, 75 mpk, IV, single injection

CT-03 – MCL1

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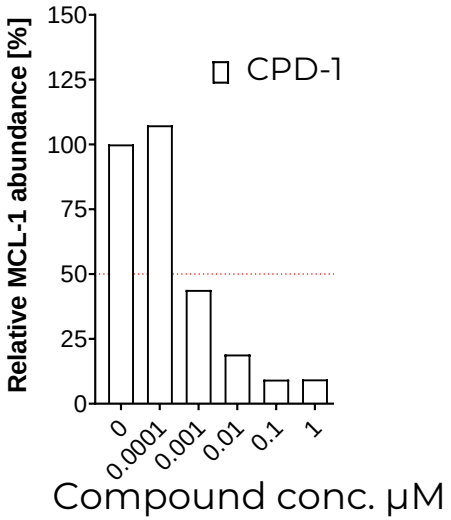
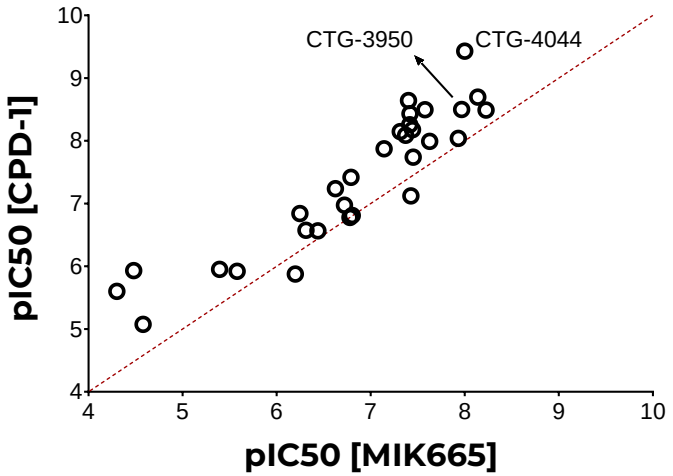
Ultrapotent compounds active *ex vivo* and in cancer cell lines

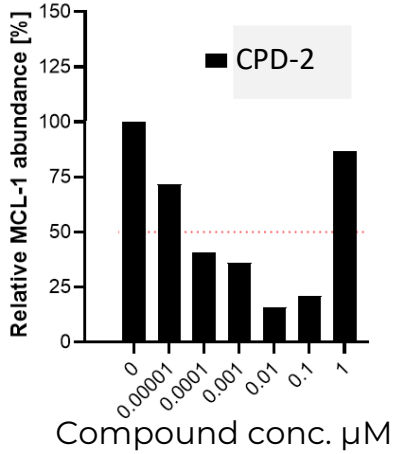


DC50 ~1 nM



CPD-1 was tested in the panel of 30 PDX AML cells ex vivo

CPD-1 shows 2-10x better activity than the clinical MCL-1 inhibitor, MIK665 (Novartis/Servier)



CPD-2 shows remarkable DC50 <100 pM

CPD-1 constitutes a great potential for ADC development

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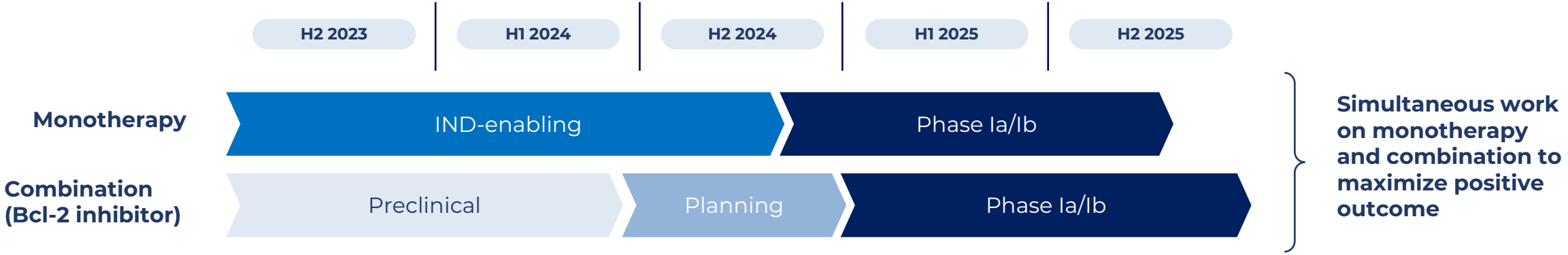
Development timeline – CT-03

Bifunctional Degrader

Initial indications:
blood cancers, subsequently solid tumours

Degradation profile:
Selective first-in-class MCL1 degrader

Expected milestones:
- IND/CTA approval in Q3/4 2024
- Initiation of Phase I clinical trial Q4 2024
- Phase Ia/Ib top-line data reported 2025



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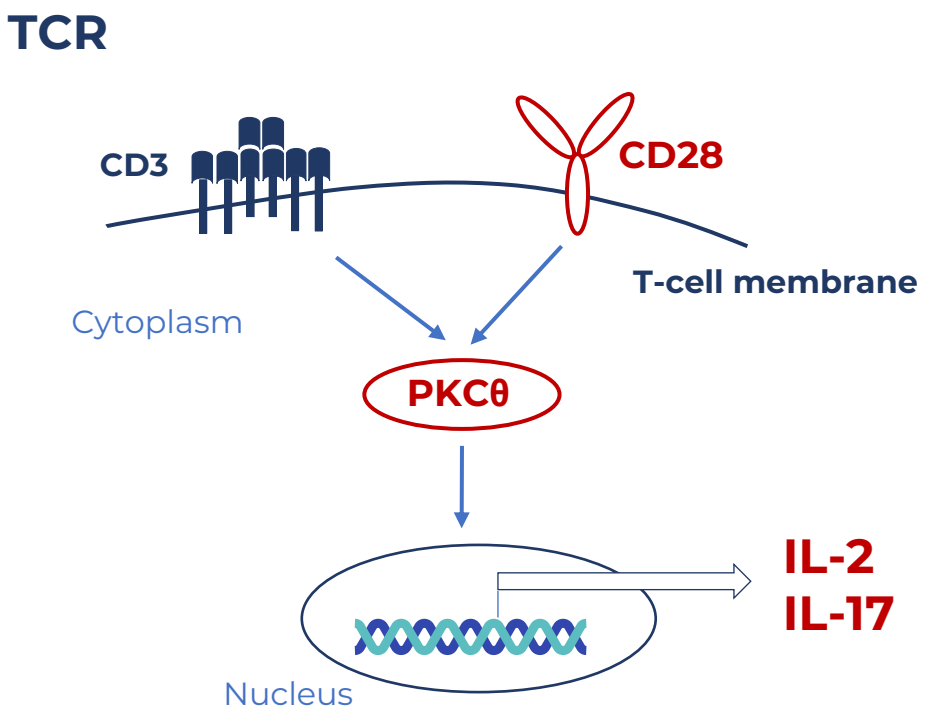
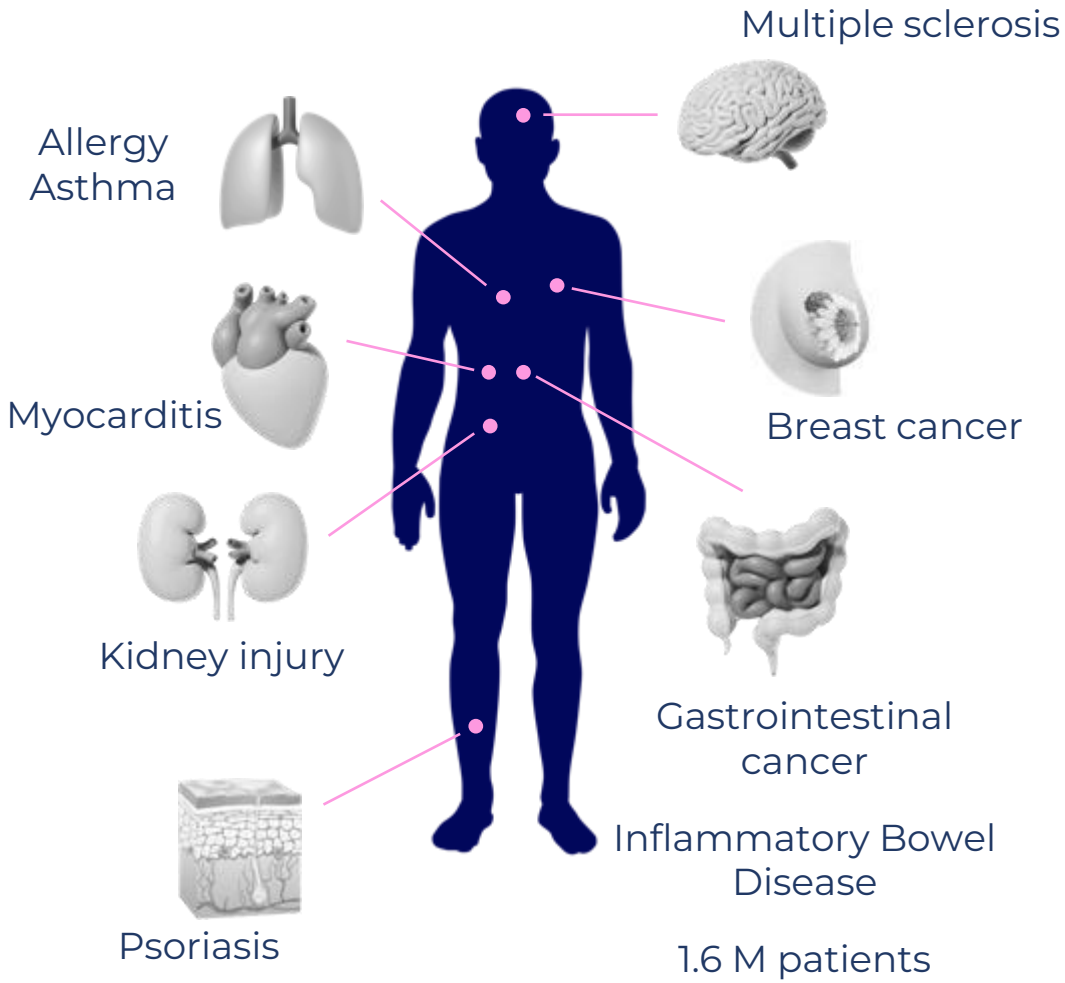
CT-05: PKC$_\theta$ an inadequately drugged high value target





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Unique opportunity tailored for bifunctional degraders

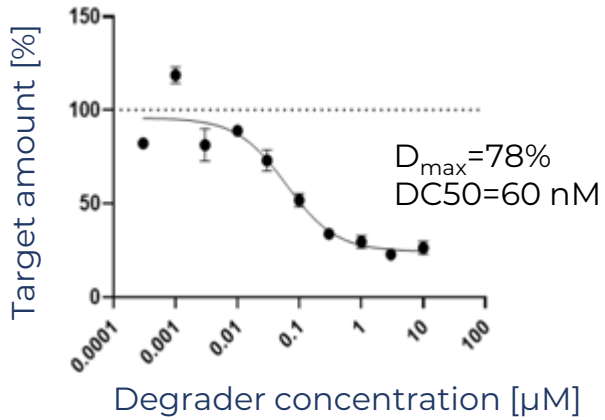
Multiple PKC$_\Theta$ inhibitors were in clinical trials:

1st generation, e.g. pan-PKC Sotrastaurin – many side effects

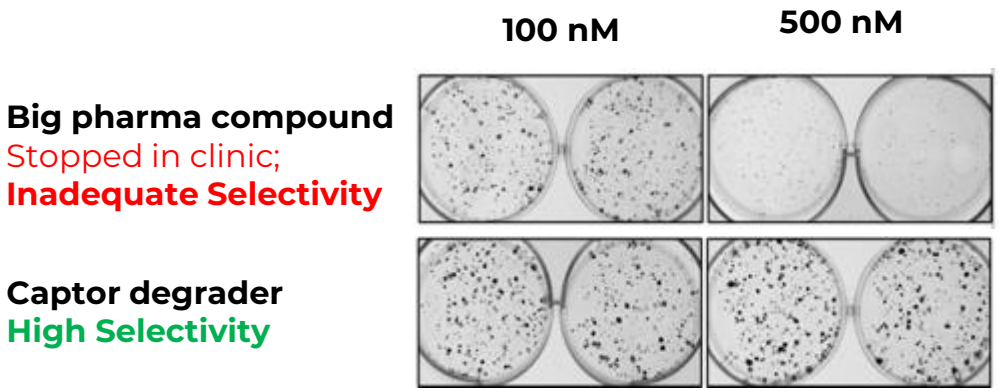
2nd generation, e.g. Astellas, AbbVie, Celgene - PKC$_\Theta$-selective but showed unknown off-targets or poor coverage

Recent revival, allosteric compound deal: Exscientia-BMS

Potent degradation in immune cells



Inhibitor shows significant effects on non-immune cells
Degrader has no effect in non-immune cells



2023 expected milestones

Demonstration of ***in vivo*** **efficacy** in inflammation model

Degradation with BIDs offers selectivity and coverage umatched by inhibitors

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Captor Partnering

Existing collaborations:

- Sosei Heptares collaboration was extended and continues as planned
- Ono partnership is advancing well with positive data confirming our approach

Pipeline partnering strategy:

- CT-01 + CT-03: Early clinical trials for human POC and partner at point of optimum risk/reward
- CT-02 + CT-05 – Preclinical POC in 2023 to support partnering discussions

Platform partnering opportunities:

- Novel E3 Ubiquitin Ligase data has generated interest & contact from pharma partners
- Additional discovery collaborations against new targets (outside of our pipeline)
- New area: applying high-potency degraders in Antibody Drug Conjugates (ADCs)

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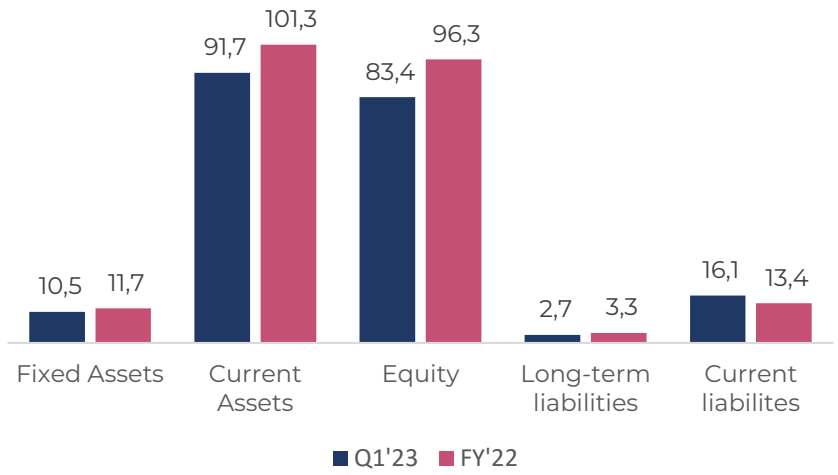
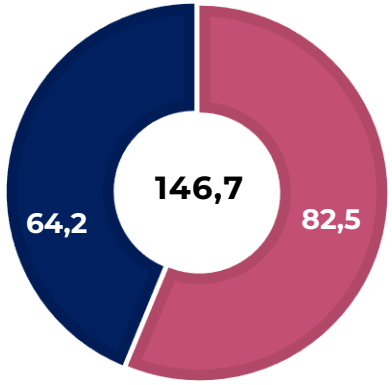
FINANCIALS AND PLANS FOR THE FUTURE

Strong balance sheet and cash position

Consolidated statement of financial position (PLN, M)



Cash position



Available funding secured (PLN M; as of 31/03/2023):

Total : PLN 146,7 M

- PLN 82,5 M
  cash, short-term bonds
- PLN 64,2 M
  grant funding agreements with NCBR

R&D costs in Q1**:**

Total : PLN 15,6 M

Cash outflow in Q1**:**

Total : PLN 8,3 M

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Next steps

2023

- **CT-01**: Phase Ia/Ib initiation in liver cancer patients
- **CT-02** and **CT-05**: *In vivo* proof of concept in autoimmunity
- In cell degradation of target with novel E3 ligase-based degrader

2024

- **CT-03**: Phase Ia/Ib initiation in haematological cancer patients
- **CT-01**: Clinical readouts: safety, pharmacology, & mechanism
- First degrader of a new target based on novel E3 ligase
- New partnering in immunology

Captor Therapeutics®

Q&A SESSION

Captor Therapeutics®

Captor Therapeutics S.A
ul. Duńska 11
54-427 Wrocław, Poland

Captor Therapeutics GmbH
Hegenheimermattweg 167A
4123 Allschwil, Switzerland

Contact: t.shepherd@captortherapeutics.com