Captor Therapeutics®

Targeted Protein Degradation Q1 2024 Update

- **DM Pekao -**

All images by Piotr Piatek

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Advanced therapies from targeted protein degradation

Captor Therapeutics: Warsaw Exchange listed biotech (WSE: CTX): ~105 FTEs dedicated to targeted protein degradation (TPD) with facilities in Poland and Switzerland

$2 billion innovation support program in Poland allows a capital sparing R&D model

- Secured >$53m EU / Polish non-dilutive funds to date

Fully-owned, differentiated, oncology and inflammation pipeline:

- Tissue-activated degrader of GSPT1 for liver cancer: potential best in class profile, CTA H1 2024
- Kinetics-optimized degrader of MCL-1 for heme & solid tumors, potential best in class profile; CTA/IND H2 2024/ Q1 2025
- 2 series of selective molecular glue NEK7 degraders - systemic series for chronic inflammation and metabolism indications; and brain penetrant series for neuroinflammation, *in vivo* POC established
- Potential first-in-class selective degrader of PKCϴ for autoimmune indications in *in-vivo* studies

Discovery platform: Optigrade™

- Empowers both molecular glue and bifunctional degrader discovery
- Industry-leading protein engineering, structural biology and biophysics team >30 internal FTEs
- Novel E3 ligases for TPD (targeted protein degradation demonstrated with the E3 Ligase KLHDC2)
- Ultra-potent, selective MCL-1 & GSPT1 degraders potentially suitable for use as ADC payloads
- Strategic partnership with Ono Pharmaceutical to develop degraders of a neurodegeneration target

Fully owned pipeline

Program	Primary Target	Indications	Modality	Discovery	Preclinical*	IND Filing	Phase IA / IB	Phase II
CT-01	GSPT1	Hepatocellular carcinoma, Lung cancer, NET tumors	MG					
CT-02	NEK7	Autoimmunity, CNS, Metabolism, Oncology	MG					
CT-03	MCL-1	Liquid & solid tumors	BIFD					
CT-05	PKCΘ	Autoimmunity, Oncology, Transplantation, Metabolism	BIFD					
New target projects		Autoimmunity, Cancer	MG BIFD					
New E3 ligase degraders		Autoimmunity, Cancer	MG BIFD					

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

Assumed stage at the end of 2025

An experienced leadership team

Tom Shepherd, Ph.D.
Chief Executive Officer

- 30 years in Biotech leadership positions
- Led 12 licensing transactions resulting in > $3 B in sales
- 6 private investment rounds and 3 IPOs.

Michal Walczak, Ph.D.
Co-founder
Chief Scientific Officer

- Ph.D. ETH Zurich,
- Post-doc FMI Basel (Novartis Research Foundation) on TPD
- 10 years in drug discovery and TPD

Anna Pawluk, Ph.D.
Head of Operations

- Ph.D. University of Wroclaw
- MBA WSH in Wroclaw
- 15 years of R&D experience

Sylvain Cottens, Ph.D.
Co-founder
SVP Chemistry

- Ph.D. EPFL Lausanne,
- Post-doc Caltech, (USA)
- Scientific expert & leader with 25+ years at Novartis
- Co-inventor of Afinitor and co-developer of Gilenya (both blockbuster drugs)

EDUCATION

University of Strathclyde Glasgow · London Business School · Universytet Wrocławski · ETH · Universytet Wrocławski · WSH Wyższa Szkoła Handlowa we Wrocławiu · EPFL

PREVIOUS EXPERIENCE

BAUSCH Health · kymab · FMI Friedrich Miescher Institute for Biomedical Research · NOVARTIS

New members of the leadership team

Andrew Saunders DPM, FFPM
Chief Medical Officer

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

Donald Coppen, Ph.D.
Business Development Director

- PhD, University of Southampton
- MBA, Cranfield School of Management
- 20 years' experience in business development:
- Biocompatibles plc [Acquired by BTG plc for £177M]
- Algeta ASA [Acquired by Bayer for $2.9B]
- Consultant to various UK biotechs
- Mereo BioPharma plc [Ultragenyx >$300M out-license]

Tomas Drmota, Ph.D.
VP Early Discovery

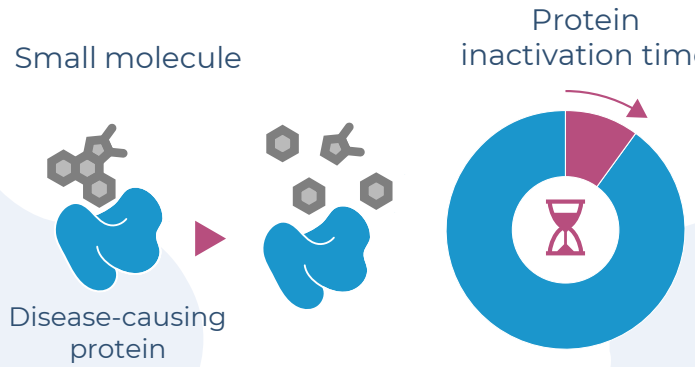
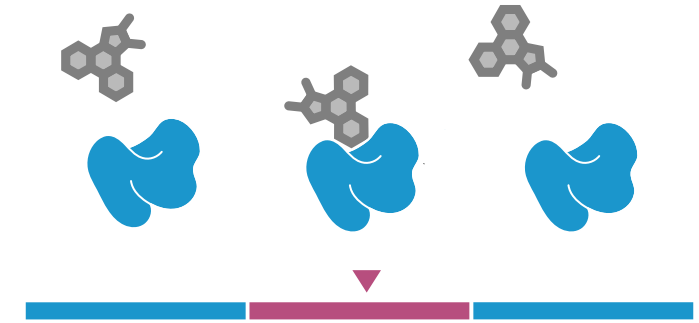
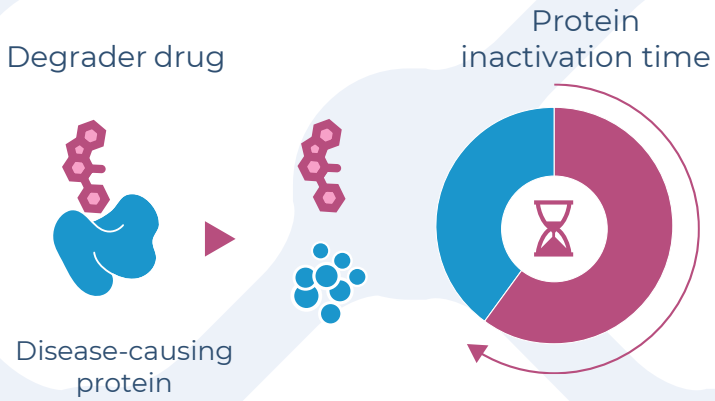
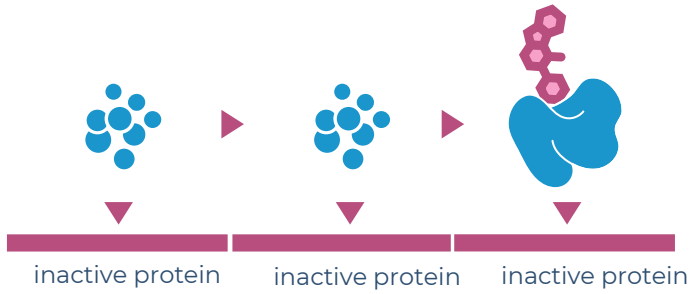
- PhD, Charles University Prague
- University of Glasgow, Biochemistry and Molecular Biology
- Tufts University, School of Medicine Boston
- 25 years' experience in preclinical drug development for cardiovascular, metabolism, respiratory, autoimmunity and immuno-oncology therapeutic areas

EDUCATION

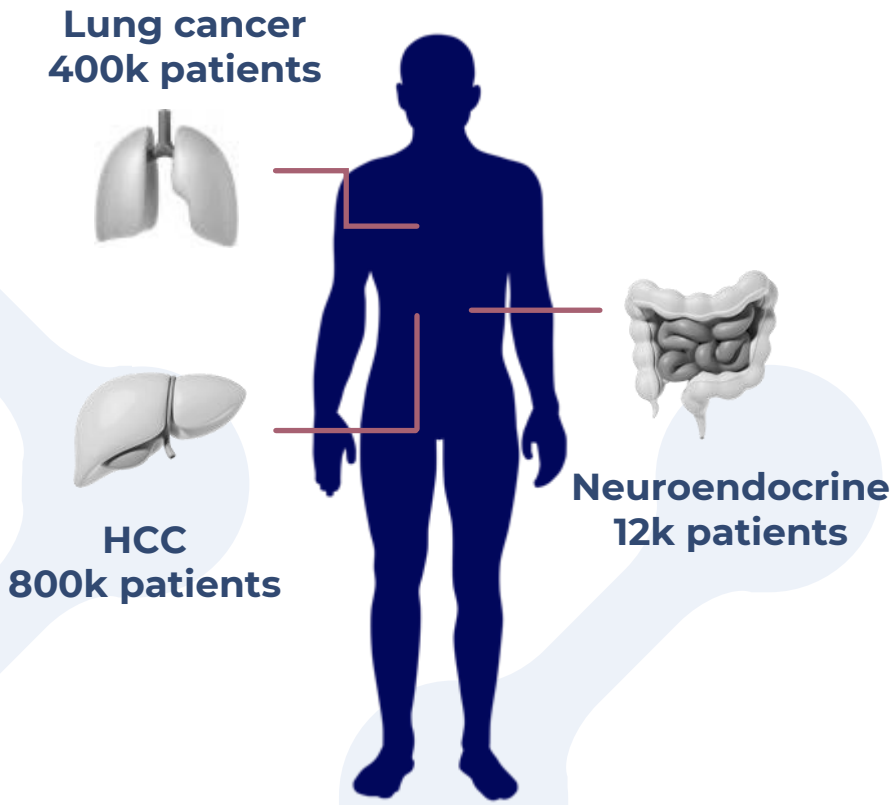
PREVIOUS EXPERIENCE

6

TPD: A new dawn in drug discovery

	Prolonged Pharmaco-dynamics	Different pharmacology	Outcomes
INHIBITION		 Protein only inactive when bound to drug	**Inhibitors:** • Frequent dosing • More side effects
DEGRADATION		 After TPD, same drug degrades other proteins	**TPD drugs:** • Less frequent dosing • Fewer side effects

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-1β production – a well-establish pro-carcinogenic factor. Reduction of IL-1β levels enables activation of the immune response

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

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

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Liver cancer

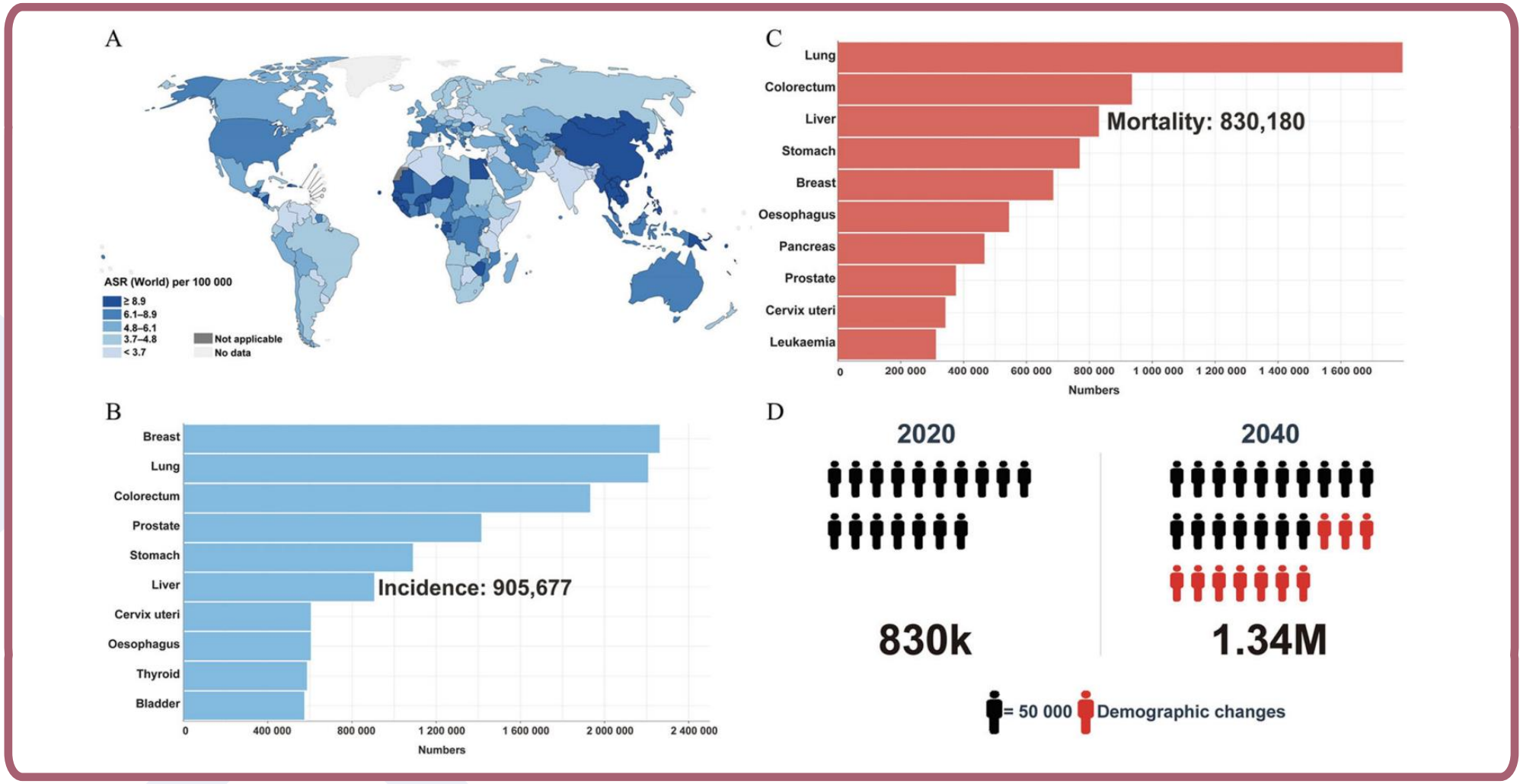
Liver cancer

- Fifth most common cancer
- Second most frequent cause of cancer-related death globally
  - 905,000 new cases and 830,000 deaths per year
- 7% of all cancers

Hepatocellular Carcinoma (HCC)

- Accounts for approximately 90% of primary liver cancers
- Constitutes a major global health problem
- Metabolic diseases (e.g. obesity, T2D) are major drivers of new cases

Epidemiology of Hepatocellular carcinoma



Market estimates for systemic HCC therapies of **$10.5-12.9B by 2030**, with a **CAGR of 15-20%**

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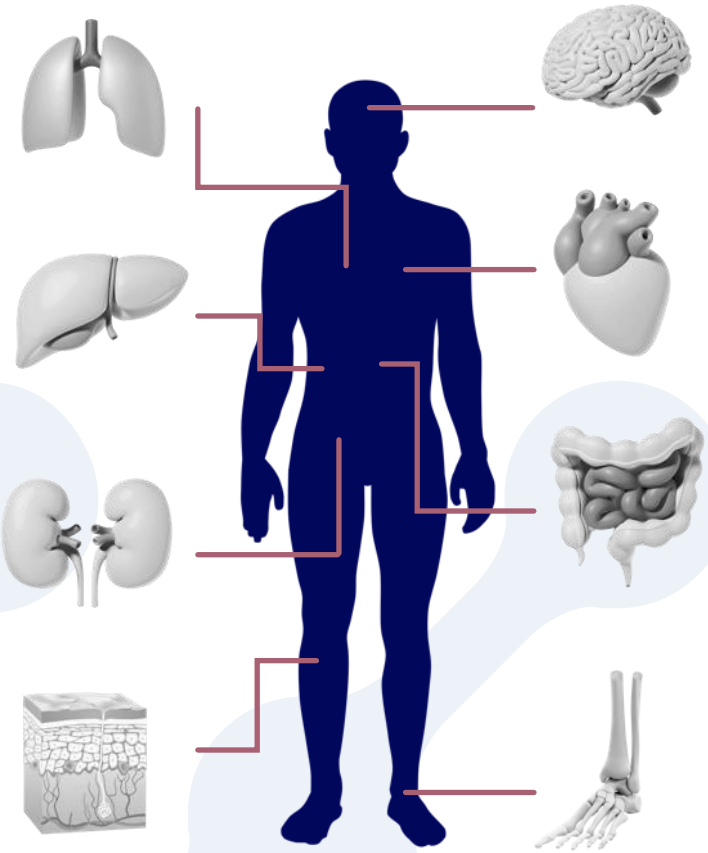
CT-01: Clinical trials in the short term

In vitro and in vivo pharmacology and GLP-toxicology	✓
Drug substance for clinical trials	✓
Documentation for clinical trials	✓
Capsule preparation	**Underway**
CTA submission	**Scheduled for H1**
First patient enrolled	**Autumn 2024**

CT-02: Vast market potential for inflammasome modulators



Allergy
Asthma

Fibrotic diseases

Lupus nephritis

Psoriasis

Neuroinflammation (AD, PD, MS, ALS)

Myocardial infarction
Diabetic cardiomyopathy

Inflammatory Bowel Disease

Gout
Rheumatoid Arthritis

NEK7 degradation inhibits the possibility of inflammasome formation and, consequently, the production of inflammatory cytokines leading to the reduction of symptoms of immune-related diseases

Two series of potent NEK7 degraders:

CPT-513 - systemic therapy for the treatment of **autoimmune disorders**

CPT-101 - therapy of inflammatory **neurodegenerative disorders**

Neuroinflammation and systemic inflammation markets are in excess of $30bn in 2030 each

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CT-02: Spectacular results for two different strategies

Systemic lead compound

Brain-penetrant lead compound

✓

Potent degradation of NEK7 in monkeys for 24h with single dose

✓

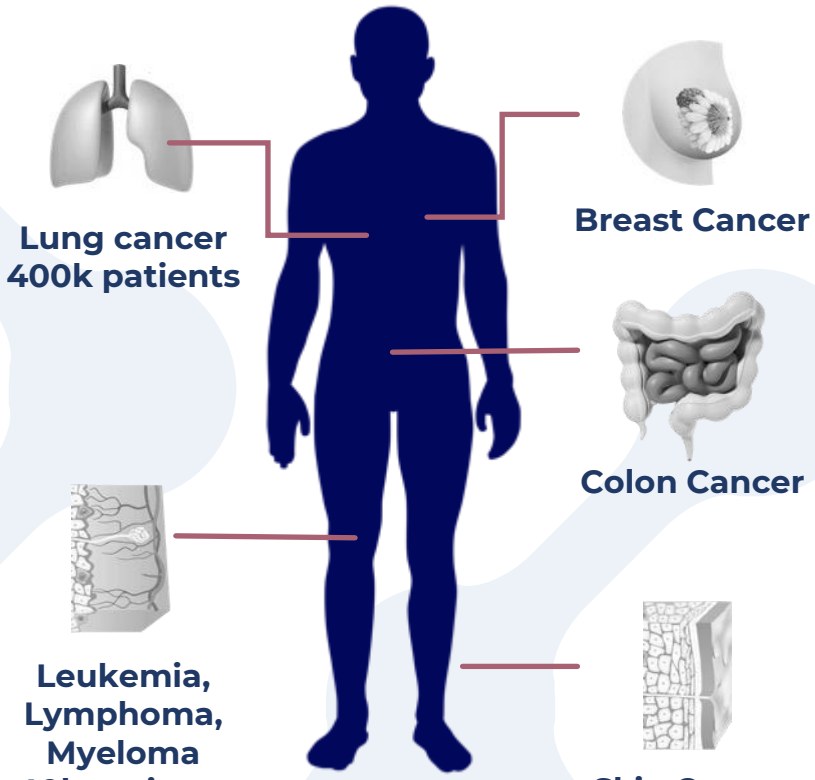
In vivo PoC in systemic model

In vivo PoC in neuroinflammation model

UNDERWAY

Both compounds display excellent drug-like properties and induce rapid and lasting degradation of NEK7 in monkeys

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 hematological and solid tumors

Degradation of inhibition of MCL-1 protein directly attenuates tumors *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

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MCL-1 as a target in numerous anticancer therapies

MCL-1 is a key mechanism of drug resistance in cancer cells

Highly attractive target serving as a major survival signal in numerous cancers

Hematological malignancies

Multiple Myeloma (MM)	Acute Myeloid Leukemia (AML)	Non-Hodgkin Lymphoma (NHL)
$33bn by 2030	$6bn by 2028	$16bn by 2032

Selected solid tumors

Small cell lung cancer (SCLC)	Non-small cell lung cancer (NSCLC)	Triple-negative breast cancer (TNBC)

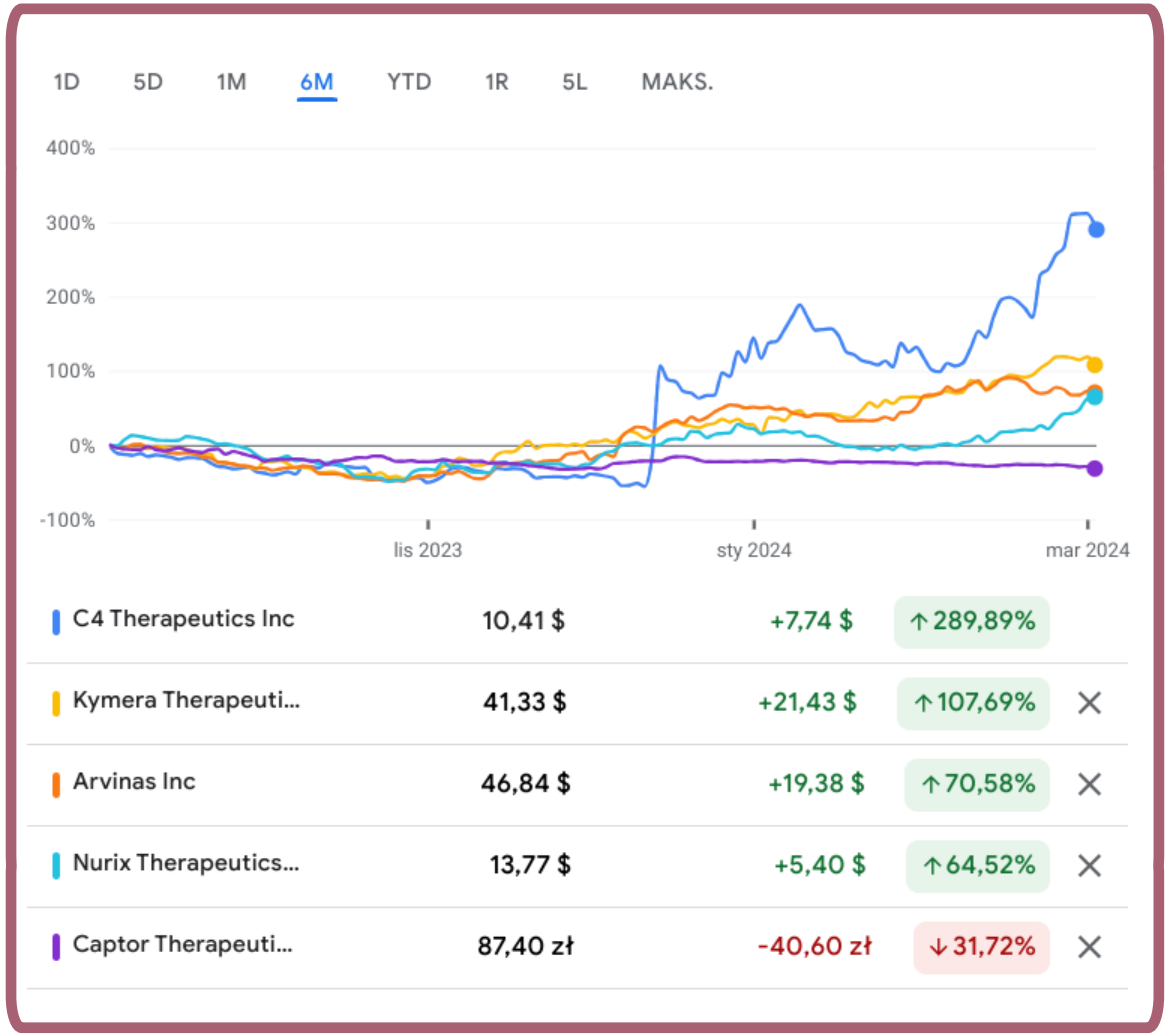
Market value of MCL-1 drugs in liquid tumors is in excess of $50bn in 2030

BCC Research, Spherical Insights

CT-03: Milestones

MCL-1 degraders with very high potency in cancer models *in vitro* and *in vivo*	✓
Cardiosafety *in vivo* in monkeys by troponin level analysis	✓
Early toxicology	**Underway**
IND-enabling studies initiation	**H2 2024**
IND approval	

Steep rebound of XBI and other TPD companies in the last 6 months



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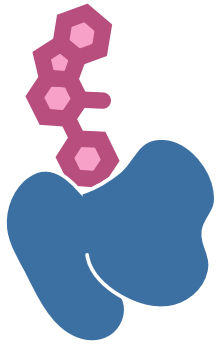
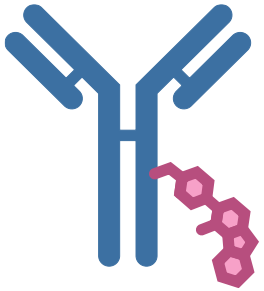
Business opportunities beyond our pipeline

Degraders for new collaborative drug targets
Similar to our partnership with Ono Pharmaceutical, this kind of collaboration can bring non-dilutive funding and build expertise in a new area



Degraders working through novel ligases
Novel ligases have the potential to provide the next generation of degrader drugs. Captor has demonstrated degradation exploiting the KLHDC2 ligase

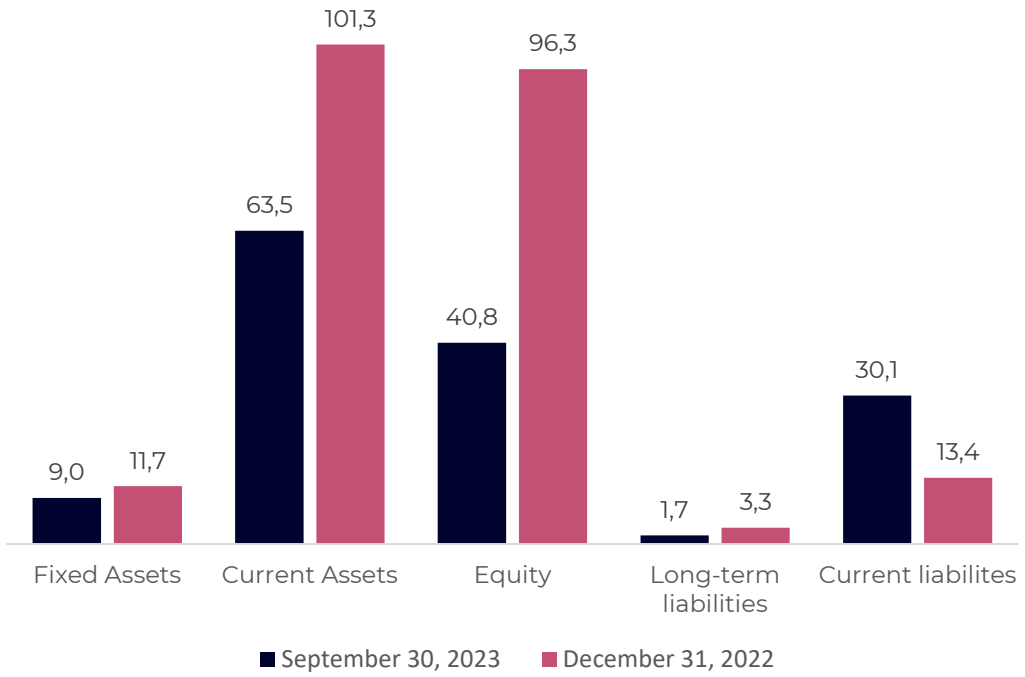
Degrader Antibody Conjugates (DAC)
A very hot area seen as an evolution of ADC drugs.
Many ADC companies are seeking access to degraders that can be used as payloads and coupled to antibodies which target cancer cells



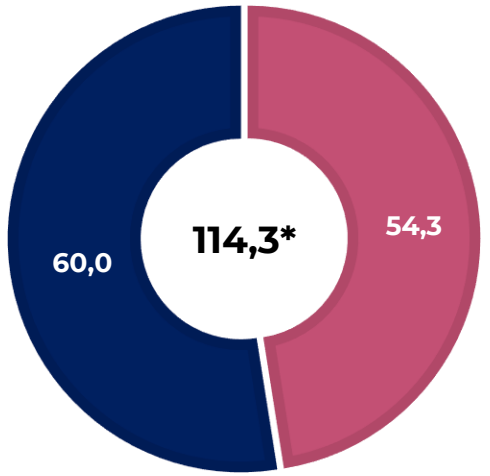
Discussions are underway with companies in all three areas

Strong balance sheet and cash position

Consolidated statement of financial position (PLN, M)



Cash position



Available funding secured
(PLN M; as of September 30, 2023):

Total : PLN 114,3 M*

- PLN 54,3
  cash, short-term bonds
- PLN 60,0
  available grants (NCBR; ABM)

*** The amount does not include PLN 40M from capital raising completed in October 2023 and phasing part from NCBiR**

R&D costs in Q1-Q3 2023**:**

Total : PLN 50,3 M

Cash outflow in Q1-Q3 2023**:**

Total : PLN 32,6 M

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