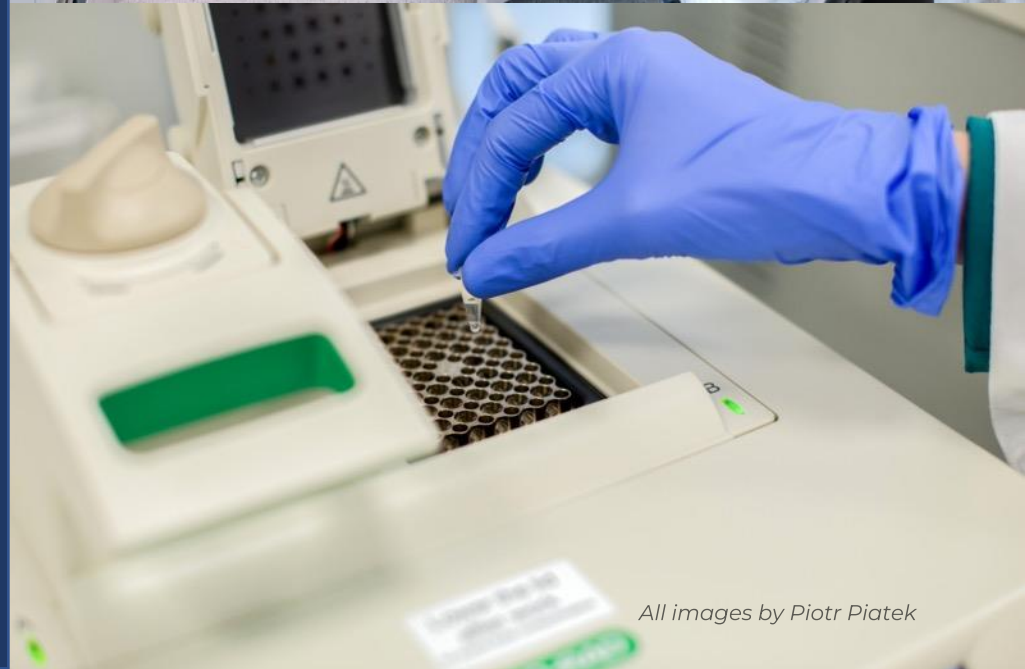




# H1 2023 update

Corporate Presentation

September 2023



All images by Piotr Piatek

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# CORPORATE SUMMARY

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# Captor Therapeutics – key take-aways in H1 2023

## Corporate

- ✓ Announcement of the **next steps in the Strategy** - increase of authorised share capital
- ✓ **Strengthening** of the scientific and business team
- ✓ Cooperation with **international advisors**: Wedbush and M.S.Q Ventures
- ✓ **Collaboration with Ono** has started well with excellent cooperation between the companies and an improvement in cash flow for Captor
- ✓ Several pharma companies enter into **Confidentiality Agreements** to look at Captor projects

## R&D

- ✓ **Target announcements** in CT-01, CT-02 and CT-05
- ✓ **Key R&D Announcements:**
  - ✓ **CT-01**  
Completed in-life part of GLP-tox – no gross findings
  - ✓ **CT-02**  
Partnering discussions initiated
  - ✓ **CT-03:**  
MTD and DRF in monkeys completed; no heart related issues in supraefficacious concentrations
  - ✓ **CT-05:**  
Partnering discussions initiated

## Optigrade™ discovery platform

- ✓ New E3 Ligases - degradation confirmed for **two new kinases (PTK2b and Wee1)**
- ✓ New project: **CT-09 targeting intrinsically-disordered protein in oncology**

## PLN 38,2 M incurred for R&D

Cash flow supported by non-dilutive grant funding which helps preserve capital in these difficult market conditions.

Company started a new research project, co-financed with **PLN 52.2 million by the Medical Research Agency (ABM)**, related to the development of an anti-cancer therapy for the treatment of patients with colorectal cancer and other types of cancer.

**The company has obtained shareholders' consent for introduction of authorized share capital and issue shares on this basis, which will take place at the most favorable time for it.**

# Roadmap to our strategic objectives

Fully-owned portfolio of owned clinical and preclinical assets, while sharing development or commercial risks with partners at the optimum time for each asset.

4 active pipeline projects\*

Clinical trials in patients of 2 lead pipeline assets – CT-01 and CT-03

Further preclinical work on CT-02 and CT-05, with partnering or licensing at preclinical stage

Optigrade™ Platform

2 new collaborative areas – Novel ligases and ADCs  
Leverage our platform for additional non-dilutive funding and validation  
Source of new early pipeline projects

**Nurix – Seagen deal for DACs – Degradable-Antibody Conjugates**

# Significant strengthening of the team



## Donald Coppen

Business Development Director

- PhD Southampton University
- MBA Cranfield University
- Over 20 years experience in Business Development & Alliance Management



## Andrew Saunders

Chief Medical Officer

- MD Trinity College Dublin
- Over 20 years of experience in conducting clinical trials
- Past experience in hemato-oncology and solid tumours



The University of Dublin



## Tomas Drmota

VP Early Discovery

- Ph.D. Charles Univ. Prague
- Post-doc Univ. of Glasgow GPCRs research
- 15 years AstraZeneca drug discovery/development projects leadership
- Program manager SOTIO biologicals-cell therapy



### EDUCATION

### PREVIOUS EXPERIENCE



# International financial advisors



- Exclusive strategic financial advisor
- Identification and evaluation of potential strategic opportunities:
  - licensing or partnering transaction
  - asset transaction
  - strategic transaction regarding shares or assets
- Maximizing value for the Company's shareholders
- Long-term cooperation



- Specialist healthcare business development broker focused on Greater China
- Objective is to establish potential partnerships in Greater China area (China, Taiwan, Hong-Kong, Macau)
- In 2023 MSQ an advisor to C4 Therapeutics by the licensing agreement in Greater China with Betta Pharmaceuticals - \$10M upfront/ \$25M equity investment/ up to \$357M in milestones

# Ono Pharmaceutical - a strong partner for Captor

Worldwide drug discovery collaboration with Ono Pharmaceutical to develop novel small molecule degrader drugs against a currently undrugged target of interest in neurodegenerative diseases.

## R&D Capabilities Combining In-house Drug Discovery and Open Innovation



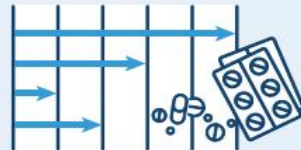
Number of joint research projects with universities, biopharmaceutical companies, etc.  
(As of end of March 2022)

About **200**

Number of approvals obtained  
(FY2021)

**7**  
approvals

## A Pipeline that Continuously Markets New Drugs



Number of clinical trials conducted  
(FY2021)

**82**  
trials

Number of new products launched and additional indications approved  
(FY2017 to FY2021)

**31**  
products

## Continuing Vigorous Investment in Drug Development



R&D costs  
(FY2021)

**¥75.9**  
billion

R&D cost-to-revenue ratio  
(FY2021)

**21.0**  
%

## Other ONO partnerships



## Payment of remuneration to the Company:

- Fee upon signature of the collaboration agreement
- Ono pays all research costs of Captor
- Milestones payments as products progress

This collaboration shows that Captor's Optigrade™ TPD platform is attractive for international pharma partners as well as allowing the Company to enter a new disease area at minimal cost.



# DEVELOPMENT OF PROJECTS

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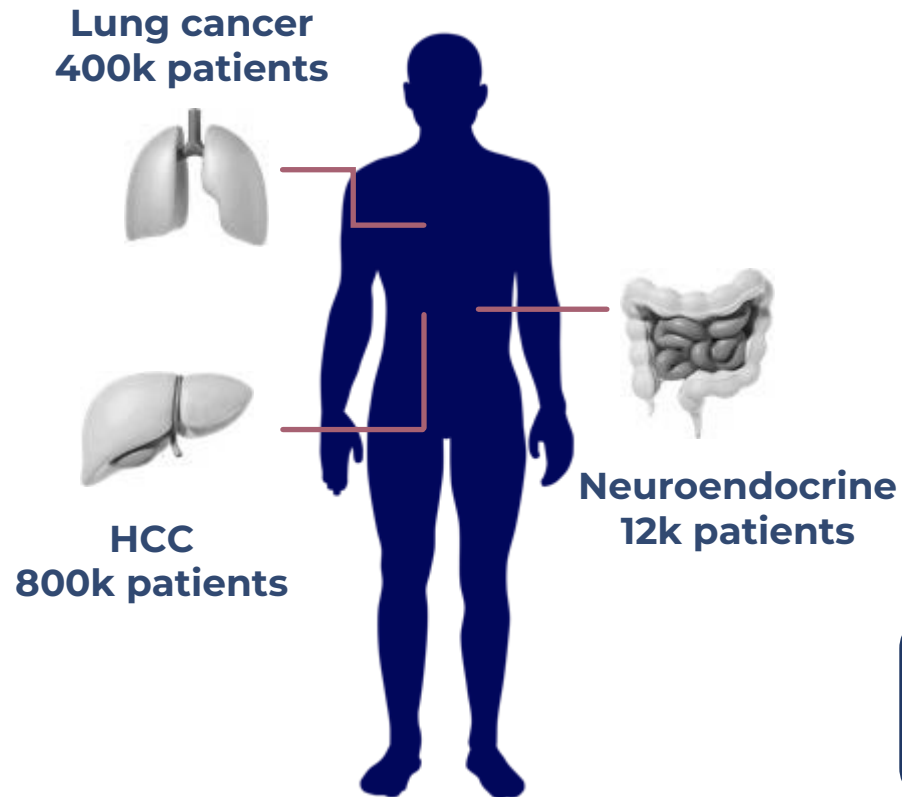
# Fully owned pipeline

#	Target	Indications	Modality	Discovery	Preclinical*	IND Filing	Phase IA / IB
CT-01	GSPTI, NEK7, SALL4	Hepatocellular carcinoma, Lung cancer, NET tumours	MG				
CT-02	NEK7	Autoimmunity, CNS, Metabolism, Oncology	MG				
CT-03	MCL-1	Liquid & solid tumours	BID				
CT-05	PKCθ	Autoimmunity, Oncology, Transplantation, Metabolism	BID				
CT-09	IDP** target	CRC, Haemato-oncology, I/O	MG				
	New E3 ligase degraders	Autoimmunity, Cancer	MG BID				

\*Preclinical stage include IND-enabling studies, **BID** – Bi-functional Degradere; **MG** – Molecular Glue, \*\* - IDP – Intrinsically Disordered Protein

Assumed stage at the end of 2025

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



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

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

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

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

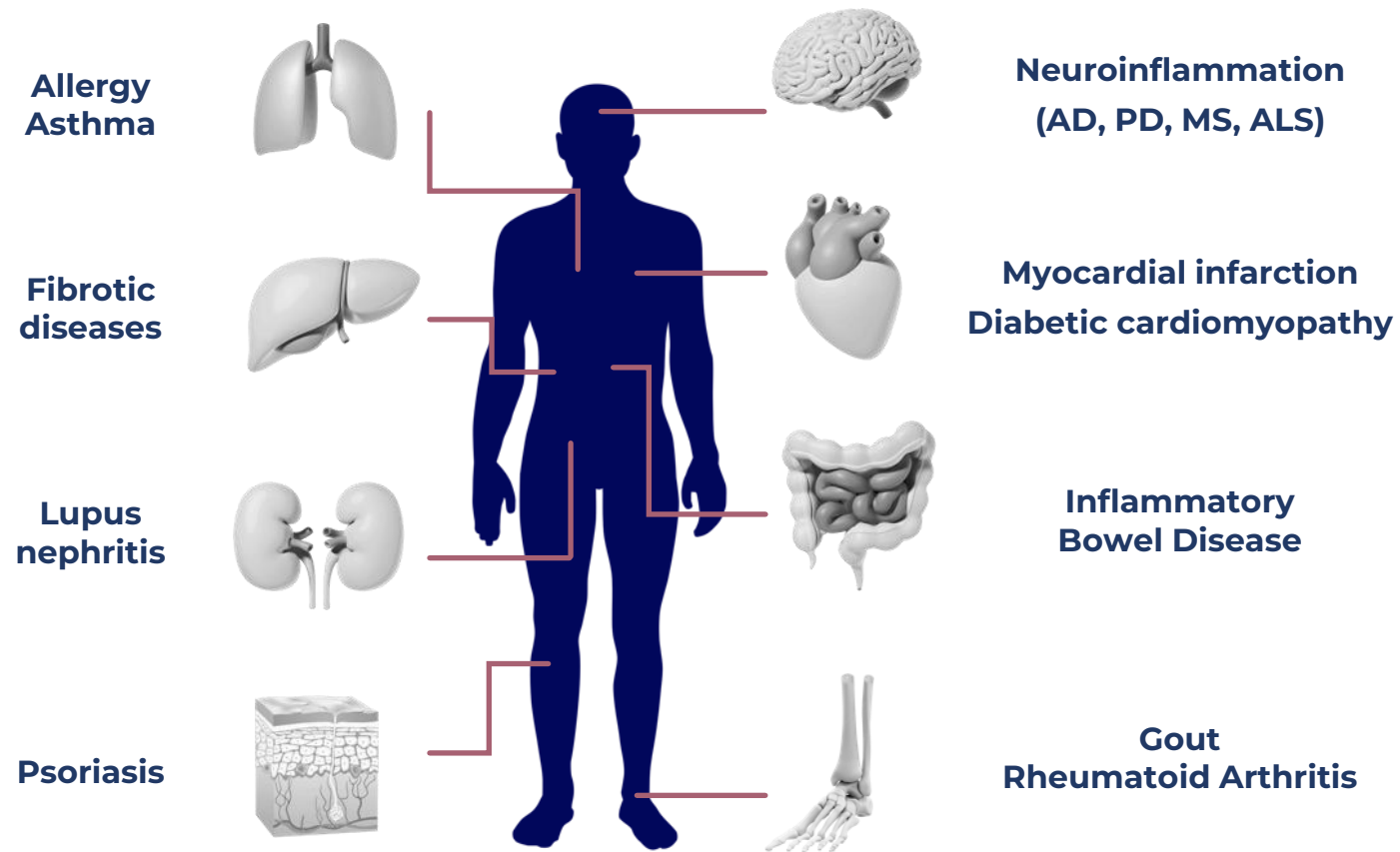
A unique degradation profile combined with target tissue pro-drug activation for liver, lung and neuroendocrine cancers

# Recent achievements

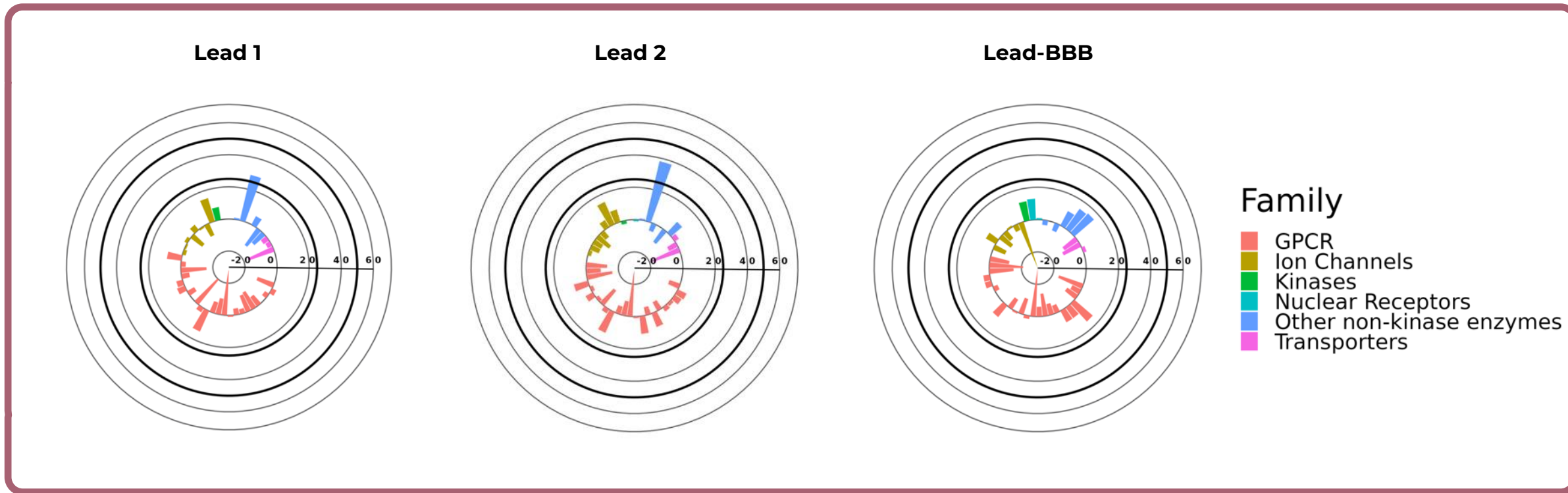
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- Completion of GLP-tox in life phase with no gross findings
  - Awaiting histopathology and full report
- Demonstrated combination with everolimus potentiates degradation of target proteins
- Status:
  - Production of a GMP-batch and drug product for clinical studies ongoing
  - IB in preparation
  - Finalising appointment of CRO to supervise clinical study

# CT-02: Vast market potential for inflammasome modulators

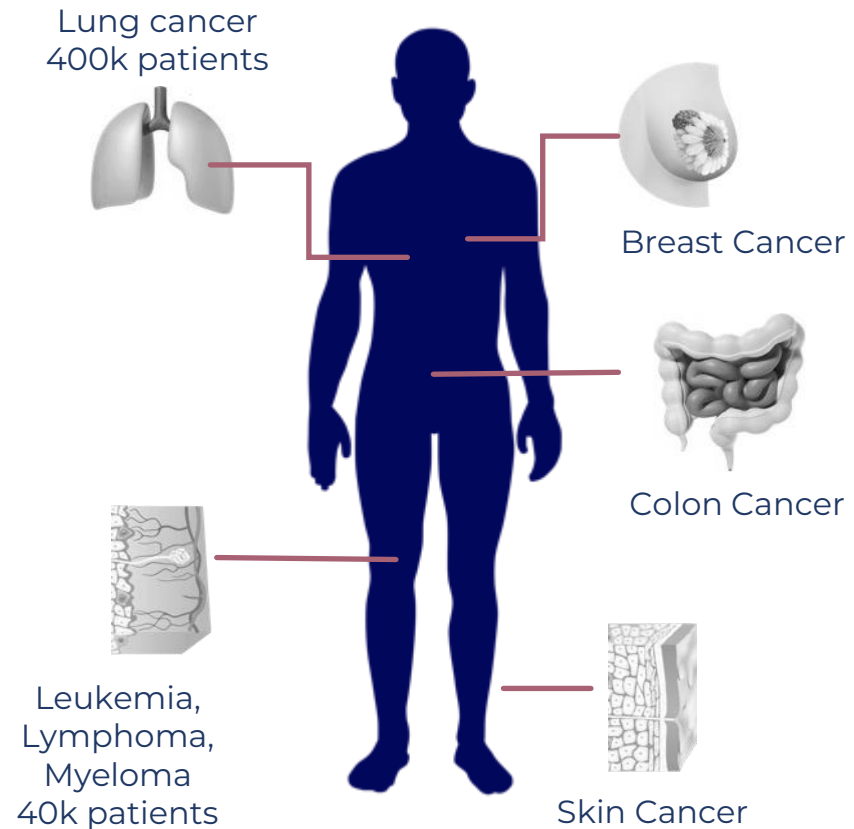


# Lead compounds show excellent selectivity profile / safety profile



**All the lead compounds demonstrate high safety profile in CEREP panel**

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



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

A critical resistance mechanism in haematological and solid tumours

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

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

Use of inhibitors causes accumulation of MCL1 in cancer cells

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

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

# Milestones

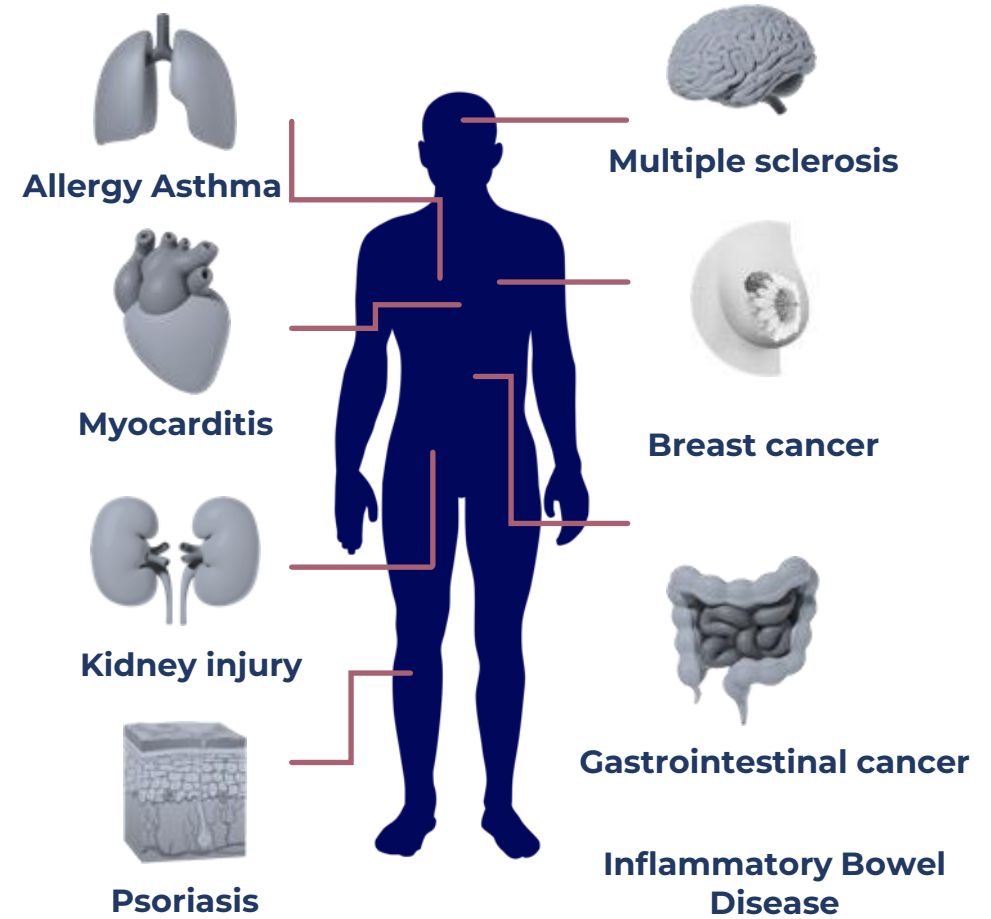
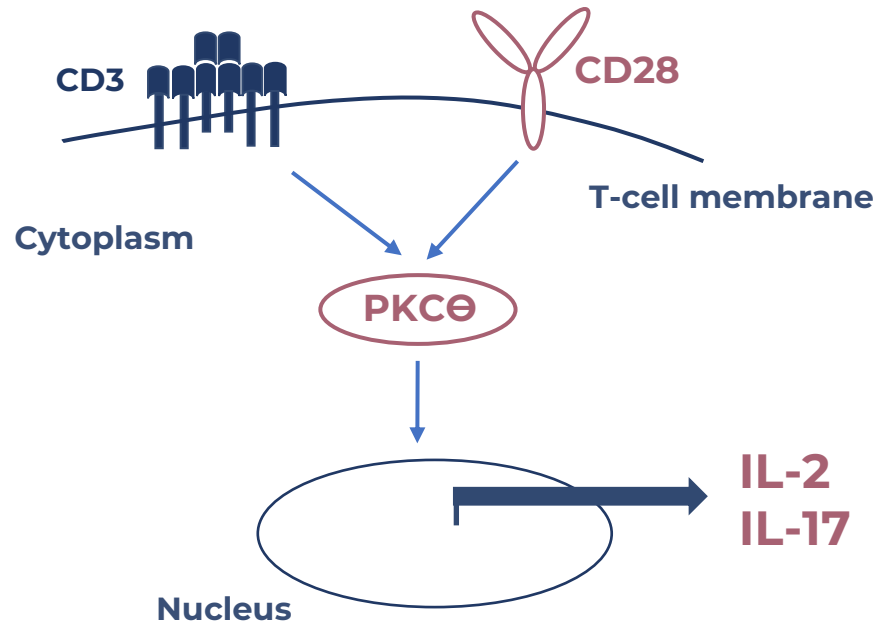
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- Two MCL-1 bifunctional degraders have been developed with high potency (DC<sub>50</sub>) of <1 nM and 100 nM
- Both compounds potently inhibit tumor growth *in vivo*
- Both compounds do not affect NHP troponin-I (a marker of heart muscle damage) levels at doses higher than the effective doses
- ~1 kg of a first compound was produced and is a single synthesis step from the second compound
- Candidate selection planned for Q4 2023



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

TCR

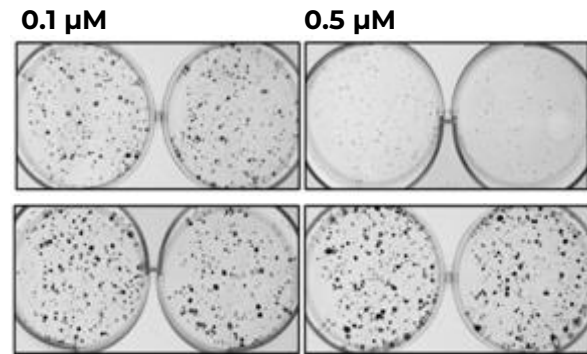


# Best-in-class selectivity

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

BigPharma compound  
**ABS-911**  
 Inadequate Selectivity

Lead compound  
 High Selectivity

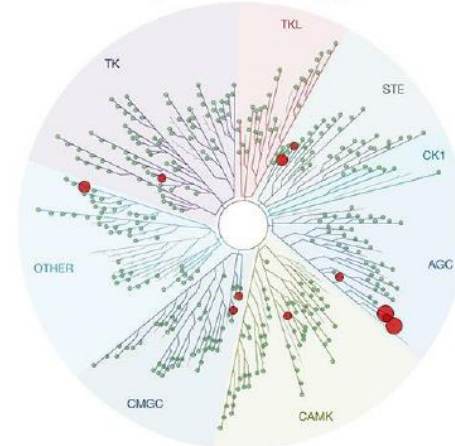


Compound	IC <sub>50</sub>	I <sub>max</sub>	DC <sub>50</sub>
Lead compound	55 nM	82 %	29 nM
ABS-911	98 nM	99 %	N/A

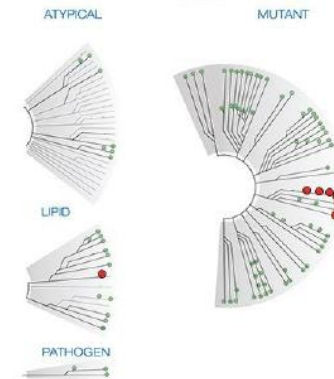
Excellent selectivity against a large number of kinases

## KinomeScan results TREEspot™ Interaction Map panel of ca. 450 kinases

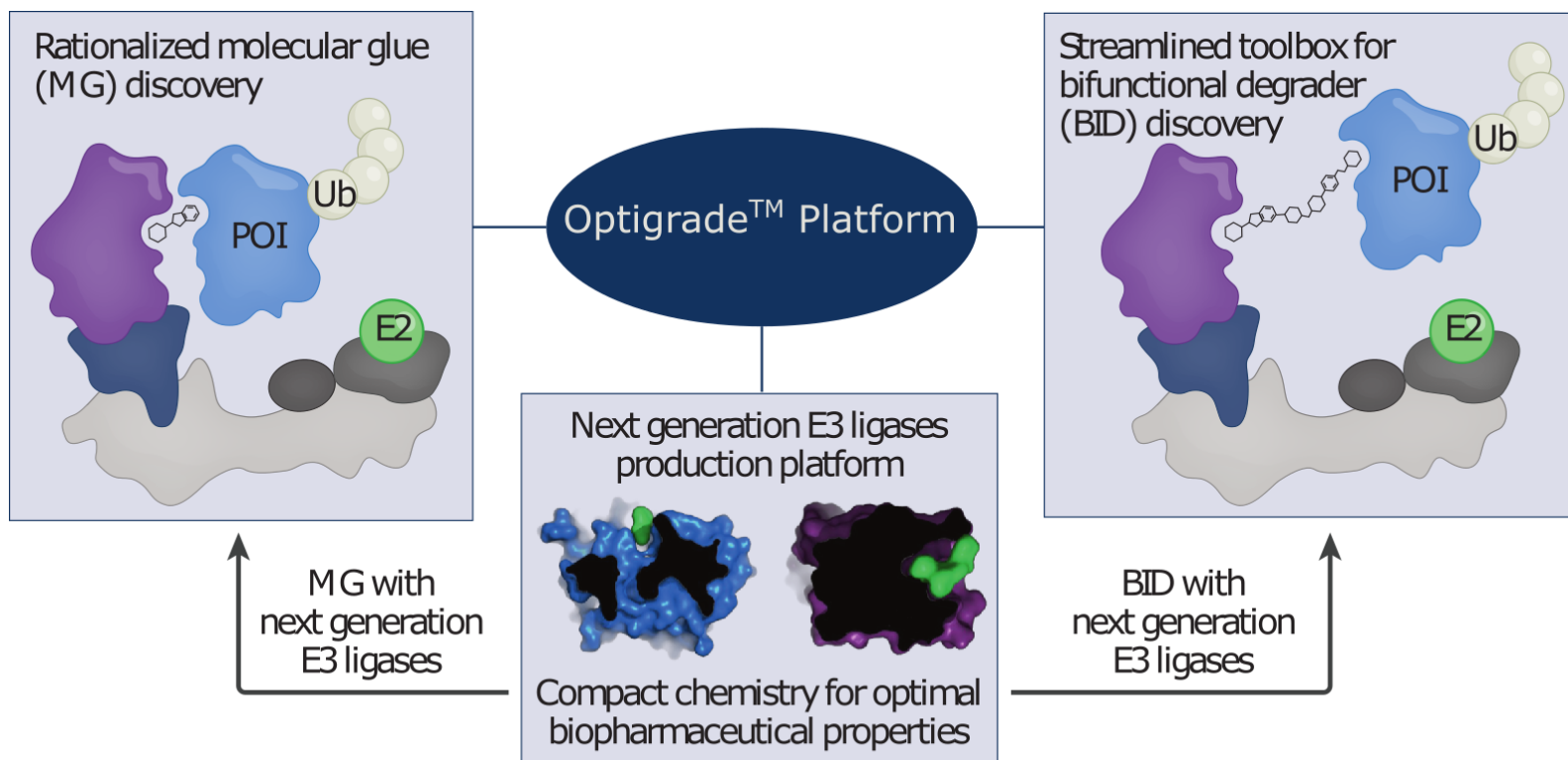
ABT-673 @ 10000nM



Percent Control



# Optigrade™ discovery platform – importance of structure & chemistry

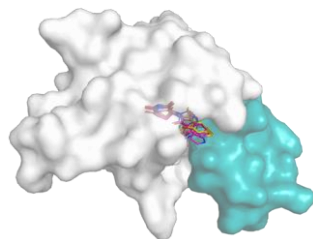


**Optigrade™ – addressing Molecular Glues, Bifunctional Degraders and novel Ubiquitin E3 Ligases**  
**Leading chemistry expertise in creating “activated” degraders to increase intracellular potency**

# Optigrade™ discovery platform

Molecular  
Glue

CT-09



New E3  
Ligases

- **First-in-class degrader** of an Intrinsically Disordered Protein (IDP)
- **High commercial potential** in CRC, hematological cancers and immuno-oncology
- Disordered architecture **precludes of development of classical inhibitors**

#### To date we have:

1. Identified hits
2. Solved X-ray structure of the target in complex with E3 ligase and a hit compound

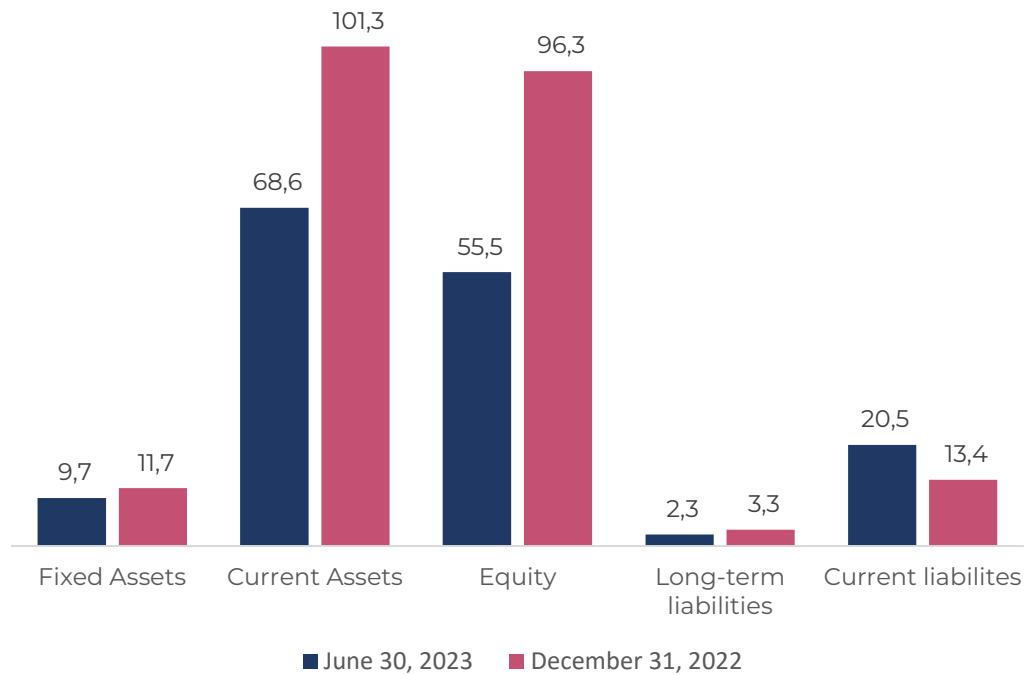
- Demonstrated **first degradation** of a target protein using the **novel ligase KLHDC2**
- Recently demonstrated **degradation of 2 additional new kinases with KLHDC2**
- **Established a production workflow** for the Kelch family of novel ligases

# FINANCIALS AND PLANS FOR THE FUTURE

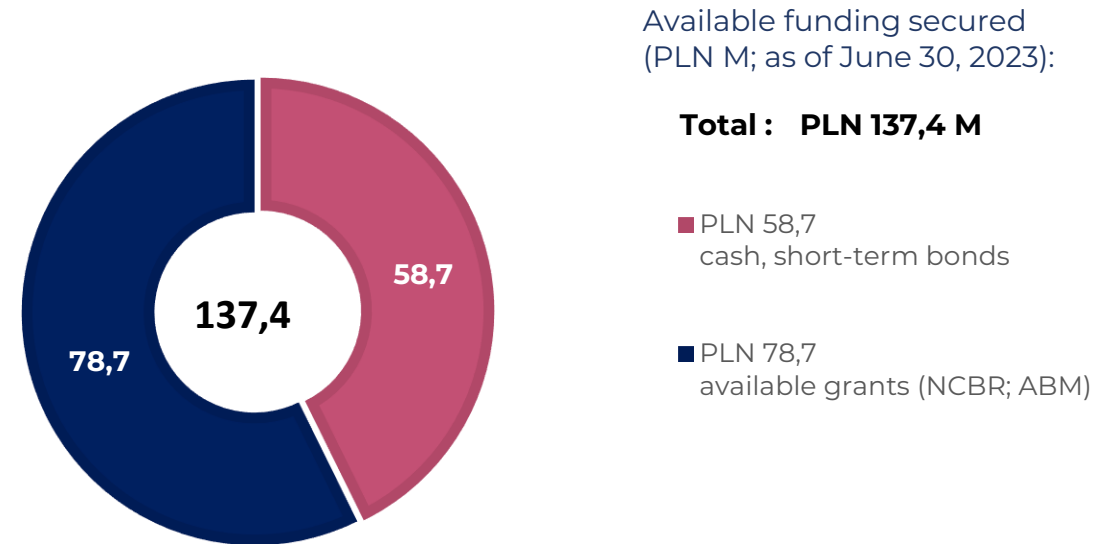
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# Strong balance sheet and cash position

Consolidated statement of financial position (PLN, M)



Cash position



R&D costs in H1 2023:

**Total : PLN 38,2 M**

Cash outflow in H1 2023:

**Total : PLN 32,2 M**

# Next steps

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## 2023

- **CT-01:** Submit Clinical Trial Authorisation application
- **CT-02** and **CT-05:** *In vivo* proof of concept in autoimmunity
- In cell degradation of target with novel E3 ligase-based degrader

## 2024

- **CT-03:** Submit Clinical Trial Authorisation application
- **CT-01:** Clinical readouts: safety, pharmacology, & mechanism
- First degrader of a new target based on novel E3 ligase
- New partnering in immunology

# Q&A SESSION

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4123 Allschwil, Switzerland

Contact: [relacje.inwestorskie@captortherapeutics.com](mailto:relacje.inwestorskie@captortherapeutics.com)



## Projects co-financed by the European Regional Development Fund:

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

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

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)



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

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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)

