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Q1 2024 Results

All images by Piotr Piatek

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Fully owned pipeline

Programme	Primary Target	Indications	Modality	Discovery	Preclinical*	IND Filing	Phase IA / IB	Phase II
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*

Assumed stage at the end of 2025

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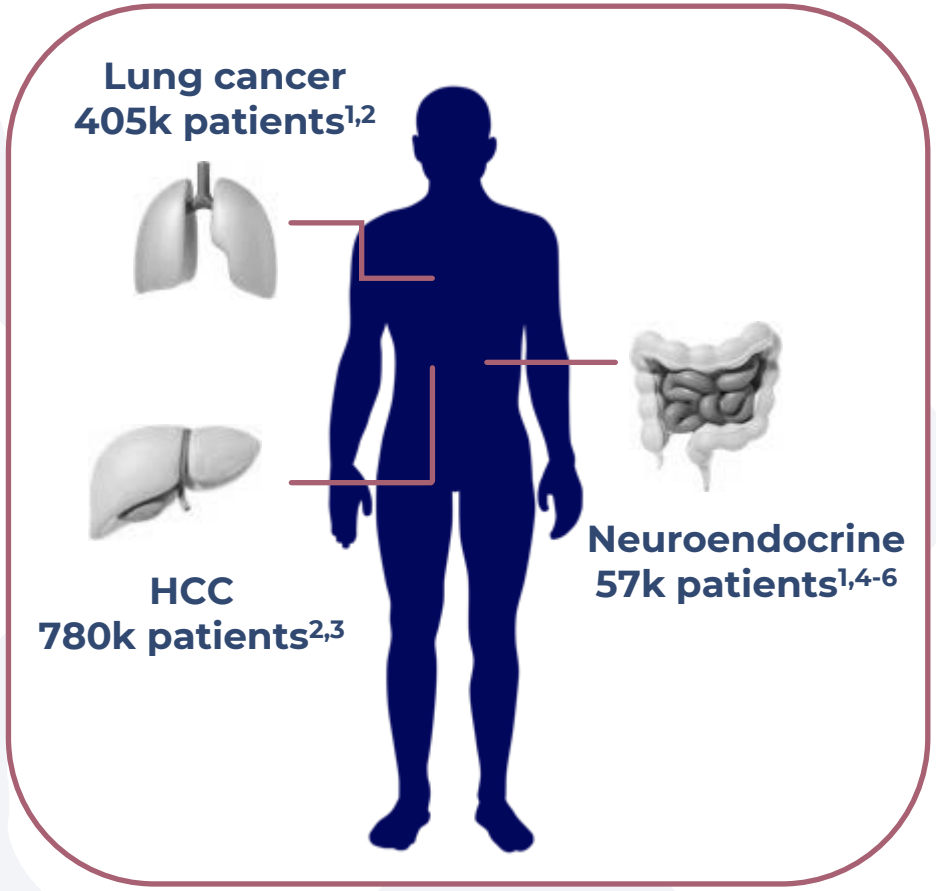
CT-01 (CPT-6281): First-in-Class GSPT1 Targeted Degrader for Hepatocellular Carcinoma (HCC)

CPT-6281 – first-in-class molecular glue degrader for hepatocellular carcinoma



The unique degradation profile of CPT-6281 leads to an Integrated Stress Response (ISR) and induction of apoptosis in HCC cells, while reduction of IL-1β levels in the tumor microenvironment may enable 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, breast and neuroendocrine cancer

(1) Semin Cancer Biol. 2006 Aug;16(4):253-64
(2) https://gco.iarc.fr/today/en/
(3) Nat Rev Dis Primers 7:6 (2021)
(4) Sci Rep. 2021 Apr 12;11(1)
(5) Endocr Connect. 2023 Nov 23;12(12)
(6) JAMA Oncol. 2017 Oct 1;3(10):1335-1342

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HCC treatment with a GSPT1 degrader - status

Molecular Glue

- **Initial indication**
  - Hepatocellular carcinoma
- **Degradation profile**
  - GSPT1, NEK7
  - **Liver and lung activated pro-drug**
- **Strong differentiation from other GSPT1 degraders (BMS, MonteRosa)**
  - Best-in-class degradation profile
  - Tissue restrained prodrug expands therapeutic window
- **Development activities**
  - GLP-tox and GMP manufacturing complete
  - Drug product (capsules): first batches on stability studies
- **Expected milestones in 2024**
  - Clinical Trial Application submission and approval in Europe
  - Initiation of Phase 1 clinical trials in hepatocellular carcinoma in H2

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Development status – CPT-6281

In vitro and *in vivo* pharmacology studies	✓
Drug Substance synthesis optimization and manufacture for tox	✓
MTD/DRF tox studies in rats and NHP	✓
DMPK studies	✓
Appointment of CRO to supervise clinical study	✓
GLP toxicology studies	✓
Drug Substance GMP manufacture	✓
Drug Product development and GMP manufacture	✓
PK & PD assays development for the clinic	**Stability testing**
Investigator's brochure for clinical trials	**Ongoing**
	Final draft

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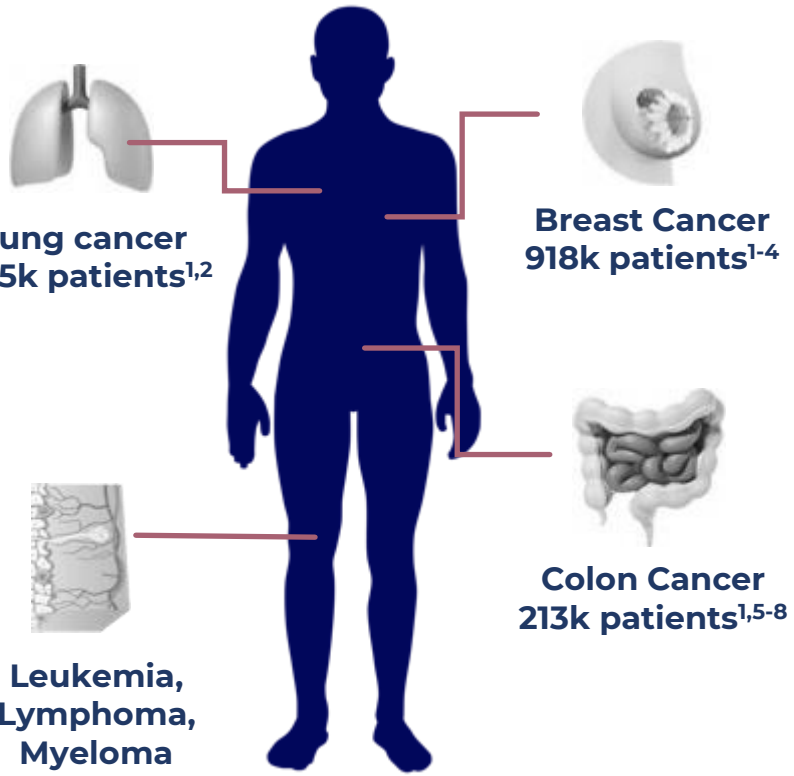
CT-03: First-in-Class MCL-1 Degraders for Liquid & Solid tumors

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 tumors‡

Degradation or 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 MCL-1

Degraders have a different mode of action, without accumulation of MCL-1

Degradation of ~70% of MCL-1 induces apoptosis, while inhibitors require almost 100% of target coverage and cause accumulation of the MCL-1

This, together, with optimized clearance expands the therapeutic window from the perspective of toxicity

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

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MCL-1: a high potential cancer target

Highly attractive target with application in numerous cancer markets

Haematological malignancies

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

Acute Myeloid Leukaemia (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]

Despite years of effort from Pharma, no MCL-1 targeting drug has been approved and several inhibitors have been associated with toxicity

Captor's lead degraders,CPT-908 and CPT-2036 have not shown evidence of cardiotoxicity to date, in keeping with their different mode of action

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

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Strong pharma interest in MCL-1 target – MCL-1 inhibitors

Compound	Company	Program Status	Clinical Trials	Study Size (Pts)
MIK665/ S64315	Novartis/ Servier	**Stopped**	NCT03672695	37
			NCT04702425	37
			NCT02979366	38
AMG 176	Amgen/ Beigene	**Stopped**	NCT02992483	31
			NCT02675452	142
			NCT05209152	9
			NCT03797261	9
AMG 397	Amgen/ Beigene	**Stopped**	NCT03465540	24
AZD5991	Astrazeneca	**Stopped**	NCT03218683	78
ABBV-467	Abbvie	**Stopped**	NCT04178902	8
PRT1419	Prelude Therapeutics	**Stopped**	NCT05107856	21
			NCT04837677	26
			NCT04543305	16
GS-9716	Gilead Sciences	**On-going**	NCT05006794	195e

Several Phase 1 clinical trials were started

To-date, 6 of the 7 MCL-1 inhibitor programs were stopped by companies

For 5 of the 6 compounds stopped, cardiac toxicity or elevated **cardiac troponin I** was observed in the study

Captor Therapeutics has the only MCL-1 degrader we are aware of in development

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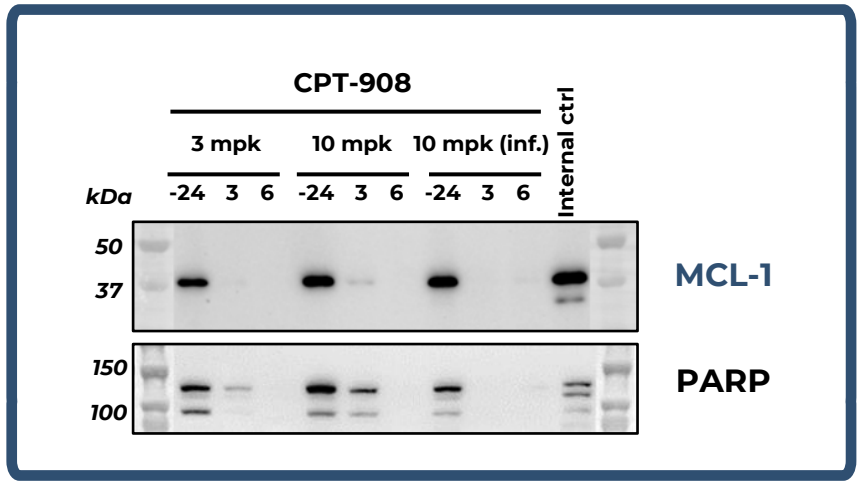
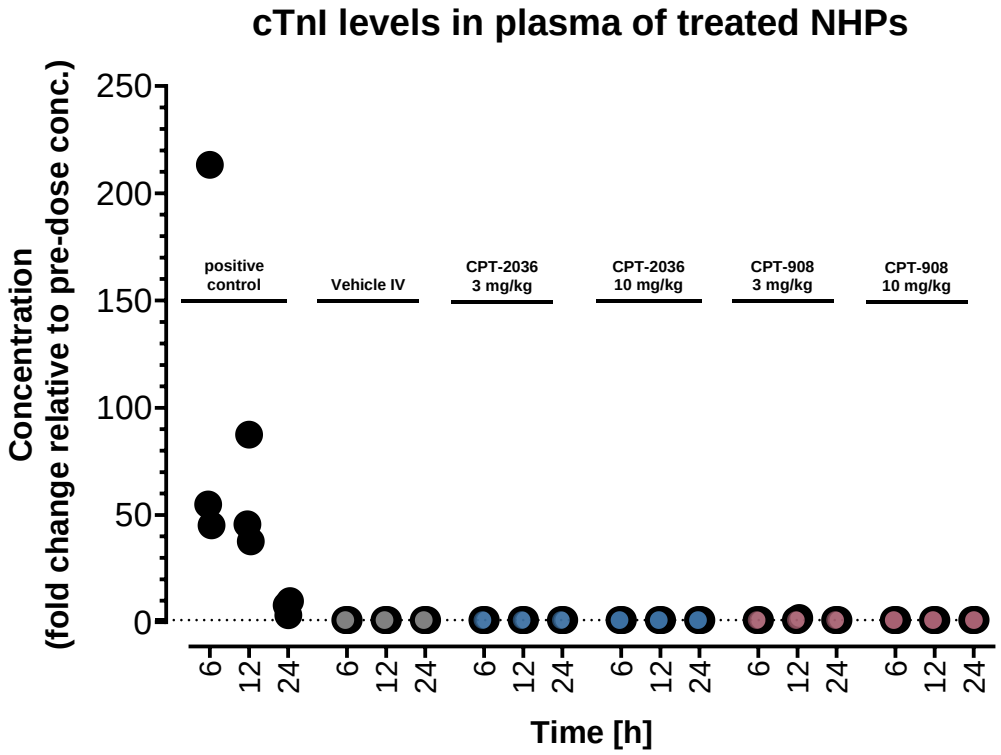
Cardiotoxicity marker Troponin I in plasma of NHPs after MCL-1 degrader dosing





No observed changes in cardiac troponin levels were significantly different from the vehicle control

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

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Status: CT-03

Bifunctional Degrader

- **Initial indications**
  - Blood cancers, subsequently solid tumors
- **Degradation profile**
  - Selective first-in-class MCL-1 degraders
- **Development activities**
  - Efficacy proven *in vivo*
  - MTD completed, DRF ongoing
  - Candidate selection studies underway
  - No indicators of cardiac safety issues
- **Expected milestones**
  - Candidate selection planned for 2024
  - IND-enabling studies 2024

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CT-02: First-in-Class NEK7 Degraders for Autoimmune & Neurodegenerative Diseases

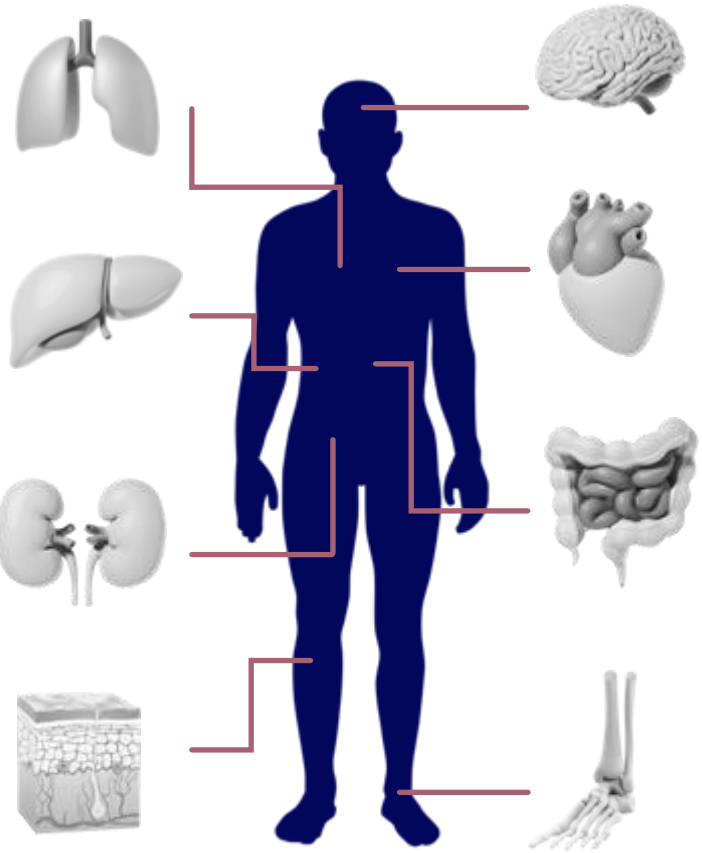
CT-02: Vast market potential for inflammasome modulators

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 inflammasome formation and, consequently, the production of inflammatory cytokines leading to the reduction of symptoms of immune-related diseases.

Recent publications demonstrate the potential role of CNS inflammasome in weight loss

Two series of potent NEK7 degraders:

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

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

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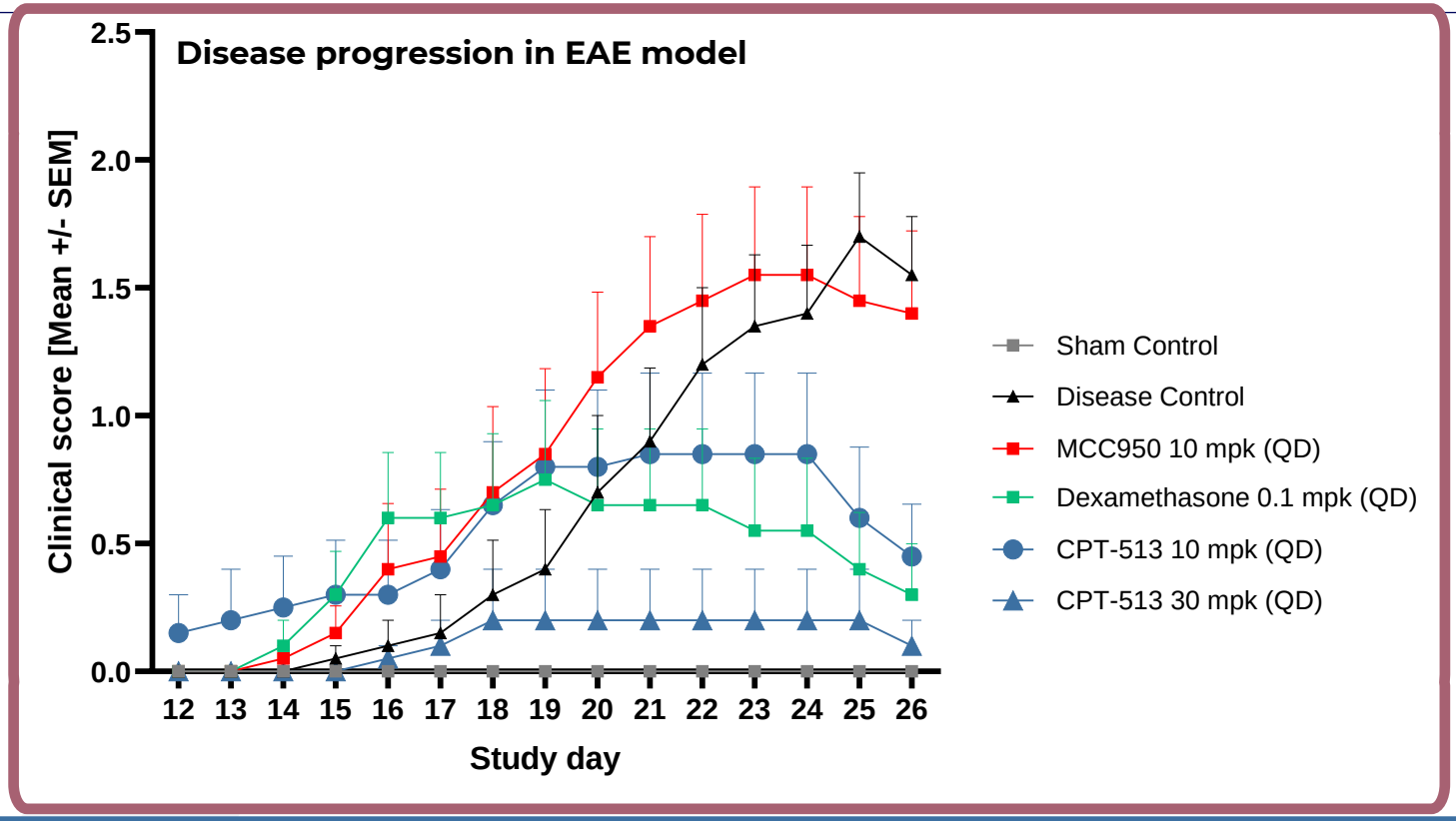
High efficacy of CPT-513 with oral dosing in EAE mouse model *in vivo*



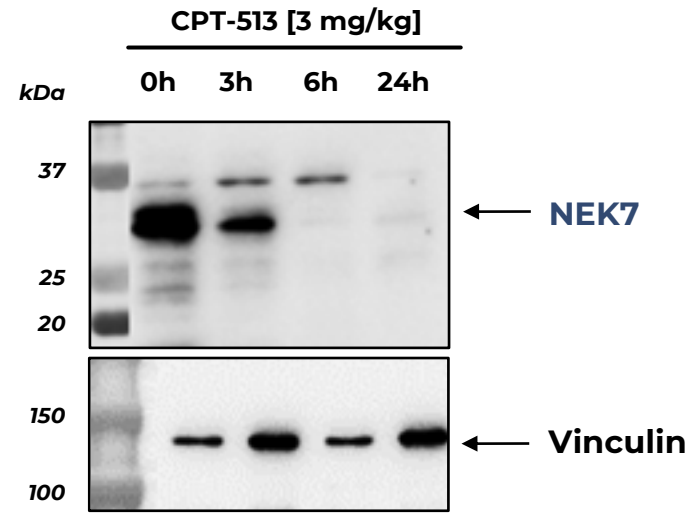
C57BL/6 mice; females; 10 mice in each group;

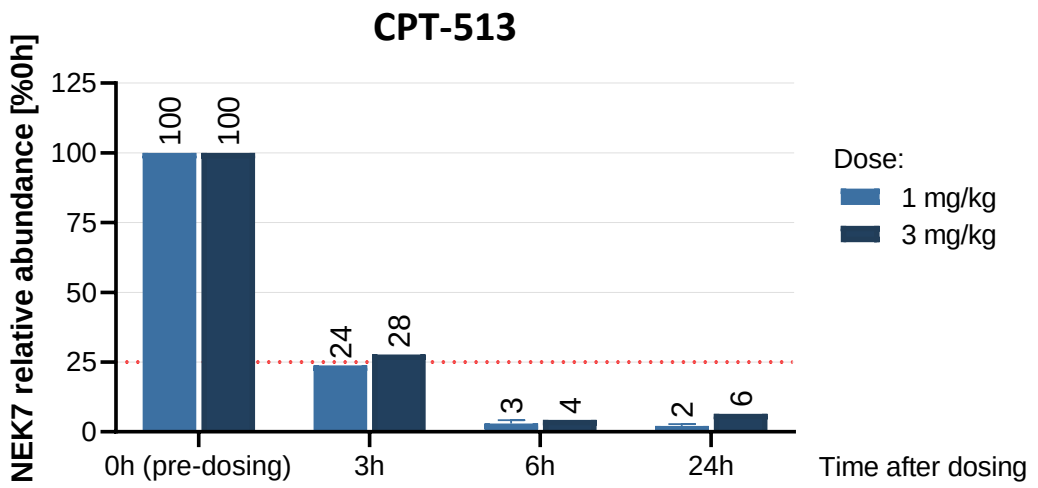
Clinical score/disease index monitored based on a predefined scale including motor skills and tail / limb weakness;

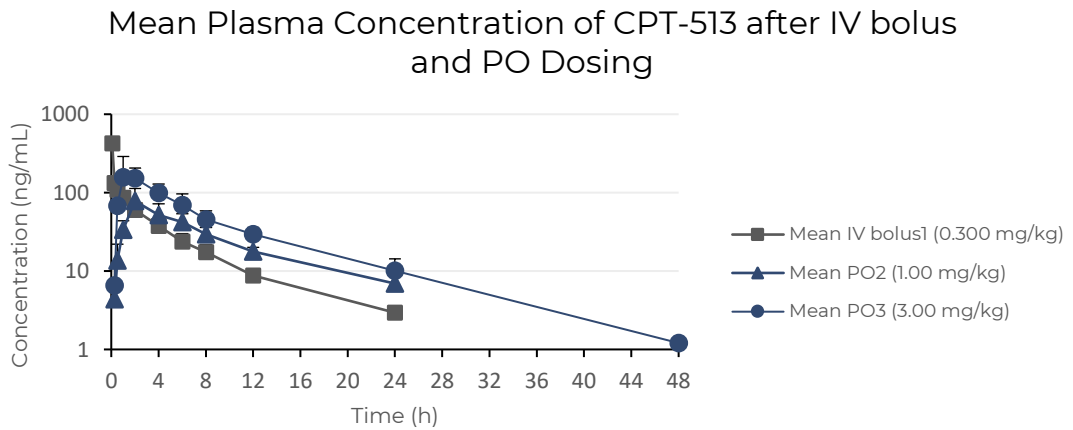
Treatment for 16 days did not induce any side effects; body weight in the groups treated with NEK7 degraders was higher than in the group treated with Dexamethasone.

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

CPT-513 efficiently covers and degrades NEK7 in NHPs







NEK7 degradation is observed in monkey PBMCs isolated at 3, 6, and 24 h after CPT-513 PO [1 & 3 mg/kg] administration

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CT-02: Excellent degraders from two different strategies

Two series of potent NEK7 degraders - in **autoimmune diseases** (CPT-513) and **neurodegenerative disorders** (CPT-101, brain-penetrant series)

Activity confirmed both *in vitro* on 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 show excellent drug-like properties

In vivo proof of efficacy in disease models

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Business Development

Business development update

- The recent compelling data on our differentiated pipeline drug candidates and the increase in our BD resources has resulted in increased activity
- The *in-vivo* proof of concept data in CT-02 is a key trigger for partnering discussions that is now met
- For example, at the recent BIO conference in San Diego, we had business meetings with 37 different companies and investors, including 9 of the top 10 global pharma companies, as well as leading Asian pharma companies interested in regional rights to our products
- The number of discussions under confidentiality agreements has increased during Q1
- These discussions may, or may not lead to transactions, but we are confident that the increased activity is a sign of the attractiveness of our unique pipeline

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Finance Highlights

Highlights from the financial results in first quarter 2024

- Increase in revenues from collaborations from PLN 1,5M to PLN 4,5M
- Decrease in grant revenues from PLN 2,7M to PLN 1,5M
- Narrowing loss from PLN 14,2M to PLN 9,5M due to a focus on lead projects, the timing of expenditure on CT-01 costs, and lower employee benefit costs

- After the end of the reporting period, signature of new grant phasing agreements for both CT-03 & CT-01 for a total value of PLN 11,7M

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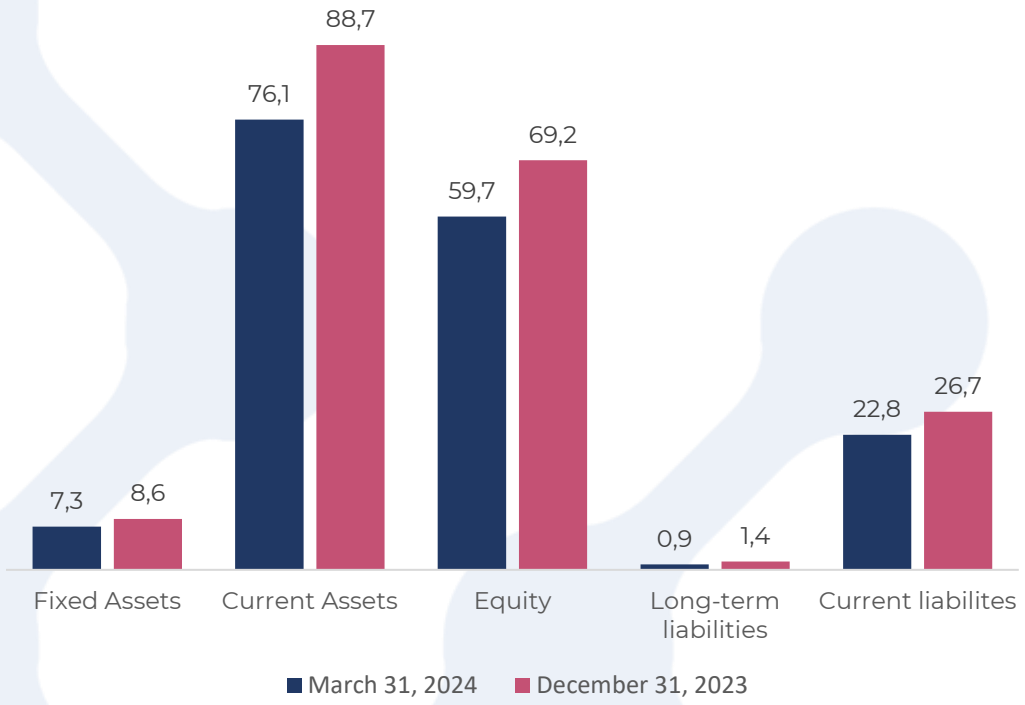
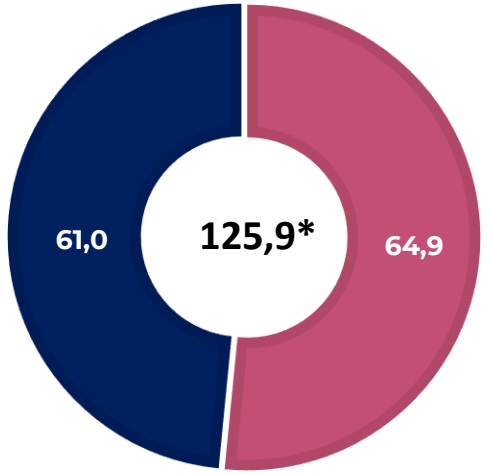
Balance sheet and cash position

Consolidated statement of financial position (PLN, M)



Cash position



Available funding secured (PLN M; as of March 31, 2024):

Total : PLN 125,9 M*

- PLN 64,9 M cash
- PLN 61,0 M available grants (NCBR; ABM)

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

R&D costs in Q1 2024**:**

Total : PLN 13,0 M

Net Operational Cash Outflow in Q1 2024**:**

Total : PLN 10,3 M

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