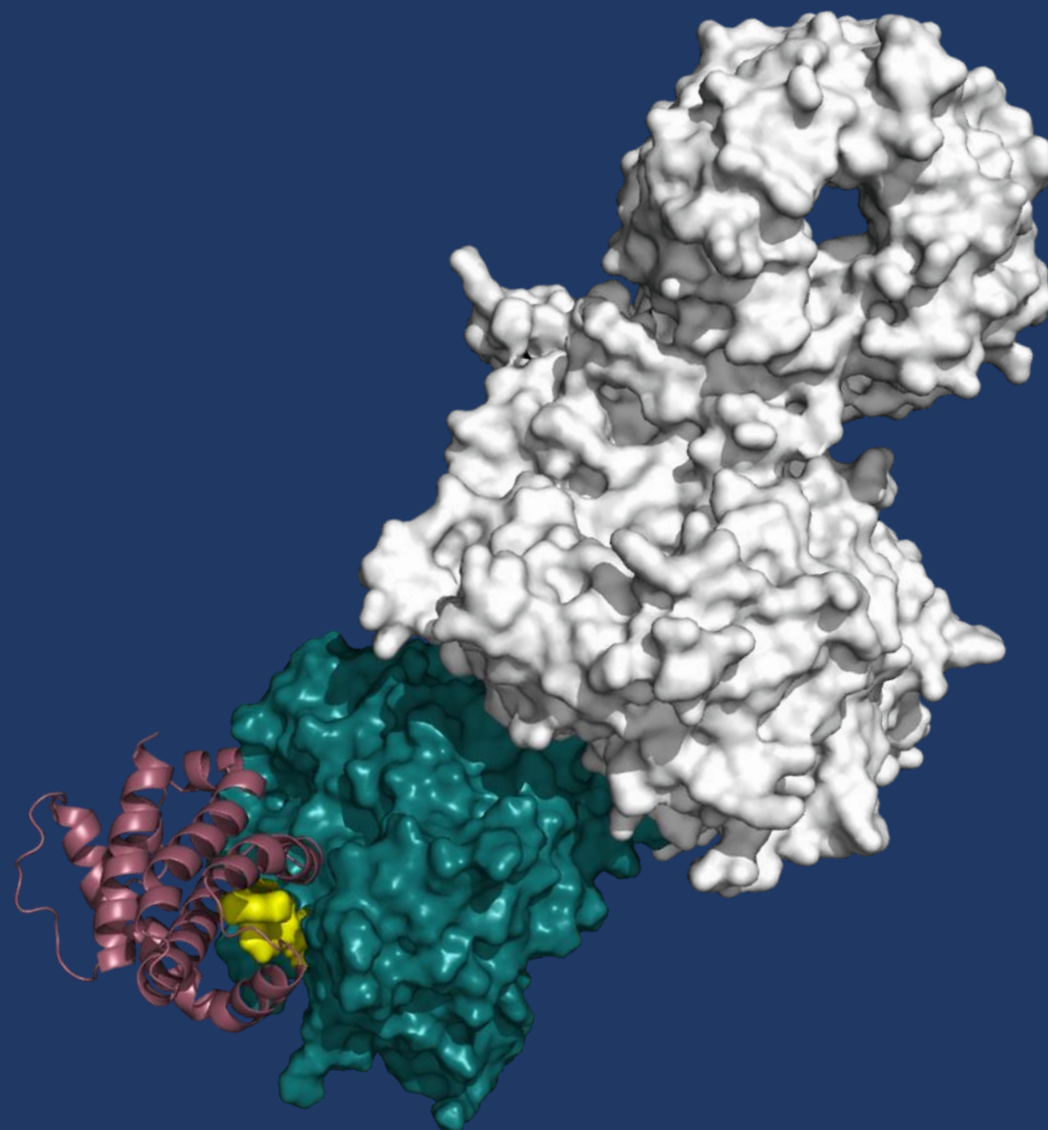




Captor  
Therapeutics®

*Pioneering targeted protein  
degraders for human health*

2026



# Legal notice

This document and the information contained herein (unless otherwise indicated) have been prepared by Captor Therapeutics S.A. (the "Issuer") solely for informational purposes. For this notice, the presentation that follows shall mean and include the slides that follow, the oral presentation of the slides by the Issuer or any person on behalf of the Issuer, any question-and-answer session that follows the oral presentation, hard copies of this document, and any materials distributed at, or in connection with the presentation (collectively, the "Presentation"). By attending the meeting at which the Presentation is made, or by reading the Presentation, you will be deemed to have (i) agreed to all of the following restrictions and made the following undertakings and (ii) acknowledged that you understand the legal and regulatory sanctions attached to the misuse, disclosure or improper circulation of the Presentation.

The information contained in this Presentation may not be reproduced or redistributed in any way, in whole or in part, to any other person without the prior written consent of the Issuer. This Presentation does not purport to contain all the information that may be required by the recipient to assess the Issuer or its securities. The Issuer prepared this Presentation based on the information which it has and from sources believed to be reliable. To the extent available, the industry, market, and competitive position data contained in this Presentation come from official or third-party sources. There is no guarantee of the accuracy or completeness of such data.

This Presentation contains neither a complete nor a comprehensive financial or commercial analysis of the Issuer, nor does it present its position or prospects in a complete or comprehensive manner. The Issuer has prepared the Presentation with due care, however certain inconsistencies or omissions might have appeared in it. Therefore it is recommended that any person who intends to undertake any investment decision regarding any security issued by the Issuer shall only rely on information released as an official communication (i.e., current/periodic reports) in accordance with the legal and regulatory provisions.

This Presentation may contain certain forward-looking statements, forecasts, estimates, projections, and opinions ("Forward-looking Statements"). By their nature, Forward-looking Statements involve known and unknown risks, uncertainties, assumptions, and other factors because they relate to events and depend on circumstances that will occur in the future whether or not outside the control of the Issuer. No representation is made or will be made that any Forward-looking Statements will be achieved or will prove to be correct. Actual future results and operations could vary materially from the Forward-looking Statements. Similarly, no representation is given that the assumptions disclosed in this Presentation upon which Forward-looking Statements may be based are reasonable. The recipient acknowledges that circumstances may change and the contents of this Presentation may become outdated as a result. The assumptions included herein do not constitute profit forecasts or profit estimates.

No warranties or representations can be made as to the comprehensiveness or reliability of the information contained in this Presentation. Neither the Issuer nor its directors, managers, advisers or representatives of such persons shall bear any liability that might arise in connection with any use of this Presentation. Furthermore, no information contained herein constitutes an obligation or representation of the Issuer, its managers or directors, its shareholders, subsidiary undertakings, advisers or representatives of such persons. Data contained in this Presentation is valid as of the day of its preparation. Consequently, this Presentation will not be subject to changes, updates or modifications to account for events which might occur after this day.

This Presentation does not constitute or form part of, and should not be construed as, an offer to sell or issue, or the solicitation of an offer to purchase, subscribe to, or acquire the Issuer or the Issuer's securities, or an inducement to enter into investment activity in any jurisdiction in which such offer, solicitation, inducement or sale would be unlawful before registration, exemption from registration or qualification under the securities laws of such jurisdiction. No part of this Presentation, nor the fact of its distribution, should form the basis of, or be relied on in connection with, any contract or commitment or investment decision whatsoever. This presentation is not for publication, release, or distribution in any jurisdiction where to do so would constitute a violation of the relevant laws of such jurisdiction nor should it be taken or transmitted into such jurisdiction.

# Captor Therapeutics: a clinical stage TPD company



- biotechnology company based in Wrocław (Poland) and Basel (Switzerland)
- all projects developed in Captor's **own drug discovery platform**
- develops **first-in-class drugs**
- one of pioneers of **targeted protein degradation** (TPD)
- **viadrudomide (CT-01)** is in a **clinical trial** for **liver cancer**
- **vratitoclax (CT-03)** will be tested in an **investigator-initiated trial** at **MD Anderson Cancer Center**
- several drug projects for autoimmune diseases – especially **NEK7 degraders**
- beneficiary of numerous grants, including EIC Accelerator in 2025 – one of twelve biotech companies from the EU
- **~80 FTEs on board, 50% are PhDs** – mainly young scientists from Wrocław, Poland
- several biotechnology veterans
- labs in Wrocław, Poland – Western standards at low cost
- relatively high share of in-house work – limited outsourcing
- shares listed on **Warsaw Stock Exchange**
- ~600 m2 of laboratory space equipped with state-of-the-art machinery

# An experienced leadership team



**Michal Walczak, PhD**  
Co-founder  
Chief Executive Officer  
Chief Scientific Officer

- 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



**Adam Łukojć, PhD, CFA**  
Chief Financial Officer

- PhD Kozminski University
- Certificates and licences: CFA, PRM, investment advisor
- 20 years of experience in the capital market



**Adam Ostrowski, MD**  
Chief Medical Officer

- MD, specialization in Internal Medicine & Clinical Oncology
- 30+ years in patient care & systemic treatment of solid tumors
- Head of Clinical Oncology Department & Medical Director at a regional hospital in Poland
- Clinical research expert across preclinical, translational & Phase I-IV trials



**Sylvain Cottens, PhD**  
Co-founder & SVP Chemistry

- PhD EPFL Lausanne
- Post-doc at Caltech
- Scientific expert & leader 25+ years at Novartis
- Involved in two blockbuster drugs (co-inventor of Afinitor & co-developer of Gilenya)



**Tom Shepherd, PhD**  
Strategy Consultant

- Ex-Captor CEO
- PhD University of Strathclyde
- CPE London Business School
- 30 years in Biotech
- Led 12 licensing deals >€3B sales
- 6 private investment rounds & 3 IPOs

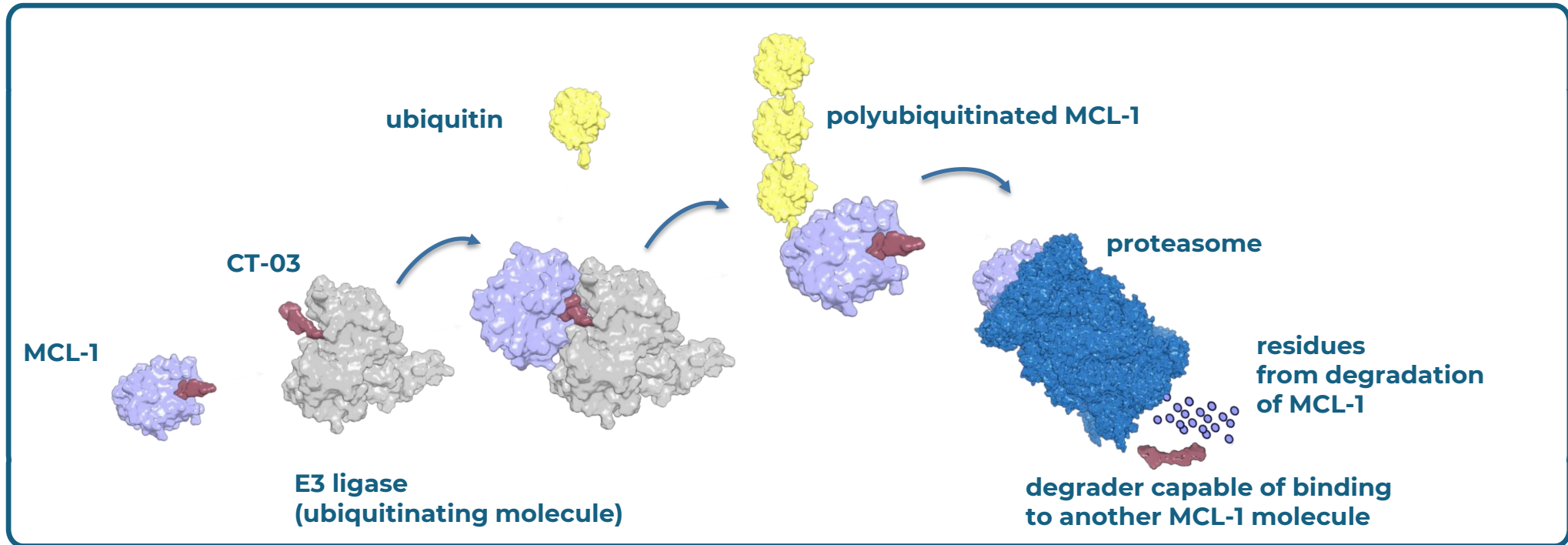


# Pipeline

Project name	Target	Indications	Modality	Discovery	Pre-clinical	IND-enabling	Phase I
Viadrudomide (CT-01)	GSPT1 & NEK7	Hepatocellular carcinoma, lung cancer, rare cancers	MG	▶			
Vratitoclax (CT-03)	MCL-1	Liquid & solid tumors	BID	▶			
CT-02B	NEK7	Neuroinflammation (Parkinson's disease, ALS, MS)	MG	▶			
CT-02S	NEK7	Systemic autoimmunity (IBD, gout, dermatology)	MG	▶			
CT-05, PKCtheta	PKCθ	Autoimmunity, transplantation, metabolism	BID	▶			
---	Undisclosed	Oncology, autoimmunity, CNS, rare diseases	MG/BID	▶			
---	Novel E3 ligases	Oncology, autoimmunity	MG/BID	▶			

MG - molecular glue, BID - bifunctional degrader

# Mechanism of action of TPD-based drugs



Very low doses due to catalytic action (shown for CT-01, CT-02 or MonteRosa GSPT1 degrader)

Overcoming cancer resistance (shown for CT-03 or ARV-766 from Arvinas)

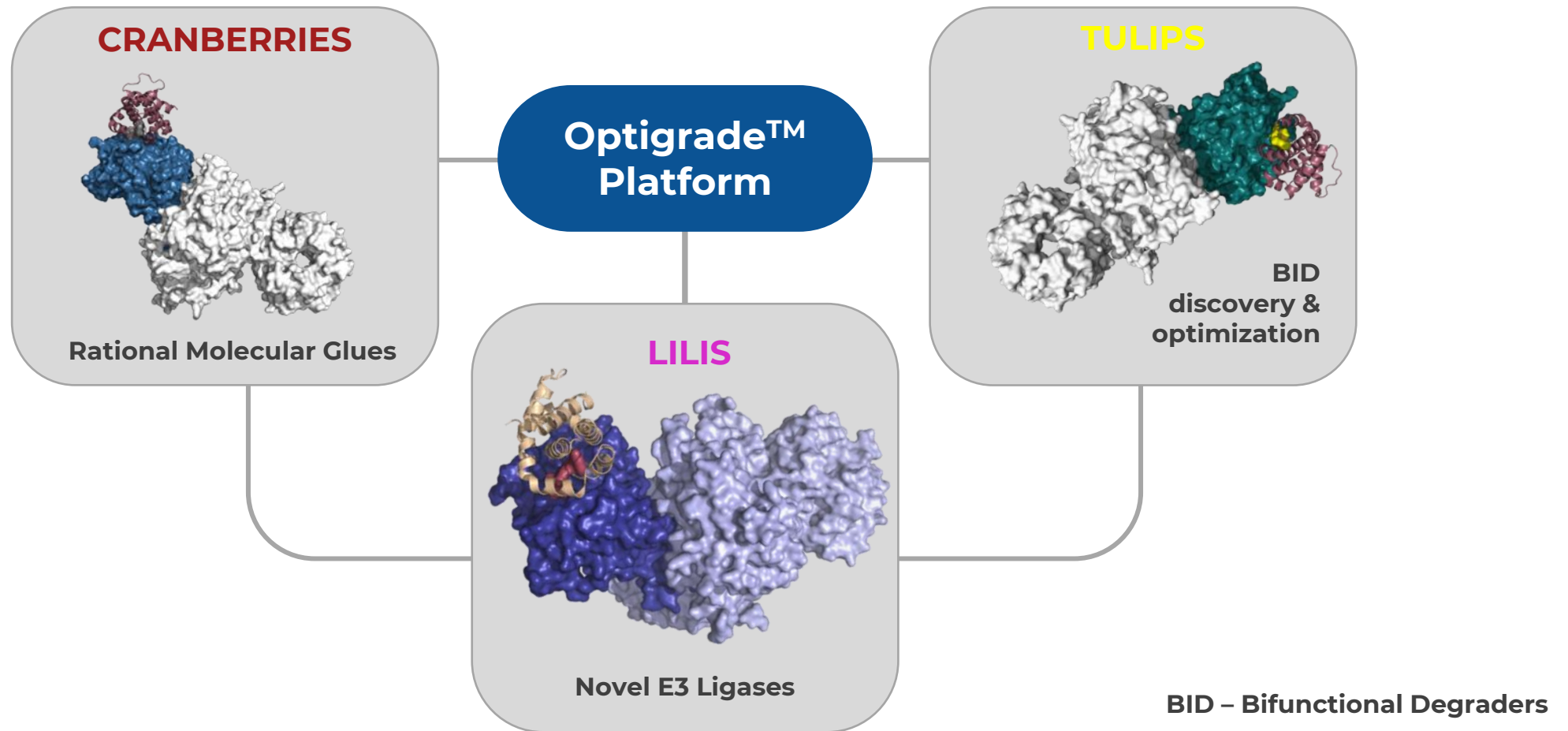
Targeting undrugged scaffolding proteins (shown for CT-02 or STAT6 degraders from Kymera)

# 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

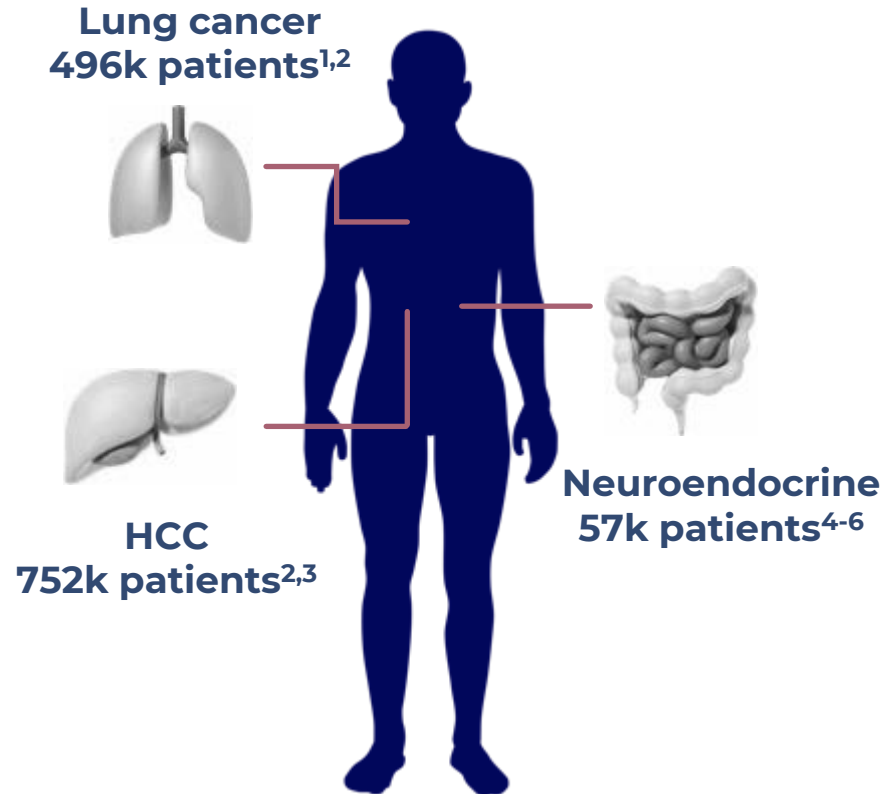


Optigrade™ – addressing Molecular Glues, Bifunctional Degraders and novel E3 Ubiquitin Ligases

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

---

# A first-in-class molecular glue prodrug degrader of GSPT1 & NEK7



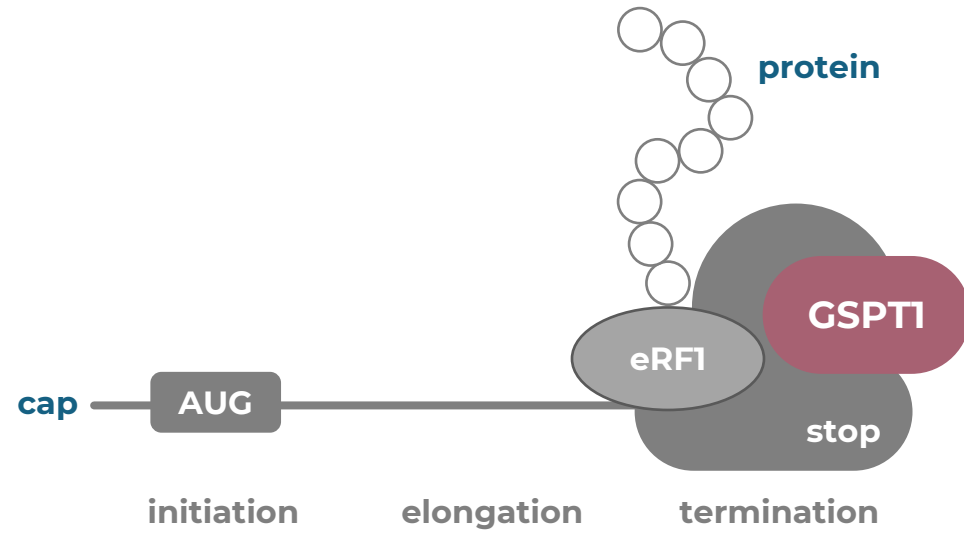
**GSPT1** degradation leads to an Integrated Stress Response (ISR) and induction of apoptosis in HCC cells

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

**Viadrudomide 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 translation<sup>1</sup>

GSPT1 and eRF1 form a translation termination complex that facilitates the nonsense mediated mRNA Decay<sup>2,3</sup>

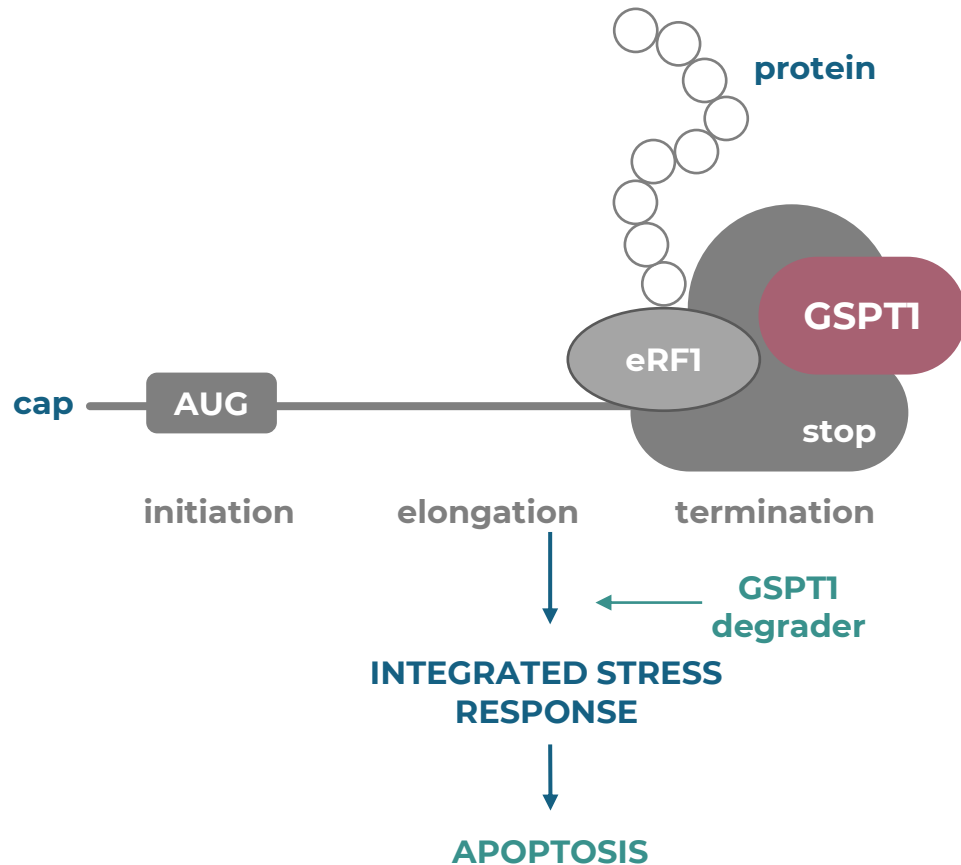
### Clinical opportunity

Targeting protein translation via GSPT1 degradation offers treatment options for:

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 $\beta$  depletion



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

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

**Viadrudomide** 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.

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

Downregulation of NEK7/IL-1 $\beta$  reduces inflammation and improves outcomes of treatments

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

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

## 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 \$3 billion could grow exponentially through extending overall survival

## Different from mono-GSPT1 degraders

Targets cancer cell proliferation (GSPT1) & tumor microenvironment (NEK7/IL-1 $\beta$ )

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

**Viadrudomide synergizes with everolimus and potentially with standard of care  
via inflammation reduction**

# Standard of care fails to significantly extend lives of HCC patients

## Improving modest survival: a path to accelerated approval

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

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

# Viadrudomide is highly differentiated among GSPTI degraders

Characteristics of viadrudomide may provide disease-specific efficacy and high safety

Feature	Viadrudomide	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

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

Tissue-activated degrader; dual selectivity



Systemic degrader

multiple other



multiple other

Antibody-conjugated degraders (DAC)

Class

Discovery

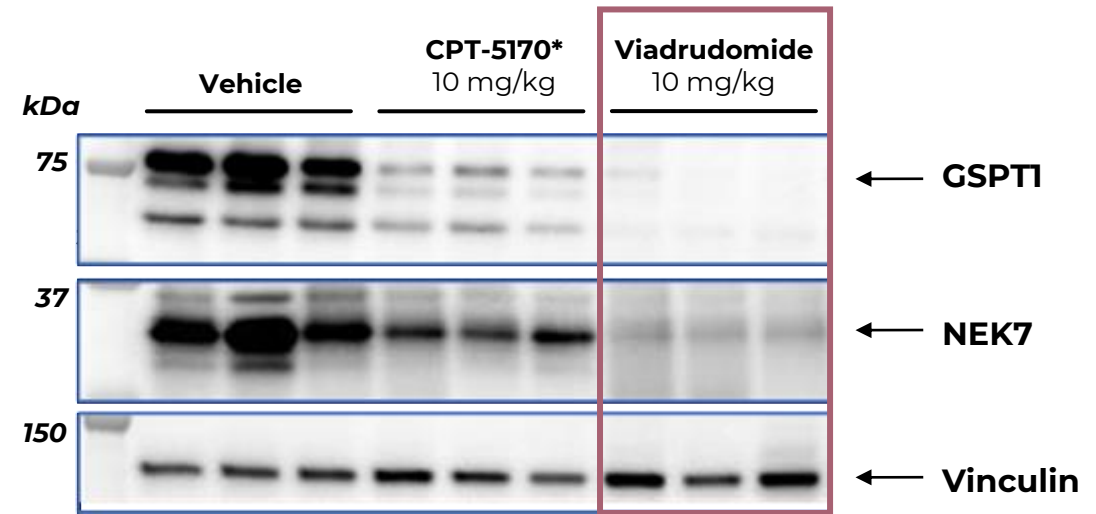
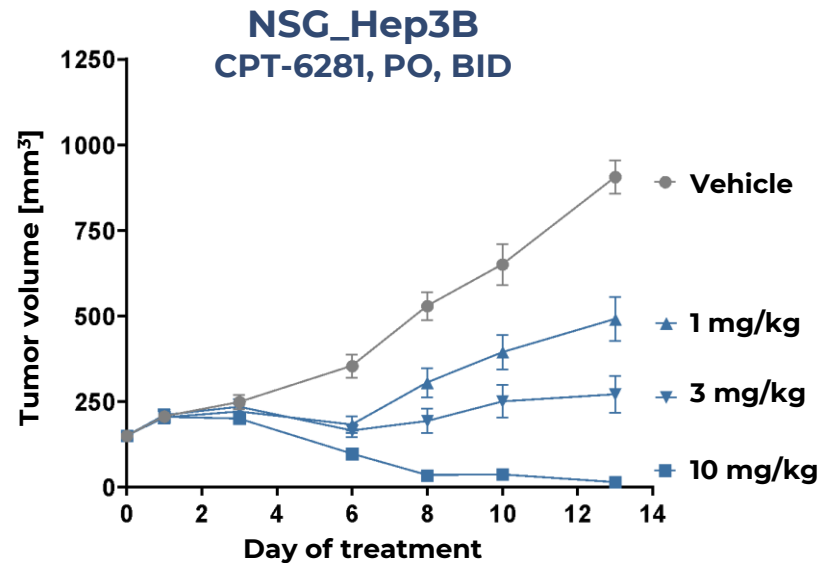
Preclinical

Phase I/II



# Viadrudomide potently regresses fast growing HCC tumors in mice

Viadrudomide efficiently degrades GSPT1 and NEK7

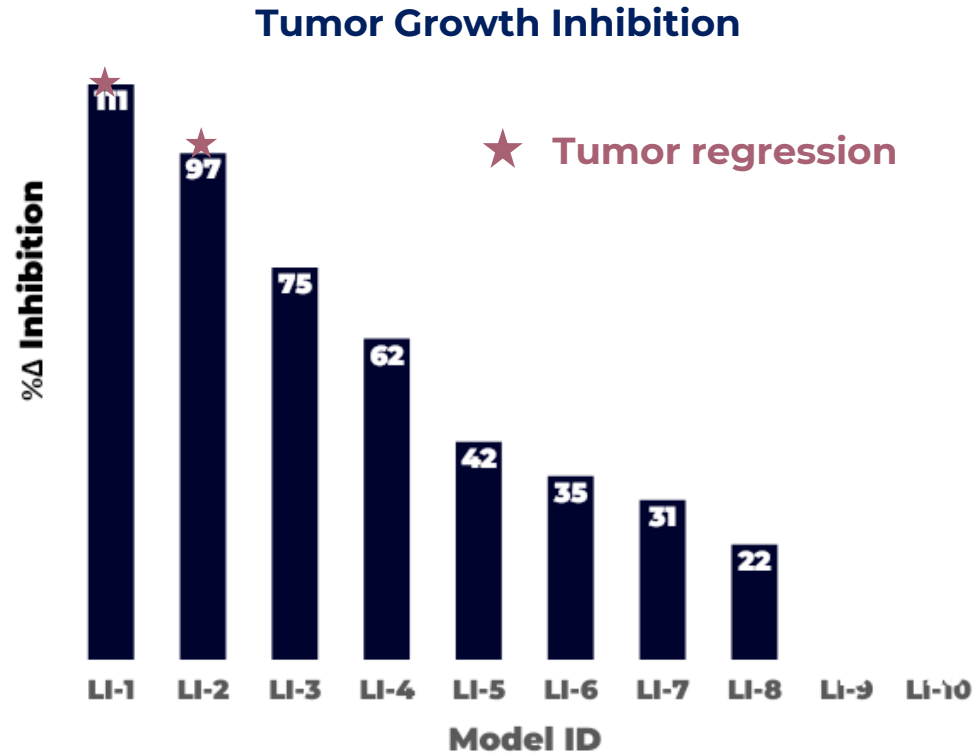


Regression of large tumors (~ 150 mm<sup>2</sup>) observed at doses as little as 10 mg/kg BID administered orally  
\*CPT-5170: an early lead compound in the project

Viadrudomide 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

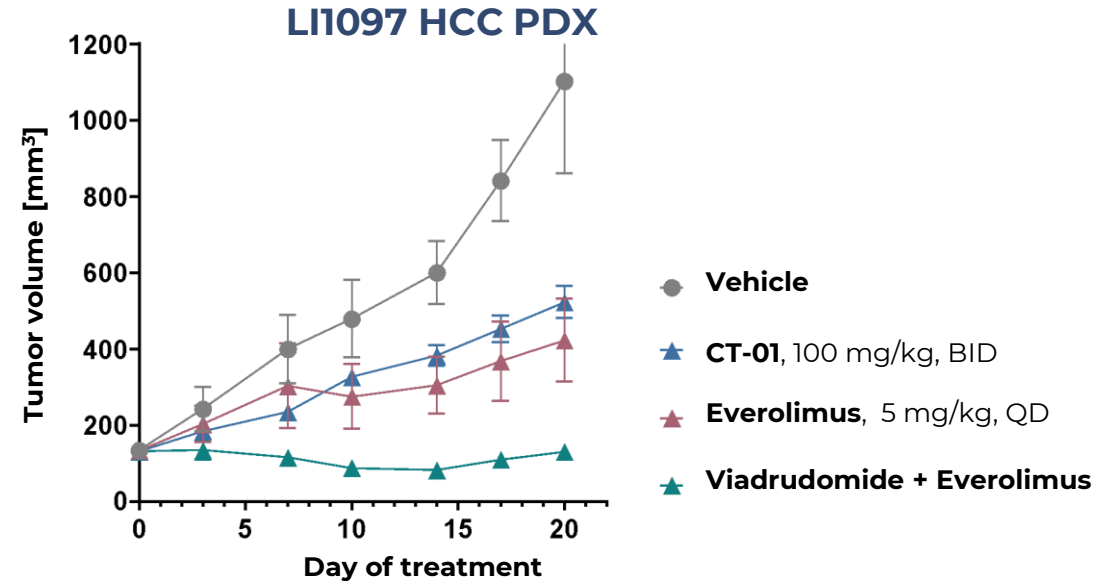
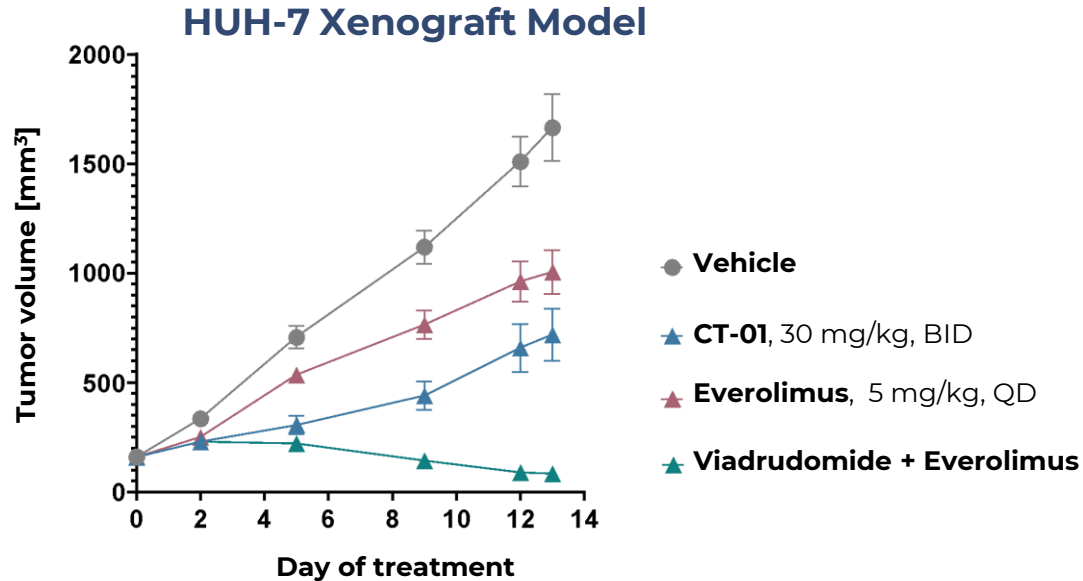
2 models show complete response

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 viadrudomide in combination with everolimus

Everolimus (EVE) and viadrudomide display strong synergy *via* molecular pathway cross-talk

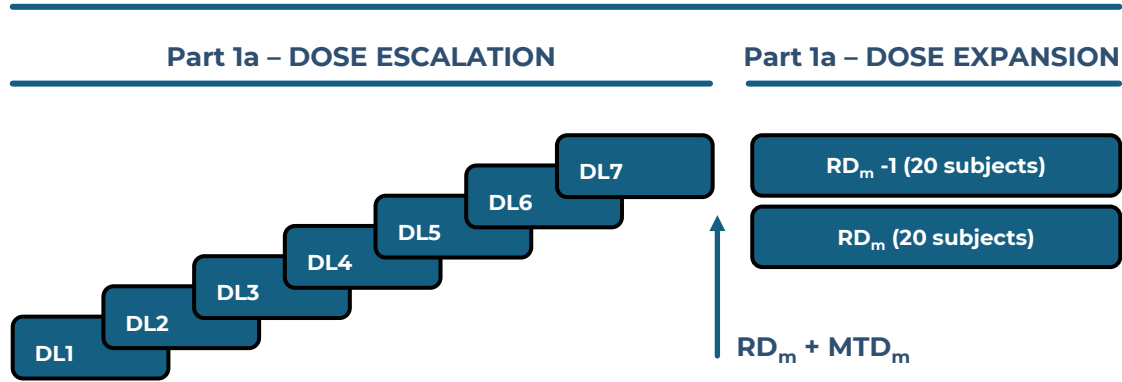


Everolimus is an approved drug in oncology

Combination with everolimus sensitizes non-responders and poor responders to viadrudomide

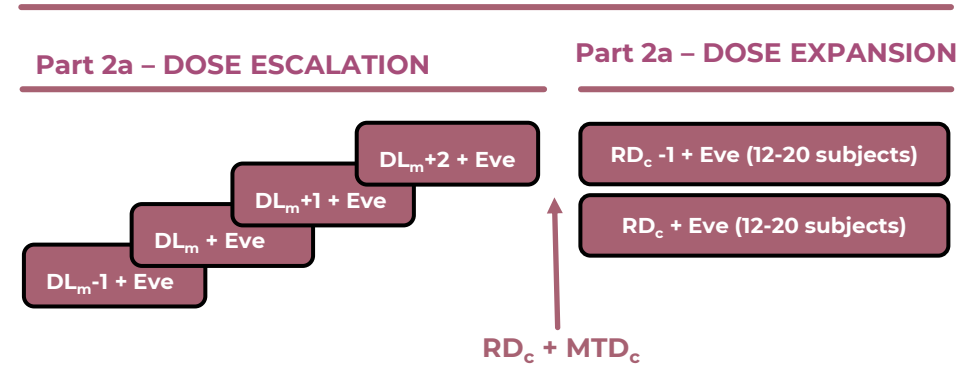
# Phase I in HCC patients - study design

## PART 1 – MONOTHERAPY



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

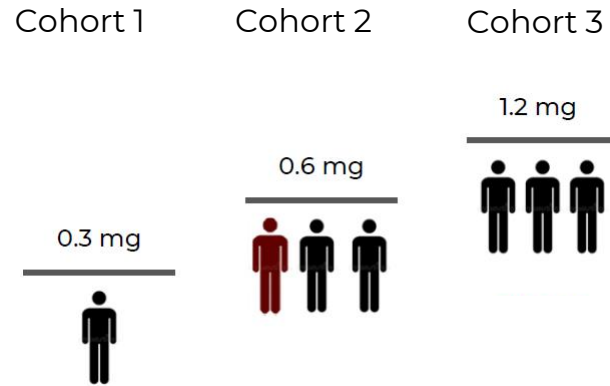


## PART 2 – COMBOTHERAPY VIADRUDOMIDE + EVEROLIMUS

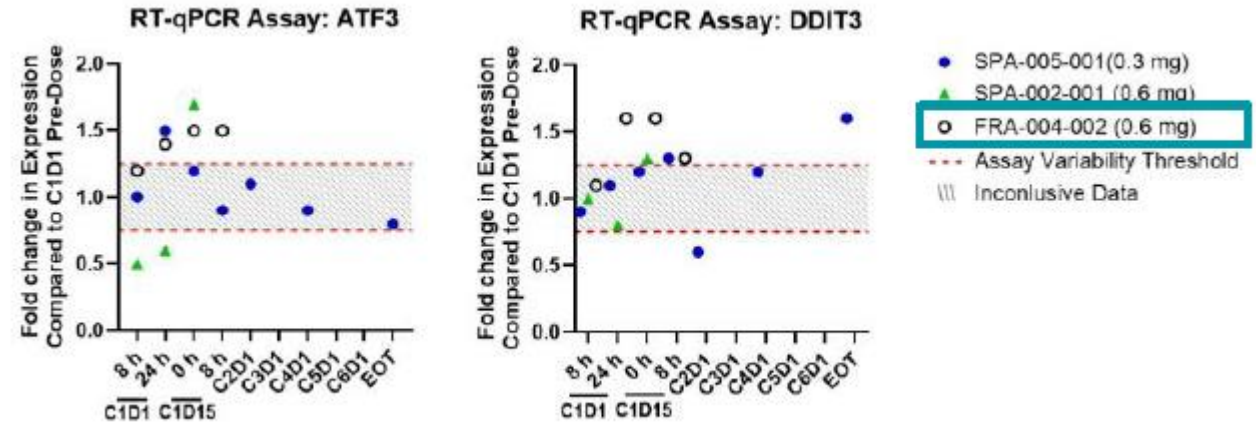
# Progress in the treatment of terminally ill HCC patients

## Increased levels of ATF3 & DDIT3 – biomarkers indicating cell death

### Viadrudomide (CT-01)



Prodrug released in liver  
 Increased active drug concentration expected in liver  
 Liver biopsy possible from Cohort 3 onwards



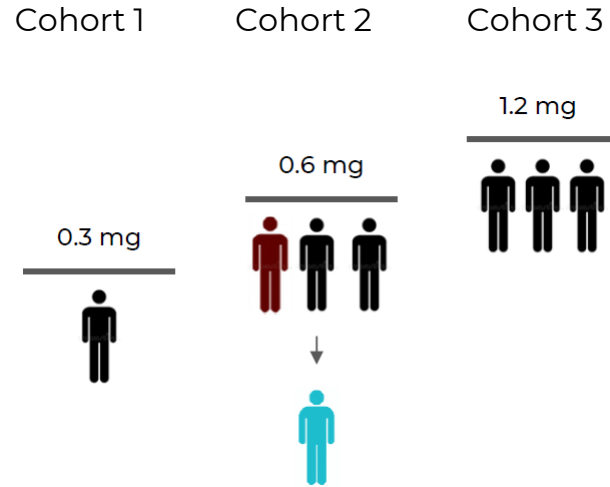
*No significant adverse effects have been observed to date*

Information accurate at the time of publication: 3 February 2026

# Progress in the treatment of terminally ill HCC patients

In one of the patients from cohort 2 disease inhibition is seen

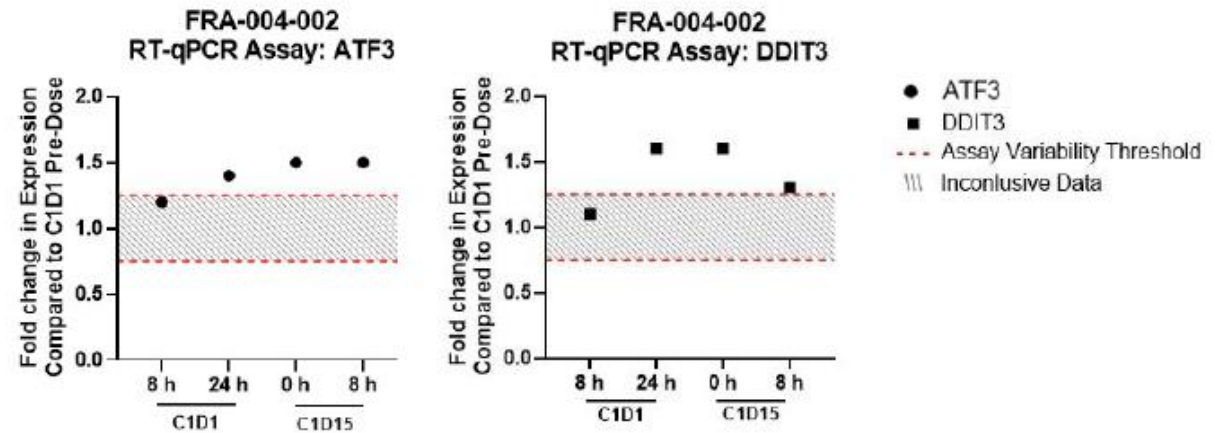
## Viadrudomide (CT-01)



„Stable disease“;  
treatment continues

*No significant adverse effects have  
been observed to date*

## FRA-004-002



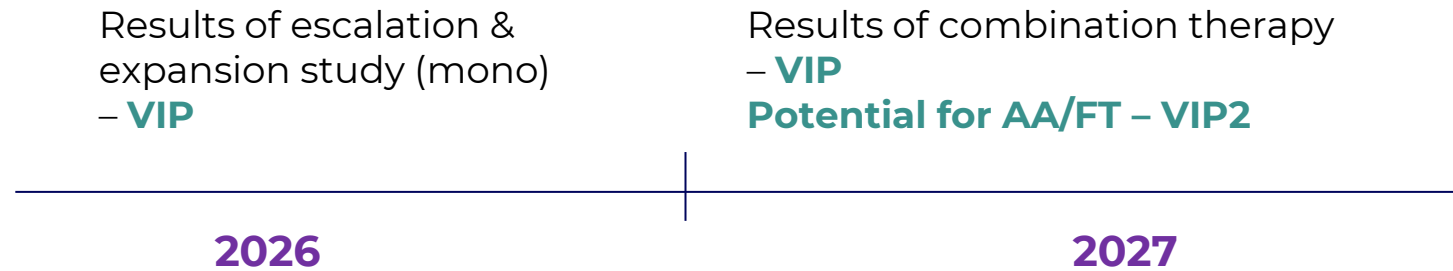
# What we expect in 2026

## Readouts in the coming months will determine project's value

Completing escalation and expansion studies in monotherapy – **Value Growth Point**

Escalation dose study in combination therapy

Potential expansion to new indications, e.g. CCA or other cancers without treatment options



# The only-in-class potential of highly differentiated viadrudomide

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

# Possible further development

## Other indications:

- **cholangiocarcinoma,**
- **lung cancer,**
- **breast cancer,**
- **neuroendocrine cancer.**

## Combotherapies – **viadrudomide** could possibly be combined with:

- **everolimus** – *synergy has been proven in animal models,*
- **anti-PD-1 antibodies,**
- **anti-PD-L1 antibodies.**

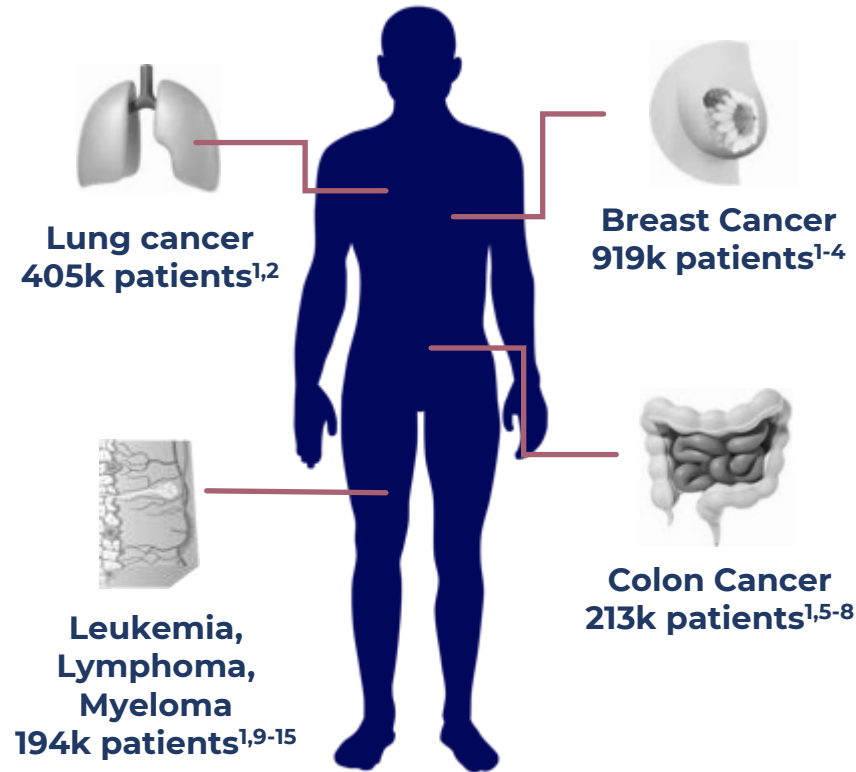
## ADC payload – **viadrudomide** could possibly be used as an ADC payload.

# Vratitoclax (CT-03): First-in-Class MCL-1 Degradator for Liquid & Solid Tumors

---

# 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% target coverage, leading to MCL-1 accumulation† and necrosis-mediated cardiotoxicity§

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

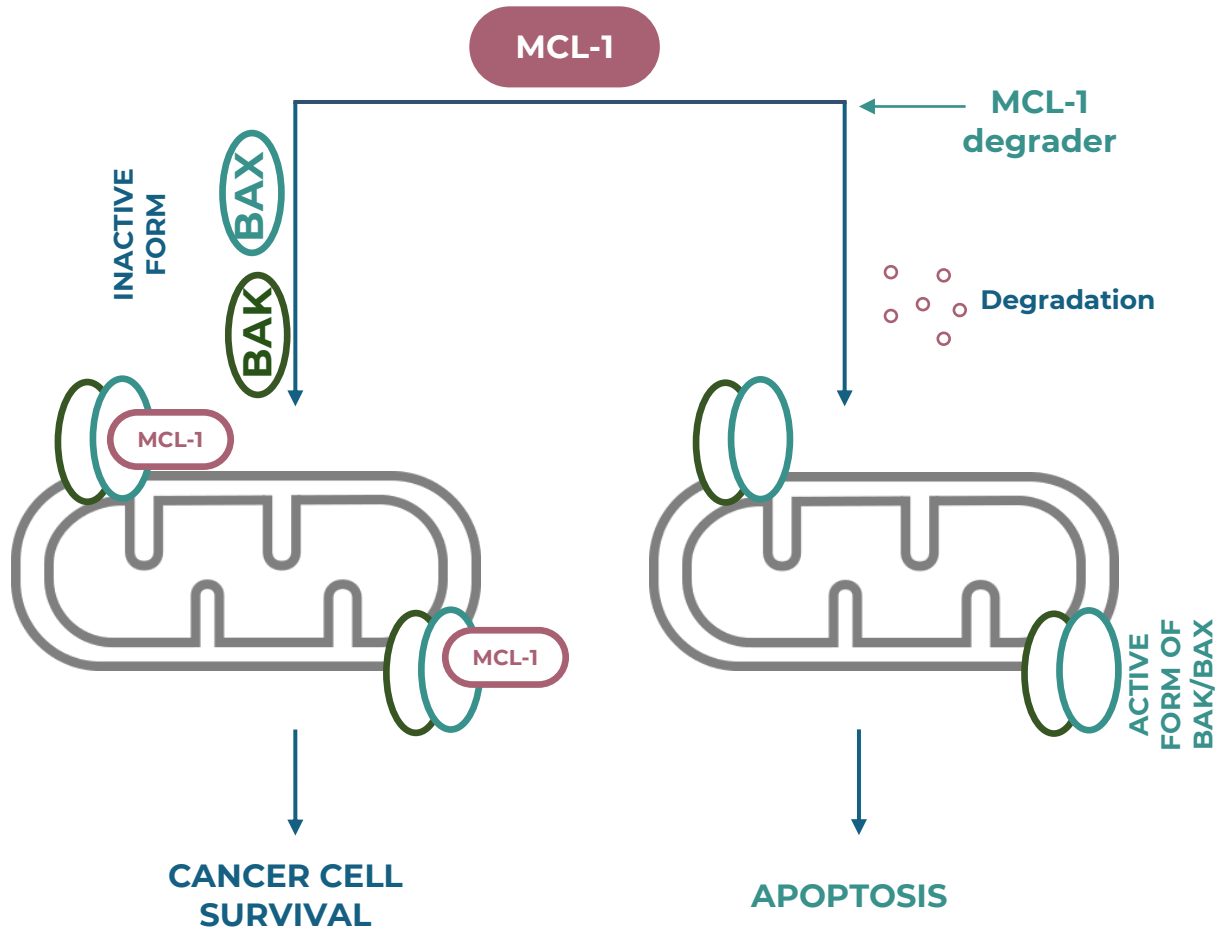
6. Cell Death Dis. 2022 13(1): 63  
7. Colorectal Dis 2022 24(11): 1295-1307  
8. Ann Fam Med. 2016 14(3): 215-20  
9. Exp Hematol Oncol. 2020 Jun 19;9:14  
10. Hum Pathol. 2004 Sep;35(9):1095-100

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

# 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**<sup>1</sup>.

Studies show MCL-1-dependent cell growth in liquid (leukemia, lymphoma, myeloma) and solid tumors (breast and lung cancers<sup>2</sup>).

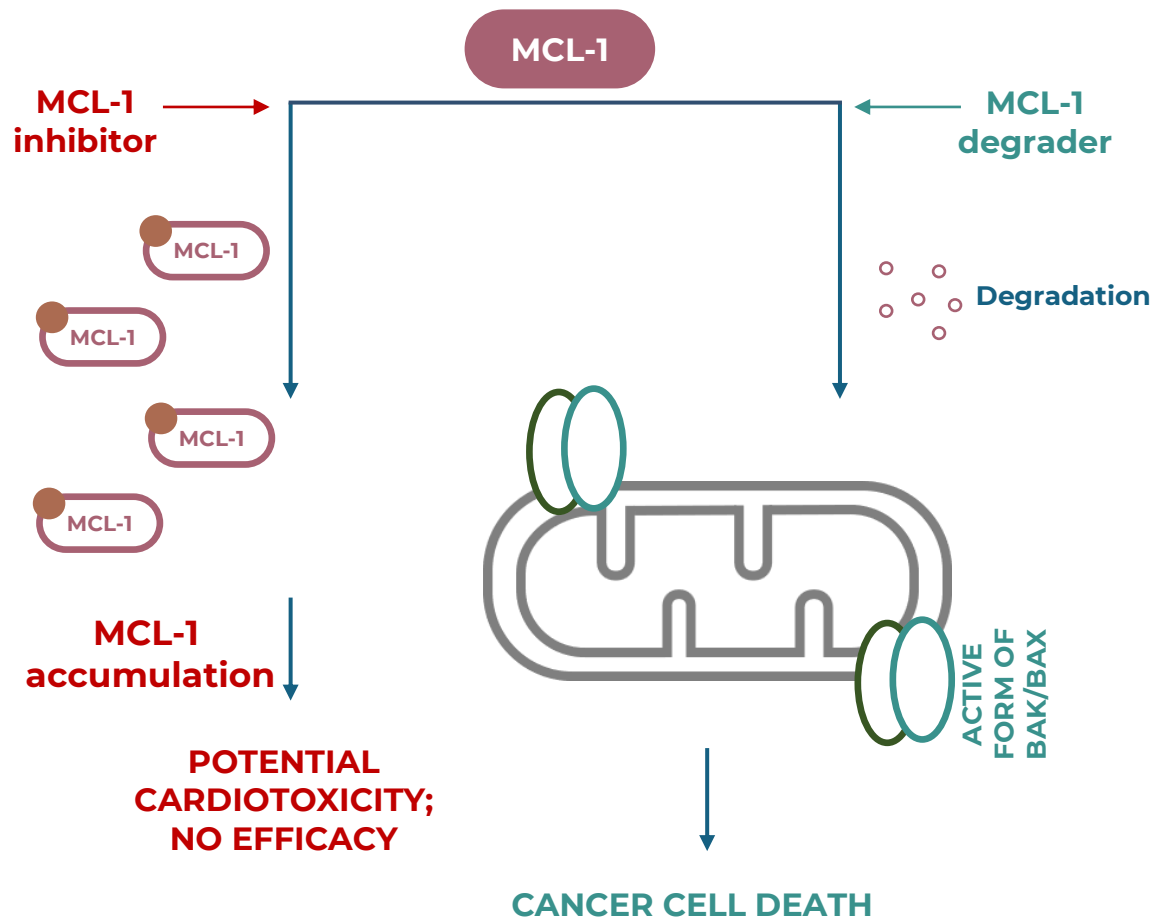
Monoallelic KO of MCL-1 in mice is viable, does not show signs of cardiac damage<sup>3</sup> or a gross phenotype, and shows resistance to selected liquid tumors.

Numerous systemic and targeted therapies drive the clonal selection of cells towards increased levels of MCL-1, like in AML refractory to venetoclax<sup>4</sup>.

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 cells<sup>1</sup>.

As a result of this 5- to 12-fold MCL-1 accumulation, adverse events occur<sup>2</sup>.

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.**

1. Singh R et al. Nature Reviews, 2019; 20: 175-193  
2. Kotschy A et al. Nature, 2016; 538(7626): 477-482

# Over \$100 billion 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 2030<sup>1</sup>

Acute Myeloid Leukaemia

\$6B by 2028<sup>2</sup>

Non-Hodgkin Lymphoma

\$16B by 2032<sup>3</sup>

## Selected solid tumors

Small cell lung cancer (SCLC)

\$6.5B by 2031<sup>4</sup>

Non-small cell lung cancer

\$36.9B by 2031<sup>5</sup>

Triple-negative breast cancer

\$1.5B by 2030<sup>6</sup>

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

<sup>1</sup>Allied Market Research

<sup>2</sup>BCC Research

<sup>3</sup>Spherical Insights

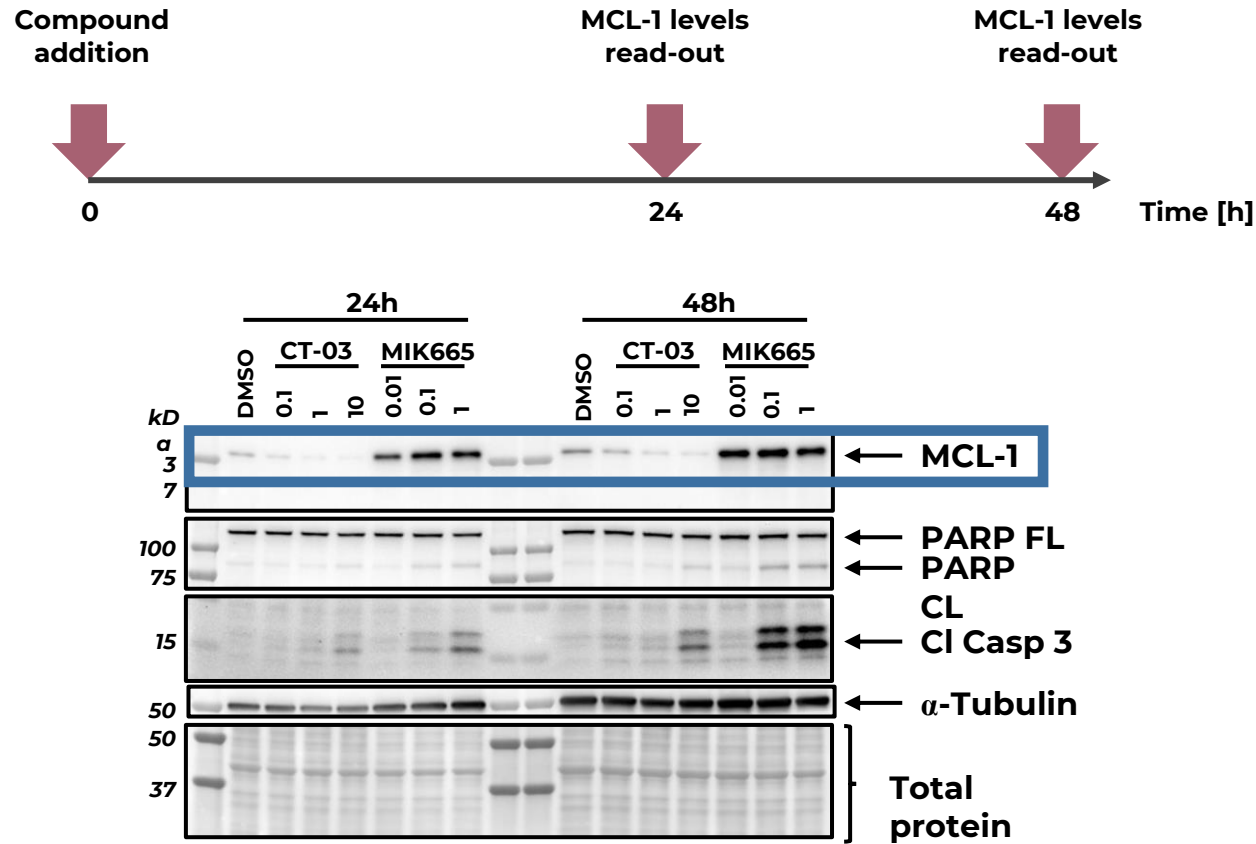
<sup>4</sup>HealthcareAnalyst

<sup>5</sup>Allied Market Research

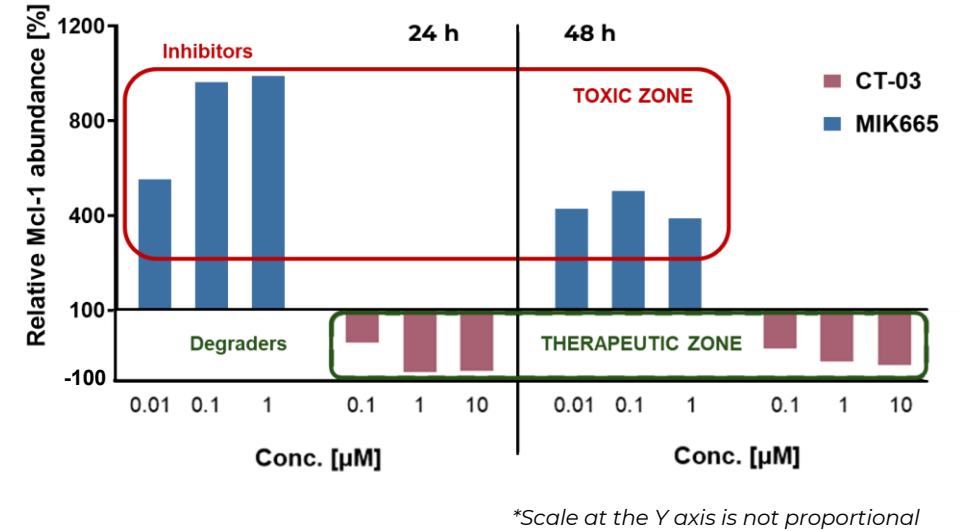
<sup>6</sup>Databridge Market Research

# MCL-1 degraders remain safe to healthy tissues

Inhibitors induce MCL-1 accumulation that is toxic to healthy tissues



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

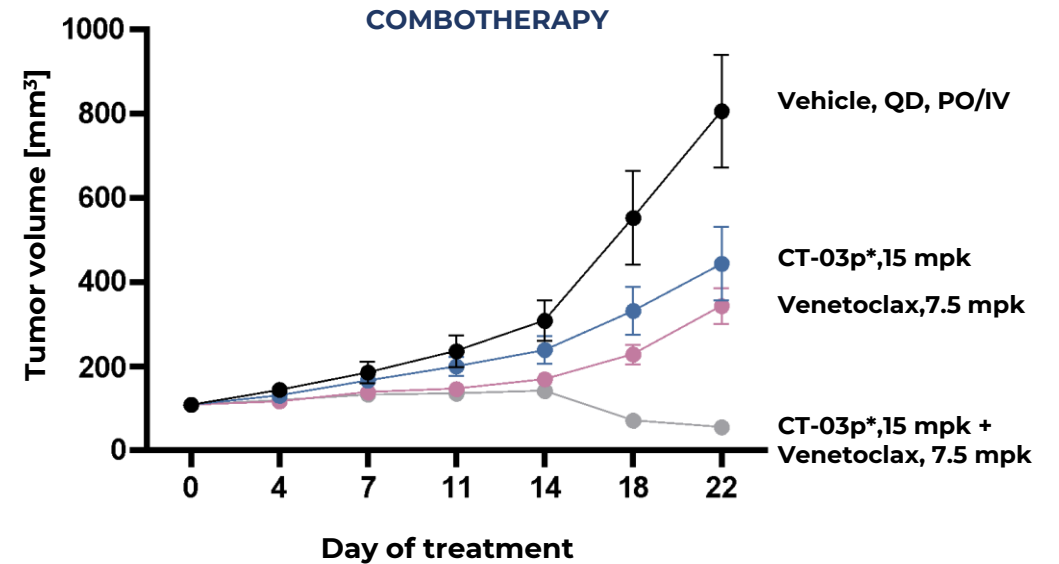
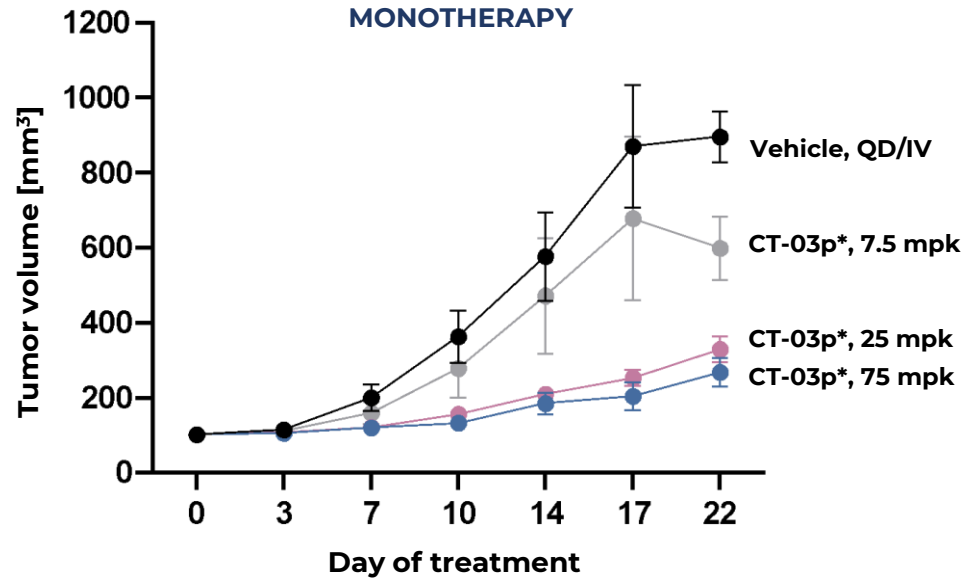


Inhibitors induce MCL-1 accumulation that persists even after compound washout

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



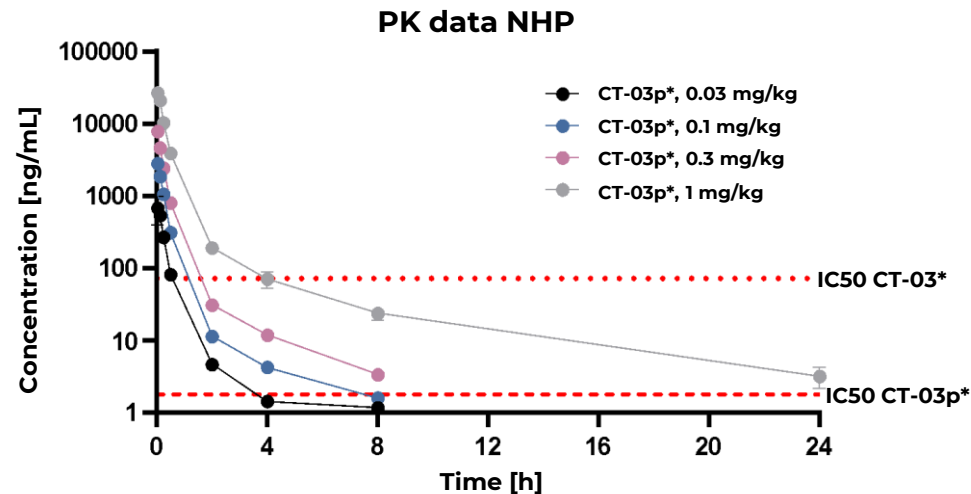
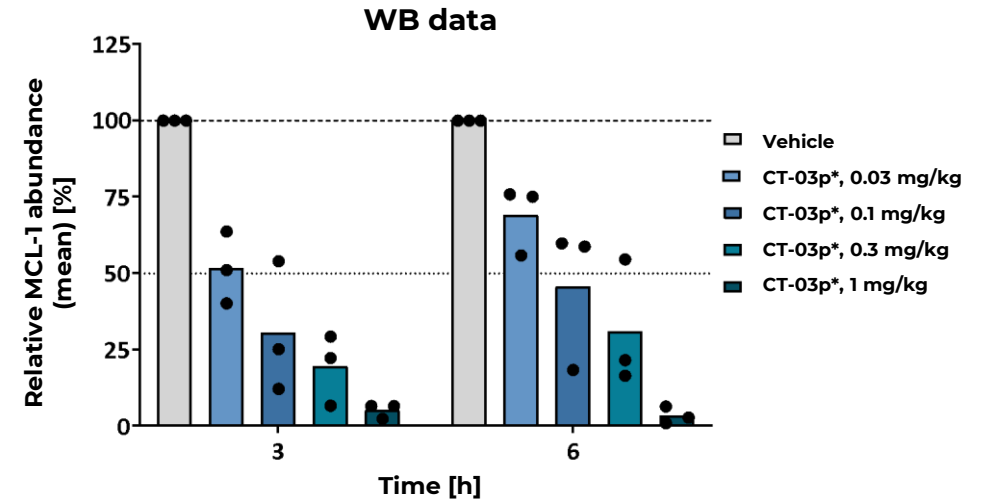
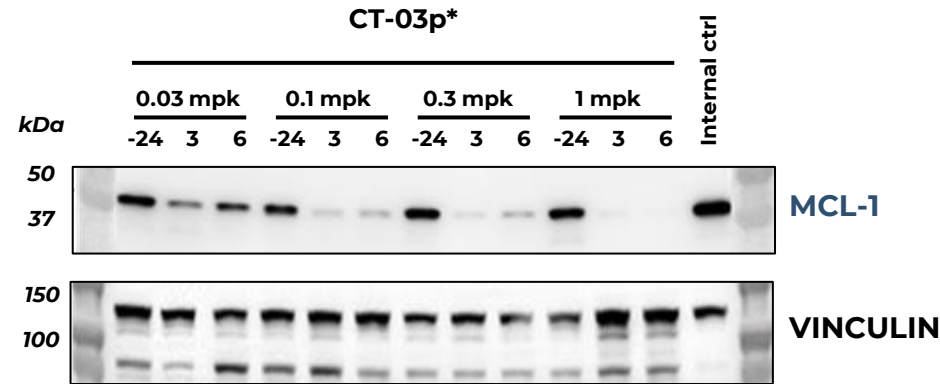
CT-03p\* - active isomer of CT-03p

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



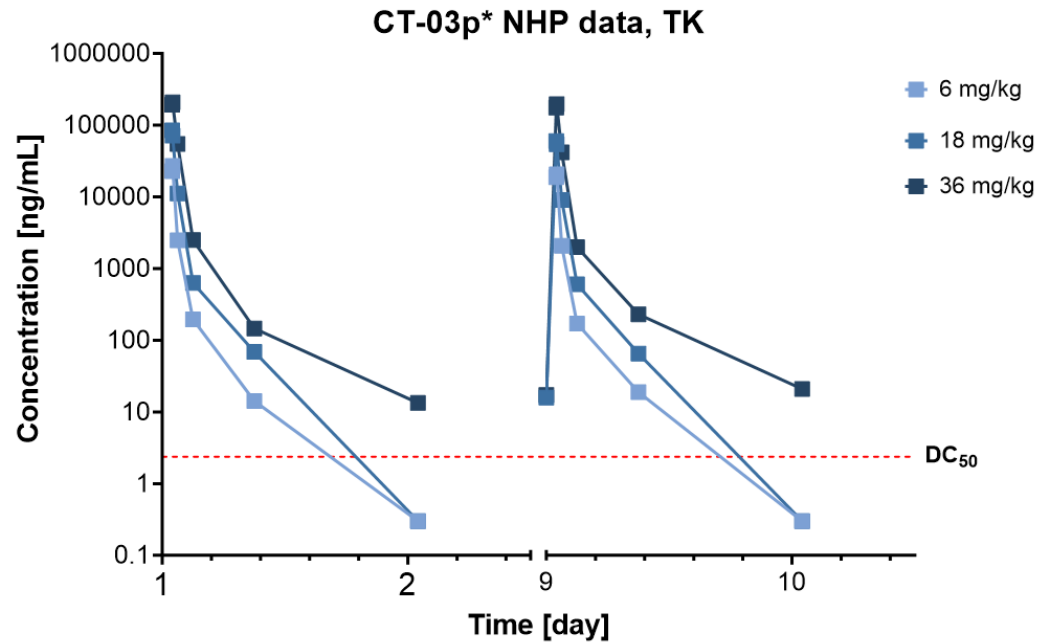
Rapid and dose-dependent degradation of MCL-1 observed in NHP after single IV administration

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

Supports translation of MCL-1 degradation to higher species

# Vratitoclax 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

# Vratitoclax: 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

# Differentiating MCL-1 degrader vs. MCL-1 inhibitors

## Clinical development of MCL-1 inhibitors limited by cardiotoxicity issues

- Literature & experimental data now demonstrate a MOA 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 cardiac safety issues were observed at doses 120-fold higher in monkeys than those required for DC50
  - Cardiac Troponin-I levels in monkeys are not affected by MCL-1 degradation
- Safety MTD/DRF study in monkeys showed no cardiotoxicity
- IND-enabling studies ongoing

# Investigator-Initiated Trial

## Press release dated November 27th, 2025:

Captor Therapeutics S.A. (WSE: CTX), an innovative biopharmaceutical company, today announced it has entered into a Memorandum of Understanding with The University of Texas MD Anderson Cancer Center to support an Investigator-Initiated Trial (IIT) for Captor's lead asset - an MCL-1 protein degrader for the treatment of hematological malignancies.

The clinical trial builds upon preclinical research conducted at MD Anderson by Michael Andreeff, M.D., Ph.D., professor of Leukemia, and Bing Carter, Ph.D., professor of Leukemia, in collaboration with Captor. Amongst the findings, the clinical candidate, CT-03p, was shown to decrease MCL-1 protein levels, induce death of leukemia cells, and synergize with BCL-2 inhibition in cells resistant to venetoclax, CT-03p, or both. Updated results from this research will be presented at the American Society of Hematology Annual Meeting December 2025.

Abhishek Maiti, M.D., assistant professor of Leukemia at MD Anderson, will serve as the trial's principal investigator.

# Summary: vratitoclax – a 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 data now demonstrate a MOA for inhibitor-induced toxicity
  - Protein degradation modality provides greater target coverage and is well tolerated
    - No cardiac safety issues were observed at doses 120-fold higher in monkeys than those required for DC50
  - MCL-1 degraders are synergistic with venetoclax using *in vitro* & *in vivo* AML models
- **Vratitoclax: a first-in-class opportunity**
  - 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 monkeys showed no cardiotoxicity
  - IND-enabling studies ongoing

# Possible further development

## Other indications:

- **triple negative breast cancer** (TNBC),
- **non-small cell lung cancer** (NSCLC),
- **colon cancer,**
- **venetoclax-resistant cancers.**

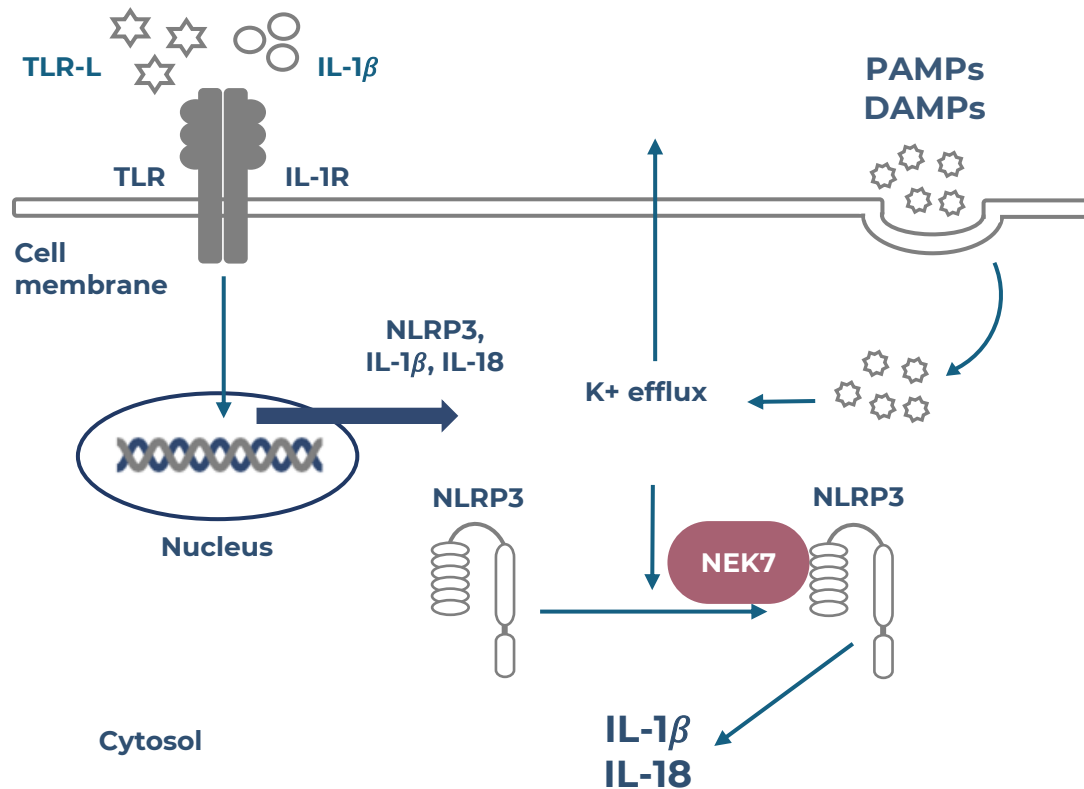
## Combotherapies – **vratitoclax** could possibly be combined with:

- **venetoclax** – *synergy has been proven in animal models,*
- **venetoclax and azacitidine,**
- **ibrutinib (BTK inhibitor)**
- **trametinib (MEK1/2 inhibitor)**
- **everolimus (mTOR inhibitor)**

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

---

# NEK7 as a new target of the NLRP3 inflammasome pathway



## 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<sup>+/-</sup> mice show no internal anatomical or growth abnormalities.

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

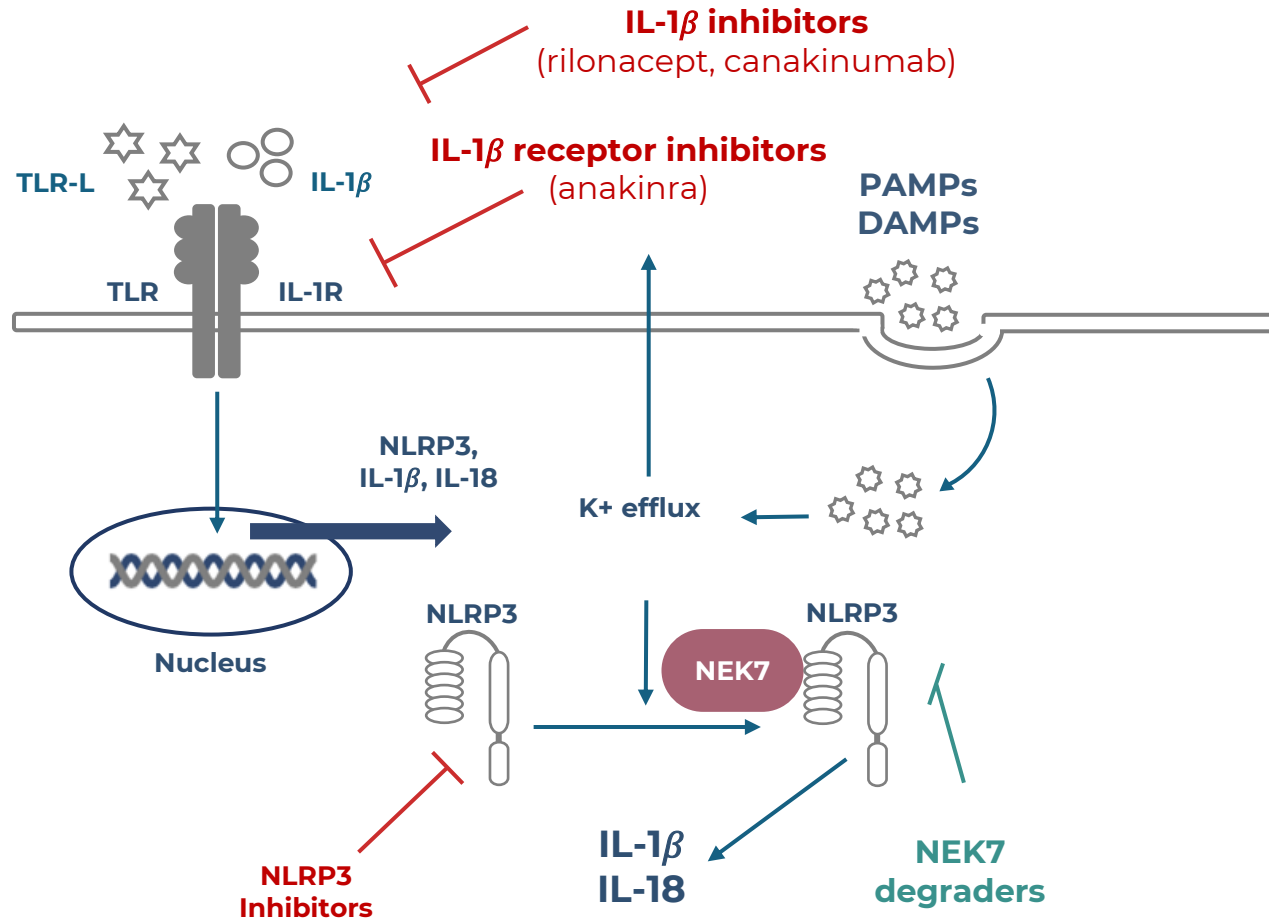
CAPS syndromes (FCAS, MWS, NOMID)

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\*.

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 as a new target of the NLRP3 inflammasome pathway



## Differentiation

### From anti-IL-1 $\beta$ 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

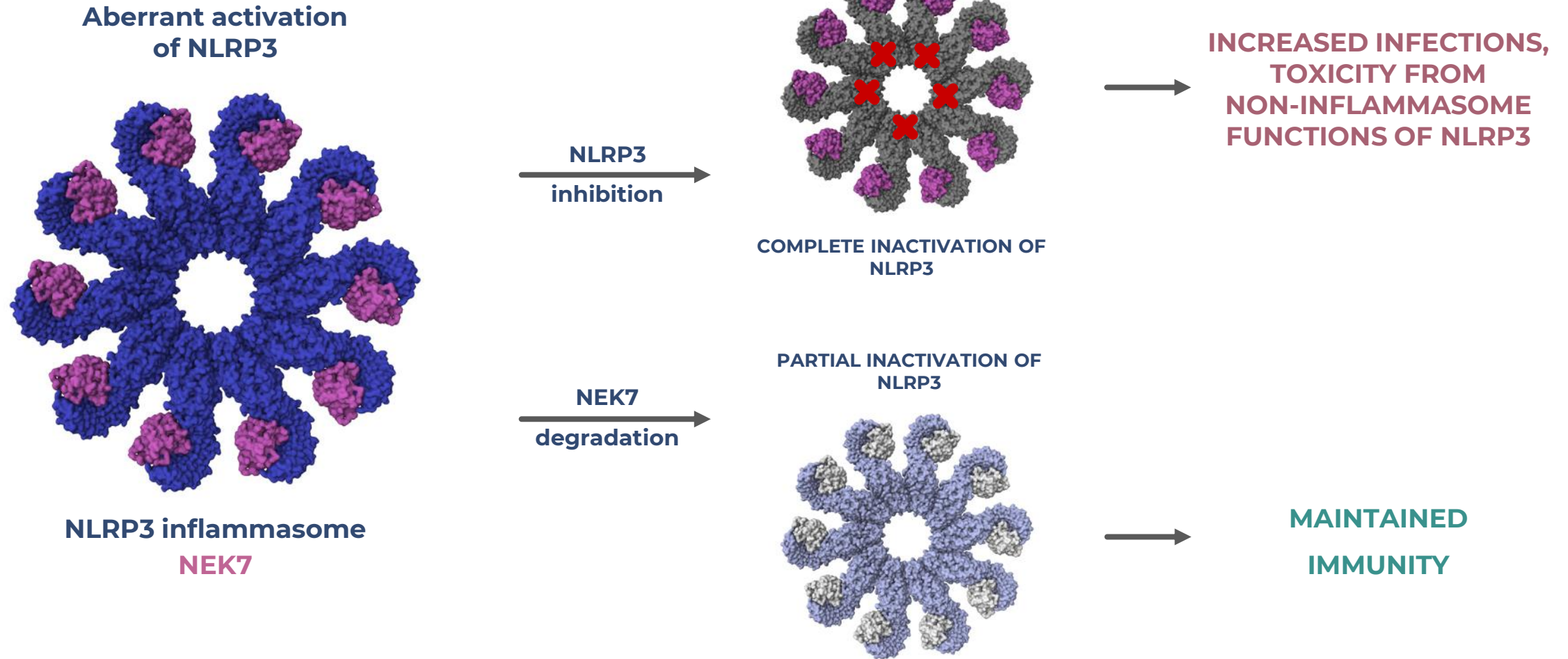
Complete IL-1 $\beta$  shutdown potentially manifests in recurring infections

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)

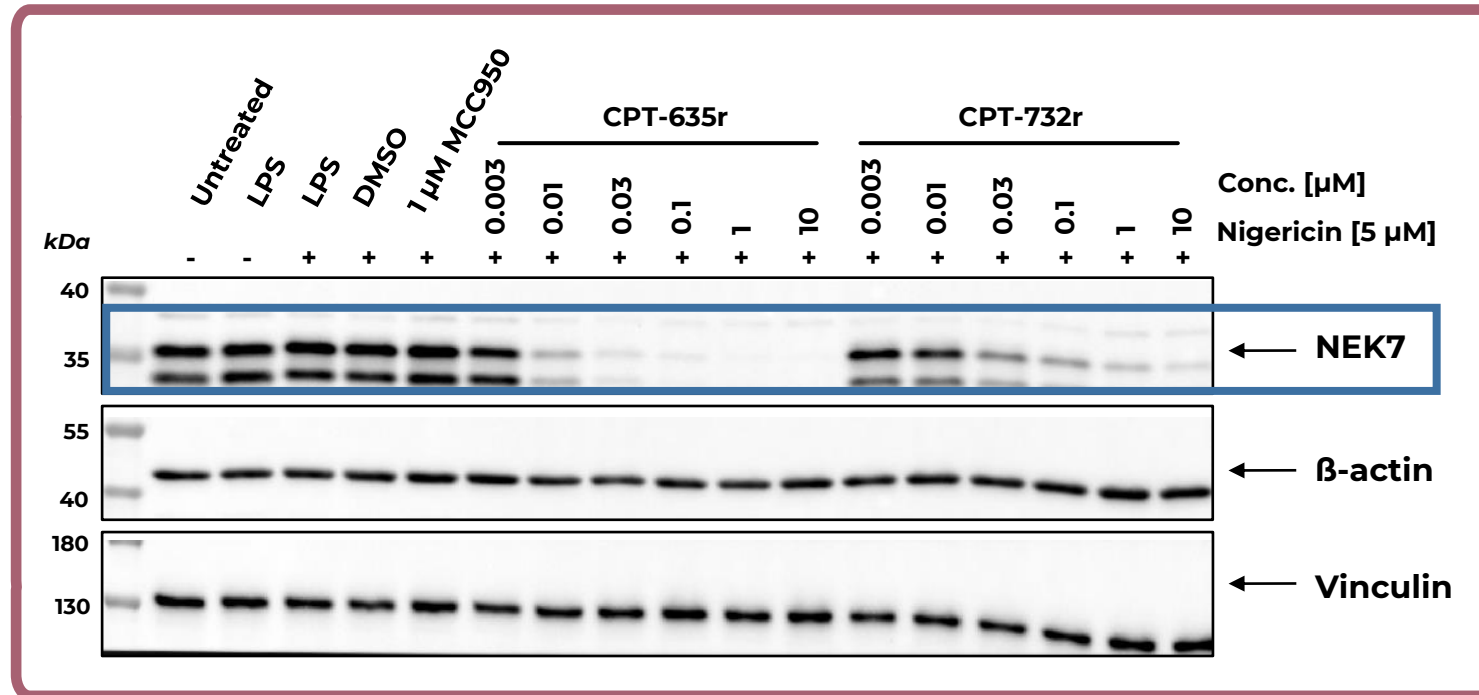
1. Molina-Lopez et al; Nature Communications, vol 15, (2024); <https://www.ema.europa.eu/en/medicines/human/EPAR/ilaris>  
2. <https://www.ema.europa.eu/en/medicines/human/EPAR/kineret>  
\*own results conducted by Captor Therapeutics

# NEK7 degradation as a differentiated intervention in NLRP3 pathway

Degradation of NEK7 provides therapeutic benefit and maintains basal immunity



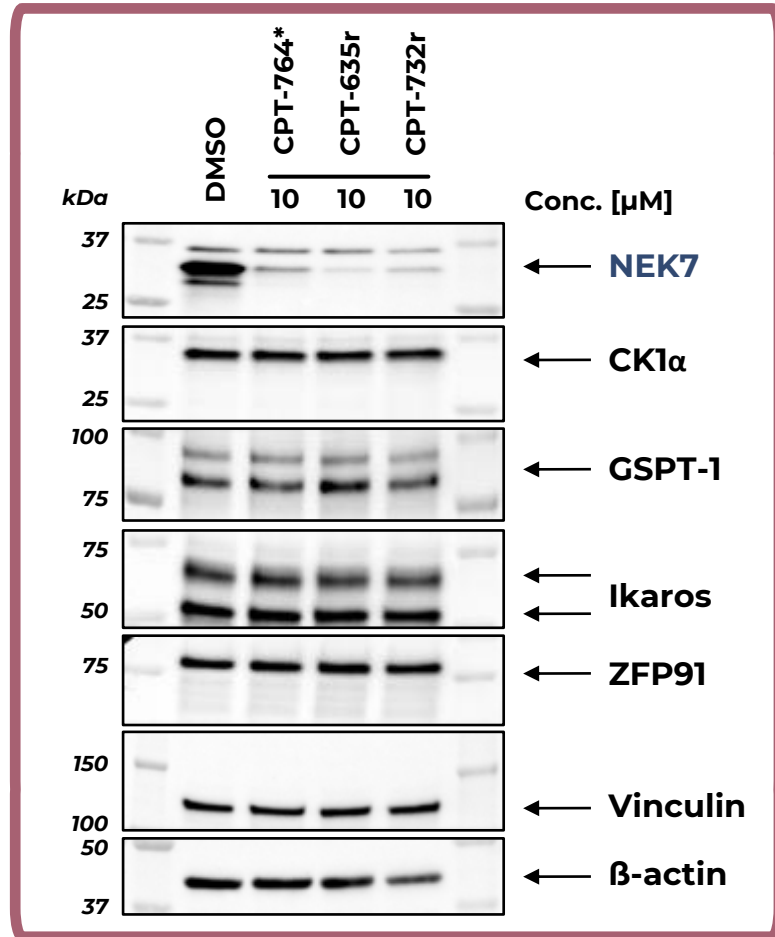
# Potent degradation of NEK7 in human macrophages *in vitro*



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

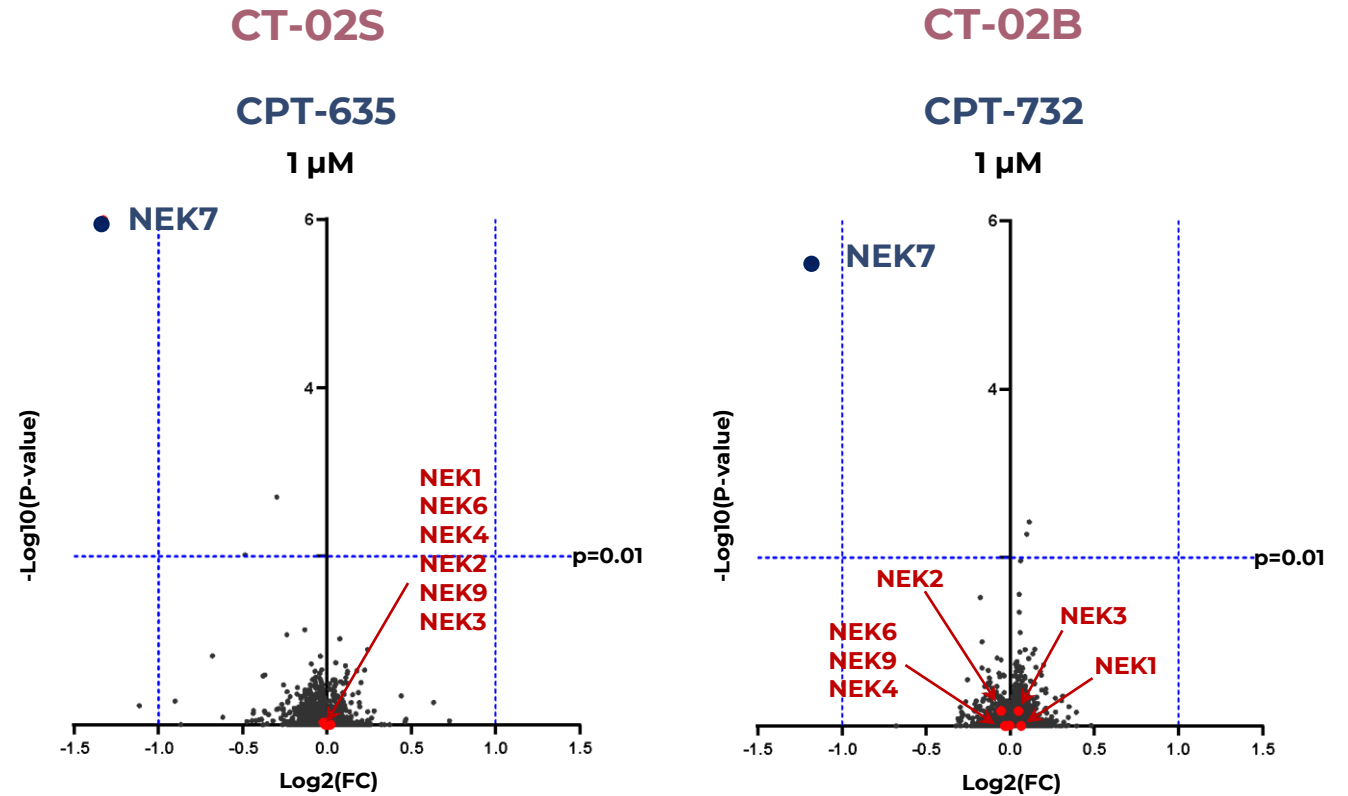
Human PBMC differentiated into macrophages with M-CSF; treatment with compounds – 24h; inflammasome activation: LPS – 3h, Nigericin – 1h  
MCC950 – NLRP3 inhibitor (Roche/ Inflazome); CPT-635r – racemate of CPT-635, CPT-732r – racemate of CPT-732

# High selectivity of NEK7 molecular glue degraders



\*Early lead compound

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

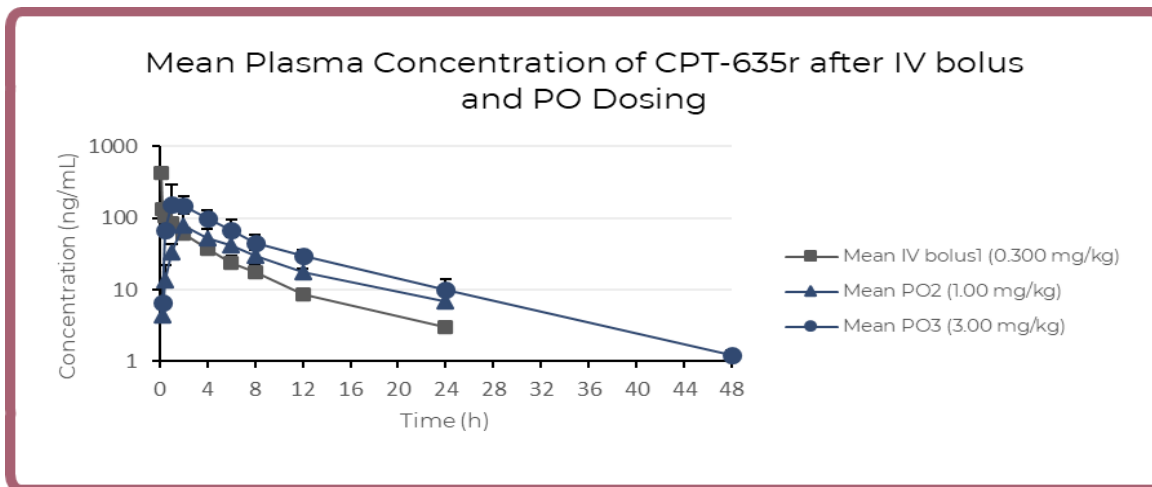
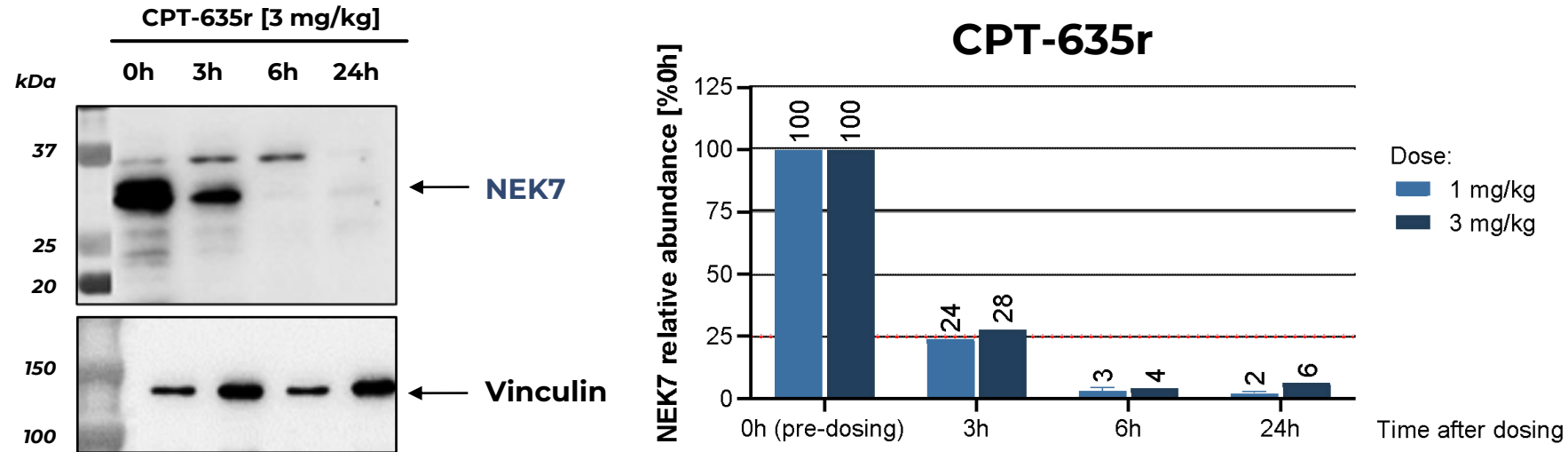


Confirmed degradation of NEK7 in hPBMCs  
No off-targets, even at high concentrations

CT-02 (NEK7)

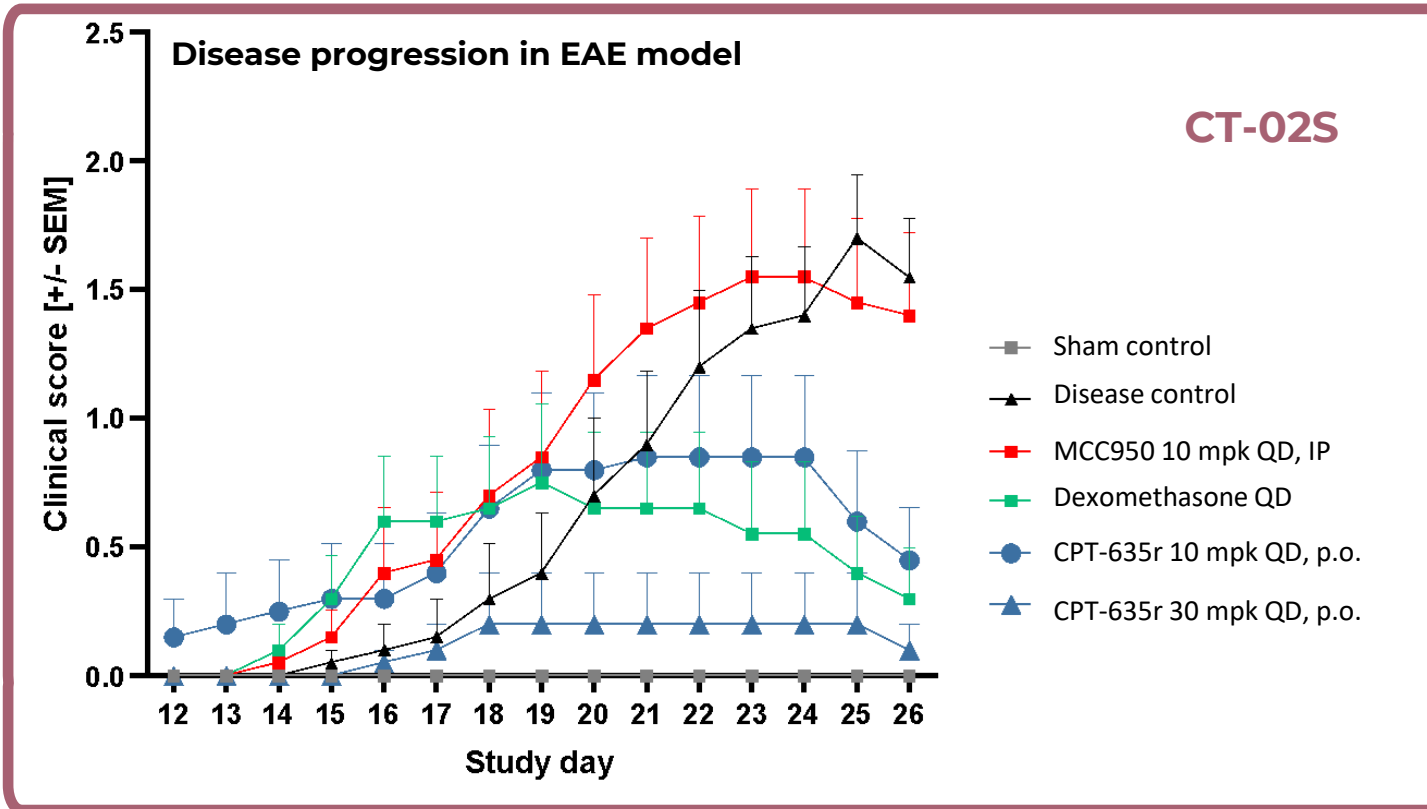
# CPT-635r efficiently covers & degrades NEK7 in NHPs after a single dose

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

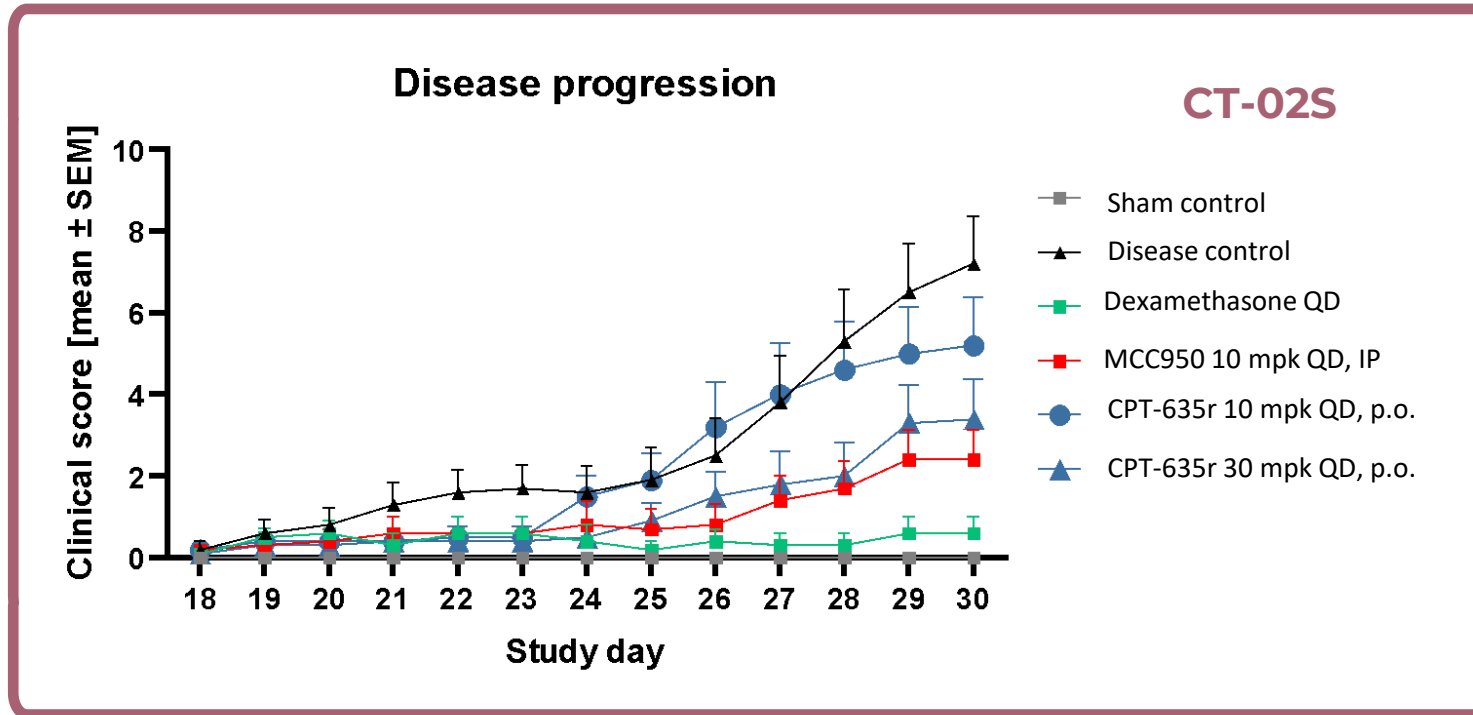
Treatment for 16 days did not induce any side effects

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

MOG<sub>35-55</sub> 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

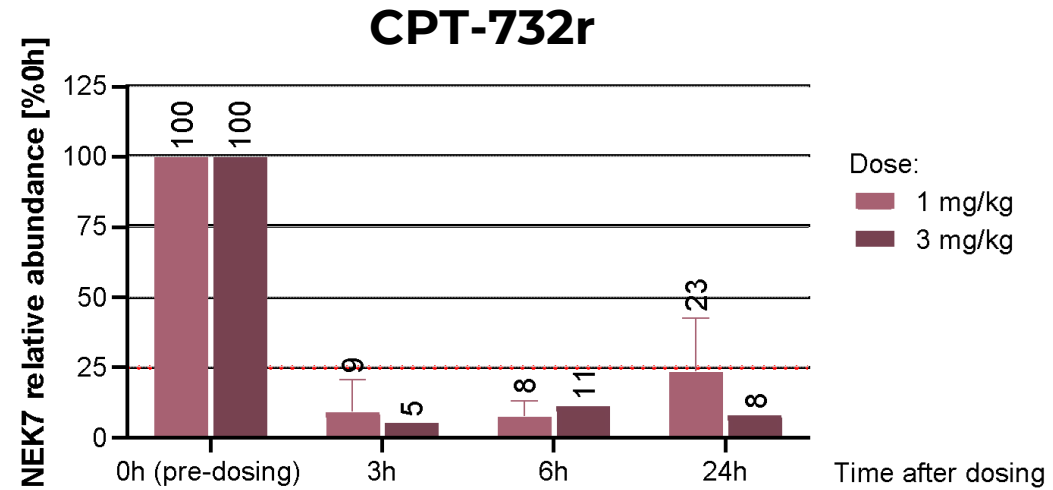
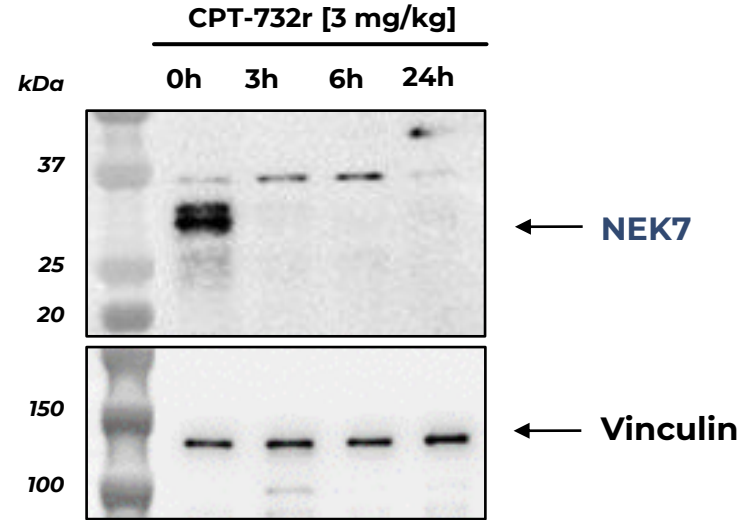
Treatment over 32 days did not induce any side effects

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)

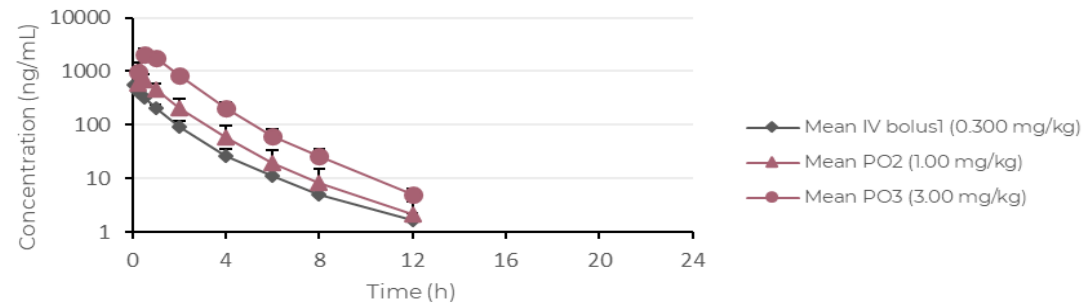
T/B-cell driven; Collagen-Induced Arthritis

# 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

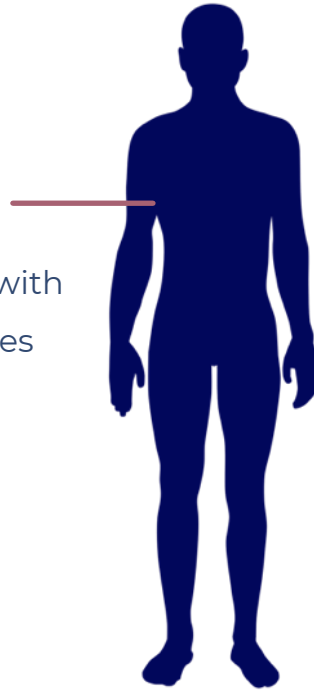
**CT-02S**  
CPT-635

## 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

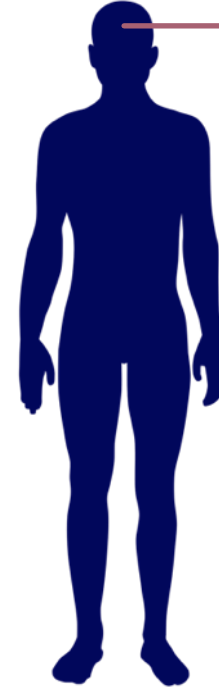
# CT-02B: Breakthrough for Neurodegeneration

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**

# Research Collaboration and License Option Agreement

Captor (CTX) signed agreement with a U.S. public pharmaceutical company

CTX granted a paid, time-limited exclusivity to conduct research on the Captor's proprietary NEK7 degraders in Partner's animal models

Granting satisfactory research results, the Partner will have an option to enter into an exclusive license agreement.

Captor may be entitled to:

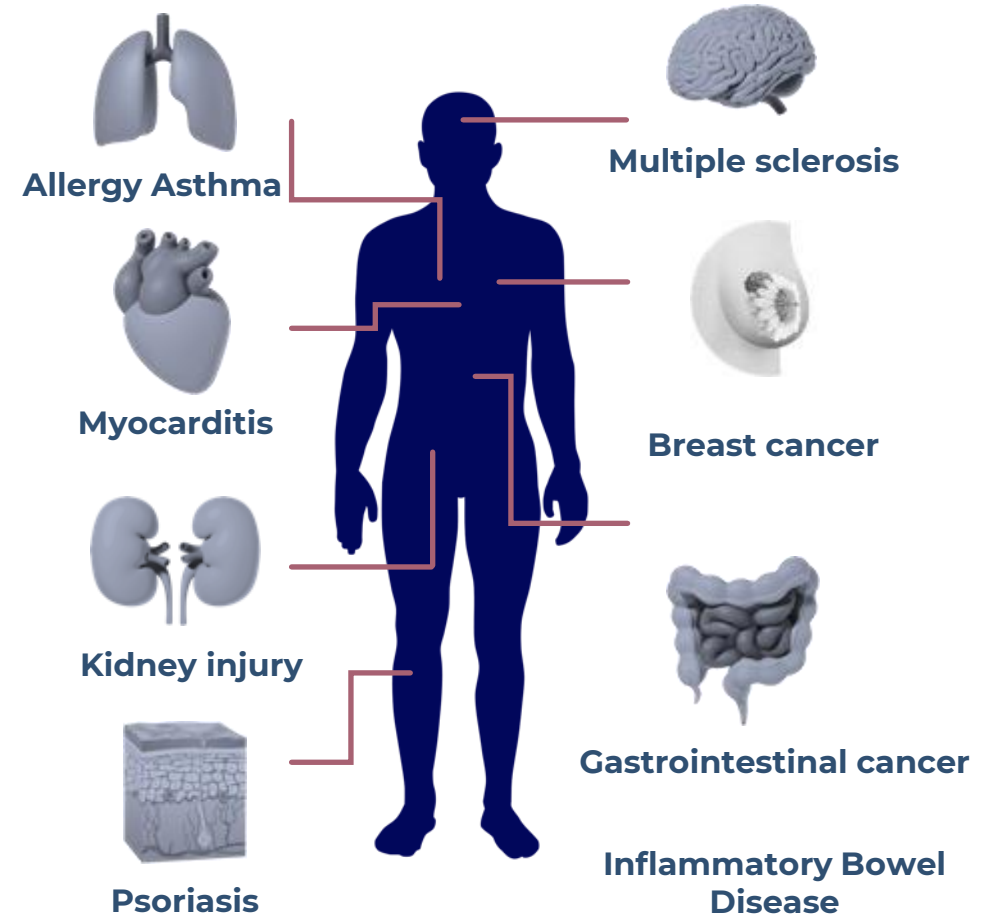
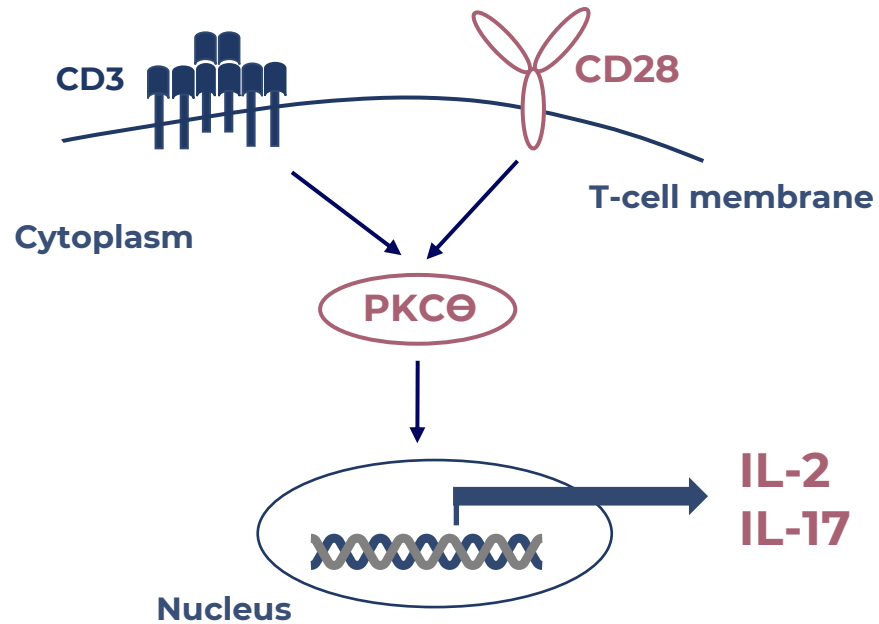
- Receive an upfront payment in the amount of a "mid-single digit million USD".
- Receive development and commercial milestone payments totaling up to a "triple-digit million USD".
- Receive royalties on future net sales of products based on the license.

# CT-05: First-in-Class PKC $\theta$ Degraders for Autoimmune Disorders

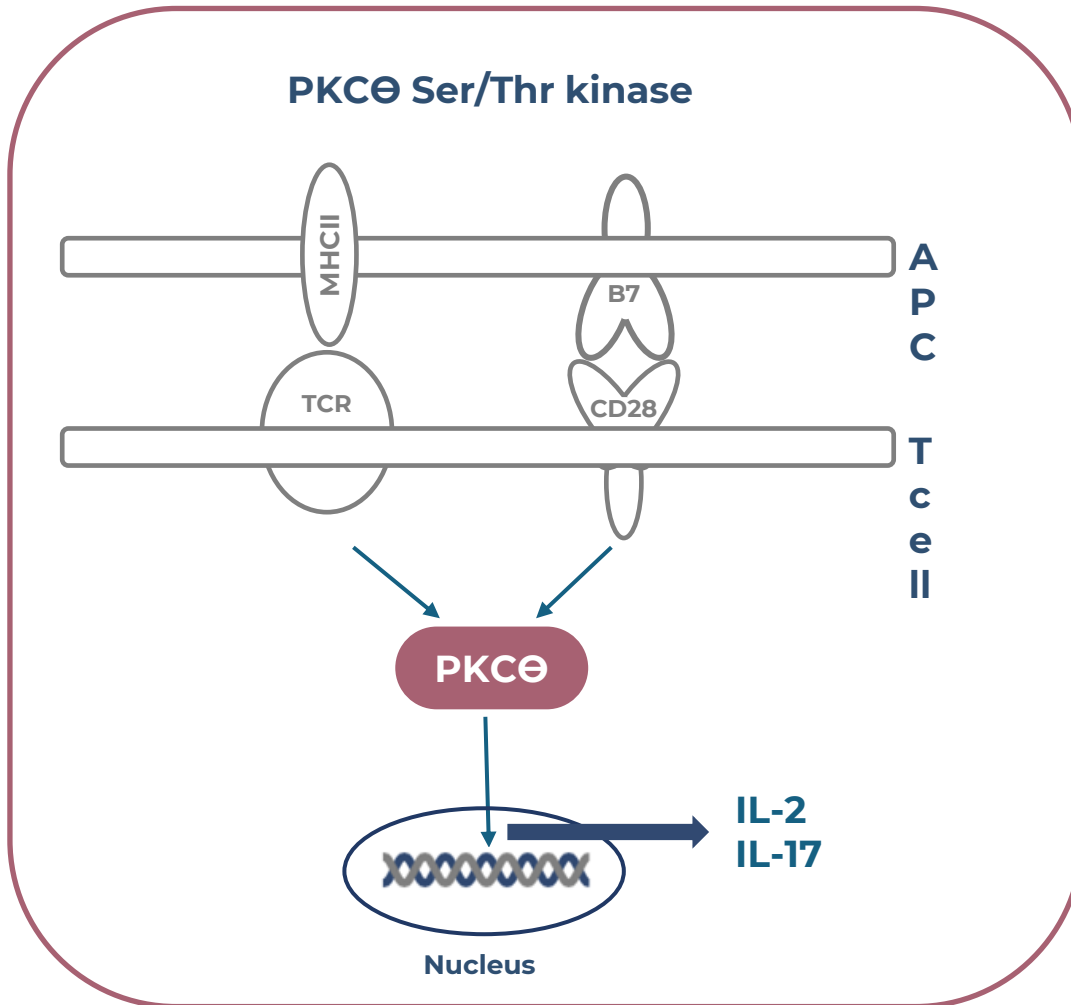
---

# PKC $\theta$ : an undrugged high value target

TCR



# PKC $\theta$ Biology and target rationale



## Target Biology and rationale

PKC $\theta$  has a thoroughly established role in regulatory and effector T cell functions<sup>1,2</sup>

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

## Human and mouse genetics

PKC $\theta$  KO mice show impaired *in vivo* T cell activation, decreased IL-17 production and are protected from T cell-mediated inflammatory diseases (EAE, colitis)<sup>7,8</sup>

## Clinical pathway validation

PKC $\theta$  inhibitor – Sotrastaurin (AEB071) – has been shown effective in preventing IL-17 production and to have a potential for therapeutic option in psoriasis<sup>9-11</sup>

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- $\theta$  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  $\theta$ -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 $\theta$ degrader as an antibody-like activity in a pill

Established a screening workflow that allows for discovery of PKC $\theta$  degraders superior to existing inhibitors

Highly selective for PKC $\theta$  with no off-target toxicity

Early stage of lead optimisation with 2 compounds has demonstrated:

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

*In vivo*: degradation of PKC $\theta$  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	
<b>Stage</b>	<b>GID4</b> Ligands available & degraders tested	<b>Ligase A*</b> PROTACability confirmed	<b>KLHDC2</b> PROTACability confirmed	<b>Ligase B</b> Fragments & X-ray structures	<b>Ligase C</b> Fragments & X-ray structures
<b>Key feature</b>	SRS of an evolutionarily conserved, multi-subunit E3 complex (HMGCS1, ARHGAP11A, DDX17 – among endogenous targets)	Expressed in cytosol & nucleus, essential for some cancers; <b>Not expressed in liver</b>	Ubiquitously expressed, predominantly in the nucleus	Benign KO prevents autoimmune disease	Upregulated in solid tumors
<b>Known in TPD</b>	YES	NO	YES	NO	NO
<b>Status at Captor</b>	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

---

# Equity summary

Ticker: **CTX PW**

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

Shares outstanding: **6,353,397** (April 29, 2025)

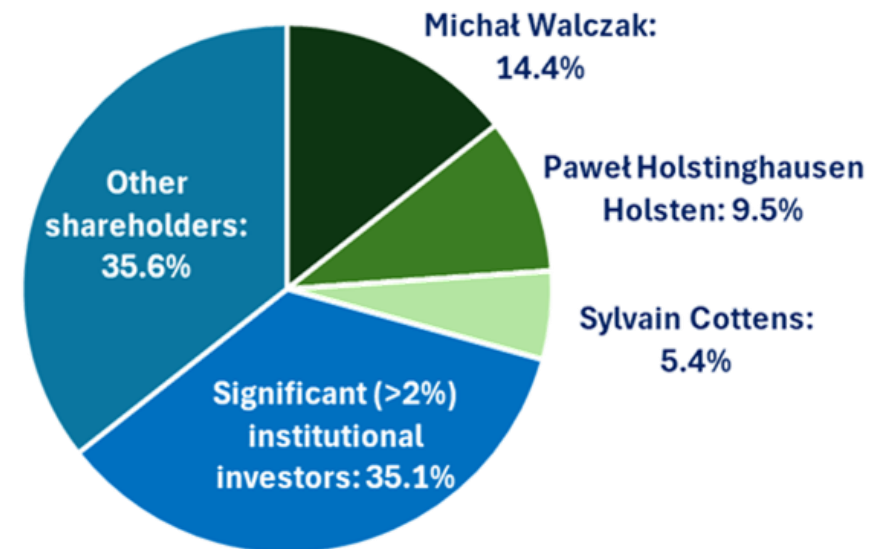
ISIN: **PLCPTRT00014**

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

## Top shareholders

Michał Walczak	Founder, CEO & CSO	14.4%
Paweł Holstinghausen Holsten	Early investor	9.5%
PTE Allianz Polska	Pension fund	7.9%
PTE PKO BP Bankowy	Pension fund	6.8%
TFI Allianz Polska SA	Mutual funds	6.8%
PTE Nationale Nederlanden	Pension fund	5.8%
Sylvain Cottens	Founder, Head of Chemistry	5.4%
PTE PZU	Pension fund	3.1%
Norway	Sovereign wealth fund	2.5%
PTE Generali	Pension fund	2.4%
Other shareholders		35.6%

Source: stooq.pl (April 29, 2026)



# Financial summary

## Income Statement and Balance Sheet, PLN mln

	2025	2024
<b>Revenues:</b>		
Collaboration revenue	4.7	15.8
Grant revenue	7.1	5.8
Other revenues (incl. financial)	4.5	1.1
	<b>16.3</b>	<b>22.7</b>
<b>Costs:</b>		
Third-party services	29.7	29.8
Salaries and employee benefits	19.8	21.1
Depreciation (incl. from leasing)	4.4	5.1
Materials and energy	3.1	3.7
Other costs (incl. financial)	0.7	1.5
	<b>58.3</b>	<b>61.1</b>
<b>Loss</b>	<b>42.0</b>	<b>38.4</b>
<b>Share issues</b>	<b>32.9</b>	<b>0.0</b>
<b>Cash &amp; investments EOP</b>	<b>32.1</b>	<b>39.3</b>

## Highlights:

- Captor has so far been financed primarily through equity, secondarily through grants, and thirdly through revenues from research collaborations.
- In March 2026, we carried out a share issue, after which our cash runway was extended to Q3 2027.
- Operating in Poland allows us to achieve Western standards at low costs.
- Cash burn is reduced by Polish and EU grants.

# Recent corporate developments

---

# EIC Accelerator grant

The most recent grant agreement:

- ✓ 2.5 mln EUR in an EU programme EIC Accelerator,
- ✓ one of 71 companies chosen from 1,211 applicants from across Europe,
- ✓ one of 12 biotechnology companies,
- ✓ financing the clinical trial of vratitoclax in combination with venetoclax, in R/R AML.

# New employee share option programme

Participants in the program will receive share options:

- ✓ for 220,000 new shares,
- ✓ exercisable at a price of **PLN 172** per share (closing price on the announcement date: **PLN 36.10**),
- ✓ exercisable in early October 2028,
- ✓ exercisable only by individuals who will work at Captor until the end of August 2028.

# Share issue

Recent share issue:

- ✓ 800,000 shares were issued in March 2026 (dilution of 14.5%).
- ✓ PLN 65.6 mln raised extends cash runway until Q3 2027.
- ✓ Share issue was oversubscribed and sold at less than 1% discount to market price.
- ✓ EIC Fund, an investment arm of European Commission, invested alongside existing shareholders.
- ✓ Two shareholders – local pension funds – reported exceeding 5% of the votes.

# Beyond the Pipeline

---

# Captor's Research in Print

Ligand-Induced Conformational Plasticity of the CTLH E3 Ligase Receptor GID4

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

A Robust Crystallographic Platform for High-Throughput  $\beta$ -Catenin Ligand Discovery

Ligand-Induced Conformational Plasticity of the CTLH E3 Ligase Receptor GID4

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



Captor  
Therapeutics<sup>®</sup>  
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 

Beta Catenin A Key, Yet Elusive Target in Oncology 

The Master Switch Resetting Autoimmunity with PKC  $\theta$  degraders 

# In the Spotlight

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



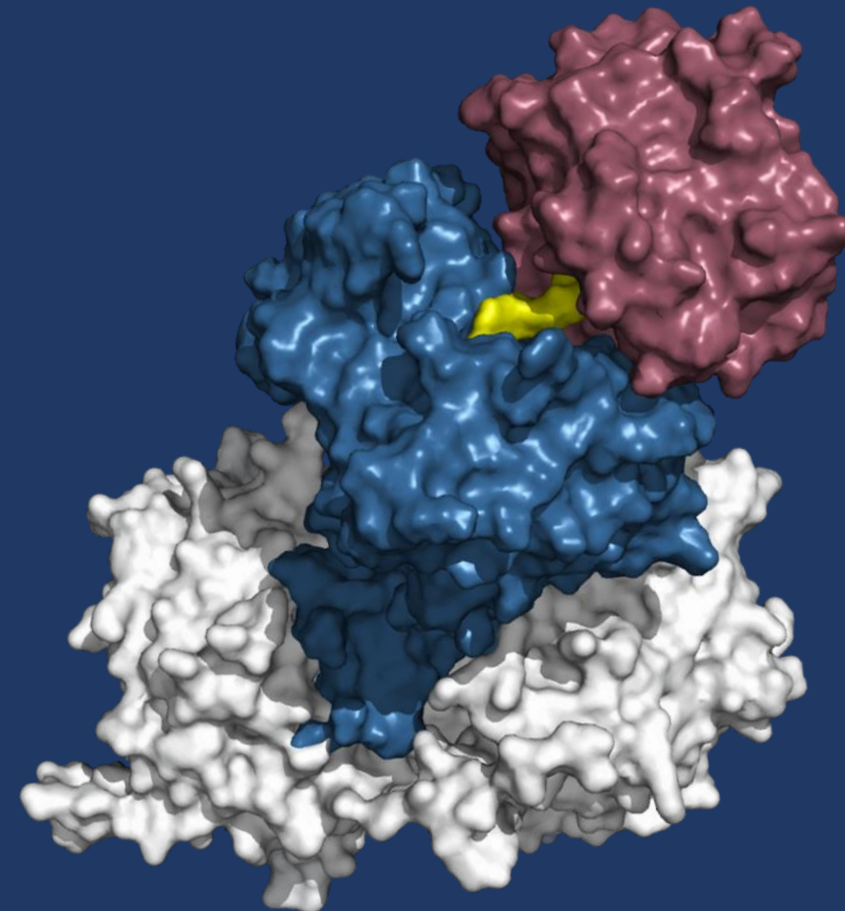
European Innovation Council (EIC) Accelerator grant for CT-03

European  
Innovation  
Council



Interview with Politykzdrowotna.com on discovering drug candidates in oncology





**Captor Therapeutics S.A.**



ul. Duńska 11  
54-427 Wrocław, Poland

**Captor Therapeutics GmbH**



Hegenheimermattweg 167A  
4123 Allschwil, Switzerland

Contact: [investor.relations@captortherapeutics.com](mailto:investor.relations@captortherapeutics.com)

## Projects co-financed by the European Union:

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)

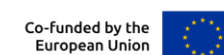
Inducing apoptosis with small molecules as therapeutic intervention in multiple severe malignancies – Stage II (FENG.01.01-IP.01-1002/23)

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)

A First-in-Class MCL-1 Degradator to Promote Apoptosis in Therapy-Resistant Liquid and Solid Tumours (Project no. 101218723)



## 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)

