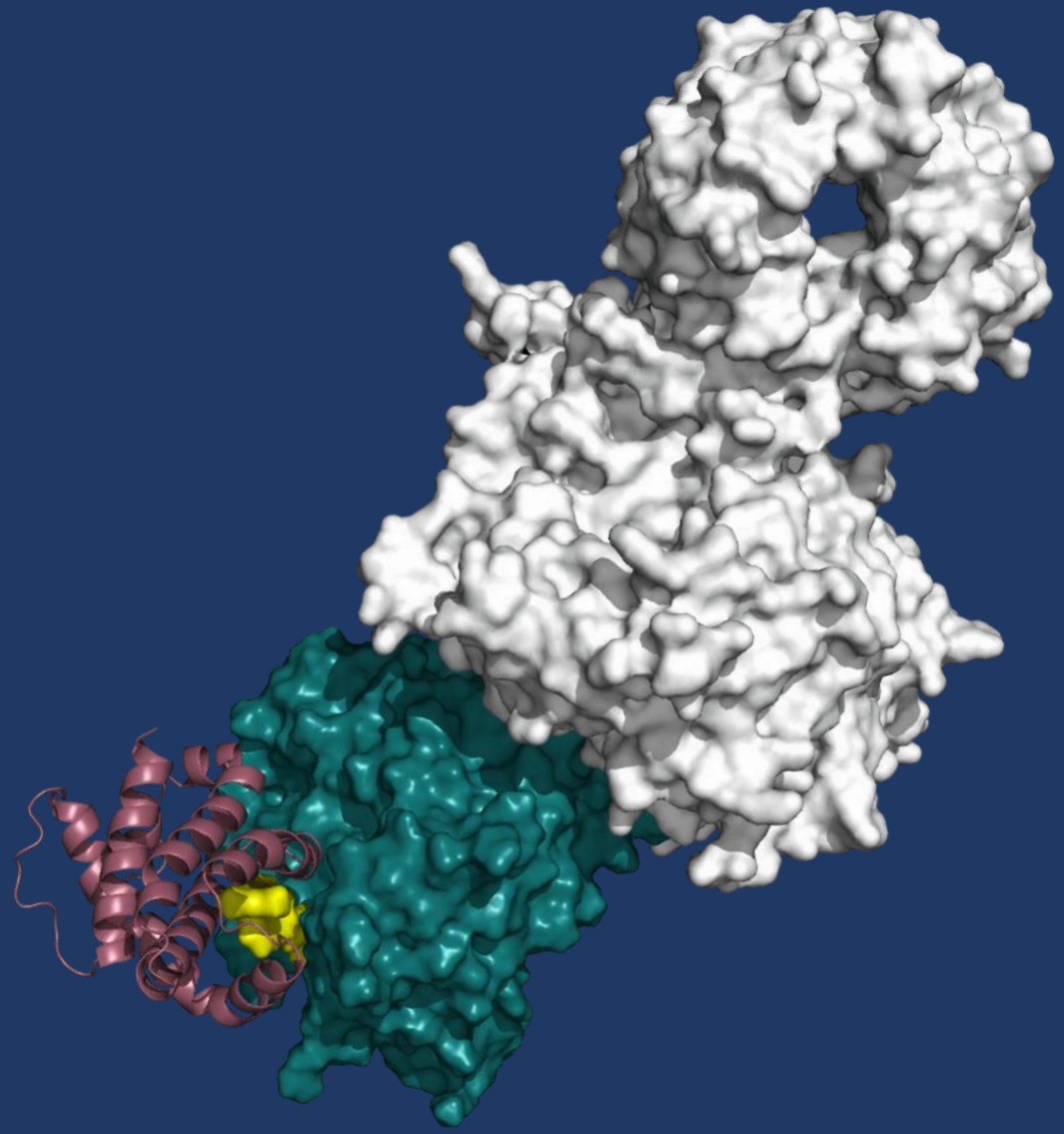
Captor Therapeutics®

pioneering targeted protein degraders for human health

October 2024



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®

Captor targets difficult-to-drug proteins for human health

Captor Therapeutics (WSE: CTX), Polish & Swiss company listed on Warsaw Exchange

- Focused on **Targeted Protein Degradation (TPD)** drug discovery and development
- Five fully-owned drug candidates in oncology and immunology
- Discovery of first- / best-in-class molecules characterized by prolific structure- and biophysics-enabled technologies

Multilple early clinical value inflection points over next 24 months

- Small molecule TPD candidates targeting **GSPT1**, **NEK7 & MCL-1 advancing into clinic in 2025/2026**
- **Positive preclinical proof of concept data and differentiated safety** across oncology, inflammation & neuroinflammation

*"Structure based drug design Provides a specific, efficient and rapid process for lead compound discovery and optimization. Researchers have discovered highly potent and selective molecular glues with SBDD..." ***

Capital sparing business model

- **~$30 million** in cash + unused grants as of 30 June 24
- Cash for operations **through 3Q25**
- >$52m in EU / Polish non-dilutive funding secured by Captor to date
- Raised $48m* in 2021 IPO & subsequent SPO (PIPE)

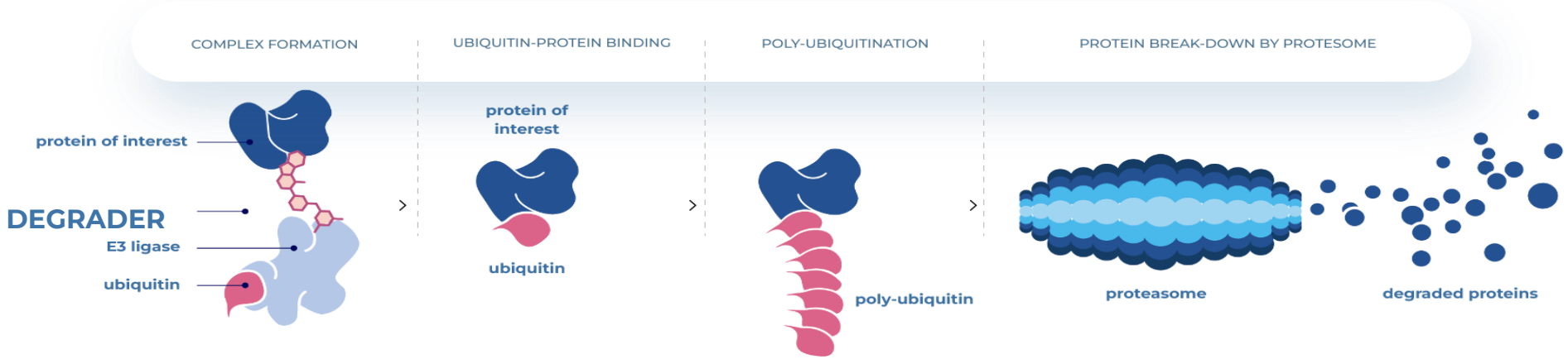
Management-controlled capital structure

*US$1: PLN3.96

Biochemistry 2023, 62, 601−623

Captor Therapeutics®

Targeted Protein Degradation set to unlock up to $974* bn by 2030



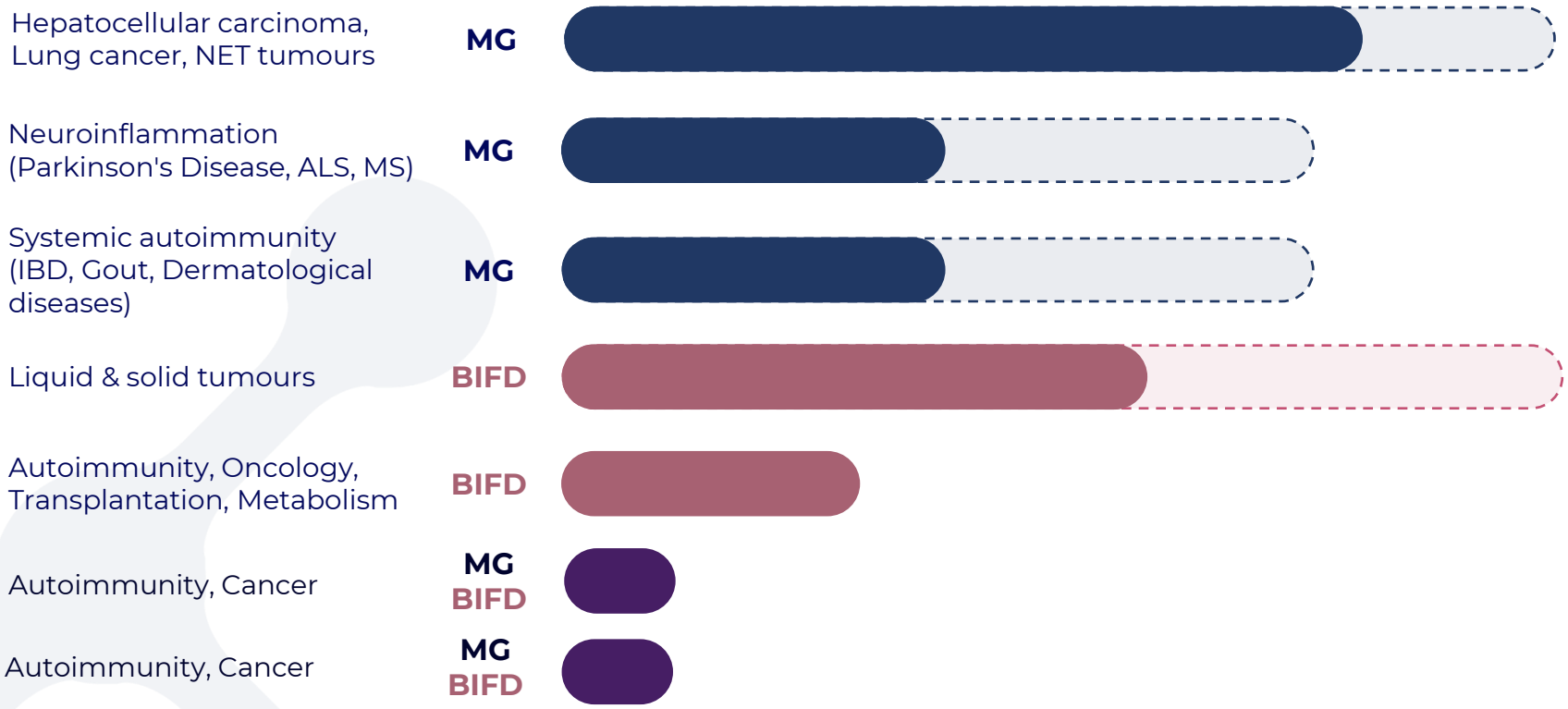
	Degraders	Inhibitors	mAbs	siRNA
Removing multiple pathological functions	✓✓✓	✗	✗	✓✓✓
Oral availability	✓✓✓	✓✓✓	✗	✗
Uncoupling PK from PD = prolonged efficacy	✓✓✓	✗	✗	✓✓✓
Overcoming mutational resistance	✓✓✓	✓	✓✓	✓✓✓
Targeting scaffolding function	✓✓✓	✗	✗	✓✓✓
Brain-penetration	✓✓	✓✓✓	✗	✗
Accessing undrugged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

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

Captor Therapeutics®

Wholly-owned pipeline

Programme	Primary Target	Indications	Modality
CT-01	GSPT1	Hepatocellular carcinoma, Lung cancer, NET tumours	**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 tumours	**BIFD**
CT-05	PKCϴ	Autoimmunity, Oncology, Transplantation, Metabolism	**BIFD**
New target projects		Autoimmunity, Cancer	**MG** **BIFD**
New E3 ligase degraders		Autoimmunity, Cancer	**MG** **BIFD**



†CT-02B - Brain-penetrant
‡CT-02S - Systemic

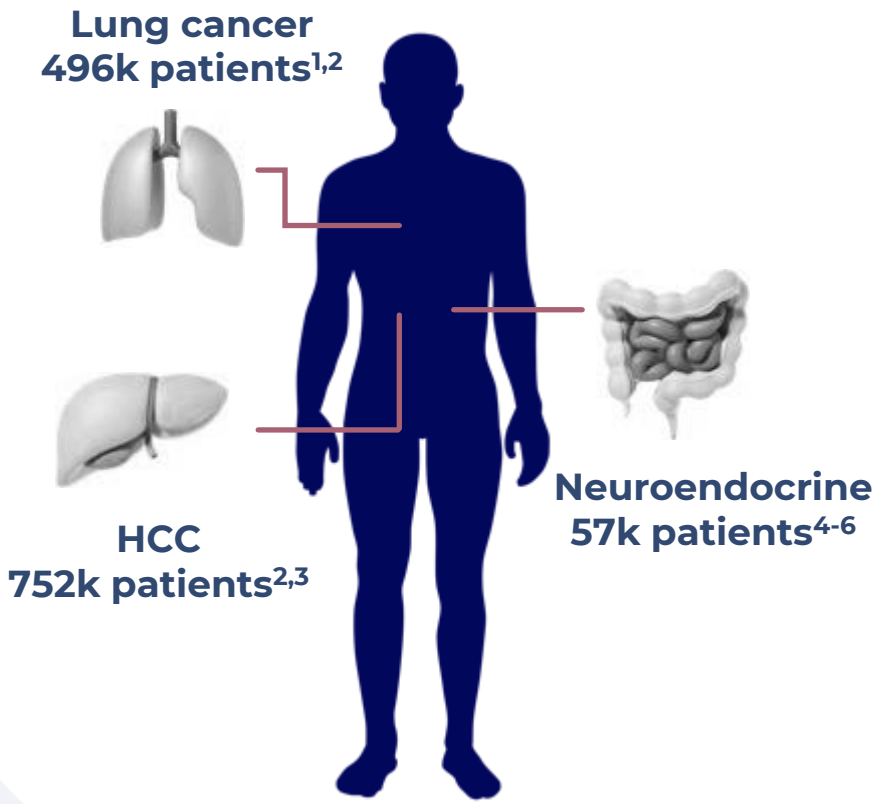
Preclinical stage include IND-enabling studies,* ***BIFD:* *Bi-functional Degrader;* ***MG:*** *Molecular Glue*

Projected stage at the end of 2025

Captor Therapeutics®

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

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



"Molecular glue" type targeted protein degraders tighten and simplify the connection of an E3 ligase with a disease-causing protein for ubiquitination and subsequent degradation

GSPT1 protein functions as a tumor promoter in human liver cancer cells. GSPT1 degradation leads to an Integrated Stress Response (ISR) and induction of apoptosis in hepatocellular carcinoma (HCC) cells.

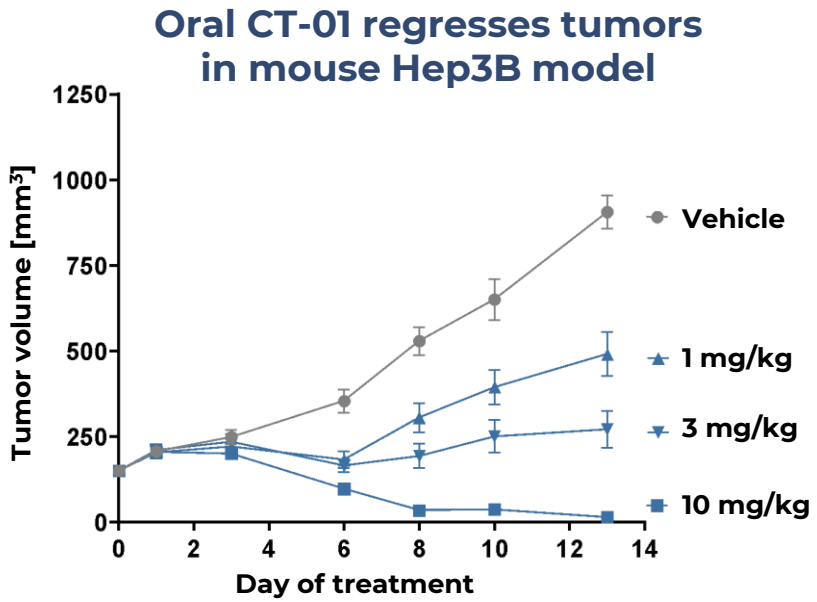
NEK7 degradation leads to reduction of IL-1β production – a well-established pro-carcinogenic factor. Reduction of IL-1β levels in the tumor microenvironment enables activation of the immune response. In addition, NEK7 is required for assembly and activation of NLRP3 inflammosome.

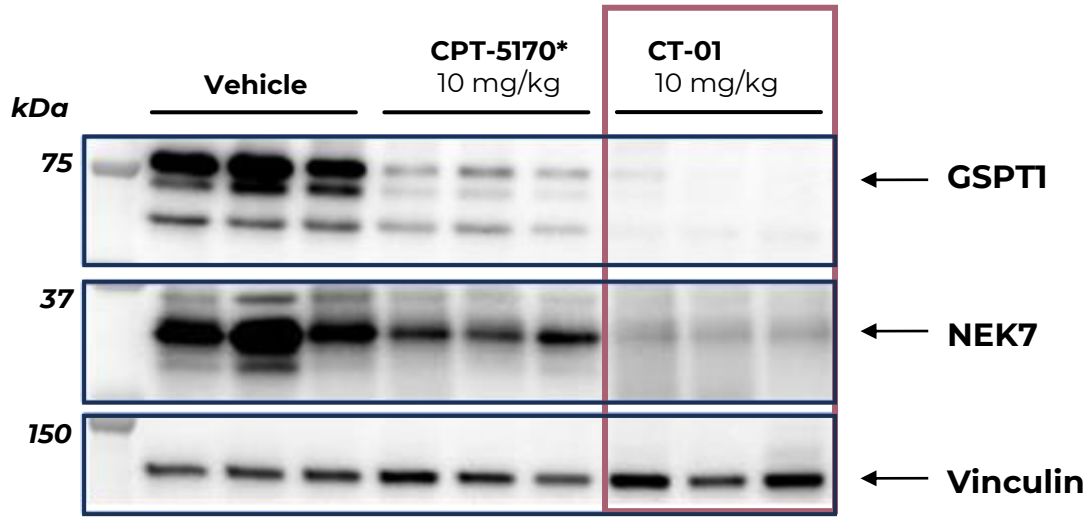
CT-01 is a tissue selective pro-drug; the small molecule orally available molecular glue degrader targeting GSPT1 and NEK7 is activated by an enzyme present at high levels in liver, lung and certain gastrointestinal tumors

(1) Semin Cancer Biol. 2006 Aug;16(4):253-64
(2) https://gco.iarc.fr/today/en/
(3) J Hepatol. 2022 Dec;77(6):1598-1606
(4) Semin Cancer Biol. 2006 Aug;16(4):253-64
(5) Endocr Connect. 2023 Nov 23;12(12)
(6) JAMA Oncol. 2017 Oct 1;3(10):1335-1342

Captor Therapeutics®

Highly potent oral CT-01 administration regresses tumors in mice





Regression of large tumors (~ 150 mm^2) at doses as low as 10 mg/kg BID administered orally from 6 days

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

Oral CT-01 strongly inhibits liver cancer growth in Hep3B model at all tested doses suggesting potent degradation of the target

Captor Therapeutics®

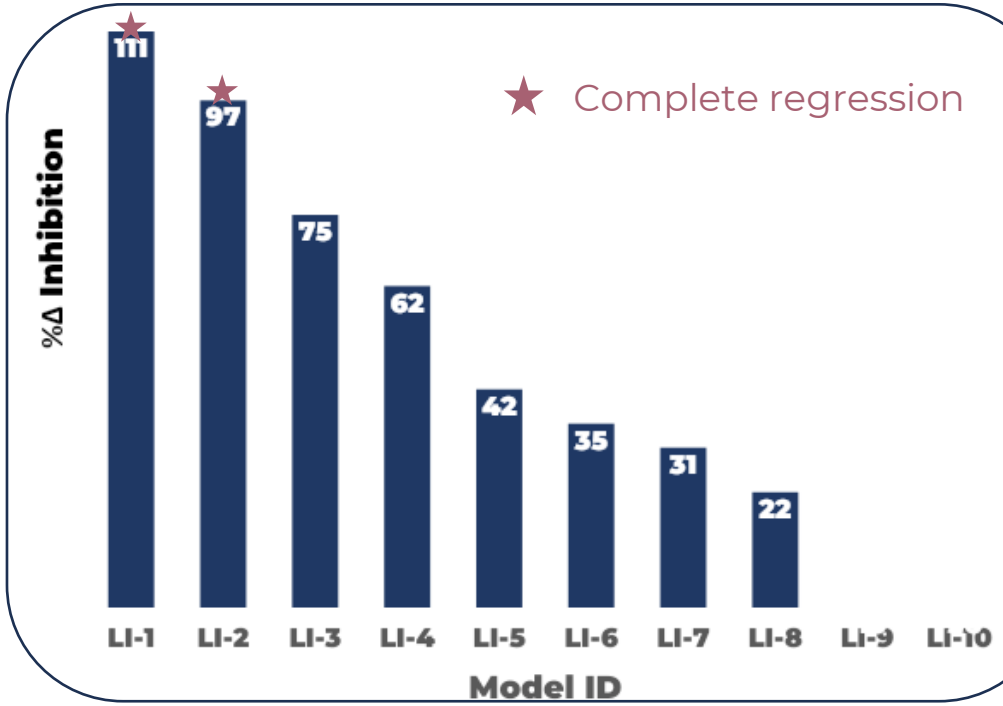
CT-01 inhibits human hepatocellular carcinoma (HCC) tumor growth in patient derived xenograft (PDX) mouse models

CrownBio
CONNECTING SCIENCE TO PATIENTS

PDX Models/

- 10 random HCC tumor samples
  - from different patients
  - with no drug target selection
- n=30
- **CT-01**, 100mg/kg, BID

Tumor Growth Inhibition



Each data point includes one tumor type in 3 PDX mice

Efficacy demonstrated in 8/10 PDX tumors;
tumor regression >50% in 4 HCC tumors - 2 tumors with complete regression

Captor Therapeutics®

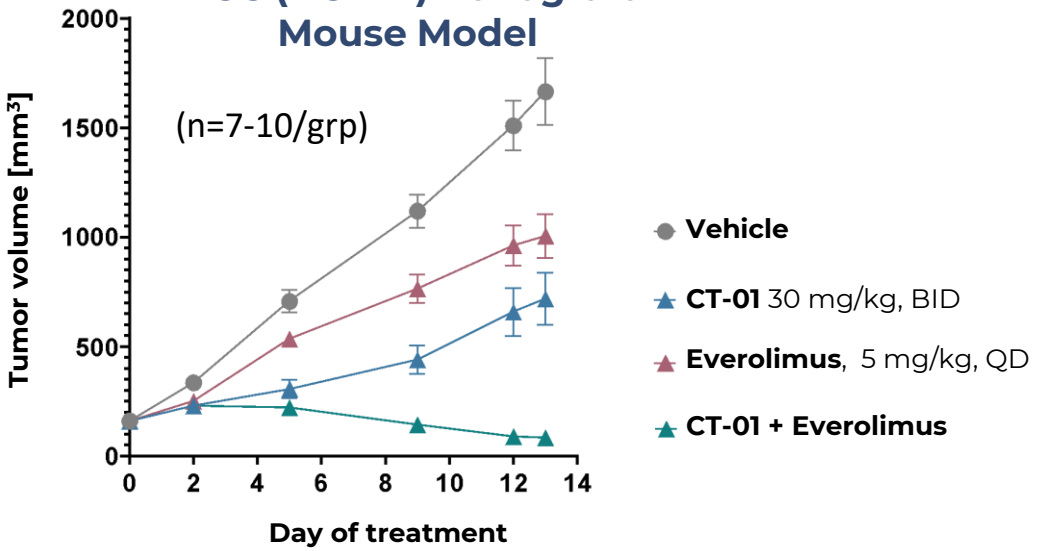
Clear synergy of CT-01 in combination with everolimus in HCC PDX model

CrownBio
CONNECTING SCIENCE TO PATIENTS

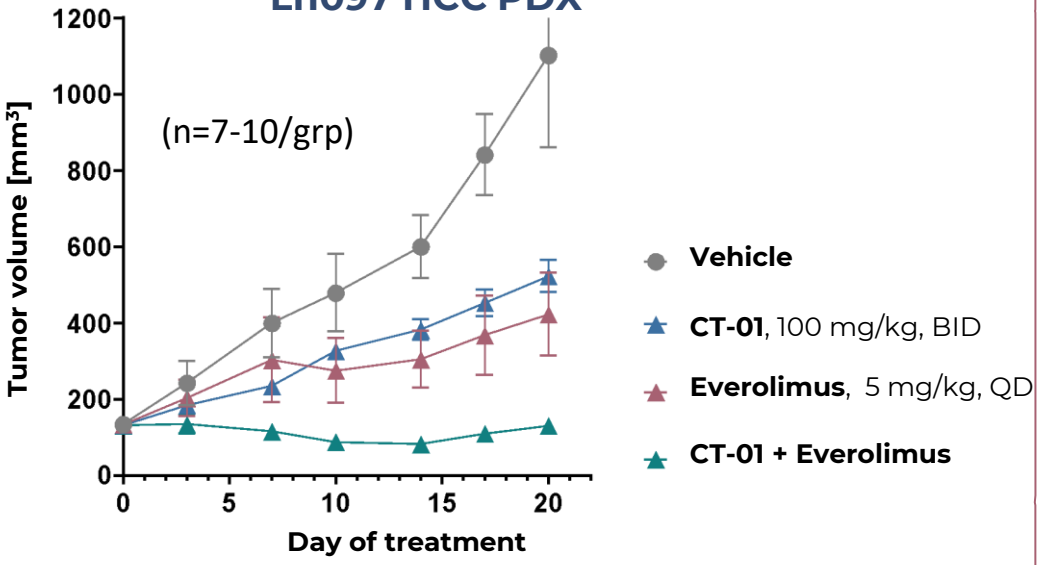
PDX Models/

Everolimus
- approved anticancer drug (kidney, breast & brain cancers)
- well-known for synergy with thalidomide (1st gen TPD)
- shows clear synergy in combination with CT-01

HCC (HUH-7) Xenograft Mouse Model



LI1097 HCC PDX



CT-01 combination with everolimus sensitizes poorly or non-responding tumor models

Captor Therapeutics®

Best-in-class potential of highly differentiated CT-01

- **Strong differentiation from other GSPT1 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**
  - GSPT1, 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)
- **Development activities**
  - Clinical Trial Authorization Application submitted in Europe
  - Initiation of Phase 1 clinical trials in hepatocellular carcinoma Q1 2025

Captor Therapeutics®

Captor Therapeutics®

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

Degradation is the better therapeutic strategy when targeting MCL-1

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

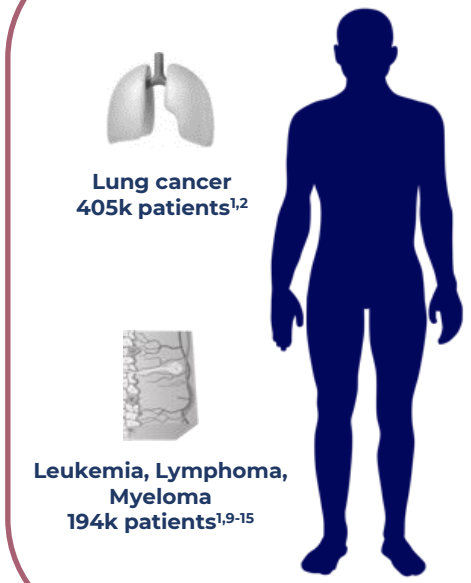
A critical resistance mechanism in hematological and solid tumors‡, cancer cells require very high levels to avoid induction of apoptosis

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

Inbitors ≠ degraders

Inhibitors bind to MCL-1 transiently blocking its activity; but when an inhibitor leaves the system MCL-1 remains. Most MCL-1 inhibitors were terminated as they caused accumulation of MCL-1†, which caused cardiotoxicity through necrosis§.

Captor is developing MCL-1 degraders, which remove MCL-1 and trigger cell-death, completely avoiding accumulation of MCL-1.



- ✓ CT-03's lack of accumulation of MCL-1 protein in heart tissue expands its therapeutic window compared to MCL-1 inhibitors
- ✓ CT-03 works via hit-and-run degradation, rapid induction of apoptosis and fast clearance at therapeutic doses
- ✓ Short-term degradation of ≈70% of MCL-1 irreversibly induces apoptosis in cancer cells

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
§iScience. 2020 April; 23(4): 101015

Captor Therapeutics®

MCL-1: a high potential cancer target

Highly attractive target with application in numerous cancer markets

Hematological malignancies

Multiple Myeloma (MM)
Est. $53B by 2030[1]

Acute Myeloid Leukemia (AML)
Est. $6B by 2028[2]

Non-Hodgkin Lymphoma (NHL)
Est. $16B by 2032[3]

Selected solid tumors

Small cell lung cancer (SCLC)
Est. $6.5B by 2031[4]

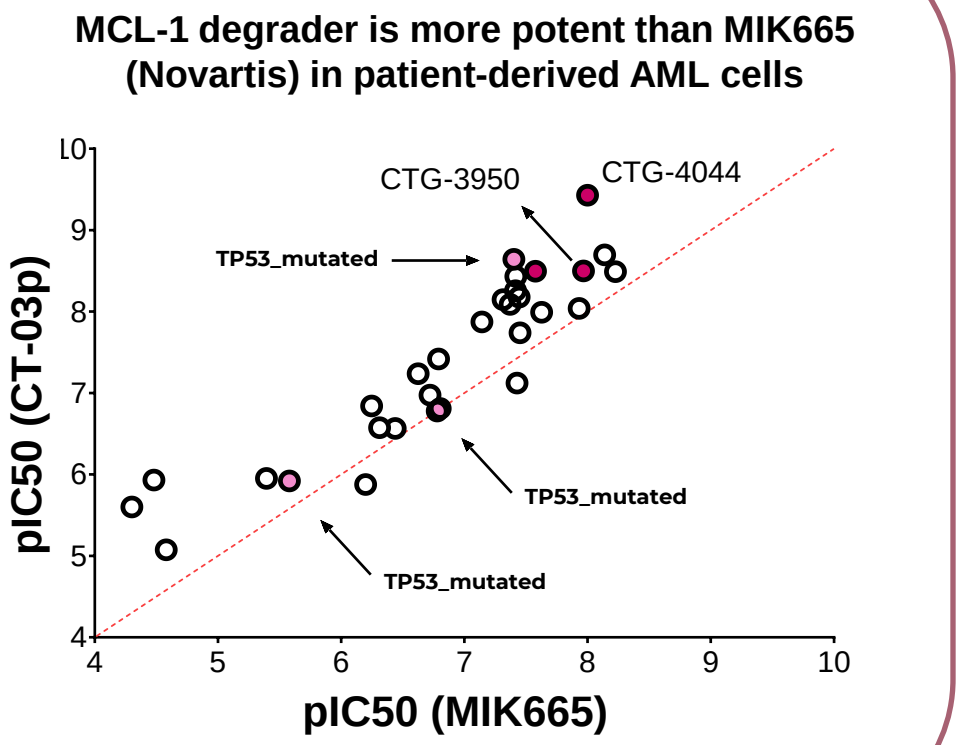
Non-small cell lung cancer (NSCLC)
Est. $36.9B by 2031[5]

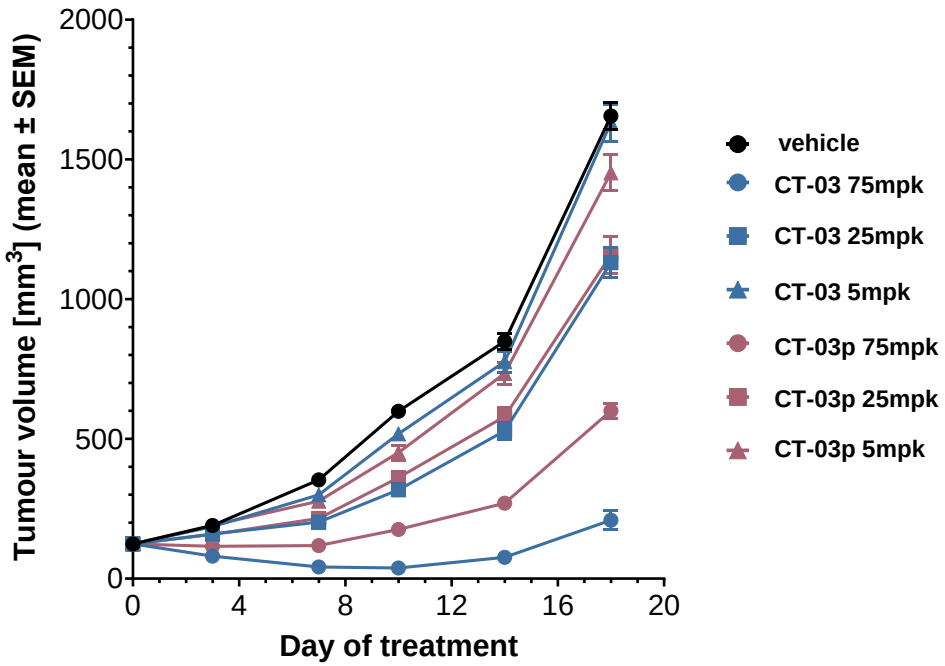
Triple-negative breast cancer (TNBC)
Est. $1.5B by 2030[6]

[1] Allied Market Research
[2] BCC Research
[3] Spherical Insights
[4] iHealthcareAnalyst
[5] Allied Market Research
[6] Databridge Market Research

Captor Therapeutics®

High potency of MCL-1 degraders in AML patient samples *ex vivo* & *in vivo* leukemia model

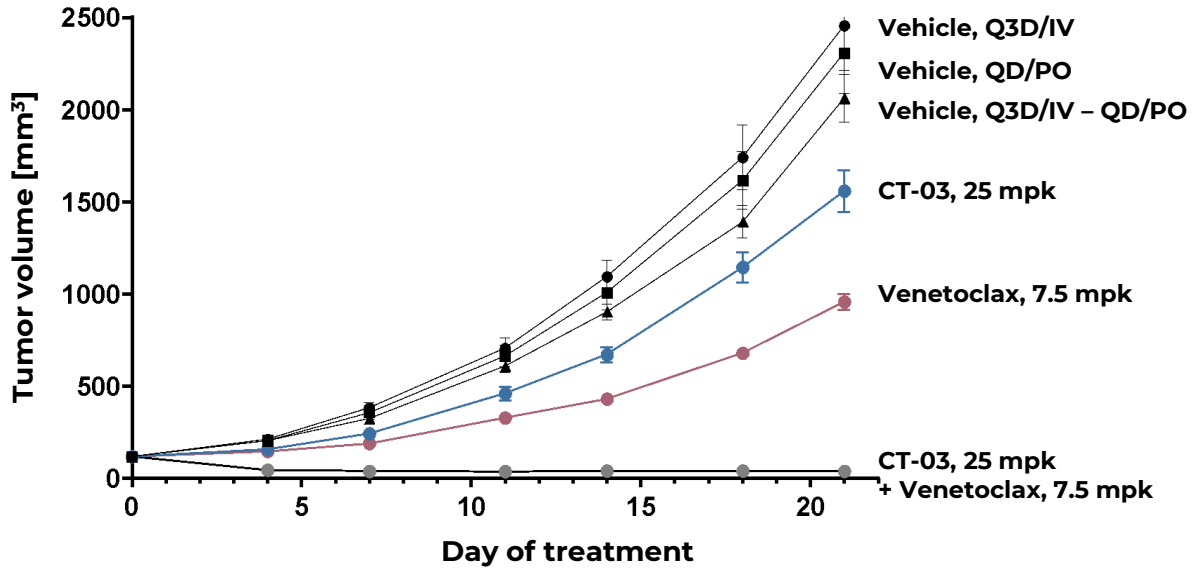




Q3D, IV, n=7-10/grp

CT-03p (prodrug) is more potent than MIK665 (Novartis) in a panel of 30 patient-derived cells (PDCs)and shows nM activity in cells refractory to gilteritinib and venetoclax

Combined of MCL-1 degrader with venetoclax regresses AML tumors in mice



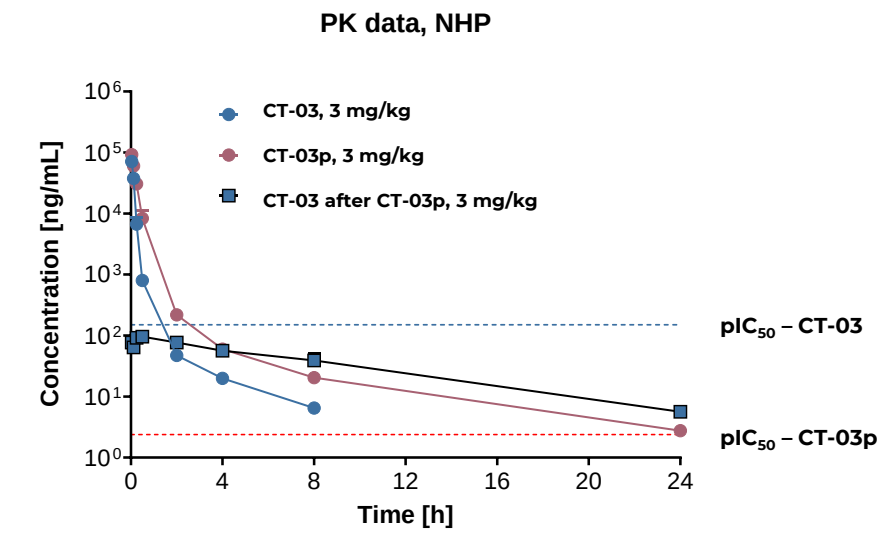
Regression of large tumors was observed for both CT-03 and venetoclax, with the combination providing the best outcome

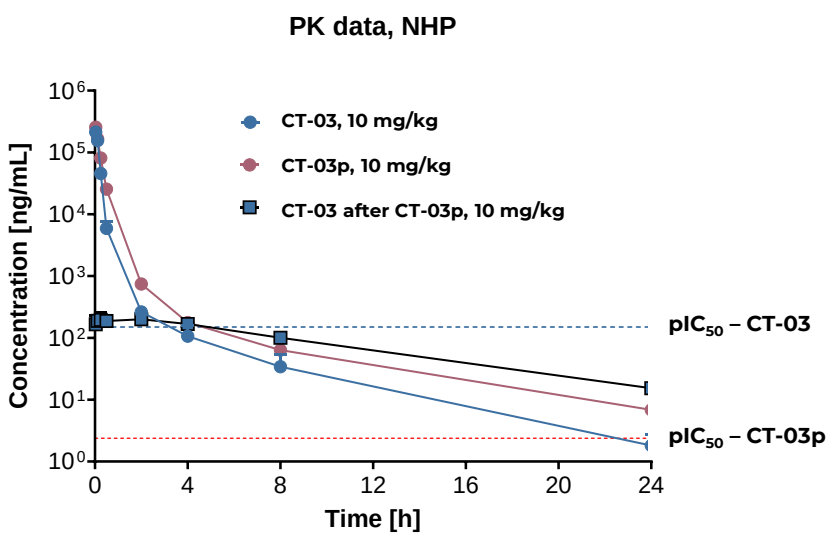
CT-03 was administered 8 times, every 3 days (Q3D) intravenously and venetoclax was administered daily (QD) orally; n=7-10/grp

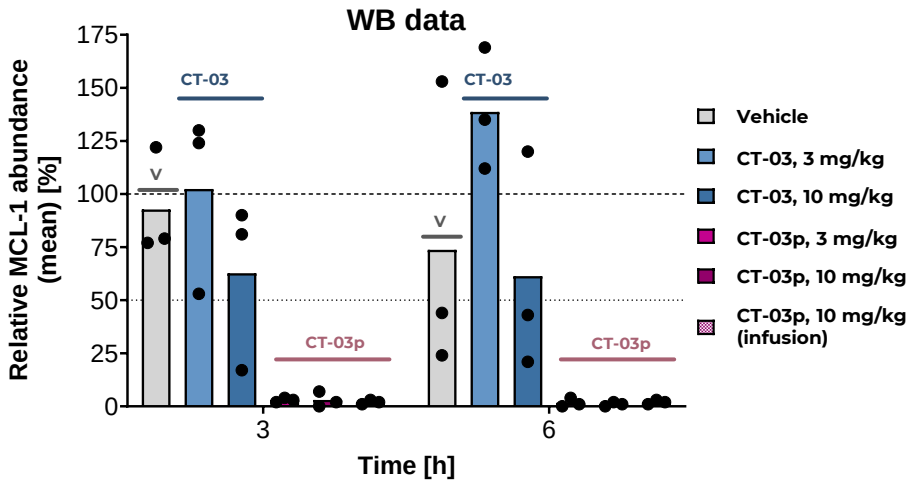
CT-03 in combination with venetoclax strongly inhibits cancer growth in MV4-11 Human Leukaemia Xenograft Model

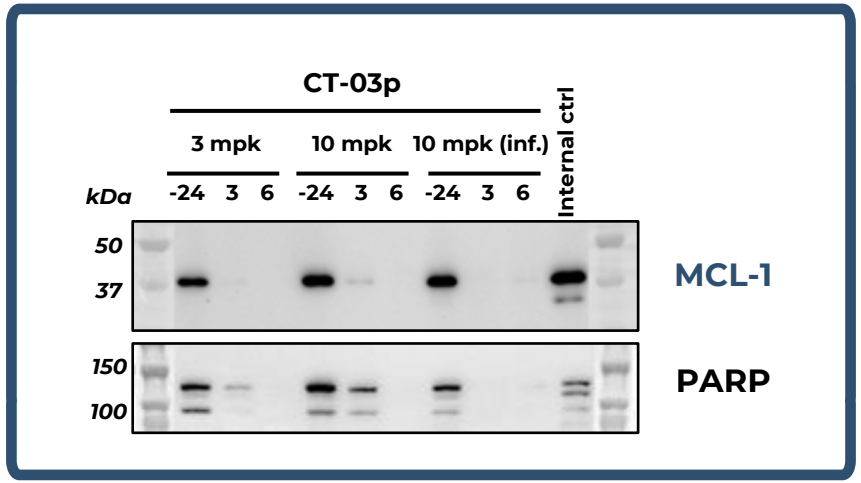
Captor Therapeutics®

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



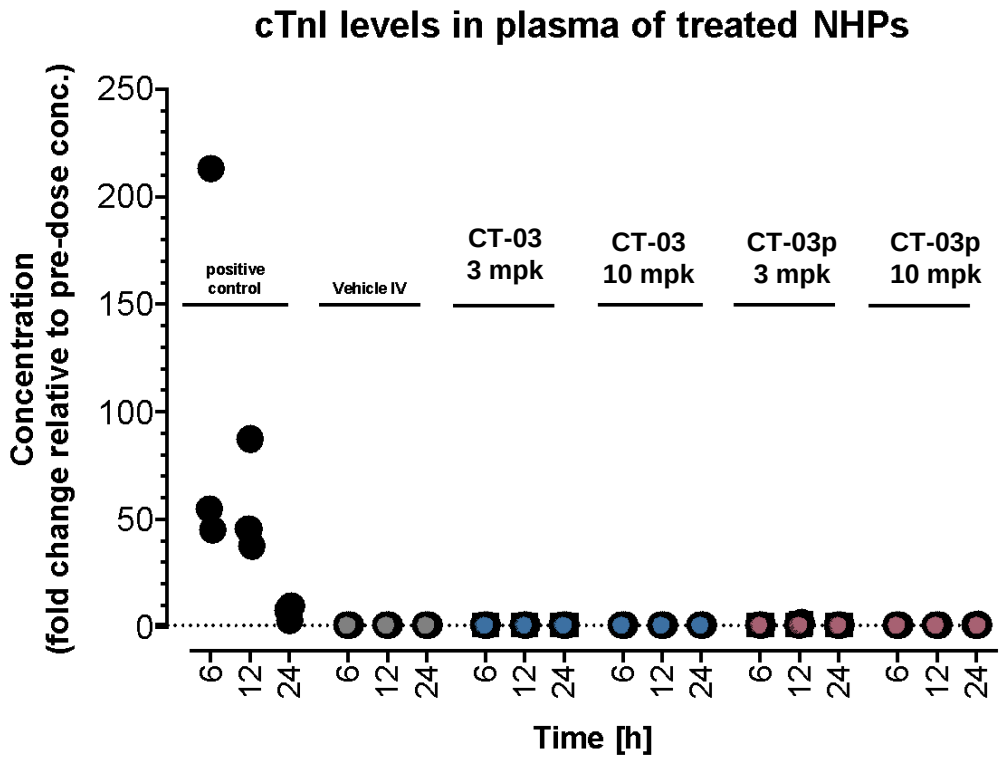






Captor Therapeutics®

Cardiotoxicity marker Troponin I in plasma of NHPs after CT-03 dosing



CT-03 & CT-03p: no observed increase in troponin-I levels

***Cardiotoxic positive control - Isoproterenol 3mg/kg, Vasopressin 0.3mg/kg**

Captor Therapeutics®

CT-03 candidate drug with unmatched therapeutic window

- **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
- **Expected milestones**
  - IND-enabling studies completion in H2 2025

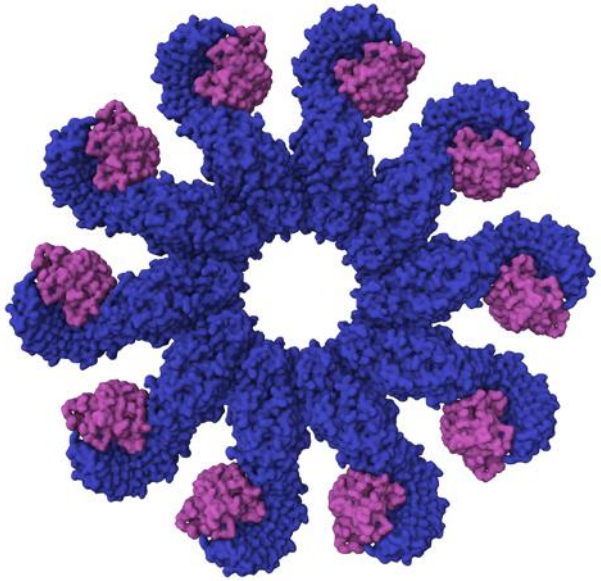
Captor Therapeutics®

CT-02 Series - First-in-Class NEK7 Degraders

- CT-02S (Autoimmune)
- CT-02B (Neuroinflammation)

Intervention in NLRP3 pathway *via* NEK7 degradation

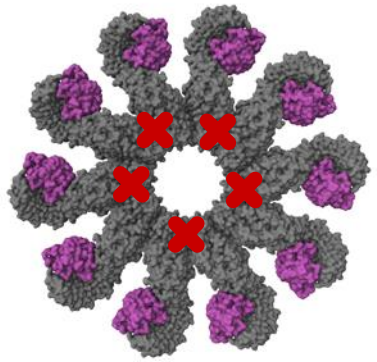
Aberrant activation of NLRP3



NLRP3 inflammasome

NEK7

NLRP3 inhibition →



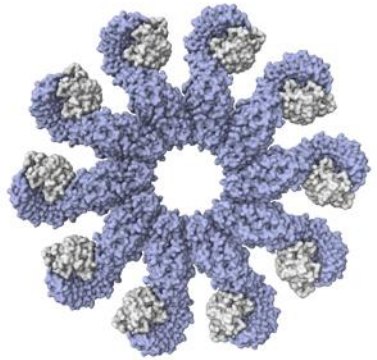
COMPLETE INACTIVATION OF NLRP3

→ **INCREASED INFECTIONS *via* REDUCED IMMUNITY**

Toxicity from other functions of NLRP3

NEK7 degradation →

PARTIAL INACTIVATION OF NLRP3



→ **MAINTAINED IMMUNITY**

Captor Therapeutics®

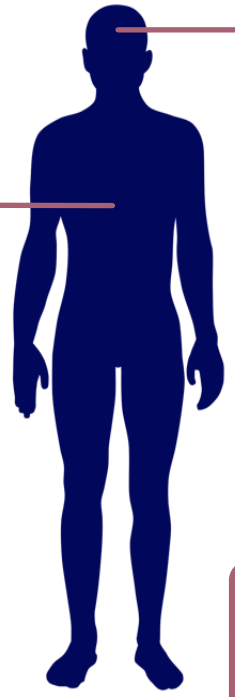
Significant market opportunities for Captor's NEK7 degraders

CPT-635
Systemic

Peripheral autoimmunity

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

Three significant therapeutic areas:



CPT-732
Brain Penetrant

Neuroinflammation

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

Obesity/ Metabolic	Autoimmune	Cardiovascular
16% living with obesity worldwide[5]	5-10% of global population[6]	19.8M deaths in 2022 due to CVD[7]
Global market size (2030):		
$100B[1]	$10.9B[2]	$124.9B[3]

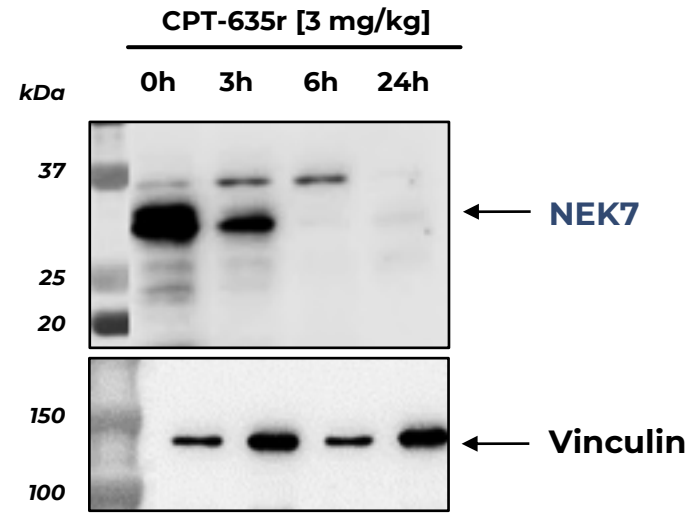
- Parkinson's: 8.5M patients worldwide (2019)[5]
- Alzheimer's: 6.7M patients in US (2023)[10]
- Huntington's: 388,000 patients worldwide[8]
- ALS: 345,000 patients worldwide[9]
- Potential in weight loss / obesity through CNS effect)

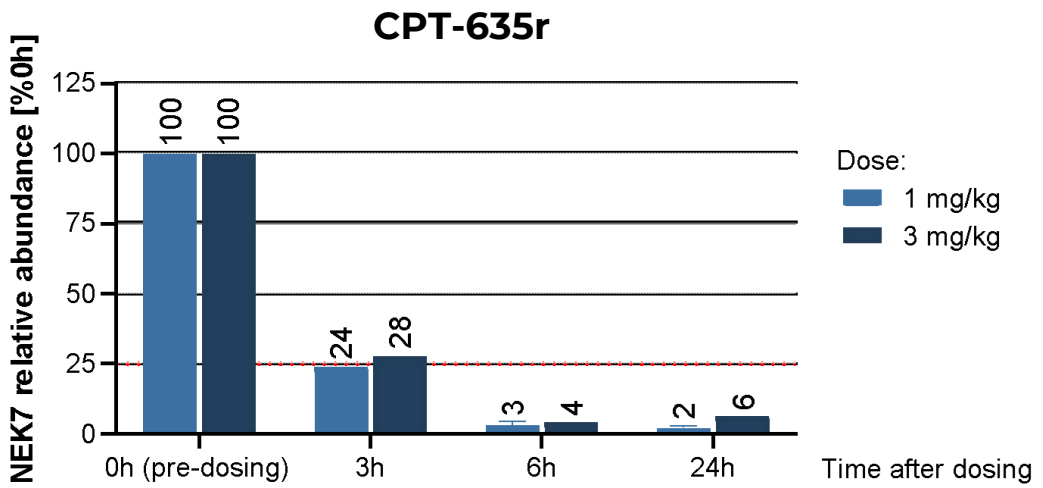
Neuroinflammation market is estimated at $4.9B[4] (2030)

1. https://www.goldmansachs.com/intelligence/pages/anti-obesity-drug-market.html
2. https://www.databridgemarketresearch.com/reports/global-autoimmune-disease-treatment-market
3. https://www.researchandmarkets.com/report/cardiovascular
4. https://www.researchandmarkets.com/report/neurodegenerative-disease-drug
5. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight
6. J Autoimmun. 2010 May;34(3):J168-77.
7. J Am Coll Cardiol. 2023 Dec 19;82(25):2350-2473
8. https://pubmed.ncbi.nlm.nih.gov/36161673/
9. https://pubmed.ncbi.nlm.nih.gov/31797084/
10. https://alz-journals.onlinelibrary.wiley.com/

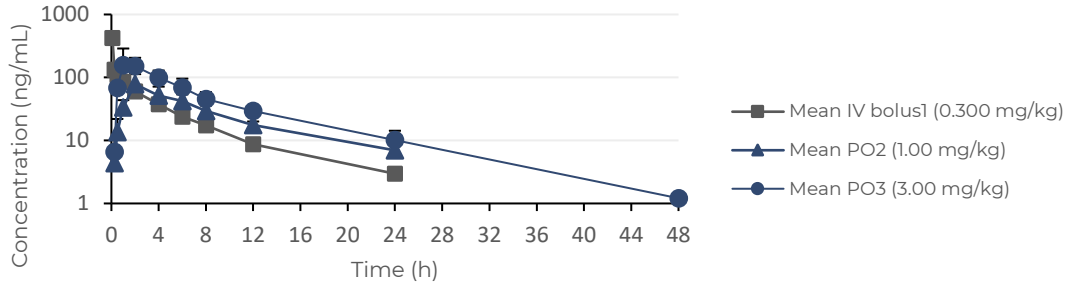
Captor Therapeutics®

CPT-513 efficiently covers & degrades NEK7 in NHPs after a single dose





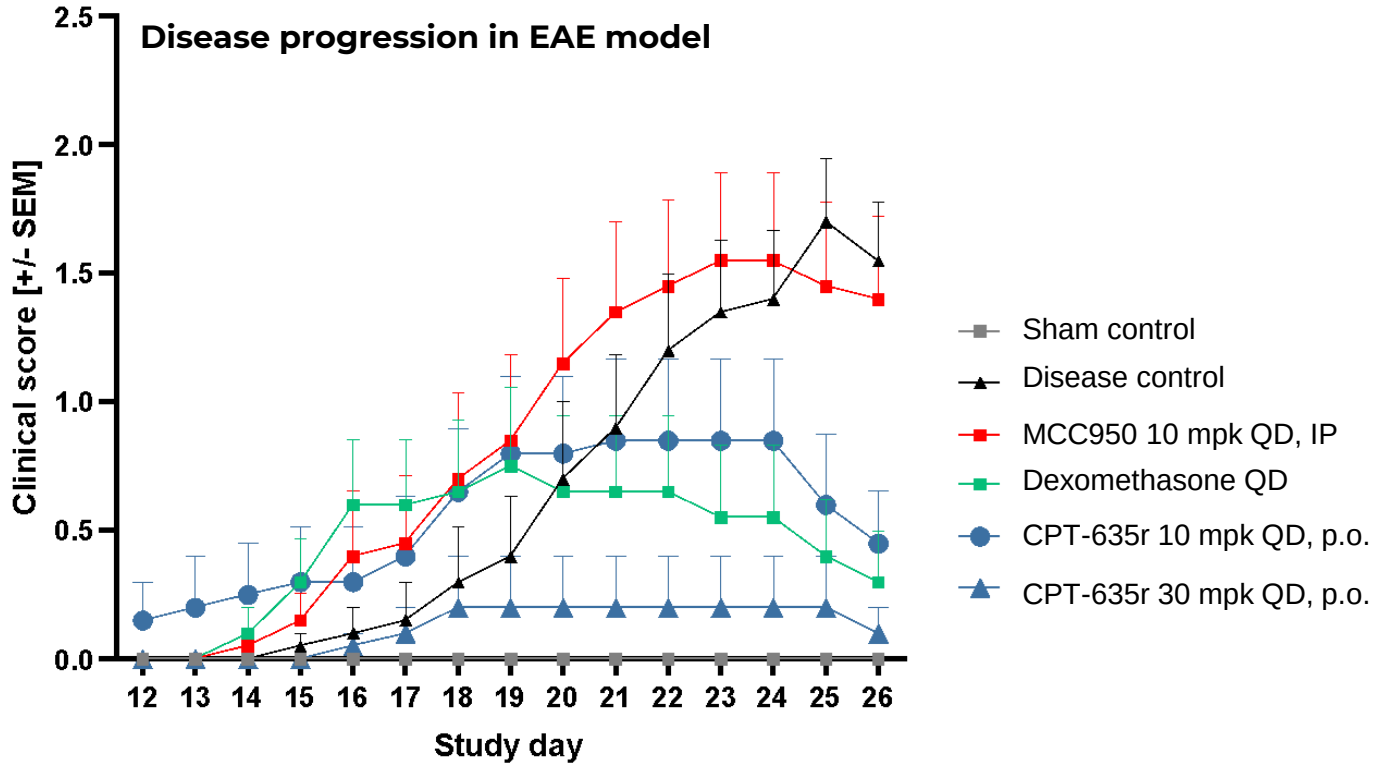
Mean Plasma Concentration of CPT-635r after IV bolus & PO dosing



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

Captor Therapeutics®

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

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_{35-55} Induced Experimental Autoimmune Encephalomyelitis (EAE) In Mice

CT-02: Excellent degraders from two different strategies

Two series of potent NEK7 degraders - in **autoimmune diseases** (CPT-635) and **neurodegenerative disorders** (CPT-732, brain-penetrant series)

Activity confirmed both *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 clean CEREP panel

PK/PD results in monkeys show excellent drug-like properties

Captor NEK7 degraders also degrade mouse NEK7 in addition to Human and Primate

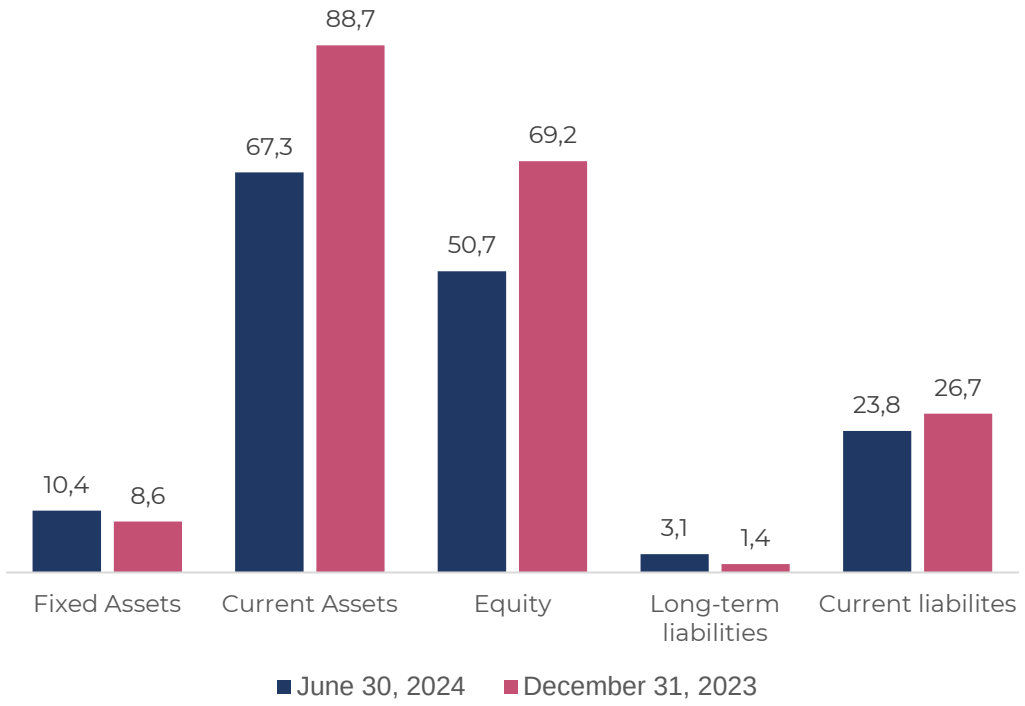
In vivo proof of efficacy in disease mouse models with no signs of toxicity

Captor Therapeutics®

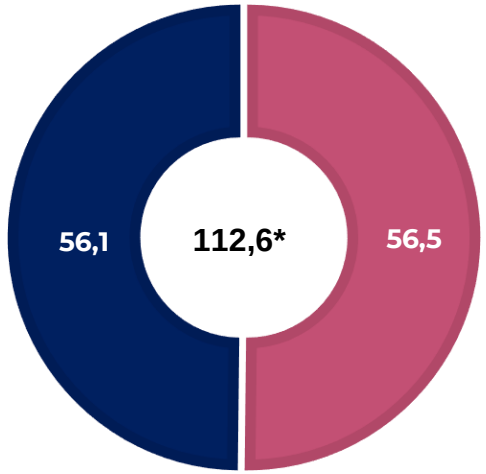
Finance Highlights

Balance sheet and cash position

Consolidated statement of financial position (PLN, M)



Cash position



Available funding secured (PLN M; as of June 30, 2024):

Total : **PLN 112,6***

- PLN M 56,5 cash
- PLN M 56,1 available grants (NCBR; ABM)

*** Amount includes grant awarded for phasing in CT-03 and CT-01 project.**

R&D costs in H1 2024**:**

Total : PLN 25,2 M

Net Operational Cash Outflow in H1 2024**:**

Total : PLN 17,5 M
(H1 2023 -PLN 31,5 M)

Captor Therapeutics®

Captor Therapeutics®

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

Captor Therapeutics GmbH
Hegenheimermattweg 167A
4123 Allschwil, Switzerland

Contact: investor.relations@captortherapeutics.com