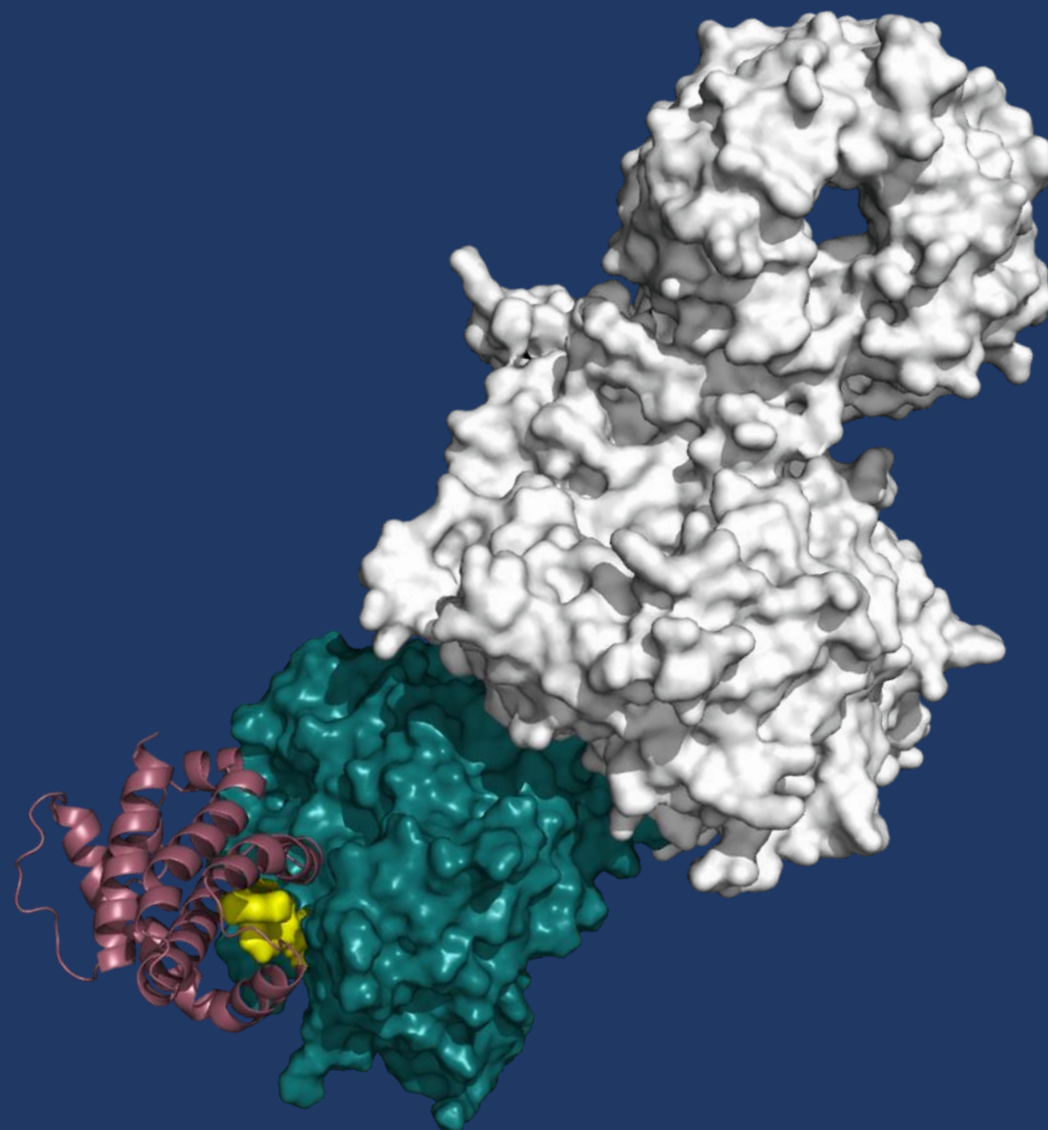




Captor
Therapeutics®

*Pioneering targeted protein
degraders for human health*

H2 2025



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Beyond the Pipeline

Captor's Research in Print

Targeted degradation of GSPT1 and NEK7 by a molecular glue prodrug for treatment of HCC

A Robust Crystallographic Platform for High-Throughput β -Catenin Ligand Discovery

Exploration of chemical probes and conformational flexibility of GID4 - the substrate receptor of human CTLH E3 ligase complex

Expression screen of TNFR1R347A, MyD88, IRAK4 death domains in E. coli followed by purification and biophysical characterization of TNFR1R347A death domain

Uniting the protein degradation generation

Expression Strategies for Recombinant HECT E3 Ligases in Escherichia coli

Comparative analysis of biophysical methods for monitoring protein proximity induction in the development of small molecule degraders

Targeted Protein Degradation: "The Gold Rush is On!"

Captor Express

CT 01: An innovative drug for liver cancer 

Technology Platform 

Changing the face of cancer treatment with MCL-1 degraders 

Redefining the future of cancer treatment with MCL-1 degraders 



In the Spotlight

CT-01 (ABS-752) featured by Drug Hunter as Molecule of the Month, June 2025



European Innovation Council (EIC) Accelerator grant for CT-03

European
Innovation
Council



Interview with Politykzdrowotna.com on discovering drug candidates in oncology



An experienced leadership team



Michal Walczak, PhD

Co-founder
CEO and CSO

- PhD ETH Zurich
- Post-doc at FMI Basel (Novartis Research Foundation) on TPD
- 14 years of drug discovery & TPD experience
- Co-founder of Alphamoon.ai



Anna Pawluk, PhD & MBA
Chief Operating Officer

- PhD Wroclaw University
- MBA WSH in Wroclaw
- 20 years of R&D experience



Tomáš Drmota, PhD
Chief Technology Officer

- PhD Charles University
- Post-doc at the University of Glasgow
- 25 years of R&D experience



Sylvain Cottens, PhD

Co-founder & SVP Chemistry

- PhD EPFL Lausanne
- Post-doc at Caltech
- Scientific expert & leader with 25+ years at Novartis
- Involved in 2 blockbusters (Afinitor & Gilenya)



Adam Łukojć, PhD

Chief Financial Officer

- Doctorate: Kozminski University
- Certificates and licences: Chartered Financial Analyst, Professional Risk Manager; investment advisor
- 20 years of experience in the capital market



Andrew Saunders DPM, FFPM

Chief Medical Officer

- MB BCh BAO BA, Medicine, Trinity College, Dublin
- FFPM, Royal College of Physicians, London
- 25 years in oncology clinical development, including global responsibility for Rituximab



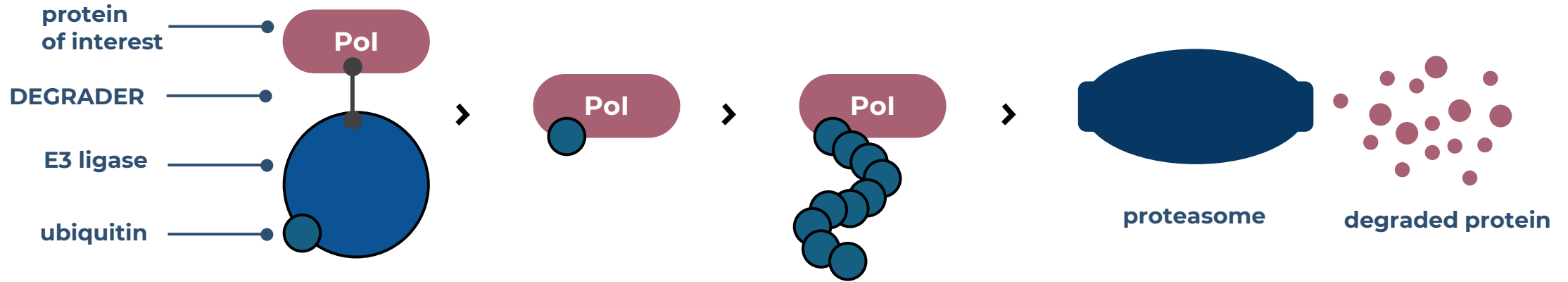
Robert Dyjas, MD, PhD

Head of Medical Affairs and Clinical Development

- MD, PhD, specialization in Internal Medicine (Medical University of Silesia)
- 20 years in clinical drug development, with strong focus on early phase trials of novel small molecules



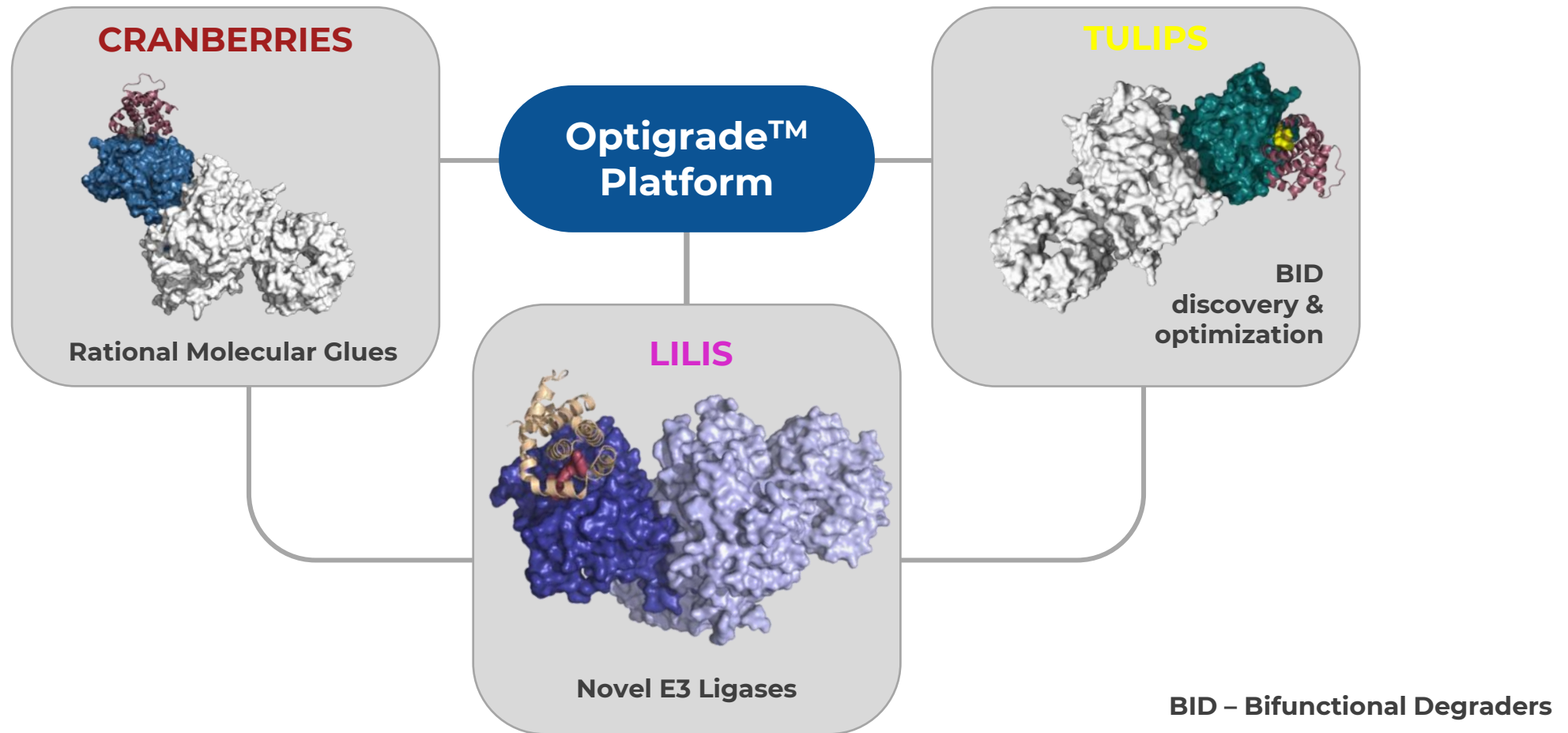
Targeted Protein Degradation expected to unlock \$974* bn by 2030



	Degraders	Inhibitors	mAbs	siRNA
Removing multiple pathological functions	✓✓✓	✗	✗	✓✓✓
Oral bioavailability	✓✓✓	✓✓✓	✗	✗
Uncoupling PK from PD = prolonged efficacy	✓✓✓	✗	✗	✓✓✓
Overcoming mutational resistance	✓✓✓	✓	✓✓	✓✓✓
Targeting scaffolding function	✓✓✓	✗	✓✓	✓✓✓
Brain-penetration	✓✓	✓✓✓	✗	✗
Accessing undrugged proteins	✓✓✓	✓	✗	✓✓✓

*Biopharmaceuticals Market by Type and Application: Global Opportunity Analysis and Industry Forecast, 2022-2030

Optigrade™ discovery platform – importance of structure



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

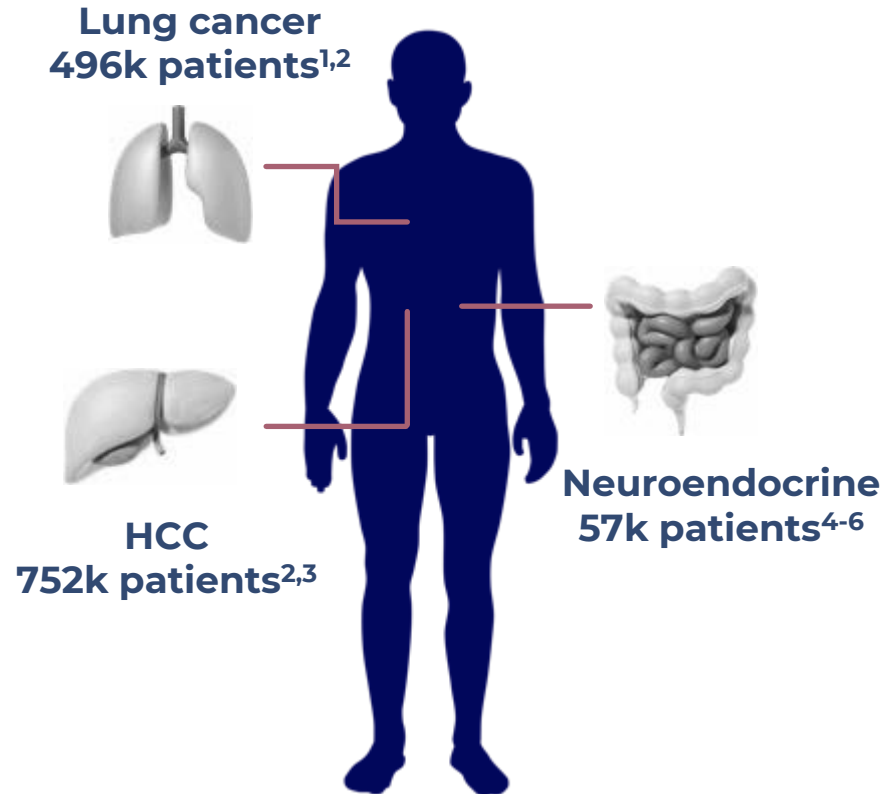
Fully-owned pipeline

#	Target	Indications	Modality	Discovery	Pre-clinical	CTA-enabling	Phase I
CT-01	GSPT1 & NEK7	HCC, Lung cancer, Rare cancers	MG				
CT-02B	NEK7	Neuroinflammation (Parkinson's Disease, ALS, MS)	MG				
CT-02S	NEK7	Systemic autoimmunity (IBD, Gout, Dermatological diseases)	MG				
CT-03	MCL-1	Liquid & solid tumors	BID				
CT-05	PKCθ	Autoimmunity, Transplantation, Metabolism	BID				
	Undisclosed	Oncology, Autoimmunity, CNS, Rare	MG/BID				
	Novel E3	Oncology, Autoimmunity	MG/BID				

BID – Bifunctional Degradator
MG – Molecular Glue

CT-01: First-in-Class GSPT1 Targeted Degradator for Hepatocellular Carcinoma (HCC)

CT-01: A first-in-class molecular glue prodrug degrader of GSPT1 & NEK7



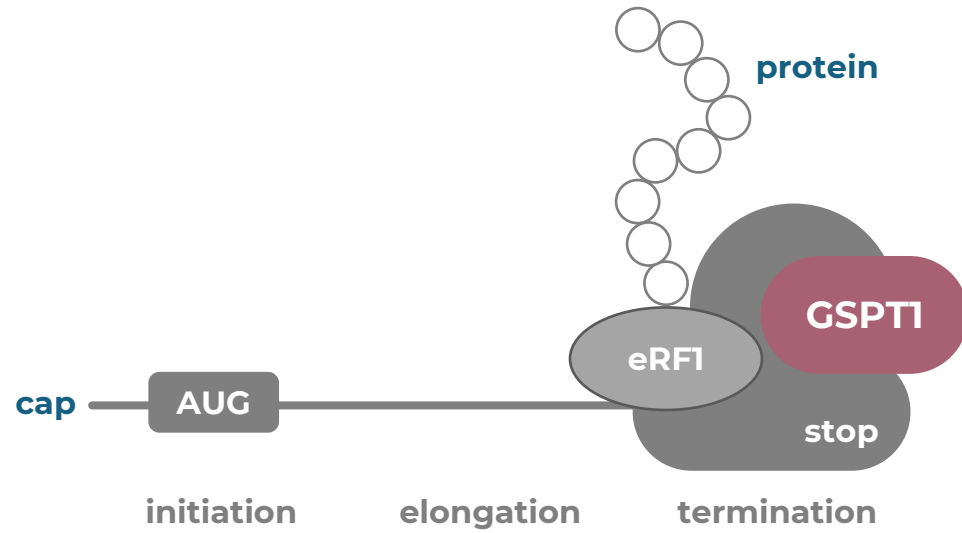
GSPT1 degradation leads to an Integrated Stress Response (ISR) and induction of apoptosis in HCC cells

NEK7 degradation leads to reduction of IL-1 β production – a well-established pro-carcinogenic factor. Reduction of IL-1 β levels in the tumor microenvironment enables activation of the immune response

CT-01 is a prodrug activated by an enzyme present at high levels in the liver, lungs and certain gastrointestinal tumors

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

Degradation of GSPT1 halts proliferation of cancer cells



1. Hellen C. U. T., *Cold Spring Harb Perspect Biol*, 2018
2. Salas-Marco, J. & Bedwell, D. M., *Mol Cell Biol*, 2004
3. Kurosaki, T. & Maquat, L. E., *J Cell Sci*, 2016

Target Biology and Therapeutic Rationale

G1 to S phase transition 1 protein (GSPT1, eRF3a)

is a translation termination factor that regulates mRNA translation¹

GSPT1 and eRF1 form a translation termination complex that facilitates the nonsense mediated mRNA Decay^{2,3}

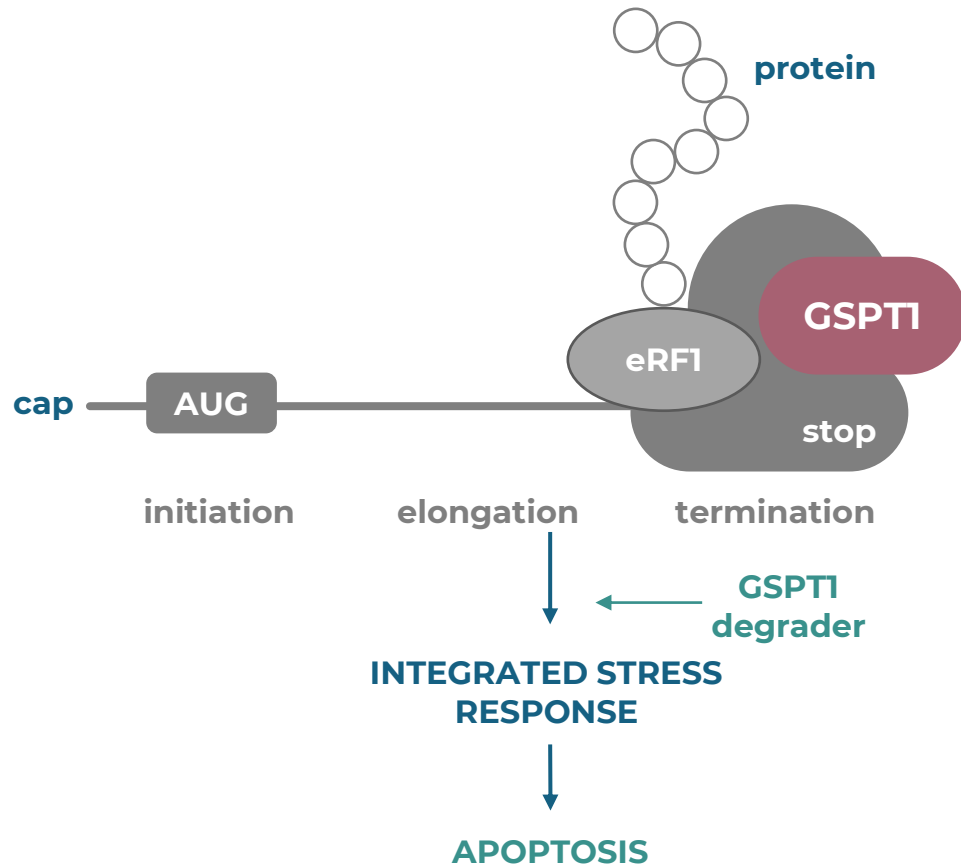
Clinical opportunity

Targeting protein translation GSPT1 degradation offers treatment options of:

1. Hepatocellular Carcinoma (HCC)
2. Lung cancer
3. Breast cancer
4. Gliomas
5. Rare cancers, e.g.: hepatoblastoma, angio- and liposarcomas

Degradation of GSPT1 induces apoptotic death in cancer

NEK7 degradation attenuates tumor-microenvironment *via* IL-1 β depletion



GSPT1 degradation leads to apoptosis via induction Integrated Stress Response (ISR)

An excellent opportunity for targeting of cancer cells that require translational adaptations and efficient protein synthesis

CT-01 degrader is a pro-drug converted by an enzyme elevated in the inflamed liver, lungs and blood-brain barrier.

The active molecule is released in HCC and features:

- 1) poor cell membrane penetration and
- 2) fast clearance, both of which significantly expand the therapeutic window.

CT-01 degrades also NEK7, whose pro-carcinogenic role is manifested in stabilization of MDSCs and TAMs in Tumor Micro-Environment (TME)

Downregulation of NEK7/IL-1 β reduces inflammation and improves outcomes of treatments

Dual GSPT1 & NEK7 degrader is in Phase 1 for liver cancer

CT-01 due to its mechanism & dual activity could become the-only-in class drug

ABS-752 - Phase 1 clinical studies in hepatocellular carcinoma (HCC)

Dose escalation initiated in May 2025

High unmet medical need in advanced HCC

Est. market size of \$3billion could grow exponentially through extending overall survival

Different from mono-GSPT1 degraders

Targets cancer cell proliferation (GSPT1) & tumor microenvironment (NEK7/IL-1 β)

Prodrug - activated by VAP-1 overproduced in diseased liver tissues and in other cancers

CT-01 synergizes with everolimus and potentially with standard of care *via* inflammation reduction

Standard of care fails to significantly extend lives of HCC patients

Improvement of modest survival of patients offers accelerated path to approval

Line of therapy	Therapy	Survival Benefit vs Sorafenib [months]	FDA Approval
1	Tecentriq + Avastin	+5.8 ¹	uHCC / mHCC
1	Imfinzi + Imjudo	+2.7 ²	uHCC
1/2	Nexavar	0.0 ³	uHCC
2	Opdivo	+1.7 ⁴	uHCC (Post sorafenib)
2	Cabometyx	+2.2 ⁵	uHCC (Post sorafenib)

Market Research Provider	Base (Year / \$B)	Future (Year / \$B)	CAGR (%)
Vision Research Reports ⁶	2024: \$3.2	2033: \$11.6	15%
SNS Insider ⁷	2022: \$2.9	2030: \$12.9	20%
Skyquest ⁸	2022: \$2.7	2030: \$11.4	20%
Research and Markets ⁹	2022: \$2.4	2030: \$7.8	15%
Polaris ¹⁰	2021: \$2.2	2030: \$10.4	20%

Annual growth rate of HCC cases is 15-20%

uHCC – unresectable HCC
mHCC – metastatic HCC

(1) J Hepatol. 2022;76(4):862-873 | (2) NEJM Evid 2022;1(8) | (3) N Engl J Med 2008; 359:378-390 | (4) Lancet Oncol 2022 Jan;23(1):77-90 | (5) N Engl J Med 2018 Jul 5;379(1):54-63
(6) <https://www.visionresearchreports.com/liver-cancer-drug-market/40952> | (7) <https://www.snsinsider.com/reports/liver-cancer-therapeutics-market-3215> | (8) <https://www.skyquest.com/report/liver-cancer-drugs-market>
(9) <https://www.researchandmarkets.com/reports/5899559/liver-cancer-drug-market-size-share-and-trends> | (10) <https://www.polarismarketresearch.com/industry-analysis/global-liver-cancer-market>

CT-01 is highly differentiated among GSPTI degraders

Characteristics of CT-01 may provide disease-specific efficacy and high safety

Feature	CT-01 (Captor)	Antibody-conjugated degraders	Systemic degraders
Targeting	Degradation of GSPTI and NEK7	Degradation of GSPTI	Degradation of GSPTI
Pharmacology	Cell cytotoxicity, TME* attenuation, inflammation reduction	Cell cytotoxicity	Cell cytotoxicity
Mechanism	Tissue-activated prodrug	Antibody delivery mechanism	Systemically distributed
“In class” status	First- and the-only-in-class	First/Best	First/Best
Disease selection	Activating enzyme-directed: HCC, lung cancer, rare cancers	Antigen-directed, e.g. liquid tumors	None, basket trial screening
Route of administration	Oral	Injectable	Oral
Potential weaknesses	Limited to selected diseases due to enzymatic activation	Tissue distribution, subpar efficacy, limited to selected antigens	Systemic toxicities reported (hypocalcemia, hypotension, thrombocytopenia)

TME* - Tumor MicroEnvironment

Competitive landscape of GSPT1 degraders is highly attractive for CT-01

The-only-in-class status of CT-01 offers strong partnering & commercialization position

Tissue-activated degrader; dual selectivity



Systemic degrader

multiple other



multiple other



Antibody-conjugated degraders (DAC)



Class

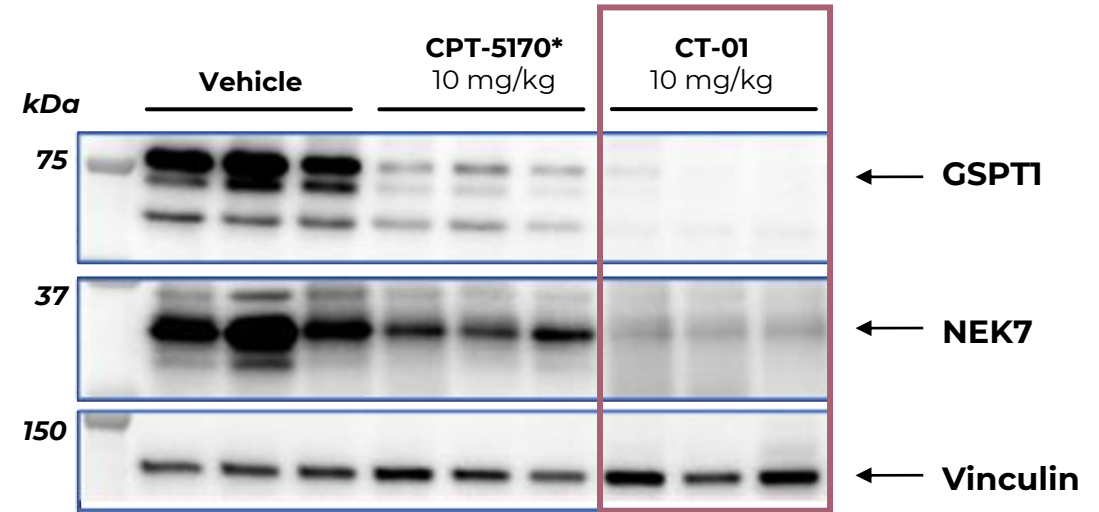
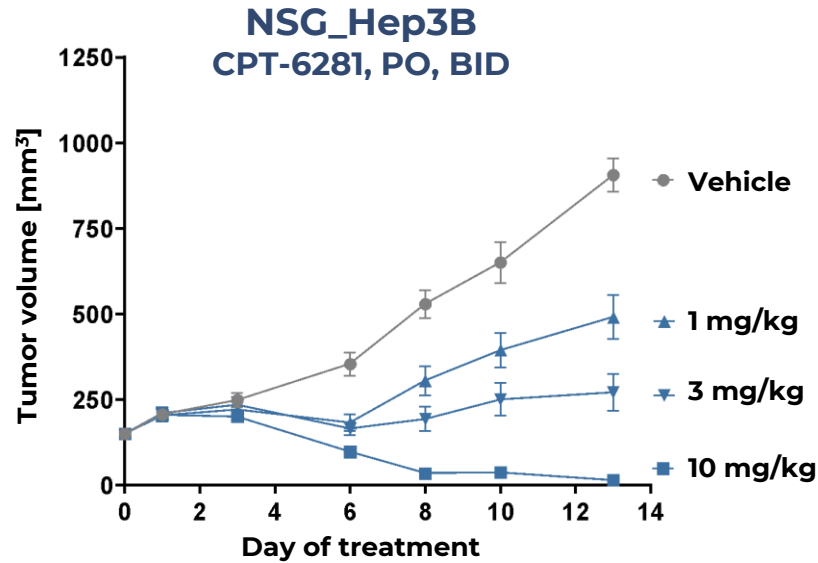
Discovery

Preclinical

Phase I/II

CT-01 potently regresses fast growing HCC tumors in mice

CT-01 efficiently degrades GSPT1 and NEK7



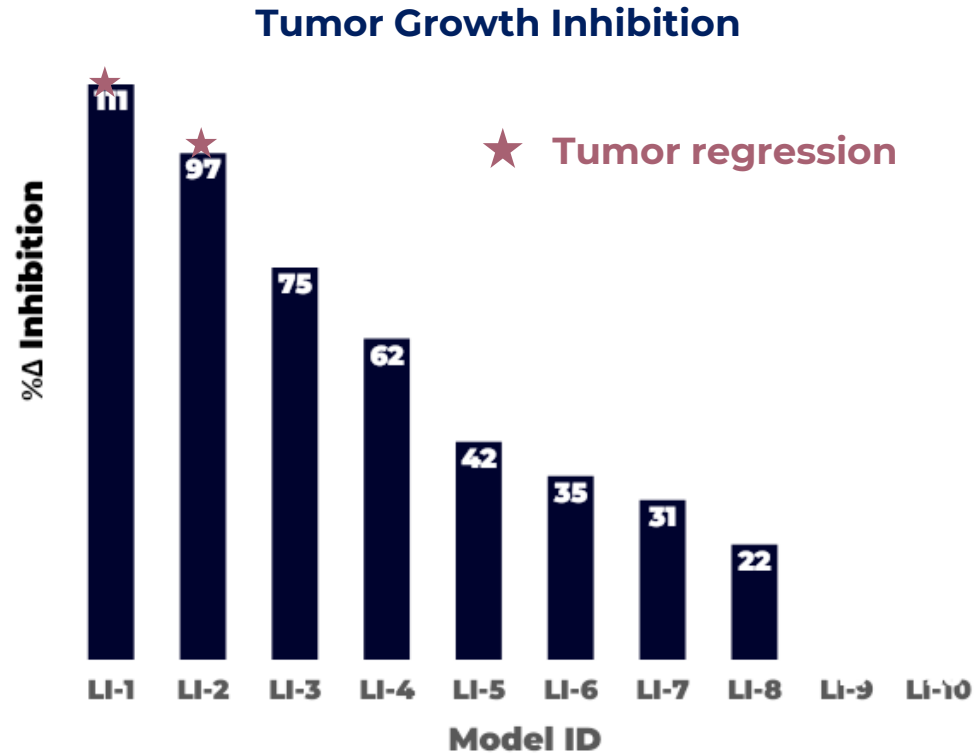
Regression of large tumors (~150 mm²) observed at doses as little as 10 mg/kg BID administered orally

*CPT-5170: an early lead compound in the CT-01 project

CT-01 regresses liver cancer in HCC models including aggressive Hep3B model

Convincing tumor growth inhibition in HCC PDX models

Results from Patient Derived Xenografts (PDXs) strongly translate to humans



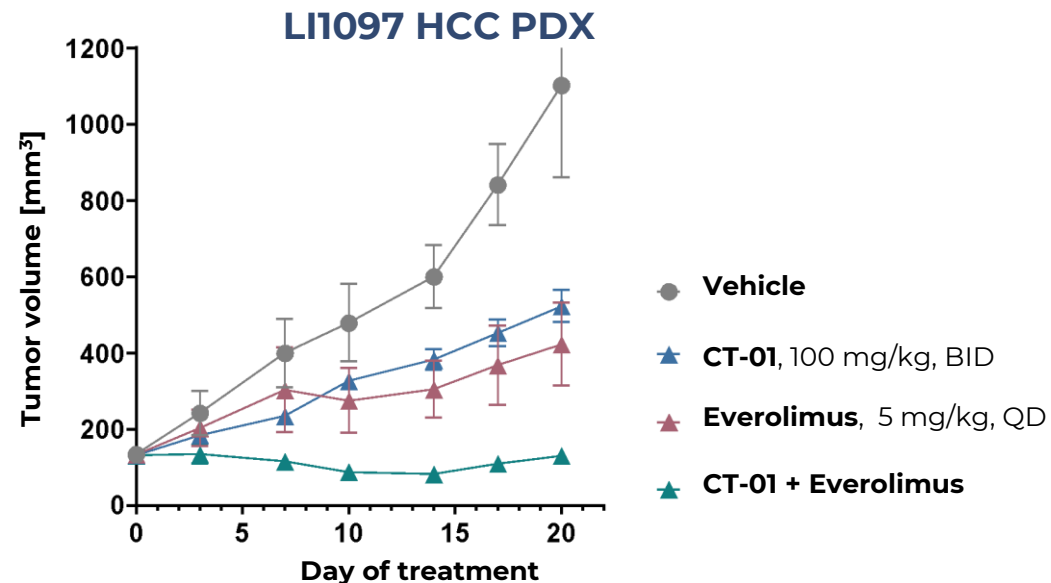
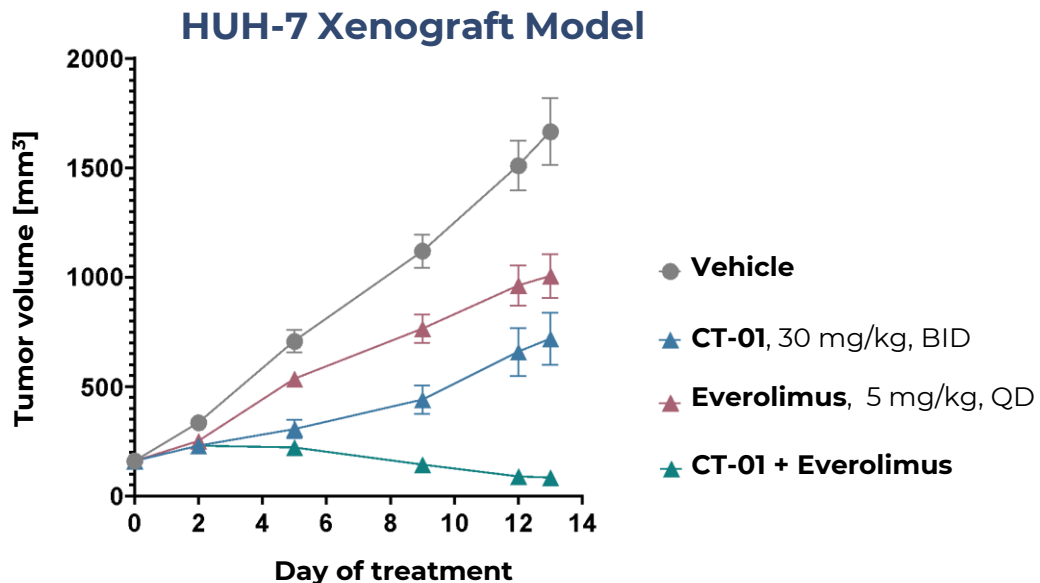
10 randomly selected HCC patient samples to ensure patient-like responses
Tumor growth inhibitions (TGI) of 40% or more observed in 5 out of 10 cases
2 models show complete response

100mg/kg, BID, p.o., N=3

8 out of 10 PDXs respond to treatment while in 5 out of 10 tumors are inhibited by >40%

Strong synergy of CT-01 in combination with everolimus (eve)

Everolimus and CT-01 display strong synergy *via* molecular pathway cross-talk

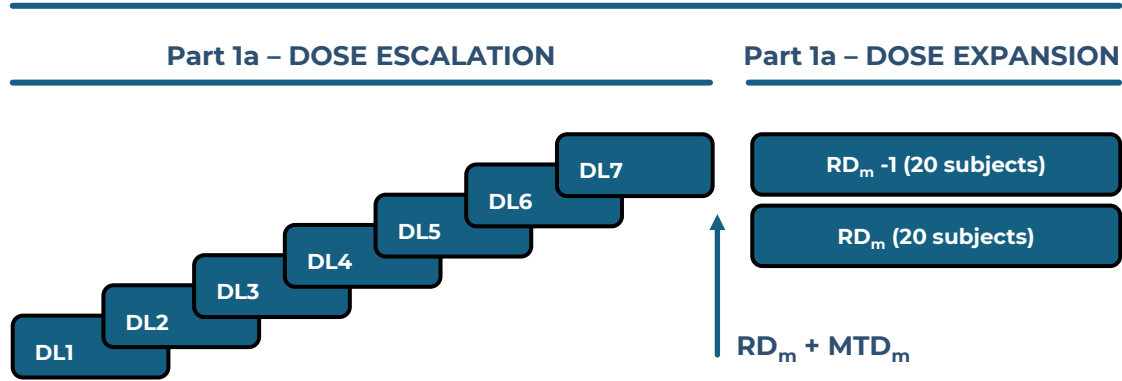


Everolimus is an approved drug in oncology

Combination with eve sensitizes non-responders and poor responders to CT-01

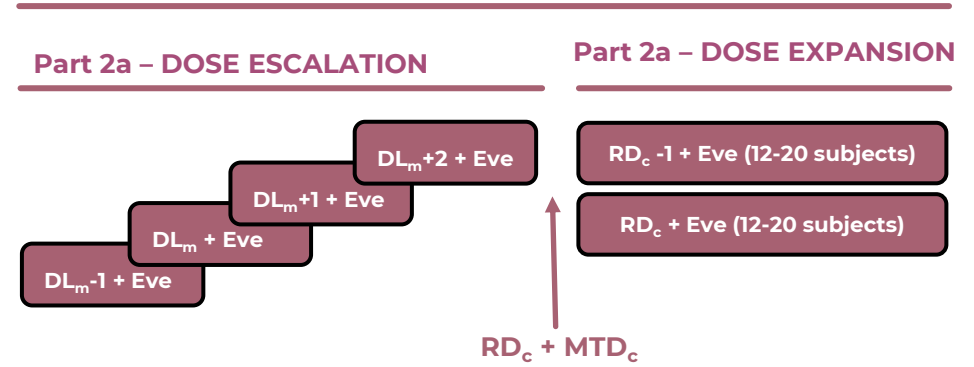
Phase I in HCC patients - study design

PART 1 – MONOTHERAPY – CT-01



Parallel recruitment may start from Part 1a DL3 and Part 2a DL1 if 2 DLs of Part 1a are considered safe by SMC

PART 2 – COMBOTHERAPY – CT-01



PART 2 – COMBOTHERAPY CT-01 + EVEROLIMUS

The-only-in-class potential of highly differentiated CT-01

CT-01 opportunity due to high disease burden, fast growing market & unique activity

Strong differentiation from other GSPTI degraders (BMS, Monte Rosa)

Best-in-class degradation profile

Active degrader lingers inside cancer cells after activation (poor cell penetration after prodrug conversion)

Active degrader is rapidly cleared from systemic circulation

Degradation profile

GSPTI, NEK7

Activated in diseased liver, lung, adipocytes and inflamed blood-brain barrier

Initial indications

hepatocellular carcinoma (HCC)

lung cancer

brain tumors

rare cancers (hepatoblastoma, lipo- and angiosarcoma)

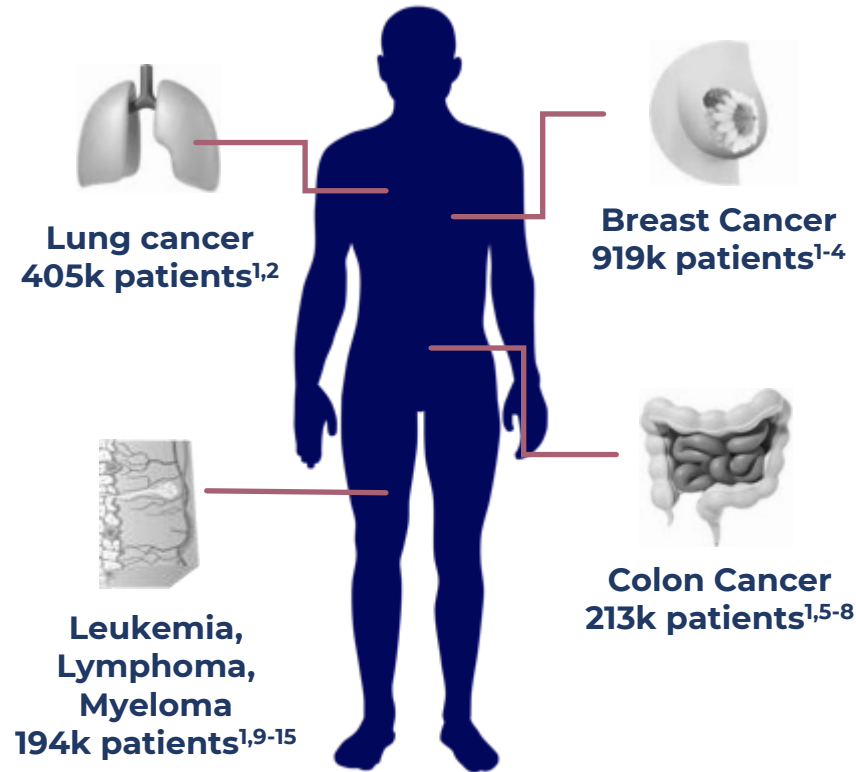
Status

Dosing patients in phase Ia

CT-03: First-in-Class MCL-1 Degraders for Liquid & Solid Tumors

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

MCL-1 constitutes a gate to cancer immortality



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

A critical resistance mechanism in hematological and solid tumors‡, cancer cells require very high levels to avoid induction of apoptosis

Degradation or inhibition of MCL-1 protein directly attenuates tumors in vivo as monotherapy & sensitizes tumors for other therapies

Inhibitors require prolonged, almost 100% of target coverage and cause accumulation of MCL-1†, cardiotoxicity through necrosis§

Short-term degradation of ≈70% of MCL-1 irreversibly induces apoptosis in cancer cells

This, together, with optimized clearance expands the therapeutic window of degraders

1. <https://gco.iarc.fr/today/en/>
2. Semin Cancer Biol. 2006 16(4):253-64
3. Cell Death Dis 2018 9(2): 19
4. Breast Cancer Res. 2016 18(1): 125
5. Int J Mol Sci. 2019 20(3): 5999

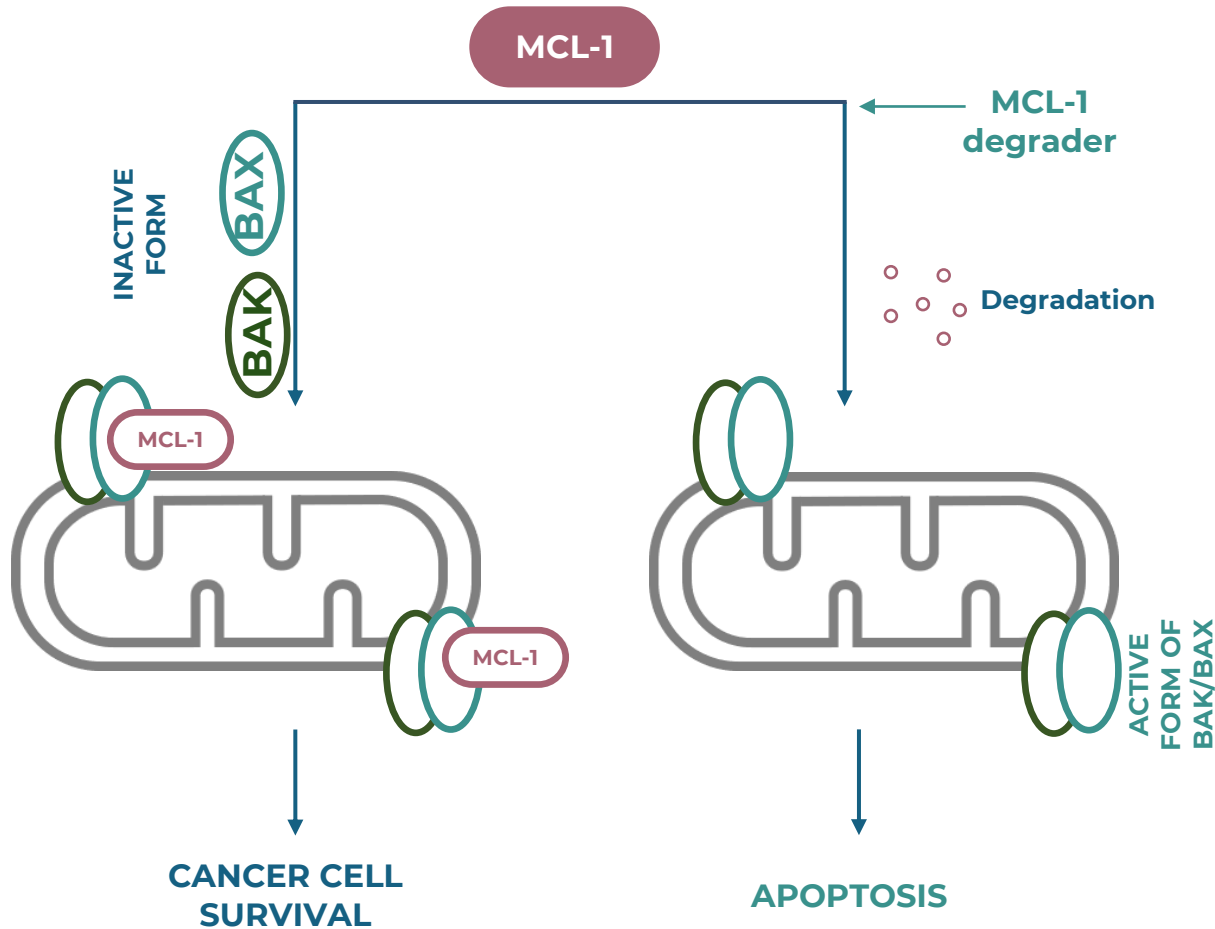
6. Cell Death Dis. 2022 13(1): 63
7. Colorectal Dis 2022 24(11): 1295-1307
8. Ann Fam Med. 2016 14(3): 215-20
9. Exp Hematol Oncol. 2020 Jun 19;9:14
10. Hum Pathol. 2004 Sep;35(9):1095-100

11. ACS Key Statistics for AML, CLL, Lymphoma
12. Curr Treat Options Oncol. 2020 Jun 29;21(8):66
13. Int J Mol Sci. 2024 Jan 27;25(3):1589
14. Blood Rev. 2020 Nov;44:100672
15. Leukemia. 2013 Jun;27(6):1381-90

†Front Oncol. 2023 Jul 31;13:1226289
‡Apoptosis. 2023 Feb;28(1-2):20-38
§Science. 2020 April; 23(4): 101015

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

MCL-1 is over-produced in up to 30% of cancers with acquired resistance



MCL-1 biology and clinical relevance

MCL-1 is a well-characterized oncogenic protein with a key role in **evading apoptosis** and **promoting the survival of cancer cells**¹.

Studies show cell growth dependency of MCL-1 levels in liquid (leukemia, lymphoma, myeloma) and solid tumors (breast and lung cancers)².

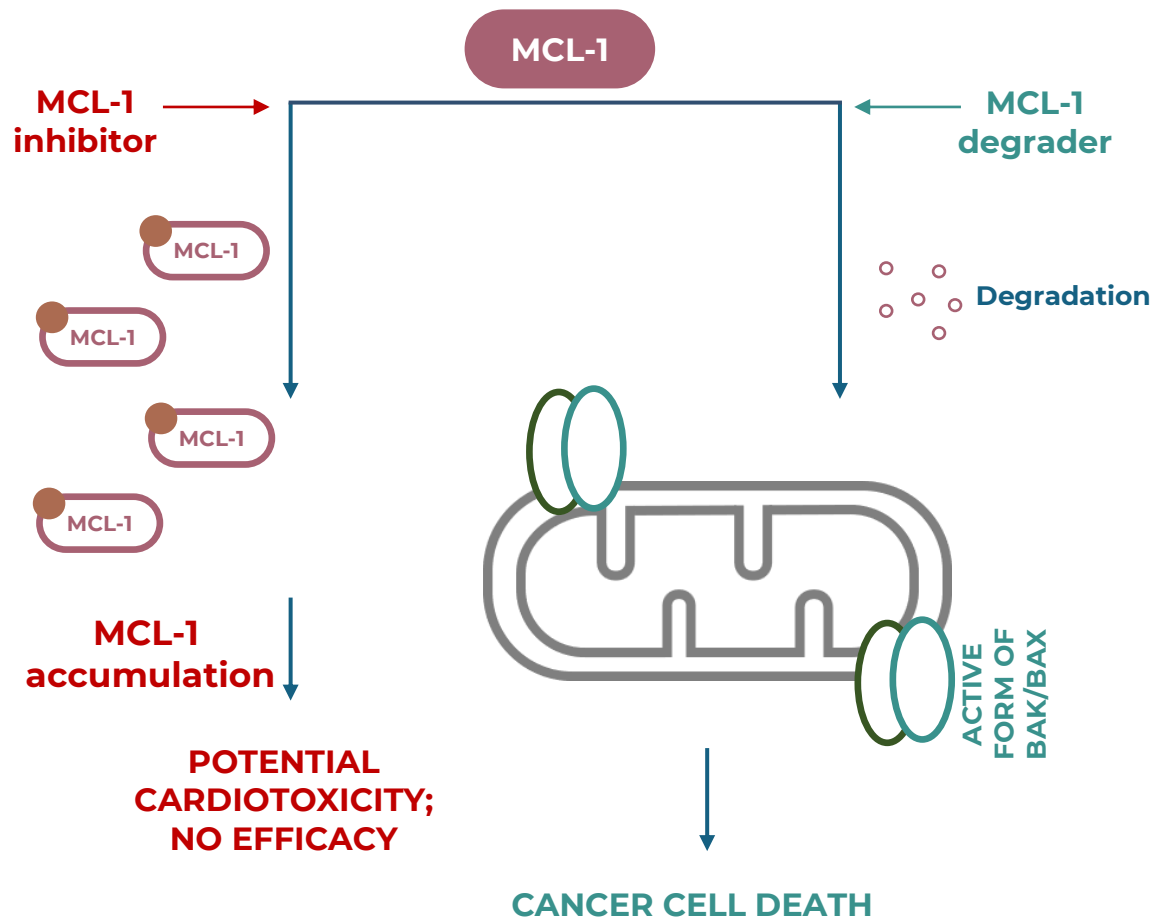
Monoallelic KO of MCL-1 in mice is viable and do not show signs of cardiac damage³ or gross phenotype, and show resistance to selected liquid tumors.

Numerous systemic and targeted therapies drive the clonal selection of cells towards increased levels of MCL-1, like in AML refractory to venetoclax⁴.

1. Singh R et al. Nature Reviews, 2019; 20: 175-193
2. Kotschy A et al. Nature, 2016; 538(7626): 477-482
3. Brinkmann K et al. Cell Death Differ, 2017; 24(12): 2032-2043
4. Garcia S et al. Cancers, 2024; 16(6): 1091

Targeted Protein Degradation is the only modality to break MCL-1

Classical drugs induce dramatic MCL-1 accumulation in healthy and cancer cells



MCL-1 degraders advantage over inhibitors

MCL-1 inhibitors induce strong accumulation of this protein in cells¹.

As a result this 5-12 fold MCL-1 accumulation adverse events occur².

Because of the adverse events, i.e. cardiotoxicity MCL-1 inhibitors have not progressed in development

Degrader drugs rapidly remove MCL-1 and instantly induce cancer cell death.

They also allow for homeostatic levels of MCL-1 in cardiac myocytes and other healthy tissues, which provides a healthy therapy.

Over \$100billion market potential of MCL-1 targeting agents

Classical drugs failed to address needs of this market due to their poor safety

Haematological malignancies

Multiple Myeloma

\$53B by 2030¹

Acute Myeloid Leukaemia

\$6B by 2028²

Non-Hodgkin Lymphoma

\$16B by 2032³

Selected solid tumors

Small cell lung cancer (SCLC)

\$6.5B by 2031⁴

Non-small cell lung cancer

\$36.9B by 2031⁵

Triple-negative breast cancer

\$1.5B by 2030⁶

Thanks to a different mode of action, MCL-1 degraders show great safety profile in relevant species

¹Allied Market Research

²BCC Research

³Spherical Insights

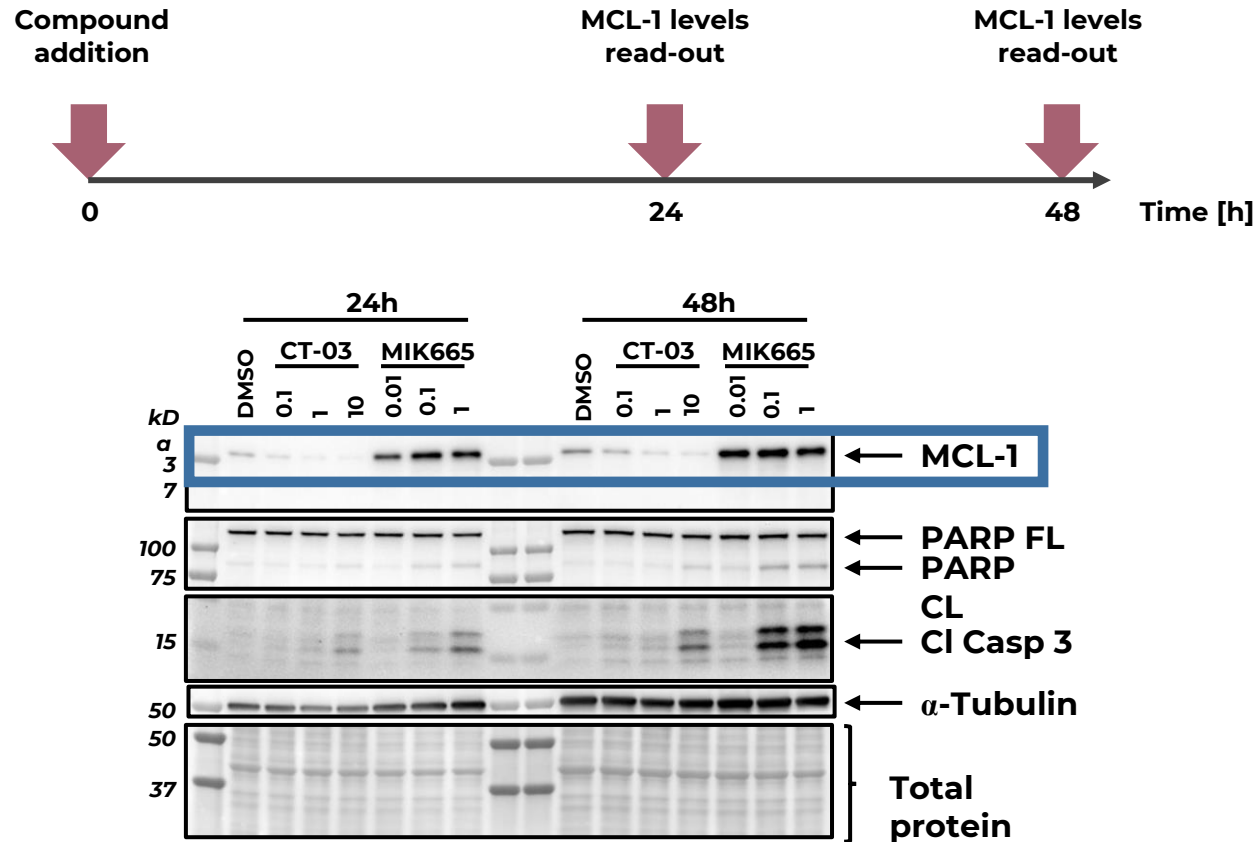
⁴iHealthcareAnalyst

⁵Allied Market Research

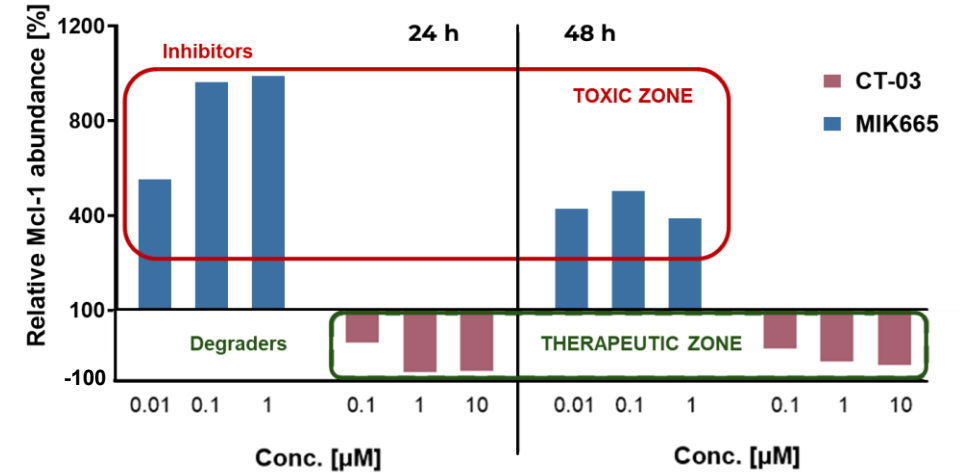
⁶Databridge Market Research

MCL-1 degraders remain safe to healthy tissues

Inhibitors induce MCL-1 accumulation toxic to healthy tissues



MCL-1 degradation with CT-03 and accumulation with MIK665 in hiPSC-Cardiomyocytes after 24 and 48h incubation



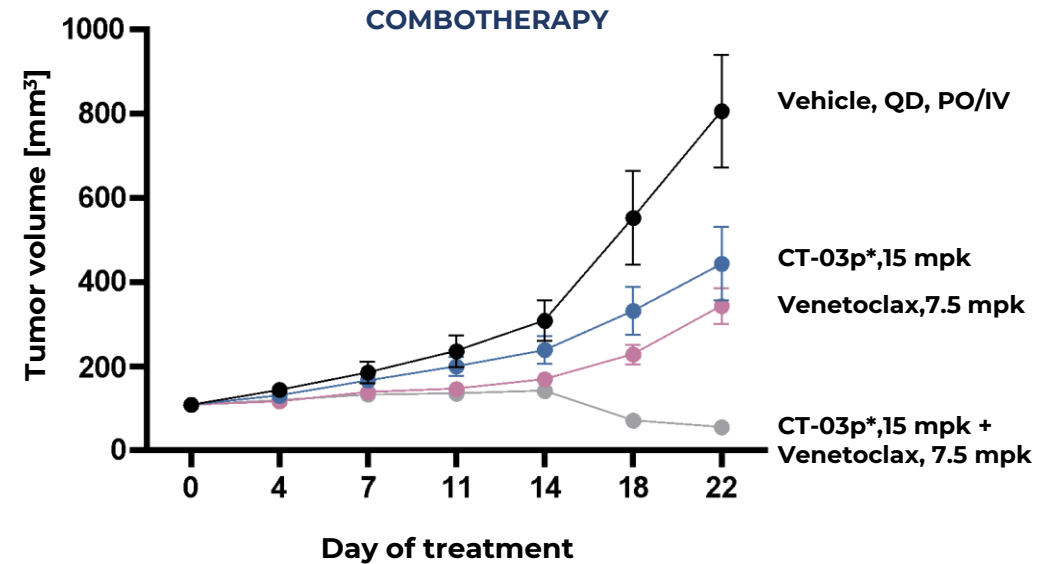
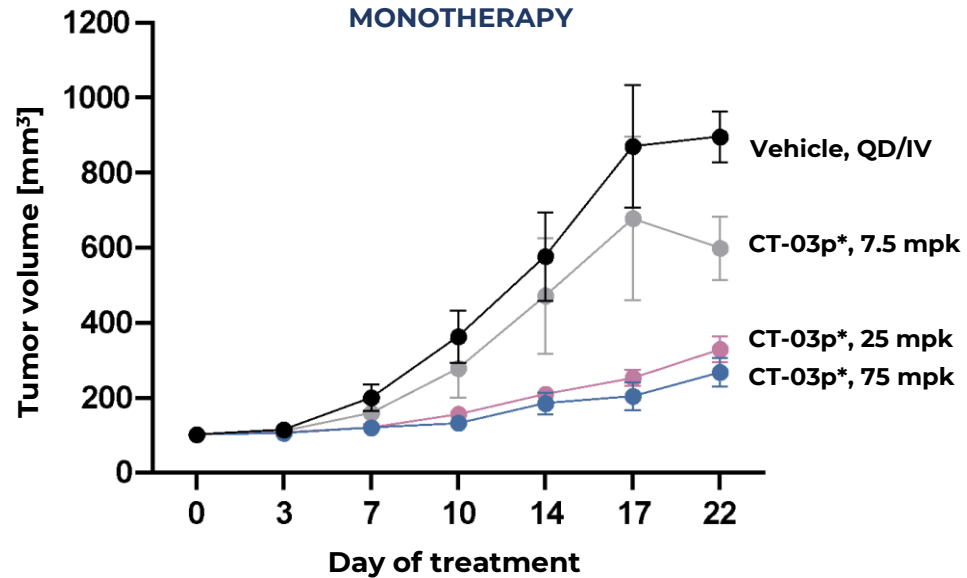
*Scale at the Y axis is not proportional

Inhibitors induce MCL-1 accumulation lasting even after compound wash-out

Degraders transiently remove MCL-1 in healthy cells

High potency of MCL-1 degraders in an *in vivo* AML model

MCL-1 degraders demonstrate strong activity in mono- and combination therapies



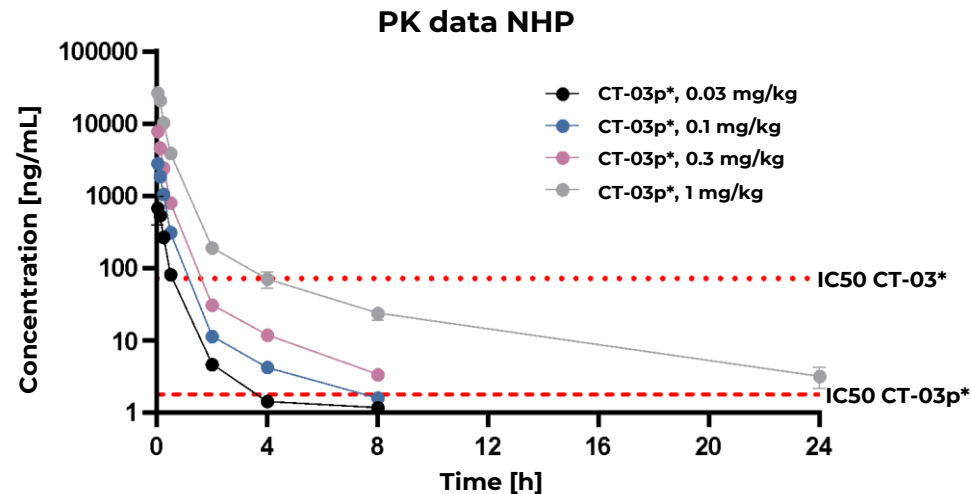
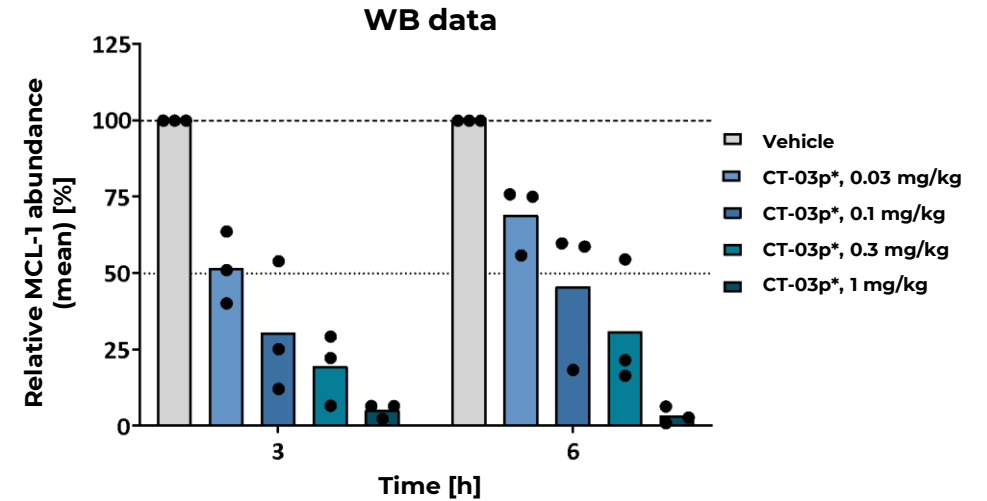
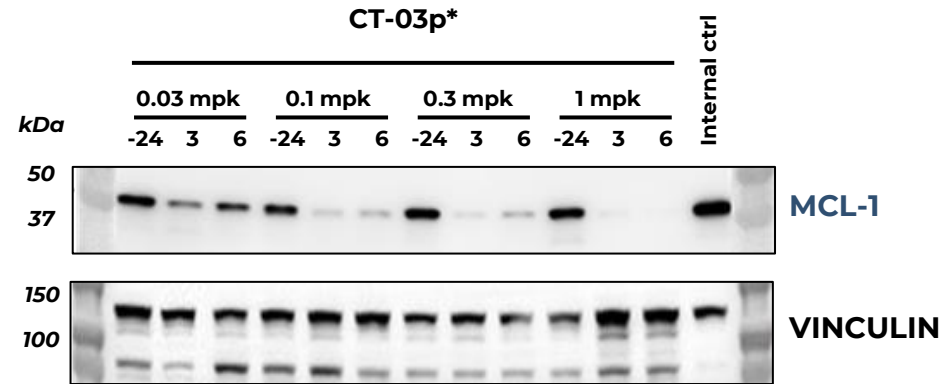
CT-03p* - active isomer of CT-03p

CT-03p* was administered 2 days ON and 5 days OFF for 4 weeks intravenously and venetoclax was administered daily (QD) orally for 4 weeks. N=9-10/group

CT-03p* (active isomer of CT-03p) suppresses the growth of MV-4-11 human leukemia xenograft model in NOD/SCID mice as a monotherapy and, even more potently, in combination with Venetoclax

Degradation of MCL-1 in NHP after single IV dose of degraders

Complete degradation of MCL-1 is safe to non-human primates



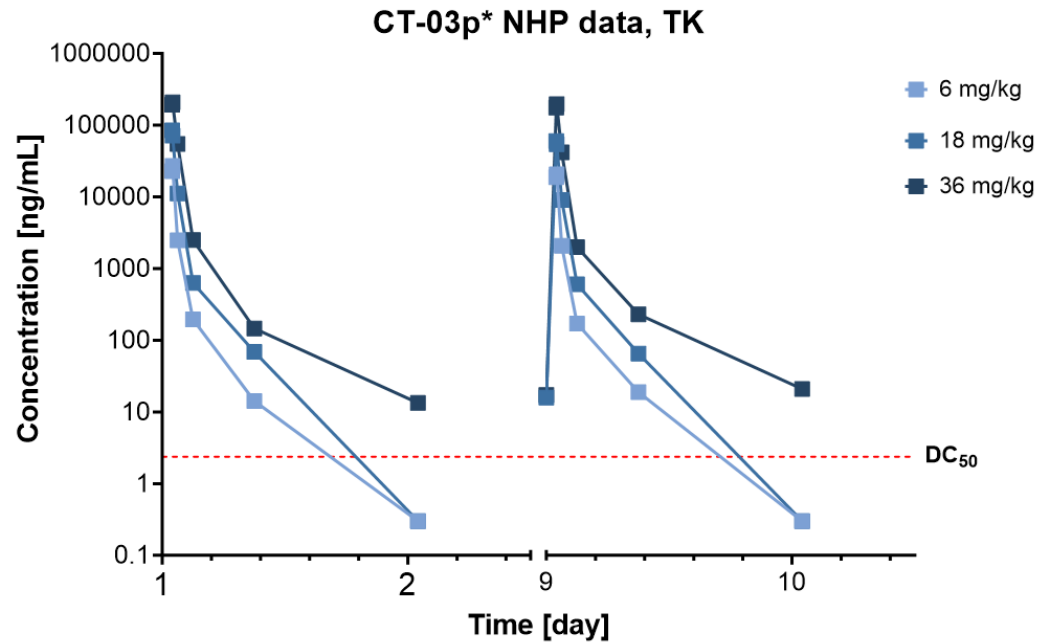
Rapid and dose-dependent degradation of MCL-1 observed in NHP after single IV administration

Clear correlation between plasma exposure (PK) and extent of protein degradation

Supports translation of MCL-1 degradation to higher species

CT-03p shows great toxicokinetic profile

TK and PK profiles demonstrate lasting and complete target coverage



At all doses tested, CT-03p* shows plasma concentrations for 20 hours or longer at levels exceeding the estimated effective dose (DC50).

Even the highest dose, 36 mg/kg, (70-120x above pHED) shows cardiosafety

MCL-1 degraders show high efficacy and broad therapeutic window in relevant species

CT-03 candidate drug with unmatched therapeutic window

First-in-class MCL-1 degrader is poised to unlock the cancer resistance market

Strong differentiation from MCL-1 inhibitors

Pharmacology of MCL-1 degradation vs. pharmacology of accumulation (inhibitors)

No accumulation of MCL-1 protein

No cardiotoxicity observations in MTD, DRF in NHPs by any means

Very high degradation potency in mouse models, in NHP and in human cells *ex vivo*

Candidate drug in place

Initial indications

Hematological cancers

Solid tumors

Status

IND-enabling studies

Differentiation of MCL-1 degrader vs MCL-1 inhibitors

Clinical development of MCL-1 inhibitors limited by cardiotoxicity issues

- Literature & experimental MOA now demonstrated for inhibitor induced toxicity
- MCL-1 inhibitors cause MCL-1 accumulation in healthy tissue and necrosis

MCL-1 degraders - no cardiotoxicity is observed in all pre-clinical *in vitro* & *in vivo* studies

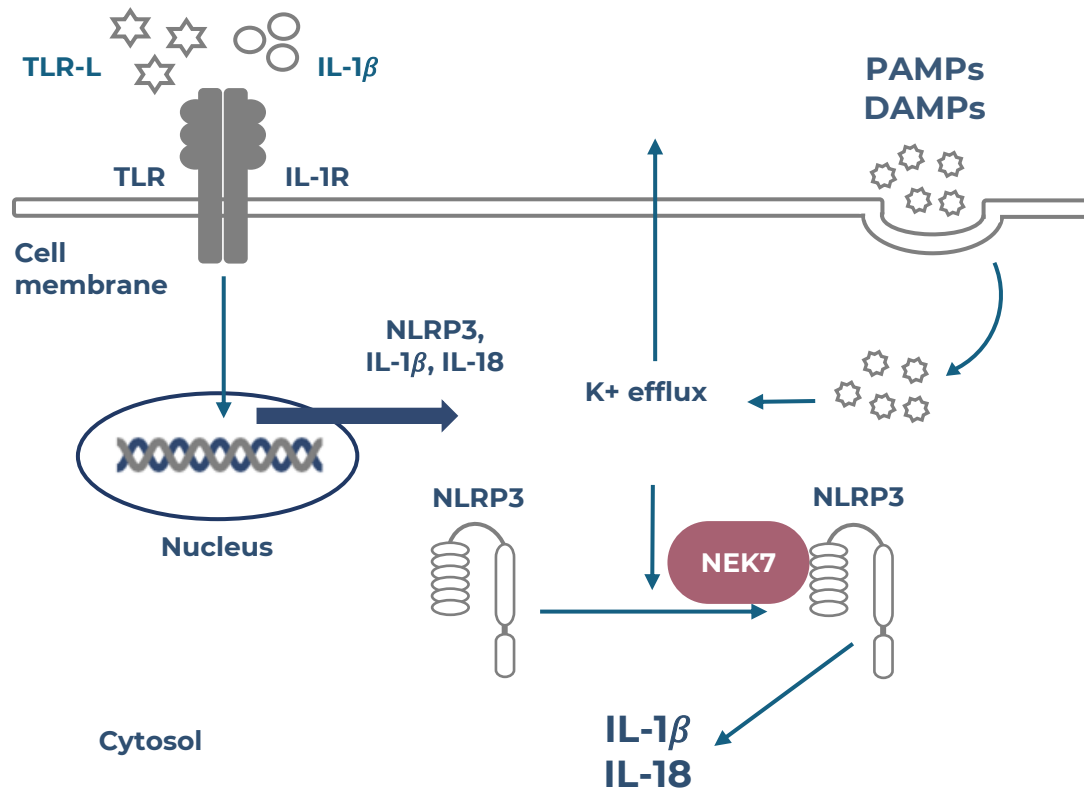
- MCL-1 degradation has no effect on cardiomyocytes *in vitro*
- No cardio safety issues observed at 120x higher doses in monkey than required for DC50
 - Cardiac Troponin-I levels in monkey are not affected by MCL-1 degradation
- Safety MTD/DRF study in monkey showed no cardiotoxicity
- IND-enabling studies ongoing – GLP tox report 4Q2025

Summary: first-in-class MCL-1 degrader

- **MCL-1 a well validated oncology target**
- **Differentiation of degrader vs inhibitor modalities**
 - MCL-1 inhibitors show cardiotoxicity in the clinic
 - Strong literature & experimental MOA now demonstrated for inhibitor induced toxicity
 - Protein degradation modality provides greater target coverage and is well tolerated
 - No cardio safety issues observed at 120x higher doses in monkey than required for DC50
 - MCL-1 degraders are synergistic with venetoclax using *in vitro* & *in vivo* AML models
- **First-in-class opportunity: clinical candidate ABX-629**
 - Phase 1 study in high-risk MDS & relapsed/refractory AML planned
 - Active as monotherapy and shows synergy in combo with venetoclax *in vivo*
 - Safety MTD/DRF study in monkey showed no cardiotoxicity
 - IND-enabling studies ongoing – GLP tox report 4Q2025

CT-02: First-in-Class NEK7 Degraders for Autoimmune (CT-02S), Neuroinflammation (CT-02B)

NEK7 as a new target of the NLRP3 inflammasome pathway



1. Shi et al; Nature Immunology, vol 17 (2016);
2. Sharif et al.; Nature, vol 570, (2019);
3. He et al.; Nature, vol 530, (2016);
4. Walle and Lamkanafi; Nature Reviews Drug Discovery vol 23
*own results conducted by Captor Therapeutics

NEK7 overview

NEK7 is master regulator of the NLRP3 inflammasome complex through its scaffolding function

NEK7 KO/KD in mouse abrogates production of IL-1 beta in response to stimulating factors.

Haploinsufficient, NEK7^{+/-} mice show no internal anatomical or growth abnormalities.

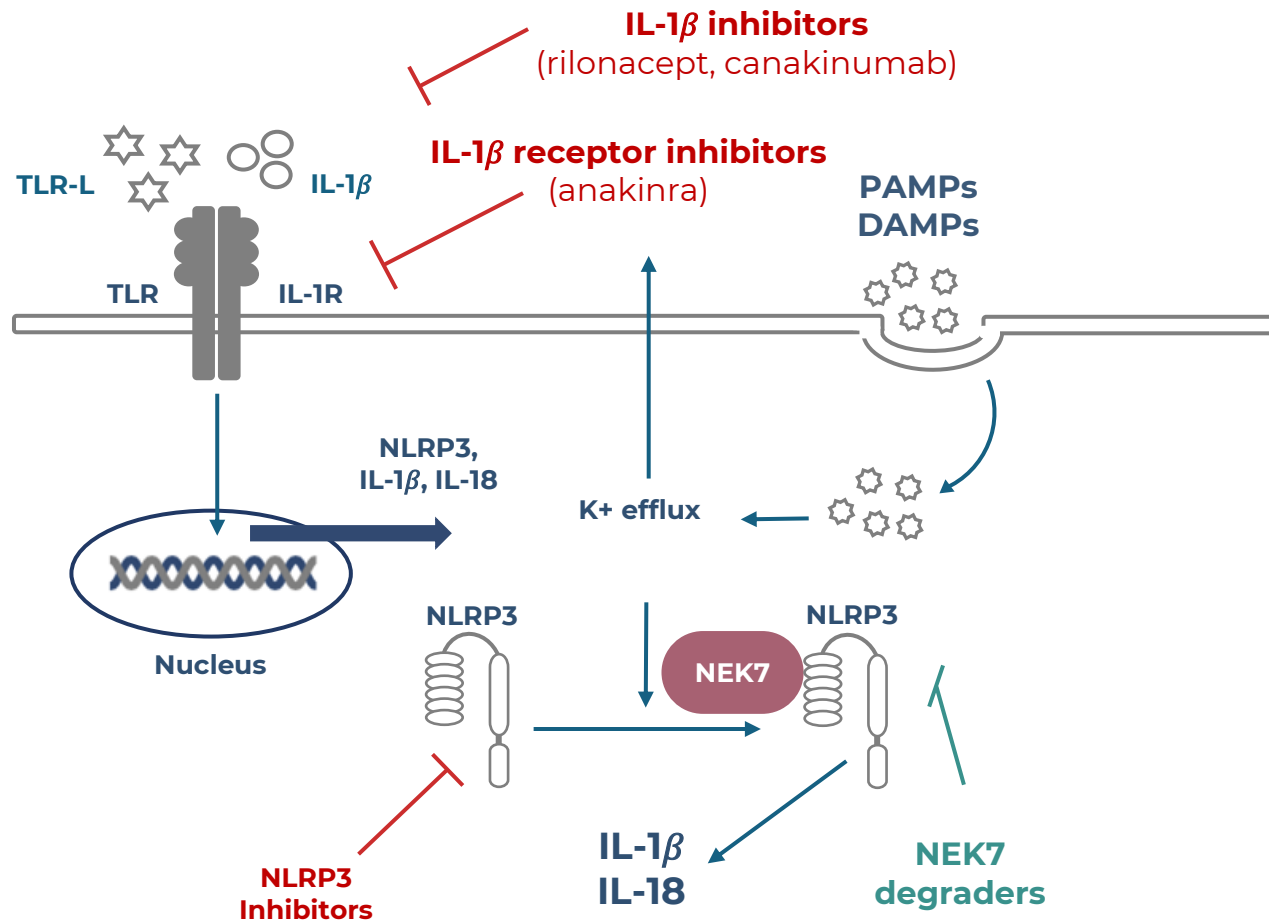
Antagonists of IL-1β or IL-1R are approved in:

CAPS syndromes (FCAS, MWS, NOMID)

Inflammatory disorders, e.g. familial mediterranean fever (FMF), tumor necrosis factor receptor associated periodic syndrome (TRAPS), hyperimmunoglobulin D syndrome (HIDS) / mevalonate kinase deficiency (MKD), Still's disease, and gouty arthritis.

Degradation of NEK7 inhibits the production of pro-inflammatory cytokines in *in vitro* models and halts disease progression in pre-clinical mouse models of chronic NLRP3-related diseases*.

NEK7 as a new target of the NLRP3 inflammasome pathway



Differentiation

From anti-IL-1 β antagonists:

Once daily oral administration instead of injection
Uncoupling pharmacokinetics from pharmacodynamics potentially offers a better safety profile

From NLRP3 inhibitors:

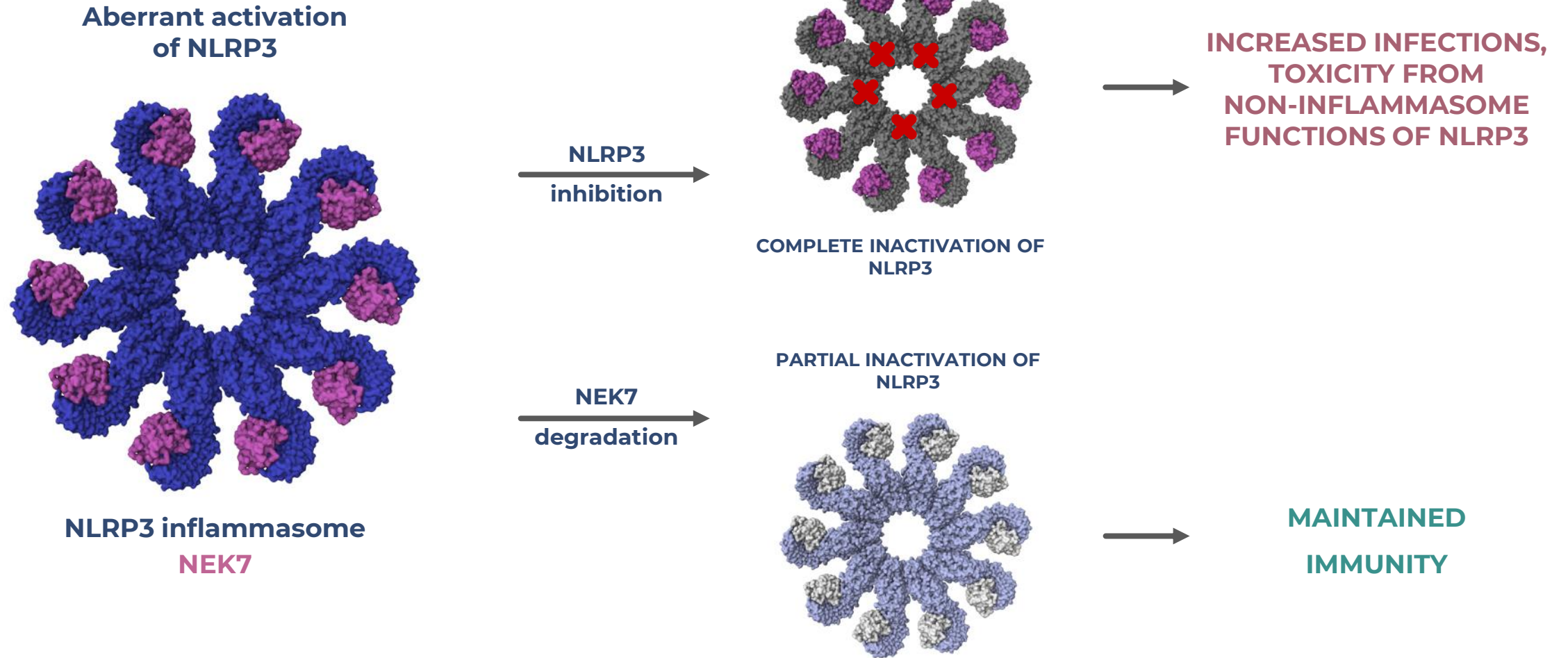
Uncoupling pharmacokinetics from pharmacodynamics potentially offers a better safety profile and prolonged efficacy
High safety profile: due to multiple functions of NLRP3 outside of the inflammasome, there are serious safety concerns about NLRP3 inhibitors

Complete IL-1 β shutdown potentially manifests in recurring infections

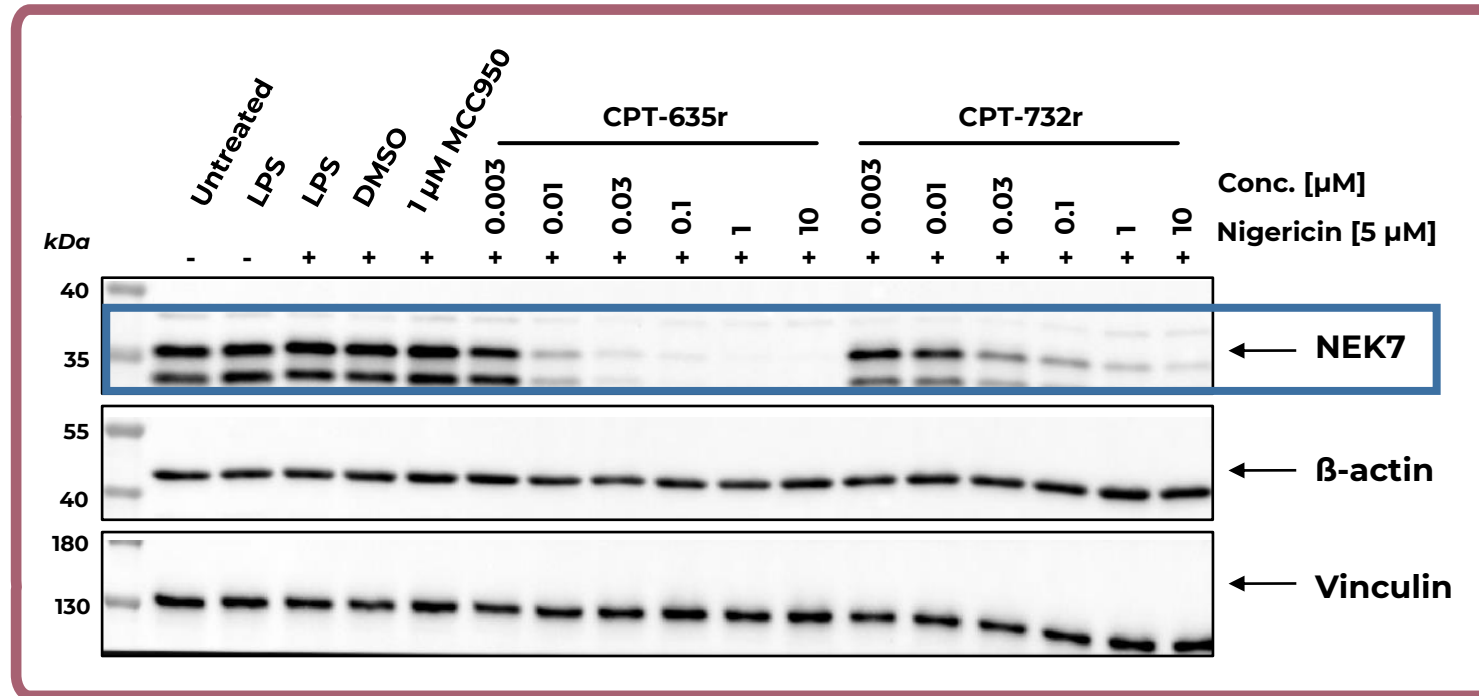
NLRP3 requires high coverage by inhibitors, which is recapitulated in increased frequency of dosing (BID) of some of the clinical compounds, e.g. DFV890 (Novartis)

NEK7 degradation as a differentiated intervention in NLRP3 pathway

Degradation of NEK7 provides therapeutic benefit and maintains basal immunity



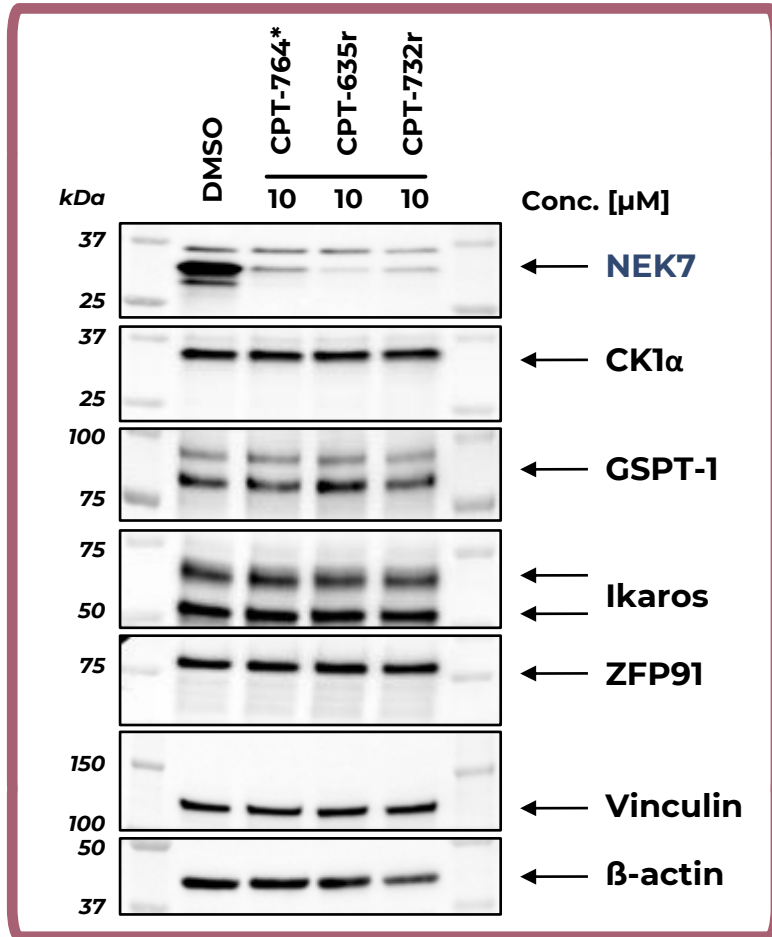
Potent degradation of NEK7 in human macrophages *in vitro*



CPT-635r (CT-02S) and CPT-732r (CT-02B) degrade NEK7 protein dose-dependently in human PBMC-derived macrophages with LPS+Nigericin activated inflammasome

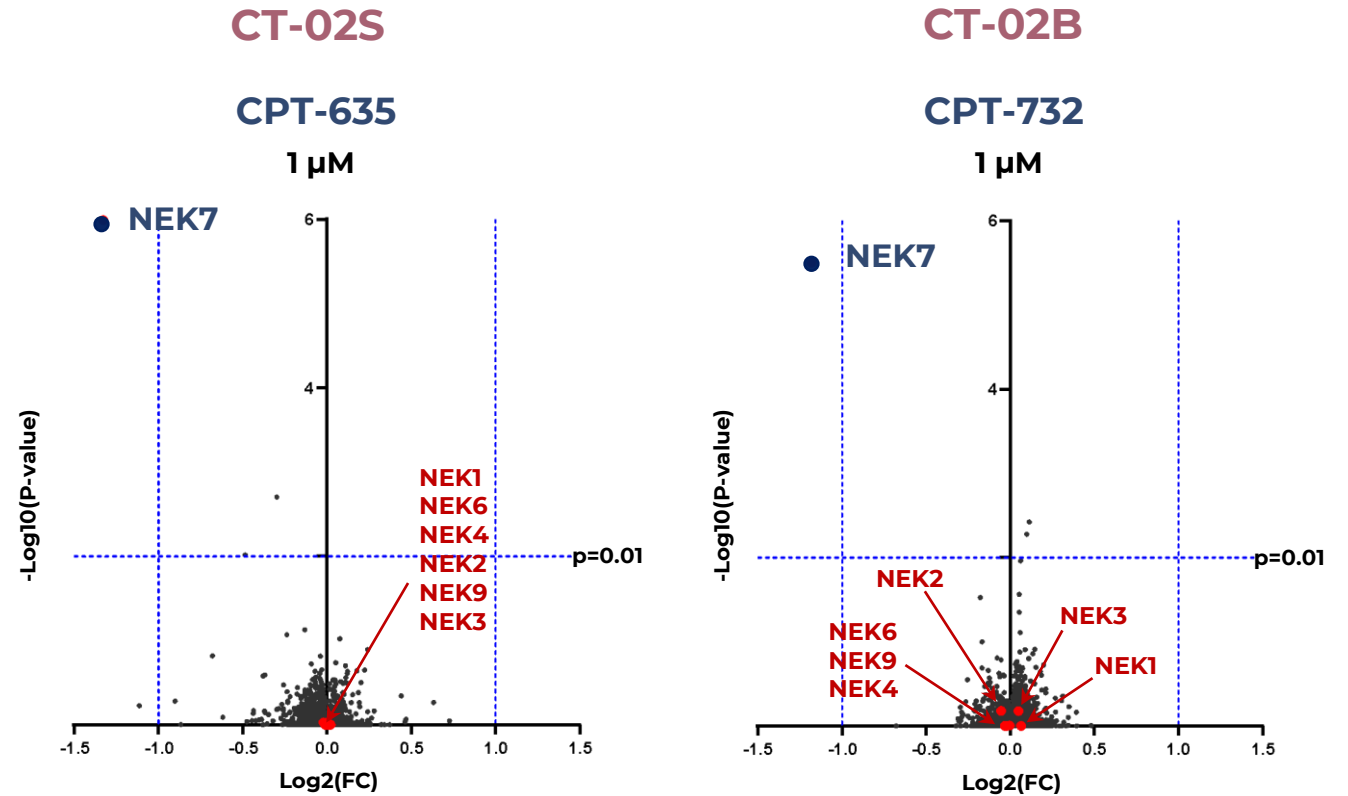
Human PBMC differentiated into macrophages with M-CSF; treatment with compounds – 24h; inflammasome activation: LPS – 3h, Nigericin – 1h
MCC950 – NLRP3 inhibitor (Roche/ Inflazome); CPT-635r – racemate of CPT-635, CPT-732r – racemate of CPT-732

High selectivity of NEK7 molecular glue degraders



*Early lead compound

CPT-635r – racemate of CPT-635, CPT-732r – racemate of CPT-732

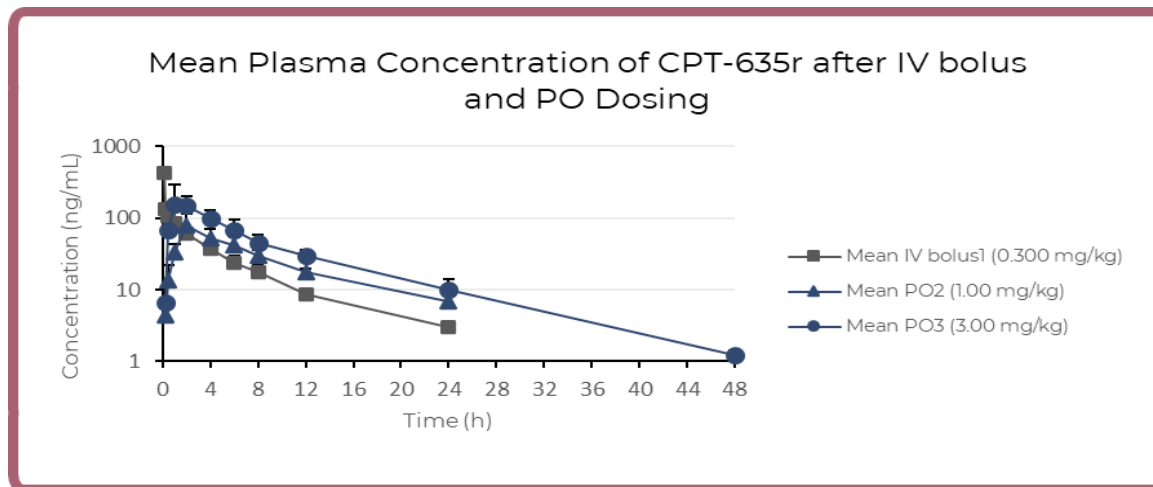
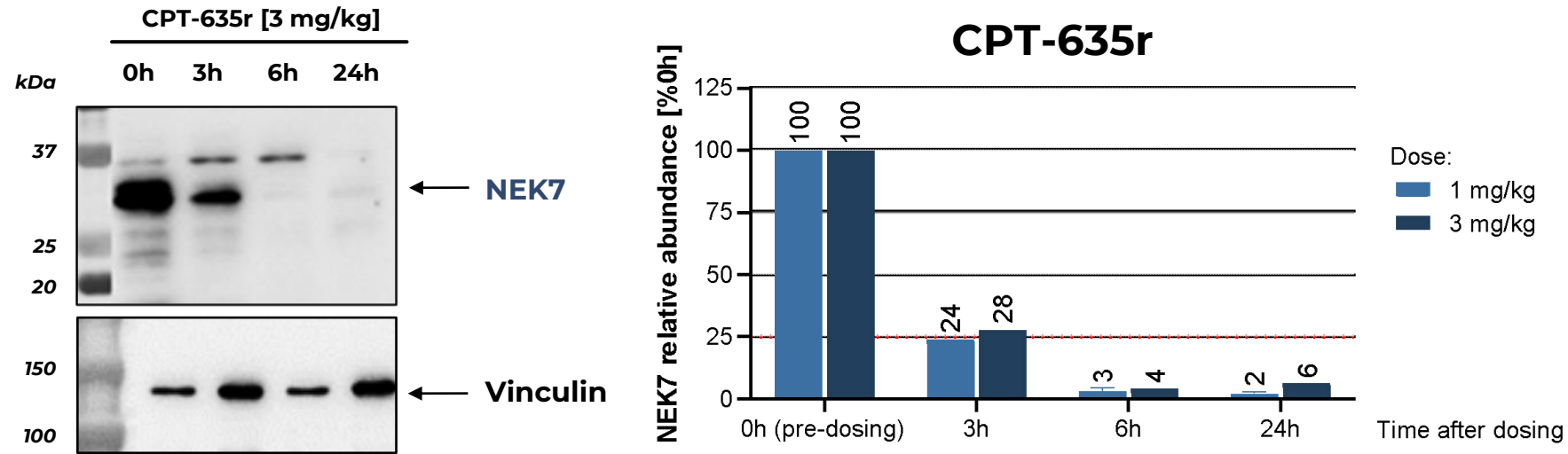


Confirmed degradation of NEK7 in hPBMCs
No off-targets, even at high concentrations

CT-02 (NEK7)

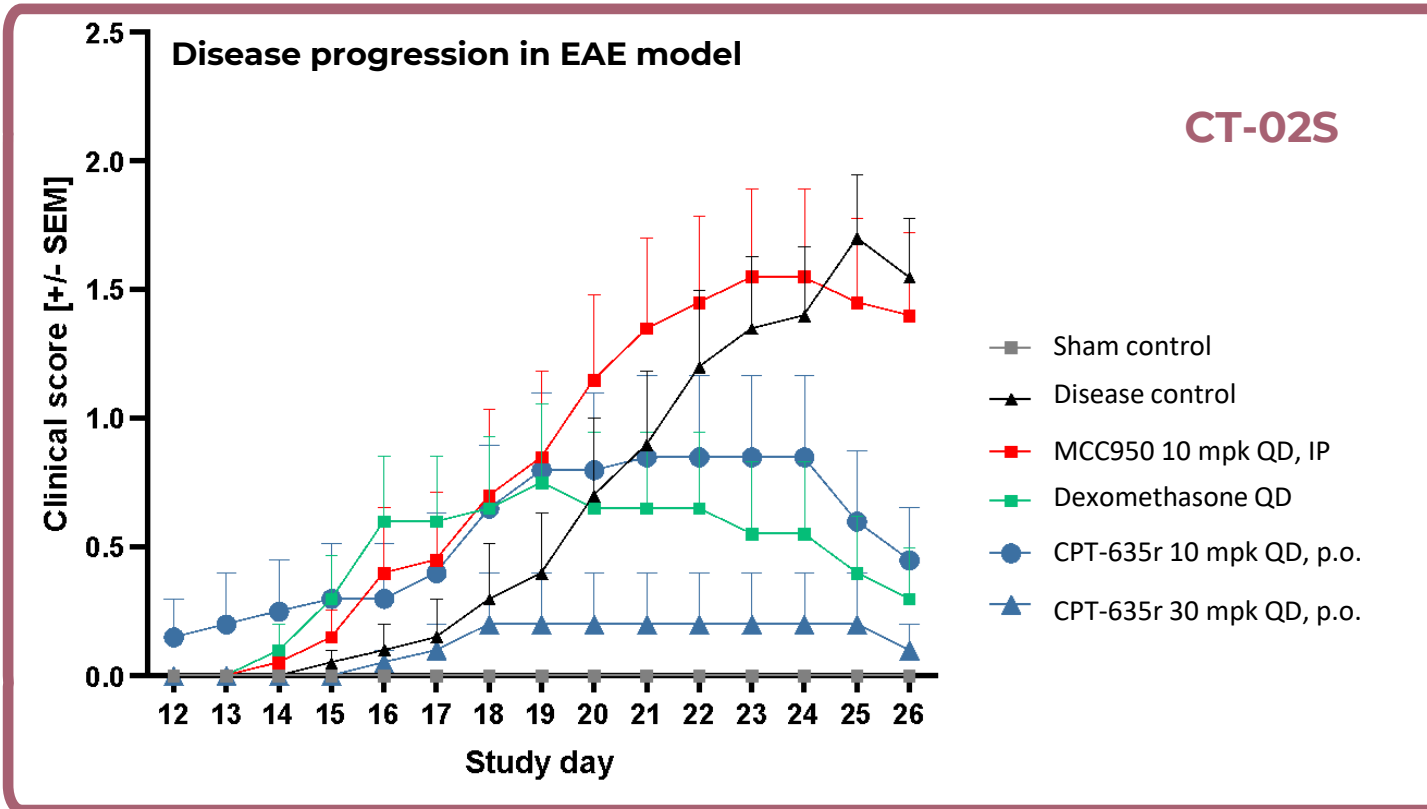
CPT-635r efficiently covers & degrades NEK7 in NHPs after a single dose

CT-02S



NEK7 degradation in monkey PBMCs *ex vivo* at 3, 6, and 24 h after PO administration of CPT-635r [1 & 3 mg/kg]

High efficacy of CPT-635r with oral dosing in EAE mouse model *in vivo*



Clinical Score	Clinical Sign
0	Normal mouse; no overt signs of disease
0.5	Tail weakness/partial tail weakness (50% of length)
1	Loss of tail tonicity (complete tail paralysis)/ Limp tail
1.5	Limp tail and weakness in one hind limb
2	Partial hind limb paralysis/ Limp tail and weakness in both hind limbs
2.5	Both hind limbs have some movement, but both are dragging at the feet

Dosing: QD (once a day)
 Route of administration:
 CPT-513, dexamethasone - PO
 MCC950 - IP

C57BL/6 female mice (10 mice per group)

Clinical score/ disease index monitored based on a predefined scale including motor skills and tail / limb weakness

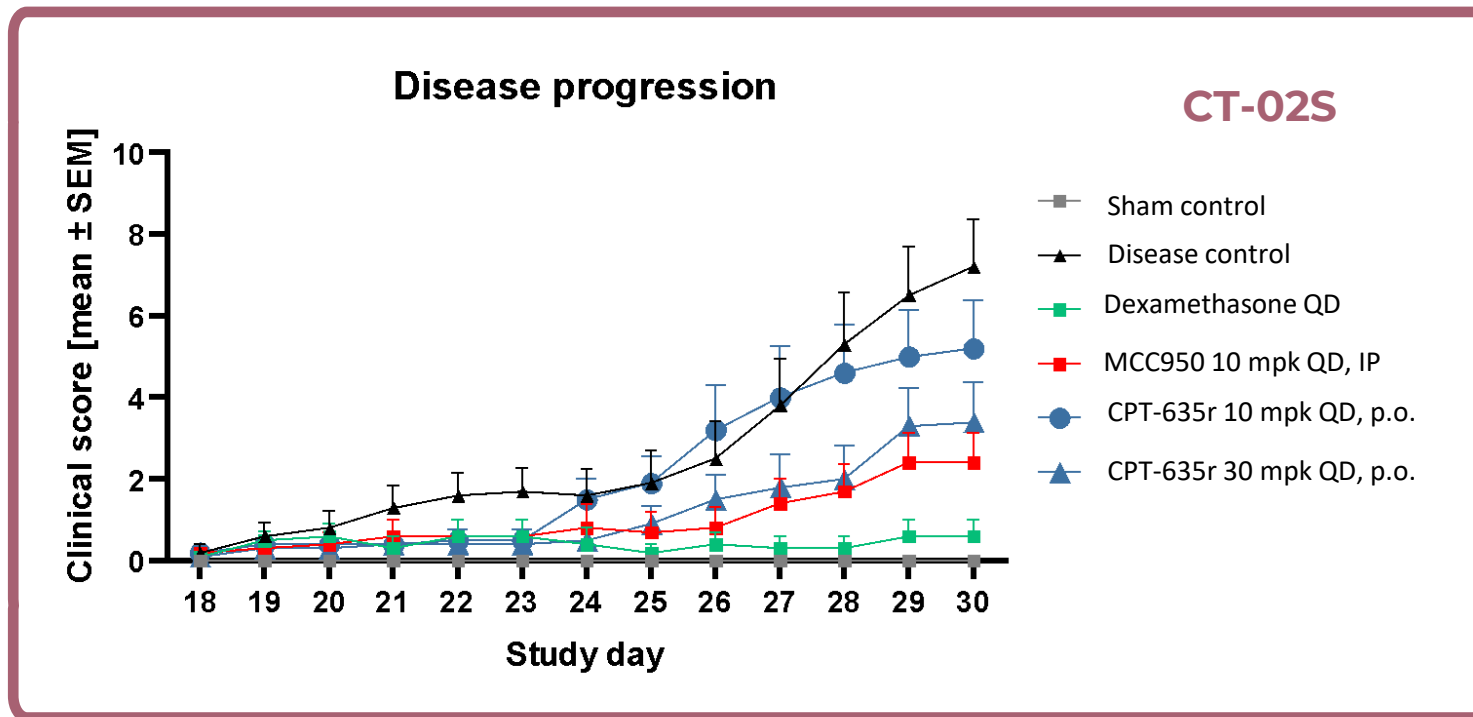
Treatment for 16 days did not induce any side effects

Body weight in the groups treated with NEK7 degraders was higher than in the group treated with Dexamethasone

Note: CPT-635r is approximately 40% less potent (DC50) in murine systems compared to human / primate

MOG₃₅₋₅₅ Induced Experimental Autoimmune Encephalomyelitis (EAE) In Mice

Therapeutic potential of NEK7 degraders in Collagen-Induced Arthritis model



Clinical Score	Clinical Sign
0	No redness or swelling
1	Slight swelling in ankle and or redness in one toe
2	Progressive swelling from ankle to midfoot and or involvement of more than two toes
3	Swelling and inflammation in entire foot
4	Swelling and inflammation in entire foot including toes

Dosing: QD (once a day)
 Route of administration:
 CPT-513, dexamethasone - PO (oral)
 MCC950 - IP (intraperitoneally)

Male DBA1/J Mice (10 mice per group)

Clinical score/ disease index was assessed by trained, blinded personnel for swelling of digits/paws and erythema

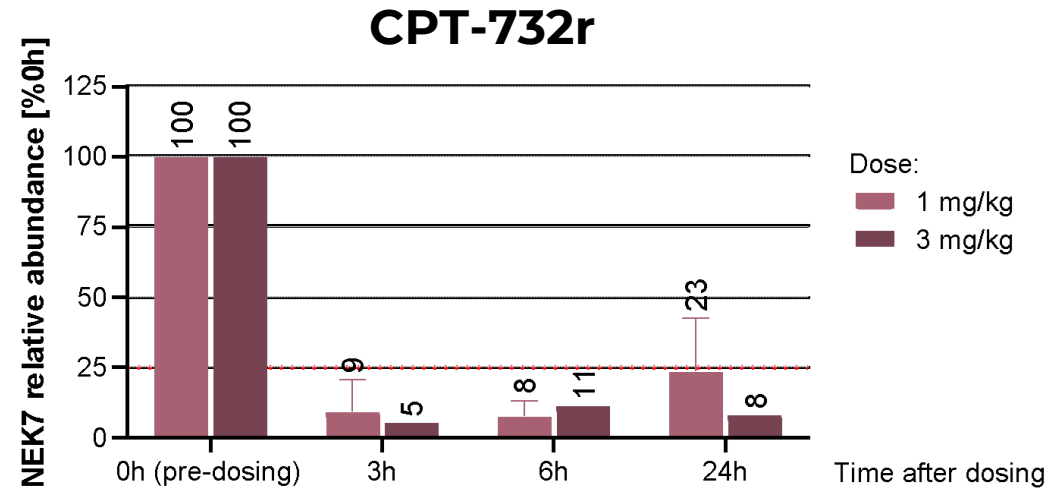
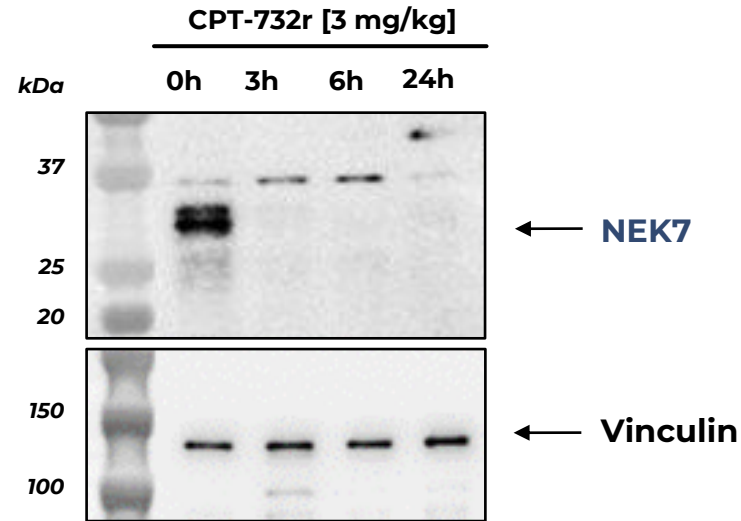
Treatment over 32 days did not induce any side effects

Treatment with CPT-635r at a dose of 30 mg/kg reduced the clinical score by approximately 50% compared to the control group, to a degree comparable to MCC950. Dose-dependent therapeutic effect is observed (30 mpk vs.10 mpk)

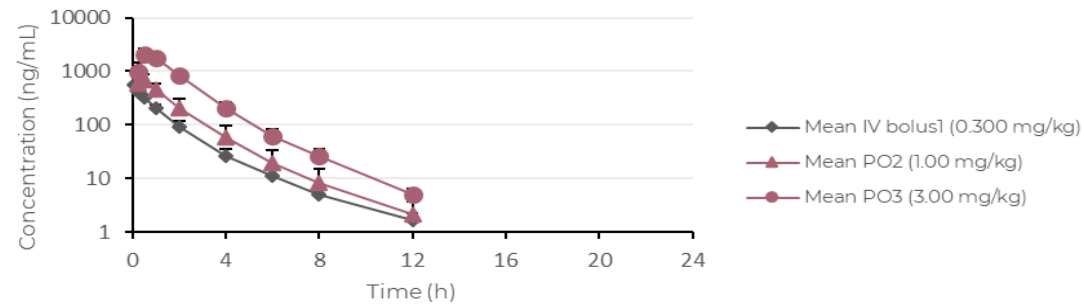
T/B-cell driven; Collagen-Induced Arthritis

CPT-732r efficiently covers & degrades NEK7 in NHPs after a single dose

CT-02B



Mean Plasma Concentration of CPT-732r after IV bolus and PO dosing



NEK7 degradation observed in monkey PBMCs isolated at 3, 6, and 24 h after PO administration of CPT-732r [1 & 3 mg/kg]

CT-02B
(NEK7)

CT-02S: Transforming the Treatment of Autoimmune Disorders

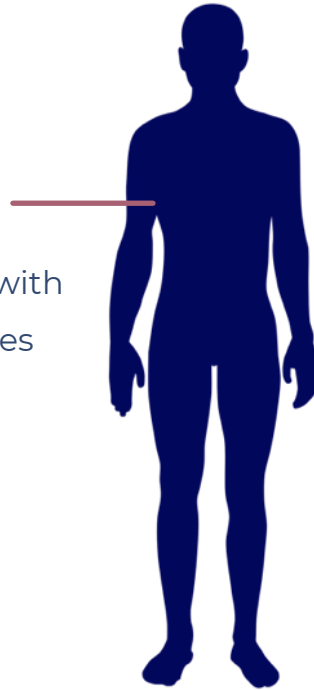
CT-02S
CPT-635

Peripheral autoimmunity

NEK7 degraders could be used in combination with available therapeutics to manage comorbidities

Three significant therapeutic areas:

Autoimmune
Metabolic
Cardiovascular



Activity confirmed *in vitro* in mouse, monkey and human cells and *in vivo* in mice and monkeys

Specificity-driven safety demonstrated in *in vitro* analysis, *in vivo* tolerability studies and confirmed in the clean CEREP panel

PK/PD results in monkeys illustrate the attractive features of drug candidates

In vivo proof of efficacy in disease models with no signs of toxicity

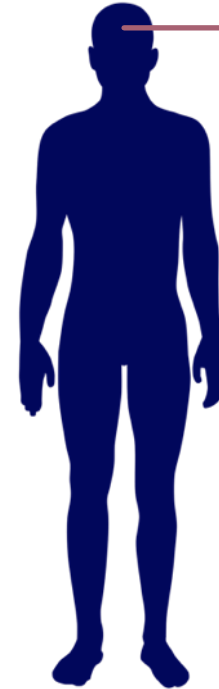
CT-02B: Breakthrough for Neurodegeneration

Activity confirmed *in vitro* in mouse, monkey and human cells and *in vivo* in mice and monkeys

Specificity-driven safety demonstrated in *in vitro* analysis, *in vivo* tolerability studies and confirmed in the clean CEREP panel

PK/PD results in monkeys illustrate the attractive features of drug candidates

In vivo proof of efficacy in disease models with no signs of toxicity



CT-02B
CPT-732

Neurodegenerative diseases

Growing evidence for role of pathological activation of innate immunity in the pathogenesis of NDDs
Limited treatment strategies available: opportunity to target neuroinflammation via NEK7 degradation & inhibit disease progression

Alzheimer's

Parkinson's

Multiple Sclerosis

Huntington's

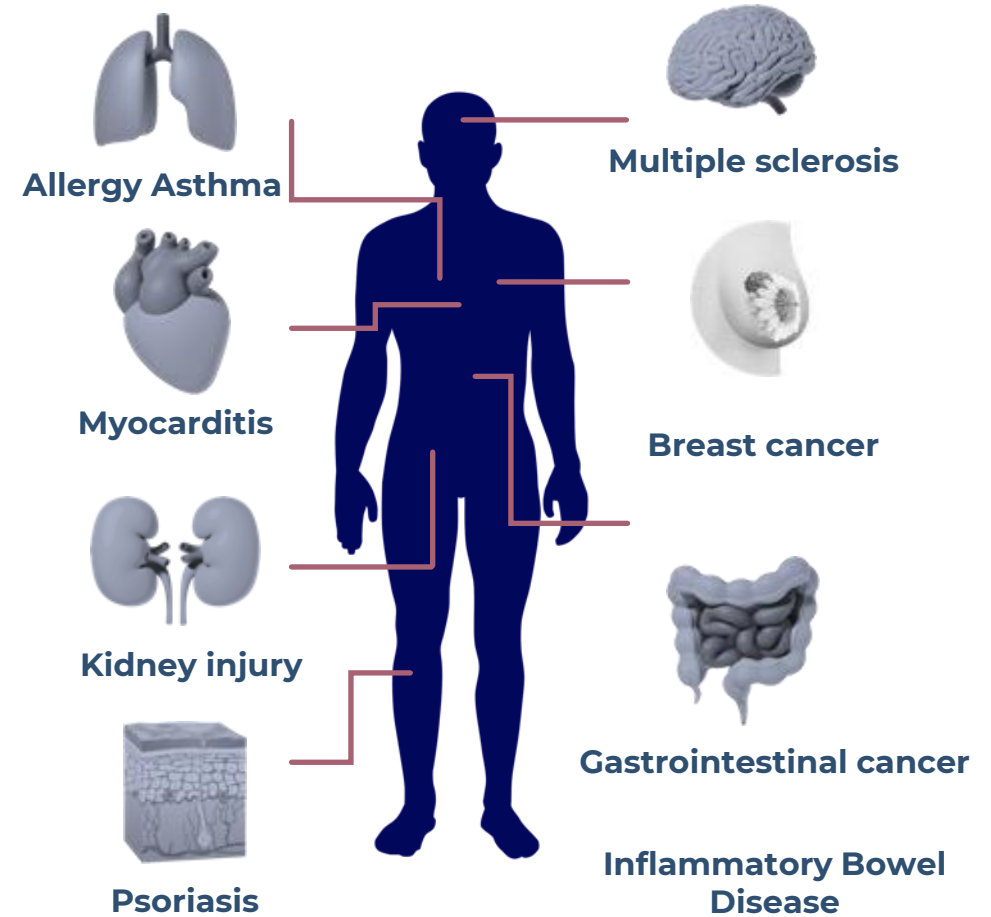
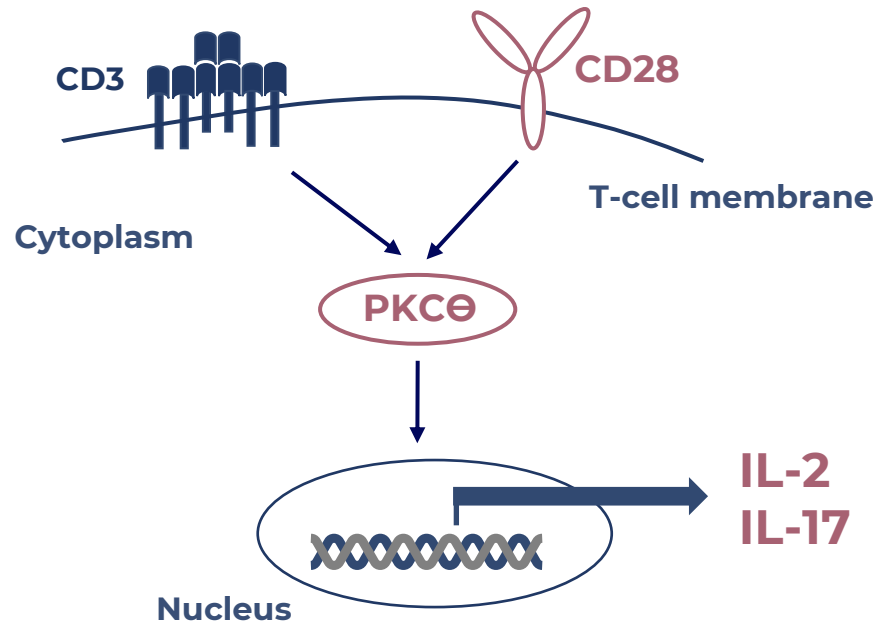
ALS

Metabolic

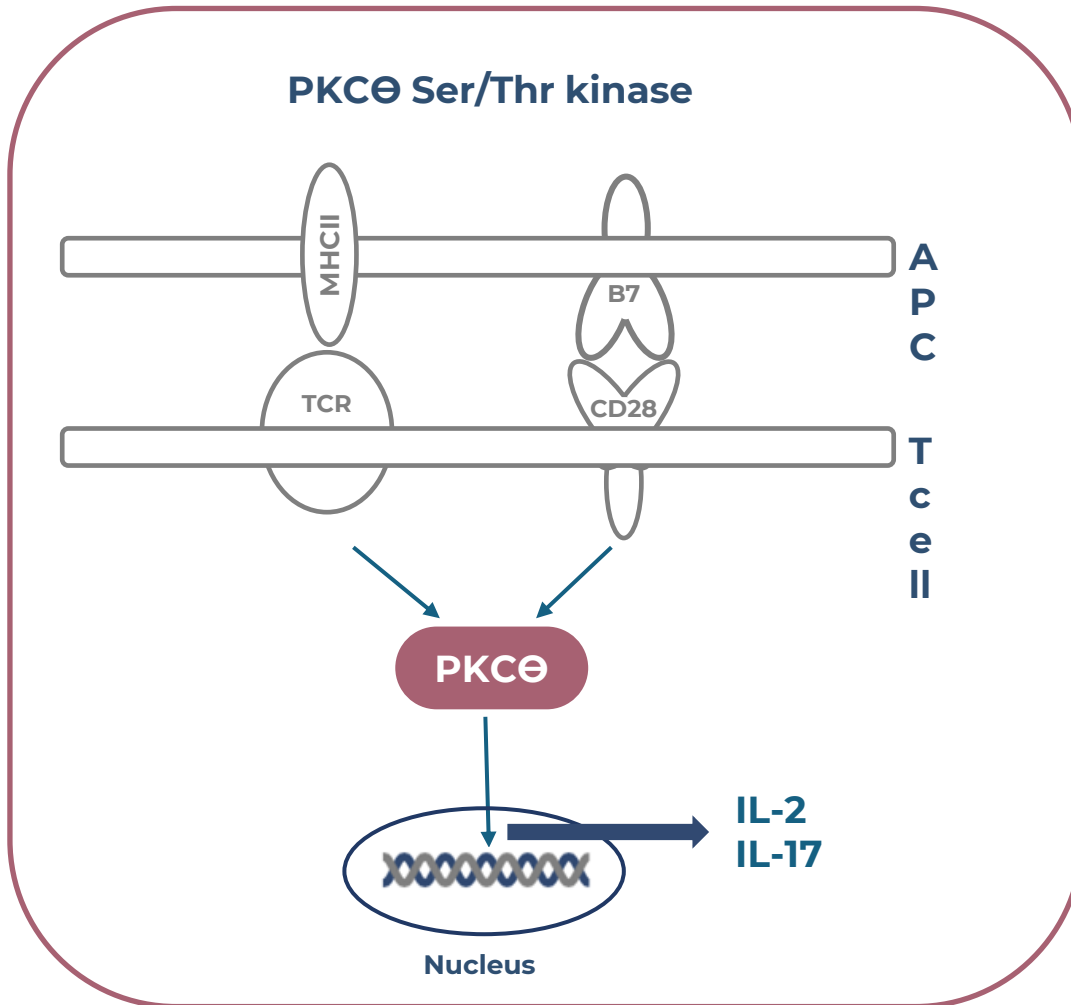
CT-05: First-in-Class PKC θ Degraders for Autoimmune Disorders

PKC θ : an undrugged high value target

TCR



PKC θ Biology and target rationale



Target Biology and rationale

PKC θ has a thoroughly established role in regulatory and effector T cell functions^{1,2}

PRCKQ locus was shown associated with several immune-related diseases in multiple GWAS studies (type I diabetes, rheumatoid arthritis, celiac disease)³⁻⁶

Human and mouse genetics

PKC θ KO mice show impaired *in vivo* T cell activation, decreased IL-17 production and are protected from T cell-mediated inflammatory diseases (EAE, colitis)^{7,8}

Clinical pathway validation

PKC θ inhibitor – Sotrastaurin (AEB071) – has been shown effective in preventing IL-17 production and to have a potential for therapeutic option in psoriasis⁹⁻¹¹

Currently, undergoing clinical evaluation is a novel inhibitor from Exscientia / BMS

1. PKC-theta in regulatory and effector T cell functions, Brezar V., 2015, Front. Immunol. 6
2. Intervention of PKC- θ as an immunosuppressive regimen, Sun Z., 2012, Front Immunol. 3: 225
3. Meta-analysis of genome-wide association study data identifies additional type 1 diabetes risk loci, Cooper J.D., 2008, Nat. Genet. 40, 1399-1401
4. Common variants at CD40 and other loci confer risk of rheumatoid arthritis, Raychaudhuri S., 2008, Nat. Genet. 40, 1216-1223
5. Genome-wide association study meta-analysis identifies seven new rheumatoid arthritis risk loci, Stahl E.A., 2010, Nat. Genet. 42, 508-514
6. Meta-analysis of genome-wide association studies in celiac disease and rheumatoid arthritis identifies fourteen non-HLA shared loci, Zhernakova A., 2011, PLoS Genet. 7, e1002004

7. Mice deficient in PKC theta demonstrate impaired *in vivo* T cell activation and protection from T cell-mediated inflammatory diseases, Anderson K., 2006, Autoimmunity, 6: 469-487
8. Resistance to experimental autoimmune encephalomyelitis and impaired IL-17 production in protein kinase C θ -deficient mice, Tan S-L., 2006, J Immunol. 176(5): 2872-2879
9. The PKC inhibitor AEB071 may be a therapeutic option for psoriasis, Skvara H., 2008, J Clin Invest. 118(9): 3151-9
10. The protein kinase C inhibitor sotrastaurin allows regulatory T cell function, de Weerd A., 2013, Clin Exp. Immunol. 175(2): 296-304
11. Targeting PKC in Human T Cells Using Sotrastaurin (AEB071) Preserves Regulatory T Cells and Prevents IL-17 Production, He X., 2013, J Invest dermatol. 134(4): 975-983

First-in-class PKC θ degrader as an antibody-like activity in a pill

Established a screening workflow that allows for discovery of PKC θ degraders superior to existing inhibitors

Highly selective for PKC θ with no off-target toxicity

Early stage of lead optimisation with 2 compounds has demonstrated:

In vitro: degradation of PKC θ in mouse & human T-cell line & inhibition of IL-2 and IL-17 in human T-cells

In vivo: degradation of PKC θ in mouse splenocytes

Optigrade™ Targeted Protein Degradation Platform

Molecular glues

Bifunctional Degraders

Novel E3 ligases

LiLis™ program: developing novel E3 ligases beyond CRBN

Expanding the range of targets for effective degradation

CRBN down regulation-driven resistance mechanisms in cancer

Crowded IP space for CRBN binders

Opportunity for cell type or cell compartment specificity

**In-house developed E3 ligase production platform and is generating leads
for novel E3s**

LiLis™: new E3 Ligases

	N-degron pathway			DesCEND pathway	
Stage	GID4 Ligands available & degraders tested	Ligase A* PROTACability confirmed	KLHDC2 PROTACability confirmed	Ligase B Fragments & X-ray structures	Ligase C Fragments & X-ray structures
Key feature	SRS of an evolutionarily conserved, multi-subunit E3 complex (HMGCS1, ARHGAP11A, DDX17 – among endogenous targets)	Expressed in cytosol & nucleus, essential for some cancers; Not expressed in liver	Ubiquitously expressed, predominantly in the nucleus	Benign KO prevents autoimmune disease	Upregulated in solid tumors; absent in heme precursor cells => no heme AE
Known in TPD	YES	NO	YES	NO	NO
Status at Captor	Biophysical, cellular, permeability assays; protein production & crystallography platform; MedChem			Biophysical, cellular, permeability assays; protein production & crystallography platform	

**Ligands interacting with two homologues E3 ligases of the N-end pathway*

Finance Highlights

Financial and equity summary

Income Statement and Balance Sheet, PLN mln

	1H 2025	2024
Revenues:		
Collaboration revenue	4.6	15.8
Grant revenue	2.9	5.8
Other revenues	3.6	1.1
Costs:		
Salaries and employee benefits	11.8	21.1
Third-party services	14.6	29.8
Depreciation (incl. from leasing)	2.5	5.1
Materials and energy	2.1	3.7
Other costs (incl. financial)	0.6	1.5
Balance sheet (end of period):		
Cash and equivalents	48.7	72.2
Debt	0.0	0.0

Shares outstanding: **5,528,709** (October 2, 2025)

Market: **Warsaw Stock Exchange** (main market)

ISIN: **PLCPTRT00014**

Ticker: **CTX PW**

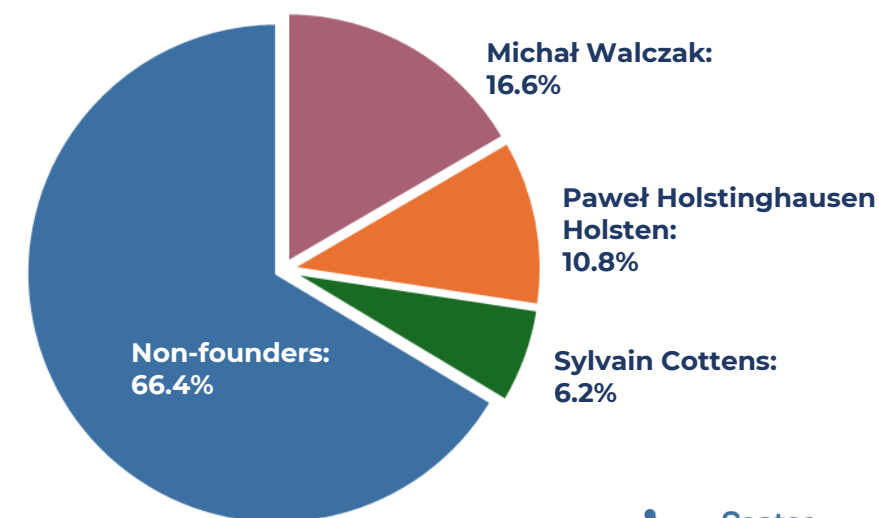
Analyst coverage: **Ipopema, mBank, Noble Securities, PKO BP, Trigon**

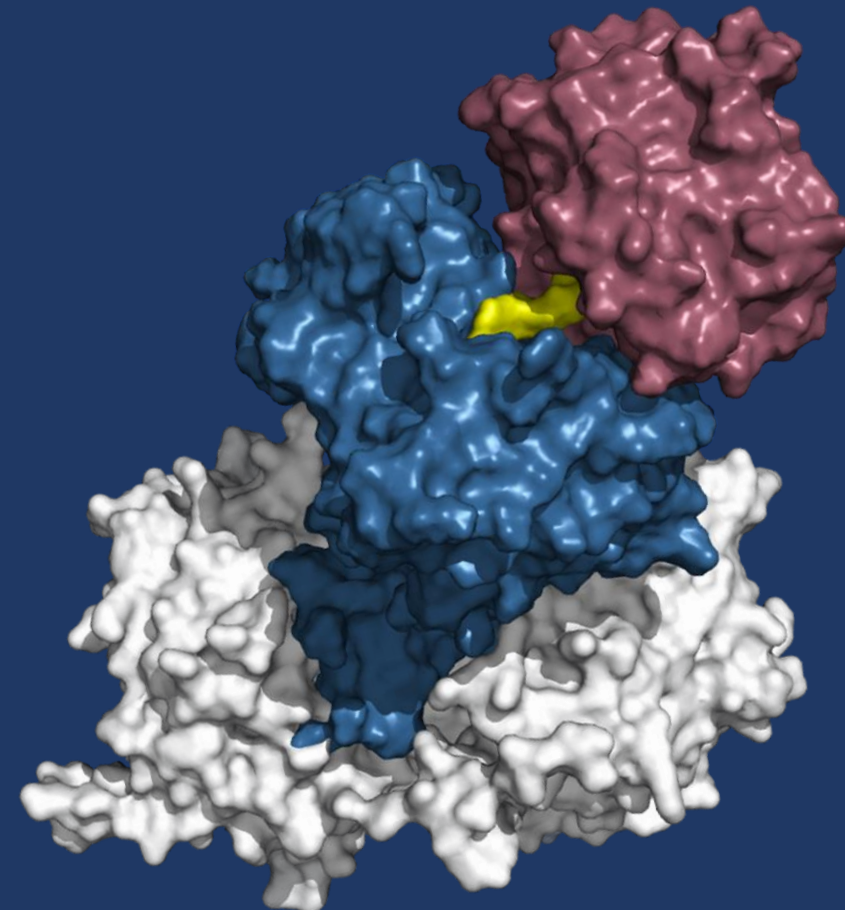
E-mail: **investor.relations@captortherapeutics.com**

Top shareholders

Michał Walczak	Founder, CEO & CSO	16.6%
Paweł Holstinghausen Holsten	Founder	10.8%
TFI Allianz Polska SA	Mutual funds	9.1%
Sylvain Cottens	Founder, Head of Chemistry	6.2%
OFE Nationale Nederlanden	Pension fund	6.2%
OFE Allianz Polska	Pension fund	4.2%
TFI PZU SA	Mutual funds	3.6%
OFE PKO BP Bankowy	Pension fund	3.5%
OFE PZU Złota Jesień	Pension fund	3.4%
TFI Quercus SA	Mutual funds	2.7%
OFE Generali	Pension fund	2.6%
Other shareholders		31.2%

Source: *stooq.pl* (Oct 2, 2025), H1 2025 report of Captor Therapeutics





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Projects co-financed by the European Regional Development Fund:

Discovery and development of a new clinical drug candidate for the eradication of cancer stem cell in the treatment of hepatocellular carcinoma, through degradation of oncofetal transcription factor” (POIR.01.01.01-00-0740/19-00)

Discovery and development of a new clinical drug candidate for the eradication of cancer stem cell in the treatment of hepatocellular carcinoma, through degradation of oncofetal transcription factor - Stage II
(FENG.01.01.IP.01-1001/23-00)

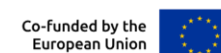
Inducing apoptosis with small molecules as therapeutic intervention in multiple severe malignancies (POIR.01.01.01-00-0956/17-01)

Inducing apoptosis with small molecules as therapeutic intervention in multiple severe malignancies – Stage II (FENG.01.01-IP.01-1002/23)

Application of targeted protein degradation technology in the treatment of psoriasis and rheumatoid arthritis (POIR.01.02.00-00-0079/18-00)

Development of an integrated technology platform in the field of targeted protein degradation and its implementation to the pharmaceutical market (POIR.01.01.01-00-0931/19-00)

Discovery and development of non-toxic ligase ligands and their application in the treatment of autoimmune diseases
(POIR.01.01.01-00-0741/19-00)



Project co-financed by the state budget from the Medical Research Agency:

Design and clinical development of a first-in-class small-molecule drug candidate for the treatment of colorectal cancer based on the stimulation of immune cells to increase anti-cancer activity through induced protein degradation (2022/ABM/06/00001 - 00)

